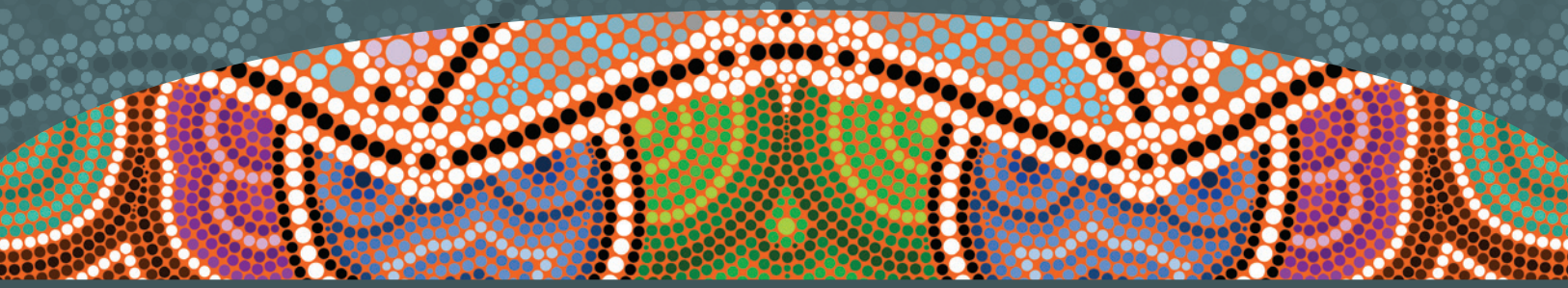
THIRD EDITION

MEDICAL-SURGICAL NURSING

CRITICAL THINKING FOR PERSON-CENTRED CARE

LEMONE • BURKE • BAULDOFF • GUBRUD • LEVETT-JONES • HALES
BERRY • CARVILLE • DWYER • KNOX • MOXHAM • RAYMOND • REID-SEARL

VOLUMES 1–3



ALWAYS LEARNING

PEARSON

THIRD AUSTRALIAN EDITION

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VOLUMES 1–3

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This book contains photographs of people from Aboriginal and Torres Strait Islander communities. It may cause distress to some Indigenous Australians to view photographs of any of these people who may have subsequently died.

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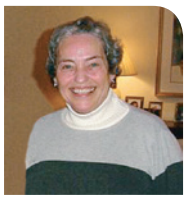
ABOUT THE **AUTHORS**



PRISCILLA LEMONE-KOEPLIN RN, DSN, FAAN

Priscilla LeMone-Koeplin spent most of her career as a nurse educator, teaching medical–surgical nursing and pathophysiology at all levels from diploma to doctoral students. She has a diploma in nursing from Deaconess College of Nursing (St Louis, Missouri), baccalaureate and master's degrees from Southeast Missouri State University, and a doctorate in nursing from the University of Alabama-Birmingham. She is retired as an Associate Professor Emeritus, Sinclair School of Nursing, University of Missouri, but continues to keep up to date in nursing both as an Adjunct Associate Professor at the Ohio State University, College of Nursing and as an author of nursing textbooks.

Dr LeMone-Koeplin won numerous awards for scholarship and teaching during her more than 30 years as a nurse educator. She is most honoured for receiving the Kemper Fellowship for Teaching Excellence from the University of Missouri, the Unique Contribution Award from the North American Nursing Diagnosis Association, and for being selected as a Fellow in the American Academy of Nursing. Dr LeMone-Koeplin currently lives in Ohio. She enjoys travelling, gardening, knitting and reading fiction.

She believes that her education gave her solid and everlasting roots in nursing. Her work with students has given her the wings that have allowed her love of nursing and teaching to continue through the years.

KAREN M. BURKE RN, MS

Karen Burke has been a nurse educator for much of her career, teaching basic and advanced medical–surgical nursing and pathophysiology. She retired as director of the nursing and health occupations programs at Clatsop Community College in Astoria, Oregon, subsequently serving as nursing education consultant and program manager for the Oregon State Board of Nursing. She currently provides consulting services for nursing and higher education.

Ms Burke earned her diploma in nursing from Emanuel Hospital School of Nursing in Portland, Oregon, later completing baccalaureate studies at Oregon Health & Science University, and a master's degree in nursing at the University of Portland. She has been actively involved in nursing education and clinical nursing education reform, as well as in initiatives to address the nursing faculty shortage. Ms Burke is co-author of another text, *Medical–Surgical Nursing Care* (3rd edition), with Elaine Mohn-Brown and Linda Eby.

Ms Burke highly values the nursing profession and the importance of providing a strong education in the art and science of nursing for students preparing to enter the profession, no matter which educational path is being pursued.

GERENE BAULDOFF RN, PhD, FAAN

Gerene Bauldoff is a Professor of Clinical Nursing at The Ohio State University College of Nursing in Columbus, Ohio. She has been a nurse educator for 14 years, teaching medical–surgical nursing, translational science and evidence-based practice courses at the baccalaureate, master's, and doctoral levels. Prior to her nursing educator role, her clinical background included home health nurse, lung transplant coordinator and pulmonary rehabilitation coordinator. Dr Bauldoff has a diploma from the Western Pennsylvania Hospital School of Nursing, Pittsburgh, Pennsylvania, and a BSN from LaRoche College in Pittsburgh. Her graduate education is from the University of Pittsburgh, with an MSN in medical–surgical nursing (cardiopulmonary clinical nurse specialist) and a PhD in nursing in 2001, training under Leslie Hoffman, PhD, RN, FAAN.

Dr Bauldoff is an active member of multiple professional organisations including the American Academy of Nursing (AAN), Sigma Theta Tau International Honor Society of Nursing, the American Association of Cardiovascular and Pulmonary

Rehabilitation (AACVPR), the American Thoracic Society Nursing Assembly and the American College of Chest Physicians (ACCP). She is a recognised expert in medical–surgical nursing, focusing on the care of the patient with chronic pulmonary disease, serving on committees focusing on patient-centred outcomes in pulmonary rehabilitation. She has been honoured with fellowships in the AAN, AACVPR and ACCP. In 2013, Dr Bauldoff was the keynote speaker at the Nanning Nursing Education Conference in Nanning, China, attended by representatives from more than 100 nursing schools in China.

Dr Bauldoff views nursing as the greatest profession, using scientific evidence to provide the highest quality of care while maintaining the personal relationship with patients and their families. Her experiences provide her with insights and lessons learned that she shares with her students.

Dr Bauldoff resides in central Ohio. She enjoys travelling, walking, bicycling, golf and spending time with her family and friends.

PAULA GUBRUD-HOWE RN, MS, EdD, FAAN

Paula Gubrud-Howe is Senior Associate Dean for Academic Affairs and an Associate Professor at Oregon Health and Science University (OHSU) School of Nursing. She has more than 25 years of experience as a nurse educator involving multiple levels of programs from LPN to doctoral education. Dr Gubrud-Howe is a founding leader and co-director of the Oregon Consortium for Nursing Education, an award-winning consortium that includes the five campuses of OHSU and nine community colleges. She also has more than 20 years of experience in medical–surgical nursing, critical care, home health and hospice. Dr Gubrud-Howe earned a baccalaureate degree in nursing from Walla Walla University (1980), an MS in community-based nursing from OHSU (1993) and an EdD in post-secondary education from Portland State University (2008). She is a frequently invited speaker at national and international nursing education conferences and consults with other states and countries on the development of competency-based curricula and nursing education consortiums designed to promote academic progression in nursing education. Her research activity is focused on clinical education redesign and the integration of simulation into nursing curricula.

Dr Gubrud-Howe is passionate about nursing and the opportunities it provides to members of the profession. She values the sacred relationship nurses experience with patients as they promote health, treat illness and provide comfort and palliative care. She believes the nation's health depends on highly qualified nurses who are dedicated to lifelong learning in pursuit of evidence-based, patient-centred care.

TRACY LEVETT-JONES RN, PhD, MEd & Work, BN, DipAppSc(Nur)

Professor Tracy Levett-Jones is the Deputy Head of School (Teaching and Learning), School of Nursing and Midwifery, and the Director of the Research Centre for Health Professional Education at the University of Newcastle. Her research interests include clinical reasoning, interprofessional education, empathy, belongingness, cultural competence, simulation and patient safety. Tracy has authored 10 books, the most recent being *Clinical reasoning: Learning to think like a nurse* and *Critical conversations for patient safety*, as well as over 150 book chapters, reports and peer-reviewed journal articles. Tracy has been the recipient of nine research awards and 10 teaching awards including an Australian Learning and Teaching Council (ALTC) Award for Teaching Excellence (2010), a NSW Minister for Education and Training Quality Teaching Award (2007) and a Pearson/Australian Nurse Teacher Society Nurse Educator of the Year Award (2011). She has been awarded nearly $2 million in grant funding and has led and been involved in a number of Category 1 funded projects.

TRUDY DWYER RN, PhD, MClinEdu, GCFlexLearn, BHlthScn(Nsg), NrCert, ICUNsgCert

Trudy Dwyer completed a hospital-based nursing program at the Rockhampton Hospital, Queensland, where she developed an enthusiasm for critical care nursing and travel. Trudy currently holds the position of Associate Professor (Research Intensive) at CQUniversity. She has extensive experience coordinating undergraduate courses/programs and research higher-degree supervision. Her research interests include patient safety, recognition of deterioration, resuscitation and simulation. She has published in international refereed scholarly journals, book chapters and is the co-author of the highly successful *Student nurse: Clinical survival guide*.

LORNA MOXHAM RN, PhD (CQU), MHN, DAS(Nsg) (MIHE), Med (UNSW), GCOH&S (CQU), GCQualMgmt (CQU), BHSc (UWS), Cert IV (Training & Assessment) (CQIT), FACMHN, FACON

Professor Lorna Moxham is a 3-year specialist hospital-trained psychiatric nurse. She completed her BHSc at the University of Western Sydney and is passionate about the nursing profession, actively contributing at regional, state, national and international levels. Lorna has spent time in regional Queensland, regional New South Wales and metropolitan New South Wales and has served on many ministerial committees as a member and chair. In addition, she has held several leadership and governance roles, both within the tertiary education sector and in nursing. Lorna is currently the inaugural Professor of Mental Health Nursing and also the leader of the Living Well, Longer theme in the Global Challenges Program at the University of Wollongong. Lorna has successfully supervised numerous higher degree by research students to on-time completion, all of whom have published their work. Lorna thinks of being a nurse as 'the very best job in the world'.

KERRY REID-SEARL RN, RM, PhD, BHSc (Nsg), MClinEdu, MRCNA, FCN

Kerry Reid-Searl is a Professor in the School of Nursing and Midwifery at CQUniversity. Kerry is a current clinician in the area of paediatrics. She has a long career as a nurse academic and has been involved in the undergraduate degree for 25 years. Kerry is also a supervisor of master's and PhD research students. Kerry's research interests include patient safety, simulation, paediatrics and wound care. Kerry has been the recipient of 10 teaching awards including two Australian Learning and Teaching Citations for her outstanding contribution to student learning and an Australian University Teaching Excellence Award. She was named Pearson/Australian Nurse Teacher Society Nurse Teacher of the Year in 2009 and in 2013 received the Simulation Australia Achievement award. Kerry has become known nationally and internationally for her pioneering work in creating, designing and researching two innovative simulation strategies called Mask Ed (KRS Simulation) and Pup Ed (KRS Simulation).

KAMAREE BERRY RN, PhD candidate, MEd Stds (Hon), PGDip Clinical Nursing (Perioperative), BN, MACN, RAA

Kamaree's career spans more than 20 years and commenced with the Australian Army where she trained as a medic and then became a nursing officer at the rank of lieutenant. She has been employed in the public, private and academic sectors in a number of roles spanning from an RN, senior lecturer and academic undergraduate chair to General Manager, Learning.

A successful career has provided her with the opportunity to teach at both undergraduate and postgraduate levels, along with employment within the corporate and commercial sector at a national level. Underpinning Kamaree's philosophy and values of learning, development and professional education is her strong conviction of innovative leadership and direction for both staff and students. She values the importance of continuing professional education and development; this has been acknowledged by her peers making use of her extensive expertise and contemporary approach to teaching and learning, curriculum development and review, program designs, staff and student mentorship, and currency on educational trends within the nursing profession across a number of specialties. She has been awarded a Bachelor of Nursing, Postgraduate Diploma Clinical Nursing—Perioperative, a Master of Educational Studies (Hons) and has completed her doctoral thesis.

KERYLN CARVILLE PhD, RN, STN (Cred)

Professor Primary Health Care & Community Nursing

Silver Chain Group and Curtin University, Western Australia.

Keryln has extensive clinical experience and is committed to research and education within the domains of wound and ostomy care. She was appointed a Fellow of the Australian Wound Management Association (now Wounds Australia) in 2006. She is Chair of the Australian Pressure Injury Advisory Panel and Chair Pan Pacific Pressure Injury Alliance, Chair of the Wounds Australia Wound Standards Committee and sits on the International Wound Infection Institute Committee. Keryln was awarded the Western Australia Health Lifetime Achievement Award for Nursing in 2010.

MAJELLA HALES RN, MAppSci, GCHE, BN

Majella Hales works as a casual academic at the Australian Catholic University in Brisbane. Originally hospital trained, she has worked in nursing for over 25 years. She maintains her clinical experience by undertaking agency shifts in critical care units across South-East Queensland and provides clinical facilitation for undergraduate nursing students for various local universities. Majella authored several chapters of Kozier & Erb's *Fundamentals of nursing* Volumes 1–3 and co-authored *Principles of pathophysiology* with Associate Professor Shane Bullock. Along with journal articles and conference presentations, she has also produced the skills DVD for Tollefson's *Clinical psychomotor skills* text and adapted the American case study resource *The neighbourhood*. Majella is a co-owner of Sciencopia, a company producing informative, novel and fun educational resources for academics, students and healthcare professionals. One of Sciencopia's first products—*Essential Aussie drugs: A little pocket book of common Aussie drug facts*—is a great resource for assisting individuals to improve their drug knowledge and safety in the ever-changing and complex world of pharmacology and drug administration.

NICOLE KNOX RN, BN, MN, GDipAdultEd, GCICU

Nicole Knox has been working in nursing for 20 years, including roles as a nursing academic, clinical nurse specialist, clinical educator and nurse unit manager.

The major part of Nicole's nursing career has been in critical care nursing, particularly intensive care, for which she completed a graduate certificate. She enjoys clinical education and this guided her decision to pursue a career in academia. She has worked in several universities in Sydney where her roles have included unit coordination, teaching and research. She is currently a sessional academic at Western Sydney University. She has a love of education and values the opportunity to introduce the next generation of nurses to the profession.

DEBRA RAYMOND RN, MCP, BHlthSc, DipHealthScNsg

Debra Raymond has been nursing since she was 15 years of age, starting in a nursing home as an Assistant in Nursing. She then completed her enrolled nursing aide course, worked as an EN and was encouraged to do further studies, from which she gained a Diploma of Health Science Nursing, Bachelor of Health Science and Master of Clinical Practice. She received an International Nurses Day award which supported her travels to France to present her research projects at the Emergency Medicine Conference.

The major part of Debra's nursing career has been in critical care nursing. Debra's enjoyment of education prompted her decision to enter academia. She then started facilitating nursing students in the clinical setting. She has continued in education and is now an Associate Lecturer. Debra is also a PhD candidate and unit coordinator for an undergraduate unit.

Debra believes that her career progression in the nursing profession has given her an appreciation of the endless opportunities in this wonderful, and at times challenging, profession. Her love for education and the wish to make a difference in nursing have guided her throughout her career.

Debra is married, and with the support of her husband and wonderful, talented daughter she will continue pursuing her goal of making a difference in her chosen nursing profession.

CONTRIBUTORS

We extend deep, sincere thanks to our contributors who gave their time, effort and expertise so willingly to the development and writing of chapters and resources that help foster our goal of achieving nursing excellence through building clinical competence.

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PREFACE

This is a wonderful time to become a nurse in Australia, with opportunities that are exciting and far reaching. As always, there is a need for committed nurses whose practice is based on a strong foundation of knowledge, clinical skills and the ability to think critically in challenging and complex situations. This book has been written to help you develop these professional attributes and skills.

This third Australian edition of *Medical–surgical nursing: Critical thinking for person-centred care* will help prepare you for your nursing journey. It will challenge you to excel and support you as you learn.

Throughout this book, we demonstrate how competent nurses provide person-centred care that is empathetic, holistic and individualised, and is respectful of the person's age, ethnicity, culture and psychosocial status.

Our goal—helping you to excel as a nurse

In writing this book, our commitment has been to provide students with a strong knowledge base, an understanding of contemporary practice issues in Australia and the capacity for sound clinical reasoning. These professional attributes will allow you to provide nursing care that is safe and effective. This easily understood, straightforward Australian edition integrates the following concepts: epidemiology, pathophysiology, pharmacology, legal and ethical issues, therapeutic communication, interprofessional communication and cultural competence. The use of effective design principles and learning strategies such as advanced organisers, special features, colourful illustrations and critical thinking exercises will support your learning and application to practice.

This textbook has been designed to:

- emphasise a person-centred philosophy whereby the person who is the recipient of care is seen as an integral member of the team and consideration of their needs and wishes is paramount
- foster critical thinking and clinical reasoning skills as the basis for nursing excellence in clinical practice
- recognise the nurse's role as an essential member of the interprofessional healthcare team.

We are confident that this text will support your learning and professional practice, and we wish you well as you undertake your nursing journey.

Organisation

The book contains 52 chapters in 14 units. Units 1 and 2 provide an overview of medical–surgical nursing, the meaning of health and illness, and alterations in patterns of health in particular populations, contexts and situations. The remaining units are based on alterations in human structure and function. Each unit has a focus on altered health states and opens with an assessment chapter, which draws upon the student's prerequisite knowledge and serves to reinforce basic principles of anatomy and physiology as applied to assessment in both health and illness. Following the assessment chapter, each nursing care chapter focuses on major conditions and diseases and includes three key components:

1. ***Pathophysiology*** The discussion of each *major* illness or condition begins with incidence and prevalence, an overview of pathophysiology and disease manifestations and complications.
2. ***Interprofessional care*** The role of both nurses and the other members of the healthcare team in managing illness is then profiled. This section includes information about specific tests necessary for diagnosis, medications, surgery and treatments, fluid management, dietary management and complementary and alternative therapies.
3. ***Nursing care*** Nursing care within a context of priority nursing diagnoses and interventions is then provided and rationales outlined for each intervention. This section also takes into account that health promotion and illness prevention are critical nursing roles in contemporary healthcare.

Finally, for each major disorder or condition, a narrative *Nursing care plan* is provided with a brief case study, followed by the steps of the nursing process.

Chapter highlights This end-of-chapter section concludes with multiple-choice revision questions to reinforce comprehension of the chapter content. (The correct answers with rationales are found in the Instructors' Manual.)

Language and terminology

In developing this text we have used terminology that is familiar and applicable to most Australians. While person-centred care is most often used to reflect our philosophical stance, the term 'patient' is also used as appropriate throughout the text and according to the context of care being described.

Indigenous Australians

Throughout the text we have integrated issues relevant to the Indigenous Australian population. In covering these issues we have acknowledged the importance of using non-discriminatory and appropriate language to describe groups of people, policies and events, and have thus followed the guidelines set out by NSW Health in its publication *Communicating positively: A guide to appropriate Aboriginal terminology*.

Nursing diagnoses

In this updated edition of *Medical–surgical nursing* we refer to the well-known nursing process as a logical approach to managing nursing care. Within this process we refer to diagnostic terminologies that are typical of those used by Australian nurses.

What's new in the third edition

This edition of *Medical–surgical nursing* has two new features:

1. National Patient Safety Standards from the Australian Commission on Quality and Health Care. The relevant standards have been added to the chapters where applicable as they relate to patient safety .
2. Translation to practice boxes. These boxes focus on research into specific topics and how this relates to current nursing care and the application of evidence in clinical settings.

Other changes include:

- Chapter 1 'Medical–surgical nursing' includes a more detailed explanation of how to write a nursing diagnosis.
- All chapters have been updated with newer research and evidence-based practice throughout; Chapter 35 'Nursing care of people with ventilation disorders' has been updated with newer research with reference to the emerging zoonotic viral pneumonias SARS and MERS.
- Chapter 51 'Community care' (previously chapter 3) now resides In Unit 14 'Special topics in medical–surgical nursing'.

Visual engagement and accuracy

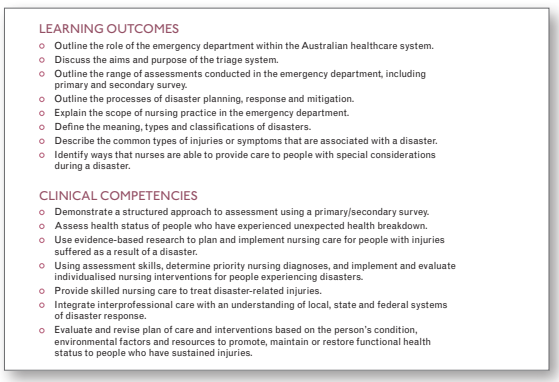
The authors understand the importance of not only making the text visually engaging but also ensuring that any visual representations accurately reflect nursing in Australia. For this reason the photographs featured in *Medical–surgical nursing* have been carefully selected to ensure that they accurately depict Australian nursing equipment, uniforms, clinical settings, processes and procedures.

Culturally competent nursing

Chapter 1 introduces the concept of culturally competent healthcare with particular attention to the culture and history of Indigenous peoples in Australia. *Focus on cultural diversity* boxes present cultural nursing in context and highlight the importance of acknowledging the dignity, culture, values, beliefs and rights of not only Indigenous Australians but also people from all cultural and ethnic backgrounds. These themes are threaded throughout the text, with direct reference to the Nursing and Midwifery Board of Australia's (NMBA) *Registered Nurse Standards for Practice (2016)*.

GUIDED **TOUR**

We carefully reviewed the US edition of this book to ensure current content and the necessary knowledge to educate the next generation of nurses in Australia. Key features of the Australian edition include:



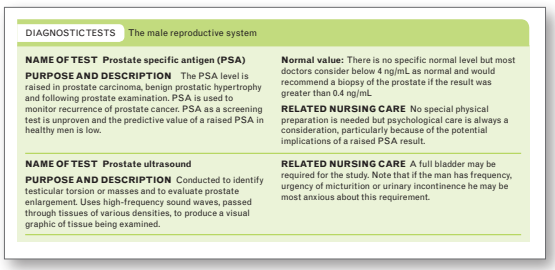
LEARNING OUTCOMES

- Outline the role of the emergency department within the Australian healthcare system.
- Discuss the aims and purpose of the triage system.
- Outline the range of assessments conducted in the emergency department, including primary and secondary survey.
- Outline the processes of disaster planning, response and mitigation.
- Explain the scope of nursing practice in the emergency department.
- Define the meaning, types and classifications of disasters.
- Describe the common types of injuries or symptoms that are associated with a disaster.
- Identify ways that nurses are able to provide care to people with special considerations during a disaster.

CLINICAL COMPETENCIES

- Demonstrate a structured approach to assessment using a primary/secondary survey.
- Assess health status of people who have experienced unexpected health breakdown.
- Use evidence-based research to plan and implement nursing care for people with injuries suffered as a result of a disaster.
- Using assessment skills, determine priority nursing diagnoses, and implement and evaluate individualised nursing interventions for people experiencing disasters.
- Provide skilled nursing care to treat disaster-related injuries.
- Integrate interprofessional care with an understanding of local, state and federal systems of disaster response.
- Evaluate and revise plan of care and interventions based on the person's condition, environmental factors and resources to promote, maintain or restore functional health status to people who have sustained injuries.

Learning Outcomes show you the knowledge you'll gain, while **Clinical Competencies** demonstrate how you will apply that knowledge.



DIAGNOSTIC TESTS The male reproductive system

NAME OF TEST **Prostate specific antigen (PSA)**

PURPOSE AND DESCRIPTION The PSA level is raised in prostate carcinoma, benign prostatic hypertrophy and following prostate examination. PSA is used to monitor recurrence of prostate cancer. PSA as a screening test is unproven and the predictive value of a raised PSA in healthy men is low.

Normal value: There is no specific normal level but most doctors consider below 4 ng/mL as normal and would recommend a biopsy of the prostate if the result was greater than 0.4 ng/mL

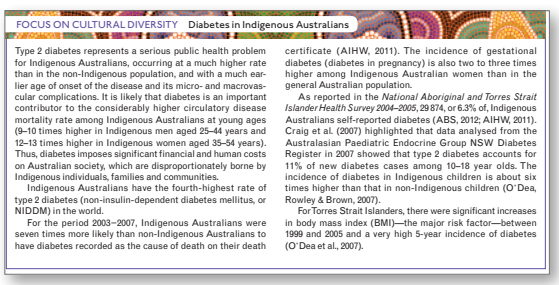
RELATED NURSING CARE No special physical preparation is needed but psychological care is always a consideration, particularly because of the potential implications of a raised PSA result.

NAME OF TEST **Prostate ultrasound**

PURPOSE AND DESCRIPTION Conducted to identify testicular torsion or masses and to evaluate prostate enlargement. Uses high-frequency sound waves, passed through tissues of various densities, to produce a visual graphic of tissue being examined.

RELATED NURSING CARE A full bladder may be required for the study. Note that if the man has frequency, urgency of micturition or urinary incontinence he may be most anxious about this requirement.

Diagnostic Tests include diagnostic test tables and a narrative summary. The tables include the name of the test, the purpose and description of the test, and related nursing care.



FOCUS ON CULTURAL DIVERSITY Diabetes in Indigenous Australians

Type 2 diabetes represents a serious public health problem for Indigenous Australians, occurring at a much higher rate than in the non-Indigenous population, and with a much earlier age of onset of the disease and its micro- and macrovascular complications. It is likely that diabetes is an important contributor to the considerably higher circulatory disease mortality rate among Indigenous Australians at young ages (9–10 times higher in Indigenous men aged 25–44 years and 12–13 times higher in Indigenous women aged 35–54 years). Thus, diabetes imposes significant financial and human costs on Australian society, which are disproportionately borne by Indigenous individuals, families and communities.

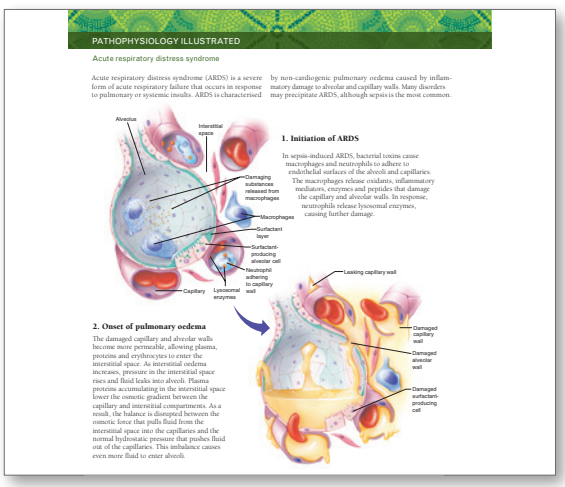
Indigenous Australians have the fourth-highest rate of type 2 diabetes (non-insulin-dependent diabetes mellitus, or NIDDM) in the world.

For the period 2003–2007, Indigenous Australians were seven times more likely than non-Indigenous Australians to have diabetes recorded as the cause of death on their death certificate (AIHW, 2011). The incidence of gestational diabetes (diabetes in pregnancy) is also two to three times higher among Indigenous Australian women than in the general Australian population.

As reported in the *National Aboriginal and Torres Strait Islander Health Survey 2004–2005*, 29 874, or 6.3% of, Indigenous Australians self-reported diabetes (ABS, 2012; AIHW, 2011). Craig et al. (2007) highlighted that data analysed from the Australasian Paediatric Endocrine Group NSW Diabetes Register in 2007 showed that type 2 diabetes accounts for 11% of new diabetes cases among 10–18 year olds. The incidence of diabetes in Indigenous children is about six times higher than that in non-Indigenous children (O'Dea, Rowley & Brown, 2007).

For Torres Strait Islanders, there were significant increases in body mass index (BMI)—the major risk factor—between 1999 and 2005 and a very high 5-year incidence of diabetes (O'Dea et al., 2007).

Focus on Cultural Diversity boxes demonstrate how culture, age and gender produce differences in incidence, prevalence and mortality.



PATHOPHYSIOLOGY ILLUSTRATED

Acute respiratory distress syndrome

Acute respiratory distress syndrome (ARDS) is a severe form of acute respiratory failure that occurs in response to pulmonary or systemic insults. ARDS is characterised by non-cardiogenic pulmonary oedema caused by inflammatory damage to alveolar and capillary walls. Many disorders may precipitate ARDS, although sepsis is the most common.

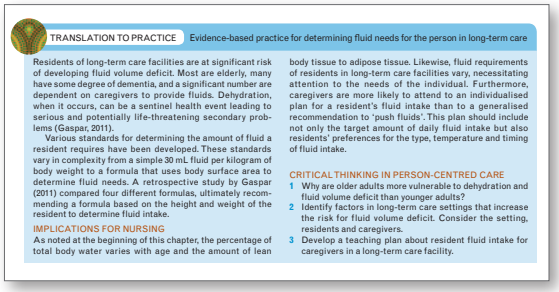
1. Initiation of ARDS

In sepsis-induced ARDS, bacterial toxins cause macrophages and neutrophils to adhere to endothelial surfaces of the alveoli and capillaries. The macrophages release oxidants, inflammatory mediators, enzymes and peptides that damage the capillary and alveolar walls. In response, neutrophils release lysosomal enzymes, causing further damage.

2. Onset of pulmonary oedema

The damaged capillary and alveolar walls become more permeable, allowing plasma, proteins and erythrocytes to enter the interstitial space. As interstitial oedema increases, pressure in the interstitial space rises and fluid leaks into alveoli. Plasma proteins accumulating in the interstitial space lower the osmotic gradient between the capillary and interstitial compartments. As a result, the balance is disrupted between the osmotic force that pulls fluid from the interstitial space into the capillaries and the normal hydrostatic pressure that pushes fluid out of the capillaries. This imbalance causes even more fluid to enter alveoli.

Pathophysiology Illustrated art brings physiological processes to life.



TRANSLATION TO PRACTICE Evidence-based practice for determining fluid needs for the person in long-term care

Residents of long-term care facilities are at significant risk of developing fluid volume deficit. Most are elderly, many have some degree of dementia, and a significant number are dependent on caregivers to provide fluids. Dehydration, when it occurs, can be a sentinel health event leading to serious and potentially life-threatening secondary problems (Gaspar, 2011).

Various standards for determining the amount of fluid a resident requires have been developed. These standards vary in complexity from a simple 30 mL fluid per kilogram of body weight to a formula that uses body surface area to determine fluid needs. A retrospective study by Gaspar (2011) compared four different formulas, ultimately recommending a formula based on the height and weight of the resident to determine fluid intake.

IMPLICATIONS FOR NURSING

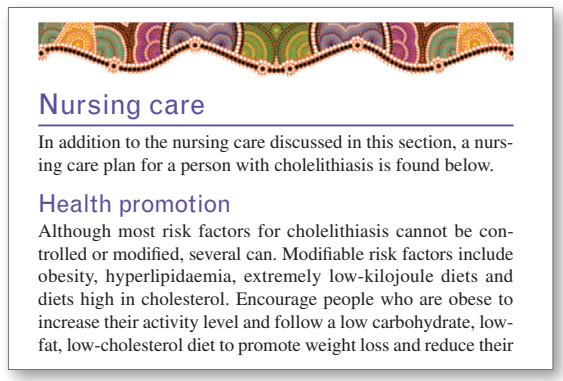
As noted at the beginning of this chapter, the percentage of total body water varies with age and the amount of lean body tissue to adipose tissue. Likewise, fluid requirements of residents in long-term care facilities vary, necessitating attention to the needs of the individual. Furthermore, caregivers are more likely to attend to an individualised plan for a resident's fluid intake than to a generalised recommendation to 'push fluids'. This plan should include not only the target amount of daily fluid intake but also residents' preferences for the type, temperature and timing of fluid intake.

CRITICAL THINKING IN PERSON-CENTRED CARE

1 Why are older adults more vulnerable to dehydration and fluid volume deficit than younger adults?
2 Identify factors in long-term care settings that increase the risk for fluid volume deficit. Consider the setting, residents and caregivers.
3 Develop a teaching plan about resident fluid intake for caregivers in a long-term care facility.

Translation to Practice boxes focus on how research relates to current nursing care and application of evidence in clinical settings.

Nursing Care sections detail the assessment and planning aspects relating to specific conditions and outlines potential pain and risks.

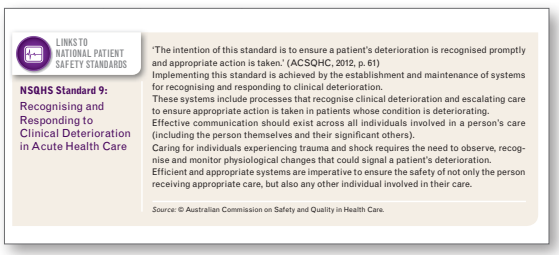


Nursing care

In addition to the nursing care discussed in this section, a nursing care plan for a person with cholelithiasis is found below.

Health promotion

Although most risk factors for cholelithiasis cannot be controlled or modified, several can. Modifiable risk factors include obesity, hyperlipidaemia, extremely low-kilojoule diets and diets high in cholesterol. Encourage people who are obese to increase their activity level and follow a low carbohydrate, low-fat, low-cholesterol diet to promote weight loss and reduce their



LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 9:
Recognising and Responding to Clinical Deterioration in Acute Health Care

'The intention of this standard is to ensure a patient's deterioration is recognised promptly and appropriate action is taken.' (ACSQHC, 2012, p. 61)

Implementing this standard is achieved by the establishment and maintenance of systems for recognising and responding to clinical deterioration.

These systems include processes that recognise clinical deterioration and escalating care to ensure appropriate action is taken in patients whose condition is deteriorating.

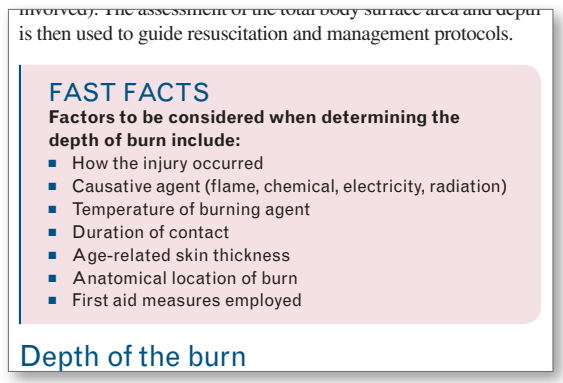
Effective communication should exist across all individuals involved in a person's care (including the person themselves and their significant others).

Caring for individuals experiencing trauma and shock requires the need to observe, recognise and monitor physiological changes that could signal a patient's deterioration.

Efficient and appropriate systems are imperative to ensure the safety of not only the person receiving appropriate care, but also any other individual involved in their care.

Source: © Australian Commission on Safety and Quality in Health Care.

Links to National Patient Safety Standards boxes appear in the chapters where applicable to demonstrate how concepts relate back to patient safety standards.



involved). The assessment of the total body surface area and depth is then used to guide resuscitation and management protocols.

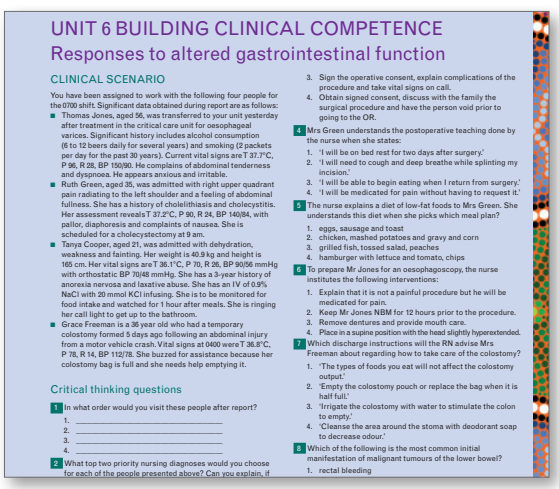
FAST FACTS

Factors to be considered when determining the depth of burn include:

- How the injury occurred
- Causative agent (flame, chemical, electricity, radiation)
- Temperature of burning agent
- Duration of contact
- Age-related skin thickness
- Anatomical location of burn
- First aid measures employed

Depth of the burn

Fast Facts boxes highlight and summarise important data about the prevalence and incidence of selected disorders in Australia, and of other featured content.



UNIT 6 BUILDING CLINICAL COMPETENCE

Responses to altered gastrointestinal function

CLINICAL SCENARIO

You have been assigned to work with the following four people for the 0700 shift. Significant data obtained during report are as follows:

- Thomas Jones, aged 56, was transferred to your unit yesterday after treatment in the critical care unit for oesophageal varices. Significant history includes alcohol consumption (6 to 12 beers daily for several years) and smoking (2 packets per day for the past 30 years). Current vital signs are T 37.7°C, P 96, R 28, BP 150/90. He complains of abdominal tenderness and dyspnoea. He appears anxious and irritable.
- Ruth Green, aged 35, was admitted with right upper quadrant pain radiating to the left shoulder and a feeling of abdominal fullness. She has a history of cholelithiasis and cholecystitis. Her assessment reveals T 37.2°C, P 90, R 24, BP 140/84, with pallor, diaphoresis and complaints of nausea. She is scheduled for a cholecystectomy at 9 am.
- Tanya Cooper, aged 21, was admitted with dehydration, weakness and fainting. Her weight is 40.9 kg and height is 165 cm. Her vital signs are T 36.1°C, P 70, R 26, BP 90/56 mmHg with orthostatic BP 70/48 mmHg. She has a 3-year history of anorexia nervosa and laxative abuse. She has an IV of 0.9% NaCl with 20 mmol KCl infusing. She is to be monitored for food intake and watched for 1 hour after meals. She is ringing her call light to get up to the bathroom.
- Grace Freeman is a 36 year old who had a temporary colostomy formed 5 days ago following an abdominal injury from a motor vehicle crash. Vital signs at 0400 were T 36.8°C, P 78, R 14, BP 112/78. She buzzed for assistance because her colostomy bag is full and she needs help emptying it.

Critical thinking questions

1 In what order would you visit these people after report?
1. _____
2. _____
3. _____
4. _____

2 What top two priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if

3. Sign the operative consent, explain complications of the procedure and take vital signs on call.
4. Obtain signed consent, discuss with the family the surgical procedure and have the person void prior to going to the OR.

4 Mrs Green understands the postoperative teaching done by the nurse when she states:
1. 'I will be on bed rest for two days after surgery.'
2. 'I will need to cough and deep breathe while splinting my incision.'
3. 'I will be able to begin eating when I return from surgery.'
4. 'I will be medicated for pain without having to request it.'

5 The nurse explains a diet of low-fat foods to Mrs Green. She understands this diet when she picks which meal plan?
1. eggs, sausage and toast
2. chicken, mashed potatoes and gravy and corn
3. grilled fish, tossed salad, peaches
4. hamburger with lettuce and tomato, chips

6 To prepare Mr Jones for an oesophagoscopy, the nurse institutes the following interventions:
1. Explain that it is not a painful procedure but he will be medicated for pain.
2. Keep Mr Jones NBM for 12 hours prior to the procedure.
3. Remove dentures and provide mouth care.
4. Place in a supine position with the head slightly hyperextended.

7 Which discharge instructions will the RN advise Mrs Freeman about regarding how to take care of the colostomy?
1. 'The types of foods you eat will not affect the colostomy output.'
2. 'Empty the colostomy pouch or replace the bag when it is half full.'
3. 'Irrigate the colostomy with water to stimulate the colon to empty.'
4. 'Cleanse the area around the stoma with deodorant soap to decrease odour.'

8 Which of the following is the most common initial manifestation of malignant tumours of the lower bowel?
1. rectal bleeding

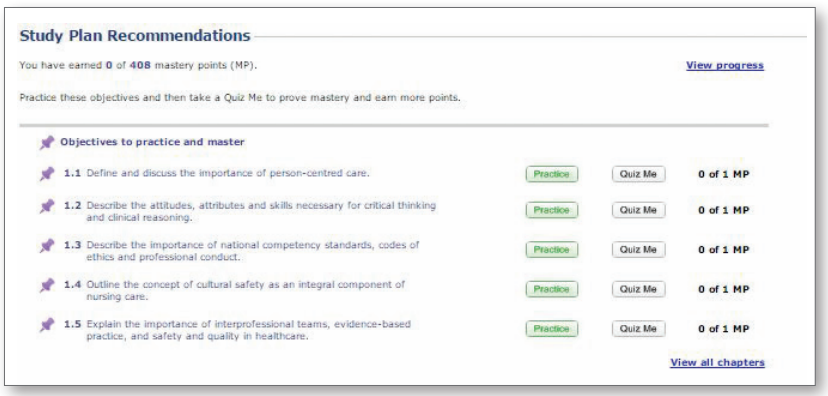
An end-of-unit review for each of the units, called **Building Clinical Competence**, synthesises what you have learned in the unit and applies the knowledge to specific cases. The feature includes:

- A **clinical scenario** involving a priority issue reflection piece that synthesises the underlying concepts and includes a variety of questions that allow students to apply different skills.
- A **case study** with concept map that further synthesises material using the nursing process.

STUDENT AND EDUCATOR **SUPPORT**

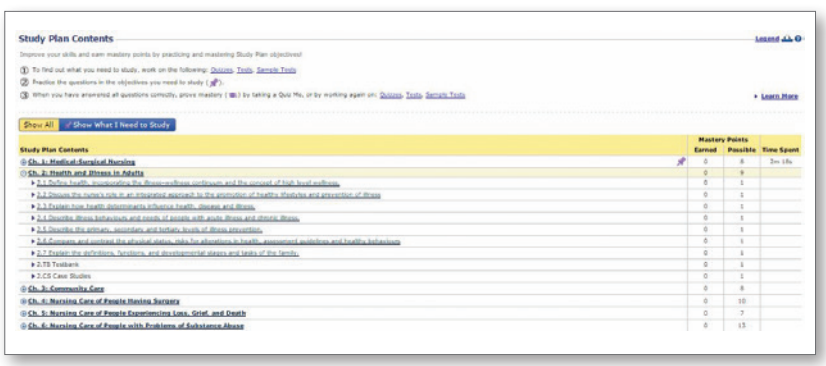
MyNursingLab for Medical–Surgical Nursing 3e

A guided tour for students and educators



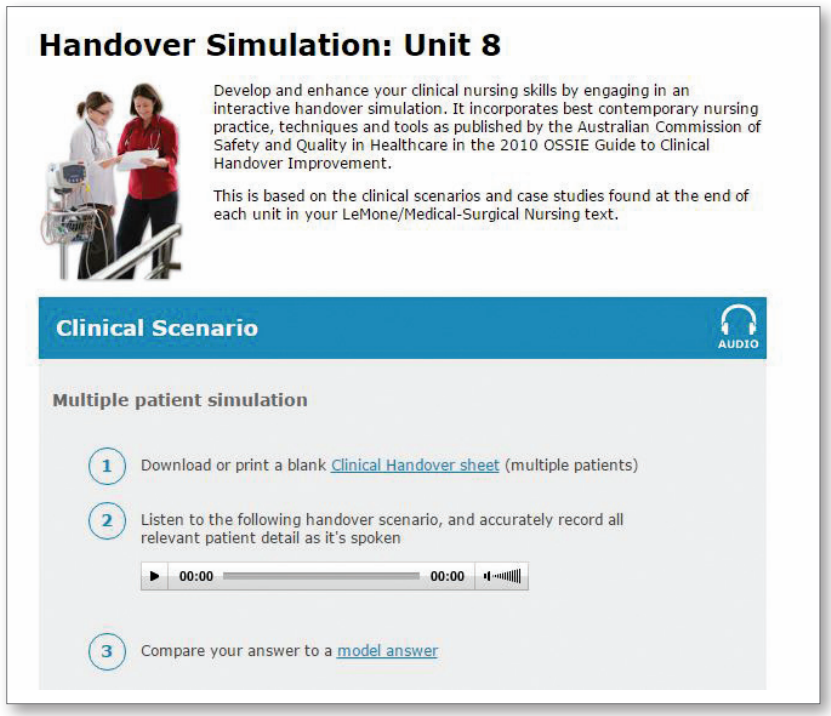
Study Plan: A study plan, tagged to the revised NMBA Standards allows students to clearly see which topics they have mastered and, more importantly, which they need to work on.

Practice: MyNursingLab comes with pre-loaded assignments covering in-chapter content, all of which are automatically graded.



Learning Resources: The following links to additional learning resources are incorporated into the Study Plan or Multimedia Library:

- the relevant section of the eText, so students can review key concepts
- video, simulation and case studies which ensure that each individual is able to comprehend the course material and apply it to real-world scenarios.



Educator resources

A suite of resources is provided to assist with delivery of the text, as well as to support teaching and learning:

- *Instructor's Manual.* This manual provides educators with detailed, accuracy-verified solutions to the in-chapter problems in the book.
- *Test Bank.* The Test Bank provides a wealth of accuracy-verified testing material. Updated for this new edition, each chapter offers a wide variety of question types arranged by learning objective and tagged by NMBA Standards. Each Test Bank question can also be assigned to students and auto-graded in MyNursingLab.
- *Digital Image PowerPoint Slides.* All the diagrams and tables from the text are available for lecturer use in chapter-based PowerPoint slides.

ACKNOWLEDGEMENTS

A collaborative adventure

Medical–surgical nursing: Critical thinking for person-centred care, third Australian edition, is the result of an exciting and productive collaboration between academic staff and clinicians across Australia. Authors and reviewers alike have been generous in sharing their expertise and crafting a textbook that is uniquely Australian and directly relevant to contemporary practice. The following people are to be acknowledged for carefully reviewing the chapters and providing a thoughtful and constructive critique:

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MAPPING TO THE NMBA *REGISTERED NURSE STANDARDS FOR* ***PRACTICE***

AUTHOR: Trish Burton

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
Unit 1 Dimensions of Medical–Surgical Nursing			
1	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Employs clinical reasoning in applying the nursing process to knowledgeable, safe, person-centred, culturally safe care, in Table 1.1 Using critical thinking in the nursing process, p. 5
		1.2. Develops practice through reflection on experiences, knowledge, actions, feelings and beliefs to identify how these shape practice	Employs the Clinical Reasoning Cycle to inform nursing practice, in the Clinical Reasoning Cycle, p. 7
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Uses the nursing process as a model of patient care, in Table 1.1 Using critical thinking in the nursing process, p. 5
	5. Develops a plan for nursing practice	5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Uses the nursing process as a model of patient care, in Table 1.1 Using critical thinking in the nursing process, p. 5
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Uses the nursing process as a model of patient care, in Table 1.1 Using critical thinking in the nursing process, p. 5
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Uses the nursing process as a model of patient care, in Table 1.1 Using critical thinking in the nursing process, p. 5
2	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Conducts an assessment that is sensitive to the risk factors of family developmental stages and tasks to person-centred care, in Table 2.10 Family-related risk factors for alterations in health, p. 31
	2. Engages in therapeutic and professional relationships	2.1. Communicates effectively, and is respectful of a person's dignity, culture, values, beliefs and rights	Is sensitive to the associated risk factors of family developmental stages and tasks for the person and family, in Table 2.10 Family-related risk factors for alterations in health, p. 31

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts an assessment that is sensitive to the physical changes in the older adult years to promote, restore and maintain health when planning and implementing care, in Table 2.8 Physical changes in the older adult years, p. 28
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Considers the physical changes in the older adult years in planning the care of the person, in Table 2.8 Physical changes in the older adult years, p. 28 Considers the risk factors of family developmental stages and associated tasks in planning the care of the person, in Table 2.10 Family-related risk factors for alterations in health, p. 31
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Is cognisant of the physical changes in the older adult years in implementing care of the person, in Table 2.8 Physical changes in the older adult years, p. 28 The risk factors of family developmental stages and associated tasks are considered during the care of the person, in Table 2.10 Family-related risk factors for alterations in health, p. 31
Unit 2 Alterations in Patterns of Health			
3	1. Thinks critically and analyses nursing practice	1.4. Complies with legislation, regulations, policies, guidelines and other standards or requirements relevant to the context of practice when making decisions	Complies with the legal requirement of patient safety in the preoperative phase, pp. 52, 55, 56
		1.5. Uses ethical frameworks when making decisions	Is aware of advocate role in relation to person-informed consent, in Legal requirements, p. 37
	2. Engages in therapeutic and professional relationships	2.5. Advocates on behalf of people in a manner that respects the person's autonomy and legal capacity	Facilitates informed consent for the person, in Legal requirements, p. 37
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Notifies anaesthetist of all prescribed and over-the-counter drugs as surgical risk factors in care of the person, in Table 3.2 Nursing implications for surgical risk factors, p. 40 Works closely with the surgeon during the intraoperative and postoperative phases, p. 56
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Considers the assessment required for surgical risk factors in person-centred care, in Table 3.2 Nursing implications for surgical risk factors, pp. 38–39

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans for the nursing implications for surgical risk factors in person-centred care, in Table 3.2 Nursing implications for surgical risk factors, pp. 38–39
			Ensures that the safety guidelines and checklists in the preoperative phase are included in the plan of care, pp. 52, 55, 56
			Includes in the plan that the older adult has an increased risk of complications in the postoperative period, in Table 3.6 Nursing interventions for older people having surgery, p. 61
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Adheres to safety guidelines and completes checklists in the preoperative phase, pp. 52, 55, 56
			Nursing interventions are in response to the older adult having an increased risk of complications in the postoperative period, in Table 3.6 Nursing interventions for older people having surgery, p. 61
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates completed checklists in the preoperative phase, pp. 52, 55, 56
			Evaluates interventions to reduce the increased risk of complications in the postoperative period, in Table 3.6 Nursing interventions for older people having surgery, p. 61
4	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Promotes trust in the therapeutic relationship with the person experiencing loss and grief, in Nursing care plan, pp. 81–82
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Makes assessments for appropriate nursing interventions, in Box 4.3 Providing comfort for the person nearing death, p. 76
			Provides comprehensive nursing care for the person experiencing loss and grief, in Nursing care plan, pp. 81–82
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans for appropriate nursing interventions, in Box 4.3 Providing comfort for the person nearing death, p. 76
			Plans for comprehensive nursing care for the person experiencing loss and grief, in Nursing care plan, pp. 81–82
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Implements appropriate nursing interventions, in Box 4.3 Providing comfort for the person nearing death, p. 76
			Provides comprehensive nursing care for the person experiencing loss and grief, in Nursing care plan, pp. 81–82

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates appropriate nursing interventions, in Box 4.3 Providing comfort for the person nearing death, p. 76
			Evaluates comprehensive nursing care for the person experiencing loss and grief, in Nursing care plan, pp. 81–82
5	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses, and uses the best available evidence, that includes research findings, for safe, quality practice	Uses evidence-based research to plan and implement nursing care for people experiencing withdrawal symptoms of tobacco use, in Translation to practice, p. 90
	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Refers person to healthcare professionals to assist with nicotine withdrawal, in Table 5.3 Guide for the management of nicotine-dependent inpatients, p. 89
			As part of collaborative care, consults and plans interventions in conjunction with the doctor and dietitian about management during alcohol withdrawal, in Nursing care plan, p. 107
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Assesses for nicotine withdrawal, in Table 5.3 Guide for the management of nicotine-dependent inpatients, p. 89; Translation to practice, p. 90
			Assesses for alcohol withdrawal, in Nursing care plan, p. 107
	5. Develops a plan for nursing practice	5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Plans interventions for nicotine withdrawal, in Translation to practice, p. 90
			Plans specific interventions (stress management, coping skills, nutrition, relapse prevention and healthy lifestyle choices) for alcohol withdrawal, in Nursing care plan, p. 107
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides interventions for nicotine withdrawal, in Table 5.3 Guide for the management of nicotine-dependent inpatients, p. 89; Translation to practice, p. 90
			Provides needs-specific nursing interventions for alcohol abuse and medical conditions, in Box 5.1 Principles of nursing care in relation to alcohol, p. 92
			Provides interventions for alcohol withdrawal, in Nursing care plan, p. 107
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates interventions for nicotine withdrawal, in Table 5.3 Guide for the management of nicotine-dependent inpatients, p. 89
			Evaluates interventions for alcohol withdrawal, in Nursing care plan, p. 107

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
6	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	In the disaster setting, communicates Disaster triage category, in Table 6.3 Disaster triage system, by category, p. 116
	4. Comprehensively conducts assessments	4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Initially assesses the person using the primary survey, in Table 6.3 Disaster triage system, by category, p. 116 Assesses the person with trauma, in Nursing care Plan, pp. 124–125
	5. Develops a plan for nursing practice	5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Provides comprehensive nursing care for a person with trauma, in Nursing care Plan, pp. 124–125
		5.5. Coordinates resources effectively and efficiently for planned actions	In the disaster setting, plans the use of resources, in Table 6.3 Disaster triage system, by category, p. 116
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides comprehensive nursing care for a person with trauma, in Nursing care plan, pp. 124–125
	7. Evaluates outcomes to inform nursing practice	7.1 Evaluates and monitors progress towards the expected goals and outcomes	Evaluates comprehensive nursing care for a person with trauma, in Nursing care plan, pp. 124–125
Unit 3 Pathophysiology and Patterns of Health			
7	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health assessment and maps pedigree, in Box 7.11 Adult indicators for a referral to a genetic specialist, p. 151
	5. Develops a plan for nursing practice	5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Develops a nursing care plan, in Box 7.11 Adult indicators for a referral to a genetic specialist, p. 151 Develops a nursing care plan, including integrating genetic concepts into education for people and their families, in Education, p. 151
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Delivers nursing care, in Box 7.11 Adult indicators for a referral to a genetic specialist, p. 151; Education, p. 151
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates a nursing care plan, in Box 7.11 Adult indicators for a referral to a genetic specialist, p. 151
8	1. Thinks critically and analyses nursing practice	1.5. Uses ethical frameworks when making decisions	Ensures the person is an active participant in planning nursing care, in Nursing care, pp. 176–177
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Conducts education to promote pain relief, in Medication administration, p. 170

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a nursing assessment in relation to pain, in Nursing care, pp. 172–175 Makes assessments for a nursing care plan, in Nursing care, pp. 176–177
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Conducts a nursing assessment, in Nursing care, pp. 172–175
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Plans for administering medication safely and effectively, in Medication administration, p. 170 Develops a nursing care plan, in Nursing care, pp. 176–177 Revises plan of care according to the person's response to interventions and need for control, in Nursing care plan, p. 178
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Administers medication safely and effectively, in Medication administration, p. 170
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates the person's response to pain relief measures throughout the nursing process, in Nursing care plan, pp. 173, 178
		7.2 Revises the plan based on the evaluation	Evaluates effectiveness of interventions to relieve pain, re-treats or adjusts doses of medication and intervenes as necessary, in Nursing care plan, p. 173
9	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Employs effective communication processes to facilitate education of the person, in Nursing care plan, p. 215
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Communicates nursing assessment of serum potassium levels and ECG findings to doctor, in Nursing care plan, p. 215
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Assesses a person with fluid volume excess, in Nursing care plan, p. 200 When caring for a person with hypokalaemia, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, p. 211 When caring for a person with hyperkalaemia, carries out the processes involved in the assessment phase that specifically address the multidisciplinary team, in Nursing care plan, p. 215

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	5. Develops a plan for nursing practice	5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Plans for the administration of medication safely and effectively, in Medication administration, p. 210 Determines priority nursing diagnoses, based on assessment data, to select and implement individualised nursing interventions, in Nursing care plan, p. 211 Plans education for promoting a healthy diet and safe medication management, in Nursing care plan, p. 215 Integrates interprofessional care into care of a person with altered fluid, electrolyte and acid–base balance, in Nursing care plan, p. 215
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Assesses and monitors the person's fluid, electrolyte and acid–base balance, in Nursing care plan, p. 200 Administers fluids and medications knowledgeably and safely, in Medication administration, p. 210 When caring for a person with hypokalaemia, carries out the processes involved in the implementation phase that specifically address the person's needs, in Nursing care plan, p. 211 Delivers pertinent information to the person and their family about diet and medications used to restore, promote and maintain fluid, electrolyte and acid–base balance, in Nursing care plan, p. 215 When caring for a person with hyperkalaemia, carries out the processes involved in the intervention phase that specifically address the multidisciplinary team, in Nursing care plan, p. 215
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	When caring for a person with hyperkalaemia, carries out the processes involved in the evaluation phase that specifically address the multidisciplinary team, in Nursing care plan, p. 215
10	4. Comprehensively conducts assessments	4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts assessment of a person with multi-trauma within the emergency department, in Nursing care plan, p. 258
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans comprehensive nursing care for a person with multi-trauma within the emergency department, in Nursing care plan, p. 258

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Demonstrates comprehensive nursing care for a person with multi-trauma within the emergency department, in Nursing care plan, p. 258
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates responses to medical and surgical interventions for people sustaining multi-trauma, in Nursing care plan, p. 258
11	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Demonstrates awareness of the procedures for standard precautions for all hospitalised people, in Table 11.9 Transmission-based precautions, p. 312
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Recognises that adherence to transmission-based precautions depends on effective communication with the person, in Table 11.9 Transmission-based precautions, p. 312
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Conducts education for taking antibiotic medication for an infection, which includes the person notifying the care provider about adverse effects, taking or avoiding specific foods and drugs, and fluid intake, in Medication administration, pp. 308–311
			Communicates contact precautions to prevent the spread of infection between people, healthcare professionals and visitors, Table 11.9 Transmission-based precautions, p. 312
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person who requires immunisation, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 295
			Assesses for a history of hypersensitivity to antibiotics, in Medication administration, pp. 308–311
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts a comprehensive assessment for the detection of an infection, in Nursing care of the older adult, p. 305
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Determines priority nursing diagnosis, based on assessment data, to select and implement individualised nursing interventions for people, in Nursing care plan, p. 295
			Plans for contact precautions to prevent the spread of infection between people, healthcare professionals and visitors, Table 11.9 Transmission-based precautions, p. 312

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
		5.4. Plans and negotiates how practice will be evaluated and the time frame of engagement 5.5. Coordinates resources effectively and efficiently for planned actions	Plans for antibiotic administration, which includes fluid dilution, length of administration and compatibility with other medications, in Medication administration, pp. 308–311
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	When caring for a person who requires immunisation, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 295 At the intervention stage monitors the response of the person for adverse effects, including administration site and allergic reactions, in Medication administration, pp. 308–311 Coordinates contact precautions to prevent the spread of infection between people, healthcare professionals and visitors, Table 11.9 Transmission-based precautions, p. 312
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	At the evaluation stage monitors the response of the person for adverse effects, including administration site and allergic reactions, in Medication administration, pp. 308–311
12	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Recognises that taking antiretroviral nucleoside analogues can induce adverse reactions, as well as prolonging life, in Medication administration, p. 348 Uses evidence-based practice in providing nursing care for a person with HIV, in Nursing care plan, p. 354
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with HIV, carries out the processes involved in the assessment phase, in Nursing care plan, p. 354
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans for safe nursing management of antiretroviral agents, in Medication administration, p. 348 Uses evidence-based practice to plan and implement nursing care for people with HIV, carries out the processes involved in the assessment, planning and intervention phases, in Nursing care plan, p. 354
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides appropriate and safe nursing management of antiretroviral agents, in Medication administration, p. 348 When caring for a person with HIV, carries out the processes involved in the intervention phase, in Nursing care plan, p. 354

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
13	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Communicates nursing assessment of any abnormalities for a person with cancer, in Box 13.11 Australia-modified Karnofsky Performance Scale, p. 392
			Works together with the healthcare team to provide optimal care, in Nursing care plan, pp. 394–395
			Refers the person to specific care providers in relation to groups of complications, in Box 13.13 When to call for help, p. 398
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a comprehensive health assessment for a person with cancer and provides ongoing monitoring of status, in Box 13.11 Australia-modified Karnofsky Performance Scale, p. 392
			When caring for a person with cancer, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, pp. 394–395
			Provides individualised assessment of the person and family, in Nursing care plan, pp. 394–395
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of chemotherapeutic drugs and adjunct agents, in Table 13.10 Classification of chemotherapeutic drugs, pp. 383–384
			Prioritises nursing diagnosis based on assessment data and implements appropriate nursing interventions for people with cancer during cancer diagnosis, treatment and rehabilitation, in Nursing care plan, pp. 394–395
			Uses the nursing process as a framework for planning individualised care and integrating interprofessional care for people with cancer to meet their healthcare needs, in Nursing care plan, pp. 394–395
			Designs and provides individualised education to the person and family to restore, promote and maintain the person's functional status, in Box 13.13 When to call for help, p. 398
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides ongoing monitoring of status, in Box 13.11 Australia-modified Karnofsky Performance Scale, p. 392
			When caring for a person with cancer, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care plan, pp. 394–395

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Safely administers chemotherapeutic medications and other medications for pain, nausea and vomiting, mucositis or anaemia, in Table 13.10 Classification of chemotherapeutic drugs, pp. 383–384
			Provides individualised nursing care for the person and family, in Nursing care plan, pp. 394–395
			Conducts education for the recognition of complications and notifying the care provider of the complications, in Box 13.13 When to call for help, p. 398
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Assesses functional health status of people with cancer and monitors, documents and reports abnormal manifestations, in Box 13.11 Australia-modified Karnofsky Performance Scale, p. 392
			Evaluates individualised nursing care for the person and family, in Nursing care plan, pp. 394–395
Unit 4 Responses to Altered Integumentary Structure and Function			
14	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 419–420
	4. Comprehensively conducts assessments	4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts a health history for a person with an alteration in the integument, in Functional health pattern interview, p. 417
			Conducts and /or assists in the collection of skin, blood and tissue samples, in Diagnostic tests, pp. 419–420
			Conducts a physical assessment for a person with an alteration in the integument, in Integumentary assessments, pp. 421–427
15	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Recognises that people who are at risk of a pressure injury or have a pressure injury require specific nursing management protocols, in Box 15.13 Nursing care of the person at risk of a pressure injury and the person with a pressure injury, pp. 468–469
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Provides information on how to manage dry skin and pruritis, in Box 15.1 Teaching to reduce dry skin and relieve pruritis, p. 432
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Works together with the healthcare team to provide optimal care for a person with a pressure injury, in Nursing care plan, pp. 469–470

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	4. Comprehensively conducts assessments	4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	When caring for a person with a pressure injury, carries out the processes involved in the assessment phase that specifically address the person's needs, in Box 15.13 Nursing care of the person at risk of a pressure injury and the person with a pressure injury, pp. 468–469; Nursing care plan, pp. 469–470
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan of care	Plans for the safe nursing management of antifungal agents, in Medication administration, pp. 440–441
			Uses evidence-based research to plan nursing care for people with pressure injuries and skin tears, in Box 15.13 Nursing care of the person at risk of a pressure injury and the person with a pressure injury, pp. 468–469
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	When planning care for a person with a pressure injury, integrates interprofessional care, in Nursing care plan, pp. 469–470
		5.6. Plans nursing care in consultation with individuals/ groups, significant others and the interprofessional team	Plans an education session to promote the reduction of dry skin and relieving pruritis, in Box 15.1Teaching to reduce dry skin and relieve pruritis, p. 432
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Conducts education to promote the self-care of pruritis, in Box 15.1 Teaching to reduce dry skin and relieve pruritis, p. 432
			Administers topical, oral and injectable medications used to treat integumentary disorders knowledgeably and safely, in Medication administration, pp. 440–441
			When caring for a person with a pressure injury, carries out the processes involved in the intervention phase that specifically address the person's needs, in Box 15.13 Nursing care of the person at risk of a pressure injury and the person with a pressure injury, pp. 468–469; Nursing care plan, pp. 469–470
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	When caring for a person with a pressure injury, carries out the processes involved in the evaluation phase that specifically address the multidisciplinary team, in Nursing care plan, pp. 469–470
16	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates in an effective way education to promote burn prevention, in Box 16.1 Burn prevention tips, p. 479

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	4. Comprehensively conducts assessments	4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	When caring for a person with a major burn, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, pp. 502–504
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans education to promote burn prevention, in Box 16.1 Burn prevention tips, p. 479
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	When caring for a person with a major burn, carries out the processes involved in the planning phase that specifically address the person's needs, in Nursing care plan, pp. 502–504
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides teaching appropriate for prevention of burns, in Box 16.1 Burn prevention tips, p. 479 When caring for a person with a major burn, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care plan, pp. 502–504
Unit 5 Responses to Altered Endocrine Function			
17	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 520–524
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration in the endocrine system, in Functional health pattern interview, pp. 518–519
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of blood and urine samples, and radiographical studies, in Diagnostic tests, pp. 520–524 Conducts a physical assessment for a person with an alteration in the endocrine system, in Endocrine assessments, pp. 525–527
18	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Recognises that a person who is prescribed medication for hyperthyroidism requires specific medication management protocols education, in Medication administration, p. 534
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates effectively health education information to promote safe administration of anti-thyroid preparations, in Medication administration, p. 540

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
		2.3. Recognises that people are the experts in the experience of their life	Conducts person-centred education to promote appropriate administration and storage of medication for hypothyroidism, in Medication administration, p. 540
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with hypothyroidism, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, p. 542
			Conducts a comprehensive nursing assessment with consideration of the manifestations of Cushing's syndrome and Addison's disease, in Manifestations, pp. 547, 551
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Assesses respiratory function, in Nursing care of the person, p. 535
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans to provide appropriate teaching for self-medicating with thyroid hormone, in Medication administration, p. 534
			Plans education to ensure the person knows that hormone replacement is for life and knows how to take medications, in Medication administration, p. 540
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a person with hypothyroidism, in Nursing care plan, p. 542
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Conducts education to promote safe administration of anti-thyroid preparations, in Medication administration, p. 534
			Implements respiratory management of the person having a thyroidectomy, in Nursing care of the person, p. 535
			Conducts education to promote adherence to medication regime for hypothyroidism, in Medication administration, p. 540
			When caring for a person with hypothyroidism, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care plan, p. 542
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates respiratory management of the person having a thyroidectomy, in Nursing care of the person, p. 535

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
19	2. Engages in therapeutic and professional relationships	2.3. Recognises that people are the experts in the experience of their life	Conducts person-centred education to promote administration of: insulin via injection, in Medication administration, p. 571; oral hypoglycaemic agents, in Medication administration, pp. 577–578; foot care, in Meeting individualised needs, p. 593
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with type 1 diabetes, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, pp. 592–593
			Make assessments for appropriate nursing care for the person with type 1 diabetes, in Nursing care plan, pp. 592–593
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Assesses for patterns of hypoglycaemia and hyperglycaemia in people with diabetes mellitus and provides ongoing monitoring of the status of the person, in Box 19.6 Guidelines for insulin adjustment, p. 574
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for the person with type 1 diabetes, in Nursing care plan, pp. 592–593
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Plans for education to promote administration of: insulin via injection, in Medication administration, p. 571; oral hypoglycaemic agents, in Medication administration, pp. 577–578
			Devises a teaching plan for the relationship of hygiene, neuropathy and impaired microcirculation to infection; teaches the principles and procedures of effective foot care, in Meeting individualised needs, p. 593
		5.4. Plans and negotiates how practice will be evaluated and the time frame of engagement	Plans for the administration of medication safely and effectively, in Box 19.7 Techniques to minimise painful injections, p. 575
			Provides safe and appropriate nursing care for the person with type 1 diabetes, in Nursing care plan, pp. 592–593
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Assesses for patterns of hypoglycaemia and hyperglycaemia in people with diabetes mellitus and provides ongoing monitoring of the status of the person, in Box 19.6 Guidelines for insulin adjustment, p. 574
			Administers medication safely and effectively, in Box 19.7Techniques to minimise painful injections, p. 575

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a person with type 1 diabetes, carries out the processes involved in the planning phase that specifically address the person's needs, in Nursing care plan, pp. 592–593
			Provides safe and appropriate nursing care for the person with type 1 diabetes, in Nursing care plan, pp. 592–593
			Conducts education to promote administration of: insulin via injection, in Medication administration, p. 571; oral hypoglycaemic agents, in Medication administration, pp. 577–578
			Conducts education to promote effective foot care in diabetics, in Meeting individualised needs, p. 593
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates for patterns of hypoglycaemia and hyperglycaemia in people with diabetes mellitus and provides ongoing monitoring of the status of the person, in Box 19.6 Guidelines for insulin adjustment, p. 574
			Evaluates nursing care for the person with type 1 diabetes, in Nursing care plan, pp. 592–593

MAPPING TO THE NMBA *REGISTERED NURSE STANDARDS FOR* ***PRACTICE***

AUTHOR: Trish Burton

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
Unit 6 Responses to Altered Gastrointestinal Function			
20	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 619–623
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration or at risk of alterations in nutrition and gastrointestinal function, in Functional health pattern interview, pp. 616–617
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of gastric secretions, blood and tissue samples, and radiographical studies, in Diagnostic tests, pp. 619–623
			Conducts a physical assessment of nutritional status and the gastrointestinal system, in Nutritional and Gastrointestinal assessments, pp. 624–633
21	1. Thinks critically and analyses nursing practice	1.3. Respects all cultures and experiences, which includes responding to the role of family and community that underpin the health of Aboriginal and Torres Strait Islander peoples and people of other cultures	Considers the person's cultural background when providing care, in Nursing care plan, p. 654
	2. Engages in therapeutic and professional relationships	2.3. Recognises that people are the experts in the experience of their life	Conducts education that is sensitive to the person's age group and experiences, in Meeting individualised needs, p. 646
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Notifies the doctor in relation to hypersensitivity to iodine or seafood, in Medication administration, p. 650
			Includes dietitian in the evaluation of nutritional needs, in Nursing care plan, p. 654

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with obesity, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, p. 644
			Provides comprehensive assessment for a person with malnutrition, in Nursing care plan, p. 654
			Includes cultural practices in relation to nutritional assessment of the person and subsequent planning, in Nursing care plan, p. 654
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Monitors vitamin and mineral manifestations, in Medication administration, p. 650
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Uses assessment data to determine priority nursing diagnoses and to select and implement nursing interventions for a person with obesity, in Nursing care plan, p. 644
			Plans and provides family teaching to restore, promote and maintain nutritional status in the older person, in Meeting individualised needs, p. 646
			Plans for safe nursing management of medication, in Medication administration, p. 650
			Adapts cultural values and variations into the plan of care for a person with a nutritional disorder, in Nursing care plan, p. 654
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates interprofessional care into the plan of care for a person with malnutrition, in Nursing care plan, p. 654
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	When caring for a person with obesity, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care plan, p. 644
			Conducts education for maintaining nutritional status, in Meeting individualised needs, p. 646
			Implements medication management of the person, in Medication administration, p. 650
			Administers medications and enteral and parenteral nutrition knowledgeably and safely, in Medication administration, p. 650

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Provides comprehensive nursing care for a person with malnutrition, in Nursing care plan, p. 654
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	Evaluates medication management of the person, in Medication administration, p. 650
			Provides comprehensive nursing care for a person with malnutrition, in Nursing care plan, p. 654
22	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to peptic ulcer disease, in Nursing care plan, p. 691
		1.2. Practises within a professional and ethical nursing framework	Considers the person's cultural background when providing care, in Nursing care plan, p. 667
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Is sensitive to the needs of the person with gastric cancer and their family in relation to diagnosis, in Nursing care plan, p. 698
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Includes dietitian in the evaluation of nutritional needs, in Nursing care plan, p. 667
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Demonstrates assessment in nursing care for a person with oral cancer, in Nursing care plan, p. 667
			When caring for a person with oral cancer, carries out the processes involved in the assessment phase that specifically address the person's energy needs and enteral feeding, in Nursing care plan, p. 667
			Includes evidence-based practice in relation to the assessment of the person with peptic ulcer disease, in Nursing care plan, p. 691
			When caring for a person with gastric cancer, carries out the processes involved in the assessment, planning and intervention phases that specifically address the person's needs, in Nursing care plan, p. 698
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Constructs and revises individualised plans of care considering the culture and values of the person with oral cancer, in Nursing care plan, p. 667
			Plans for safe nursing management of medication for GORD, gastritis and peptic ulcer disease, in Medication administration, pp. 671–672
			Plans nursing care using evidence-based research for the person with peptic ulcer disease, in Nursing care plan, p. 691

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a person with gastric cancer, determine priority nursing diagnoses and interventions based on assessed data, in Nursing care plan, p. 698
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	When caring for a person with oral cancer, coordinates and integrates interprofessional care into the plan of care that specifically address the person's energy needs and enteral feeding, in Nursing care plan, p. 667
			Plans and provides the person with gastric cancer and their family with education to promote, maintain and restore functional health in relation to diet, pain management and diagnosis, in Nursing care plan, p. 698
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Demonstrates comprehensive nursing care for a person with oral cancer, in Nursing care plan, p. 667
			When caring for a person with oral cancer, carries out the processes involved intervention phase that specifically address the person's energy needs and enteral feeding, in Nursing care plan, p. 667
			Administers medications and prescribed care knowledgeably and safely, in Medication administration, pp. 671–672
			Conducts education for a person with gastric cancer and their family in relation to diet, pain management and diagnosis, in Nursing care plan, p. 698
			When caring for a person with gastric cancer, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care plan, p. 698
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	Evaluates responses to comprehensive nursing care for a person with oral cancer, in Nursing care plan, p. 667
23	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Includes dietitian in the planning of nutritional needs, in Nursing care plan, p. 750
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides an assessment for a person having an ileostomy, in Nursing care of the person, pp. 746–747
			Provides an assessment for a person with ulcerative colitis, in Nursing care plan, p. 750

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a person with colorectal cancer, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, p. 766
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	During the postoperative phase fluid status, nasogastric drainage, wound assessment and gastrointestinal assessment are ongoing so the plan of care can be revised to ensure optimal care for a person having bowel surgery, in Nursing care of the person, p. 762
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans for safe nursing management of laxative medication, in Medication administration, pp. 708–709
			Plans for comprehensive nursing care for a person having an ileostomy, in Nursing care of the person, pp. 746–747
			Revises plan of care when necessary to provide effective interventions promoting, maintaining or restoring functional health status to a person having bowel surgery, in Nursing care of the person, p. 762
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a person with colorectal cancer, in Nursing care plan, p. 766
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates interprofessional care into care of a person with ulcerative colitis, in Nursing care plan, p. 750
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Administers medications used in the management of bowel disorders knowledgeably and safely, in Medication administration, pp. 708–709
			Provides skilled care to a person following the formation of an ileostomy, in Nursing care of the person, pp. 746–747
			Provides comprehensive nursing care for a person with ulcerative colitis, in Nursing care plan, p. 750
			When caring for a person with colorectal cancer, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care plan, p. 766
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	Evaluates responses to comprehensive nursing care for a person having an ileostomy, in Nursing care of the person, pp. 746–747

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Evaluates responses to comprehensive nursing care for a person with ulcerative colitis, in Nursing care plan, p. 750
24	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Recognises that preoperative education, discharge planning and postoperative pain management are part of an effective pain management strategy, in Translation to practice, p. 788
		1.2. Practises within a professional and ethical nursing framework	Considers the person's cultural background when providing care, in Nursing care plan, p. 789
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Describes to the person the steps in the procedure of paracentesis, in Box 24.5 Nursing implications for abdominal paracentesis, p. 806
			Provides verbal and written information about medication and complications of cirrhosis, in Nursing care plan, p. 809
		2.3. Recognises that people are the experts in the experience of their life	Involves person in the decision making for home-based care post acute pancreatitis, in Nursing care plan, p. 818
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Reports active postoperative bleeding, in Nursing care of the person, p. 789
			Involves the social worker for referral to community services, in Nursing care plan, p. 809
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Includes evidence-based practice in relation to the assessment of the person for pain management post laparoscopic cholecystectomy, in Translation to practice, p. 788
			Includes cultural practices in relation to nutritional assessment of the person with cholelithiasis and subsequent planning, in Nursing care plan, p. 789
			Assesses the person with alcoholic cirrhosis, in Nursing care plan, p. 809
			Assesses the person with a resection of the pancreas, in Nursing care of the person, p. 820
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Monitors for postoperative bleeding, in Nursing care of the person, p. 789
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Uses evidence-based practice to revise the plan of care for the person and subsequent planning for pain management post laparoscopic cholecystectomy, in Translation to practice, p. 788

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Integrates psychosocial, cultural and spiritual considerations into the plan of care for a person with cholelithiasis, in Nursing care plan, p. 789
			Prepares people for and understands the purpose and significance of the procedure of paracentesis, in Box 24.5 Nursing implications for abdominal paracentesis, p. 806
			Plans comprehensive nursing care for a person with a resection of the pancreas, in Nursing care of the person, p. 820
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates a plan for dietary, pharmacological and other interprofessional measures into nursing care and teaching of the person with alcoholic cirrhosis, in Nursing care plan, p. 809
			Plans for appropriate person and family education to promote, maintain and restore functional health status for the person with post-acute pancreatitis, in Nursing care plan, p. 818
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Monitors for postoperative bleeding, in Nursing care of the person, p. 789
			Educates the person in relation to the procedure of paracentesis, in Box 24.5 Nursing implications for abdominal paracentesis, p. 806
			Provides comprehensive nursing care for a person with alcoholic cirrhosis, in Nursing care plan, p. 809
			Conducts education for home-based care post acute pancreatitis, in Nursing care plan, p. 818
			Provides comprehensive nursing care for a person with a resection of the pancreas, in Nursing care of the person, p. 820
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	Monitors for, documents and reports expected and unexpected manifestations of postoperative bleeding, in Nursing care of the person, p. 789
			Evaluates nursing care for a person with alcoholic cirrhosis, in Nursing care plan, p. 809
			Evaluates nursing care for a person with a resection of the pancreas, in Nursing care of the person, p. 820

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
Unit 7 Responses to Altered Urinary Elimination			
25	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 837–841
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration or at risk of alterations in urinary elimination, in Functional health pattern interview, p. 836
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of urine and blood samples and radiographical studies, in Diagnostic tests, pp. 837–841
			Conducts a physical assessment of the renal system, in Renal assessments, pp. 842–843
26	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Understands that insertion of catheter to a specific length that is gender specific and the use of aseptic technique are part of an effective urinary management strategy, in Translation to practice, p. 854
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Nursing care plan, pp. 880–881
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Reports low urine output, in Nursing care of the person, p. 851
			Includes the Continence Nurse Advisor in the provision of care that specifically addresses the person's urinary elimination via stoma, in Nursing care plan, pp. 868–869
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Assesses the person with a bladder tumour, in Nursing care plan, pp. 868–869
			Assesses the functional health status of a person with a urinary tract disorder, in Nursing care of the older adult, pp. 874–875
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Monitors for postoperative bleeding and urine output, in Nursing care of the person, p. 851
			During the postoperative phase monitoring urine output, catheter drainage, stoma assessment and electrolytes is ongoing to ensure optimal care for a person having a cystectomy and urinary diversion, in Nursing care of the person, p. 868
			During the postoperative phase urine output, catheter drainage and wound assessment are ongoing to ensure optimal care for a person with a bladder neck suspension, in Nursing care of the person, p. 879

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans for safe nursing management of medication, in Medication administration, p. 850
			The preoperative phase involves education, stoma preparation and bowel care, and during the postoperative phase monitoring urine output, catheter drainage, stoma assessment and electrolytes is ongoing to ensure optimal care for a person with a cystectomy and urinary diversion, in Nursing care of the person, p. 868
			Uses evidence-based research to plan and insert a urinary catheter, in Translation to practice, p. 874
			When caring for a person with a urinary problem, carries out the processes involved in the planning phase that specifically address the person's needs, in Nursing care of the older adult, pp. 874–875
			Recognises that the preoperative phase involves education and that during the postoperative phase urine output, catheter drainage and wound assessment are ongoing to ensure optimal care for a person with a bladder neck suspension, in Nursing care of the person, p. 879
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates the interprofessional plan of care into care for a person with a bladder tumour, in Nursing care plan, pp. 868–869
			Plans education for prevention and self-care of urinary incontinence, in Nursing care plan, pp. 880–881
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Knowledgeably and safely administers prescribed medications for people with urinary tract disorders, in Medication administration, p. 850
			The preoperative phase involves education, stoma preparation and bowel care, and during the postoperative phase monitoring urine output, catheter drainage, stoma assessment and electrolytes is ongoing to ensure optimal care for a person with a cystectomy and urinary diversion, in Nursing care of the person, p. 868
			Provides comprehensive nursing care for a person with a bladder tumour, in Nursing care plan, pp. 868–869
			When caring for a person with a urinary problem, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care of the older adult, pp. 874–875

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Includes evidence-based practice in relation to the insertion of a urinary catheter, in Translation to practice, p. 874
			Provides effective nursing care for the person undergoing surgery of the bladder neck, in Nursing care of the person, p. 879
			Conducts education for home-based urinary incontinence, in Nursing care plan, pp. 880–881
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	Evaluates responses to nursing care for a person with a bladder tumour, in Nursing care plan, pp. 868–869
			Recognises that the preoperative phase involves education and that during the postoperative phase urine output, catheter drainage and wound assessment are ongoing to ensure optimal care for a person with a bladder neck suspension, in Nursing care of the person, p. 879
		7.2. Revises the plan based on the evaluation	Evaluates personal responses, revising plan of care as needed to promote, maintain or restore functional health of the individual with a cystectomy and urinary diversion, in Nursing care of the person, p. 868
27	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Medication administration, p. 893
			Involves person in the decision making for health management, in Nursing care plan, p. 927
			When caring for a person with end-stage kidney disease, involves the person and the interprofessional team in addressing the person's needs, in Nursing care plan, p. 927
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Consults the dietitian for menu planning that specifically addresses the person's needs, in Nursing care plan, p. 927
			When caring for a person with end-stage kidney disease, involves the person and the interprofessional team in addressing the person's needs, in Nursing care plan, p. 927
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with acute kidney injury, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, p. 896
			Recognises that pre-, intra- and post-dialysis care must ensure optimal management for a person requiring intermittent haemodialysis, in Nursing care of the person, p. 918

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a person with end-stage kidney disease, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, p. 927
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	During the postoperative phase monitoring urine output, catheter drainage, fluid and electrolyte balance, vital signs and wound assessment is ongoing to ensure optimal care for a person receiving a kidney transplant, in Nursing care of the person, p. 925
		4.3. Works in partnership to determine factors that affect, or potentially affect, the health and wellbeing of people and populations to determine priorities for action and/or referral	When caring for a person with end-stage kidney disease, involves the person and the interprofessional team when addressing the person's needs, in Nursing care plan, p. 927
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Assesses the person for medication education appropriate to a person with acute kidney injury and their personal circumstances, in Medication administration, p. 893
			Plans that pre-, intra- and post-dialysis care ensures optimal management for a person requiring intermittent haemodialysis, in Nursing care of the person, p. 918
			Plans for the preoperative phase of education, dialysis and immunosuppressive medication, and during the postoperative phase monitoring urine output, catheter drainage, fluid and electrolyte balance, vital signs and wound assessment is ongoing to ensure optimal care for a person receiving a kidney transplant, in Nursing care of the person, p. 925
			Based on assessment data, determines priority nursing diagnoses and interventions for a person with end-stage kidney disease, in Nursing care plan, p. 927
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Collaborates with the person and other members of the interprofessional team to prioritise and implement care, in Nursing care plan, p. 927

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Conducts education for administering medications for acute kidney injury, in Medication administration, p. 893 Monitors, documents and reports unexpected or abnormal manifestations in a person with acute kidney injury, in Nursing care plan, p. 896 Provides appropriate and effective nursing care for a person requiring intermittent haemodialysis, in Nursing care of the person, p. 918 The preoperative phase involves education, dialysis and immunosuppressive medication, and during the postoperative phase monitoring urine output, catheter drainage, fluid and electrolyte balance, vital signs and wound assessment is ongoing to ensure optimal care for a person receiving a kidney transplant, in Nursing care of the person, p. 925 When caring for a person with end-stage kidney disease, carries out the processes involved in the intervention phase that specifically address the person's needs, in Nursing care plan, p. 927
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	When caring for a person with acute kidney injury, carries out the processes involved in the evaluation phase that specifically address the person's needs, in Nursing care plan, p. 896 Recognises that pre-, intra- and post-dialysis care must ensure optimal management for a person requiring intermittent haemodialysis, in Nursing care of the person, p. 918
		7.2. Revises the plan based on the evaluation	Evaluates responses to care, revising the plan of care as needed to promote, maintain or restore functional health status for a person with a kidney transplant, in Nursing care of the person, p. 925
Unit 8 Responses to Altered Cardiovascular Function			
28	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 958–960
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration or at risk of alterations in cardiac, haematological or lymphatic function, in Functional health pattern interview, pp. 956–957

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of blood and fluid samples, and electrocardiograph and radiographical studies, in Diagnostic tests, pp. 958–960
			Conducts an electrocardiograph and interprets tracing, in Box 28.1 Electrocardiogram, pp. 960–963
			Conducts a physical assessment of the cardiac, haematological, peripheral vascular and lymphatic systems, in Cardiac assessments, pp. 969–978
29	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Advises consultation with the doctor if adverse reaction occurs, in Medication administration, p. 992
			When caring for a person with acute myocardial infarction, notifies medical officer of dangerous arrhythmias, in Nursing care plan, pp. 1016–1017
			When caring for a person with supraventricular tachycardia, notifies doctor of changes in vital signs and ECG, in Nursing care plan, p. 1037
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with coronary heart disease, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care of the person, pp. 1003–1005
			When caring for a person with acute myocardial infarction, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, pp. 1016–1017
			When caring for a person with supraventricular tachycardia, carries out the processes involved in the assessment phase that specifically address the person's needs, in Nursing care plan, p. 1037
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Recognises that the preoperative phase involves education and laboratory and diagnostic tests, and during the postoperative phase monitoring vital signs, haemodynamic monitoring, heart sounds, urine output, chest drainage and fluid and electrolyte balance, and administration of drugs are ongoing to ensure optimal care for a person with a coronary artery bypass graft, in Nursing care of the person, pp. 1003–1005

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans for education for administering medications for lowering cholesterol, in Medication administration, p. 992 Plans for safe nursing management of medication, in Medication administration, pp. 996–997 Interprets assessment data, determines priorities of care and develops and implements individualised nursing interventions for the person with coronary heart disease, in Nursing care of the person, pp. 1003–1005 Plans for the preoperative phase of education and laboratory and diagnostic tests, and during the postoperative phase monitoring vital signs, haemodynamic monitoring, heart sounds, urine output, chest drainage and fluid and electrolyte balance, and administration of drugs are ongoing to ensure optimal care for a person with a coronary artery bypass graft, in Nursing care of the person, pp. 1003–1005
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates multidisciplinary care into nursing care planning and implementation for a person with supraventricular tachycardia, in Nursing care plan, p. 1037
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides appropriate teaching for administering medications for lowering cholesterol, in Medication administration, p. 992 Administers medications and treatments to people with coronary heart disease and arrhythmias safely and knowledgeably, in Medication administration, pp. 996–997 When caring for a person with coronary heart disease, carries out the processes intervention phase that specifically address the person's needs, in Nursing care of the person, pp. 1003–1005 The preoperative phase involves education and laboratory and diagnostic tests, and during the postoperative phase monitoring vital signs, haemodynamic monitoring, heart sounds, urine output, chest drainage and fluid and electrolyte balance, and administration of drugs are ongoing to ensure optimal care for a person with a coronary artery bypass graft, in Nursing care of the person, pp. 1003–1005

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a person with acute myocardial infarction, carries out the processes involved in the implementation phase that specifically address the person's needs, in Nursing care plan, pp. 1016–1017
			When caring for a person with supraventricular tachycardia, carries out the processes involved in the implementation phase that specifically address the person's needs, in Nursing care plan, p. 1037
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	Evaluates the effectiveness of nursing interventions, revising or modifying the plan of care as needed to promote, maintain or restore functional health for the person with a coronary artery bypass graft, in Nursing care of the person, pp. 1003–1005
			Monitors the person with coronary heart disease or arrhythmias for expected and unexpected manifestations, in Nursing care plan, pp. 1016–1017
30	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to mitral valve prolapse, in Nursing care plan, pp. 1082–1083
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Nursing care of the older adult, p. 1046
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Collaborates with the medical staff, dietitian and physiotherapist, in Nursing care plan, p. 1056
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with heart failure, carries out the processes involved in the assessment phase that specifically address the person's dietary and physiotherapy requirements, in Nursing care plan, p. 1056
			Includes evidence-based assessment in relation to providing nursing care for mitral valve prolapse, in Nursing care plan, pp. 1082–1083
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	When evaluating nursing care for a person with heart failure, reinforces medication knowledge, in Nursing care plan, p. 1056
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans appropriate education and community-based care for the person with heart failure, in Nursing care of the older adult, p. 1046

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Plans appropriate and safe nursing management of medication, in Medication administration, pp. 1053–1054
			Plans nursing care for a person with heart failure, reinforces medication knowledge, in Nursing care plan, p. 1056
			Plans and prioritises evidence-based, individualised care for the person with mitral valve prolapse, in Nursing care plan, pp. 1082–1083
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Actively participates in planning and coordinating interprofessional care for the person with heart failure, in Nursing care plan, p. 1056
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Conducts education for heart failure, in Nursing care of the older adult, p. 1046
			Administers prescribed medications and treatments to individuals with cardiac disorders, in Medication administration, pp. 1053–1054
			When caring for a person with heart failure, carries out the processes involved in the intervention phase that specifically address the person's dietary and physiotherapy requirements, in Nursing care plan, p. 1056
			Includes evidence-based practice in relation to providing nursing care for mitral valve prolapse, in Nursing care plan, pp. 1082–1083
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	When caring for a person with heart failure, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the person's dietary and physiotherapy requirements, in Nursing care plan, p. 1056
			Evaluates the effectiveness of nursing care, revising the plan of care as needed to promote, maintain or restore functional health status of the person with heart failure, in Nursing care plan, p. 1056
			Evaluates evidence-based nursing care for mitral valve prolapse, in Nursing care plan, pp. 1082–1083
31	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to deep vein thrombosis, in Nursing care plan, p. 1132

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Medication administration, pp. 1129–1130
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Consults the dietitian for the person's dietary requirements, in Nursing care plan, p. 1107
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with hypertension, carries out the processes involved in the assessment phase that specifically address the person's dietary requirements, in Nursing care plan, p. 1107
			Assesses evidence-based practice in relation to providing nursing care for deep vein thrombosis, in Nursing care plan, p. 1132
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Uses research and evidence-based plans to provide individualised care for the person with deep vein thrombosis, in Nursing care plan, p. 1132
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Collaborates with the interprofessional care team in planning care for a person with hypertension, in Nursing care plan, p. 1107
			Plans person-centred and family teaching for anticoagulant therapy, in Medication administration, pp. 1129–1130
		5.6. Plans nursing care in consultation with individuals/groups, significant others and the interprofessional team	Plans safe nursing management of medication, in Medication administration, pp. 1100–1101
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	When caring for a person with hypertension, carries out the processes involved in the intervention phase that specifically address the person's dietary requirements, in Nursing care plan, p. 1107
			Safely and knowledgeably administers medications, in Medication administration, pp. 1100–1101
			Conducts education for anticoagulant therapy, in Medication administration, pp. 1129–1130
			Includes evidence-based practice in relation to providing nursing care for deep vein thrombosis, in Nursing care plan, p. 1132

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	When caring for a person with hypertension, carries out the processes involved in the evaluation phase that specifically address the person's dietary requirements, in Nursing care plan, p. 1107
			Evaluates evidence-based nursing care for deep vein thrombosis, in Nursing care plan, p. 1132
32	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to Hodgkin's disease, in Nursing care plan, p. 1182
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Nursing care plan, p. 1159
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved outcomes	Notifies the performance of handwashing as per protocol, in Nursing care plan, p. 1172
			Notifies doctor if bleeding occurs, in Nursing care plan, p. 1192
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Assesses for education for folic acid deficiency anaemia, in Nursing care plan, p. 1159
			When a person has a definitive diagnosis of acute myelocytic leukaemia, ensures further assessment is conducted, in Nursing care plan, p. 1172
			When caring for a person with acute myelocytic leukaemia, carries out the processes involved in the assessment phase that specifically address the person's dietary requirements, in Nursing care plan, p. 1172
			Assesses for evidence-based practice in relation to providing nursing care for Hodgkin's disease, in Nursing care plan, p. 1182
			When caring for a person with haemophilia, carries out ongoing assessment, in Nursing care plan, p. 1192
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans for safe nursing management of medication, in Medication administration, p. 1157
			Plans for appropriate education for the person with folic acid deficiency anaemia, in Nursing care plan, p. 1159
			Uses continuing assessment data to revise the plan of care as needed to restore, maintain or promote functional health in the person with a acute myelocytic leukaemia, in Nursing care plan, p. 1172

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Based on knowledge of pathophysiology, prescribed treatment and assessed data, identifies and prioritises nursing diagnoses for a person with Hodgkin's disease, in Nursing care plan, p. 1182
			Includes evidence-based practice in plan for providing nursing care for Hodgkin's disease, in Nursing care plan, p. 1182
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Collaborates with the interprofessional care team to plan coordinated, effective care for a person with acute myelocytic leukaemia, in Nursing care plan, p. 1172
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Safely and knowledgeably administers prescribed medications, in Medication administration, p. 1157
			Conducts education for folic acid deficiency anaemia, in Nursing care plan, p. 1159
			When a person has a definitive diagnosis of acute myelocytic leukaemia, ensures further intervention is conducted, in Nursing care plan, p. 1172
			Uses nursing research and evidence-based practice to implement individualised nursing interventions for a person with Hodgkin's disease, in Nursing care plan, p. 1182
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards expected goals and outcomes	Evaluates education for folic acid deficiency anaemia, in Nursing care plan, p. 1159
			When a person has a definitive diagnosis of acute myelocytic leukaemia, ensures further evaluation is conducted, in Nursing care plan, p. 1172
			When caring for a person with acute myelocytic leukaemia, carries out the processes involved in the evaluation phase that specifically addresses the person's dietary requirements, in Nursing care plan, p. 1172

MAPPING TO THE NMBA *REGISTERED NURSE STANDARDS FOR* ***PRACTICE***

AUTHOR: Trish Burton

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
Unit 9 Responses to Altered Respiratory Function			
33	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 1215–1217
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration or at risk of alteration in respiratory function, in Sample documentation, p. 1212 Conducts a physical assessment for a person with an alteration in the respiratory system, in Respiratory assessments, pp. 1217–1219
	4. Comprehensively conducts assessments	4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of respiratory secretions, blood samples, tissue samples, pleural fluid, pulse oximetry and radiographical studies, in Diagnostic tests, pp. 1215–1217
34	1 Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to nasal packing, in Nursing care of the person, p. 1239
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Nursing care plan, p. 1252
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for peritonsillar abscess, in Nursing care plan, p. 1233 Provides comprehensive nursing care for a person with a total laryngectomy, in Nursing care plan, p. 1252
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of medication, in Medication administration, p. 1224 Uses nursing research and evidence-based practice to plan and implement nursing care for the person with a peritonsillar abscess, in Nursing care plan, p. 1233

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Provides comprehensive nursing care for a person with a total laryngectomy, in Nursing care plan, p. 1252
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Provides appropriate education plan for the person and family in relation to diet and pain management, in Nursing care plan, p. 1252
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Safely and knowledgeably administers medications, in Medication administration, p. 1224
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for peritonsillar abscess, in Nursing care plan, p. 1233
			Provides safe and effective nursing care for a person with a total laryngectomy, in Nursing care plan, p. 1252
			Conducts education for diet and pain management, in Nursing care plan, p. 1252
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides comprehensive nursing care for a person with a total laryngectomy, in Nursing care plan, p. 1252
35	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to pneumonia, in Nursing care plan, p. 1270
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively, and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Nursing care of the older adult, p. 1275
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Uses the nursing process and evidence-based nursing research to plan and implement individualised nursing care for pneumonia, in Nursing care plan, p. 1270
			Conducts education for self-managing tuberculosis, in Nursing care plan, p. 1280
			When caring for a person having lung surgery, carries out the processes involved in the assessment and planning phases that specifically address the person's needs, in Nursing care of the person, pp. 1301–1302
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for pneumonia, in Nursing care plan, p. 1270

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Develops measures to promote ventilation and gas exchange for a person with chest tubes, in Nursing care of the person, p. 1288
			When caring for a person having lung surgery, carries out the processes involved in the assessment and planning phases that specifically address the person's needs, in Nursing care of the person, pp. 1301–1302
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Conducts education for self-managing tuberculosis, in Nursing care of the older adult, p. 1277
			Conducts education for self-managing tuberculosis, in Nursing care plan, p. 1282
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for pneumonia, in Nursing care plan, p. 1272
			Plans and provides appropriate teaching for health promotion in a person with tuberculosis, in Nursing care of the older adult, p. 1275
			Conducts education for self-managing tuberculosis, in Nursing care plan, p. 1280
			When caring for a person with chest tubes, carries out the processes involved in the planning and intervention phases that specifically address the person's needs, in Nursing care of the person, p. 1288
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Evaluates the effectiveness of nursing interventions and teaching, revising strategies and teaching plans as needed, in Nursing care plan, p. 1280
36	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to ARDS, in Nursing care plan, p. 1364
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides appropriate and safe nursing management of medication, in Medication administration, pp. 1318–1319
			Provides comprehensive nursing care for a person with COPD, in Nursing care plan, pp. 1331–1332
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for ARDS, in Nursing care plan, p. 1364

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a person with ARDS, carries out the processes involved in the assessment and planning phases that specifically address the person's needs, in Nursing care plan, p. 1364
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of medication, in Medication administration, pp. 1318–1319
			Provides comprehensive nursing care for a person with COPD, in Nursing care plan, pp. 1331–1332
			Uses data and knowledge of the effects of ARDS and prescribed treatment to identify priority nursing diagnoses and to plan care for a person with ARDS, in Nursing care plan, p. 1364
			Uses the nursing process and evidence-based nursing research to plan and implement individualised nursing care for individuals with ARDS, in Nursing care plan, p. 1364
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Coordinates safe interprofessional care and administers prescribed medications, in Medication administration, pp. 1318–1319
			Provides comprehensive nursing care for a person with COPD, in Nursing care plan, pp. 1331–1332
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for ARDS, in Nursing care plan, p. 1364
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides appropriate and safe nursing management of medication, in Medication administration, pp. 1318–1319
			Evaluates the effectiveness of nursing interventions and teaching, revising strategies and teaching plans as needed, in Nursing care plan, pp. 1331–1332
Unit 10 Responses to Altered Musculoskeletal Function			
37	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 1382–1383
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration in musculoskeletal function, in Functional health pattern interview, p. 1379
			Conducts a physical assessment of the musculoskeletal system, in Musculoskeletal assessments, pp. 1383–1388

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of blood samples, tissue samples, synovial fluid, radiographical studies, electrical activity and nerve conduction, in Diagnostic tests, pp. 1382–1383
38	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to pin site care, in Box 38.4 Nursing interventions for people in traction, p. 1401
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Conducts education for cast care, sling application, neurovascular assessment, exercise, pain medication and complications, in Box 38.6 Nursing interventions for people with fractures of the humerus, p. 1405
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Provides appropriate and safe nursing interventions for people with internal fixation, in Box 38.5 Nursing interventions for people with internal fixation, p. 1403
			Collaborates with the community nurse, physiotherapist and occupational therapist, in Nursing care plan, pp. 1409–1410
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for pin site care, in Box 38.4 Nursing interventions for people in traction, p. 1401
			Provides appropriate and safe nursing management of a cast, in Nursing care of the person, p. 1402
			When caring for a person with a hip fracture, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, pp. 1409–1410
			When caring for a person with a hip fracture, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the person's nursing in the community, physiotherapy and occupational therapy requirements, in Nursing care plan, pp. 1409–1410
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Uses evidence-based research to plan and implement nursing care for people with skeletal pin sites, in Box 38.4 Nursing interventions for people in traction, p. 1401
			Provides appropriate and safe nursing management of a cast, in Nursing care of the person, p. 1402

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a person with a hip fracture, in Nursing care plan, pp. 1409–1410
		5.2 Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Provides education appropriate for prevention and self-care of traumatic injuries of the musculoskeletal system in relation to cast care, sling application, neurovascular assessment, exercise, pain medication and complications, in Box 38.6 Nursing interventions for people with fractures of the humerus, p. 1405
			Integrates interprofessional care into care of people with musculoskeletal trauma, in Nursing care plan, pp. 1409–1410
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation for pin site care, in Box 38.4 Nursing interventions for people in traction, p. 1401
			Provides skilled cast care, in Nursing care of the person, p. 1402
			Conducts education for cast care, sling application, neurovascular assessment, exercise, pain medication and complications, in Box 38.6 Nursing interventions for people with fractures of the humerus, p. 1405
			When caring for a person with a hip fracture, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, pp. 1409–1410
			When caring for a person with a hip fracture, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the person's nursing in the community, physiotherapy and occupational therapy requirements, in Nursing care plan, pp. 1409–1410
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides appropriate and safe nursing management of a cast, in Nursing care of the person, p. 1402
			When caring for a person with a hip fracture, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the person's nursing in the community, physiotherapy and occupational therapy requirements, in Nursing care plan, pp. 1409–1410

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
39	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Provides verbal and written information about rheumatoid arthritis, in Nursing care plan, pp. 1457–1458
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Collaborates with the community nurse and physiotherapist, in Nursing care plan, pp. 1446–1447
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with osteoporosis, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1428
			When caring for a person with osteoarthritis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1446–1447
			Provides comprehensive nursing care for a person with rheumatoid arthritis, in Nursing care plan, pp. 1457–1458
			Provides appropriate and safe nursing management of surgical debridement for osteomyelitis, in Nursing care of the person, p. 1470
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of medication, in Medication administration, p. 1427
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a person with osteoporosis, in Nursing care plan, p. 1428
			Provides appropriate and safe nursing management of surgical debridement for osteomyelitis, in Nursing care of the person, p. 1470
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates interprofessional care into care of a person with osteoarthritis, in Nursing care plan, pp. 1446–1447
			Provides a teaching plan appropriate for community-based self-care of rheumatoid arthritis, in Nursing care plan, pp. 1457–1458
		5.3. Documents, evaluates and modifies plans accordingly to facilitate the agreed outcomes	Revises plan of care as needed to provide effective interventions to promote, maintain or restore functional health status for a person with osteoarthritis, in Nursing care plan, pp. 1446–1447

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	When caring for a person with osteoporosis, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1428
			Administers topical, oral and injectable medications knowledgeably and safely, in Medication administration, p. 1432
			When caring for a person with osteoarthritis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1446–1447
			Provides comprehensive nursing care for a person with osteoarthritis, in Nursing care plan, pp. 1446–1447
			Conducts education for self-managing rheumatoid arthritis, in Nursing care plan, pp. 1457–1458
			Provides skilled care of people having a surgical debridement for osteomyelitis, in Nursing care of the person, p. 1470
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	When caring for a person with osteoarthritis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1446–1447
			Provides comprehensive nursing care for a person with osteoarthritis, in Nursing care plan, pp. 1446–1447
			Provides appropriate and safe nursing management of surgical debridement for osteomyelitis, in Nursing care of the person, p. 1470
Unit 11 Responses to Altered Neurological Function			
40	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 1503–1506
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration in neurological function, in Functional health pattern interview, pp. 1501–1502
			Conducts a physical assessment of the neurological system, in Neurological assessments, pp. 1507–1513
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of CSF fluid, radiographical studies, blood flow, electrical activity and nerve conduction, in Diagnostic tests, pp. 1503–1506

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Conducts a physical assessment of the neurological system, in Neurological assessments, pp. 1507–1513
41	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to seizure disorder, in Nursing care plan, p. 1542
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Nursing care plan, p. 1542
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Communicates nursing assessment to the doctor, in Nursing care plan, p. 1536
			Reports seizure activity, in Nursing care plan, p. 1557
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with a migraine headache, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1536
			Provides appropriate and safe nursing management of a person with a subdural haematoma, in Nursing care plan, p. 1551
			Conducts a nursing assessment for a person with bacterial meningitis, in Nursing care plan, p. 1557
			Provides comprehensive nursing care for a person with bacterial meningitis, in Nursing care plan, p. 1557
			When caring for a person with a brain tumour, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1564
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of medication, in Medication administration, p. 1527
			Provides appropriate and safe nursing management of a person with a subdural haematoma, in Nursing care plan, p. 1551
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a person with a brain tumour, in Nursing care plan, p. 1564

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates interprofessional care into care of a person with a migraine headache, in Nursing care plan, p. 1536 Provides appropriate teaching and evidence-based practice to facilitate community-based care to promote safety and prevent injury, and to provide information and support necessary for long-term care of a person with a seizure disorder, in Nursing care plan, p. 1542
		5.3. Documents, evaluates and modifies plans accordingly to facilitate the agreed outcomes	Revises plan of care as needed to provide effective interventions to promote, maintain or restore functional health status for a person with bacterial meningitis, in Nursing care plan, p. 1557
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Administers oral and injectable medications knowledgeably and safely, in Medication administration, p. 1527 When caring for a person with a migraine headache, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1536 Conducts education for seizure disorder, in Nursing care plan, pp. 1542 Provides skilled care to a person with a subdural haematoma, in Nursing care plan, p. 1551 Conducts a nursing assessment for a person with bacterial meningitis, in Nursing care plan, p. 1557 Provides comprehensive nursing care for a person with bacterial meningitis, in Nursing care plan, p. 1557 When caring for a person with a brain tumour, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1564
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	When caring for a person with a migraine headache, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1536 Provides appropriate and safe nursing management of a person with a subdural haematoma, in Nursing care plan, p. 1551

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Assesses functional status of people with intracranial disorders and monitors, documents and reports abnormal findings, in Nursing care plan, p. 1557
			Provides comprehensive nursing care for a person with bacterial meningitis, in Nursing care plan, p. 1557
42	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to rapid treatment of stroke, in Translation to practice, p. 1577
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Meeting individualised needs, p. 1601
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Collaborates with the community nurse, in Nursing care plan, p. 1579
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides appropriate and safe nursing management of a person having a carotid endarterectomy, in Nursing care of the person, p. 1576
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning, implementation and evaluation of stroke, in Translation to practice, p. 1577
			When caring for a person with a stroke, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1586
			Provides comprehensive nursing care for a person with a stroke, in Nursing care plan, p. 1579
			When caring for a person with an SCI, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1593
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of medication, in Medication administration, p. 1590
			Provides appropriate and safe nursing management of a person with a carotid endarterectomy, in Nursing care of the person, p. 1576

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Uses evidence-based research to promote early recognition and treatment of the warning signs of a stroke, in Translation to practice, p. 1577 Integrates interprofessional care into care of a person with a stroke, in Nursing care plan, p. 1579 Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a person with an SCI, in Nursing care plan, p. 1593 Provides appropriate teaching to facilitate self-care of a ruptured intervertebral disc, in Meeting individualised needs, p. 1601
		5.3. Documents, evaluates and modifies plans accordingly to facilitate the agreed outcomes	Revises plan of care as needed to provide effective interventions to promote, maintain or restore functional health status for a person with a stroke, in Nursing care plan, p. 1579
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides skilled care to a person with a carotid endarterectomy, in Nursing care of the person, p. 1576 Includes evidence-based practice in relation to the assessment of the person and subsequent planning, implementation and evaluation of stroke, in Translation to practice, p. 1577 When caring for a person with a stroke, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1579 Provides comprehensive nursing care for a person with a stroke, in Nursing care plan, p. 1579 Administers oral medications knowledgeably and safely, in Medication administration, p. 1590 When caring for a person with an SCI, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1593 Conducts education for self-managing a ruptured intervertebral disc, in Meeting individualised needs, p. 1601
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides appropriate and safe nursing management of a person with a carotid endarterectomy, in Nursing care of the person, p. 1576

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning, implementation and evaluation of stroke, in Translation to practice, p. 1577
			When caring for a person with a stroke, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1579
			Provides comprehensive nursing care for a person with a stroke, in Nursing care plan, p. 1579
43	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to MS, in Nursing care plan, p. 1624
	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Collaborates with the physiotherapist and occupational therapist, in Nursing care plan, p. 1630
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides comprehensive nursing care for a person with AD, in Nursing care plan, pp. 1612–1613
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation of nursing care for MS, in Nursing care plan, p. 1624
			Provides comprehensive nursing care for a person having plasmapheresis, in Nursing care of the person, p. 1641
			When caring for a person with PD, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1630
			When caring for a person with myasthenia gravis, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1643
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of medication, in Medication administration, p. 1611
			Uses evidence-based research to design nursing interventions specific to the needs of ageing people with MS, in Nursing care plan, p. 1624
			Provides comprehensive nursing care for a person having plasmapheresis, in Nursing care of the person, p. 1641

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a person with myasthenia gravis, in Nursing care plan, p. 1643
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates interprofessional care for a person with PD, in Nursing care plan, p. 1630
		5.3. Documents, evaluates and modifies plans accordingly to facilitate the agreed outcomes	Revises plan of care as needed to provide effective interventions to promote, maintain or restore functional health status of a person with AD, in Nursing care plan, pp. 1612–1613
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Administers oral and injectable medications knowledgeably and safely, in Medication administration, p. 1611
			Provides comprehensive nursing care for a person with AD, in Nursing care plan, pp. 1612–1613
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation of nursing care for MS, in Nursing care plan, p. 1624
			When caring for a person with PD, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1630
			Provides skilled care for a person having plasmapheresis, in Nursing care of the person, p. 1641
			When caring for a person with myasthenia gravis, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1643
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides comprehensive nursing care for a person with AD, in Nursing care plan, pp. 1612–1613
			When caring for a person with PD, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1630
			Provides comprehensive nursing care for a person having plasmapheresis, in Nursing care of the person, p. 1641

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
Unit 12 Responses to Altered Visual and Auditory Function			
44	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 1667; 1678
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a health history for a person with an alteration in visual function, in Functional health pattern interview, p. 1665
		4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts and/or assists in the collection of refraction, intraocular pressure and radiographical studies, in Diagnostic tests, p. 1667
			Conducts a physical assessment of the auditory system, in Ear and hearing assessments, pp. 1679–1680
45	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to the nursing care of blindness, in Nursing care of the person, p. 1684
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Nursing care of the person, p. 1690
		2.3. Recognises that people are the experts in the experience of their life	Involves person with decision making, in Nursing care of the person, p. 1684
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Collaborates with the social worker, in Nursing care plan, p. 1703
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with glaucoma and cataracts, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1703
			When caring for a person with glaucoma and cataracts, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1703
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Plans and implements appropriate and individualised evidence-based nursing interventions and education for blindness, in Nursing care of the person, p. 1684
			Provides appropriate and safe nursing management of a person who is having eye surgery, in Nursing care of the person, p. 1690

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Provides appropriate and safe nursing management of medication, in Medication administration, p. 1701
			Using assessed data, determines priority nursing interventions and care for a person with glaucoma and cataracts, in Nursing care plan, p. 1703
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Collaborates with other members of the healthcare team to provide effective care for a person with glaucoma and cataracts, in Nursing care plan, p. 1703
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Includes evidence-based practice in relation to the planning and implementation of nursing care for blindness, in Nursing care of the person, p. 1684
			Provides appropriate care and education for a person who is having eye surgery, in Nursing care of the person, p. 1690
			Safely and effectively administers eye medications, in Medication administration, p. 1701
			When caring for a person with glaucoma and cataracts, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1703
			When caring for a person with glaucoma and cataracts, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1703
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	When caring for a person with glaucoma and cataracts, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, p. 1703
Unit 13 Responses to Altered Reproductive Function			
46	2. Engages in therapeutic and professional relationships	2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Monitors the results of diagnostic tests and reports abnormal findings, in Diagnostic tests, pp. 1736–1737
	4. Comprehensively conducts assessments	4.2. Uses a range of assessment techniques to systematically collect relevant and accurate information and data to inform practice	Conducts a health history for a man with an alteration in the male reproductive system, in Functional health pattern interview, p. 1735

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Conducts and/or assists in the collection of blood samples and cell and tissue samples, and radiographical studies, in Diagnostic tests, pp. 1736–1737
			Conducts a physical assessment of the female reproductive system, in Female reproductive assessments, pp. 1751–1754
47	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to discharge education, in Translation to practice, p. 1777
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides appropriate and safe nursing management of a man having a prostatectomy, in Nursing care of the man, pp. 1771–1772
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning, implementation and evaluation of discharge education, in Translation to practice, p. 1777
			Provides comprehensive nursing care for a man with prostate cancer, in Nursing care plan, p. 1780
			When caring for a man with prostate cancer, carries out the processes involved in the assessment, planning and implementation phases that specifically address the man's needs, in Nursing care plan, p. 1780
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of a man having a prostatectomy, in Nursing care of the man, pp. 1771–1772
			Uses evidence-based research to provide information and education to men having a radical prostatectomy, in Translation to practice, p. 1777
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a man with prostate cancer, in Nursing care plan, p. 1780
		5.3. Documents, evaluates and modifies plans accordingly to facilitate the agreed outcomes	Revises plan of care as needed to provide effective interventions to promote, maintain or restore functional health status for a man with prostate cancer, in Nursing care plan, p. 1780
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides skilled care to a man undergoing prostate surgery, in Nursing care of the man, pp. 1771–1772

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning, implementation and evaluation of discharge education, in Translation to practice, p. 1777
			Provides comprehensive nursing care for a man with prostate cancer, in Nursing care plan, p. 1780
			When caring for a man with prostate cancer, carries out the processes involved in the assessment, planning and implementation phases that specifically address the man's needs, in Nursing care plan, p. 1780
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Includes evidence-based practice in relation to the assessment of the man and subsequent planning, implementation and evaluation of discharge education, in Translation to practice, p. 1777
			Provides appropriate and safe nursing management of a man having a prostatectomy, in Nursing care of the man, pp. 1771–1772
			Provides comprehensive nursing care for a man with prostate cancer, in Nursing care plan, p. 1780
48	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates evidence-based practice in relation to breast and cervical cancer, in Translation to practice, p. 1823
		1.2. Develops practice through reflection on experiences, knowledge, actions, feelings and beliefs to identify how these shape practice	Incorporates evidence-based practice in relation to breast and cervical cancer, in Translation to practice, p. 1823
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Communicates therapeutically, in Meeting individualised needs, p. 1785
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Collaborates with the counsellor and dietitian, in Nursing care plan, pp. 1800–1801
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides appropriate and safe nursing management of a woman having a hysterectomy, in Nursing care of the woman, p. 1793
			When caring for a woman with endometriosis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1800–1801

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a woman with cervical cancer, carries out the processes involved in the assessment, planning and implementation phases that specifically address the woman's needs, in Nursing care plan, pp. 1803–1804
			Provides comprehensive nursing care for a woman with breast cancer, in Nursing care plan, p. 1821
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning of nursing care for breast and cervical cancer, in Translation to practice, p. 1823
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of a woman having a hysterectomy, in Nursing care of the woman, p. 1793
			Determines priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for a woman with cervical cancer, in Nursing care plan, pp. 1803–1804
			Provides appropriate and safe nursing management of medication, in Medication administration, p. 1818
			Uses evidence-based research to design interventions to promote early diagnosis and treatment of all Australian women with cervical and breast cancer, with particular focus on the health disparities for women from areas of social disadvantage; for example, women living in remote and rural areas of Australia or Indigenous women, in Translation to practice, p. 1823
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Provides health education appropriate for community-based self-care of sexual function, in Meeting individualised needs, p. 1785
			Integrates an interprofessional approach into care for a woman with endometriosis, in Nursing care plan, pp. 1800–1801
		5.3. Documents, evaluates and modifies plans accordingly to facilitate the agreed outcomes	Revises plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to a woman with breast cancer, in Nursing care plan, p. 1821
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Conducts education for self-managing sexual function, in Meeting individualised needs, p. 1785

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			Provides skilled care for a woman having a hysterectomy, in Nursing care of the woman, p. 1793
			When caring for a woman with endometriosis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1800–1801
			Provides comprehensive nursing care for a woman with breast cancer, in Nursing care plan, p. 1821
			When caring for a woman with cervical cancer, carries out the processes involved in the assessment, planning and implementation phases that specifically address the woman's needs, in Nursing care plan, pp. 1803–1804
			Administers medications knowledgeably and safely, in Medication administration, p. 1818
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides appropriate and safe nursing management of a woman having a hysterectomy, in Nursing care of the woman, p. 1793
			When caring for a woman with endometriosis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1800–1801
			Provides comprehensive nursing care for a woman with breast cancer, in Nursing care plan, p. 1821
49	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Provides written and verbal information for self-managing syphilis, in Nursing care plan, pp. 1844–1845
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Collaborates with relationship counsellor, in Nursing care plan, pp. 1844–1845
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	When caring for a person with gonorrhoea, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1841
			Provides comprehensive nursing care for the person who has syphilis, in Nursing care plan, pp. 1844–1845

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
			When caring for a person with syphilis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1844–1845
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Provides appropriate and safe nursing management of medication, in Medication administration, p. 1835
			Determines nursing priorities and selects and implements individualised nursing intervention for a person with gonorrhoea, in Nursing care plan, p. 1841
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Integrates interprofessional care into care of a person with syphilis, in Nursing care plan, pp. 1844–1845
			Conducts education for self-managing syphilis, in Nursing care plan, pp. 1844–1845
		5.3. Documents, evaluates and modifies plans accordingly to facilitate the agreed outcomes	Revises plan of care as needed to provide effective interventions to promote, maintain or restore functional health status for the person who has syphilis, in Nursing care plan, pp. 1844–1845
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Administers topical medications knowledgeably and safely, in Medication administration, p. 1835
			When caring for a person with gonorrhoea, carries out the processes involved in the assessment, planning and implementation phases that specifically address the person's needs, in Nursing care plan, p. 1841
			When caring for a person with syphilis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1844–1845
			Provides teaching appropriate for prevention, control and self-care of syphilis, in Nursing care plan, pp. 1844–1845
			Provides comprehensive nursing care for the person who has syphilis, in Nursing care plan, pp. 1844–1845

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	When caring for a person with syphilis, carries out the processes involved in the assessment, planning, intervention and evaluation phases that specifically address the multidisciplinary team, in Nursing care plan, pp. 1844–1845
			Provides comprehensive nursing care for the person who has syphilis, in Nursing care plan, pp. 1844–1845
Unit 14 Special Topics in Medical–Surgical Nursing			
50	1. Thinks critically and analyses nursing practice	1.1. Accesses, analyses and uses the best available evidence, that includes research findings, for safe, quality practice	Incorporates the Recovery Model as the framework for mental healthcare, in Recovery, pp. 1860–1863
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Conducts a mental state assessment of the person, in Box 50.4 Components of a mental state assessment, p. 1864
			Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation of mental health nursing care, in Recovery, pp. 1860–1863
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Uses an evidence-based approach to design interventions which promote Recovery, in Recovery, pp. 1860–1863
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Includes evidence-based practice in relation to the assessment of the person and subsequent planning and implementation of mental health nursing care, in Recovery, pp. 1860–1863
51	1. Thinks critically and analyses nursing practice	1.2. Practises within a professional and ethical nursing framework	Incorporates primary care principles in nursing service delivery, in Primary healthcare and primary care, pp. 1882–1884
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides comprehensive nursing care for the person and the community, in Primary healthcare and primary care, pp. 1882–1884
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Incorporate the principles of primary care into the provision of nursing care in the regional, rural and remote setting, in Primary healthcare and primary care, pp. 1882–1884
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides comprehensive nursing care for the person and the community, in Primary healthcare and primary care, pp. 1882–1884
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides comprehensive nursing care for the person and the community, in Primary healthcare and primary care, pp. 1882–1884
52	1. Thinks critically and analyses nursing practice	1.2. Practises within a professional and ethical nursing framework	Practises cultural safety, in Indigenous health considerations in regional, rural and remote areas, pp. 1897–1898

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	2. Engages in therapeutic and professional relationships	2.2. Communicates effectively and is respectful of a person's dignity, culture, values, beliefs and rights	Establishes a therapeutic relationship based on trust, in Establishing boundaries, p. 1905
		2.6. Uses delegation, supervision, coordination, consultation and referrals in professional relationships to achieve improved health outcomes	Consults and/or refers to specialist healthcare individuals/organisations, in Acute assessment and emergency nursing care, pp. 1901–1905
	4. Comprehensively conducts assessments	4.1. Conducts assessments that are holistic as well as culturally appropriate	Provides culturally sensitive nursing care with the person and the community as active participants, in Indigenous health considerations in regional, rural and remote areas, pp. 1897–1898
			Conducts comprehensive assessment of the person and subsequent planning, implementation and evaluation of care, including referral and transfer, in Acute assessment and emergency nursing care, pp. 1901–1905
			Provides comprehensive nursing care for a person in the community, in Establishing boundaries, p. 1905
	5. Develops a plan for nursing practice	5.1. Uses assessment data and best available evidence to develop a plan	Uses assessment findings to determine initial nursing care, referral and transfer as deemed necessary, in Acute assessment and emergency nursing care, pp. 1901–1905
		5.2. Collaboratively constructs nursing practice plans until contingencies, options, priorities, goals, actions, outcomes and time frames are agreed with the relevant people	Provides culturally sensitive nursing care with the person and the community as active participants, in Indigenous health considerations in regional, rural and remote areas, pp. 1897–1898
			Uses professional communication skills to develop therapeutic relationships and establish professional boundaries when working in regional, rural and remote communities, in Establishing boundaries, p. 1905
	6. Provides safe, appropriate and responsive quality nursing practice	6.1. Provides comprehensive, safe, quality practice to achieve agreed goals and outcomes that are responsive to the nursing needs of people	Provides culturally safe nursing care, pp. 1897–1898
			Conducts comprehensive assessment of the person and subsequent planning, implementation and evaluation of care, including referral and transfer, in Acute assessment and emergency nursing care, pp. 1901–1905
			Provides comprehensive nursing care for a person in the community, in Establishing boundaries, p. 1905

CHAPTER	STANDARD	CRITERIA	EVIDENCE-BASED EXAMPLES (INCLUDING PAGE NO.)
	7. Evaluates outcomes to inform nursing practice	7.1. Evaluates and monitors progress towards the expected goals and outcomes	Provides culturally sensitive nursing care with the person and the community as active participants, in Indigenous health considerations in regional, rural and remote areas, pp. 1897–1898 Conducts comprehensive assessment of the person and subsequent planning, implementation and evaluation of care, including referral and transfer, in Acute assessment and emergency nursing care, pp. 1901–1905 Provides comprehensive nursing care for a person in the community, in Establishing boundaries, p. 1905

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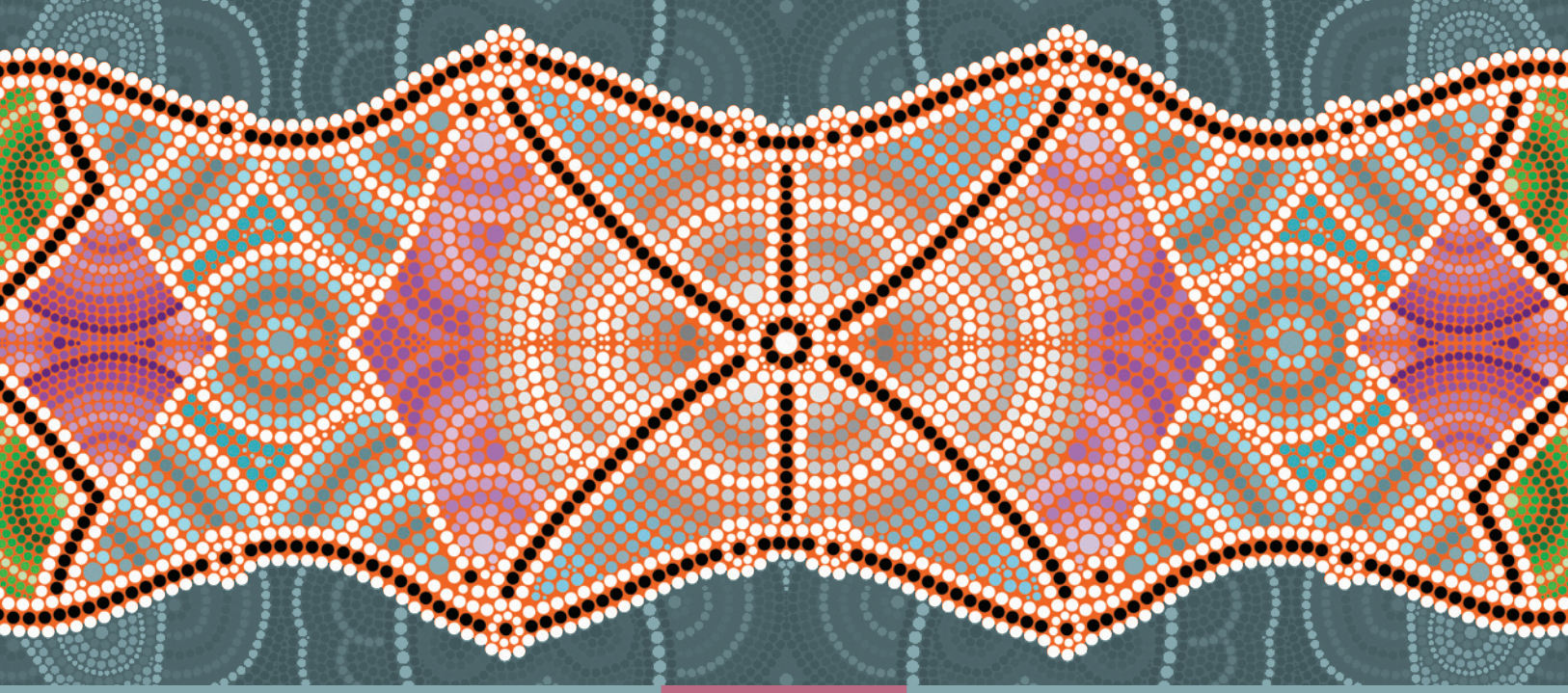

UNIT 1

DIMENSIONS OF MEDICAL–SURGICAL NURSING

CHAPTER 1

MEDICAL–SURGICAL NURSING

TRACY LEVETT-JONES, LORINDA PALMER

KEY TERMS

LEARNING OUTCOMES

- Define and discuss the importance of person-centred care.
- Describe the attitudes, attributes and skills necessary for critical thinking when providing nursing care.
- Outline the stages of the nursing process.
- Outline the stages of the Clinical Reasoning Cycle and how it was designed to positively impact on patient safety.
- Describe the importance of national competency standards, codes of ethics and codes of professional conduct as guidelines for accountable and professional nursing practice.
- Outline the concept of cultural competence as an integral component of nursing care.
- Discuss some of the legal and ethical dilemmas evident in medical–surgical nursing.
- Discuss the roles and functions of the nurse as caregiver, educator, advocate, leader/manager and researcher.

CLINICAL COMPETENCIES

- Demonstrate critical thinking and clinical reasoning when providing evidence-based, safe, person-centred and culturally competent nursing care.
- Provide clinical care within a framework that integrates, as appropriate, the medical–surgical nursing roles of caregiver, educator, advocate, leader/manager and researcher.

Medical–surgical nursing is one component in a suite of nursing services that provide healthcare within an interprofessional approach. The descriptors 'medical' or 'surgical' nursing are broad and you will encounter many specialty practice areas within the general category of medical–surgical nursing; for example, acute care, day surgery, community mental health, general practice, renal dialysis satellite centres and outpatient clinics.

The person with whom and for whom nursing care is designed and implemented may range in age from late teens to 100 years of age or even older. Medical–surgical nursing focuses on a person's response to actual or potential alterations in health and takes into account their history, community and social support network. The wide range of ages, cultural and linguistic diversity and variety of healthcare needs specific to each person make medical–surgical nursing a dynamic, challenging and rewarding area of nursing practice.

Medical–surgical nursing includes the promotion of health, prevention of illness and the care of ill, disabled and dying people across the lifespan and in diverse practice contexts. Nurses are responsible for the provision of safe, empathetic, person-centred, evidence-based care. They communicate and collaborate with patients, families and other health professionals to promote health and wellbeing. Nursing care is guided by clear thinking processes and professional, ethical and legal frameworks. This chapter provides a broad overview of the clinical practice of medical–surgical nursing, including the roles and functions of the medical–surgical nurse.

PERSON-CENTRED CARE

The terms '*person*' and '*patient*' denote the individual who is the recipient of care and may be used interchangeably, depending on the context of care. In this book we generally use the term 'person' as this aligns with the concept of person centred.

Person-centred care means seeing the *person*, not just the patient or their disease process. That is, we speak of a *person* with a disease; for example, 'In bed 4 is Mr Johns who has had an appendectomy'; rather than 'the appendectomy in bed 4', or 'Joanne requires assistance with her meals' rather than 'Joanne is a feed'.

Person-centred nurses are empathetic, respectful, ethical, open-minded and self-aware. They have a profound sense of personal responsibility for actions (moral agency) and place the 'person' at the centre of healthcare, considering the person's needs and wishes as paramount (McCormack & Titchen, 2001; Redman & Lynn, 2004). Integral to person-centred care is therapeutic communication and the nurse's commitment to understanding the person's beliefs and values, life history and cultural and/or linguistic diversity. Person-centred care is central to safe, effective and competent nursing practice. There is a body of evidence indicating that person-centred care results in improved patient outcomes; for example, decreased mortality (Meterko et al., 2010), fewer medication errors (Bolster & Manias, 2010), decreased infection and readmission rates (Isaac et al., 2010) and improved quality of life for people with dementia (Chenoweth et al., 2009).

CRITICAL THINKING

Critical thinking is a complex collection of cognitive skills and affective habits of the mind and has been described as the process of analysing and assessing thinking with a view to improving it (Paul & Elder, 2007). Critical thinking includes the ability to think about one's own thinking; this is called metacognition. To think like a nurse requires you to learn the knowledge, ideas, skills, concepts and theories of nursing, and develop your intellectual capacities to become a disciplined, self-directed, critical thinker capable of clinical reasoning (Paul & Elder, 2007). Critical thinking requires practice so that it becomes integral to your clinical decision making. Learning activities are included throughout this book to provide opportunities for practising critical thinking.

Thinking critically involves more than just cognitive (knowledge) skills. It is strongly influenced by one's attitudes and mental habits. To think critically, you must focus your attention on your attitudes and how they affect your thinking. These attitudes and mental habits include the following:

- Being able to think independently so that you make clinical decisions based on sound thinking and judgment. This means, for example, that you are not influenced by negative comments from other health professionals about a person.
- Being willing to listen to and be fair in your evaluation of others' ideas and beliefs. This involves listening carefully to other ideas and thoughts, and making decisions based on what you have learned instead of how you feel.
- Having empathy and practising empathy in a person-centred way by being able to put yourself in the place of another to better understand that person. For example, if you put yourself in the place of the person with severe pain, you are better able to understand why they are so upset when pain medications are late.
- Being fair minded, just and considerate of all viewpoints before making a decision. This means you consider the viewpoints of others that may be different from yours before reaching a conclusion. You also realise that you are constantly learning from others. You are not afraid to say, 'I don't know the answer to that question, but I will find out and let you know.'
- Being disciplined so that you do not stop at easy answers, but continue to consider alternatives.
- Being creative and self-confident. Nurses often need to consider different ways of providing care and constantly look for improved and more cost-effective methods. Confidence in your decision making is enhanced through effective critical thinking.

The major critical thinking skills are divergent thinking, reasoning, clarifying and reflection. A description of each follows.

Divergent thinking is having the ability to weigh the importance of information. This means that when you collect data (information/cues) from a person, you can sort out the data that are relevant for the care of that person from the data that are not

relevant and then explore alternatives before reaching a conclusion. Abnormal data are usually considered relevant; normal data are helpful but may not change the care you provide.

Reasoning is having the ability to discriminate between facts and guesses. By using known facts, problems are solved and decisions are made in a systematic, logical way. For example, when you take a pulse you must know the parameters of the normal pulse rate for a person of this age, the types of medications the person is taking that may alter their pulse rate, and the emotional and physical state of the person. Based on these facts, you are able to decide if the pulse rate is normal or abnormal.

Clarifying involves noting similarities and differences and sifting out unnecessary information to help focus on the present situation. For example, when caring for a person with persistent (chronic) pain, you must know the definition of persistent pain and the similarities and differences between acute pain and persistent pain.

Reflection is a crucial professional activity and one that is intrinsic to learning. It is not simply introspection but is a deliberate, orderly and structured intellectual activity (Bolton, 2001). It allows nurses to process their experience and explore their understanding of what they are doing, why they are doing it and the impact it has on themselves and others (Levett-Jones, 2007). When this activity is developed and enhanced in relation to personal and professional practice, reflection becomes a purposeful activity that leads to improvement in practice and better patient outcomes.

THE NURSING PROCESS

The **nursing process** has been described as a tool that helps nurses to think critically in order to provide a competent level of care (Alfaro-LeFevre, 2009). The activities within the nursing process define a nursing model of care, differentiating nursing from other helping professions. The nursing process can be used in any setting. The purpose of care may be to promote wellness, maintain health, restore health or facilitate coping with disability or death. Regardless of the purpose of care, the planned process of nursing allows for the inclusion of specific, holistic and person-centred care. The five steps or phases in the nursing process are assessment, diagnosis, planning, implementation and evaluation. These steps are interrelated and interdependent (see Figure 1.1).

This textbook assumes that students already have a basic understanding of the nursing process and are now ready to expand and apply that knowledge to people with medical–surgical health problems. The overview in this book serves only as a review; for more information students should read the case studies in the nursing care chapters throughout the three volumes of this textbook. Table 1.1 articulates the links between the steps of the nursing process steps and the corresponding critical thinking applications.

Assessment

Assessment is usually listed as the first step of the nursing process, but in actuality it is a critical element in each of the steps. It begins with the person's first encounter with the healthcare system and continues as long as the person requires care. During assessment, data (cues or pieces of information) about the person's health status are collected, validated, organised, clustered into patterns and communicated either verbally or in written form. Assessment serves as the basis for deriving an accurate nursing diagnosis, for planning and implementing both initial and ongoing care, and for evaluating the effectiveness of the care provided.

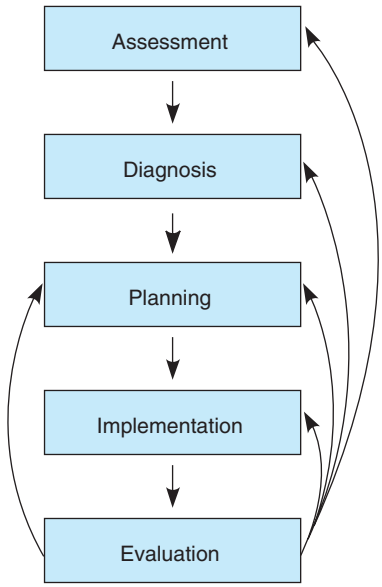

FIGURE 1.1 ■ The nursing process. Steps of the nursing process. Notice that the steps are interrelated and interdependent. For example, evaluation of the person might reveal the need for further assessment, additional nursing diagnoses and/or a revision of the plan of care

The data that the nurse collects must be holistic; that is, the nurse must carefully consider all dimensions of an individual (physical, mental, social, emotional and spiritual). The data collected are both objective and subjective. Information that the nurse perceives by the senses is *objective data*; it is seen, heard, touched or smelled and can be verified by another person (e.g. blood pressure, temperature, pulse or the presence of infected drainage). Information that is perceived only by the person experiencing it (e.g. pain, dizziness or anxiety) is *subjective data*.

Nurses assess people in two ways: through an initial assessment and through focused assessments. The initial assessment of the person, conducted through a nursing history and physical assessment, is necessary to accumulate comprehensive baseline data about health responses, to identify specific factors that contribute to these responses in an individual and to facilitate mutually established goals and outcomes of care.

Focused assessments (e.g. respiratory assessment) enable the nurse to evaluate nursing actions and make decisions about whether to continue or change interventions to meet outcomes. They also provide structure for the documentation of nursing care. In addition, focused assessments enable the nurse to identify responses to a disease process or treatment modality not present during the initial assessment, or to monitor the

TABLE 1.1 Using critical thinking in the nursing process

NURSING PROCESS STEP	CRITICAL THINKING SKILLS	QUESTIONS TO CHECK YOUR THINKING
Assessment	Selecting the correct assessment instrument Making reliable observations Distinguishing relevant from irrelevant data Distinguishing important from unimportant data Validating data Organising data Categorising data according to a framework Recognising assumptions	What assumptions am I making about the person? Are my data correct and accurate? How reliable are my sources? What data are important? Relevant? What biases do I have that might cause me to miss important information? Am I listening carefully to get the person's and family's perspective? Do I have all the facts? What other data might I need?
Diagnosis	Finding patterns and relationships among cues Identifying gaps in the data Making inferences Suspending judgment when lacking data Making interdisciplinary connections Stating the problem Examining assumptions Comparing patterns with norms Identifying factors contributing to the problem	Do I know what is within normal limits for the data? Do I have enough data to make a valid inference? What biases might I have that could affect how I see the person's problems? Do I have enough data to make a nursing diagnosis or should I make a 'possible' diagnosis? What other problems might this data suggest other than the one that seems most obvious to me?
Planning	Forming valid generalisations Transferring knowledge from one situation to another Developing evaluative criteria Hypothesising Making interprofessional connections Prioritising the person's problems Generalising principles from other sciences	Do I need help to plan interventions or am I qualified to do it? Did I remember to give high priority to the problems the person and family identified as important? What are the most important problems we need to address? What interventions worked in similar situations? Is this situation similar enough to merit using them with this person? Are there other plans that might be more agreeable to the person and therefore more likely to work? Why do I expect these interventions to be effective? Based on what knowledge?
Implementation	Applying knowledge to perform interventions Using interventions to test hypotheses	Has the person's condition changed since the plan was made? Have I overlooked any new developments? What is the person's initial response to the intervention? Are there any safety issues I have overlooked?
Evaluation	Deciding whether hypotheses are correct Making criterion-based evaluations	What are the person's responses after the interventions? Have I overlooked anything? Do the data indicate that goals were met? Does the person feel their goals were met? Does the person trust me enough to give honest answers? Am I sure the problem is really resolved? What might we have done that would have been more effective? What nursing care is still needed, if any?

Source: Nursing process and critical thinking (5th ed.) by J. M. Wilkinson (2011), pp. 325–327. Electronically reproduced by permission of Pearson Education, Inc., Upper Saddle River, NJ.

status of an actual or potential problem previously identified (Alfaro-LeFevre, 2009).

To make accurate and holistic assessments, nurses must have and use a wide variety of knowledge and skills. The ability to assess the physical, emotional and mental status of the person is essential, as is the ability to use effective communication techniques. Nurses must be knowledgeable in pathophysiology and pharmacology and be able to identify abnormal

laboratory and diagnostic test data. Finally, nurses must have a solid foundation of nursing knowledge and skills that will enable them to interpret assessment data and to use that interpretation as the basis for individualised care.

Diagnosis

The nurse examines each cluster of data (or pattern) derived from the assessment to develop appropriate nursing diagnoses. Nursing diagnoses are clinical judgments about a person's actual or potential health problems. Nursing diagnoses provide the basis for determining nursing interventions to achieve outcomes for which the nurse is accountable. Nurses then develop and implement a plan of care to address health concerns and prevent illness.

Writing a nursing diagnosis

A nursing diagnosis is generally written in two or three parts and often joined by the phrases 'related to' and 'manifested by'. The first part of the statement is the issue or problem that has been identified from the examination of the data collected during the patient assessment. The part of the statement that follows the phrase 'related to' identifies the physical, psychosocial, cultural, spiritual and/or environmental factors (aetiologies) that cause or contribute to the occurrence of the problem.

Many nurses write nursing diagnoses using the following method:

1. The problem.
2. The aetiology of the problem, which identifies the related factors.
3. The signs and symptoms, which are the defining characteristics of the problem and are indicated by the phrase 'manifested or evidenced by'.

Examples of nursing diagnoses include:

- *Faecal incontinence* related to loss of sphincter control, manifested by frequent and involuntary passage of stool.
- *Acute pain* related to inadequate education about patient-controlled analgesia (PCA) use, manifested by withdrawal, grimacing, restlessness and guarded positioning.
- *Fatigue* related to the side effects of chemotherapy, evidenced by exhaustion when undertaking activities of daily living.

Planning

During the planning step, the nurse identifies appropriate evidence-based nursing interventions (actions) and outcomes to improve health and/or to prevent or ameliorate ill health. These outcomes are usually developed collaboratively by the person and nurse and identify what the person will be able to do as a result of the care provided (Alfaro-LeFevre, 2009). For example, '30 minutes following administration of analgesic medication the person reports a reduction in pain from 8 to 3 on the numeric rating scale'.

Implementation

The implementation step is the action phase of the nursing process during which the nurse carries out planned interventions. Ongoing assessment of the person before, during and after the intervention is an essential component of implementation. Although the plan may be appropriate, many factors can influence how the person responds, making a revision to the plan necessary. For example, the nurse would not be able to encourage fluid intake if the person became nauseous. Additionally, the nurse should be aware of the interrelated nature of nursing interventions. For example, while giving a bed bath the nurse can assess the person's skin condition and at the same time use therapeutic communication to provide comfort.

Documenting interventions is the final component of implementation and is a legal requirement. Many different methods are used to document care, including problem-oriented charting, charting by exception and electronic documentation. Additionally, systems assessments are becoming increasingly common for documentation of progress notes and the development of nursing care plans.

Evaluation

The evaluation step allows the nurse to determine whether the actions taken were effective and whether to continue, revise or terminate the plan of care. The outcome criteria (goals) that were established during the planning step provide the basis for evaluation. Although evaluation is listed as the last part of the nursing process, it takes place continuously throughout each person's care. To evaluate a plan, the nurse collects data from the person and their clinical records. If the outcomes have not been accomplished, the nurse must modify the nursing diagnoses, outcomes or plan.

The nursing process in clinical practice

Experienced nurses may not consciously stop and consider each step of the nursing process. For example, when caring for a person who is haemorrhaging, the nurse would use all five steps simultaneously to meet critical, life-threatening needs. In contrast, when considering long-term needs for a person with a chronic illness or disability, the nurse makes in-depth assessments, mutually determines goals with the person, and provides documentation through a written plan of care that can be developed over time and revised as necessary by all nurses providing care. As a nurse becomes an expert clinician, the nursing process becomes so much a part of their practice that they may not even consciously consider it while providing care (Benner, 1984)

CLINICAL REASONING

Clinical reasoning is often used interchangeably with the terms 'clinical judgment', 'problem solving', 'decision making' and 'critical thinking'. While in some ways the terms are similar, clinical reasoning is a cyclical process that often leads to a series of linked clinical encounters. **Clinical reasoning** can be defined as 'the process by which nurses (and other clinicians) collect cues, process the information, come to an understanding of a person's problem or situation, plan and implement interventions, evaluate outcomes, and reflect on and learn from the process' (Levett-Jones et al., 2010, p. 516; Hoffman, 2007). Clinical reasoning can be influenced by the nurse's assumptions, perspectives, attitudes and preconceptions (McCarthy,

2003) and the capacity for self-awareness of one's cognitive biases is essential to patient safety.

Over the past decade research has identified the need for an explicit and sophisticated model to both explain how expert nurses think and as a foundation for nursing education (Levett-Jones et al., 2010, p. 516; Hoffman, 2007). A model titled the Clinical Reasoning Cycle (CRC) was developed by Australian nurse researchers to clearly articulate how nurses use sophisticated thinking skills to inform their practice decisions and enhance patient safety (see Figure 1.2). The CRC builds upon the nursing process framework and represents the multifaceted and increasingly complex nature of nursing care, and the necessity to respond appropriately to patients' needs, particularly in emergent, non-routine and unpredictable clinical situations. The CRC is integral to students' developing ability to 'think like a nurse' and its explicit meta-cognitive and reflective processes aim to identify and prevent cognitive errors that may lead to adverse patient outcomes. Nurses with inadequate clinical reasoning skills often fail to detect and appropriately respond to patient deterioration (Aiken et al., 2003). Poor clinical reasoning and critical thinking skills have been identified as a factor in more than 50% of adverse clinical events (Wilson et al., 1995).

The Clinical Reasoning Cycle consists of eight main stages or steps: *consider the patient situation, collect cues, process information, identify problems and priorities, establish goals, take action, evaluate outcomes and reflect on process and new learning.*

ACCOUNTABLE AND RESPONSIBLE NURSING PRACTICE

The Australian Nursing and Midwifery Accreditation Council (ANMAC) is responsible for accreditation of nursing and midwifery programs while the Nursing and Midwifery Board of Australia (NMBA) has responsibility for professional registration, professional codes, standards and competency issues.

In Australia there are three key documents that form the basic framework for accountable and responsible practice as a Registered Nurse. These are:

1. Nursing and Midwifery Board of Australia (NMBA) The *National Registered Nurse Standards for Practice (2016).*
2. Nursing and Midwifery Board of Australia (NMBA) *Code of Ethics for Nurses in Australia (2008).*
3. Nursing and Midwifery Board of Australia (NMBA) *Code of Professional Conduct for Nurses in Australia (2008).*

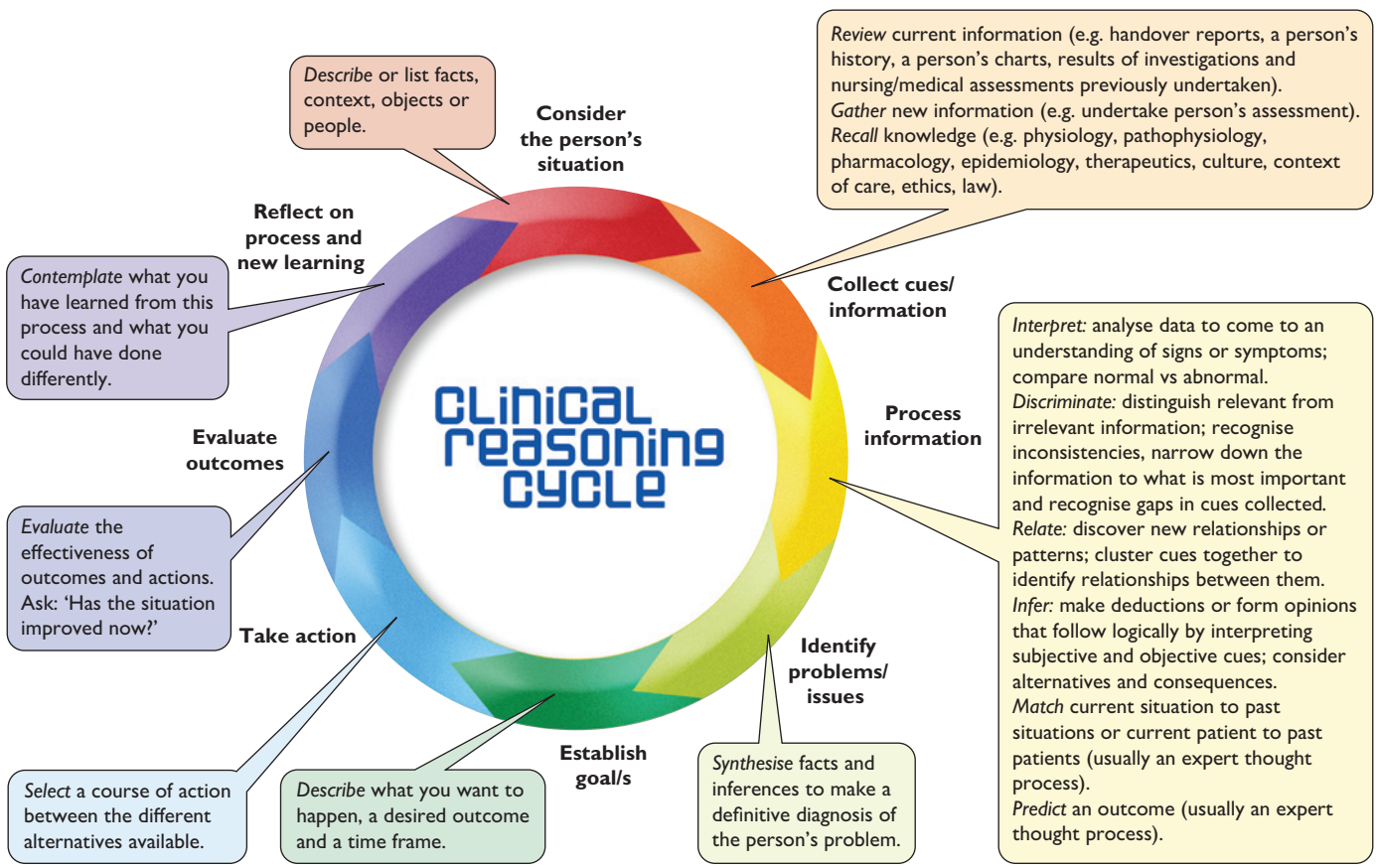

FIGURE 1.2 ■ The clinical reasoning process with descriptors

Source: T. Levett-Jones (2013). *Clinical reasoning: Learning to think like a nurse*, p. 7; adapted from T. Levett-Jones et al. (2010). The 'five rights' of clinical reasoning: An educational model to enhance nursing students' ability to identify and manage clinically 'at risk' patients. *Nurse Education Today*, *30*(6), 515–520.

These key documents are supported by other published practice standards and guidelines such as decision-making frameworks, position statements, professional boundaries and practice guidelines.

Nursing and Midwifery Board of Australia (NMBA) *National Registered Nurse Standards for Practice*

The *National Registered Nurse Standards for Practice (2016)* were developed to promote a national approach to nursing in Australia. These standards are an integral component of the nursing regulatory framework that assists nurses to deliver safe and competent care. They are the standards by which a nurse's performance is assessed to obtain and retain registration to practise as a nurse in Australia. As the ever-changing healthcare needs and expectations of Australians impact on quality and safety within the healthcare system, so must practice standards be regularly reviewed by the nursing profession. The NMBA standards for practice were reviewed and updated during 2015 and were published early in 2016. The standards are organised into domains, as illustrated in Box 1.1.

NMBA Code of Ethics

The NMBA (2013a) *Code of Ethics for Nurses in Australia (2008)* is framed by principles and standards from the United Nations and the World Health Organization (WHO) (1974). The code outlines the nursing profession's commitment to respect, promote, protect and uphold the fundamental rights of people who are both the recipients and providers of nursing and healthcare. The code provides nurses with a reference point from which to reflect on the conduct of themselves and others. It is also a guide to ethical decision making and practice (NMBA, 2013a).

BOX 1.1 Domains of the Nursing and Midwifery Board of Australia (NMBA) *National Registered Nurse Standards for Practice*

1. Thinks critically and analyses nursing practice	Nurses use a variety of thinking strategies, research and best available evidence in making decisions and providing safe, quality nursing practice within a person-centred framework.
2. Engages in therapeutic and professional relationships	Nursing practice is based on purposefully engaging in the formation and maintenance of effective therapeutic and professional relationships. This includes collegial generosity in the context of interdisciplinary and professional relationships.
3. Maintains fitness for practice and participates in lifelong learning	Registered Nurses, as regulated health professionals, are responsible and accountable for ensuring they are safe and have the capability for practice. This includes ongoing self-management and responding when there are concerns about other health professionals' fitness for practice. Registered Nurses are responsible for their professional development and contribute to the development of others. They are also responsible for providing information and education to enable people to make decisions and take action in relation to their health.
4. Comprehensively conducts assessments	Registered Nurses accurately conduct comprehensive and systematic assessments, analyse information and data and communicate outcomes as the basis of practice.
5. Develops a plan for nursing practice	Registered Nurses are responsible for the planning and communication of nursing practice. Agreed plans are developed in partnership. They are based on the Registered Nurse's comprehensive assessment, use of evidence and judgment that is documented and communicated to all the relevant persons.
6. Provides safe, appropriate and responsive quality nursing practice	Registered Nurses delegate and implement person-centred, quality and ethical goal-directed actions. These are based on comprehensive and systematic assessment, and the best available evidence to achieve planned outcomes.
7. Evaluates outcomes to inform nursing practice	Registered Nurses take responsibility for the evaluation of practice based on agreed outcomes to plan and revise practice accordingly.

Source: Nursing and Midwifery Board of Australia (NMBA) (2016). *National Registered Nurse Standards for Practice.* © Nursing and Midwifery Board of Australia, www.nursingmidwiferyboard.gov.au/News/2016-02-01-revised-standards.aspx.

BOX 1.2 Nursing and Midwifery Board of Australia Code of Ethics for Nurses—Value Statements

1. Nurses value quality nursing care for all people.
2. Nurses value respect and kindness for self and others.
3. Nurses value the diversity of people.
4. Nurses value access to quality nursing and healthcare for all people.
5. Nurses value informed decision making.
6. Nurses value a culture of safety in nursing and healthcare.
7. Nurses value ethical management of information.
8. Nurses value a socially, economically and ecologically sustainable environment promoting health and wellbeing.

Source: Nursing and Midwifery Board of Australia (NMBA) (2013a). *Code of Ethics for Nurses in Australia (2008).* © Nursing and Midwifery Board of Australia, www.nursingmidwiferyboard.gov.au/Codes-Guidelines-Statements/Professional-standards.aspx.

In addition, the *International Council of Nurses' (ICN) Code of Ethics for Nurses* (2012) specifies what nurses are accountable for in terms of people, practice, society, co-workers and the profession. The philosophical base for the ICN code is that nurses are responsible for promoting health, preventing illness, restoring health and alleviating suffering. The *Code of Ethics for Nurses in Australia* should be read in conjunction with the ICN code and related ICN position statements (NMBA, 2013a). Box 1.2 lists the eight value statements to which the Australian nursing profession is committed.

NMBA Code of Professional Conduct

Professional conduct is defined as the manner in which a person behaves while acting in a professional capacity. The *Code of Professional Conduct for Nurses in Australia (2008)* sets the minimum standards for practice that a professional person is expected to uphold, both within and outside professional domains, in order to ensure the 'good standing' of the nursing profession (NMBA, 2013b). This code is a companion to, and should be read in conjunction with, the *Code of Ethics for Nurses in Australia (2008)* (NMBA, 2013a). The code describes ten conduct statements, which are listed in Box 1.3.

BOX 1.3 NMBA Code of Professional Conduct Statements

1. Nurses practise in a safe and competent manner.
2. Nurses practise in accordance with the standards of the profession and broader health system.
3. Nurses practise and conduct themselves in accordance with laws relevant to the profession and practice of nursing.
4. Nurses respect the dignity, culture, ethnicity, values and beliefs of people receiving care and treatment, and of their colleagues.
5. Nurses treat personal information obtained in a professional capacity as private and confidential.
6. Nurses provide impartial, honest and accurate information in relation to nursing care and healthcare products.
7. Nurses support the health, wellbeing and informed decision making of people requiring or receiving care.
8. Nurses promote and preserve the trust and privilege inherent in the relationship between nurses and people receiving care.
9. Nurses maintain and build on the community's trust and confidence in the nursing profession.
10. Nurses practise nursing reflectively and ethically.

Source: Nursing and Midwifery Board of Australia (NMBA) (2013b). *Code of Professional Conduct for Nurses in Australia (2008).* © Nursing and Midwifery Board of Australia, www.nursingmidwiferyboard.gov.au/Codes-Guidelines-Statements/Professional-standards.aspx.

CULTURALLY COMPETENT NURSING

The primary focus of nursing care is the person and how they respond to their environment and experiences or situations related to health or illness. These experiences are given shape and personal meaning by **culture**—the socially inherited characteristics of a human group. The healthcare system encompasses many people (staff and patients) who are culturally diverse. This diversity includes differences in country of origin, health beliefs, sexual orientation, race, socioeconomic level and age.

Culture influences us all in our work, home and social lives. We therefore need to understand what the term 'culture' means. Rosenjack-Burcham (2002) defines culture as 'a learned world viewpoint or paradigm shared by a population or group and transmitted socially. It influences values, beliefs, customs and behaviours, and is reflected in the language, dress, food, materials and social interactions of a group' (p. 7). In Leininger's seminal work (1991) culture is described as 'the learned and transmitted values, beliefs and practices . . . the blueprint for living, remaining healthy, or for dying' (p. 36). Culture is primarily learned in our family or community life and can be shared with others. Our own culture can be experienced by us in an unconscious way and can change over time. Culture is therefore different from ethnicity, which is determined at birth. Interacting across cultures requires us to be aware of our own culture and requires an understanding of and skill in interpersonal and group communication.

Increasing cultural and ethnic diversity in most regions of the world over the past 40 years has made provision of culturally competent care essential for nurses and other health professionals (Everson et al., 2015). However, studies indicate that people from non-English-speaking backgrounds experience twice as many adverse health events as English-speaking people (Divi et al., 2007), with misunderstandings, miscommunication and culturally unsafe care by health professionals

frequently reported (Johnstone & Kanitsaki, 2008). Many factors account for culturally unsafe care, including lack of awareness, skills and empathy, as well as ethnocentrism (people's belief that their own cultural group's beliefs and values are the only acceptable ones) and prejudice.

People of every culture have the right to have their cultural values known, respected and addressed appropriately in nursing and other healthcare services (Leininger, 1991). To provide nursing care that is culturally competent, nurses must develop sensitivity to personal fundamental values about health and illness; must accept the existence of differing values; and must be respectful of, interested in, and empathetic towards people from different cultures, without being judgmental. **Cultural competence** is essential to quality care. According to Betancourt et al. (2003) cultural competence 'entails understanding the importance of social and cultural influences on patients' health beliefs and behaviours, considering how these factors interact at multiple levels of the health care delivery system, and devising interventions that take these issues into account' (p. 294).

A related concept, **cultural safety**, is 'The effective nursing practice of a person or family from another culture, as determined by that person or family' (Nursing Council of New Zealand (NCNZ), 2012, p. 32). Unsafe cultural practice comprises any action that 'diminishes, demeans or disempowers the cultural identity and wellbeing of an individual' (NCNZ, 2012, pp. 32–33).

Cultural competence and cultural safety allow nurses to connect with and understand people receiving care as well as their families and friends, their community and the profession of nursing. Through communication nurses are offered an opportunity to understand people's experience of health and illness at a personal level rather than simply at the theoretical or biomedical level. Effective communication helps to foster an environment in which culturally safe care is negotiated and delivered.

The NMBA *Code of Ethics for Nurses in Australia (2008)* (NMBA, 2013a) states that Registered Nurses:

- accept individuals/groups to whom care is provided regardless of race, culture, religion, age, gender, sexual preference, or physical or mental state
- ensure that personal values and attitudes are not imposed on others
- maintain an effective process of care when confronted by differing values, beliefs and biases.

Standard 2 of the NMBA *National Standards for Practice* (2016) refers to therapeutic relationships and indicates that Registered Nurses must 'establish, sustain and conclude therapeutic relationships in a way that is respectful and acknowledges the dignity, culture, values and beliefs and rights of a person'.

While developing a growing appreciation of the various cultural groups you come into contact with in your many nursing roles is essential, developing an appreciation and understanding of the history and culture of Aboriginal and Torres Strait Islander people (Australia's First People) is fundamental to the development of professional nurses and to nursing practice which is experienced as culturally safe. Standard 1 of the NMBA *National Standards for Practice* (2016) relates to critical thinking and analysis, and Element 1.3 advocates that Registered Nurses 'Respect peoples' culture and experiences as a core part of person-centred and evidence-based practice, which includes recognising the role of family and community that underpin Aboriginal and Torres Strait Islander cultures and health'.

In order to be effective in delivering appropriate care to Aboriginal and Torres Strait Islander people nurses need:

- awareness of important Aboriginal and Torres Strait Islander issues such as cultural differences, and specific aspects of Indigenous history and its impact on Indigenous peoples in contemporary Australian society
- the skills to interact and communicate sensitively and effectively with Indigenous peoples
- the motivation to interact successfully with Indigenous peoples in order to improve access, service delivery and patient outcomes (Farrelly & Lumby, 2009).

Undertaking this journey into the history and culture of Indigenous Australians is likely to challenge your understanding of your own culture and how your cultural values impact on the way you provide nursing care to all people.

In the chapters that follow the cultural implications of the various clinical situations are discussed and expanded upon, and you will be presented with opportunities to apply and contextualise your learning about cultural safety.

LEGAL AND ETHICAL DILEMMAS IN NURSING

A **dilemma** is a choice between two unpleasant, ethically troubling alternatives. Nurses face dilemmas almost daily—so many, in fact, that a complete discussion of them is impossible here. However, many commonly experienced dilemmas involve confidentiality, human rights and issues of dying and death. The nurse must use ethical and legal guidelines to make decisions about moral actions when providing care in these and many other situations.

The rights of each individual can result in dilemmas for nurses in the clinical setting. For example, the right to privacy and confidentiality may create a dilemma if it conflicts with the nurse's right to information that may affect their personal safety. The right to refuse treatment (including surgery, medication, nutrition and hydration) is an example of people's rights that can cause nursing dilemmas. The situation, the alternatives and the potential consequences of refusal must be carefully explored with the person.

The issues surrounding dying and death have become increasingly topical as advances in technology extend the lives of people with chronic debilitating illness and major trauma. These changes have altered concepts of living and dying, resulting in ethical dilemmas regarding quality of life and death with dignity versus technological preservation of life. Additionally, difficulties in establishing competence to make informed decisions about withholding and withdrawing treatment, and use of narcotic analgesia at the end of life, are some issues that nurses will encounter in their practice.

ROLES OF THE NURSE IN MEDICAL–SURGICAL NURSING PRACTICE

Healthcare today is a vast and complex system. It reflects changes in society, changes in the populations requiring nursing care and a philosophical shift towards health promotion rather than illness care. The roles of the medical–surgical nurse have broadened and expanded in response to these changes. Medical–surgical nurses are not only caregivers but also educators, advocates, leaders and managers, and researchers. The nurse assumes these various roles to promote and maintain health, to prevent illness and to facilitate coping with disability or death for people in a range of healthcare settings.

The nurse as caregiver

Nurses have always been caregivers. However, the activities carried out within the caregiver role have changed tremendously in the 21st century. From 1900 to the 1960s, the nurse was almost always female and was regarded primarily as the person who gave personal care and carried out doctors' orders. This dependent role has changed as a result of the increased education of nurses, research into and the development of nursing knowledge, a strong evidence base and the recognition that nurses are autonomous and well-informed professionals.

The caregiver role for the nurse today is both independent and collaborative. Nurses independently make assessments and plan and implement patient care, based on nursing knowledge and skills. Nurses must also collaborate with other members of the interprofessional healthcare team to implement and evaluate care (see Figure 1.3).

In providing comprehensive and person-centred care the nurse uses critical thinking skills to analyse and synthesise knowledge from the arts, the sciences, and nursing research and theory. The science (knowledge base) of nursing is translated into the art of nursing through caring. Caring is the means by which the nurse is connected with and concerned for the person who is the recipient of care. Thus, the nurse as caregiver is knowledgeable, skilled and empathetic. Nursing care must address not only the physical needs but also the psychosocial, cultural, spiritual and environmental needs of each person and their family. Considering all aspects of a person's being ensures a holistic approach to nursing (Sharoff, 2006). Subsumed within the concept of 'holistic healthcare' is the concept of caring for the mind, body and spirit.

FIGURE 1.3 ■ The healthcare team discusses the individualised plan of care and outcomes

Source: Arno Massee/Science Photo Library/Alamy.

The nurse as educator

The nurse's role as educator is becoming increasingly important for several reasons. There is much greater emphasis on health promotion and illness prevention; hospital stays are becoming shorter; and the number of people with chronic illnesses in our society is increasing. Early discharge of people from the hospital setting to the home means that family caregivers must learn how to perform complex skills. All these factors make the educator role essential to maintaining the person's health and wellbeing.

The framework for the role of educator is the teaching–learning process. Within this framework the nurse assesses learning needs, plans and implements teaching methods to meet those needs, and evaluates the effectiveness of the teaching. To be effective educators nurses need effective interpersonal skills and familiarity with adult learning principles (see Figure 1.4).

A major component of the educator role today is discharge planning. Discharge planning, which begins on admission to a healthcare setting, is a systematic method of preparing the person and their family for departure from the healthcare facility and for maintaining continuity of care after they leave the setting. Discharge planning also involves making referrals, identifying community and personal resources, and arranging for necessary equipment and supplies for home care.

The nurse as advocate

The person entering the healthcare system may not always be prepared to make independent decisions. However, nurses need to be aware that today's healthcare consumer is better educated

FIGURE 1.4 ■ The nurse's role as educator is an essential component of care. As part of the discharge planning process, the nurse is responsible for teaching for self-care at home

Source: © Monkey Business Images/Shutterstock.com.

about options for care, and may have very definite opinions. The nurse as patient advocate actively promotes the patient's right to autonomy and free choice. The nurse as advocate speaks for the person, mediates between the person and other people, and/or protects the person's right to self-determination. The goals of the nurse as advocate are to:

- assess the need for advocacy
- communicate with other healthcare team members
- provide teaching to the person and family
- assist and support decision making
- serve as a change agent in the healthcare system
- participate in health policy formulation.

The nurse must practise advocacy while maintaining the belief that people have the right to choose treatment options, based on information about the results of accepting or rejecting the treatment, without coercion. The nurse must also accept and respect the decisions of the person, even though they may differ from the decisions the nurse would make.

The nurse as leader and manager

All nurses are leaders and managers. They practise leadership and they manage time, people, resources and the environment in which they provide care. Nurses carry out these roles by directing, delegating and coordinating nursing activities. Nurses must be knowledgeable about how and when to delegate, as well as the legal requirements of delegation. Nurses also evaluate the quality of care provided.

Models of care delivery

Nurses are leaders and managers of patient care within a variety of models of care delivery. Models of care may include:

- task-oriented nursing
- team nursing
- patient allocation or total patient care
- primary nursing
- case management.

Task-oriented nursing refers to a model in which nurses undertake specific tasks related to nursing care across a group of people. Some examples of task allocation may be when a nurse undertakes to shower all people in a ward; another nurse may undertake the medications for the same group of people. In this model of care delivery, nursing care relates to sets of activities that are performed by nurses for people.

Team nursing is a model that 'teams' experienced permanent nurses with less experienced or casual staff to achieve nursing goals using a group approach. A team may consist of a Registered Nurse, an Enrolled Nurse and an Assistant in Nursing. The Registered Nurse is the team leader. The team leader is responsible for making assignments and has overall responsibility for patient care by team members. All team members work together, each performing the activities for which they are best prepared.

Patient allocation models were developed because nurses recognised the need for total patient care. The implementation of these types of models results in nurses getting to know the whole person, rather than people being cared for as a series of tasks. A nurse will be allocated to their patients (the number is dependent on factors such as patient need, staff mix and ward policies) and undertake all nursing care for the allocated person/people.

Primary nursing allows the nurse to provide individualised direct care to a small number of people during their entire inpatient stay. This model was developed to reduce the fragmentation of care experienced by the person and to facilitate continuity of care. In primary nursing the nurse provides care; communicates with the person, families and other healthcare providers; and carries out discharge planning.

Case management focuses on management of a caseload (group) of patients. The purpose of case management is to maximise positive outcomes and contain costs. The nurse who is case manager is usually a clinical specialist, and the caseload consists of people with similar healthcare needs. As case manager the nurse makes appropriate referrals to other healthcare providers and manages the quality of care provided, including accuracy, timeliness and cost. The case manager is also in contact with patients after discharge, ensuring continuity of care and health maintenance.

The model of care delivery implemented on a ward will depend on a range of factors, including the degree of innovation and commitment by the persons involved. Some models work better when there are sufficient numbers of highly qualified staff (RNs) to deliver care; others may focus on supporting less experienced staff using a team approach.

Delegation

Delegation is carried out when the nurse assigns appropriate work activities to other members of the healthcare team. When the nurse delegates nursing care activities to another person, that person is authorised to act in the place of the nurse, although the nurse retains the accountability for the activities performed. Delegation skills are becoming increasingly important in healthcare as facilities restructure and implement cost containment measures. Delegation depends on knowing one's own scope of practice and that of the person to whom one plans to delegate.

Nurses' **scope of practice** refers to the roles, functions, responsibilities, activities and decision-making capacity that they are educated, competent and authorised to perform. One's scope of practice is influenced by the wider environment, the specific setting, legislation, policy, education, standards and the health needs of the population. Registered Nurses have a key role in the coordination and supervision of others who assist them in the provision of care to people. A decision-making template developed by the ANMC (2007) provides guidance not only for individual practice decisions by Registered Nurses but also for decisions about if, and when, it is appropriate to delegate aspects of patient care to others.

Evaluating outcomes of nursing care

CLINICAL PATHWAYS A **clinical pathway** is a plan designed to provide healthcare, often within a multidisciplinary team. Such pathways are generally developed for specific diagnoses—usually high-volume, high-risk and high-cost case types—with the collaboration of members of the healthcare

team. This patient care management tool describes how resources will be used to achieve predetermined outcomes. It also establishes the sequence of multidisciplinary interventions, including education, discharge planning, consultations, medication administration, diagnostics, therapeutics and treatments.

The goals of clinical pathways are to:

- achieve realistic, expected person and family outcomes
- promote professional and collaborative practice and care
- ensure continuity of care
- guarantee appropriate use of resources
- reduce costs and length of stay
- provide the framework for continuous improvement.

Clinical pathways are often used in conjunction with case management models and/or quality improvement efforts. The overall goal is to design pathways that facilitate a reproducible standard of care for specific patient populations and improve the quality and proficiency of that care.

The healthcare facility determines the process for developing a clinical pathway. Information imperative to the development of any clinical pathway includes literature reviews, chart reviews and expert opinion. A typical approach is to first identify high-cost, high-volume and high-risk case types for the agency. Next, a multidisciplinary team develops a consensus around the management of the case type and a clinical pathway. The pathway is then piloted with a designated group of people, and revised based on the number and types of variances. The goal is to develop a pathway that best meets the needs of people in the particular practice setting.

When people do not achieve expected outcomes, variances (deviations from the established plan) from the clinical pathways are recorded and studied by the multidisciplinary team. In many facilities, clinical pathways are designed so that interventions and variances can be easily documented. Most documentation systems require a check-off when interventions are performed or variances occur.

In many facilities, clinical pathways are replacing traditional nursing care plans. The advantages of clinical pathways are that they are outcome driven and provide a timeline to achieve specified goals. Additionally, clinical pathways provide opportunities for healthcare workers to collaborate and establish dynamic plans of care that consider all of the people's needs. Although initially developed for acute hospitalisations, clinical pathways are now being developed to manage people in the home, outpatients and those in long-term settings.

Clinical governance

Clinical governance is defined as a systematic and integrated approach that improves quality and safety and results in optimal patient outcomes (Office of Safety and Quality in Health Care, 2001). Health jurisdictions in Australia have embraced clinical governance by attempting to hold healthcare providers accountable for the quality and safety of the care they deliver. Clinical governance places the responsibility for the quality of care jointly on organisations and on individuals within organisations. As a leader and manager within a healthcare organisation, the nurse has an important role in promoting continuous quality improvement through governance structures such as:

- clinical risk management
- clinical quality and safety frameworks
- consumer participation
- clinical effectiveness
- clinical audit
- evidence-based practice
- credentialling/professional development
- research and development.

The medical–surgical nurse is well placed to contribute to the evaluation of the quality of clinical practice through peer review, clinical audit and external accreditation processes.

The nurse as researcher

Nurses have always identified problems in patient care. Although they have developed interventions to meet specific needs, the activities often have not always been conducted within a scientific framework or communicated to other nurses through nursing literature. To develop the science of nursing, nursing knowledge is established through clinical research and then published so that the findings can be used by all nurses to provide evidence-based person-centred care. This means that all nurses must consider the researcher role as integral to nursing practice.

Over the past decade there has been much more attention placed on the importance of evidence-based healthcare and nurses are expected to use the best clinical evidence available to inform their patient care decisions. Evidence from rigorous

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 1:
Governance for Safety and Quality in Health Service Organisations

Standard 1 of the National Safety and Quality Health Service Standards (NSQHSS) specifies that:

- There are integrated systems of governance to manage patient safety and quality risks.
- Care provided by the clinical workforce is guided by current best practice.
- All members of the clinical workforce have the appropriate qualifications and skills to provide safe high-quality healthcare.
- Patient safety and quality incidents are recognised, reported and analysed so as to improve safety systems.
- Patient's rights are respected and participation in their care supported.

Source: © Australian Commission on Safety and Quality in Health Care.

studies constitutes the best type of evidence to underpin nurses' decisions and actions. Nursing care that is based on high-quality research evidence is more likely to be cost effective and result in positive patient outcomes (Joanna Briggs Institute, 2013).

Summaries of relevant nursing research are included in almost all of the nursing care chapters of this textbook. After the summary and discussion of each study, a critical thinking section specifically related to the findings of the study encourages students to apply the findings in the clinical setting.

CHAPTER HIGHLIGHTS

- Safe and effective nursing care focuses on the provision of person-centred care, working in interprofessional teams, using evidence-based practice and working within legal and ethical frameworks.
- The nursing process is an approach used by nurses to provide care to promote wellness, maintain health, restore health or facilitate coping with disability or death. The five steps of the nursing process are assessment, diagnosis, planning, implementation and evaluation.
- Clinical reasoning is a dynamic process in which nurses collect cues, process this information, come to an understanding of the situation (or patient's problem), plan and implement care, evaluate outcomes, and reflect on and learn from the process. The questioning of assumptions and the avoidance of clinical reasoning errors are central to this process.
- The clinical practice of nurses is guided by codes of conduct, codes of ethics and standards of practice.
- Nurses function as caregivers, educators, advocates, leaders and managers, and researchers to promote and maintain health, prevent illness and facilitate coping with disability or death for the adult person.

CONCEPT CHECK

1 The Nursing and Midwifery Board of Australia (NMBA) has developed a set of standards for practice. What is the primary purpose of these standards?
1 to make all nurses equal
2 to promote safe and effective nursing care
3 to reduce the number of legal actions
4 to provide a set of ethical guidelines

2 What does the nurse use in the clinical setting to make clinical judgments and decisions?
1 nursing process
2 standards of care
3 clinical reasoning skills
4 all of the above

3 Which of the following statements is true of outcomes developed during the planning phase of the nursing process?
1 Outcomes are mutually established by the person and the nurse.
2 Outcomes are mutually established by the nurse and the doctor.
3 Outcomes are mandated by institutional policies and standards.
4 Outcomes are written by the person receiving care and their family members.

4 The steps of the nursing process are used when providing care. From the list below, select the order in which the steps are most often used.
1 diagnosis
2 assessment
3 evaluation
4 implementation
5 planning

5 When nurses discuss the 'science of nursing', what does this phrase mean?
1 clinical competency
2 holistic care
3 evidence-based practice
4 practice component

6 What role does the nurse demonstrate when appraising health information?
1 advocate
2 caregiver
3 researcher
4 educator

7 What goal is a component of the nurse's role as advocate?
1 assisting and supporting the person in their decision making
2 conducting research about the effects of exercise
3 delegating responsibilities for care to others
4 performing range-of-motion exercises

8 A nurse assigns appropriate work activities to other members of her team. What role is being illustrated?
1 advocate
2 leader/manager
3 researcher
4 caregiver

9 A method of establishing a standard of care and evaluating outcomes of that standard involves:
1 writing a dress-code policy for a healthcare agency
2 creating a clinical pathway for a specific type of person
3 establishing clinical governance approaches
4 implementing a new procedure to change dressings

10 A Registered Nurse delegates vital signs assessment to an Assistant in Nursing. Who is accountable for the assessment findings?
1 the Assistant in Nursing
2 the person receiving care
3 the nurse
4 the doctor

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CHAPTER 2

HEALTH AND ILLNESS IN ADULTS

AMANDA WILSON, TRACY LEVETT-JONES

KEY TERMS

LEARNING OUTCOMES

- Define health, including the concepts of the illness–wellness continuum and high-level wellness.
- Explain what determinants of health are and how they influence health, disease and illness.
- Discuss the nurse's role in promoting healthy lifestyles and preventing illness and injury.
- Describe the different behaviours and needs of people with acute and chronic illness.
- Describe the primary, secondary and tertiary levels of illness prevention.
- Compare and contrast the physical status, changes in health, assessment guidelines and healthy behaviours of the young adult, middle adult and older adult.
- Explain the definitions, functions and developmental stages and tasks of the family.

CLINICAL COMPETENCIES

- Include knowledge of developmental levels and of activities to promote, restore and maintain health when planning and implementing care for adults.
- Include family members in teaching to promote and maintain health of the adult.

DEFINING HEALTH

The Constitution of the World Health Organization (WHO) defines **health** as 'a state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity' (WHO, 1948, p. 1). This definition is fundamental to contemporary healthcare perspectives and can be expanded to encompass the various levels of health people experience throughout their life. However, the concept of health is subjective and reflects an individual's perspective of quality of life. For example, someone with a terminal diagnosis can still enjoy life and feel 'well'—however, their definition of health will differ radically from that of a person without that diagnosis. These factors, which greatly influence nursing care, are reflected in the **illness–wellness continuum** and the concept of high-level wellness.

The illness–wellness continuum

The illness–wellness continuum represents health as a dynamic process, with high-level wellness at one extreme of the continuum and death at the opposite extreme (see Figure 2.1). During our lifetime we would place ourselves in different locations on this continuum. The continuum of health and illness was expanded to include the concept of high-level wellness, with 'good health' differentiated from 'wellness' in the following way:

> *Good health can exist as a relatively passive state of freedom from illness in which the individual is at peace with his environment . . . Wellness is an integrated method of functioning, which is oriented toward maximising the potential of which the individual is capable, within the environment where he is functioning.* (Dunn 1959, p. 4)

The philosophy of **holistic healthcare** involves the concept that person-centred care considers all facets of an individual including physical, psychosocial, cultural, spiritual and intellectual. Similarly, the elements of wellness include self-perception, environment, culture and spiritual values. To promote, maintain or restore health, both the nurse and the person receiving care need to recognise and address this framework of wellness factors.

DETERMINANTS OF HEALTH

Australia has a population of approximately 24 million people (Australian Bureau of Statistics (ABS), 2015). Australians today are living 25 years longer than their great-grandparents who were born a century ago. Females can now expect to live to 84 years and men 80 years (Australian Institute of Health and Welfare (AIHW), 2015a). There are many factors that affect health and wellness. These factors are referred to as **determinants of health** and they can influence good health or be risk factors for poor health. The following sections describe some of the major determinants of health and their impact.

Genetic make-up

Genetic make-up is a 'grab bag' of inherited features from generations of family members and provides a blueprint for health throughout life. Genetic make-up determines eye colour, and affects personality, temperament and intellectual potential; it also makes us susceptible to developing hereditary conditions. Cystic fibrosis (CF), haemophilia, type 2 diabetes, hypercholesterolaemia (elevated levels of cholesterol in the blood) and some types of cancer are examples of genetic diseases and disorders. Research into genetic make-up is providing insights into ways to diagnose, treat and even prevent these conditions. Research has also shown how quickly genetic structure changes and how our health choices can influence the genetic health of our children.

Cognitive abilities and educational level

Cognitive abilities are developed in childhood, which is why school education is considered an important determinant of health. Our level of cognitive development affects our perceptions of health and illness and may also affect health practices. Educational levels affect our ability to understand and follow guidelines for health—this is called health literacy. If a person cannot read well, written materials such as brochures or handouts on health behaviours and resources are of little value. In Australia educational levels are lower in rural and remote areas compared with major cities, with very remote areas having the lowest levels of school completion.

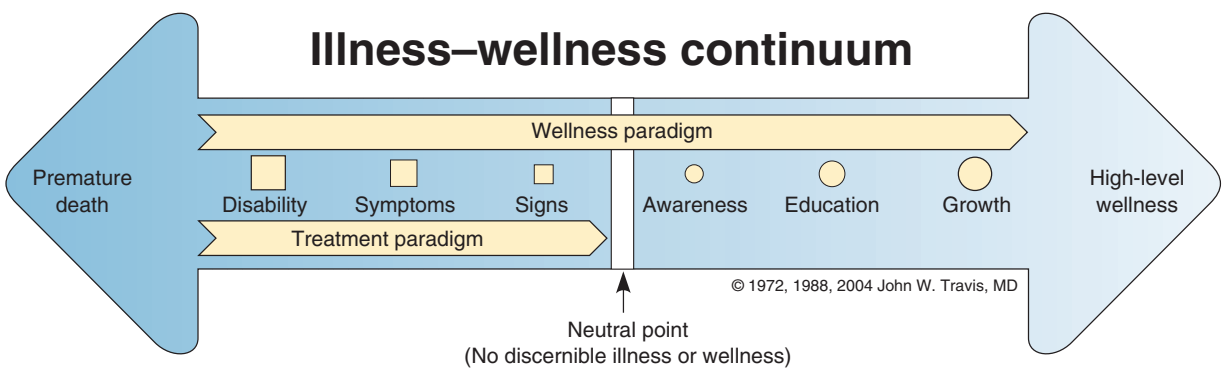

FIGURE 2.1 ■ The illness–wellness continuum

Source: Reprinted with permission from the *Wellness workbook* (3rd ed.) by John W. Travis MD & Regina Sara Ryan (2004). Berkeley, CA: Celestial Arts. © 1981, 1988, 2004 by John W. Travis. www.wellnessworkbook.com.

Race, ethnicity and cultural background

Ethnicity and cultural background influence health values and behaviours, lifestyle and illness behaviours. Every culture defines health and illness uniquely and every culture has different health beliefs and practices. Certain diseases occur at different rates in some races and ethnic groups. For example, the blood disease beta thalassaemia is a genetic disease that occurs more often in people of Italian or Greek descent. For this reason it is also known as Mediterranean anaemia.

Indigenous health

Nursing care of Aboriginal and Torres Strait Islander people is covered in Chapter 52. Many Aboriginal and Torres Strait Islander people experience disability and reduced quality of life due to poor health and many die earlier than the general Australian population. They also experience high rates of chronic disease, such as diabetes and heart disease, at much younger ages. Indigenous Australians are hospitalised for renal failure at 14 times the rate of other Australians and the rate of hospitalisation for endocrine, nutritional and metabolic diseases, including diabetes, is three times that of the rest of Australia (AIHW, 2015b). Aboriginal and Torres Strait Islander people suffer a higher burden of emotional distress and mental illness than that experienced by the wider community. The major contributing factors to these problems include persistent social and economic disadvantages, limited access to healthcare and low levels of nutrition.

A major national initiative called *Close the Gap* was developed at the 2008 Indigenous Health Summit. This initiative included a Statement of Intent between the Australian Government and Indigenous peoples of Australia 'to work together to achieve equality in health status and life expectancy between Aboriginal and Torres Strait Islander peoples and non-Indigenous Australians by the year 2030' (Australian Human Rights Commission, 2008). *Close the Gap* aims to reduce Indigenous disadvantage in terms of life expectancy, child mortality, access to early childhood education, educational achievement and employment outcomes.

Age, gender and developmental level

Age, gender and developmental level are factors in health and illness. Chronic conditions vary with age. Asthma and hay fever are common in the younger age groups, whereas arthritis and hypertensive diseases are more prevalent conditions for those aged 55 years and over. Some diseases occur only in one gender; for example, prostate cancer in men and cervical cancer in women. The older adult is more likely to experience chronic illnesses and is more at risk of serious illness or death from infectious diseases such as influenza and pneumonia.

Lifestyle and environment

The way we live directly impacts on our health. What we eat and how much, alcohol and smoking rates, use of illegal drugs, exercise patterns, stress, grief and happiness all contribute to our health and wellbeing. Too much food can result in obesity, diabetes and hypertension. Smoking causes cancers and respiratory diseases as well as contributing to heart disease and stroke. Lack of exercise is linked to multiple health problems including cardiovascular problems and mental health issues.

The environment you live and work in also impacts on your health. Occupational exposure to toxic substances, such as asbestos or coal dust, increases the risk of pulmonary disorders. Air, water and food pollution increase the risk of respiratory disorders, infectious diseases and cancers. Environmental temperature variations can result in hypothermia or hyperthermia, especially in the older adult. The World Health Organization specifies that everyone should have access to clean air and water and good-quality food.

Socioeconomic background

Socioeconomic status is mainly linked to income or wealth and has a major impact on health status. Socially and economically disadvantaged Australians are more likely to have shorter lives, higher levels of disease and lower use of preventive healthcare than those who are better off. This population tends to smoke more, exercise less, weigh more and eat less fresh food and more processed food high in fat and sugar. These are all risk factors for chronic health conditions including respiratory diseases, cancers and endocrine and cardiovascular diseases. The socioeconomic disadvantage experienced by Aboriginal and Torres Strait Islander people makes them more vulnerable to health risk factors such as smoking, alcohol misuse and domestic violence (AIHW, 2015b).

Geographical area

The geographical area in which a person lives influences their health status as access to healthcare services varies greatly between regional, rural and remote areas of Australia. For example, healthcare services in rural and remote areas can be affected by factors such as larger catchment areas, smaller populations, and fewer general and specialist medical professionals. Rural and remote area residents also have different patterns of service use. For these reasons, they generally have poorer health than people living in major cities, with higher levels of mortality, disease and health risk factors. For example, the prevalence of chronic conditions such as asthma, lung disease, cancer and arthritis in people living in rural and remote areas is more common than in people living in metropolitan areas (AIHW, 2008). Residents of the more inaccessible regions of Australia are generally disadvantaged in their educational and employment opportunities, income and access to goods and services. In some areas they also have less access to basic necessities such as fresh fruit and vegetables.

Compared with those in major urban centres, Australians living in rural and remote areas generally have less access to primary healthcare services and staff, more driving risks (such as poorer road conditions and longer travelling times), longer patient transport times and jobs with higher risks, such as primary production and mining. Preventable cancers—for example, those associated with sun exposure (melanoma) or smoking (lung, head and neck, and lip) and those detectable through screening (cervix)—have significantly higher incidence rates in rural and remote areas. Higher rates of morbidity and mortality in rural and remote areas are also partly influenced by the larger proportion of Indigenous Australians living in these areas (AIHW, 2015b).

PROMOTION OF HEALTH AND PREVENTION OF ILLNESS AND INJURY

For many years, the emphasis in nursing was on the care of acutely ill people in the hospital setting. Changes in society and healthcare have seen this emphasis shift towards preventive and community-based care. Although the primary focus of this chapter is not community health nursing, the importance of the nurse's role in health protection and promotion, as well as teaching illness prevention behaviours, is an essential component of medical–surgical nursing. Community nursing is discussed more fully in Chapter 51. Nurses promote health by teaching activities that maintain wellness, providing information about the characteristics and consequences of diseases when risk factors have been identified, and by supplying specific information about decreasing risk factors (Pender, Parsons & Murdaugh, 2006).

Programs such as the 'Immunise Australia Program' provide widespread immunisation against a large number of communicable diseases (see Table 2.1). This initiative aims to increase national immunisation rates by funding free vaccination programs, administering the Australian Childhood Immunisation register and providing information about immunisation to the general public and health professionals (Department of Health, 2015a). Nurses play an integral role in educating the public about the effectiveness of immunisation to save lives and prevent serious illness (Department of Health, 2015b). The occupational nature of healthcare workers, including nursing students, places them at risk of acquiring vaccine-preventable diseases, and health department policies generally require students and non-immune workers to be immunised.

Nurses are well placed to provide 'education and management to truncate an anticipated rising tide of CVD (cardiovascular disease) in Australia' (Carrington et al., 2009, p. 684). One of the aims of the National Health and Medical Research Council's Australian Dietary Guidelines (2013) is to reduce the risk of chronic diseases, and nurses are an integral part of the interprofessional team responsible for protecting and promoting wellness through healthy eating.

Healthy living

Practices that are known to promote health and wellness include the following:

- exercising moderately and regularly
- getting enough sleep
- limiting alcohol consumption to a moderate amount
- stopping smoking.

ACUTE AND CHRONIC ILLNESS

The National Health Priority Areas initiative is an Australian program in which all the state and territory governments work together with health experts, including clinicians, researchers, organisations and consumers, to reduce the national burden of illness (AIHW, 2015c). This initiative uses evidence-based strategies from prevention through to treatment, management and maintenance to target health areas which impose high social and financial costs on Australian society. There are nine National Health Priority Areas:

1. arthritis
2. asthma
3. cancer
4. cardiovascular disease
5. dementia
6. diabetes
7. injury
8. mental health
9. obesity.

TABLE 2.1 National Immunisation Program Schedule (NIPS)—Special Groups

AGE	VACCINE
6 months and over	• Influenza (people with medical conditions placing them at risk of serious complications of influenza)
12–18 months	• Pneumococcal conjugate (13vPCV) (Aboriginal and Torres Strait Islander people living in high-risk areas—Queensland, Northern Territory, Western Australia and South Australia)
12–24 months	• Hepatitis A (Aboriginal and Torres Strait Islander people in high-risk areas—Queensland, Northern Territory, Western Australia and South Australia)
12 months	• Pneumococcal conjugate (13vPCV) (Individuals who are medically at risk)
6 months to less than 5 years	• Influenza (Aboriginal and Torres Strait Islander people)
4 years	• Pneumococcal polysaccharide (23vPPV) (Individuals who are medically at risk)
15 years and over	• Influenza (Aboriginal and Torres Strait Islander people) • Pneumococcal polysaccharide (23vPPV) (Aboriginal and Torres Strait Islander people who are medically at risk)
50 years and over	• Pneumococcal polysaccharide (23vPPV) (Aboriginal and Torres Strait Islander people)
Pregnant women (at any stage of pregnancy)	• Influenza
65 years and over	• Influenza • Pneumococcal polysaccharide (23vPPV)

Source: Adapted from Department of Health (2015b), www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/national-immunisation-program-schedule. Reprinted with permission of the Australian Government.

BOX 2.1 The most common diseases and injuries of the Australian population

Cancer is the leading cause of disease burden, followed by cardiovascular disease, then mental disorders. There has been a decline in death rates from heart attacks since 2008.

Myocardial infarction (heart attack) rates are falling and survival rates are improving. Survival rates are improving for cancer overall. However, asthma has become more common among children and young adults and the rate of type 2 diabetes has doubled in the past two decades. Diabetes is the main cause of end-stage kidney disease and the number of people treated for this disease has tripled in the past 25 years (AIHW 2014a).

BOX 2.2 Incidence and prevalence

- *Incidence* refers to the number of new cases of an illness, disease or event occurring during a given period. Example: The number of new cases of diabetes diagnosed in 2016.
- *Prevalence* is the number of cases of a disease that exist at any given time. This includes new cases, ongoing cases and people who died during this period. Example: The total number of people who had diabetes in 2016.

See Box 2.1 for an overview of the most common diseases and injuries of the Australian population. Box 2.2 discusses incidence and prevalence.

Disease and illness

Disease and illness are terms that are often used interchangeably, but they do have different meanings. In general, nursing is concerned with illness, whereas medicine is concerned with disease.

Disease

Disease (literally meaning 'without ease') is a medical term describing alterations in the structure and function of the body or mind. Diseases usually have mechanical, biological or normative causes. Mechanical causes of disease result in damage to the structure of the body and are the result of trauma or extremes of temperature. Biological causes of disease impact on body function and can result from a variety of reasons including the normal ageing process. Normative causes are psychological but involve a mind–body interaction, so physical manifestations occur in response to the psychological disturbance.

The causes of many diseases are still unknown but common causes include:

- genetic defects
- foetal exposure to viruses, chemicals or drugs
- biological agents or toxins (including viruses, bacteria, fungi, protozoa and helminths)
- physical agents such as temperature extremes, radiation and electricity
- chemical agents such as alcohol, drugs, strong acids or bases, and heavy metals
- generalised response of tissues to injury or irritation
- changes in antibody productions, resulting in allergies or hypersensitivities
- faulty metabolic processes where production of hormones or enzymes is above or below normal.

Diseases may be classified as acute or chronic, infectious (communicable), congenital, degenerative, functional, malignant, psychosomatic, idiopathic or iatrogenic. These classifications are defined in Table 2.2. In all diseases, alterations in structure or function cause signs and symptoms (manifestations). These warning signs often prompt a person to seek health advice.

Symptoms are subjective in that the person perceives them: 'I feel nauseous and have a headache'. Signs are objective in that they can be seen and measured; for example, bleeding, vomiting, diarrhoea, limitation of movement, swelling. Pain (a subjective symptom) is the most common reason people seek healthcare.

Chronic diseases cause the greatest health problems in the world today, and the number of people with chronic diseases is rising steadily as people live longer and more populations are impacted by lifestyle, behaviour and environmental factors.

- Chronic diseases are a major health problem in all developed countries, accounting for a high proportion of deaths, disability and illness. Yet many of these diseases are preventable, or their onset can be delayed, by relatively simple measures. A summary of the characteristics of chronic diseases according to AIHW (2015d) is:
  - complex causality
  - multiple risk factors

TABLE 2.2 Disease classifications and definitions

CLASSIFICATION	DEFINITION
Acute	Rapid onset, lasts a relatively short time and is self-limiting.
Chronic	Has one or more of the following: (1) is ongoing or permanent, (2) leaves permanent disability, (3) causes irreversible pathophysiology, (4) requires rehabilitation, (5) requires a long period of care.
Communicable	Can spread from one person to another.
Congenital	Existing at or before birth.
Degenerative	Resulting from deterioration or impairment of organs or tissues.
Functional	Affects function or performance but does not have manifestations of organic illness.
Malignant	Tends to become worse and cause death.
Psychosomatic	A psychological disease causing physiological symptoms.
Idiopathic	Unknown cause.
Iatrogenic	Unintended or unnecessary harm or suffering arising from healthcare management.

- long latency periods
- a prolonged course of illness
- functional impairment or disability.

Over 75% of Australians have at least one chronic condition and as people age they are more likely to have one or more chronic conditions (ABS, 2009).

Chronic diseases range from mild to severe, and are usually characterised by periods of remission and exacerbation. During periods of remission the person may be symptom free; however, in periods of exacerbation, the symptoms reappear. Each person with a chronic disease has a unique set of responses and needs. The response of the person to the disease is influenced by the following factors:

- where in their life cycle the disease occurs
- the type and degree of limitations imposed by the disease
- the visibility of impairment or disfigurement
- the pathophysiology causing the disease
- the relationship between the impairment and functioning in social roles
- pain and fear.

These factors are highly complex. They are interrelated within each person, resulting in individualised behaviours and needs. Because there are so many different chronic diseases, and because every person will have a unique response, it is difficult to generalise about needs. However, most people with a chronic disease will need to:

- live as normally as possible—symptoms and treatment can make the person feel alienated, lonely and different
- adapt activities of daily living and self-care
- grieve the loss of physical function and structure, income, status, roles and dignity
- comply with a treatment plan
- maintain a positive self-concept and a sense of hope
- maintain a feeling of being in control
- confront the inevitability of death (Miller, 2000).

Some people with chronic disease successfully meet health-related needs whereas others do not. Nursing interventions for the person with a chronic disease focus on education and support to promote independent functioning, reduce healthcare costs and improve quality of life.

Illness

Illness is the response a person has to a disease. This response is highly individualised, because the person responds with their own perceptions of the disease as well as those of others. The concept of illness combines pathophysiological changes and psychological effects of these and the impact on the individual's roles, relationships and values, and cultural and spiritual beliefs. A person may have a disease but not see themselves as ill or they may validate feelings of illness through the comments of others: 'You look pale, do you feel OK?'.

An **acute illness** occurs rapidly, lasts for a relatively short time and is usually self-limiting (it will resolve itself). The condition may respond to self-treatment or to medical–surgical intervention. People with uncomplicated acute illnesses usually have full recovery and return to normal pre-illness functioning.

Illness behaviours are the way people cope with the changes caused by a disease. Illness behaviours are highly individualised and are influenced by age, gender, family values, economic status, culture, educational level and mental status. The concept of a sequence of illness behaviour was first defined in 1972 (Suchman, 1972):

1. *Experiencing symptoms.* The first stage of an acute illness is when someone experiences signs or symptoms that alert them to a change in their health. The most significant example is pain but other signals of illness include bleeding (haemorrhage), swelling (oedema), fever (pyrexia) or difficulty with breathing (dyspnoea). If the signs and symptoms are mild or are familiar (coughing and nasal congestion), the person will often self-medicate. It is only if the symptoms are prolonged or become more severe that the person moves to the second-stage illness behaviour. Most people would not define this stage to be an illness as they consider it a normal part of the fluctuating status of health: 'It's nothing, just a sniffle'.
2. *Assuming the sick role.* At this stage the individual accepts that their symptoms are proof of illness. They may seek external health advice and take steps to rest by not attending work or school. They may cautiously admit to illness: 'I am feeling a bit unwell'. Self-preoccupation is characteristic of this stage, so the person focuses on changes in function and ability caused by the illness. As the illness resolves, the individual resumes normal activities and may retrospectively admit to illness: 'I was feeling sick but I'm much better now'. However, if the signs and symptoms persist or become worse, the person moves to the next stage by seeking medical care.
3. *Seeking medical care.* In our society, a general practitioner or other healthcare provider usually provides the initial validation of illness. People who believe they are ill, or are encouraged to contact a healthcare provider, seek help in finding out the cause of the illness (diagnosis), how long it will last (prognosis) and the best way of treating it. If the diagnosis requires interventions, the person moves to the next stage. If the diagnosis does not require support, the person may return to normal functioning or, if dissatisfied with this answer, may seek validation from a different healthcare provider.
4. *Assuming a dependent role.* At this stage the person accepts the diagnosis, trajectory and planned treatment of the illness. As the severity of the illness increases, so does their dependent role. During this stage the person may enter a hospital for treatment and care. How an individual responds to care depends on many factors: severity of the illness, anxiety or fear about the outcome, loss of roles, their support systems available, their reaction to stress and their previous experiences with illness care.
5. *Recovery and rehabilitation.* The final stage of acute illness is recovery and rehabilitation. Institutional healthcare sees recovery begin in the hospital but completed at home. This process means education for the person and continuity of care as a major goal for nursing. The shift in settings means nursing care continues outside the hospital in community

settings and the home. The person now relinquishes their dependent role and resumes normal roles and responsibilities. The experience and knowledge gained during treatment and care may place the person on a higher level of wellness after recovery is complete. Recovery time is flexible as the severity of the illness and the method of treatment will affect this, as will the person's compliance and motivation.

ILLNESS PREVENTION

Prevention includes any measures that limit the progression of an illness at any point of its course. Three levels of illness prevention were first defined in 1965. Each level of prevention occurs at a distinct point in the development of a disease process and requires specific nursing interventions (Edelman & Mandle, 2006):

1. *Primary level of prevention.* Generalised health promotion activities as well as specific actions that prevent or delay a disease occurring, including:
   - protecting against environmental risks, such as air and water pollution
   - eating nutritious foods
   - protecting against industrial hazards
   - wearing seat belts and helmets
   - sex counselling and practising safe sex
   - being immunised
   - genetic screenings
   - not smoking and reducing alcohol intake.
2. *Secondary level of prevention.* Interventions around early diagnosis and treatment if an illness is already present. Stopping or slowing the pathological process and helping the person to return to health as soon as possible. These include:
   - screenings for diseases such as hypertension, diabetes and glaucoma
   - physical examinations and diagnostic tests for cancer
   - specific treatments; for example, the treatment of streptococcal infections of the throat will prevent secondary infections of the heart and/or kidneys.
3. *Tertiary level of prevention.* Stopping the disease process and rehabilitating the person back to their normal place in society. Tertiary prevention measures include:
   - medical or surgical treatment for an illness
   - specific rehabilitation programs for cardiovascular problems, head injuries and strokes
   - work training programs following illness or injury
   - educating the public to employ rehabilitated people to the fullest possible extent.

STAGES OF ADULTHOOD

The adult years of life can be divided into three major stages: the young adult (18 to 40 years), the middle adult (40 to 65 years) and the older adult (over 65 years). Adult developmental markers are not as clearly delineated as in the infant or child; however, specific changes do occur with ageing in intellectual, psychosocial and spiritual development, along with physical structures and functions.

The developmental theories specific to the adult, with related stages and tasks, are listed in Table 2.3. Applying a

TABLE 2.3 Theories of adult development

	THEORIST	AGE	TASK
Psychosocial development	Erikson	18–25	Identity versus role confusion • Establishing an intimate relationship with another person • Committing to work and relationships
		25–65	Generativity versus stagnation • Accepting one's own life as creative and productive • Having concern for others
		65–death	Integrity versus despair • Accepting worth of one's own life • Accepting inevitability of death
Spiritual development	Fowler	After 18	• Having a high degree of self-consciousness • Constructing one's own spiritual system
		After 30	• Being aware of truth from a variety of viewpoints
	Westerhoff	Young adult	Searching faith • Acquiring a cognitive and an affective faith through questioning one's own faith
		Middle–older adult	Owned faith • Putting faith into action and standing up for beliefs
Moral development	Kohlberg	Adult	Postconventional level Social contract/legalistic orientation • Defining morality in terms of personal principles • Adhering to laws that protect the welfare and rights of others Universal–ethical principles • Internalising universal moral principles • Respecting others; believing that relationships are based on mutual trust

TABLE 2.3 Theories of adult development (continued)

	THEORIST	AGE	TASK
Developmental tasks	Havighurst	18–35	• Selecting and learning to live with a mate • Starting a family and rearing children • Managing a home • Starting an occupation • Taking on civic responsibility • Finding a congenial social group
		35–60	• Achieving community and social responsibility • Establishing and maintaining an economic standard of living • Assisting teenage children in becoming responsible and happy adults • Developing relaxation-time activities • Relating to one's spouse as a person • Accepting and adjusting to the physiological changes of middle age • Adjusting to ageing parents
		60 and over	• Meeting community and social obligations • Establishing an affiliation with one's own age group • Establishing satisfactory physical living arrangements • Adjusting to decreasing physical strength, health, retirement, reduced income, death of spouse

Sources: Data from *Childhood and society* (2nd ed.) by E. Erikson (1963). New York: Norton; *Stages of faith: The psychology of human development and the quest for meaning* by J. W. Fowler (1981). New York: Harper & Row; *Human development and education* (3rd ed.) by R. J. Havighurst (1972). New York: Longman; *The meaning and measurement of moral development* by L. Kohlberg (1979). New York: Clark University; and *Will our children have faith?* by J. Westerhoff (1976). New York: Seabury Press.

variety of developmental theories is important to the holistic care of the adult as nurses perform assessments, implement care and provide teaching.

The young adult

In Australia, an adult is considered to be aged 18 years or over. A 'young adult' is someone aged between 18 and 25 years. A young adult is typically at the peak of physical development with all body systems functioning at maximum efficiency. During the thirties, normal physiological changes begin to occur. Table 2.4 gives a comparison of physical status for young adults during their twenties and thirties.

Risks for alterations in health

The young adult is at risk of accidents, sexually transmitted infections, substance abuse, and physical or psychosocial stressors. These risk factors may be interrelated.

INJURIES Injury is the leading cause of death, morbidity and permanent disability for Australians aged under 45 and a major source of healthcare costs. Injury causes a range of physical, cognitive and psychological disabilities that seriously affect the quality of life of individuals and their families. Factors that increase the risk of injury are age, gender, alcohol use, place of residence, ethnicity, socioeconomic status and occupation. Suicide is the leading cause of death in Australian young adults (24–44 years) followed by accidental poisoning, motor vehicle accidents, coronary heart disease and breast cancer (AIHW, 2012).

SEXUALLY TRANSMITTED INFECTIONS Sexually transmitted infections include chlamydia, genital herpes, human papillomavirus (HPV), gonorrhoea, syphilis and HIV/AIDS. Young adults who are sexually active with a variety of partners and who do not use protection such as condoms are at

TABLE 2.4 Physical status and changes in the young adult years

ASSESSMENT	20s	30s
Skin	Smooth and even	Wrinkles begin to appear
Hair	Slightly oily, shiny Balding may begin	Greying may begin Balding may begin
Vision	Snellen 20/20	Some loss of visual acuity and accommodation
Musculoskeletal	Strong, coordinated	Some loss of strength and muscle mass
Cardiovascular	Maximum cardiac output	Slight decline in cardiac output
Functional	60–90 beats/min Mean BP: 120/80	60–90 beats/min Mean BP: 120/80
Respiratory	Rate: 12–20 Full vital capacity	Rate: 12–20 Decline in vital capacity

greatest risk of these diseases. Nursing care of people with sexually transmitted infections is discussed in Chapter 49.

SUBSTANCE ABUSE The 2013 *National Drug Strategy Household Survey* found that two in five Australians smoked tobacco, drank alcohol at risky levels or used an illicit drug in 2013. However, rates of alcohol and smoking use had declined since 2010 and illicit drug use had remained stable. Misuse of prescribed medication had increased (AIHW, 2014b).

Alcohol and tobacco are the drugs most commonly used by Australians, with people starting to smoke tobacco at around 16 years. With the exception of marijuana/cannabis, the proportion of the population who have used illicit drugs at some time in their life is relatively low. However, tobacco, alcohol and illicit drug use contributes to significant illness and disease, injury, workplace concerns, violence, crime and breakdowns in families and relationships in Australia.

The most commonly reported illicit drugs used are marijuana/cannabis, followed by ecstasy, painkillers/analgesics used for non-medical purposes, and meth/amphetamine (AIHW, 2014b). Although alcohol abuse occurs at all ages, it is greater in the twenties than any other decade of life. Alcohol contributes to motor vehicle accidents and physical violence, and damages the developing foetus in pregnant women. It can cause liver disease and nutritional deficits. Nursing care of people with substance abuse issues is discussed in Chapter 5.

PHYSICAL AND PSYCHOSOCIAL STRESSORS

Physical stressors that increase the risk of illness in young adults include environmental pollutants and work-related risks such as falls, electrical hazards, mechanical injuries or exposure to toxins or infectious agents. Other physical stressors include exposure to the sun and ingestion of chemical substances such as caffeine, alcohol and nicotine.

Many different and individualised psychosocial stressors may affect young adults, including the pressure to make choices regarding education, occupation, relationships, independence and lifestyle. The young adult without adequate education or job skills may face unemployment, poverty and homelessness. Divorce is another psychological stressor that often results in loneliness, feelings of failure, financial difficulties, domestic violence and child abuse. In 2013, the divorce rate in Australia was two per 1000 people. The average time from marriage to divorce was 12 years and the median age at divorce was 45 years for men and 42 years for women (ABS, 2014).

Assessment guidelines

The following guidelines are useful in assessing the achievement of significant developmental tasks in the young adult. Does the young adult:

- feel independent from parents?
- have a realistic self-concept?
- like themselves and the direction in which their life is going?
- interact well with family?
- cope with the stresses of constant change and growth?
- have well-established bonds with significant others, such as marriage partners or close friends?
- have a meaningful social life?
- have a career or occupation?
- demonstrate emotional, social and economic responsibility for their own life?
- have a set of values that guide behaviour?
- have a healthy lifestyle?

Physical assessment of the young adult includes height and weight, blood pressure and vision. During the health history, the nurse should ask specific questions about substance use, sexual activity and concerns, exercise, eating habits, menstrual history and patterns, coping mechanisms, any familial chronic illnesses and family changes.

See Figure 2.2 and Table 2.5 for the most recent available overview of the burden of disease and Disability Adjusted Life Year (DALYs) in 15–44 year olds in Australia.

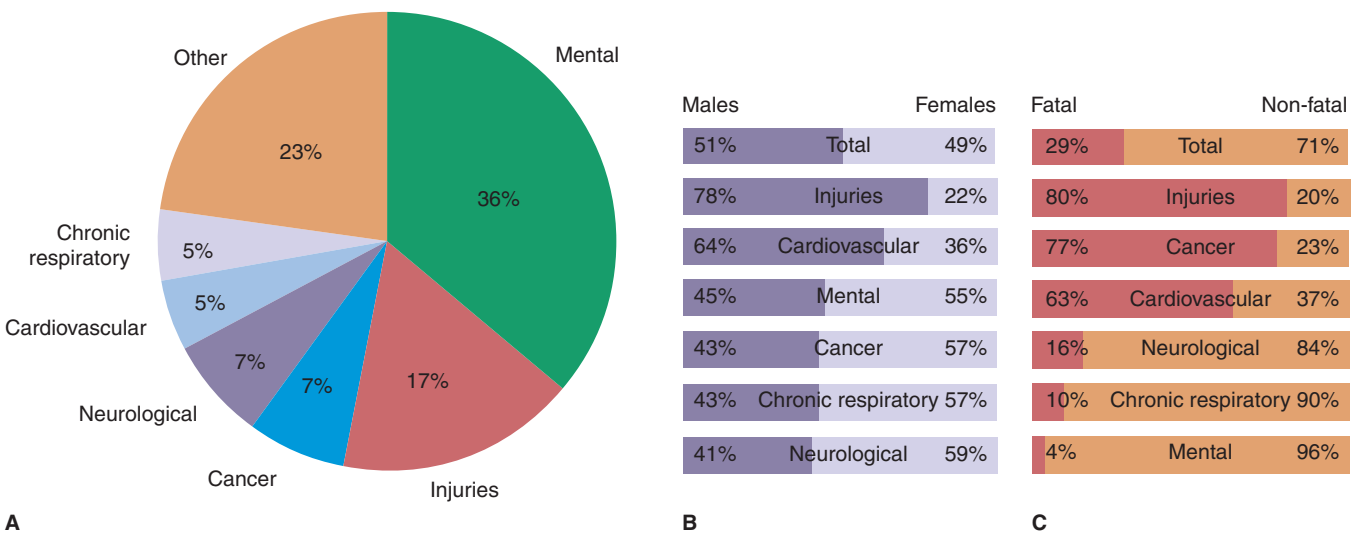

FIGURE 2.2 ■ Burden of disease disability adjusted life years (DALYs) in 15–44 year olds by broad cause group expressed as: *A,* proportions (rounded) of total, *B,* proportions by sex, and *C,* proportions due to fatal and non-fatal outcomes, Australia, 2003

Source: The burden of disease and injury in Australia 2003 by Begg et al. (2007). Canberra: AIHW. Reproduced with permission.

TABLE 2.5 Leading causes of DALYs in 15–44 year olds by sex in Australia, 2003

RANK	MALES	DALYs	% OF TOTAL	FEMALES	DALYs	% OF TOTAL
1	Anxiety and depression	42 237	13.0	Anxiety and depression	84 717	27.4
2	Suicide and self-inflicted injuries	27 592	8.5	Migraine	14 105	4.6
3	Road traffic accidents	22 845	7.1	Type 2 diabetes	12 487	4.0
4	Schizophrenia	14 376	4.4	Asthma	11 311	3.7
5	Alcohol abuse	13 953	4.3	Schizophrenia	11 064	3.6
6	Type 2 diabetes	12 868	4.0	Personality disorders	9 389	3.0
7	Heroin abuse	11 882	3.7	Breast cancer	9 068	2.9
8	Personality disorders	10 526	3.2	Infertility	8 057	2.6
9	Ischaemic heart disease	9 750	3.0	Suicide and self-inflicted injuries	7 174	2.3
10	Chronic obstructive pulmonary disease (COPD)	6 840	2.1	Road traffic accidents	6 751	2.2

Source: The burden of disease and injury in Australia 2003 by Begg et al. (2007). Canberra: AIHW. Reproduced with permission.

Promoting healthy behaviours in the young adult

Health information for the young adult is primarily provided in community settings including:

- Health-related courses and seminars at community colleges and universities provide information on exercise, alcohol and drug abuse, smoking cessation, mental health and sexual health.
- Workplace programs emphasise blood pressure monitoring, exercise, smoking cessation, nutrition guidelines and stress reduction activities.
- Community programs provide information on media, support groups, and risk factors for disease and injury.

The middle adult

Many physical status and function changes take place between ages 40 and 65. Table 2.6 lists the physical changes that normally occur in the middle years.

Risks for alterations in health

The middle adult is at risk of obesity, cardiovascular disease, cancer, substance abuse, and physical and psychosocial stressors. These factors may be interrelated.

OBESITY The middle adult often has a problem maintaining a healthy weight. Weight gain at this age is the result of consuming the same number of kilojoules/calories while physical activity decreases and basal metabolic rate falls. Obesity affects all major organ systems, increasing the risk of atherosclerosis, hypertension, elevated cholesterol and triglyceride levels, and diabetes. Obesity is also associated with heart disease, osteoarthritis and gallbladder disease.

CARDIOVASCULAR DISEASE The major risk factors, especially for coronary artery disease, include age, male gender, physical inactivity, cigarette smoking, hypertension,

TABLE 2.6 Physical changes in the middle adult years

ASSESSMENT	CHANGES
Skin	• Decreased turgor, moisture and subcutaneous fat result in wrinkles. • Fat is deposited in the abdominal and hip areas.
Hair	• Loss of melanin in hair shaft causes greying. • Hairline recedes in males and sometimes females.
Sensory	• Visual acuity for near vision decreases (presbyopia) during the 40s. • Auditory acuity for high-frequency sounds decreases (presbycusis), more commonly in men. • Sense of taste diminishes.
Musculoskeletal	• Skeletal muscle mass decreases by about age 60. • Thinning of intervertebral discs results in loss of height (about 2.5 cm). • Postmenopausal women may have loss of calcium and develop osteoporosis.
Cardiovascular	• Blood vessels lose elasticity. • Systolic blood pressure may increase.
Respiratory	• Loss of vital capacity (about 1L from age 20 to 60) occurs.
Gastrointestinal	• Large intestine gradually loses muscle tone; constipation may result. • Gastric secretions are decreased.
Genitourinary	• Hormonal changes: menopause, women (↓ oestrogen); andropause, men (↓ testosterone).
Endocrine	• Gradual decrease in glucose tolerance.

elevated blood cholesterol levels and diabetes. Other contributing factors include obesity, stress and lack of exercise. The middle adult is also at risk of peripheral vascular, cerebrovascular and cardiovascular disease.

CANCER Cancer is the leading cause of death and illness in Australia, closely followed by cardiovascular disease (Begg et al., 2007). The five most common cancers in Australia are prostate, colorectal, breast, melanoma and lung cancer. One in every two Australian males and one in every three Australian females will be diagnosed with cancer before age 85 (AIHW, 2014a). The AIHW *Australia's Health 2014* report shows that:

- 4 million women participated in Breast Screen Australia between 2011 and 2012.
- 3.7 million women participated in the National Cervical Screening Program between 2011 and 2012.
- 35% of over 2 million people invited to participate in the National Bowel Cancer Screening Program between 2011 and 2012 returned a completed bowel cancer screening kit for analysis.

Nursing care of the person with cancer is discussed in Chapter 13.

SUBSTANCE ABUSE Although middle adults use a variety of substances, the most commonly abused are alcohol, nicotine and prescription drugs. Excess alcohol use in the middle adult contributes to an increased risk of liver cancer, cirrhosis, pancreatitis, hyperlipidaemia and anaemia. Alcoholism also increases the risk of accidental injury or death and disrupts careers and relationships. Tobacco smoking increases the risk of cancers of the larynx, lung, mouth, pharynx, bladder, pancreas, oesophagus and kidney, as well as chronic obstructive pulmonary disease and cardiovascular disorders.

PHYSICAL AND PSYCHOSOCIAL STRESSORS The middle adult years are ones of change and transition, frequently resulting in stress. Both men and women must adapt to changes and declines in physical appearance and function and accept their own mortality. The age for first-time parents has risen substantially in the past few decades, and children are living at home longer, usually due to financial reasons. The middle adult becomes part of what has been called 'the sandwich generation' (Miller, 1981), caring simultaneously for children and ageing parents. Both men and women may make career changes, and approaching retirement becomes a reality. Divorce in the middle years is also a major emotional, social and financial stressor.

Assessment guidelines

The following guidelines are useful in assessing the achievement of significant developmental tasks in the middle adult. Does the middle adult:

- accept the ageing body?
- feel comfortable with and respect themselves?
- enjoy some new freedom to be independent?
- accept changes in family roles?
- enjoy success and satisfaction from work and/or family roles?
- interact well and share companionable activities with a partner?
- expand or renew previous interests?
- pursue charitable and altruistic activities?
- consider plans for retirement?
- have a meaningful philosophy of life?
- follow health promotion practices?

Physical assessment of the middle adult includes all body systems, including blood pressure, vision and hearing. Monitoring for risks and possible cancer symptoms is essential. The nurse should ask specific questions about food intake and exercise habits, substance abuse, sexual concerns, changes in the reproductive system, coping mechanisms and family history of chronic illnesses.

See Figure 2.3 and Table 2.7 for an overview of the burden of disease (DALYs) in 45–64 year olds in Australia.

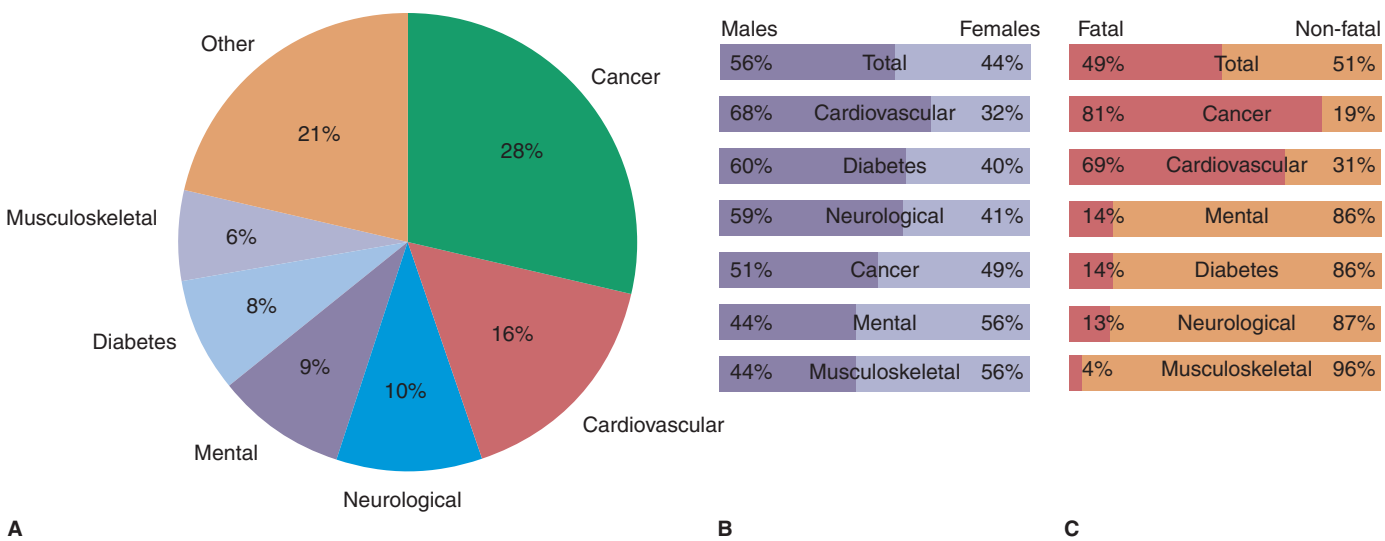

FIGURE 2.3 ■ Burden of disease (DALYs) in 45–64 year olds by broad-cause disease group expressed as: *A,* proportions (rounded) of total, *B,* proportions by sex, and *C,* proportions due to fatal and non-fatal outcomes, Australia, 2003

Source: The burden of disease and injury in Australia 2003 by Begg et al. (2007). Canberra: AIHW. Reproduced with permission.

TABLE 2.7 Leading causes of DALYs in 45–64 year olds by sex in Australia, 2003

RANK	MALES	DALYs	% OF TOTAL	FEMALES	DALYs	% OF TOTAL
1	Ischaemic heart disease	47 782	12.5	Breast cancer	32 012	10.7
2	Type 2 diabetes	32 741	8.6	Anxiety and depression	25 744	8.6
3	Lung cancer	20 861	5.5	Type 2 diabetes	22 299	7.5
4	Adult-onset hearing loss	20 847	5.5	Ischaemic heart disease	17 489	5.8
5	COPD	15 389	4.0	Lung cancer	13 475	4.5
6	Colorectal cancer	14 130	3.7	Adult-onset hearing loss	10 576	3.5
7	Stroke	13 800	3.6	COPD	10 422	3.5
8	Anxiety and depression	11 757	3.1	Colorectal cancer	9 808	3.3
9	Alcohol abuse	10 077	2.6	Stroke	9 693	3.2
10	Prostate cancer	8 953	2.3	Back pain	6 620	2.2

Source: The burden of disease and injury in Australia 2003 by Begg et al. (2007). Canberra: AIHW. Reproduced with permission.

Promoting healthy behaviours in the middle adult

Health information for the middle adult can be provided in a variety of community settings, including outpatient clinics, occupational health clinics and private practice. Examples are as follows:

- Specific programs emphasise accepting responsibility for one's own health. This type of teaching can be through seminars or on a one-to-one basis, and includes information specific to individuals with an identified need, such as smokers, women who have re-entered the workforce or men nearing retirement.
- Community and employment agencies provide information about safety hazards in the home, workplace and community.
- There are many community resources available including programs offered at alcohol/drug abuse treatment centres, clinics and health centres, counselling services, crisis intervention centres, intimate partner violence programs, and health education and promotion agencies (e.g. the Association of Relatives and Friends of the Mentally Ill, Red Cross Australia, the Cancer Council Australia, the Heart Foundation of Australia and Diabetes Australia).

The older adult

The older adult period begins at age 65, but it can be further divided into three periods: the young-old (ages 65 to 74), the middle-old (ages 75 to 84) and the old-old (age 85 and over). With increasing age a number of normal physiological changes occur (see Table 2.8).

Improving older people's health is an Australian national research priority. One area of special interest is adopting a healthy lifestyle at older ages because its benefits include the prevention of disease and functional decline, extended longevity and enhanced quality of life (WHO, 2002).

The increase in numbers of older adults has important implications for nursing. People needing healthcare in all settings will be older, requiring nursing interventions and teaching specifically designed to meet needs that differ from those of young and middle adults. Although older person nursing is a nursing specialty area, it is also an integral component of medical–surgical nursing (see Figure 2.4).

Risks for alterations in health

The older adult is at risk of alterations in health from a variety of causes. Most older adults have one chronic health problem, while many have multiple illnesses (co-morbidities). Ischaemic heart disease (coronary heart disease) and cerebrovascular diseases (notably stroke) are the two leading causes of death, accounting for about 30% of all deaths among older Australian men and women. Like the middle adult, the older adult is at risk of decreased health from obesity and a sedentary lifestyle. Other risk factors specific to this age group include accidental injuries, pharmacological effects, and physical and psychosocial stress.

INJURIES Injuries in the older adult cause many problems: illness, financial burdens, hospitalisation, self-care deficits, loss of independence and even death. The risk of injury is increased by the normal physiological changes that accompany ageing,

FIGURE 2.4 ■ The older adult population is increasing more rapidly than any other age group, making nursing of older adults an integral component of medical–surgical nursing practice

TABLE 2.8 Physical changes in the older adult years

ASSESSMENT	CHANGES
Skin	• Decreased turgor and sebaceous gland activity result in dry, wrinkled skin. Melanocytes cluster, causing 'age spots' or 'liver spots'.
Hair and nails	• Scalp, axillary and pubic hair thins; nose and ear hair thickens. Women may develop facial hair. • Nails grow more slowly; may become thick and brittle.
Sensory	• Visual field narrows, and depth perception is distorted. • Pupils are smaller, reducing night vision. • Lenses yellow and become opaque, resulting in distortion of green, blue and violet tones, and increased sensitivity to glare. • Production of tears decreases. • Sense of smell, taste and thirst decreases. • Age-related hearing loss progresses, involving middle- and low-frequency sounds. • Threshold for pain and touch increases. • Alterations in proprioception (sense of physical position) may occur.
Musculoskeletal	• Loss of overall mass, strength and movement of muscles occurs; tremors may occur. • Loss of bone structure and deterioration of cartilage in joints results in increased risk of fractures and limitation of range of motion.
Cardiovascular	• Systolic blood pressure rises. • Cardiac output decreases. • Peripheral resistance increases, and capillary walls thicken.
Respiratory	• Continued loss of vital capacity occurs as the lungs become less elastic and more rigid. • Anteroposterior chest diameter increases; kyphosis. • Although blood carbon dioxide levels remain relatively constant, blood oxygen levels decrease by 10–15%.
Gastrointestinal	• Production of saliva decreases, and decreased number of taste buds decrease accurate receptors for salt and sweet. • Gag reflex is decreased, and stomach motility and emptying are reduced. • Both large and small intestines have some atrophy, with decreased peristalsis. • The liver decreases in weight and storage capacity; gallstones increase; pancreatic enzymes decrease.
Genitourinary	• Kidneys lose mass and the glomerular filtration rate is reduced (by nearly 50% from young adulthood to old age). • Bladder capacity decreases and the micturition reflex is delayed. Urinary retention is more common. • Women may have stress incontinence; men may have an enlarged prostate gland. • Reproductive changes in men occur: – Testosterone decreases. – Sperm count decreases. – Testes become smaller. – Length of time to achieve an erection increases; erection is less full. • Reproductive changes in women occur: – Oestrogen levels decrease. – Breast tissue decreases. – Vagina, uterus, ovaries and urethra atrophy. – Vaginal lubrication decreases. – Vaginal secretions become alkaline.
Endocrine	• Pituitary gland loses weight and vascularity. • Thyroid gland becomes more fibrous and plasma decreases. • Pancreas releases insulin more slowly; increased blood glucose levels are common. • Adrenal glands produce less cortisol.

pathophysiological alterations in health, environmental hazards and lack of support systems. The two major causes of injury in the older adult are falls and motor vehicle accidents. Of these, falls with resultant hip fractures are the most significant in terms of long-term disability and death.

PHARMACOLOGICAL EFFECTS Medications are commonly used by older Australians to treat and manage illness and health conditions and include prescription pharmaceuticals, over-the-counter medications, vitamins and minerals and, increasingly, illegal drugs. The level of use of pharmaceuticals generally increases with age. The rate of prescribed medication use for Australians aged 65 years and over is about 100 per 100 general practitioner encounters, which means, on average, that every time a person in this age group sees a doctor, they are prescribed medication (AIHW 2007, p. 31).

A number of risk factors predispose the older adult to experiencing drug toxicity. Age-related changes in tissue and organ structure and function alter the absorption of both oral and parenteral medications. Low nutritional levels and decreased liver

FOCUS ON CULTURAL DIVERSITY Diversity in older Australian adults

- Australians aged 65 years and over have one of the highest life expectancies in the world.
- More than 50% of all older people in Australia are aged 65–74 years. About 30% are the middle-old and 12% are the old-old.
- By 2036, the number of Australians aged 65 years and over is expected to more than double, from 2.7 million to 6.3 million, and will represent 24% of the total population.
- Older Australians are active contributors to family and community life.
- Most older people live in their own homes—only 6% live in non-private dwellings, which include aged care homes and hospitals.
- Australian women of all cultural backgrounds tend to live longer than the men.
- The life expectancy gap between Indigenous and non-Indigenous Australians, and the very low proportion of the Indigenous population aged 65 years and over, means the 'older Indigenous' population is considered to include people aged 50 years and over.
- Most older Australians born in non-English countries come from Italy, Greece, Germany, the Netherlands and China.

Source: Data from Australian Institute of Health and Welfare (2007). *Older Australia at a glance.*

function may alter drug metabolism. The ageing kidney may not excrete drugs at the normal clearance rate. Self-administration of both prescribed and non-prescribed medications presents risks for error from confusion, forgetfulness or misreading the directions. The older adult may take several drugs at once, placing them at risk of drug–drug interaction or an adverse drug event. These events are responsible for high rates of hospital admissions and mortality in older Australians (Morgan, 2009).

PHYSICAL AND PSYCHOSOCIAL STRESSORS The older adult is exposed to the same environmental hazards as the young and middle adult, but the accumulation of years of exposure may now appear; for example, skin cancer from many years of sun exposure and hearing loss due to long-term noise pollution. Older adults (especially males) are at increased risk of respiratory disorders as a result of smoking, or from such pollutants as coal or asbestos dust. Living conditions and economic constraints may prevent the older adult from having necessary heating and cooling, contributing to thermal-related illnesses and even death. Mistreatment of older people (elder abuse) and neglect further increases the risk of injury or illness.

Psychosocial stressors for the older adult include the illness or death of a spouse, decreased or limited income, retirement, isolation from friends and family because of lack of transportation or distance, return to the home of a child or relocation to an aged care facility. A further stressor may be role loss or reversal; for example, when the wife becomes the caretaker of her chronically ill husband.

Assessment guidelines

The following guidelines are useful in assessing the achievement of significant developmental tasks in the older adult. Does the older adult:

- adjust to the physiological changes of ageing?
- manage retirement years in a satisfying manner?
- have satisfactory living arrangements and income to meet changing needs?
- participate in social and leisure activities?
- have a social network of friends and support people?
- view life as worthwhile?
- have high self-esteem?
- have the abilities to care for self or to secure appropriate help?
- gain support from a value system or spiritual philosophy?
- adapt lifestyle to their diminishing energy and ability?
- accept and adjust to their own death and that of significant others?

Physical assessment of the older adult includes a careful examination of all body systems. When taking a health history, the nurse should ask specific questions about dietary patterns; elimination; exercise and rest; use of alcohol, nicotine, over-the-counter medications, and prescription and non-prescription drugs; sexual concerns; financial concerns; and support systems.

See Figure 2.5 and Table 2.9 for an overview of the burden of disease (DALYs) in 65–74 year olds in Australia.

Promoting healthy behaviours in the older adult

Older adults derive the same benefits from health teaching as young adults and middle adults and should never be viewed as being 'too old' for healthy living practices. However, nurses need to structure teaching activities to meet age-related physiological changes, such as using large print. Health education for the older adult is provided in hospitals, aged care facilities, retirement villages, outpatient clinics, senior citizen centres and other community settings. Examples are as follows:

- Educational seminars teach about accident prevention in the home, in cars and when using public transport.
- Health screenings that are specifically aimed at the older adult.
- Community programs provide immunisation for influenza and pneumonia.
- Literature gives information about financial assistance for healthcare, crisis hotlines, community services and resources (described earlier for the middle adult), transportation and nutrition.

THE CONCEPT OF FAMILY

Although some people are totally alone in the world, most have one or more other people who are significant in their lives. These significant others may be related or bonded to the person by birth, adoption, marriage or friendship. Although not always meeting traditional definitions, people (or even pets) significant to the person are considered the person's family. The nurse

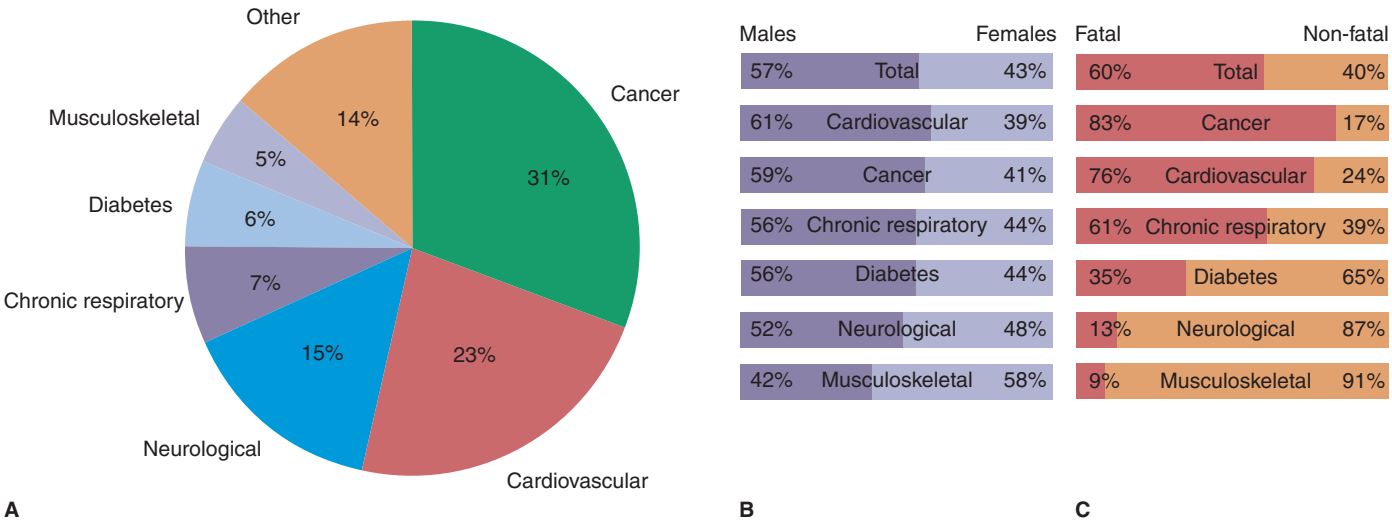

FIGURE 2.5 ■ Burden (DALYs) in 65–74 year olds by broad cause group expressed as: *A*, proportions (rounded) of total, *B*, proportions by sex, and *C*, proportions due to fatal and non-fatal outcomes, Australia, 2003

Source: Australian Institute of Health and Welfare (2007). *Older Australia at a glance.* Reproduced with permission.

TABLE 2.9 Leading causes of DALYs in 65–74 year olds by sex in Australia, 2003

RANK	MALES	DALYs	% OF TOTAL	FEMALES	DALYs	% OF TOTAL
1	Ischaemic heart disease	37 860	15.5	Ischaemic heart disease	21 052	11.4
2	Lung cancer	19 258	7.9	Type 2 diabetes	11 517	6.2
3	Type 2 diabetes	14 203	5.8	Breast cancer	10 445	5.7
4	Prostate cancer	11 950	4.9	Dementia	10 236	5.5
5	Adult-onset hearing loss	11 920	4.9	Lung cancer	9 937	5.4
6	COPD	11 693	4.8	Stroke	9 635	5.2
7	Stroke	10 938	4.5	COPD	8 855	4.8
8	Colorectal cancer	10 531	4.3	Colorectal cancer	7 513	4.1
9	Dementia	7 872	3.2	Osteoarthritis	6 088	3.3
10	Parkinson's disease	3 958	1.6	Adult-onset hearing loss	5 834	3.2

should always include the family as an integral component of care in all healthcare settings.

Definitions and functions of the family

What is a **family**? The definitions of a family are changing as society changes. According to one definition, a family is a unit of people related by marriage, birth or adoption (Duvall, 1977). A more comprehensive definition is that a family is composed of two or more people who are emotionally involved with each other. In a global society, it may not be possible for family members to live in close proximity, but they can remain emotionally involved.

Although every family is unique, all families have certain structural and functional features in common. Family structure (family roles and relationships) and family function (interactions among family members and with the community) provide the following:

- *Interdependence.* The behaviours and level of development of individual family members constantly influence and are influenced by the behaviours and level of development of all other members of the family.
- *Maintaining boundaries.* The family creates boundaries that guide its members, providing a distinct and unique family culture. This culture, in turn, provides values.
- *Adapting to change.* The family changes as new members are added, current members leave and the development of each member progresses.
- *Performing family tasks.* Essential tasks maintain the stability and continuity of the family. These tasks include physical maintenance of the home and the people in the home, the production and socialisation of family members and the maintenance of the psychological wellbeing of members.

Family developmental stages and tasks

The family, like the individual, has developmental stages and tasks. Each stage brings change, requiring adaptation, and each new stage also presents family-related risk factors for

alterations in health. The nurse must consider the person's needs both at a specific developmental stage and within a family with specific developmental tasks. Family developmental stages and developmental tasks are described next. Related risk factors and health problems for each stage are listed in Table 2.10.

The couple

The developmental tasks of the couple include adjusting to living together, establishing a mutually satisfying relationship, relating to relatives and deciding whether to have children (for those of child-bearing age).

Family with infants and preschoolers

The family with infants or preschoolers must adjust to having and supporting the needs of more than two members. Other developmental tasks of the family at this stage include developing an attachment between parents and children, adjusting to the economic costs of having more members, coping with energy depletion and lack of privacy, and

TABLE 2.10 Family-related risk factors for alterations in health

STAGE	RISK FACTORS	HEALTH PROBLEMS
Couple or family with infants and preschoolers	• Lack of knowledge about family planning, contraception, sexual and marital roles • Inadequate antenatal care • Altered nutrition: inadequate nutrition, overweight, underweight • Smoking, alcohol/drug abuse • First pregnancy before age 16 or after age 35 • Low socioeconomic status • Lack of knowledge about child health and safety • Rubella, syphilis, gonorrhoea, HIV/AIDS	• Premature pregnancy • Low-birth-weight infant Birth defects • Injury to infant or child Accidents
Family with school-age children	• Unsafe home environment • Working parents with inappropriate or inadequate resources for child care • Low socioeconomic status • Child abuse or neglect • Multiple, closely spaced children • Repeated infections, accidents and hospitalisations • Unrecognised and unattended health problems • Poor or inappropriate nutrition • Toxic substances in the home	• Behaviour problems • Speech and vision problems • Learning disabilities • Communicable diseases • Physical abuse • Cancer • Developmental delay • Obesity, underweight
Family with adolescents and young adults	• Family values of aggressiveness and competition • Lifestyle and behaviour leading to chronic illness (substance abuse, inadequate diet) • Lack of problem-solving skills • Conflicts between parent and children	• Violent death and injury • Alcohol/drug abuse • Unwanted pregnancy • Suicide • Sexually transmitted infections • Domestic abuse
Family with middle adults	• High-cholesterol diet • Overweight • Hypertension • Smoking, alcohol abuse • Physical inactivity • Personality patterns related to stress • Exposure to environment: sunlight, radiation, asbestos, water or air pollution • Depression	• Cardiovascular disease (coronary artery • disease and cerebral vascular disease) • Cancer • Accidents • Suicide • Mental illness
Family with older adults	• Age • Depression • Drug interactions • Chronic illness • Death of spouse • Reduced income • Poor nutrition • Lack of exercise • Past environment and lifestyle	• Impaired vision and hearing • Hypertension • Acute illness • Chronic illness • Infectious diseases (influenza, pneumonia) • Injuries from burns and falls • Depression • Alcohol abuse

carrying out activities that enhance the growth and development of the children.

Family with school-age children

The family with school-age children has the developmental tasks of adjusting to the expanded world of children in school and encouraging educational achievement. A further task is promoting joint decision making between children and parents.

Family with adolescents and young adults

The developmental tasks of the family with adolescents and young adults focus on transition. While providing a supportive home base and maintaining open communications, parents must balance freedom with responsibility and release adult children as they seek independence.

Family with middle adults

The family with middle adults (in which the parents are middle aged and children are no longer at home) has the developmental tasks of maintaining ties with older and younger generations and planning for retirement. If the family consists of just the middle-aged couple, they have the developmental tasks of re-establishing their relationship as a couple and possibly taking on the role of grandparents.

Family with older adults

The older adult family has the developmental tasks of adjusting to retirement, adjusting to ageing and coping with the loss of a spouse. If a spouse dies, further tasks include adjusting to living alone and perhaps selling the family home.

The family of the person with a chronic illness

The person with a chronic illness may be hospitalised for diagnosis and treatment of acute exacerbations, but their care is primarily provided at home. Chronic illness in a family member is a major stressor that causes changes in family structure and function and performing family developmental tasks.

Many factors affect family responses to chronic illness and these responses in turn affect the person's response to and perception of the illness. Factors influencing response to chronic illness include personal, social and economic resources as well as the nature and course of the disease and the demands of the illness as perceived by family members.

Support for the family is essential. The following information should be considered when performing any family assessment and developing a plan of care:

- cohesiveness and communication patterns within the family
- family interactions that support self-care
- friends and relatives available to help
- family values and beliefs about health and illness
- cultural and spiritual beliefs
- developmental level of the person and family.

It is important to remember that standardised teaching plans may not be effective. People with chronic illnesses and their families should be given the freedom to choose appropriate literature, self-help or support groups, and interactions with others who have the same illness.

CHAPTER HIGHLIGHTS

- Health is an ever-changing state affected by genetic make-up, cognitive abilities, education, ethnicity, cultural background, age, gender, developmental level, lifestyle, environment, socioeconomic background and geographical area.
- The emphasis of nursing has shifted from acute care in the hospital setting to preventive community-based care. An essential component of medical–surgical nursing is teaching health behaviours that promote and maintain functional health status.
- Illnesses may be acute or chronic and behaviours of illness follow a sequence of experiencing symptoms, assuming the sick role, seeking medical help, assuming a dependent role, recovery and rehabilitation.
- Young adults are at risk of alterations in health from injury, sexually transmitted infections, substance abuse, workplace exposure to pollutants, sun exposure and psychological stressors.
- Middle adults are at risk of alterations in health from obesity, cardiovascular disease, cancer, substance abuse, and the stresses of change and transition.
- Older adults are at risk of alterations in health from chronic illnesses, injuries, drug toxicities, and changes in income and marital status.
- The family is an integral component in planning and implementing nursing care for the adult.

CONCEPT CHECK

1 Which definition best describes wellness?
1. a complete absence of disease
2. depends on the number of chronic illnesses
3. never having to take medications
4. gaining the best potential functioning of an individual

2 Many different factors affect the health of an individual. Which of the following are included? (Select all that apply.)
1. genetic make-up
2. cognitive abilities
3. height
4. age
5. ethnicity

3 Which of the following diseases has a genetic basis?
1. tuberculosis
2. cystic fibrosis
3. appendicitis
4. indigestion

4 Primary levels of prevention are general health promotion actions that prevent or delay the occurrence of a disease. Which of the following is a primary preventive activity?
1. practising safer sex
2. screening for hypertension
3. breast self-examination
4. having surgery

5 You call your tutor to say you have the 'flu' and will not be in class. What level of illness behaviour are you demonstrating?
1 experiencing symptoms
2 assuming the sick role
3 seeking medical care
4 assuming a dependent role

6 Your nephew was born with a heart defect. How would this disorder be classified?
1 an acute illness
2 a malignant illness
3 an iatrogenic illness
4 a congenital illness

7 Of the following descriptors, which is specific to a chronic illness?
1 occurs rapidly
2 lasts for a long time
3 is self-limiting
4 lasts for a short time

8 Mr Jones, age 50, is 15 kg overweight, smokes and rarely exercises. As a middle adult these factors increase his risk of disorders of which body system?
1 cardiovascular
2 renal
3 gastrointestinal
4 nervous

9 You are asked to present a health-related program at the local senior citizen centre. What would be an appropriate topic?
1 the hazards of substance abuse
2 accident prevention in the home
3 family roles and tasks
4 treating acute illness

10 Which of the following developmental tasks are part of the life of a family with older adults if a spouse dies? (Select all that apply.)
1 coping with lack of privacy
2 planning for retirement
3 adjusting to ageing
4 coping with loss
5 relating to family

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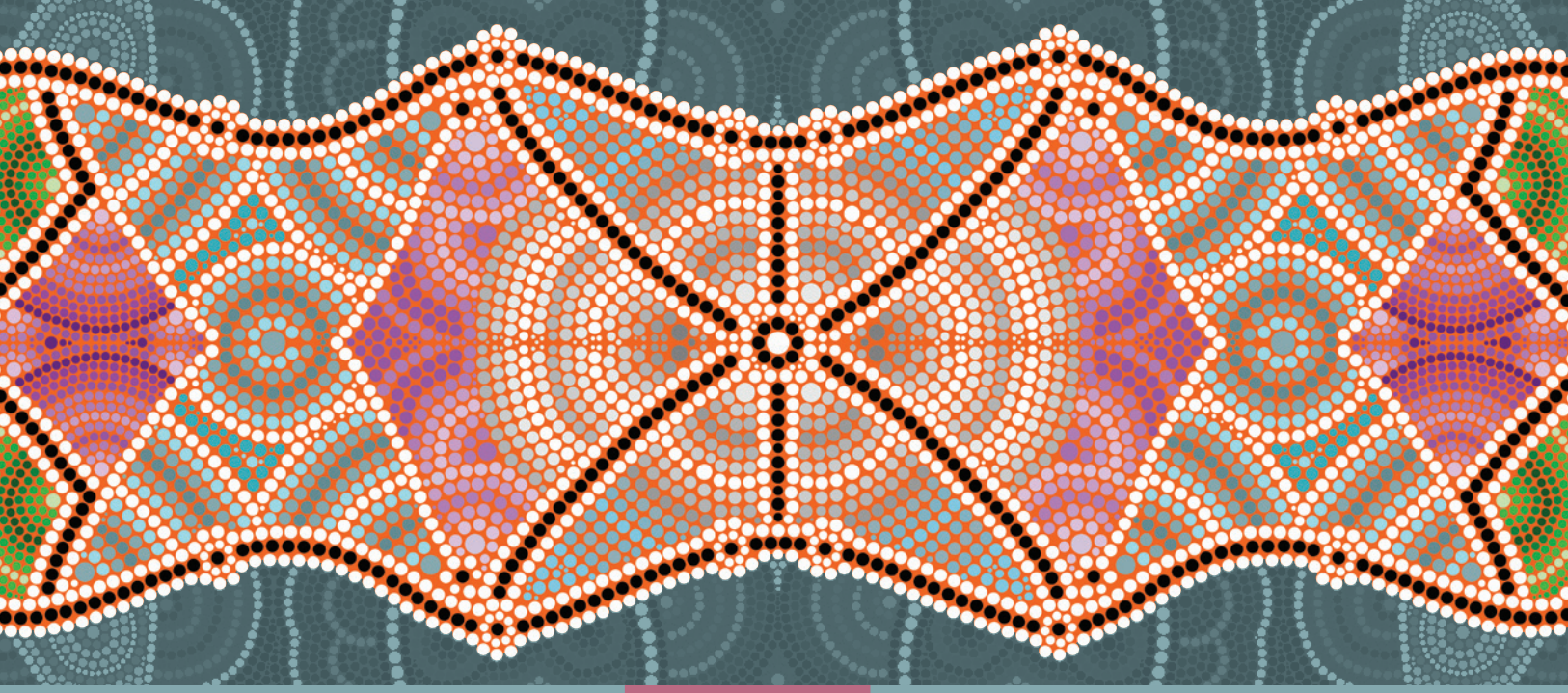

UNIT 2

ALTERATIONS IN PATTERNS OF HEALTH

CHAPTER 3

NURSING CARE OF PEOPLE HAVING SURGERY

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LEARNING OUTCOMES

- Discuss the differences and similarities between outpatient and inpatient surgery.
- Describe legal requirements and responsibilities prior to surgery.
- Describe the various classifications and risk factors of surgical procedures.
- Identify diagnostic tests used during the perioperative period.
- Describe nursing implications for medications prescribed for the person having surgery.
- Describe appropriate nursing care for people having surgery in the preoperative, intraoperative and postoperative phases of surgery.
- Identify variations in perioperative care for the older adult.
- Describe principles of pain management specific to acute postoperative pain control.
- Apply the nursing process as a framework for providing individualised care for people undergoing surgery.

CLINICAL COMPETENCIES

- Assess the physiological health status of people having surgery to determine their ability to tolerate surgery and risks of complications.
- Assess the psychosocial health status of the person having surgery and their family.
- Ensure safety of the person having surgery within the operating theatre and throughout the postoperative period.
- Participate in education for the person and their family prior to anaesthesia and prior to discharge from the facility.
- Describe the sterile field in the operating theatre, practise infection control and take occupational health and safety precautions to prevent infections and minimise hazards.
- Physiologically monitor the person during surgery. As part of the interprofessional team, promote safe practice and rehabilitation of the person having surgery.
- Monitor and control the environment to prevent accidents or injury to the person having surgery and members of the healthcare team.
- Respect the person's rights, including privacy, at all times.

KEY TERMS

Surgery is an invasive medical procedure performed to diagnose or treat illness, injury or deformity. Although surgery is a medical treatment, the nurse assumes an active role in caring for the person before, during and after surgery. **Interprofessional care** and teamwork by nurses, surgeons, anaesthetists, pharmacists and allied health professionals can prevent complications and promote optimal recovery of the person having surgery.

Perioperative nursing is a specialised area of practice. It incorporates the three phases of the surgical experience: preoperative, intraoperative and postoperative. The **preoperative** phase begins when the decision for surgery is made and ends when the person having surgery is transferred to the operating room. The **intraoperative phase** begins with the person's entry into the operating room and ends with transfer to the post anaesthesia care unit (PACU), or recovery room. The **postoperative phase** begins with the person's arrival at the PACU and ends with their complete recovery from the surgical intervention.

CLASSIFICATIONS OF SURGICAL PROCEDURES

Surgical procedures can be classified according to purpose, risk factor and urgency (see Table 3.1). Based on this information, nursing care can be individualised to meet the needs of the person having surgery.

Although the perioperative nurse works in collaboration with other healthcare professionals to identify and meet the needs of the person having surgery, the perioperative nurse has the primary responsibility and accountability for nursing care of the person undergoing surgery.

SETTINGS FOR SURGERY

People having surgery may be inpatients or outpatients. The complexity of the surgery and the recovery and expected condition of the person following the surgery are the major differences. Sometimes outpatients (i.e. people intending to be discharged home immediately following surgery) are admitted to the hospital. Cataract removal with or without lens implants, hernia repairs, tubal ligations, vasectomies, dilation and curettage (D&C), haemorrhoidectomies and biopsies are commonly performed in outpatient surgeries.

Inpatient and outpatient surgeries are performed in the same operating suites in most hospitals. There are also **day surgery units/centres** (also called *ambulatory surgery*), which are not physically connected to a hospital. Perioperative nursing roles support people having surgery irrespective of whether surgeons practise in hospitals or in freestanding surgical facilities. Some outpatient surgeries are performed in surgeons' offices rather than surgical centres. The number of outpatient surgeries has grown rapidly in the past decade as part of the effort to contain the high costs of surgery. Moreover, increasingly complex surgeries on people with complicated medical problems are now commonly performed on an outpatient basis. This increase in number of procedures and acuity level of people having surgery presents challenges for the perioperative nurse, the person having surgery and their family.

Outpatient surgery potentially offers several advantages:

- decreased cost to the person having surgery and the hospital
- reduced risk of hospital-acquired infection
- less interruption to the person's and their family's routine
- possible reduction in time lost from work and/or other responsibilities
- less physiological stress for the person and their family.

TABLE 3.1 Classification of surgical procedures

	CLASSIFICATION	FUNCTION	EXAMPLES
Purpose	Diagnostic	Determine or confirm a diagnosis	Breast biopsy, bronchoscopy
	Ablative	Remove diseased tissue, organ or extremity	Appendectomy, amputation
	Constructive	Build tissue/organs that are absent (congenital anomalies)	Repair of cleft palate
	Reconstructive	Rebuild tissue/organ that has been damaged	Skin graft after a burn, total joint replacement
	Palliative	Alleviate symptoms of a disease (not curative)	Bowel resection in a person with terminal cancer
	Transplant	Replace organs/tissue to restore function	Heart, lung, liver, kidney transplant
Risk factor	Minor	Minimal physical assault with minimal risk	Removal of skin lesions, dilation and curettage (D&C), cataract extraction
	Major	Extensive physical assault and/or serious risk	Transplant, total joint replacement, cholecystectomy, colostomy, nephrectomy
Urgency	Elective	Suggested, though no foreseen ill effects if postponed	Cosmetic surgery, cataract surgery, bunionectomy
	Urgent	Necessary to be performed within 1 to 2 days	Heart bypass surgery, amputation resulting from gangrene, fractured hip
	Emergency	Performed immediately	Obstetric emergencies, bowel obstruction, ruptured aneurysm, life-threatening trauma

Outpatient surgery also presents some disadvantages:

- less time for the nurse to establish rapport with the person and their family
- less time for the nurse to assess, evaluate and teach the person and their family
- lack of opportunity for the nurse to assess for the risk of postoperative complications that may occur after discharge
- less time for adequate pain control before discharge.

Following outpatient surgery, the person is discharged after meeting the discharge criteria of the institution and demonstrating the following:

- normal vital signs
- ability to stand and begin to walk without dizziness or nausea
- minimal pain
- ability to urinate
- orientation to time and place
- understanding of postoperative instructions.

Many similarities exist between the nursing care of people admitted for inpatient and for outpatient surgery. Physical care is provided in much the same manner in the preoperative, intraoperative and postoperative phases of surgery. The major differences lie in the degree of teaching and emotional support that must be provided for people having outpatient surgical procedures and their families. In addition to the physiological insult of surgery, the person having outpatient surgery must cope with the stress of needing to learn a great deal of information in a short span of time. The nurse teaches the person and their family in both the preoperative and postoperative periods to enable the person to perform self-care following discharge and recognise the development of complications that require further clinical management. More extensive teaching and emotional support is necessary as people requiring more complex surgical procedures and experiencing more complicated health problems undergo outpatient surgery.

CONSIDERATION FOR PRACTICE

People having outpatient surgery should wear or bring clothing that will be easy to put on after surgery and accommodate any dressings or appliances. Despite fasting, people having outpatient surgery should bring any medications such as steroids, antibiotics, anticoagulants, antivirals, diuretics, oral contraceptives, hypotensives, cardiotonics, hypoglycaemics, asthma medications, seizure medications and analgesics, as well as any herbal preparations, that they regularly use. People having surgery should consult with the surgeon and anaesthetist before taking these medications, prior to and following surgery.

LEGAL REQUIREMENTS

It is the responsibility of the surgeon who performs the procedure to obtain the person's consent for the surgical procedure. The surgeon should discuss the procedure with the person and their family in language they can understand. **Informed consent** is disclosure of the risks associated with the intended procedure or operation to the person, and includes completion of a legal document required for certain diagnostic procedures or therapeutic measures, including surgery. The language of the document varies according to the statutory and common law of each state. This legal document protects the person undergoing surgery, the nurse, the surgeon and the healthcare facility.

Informed consent includes provision of the following information prior to a person signing a consent for medical procedure treatment form:

- need for the procedure in relation to the diagnoses
- description and purpose of the proposed procedure
- possible benefits and potential risks
- likelihood of a successful outcome
- alternative treatments or procedures available
- anticipated risks should the procedure not be performed
- surgeon's advice about what is needed
- the person's right to refuse treatment or withdraw consent.

The nurse may be present when the preceding information is provided. Later, the nurse can discuss the information with the person and their family, if necessary. If the person having surgery has questions or concerns that were not discussed or made clear, or if the nurse questions the person's understanding, the surgeon is responsible for supplying further information. Following a thorough discussion of the consent for operation or special procedures, the person having surgery voluntarily signs the form. Depending on various state and hospital policies, the nurse may also sign the form, indicating that the correct person is signing the form and that the person was alert and aware of what was being signed. In many circumstances the person having surgery is admitted to the surgical unit or day surgery with an already completed consent form.

PERIOPERATIVE RISK FACTORS

Prior to planning and implementing care for the person having surgery, the nurse must first assess their needs and the factors that may increase the risks associated with surgery. The type of surgical procedure directs the assessment and interventions planned by the nurse. However, a complete assessment is also necessary to identify *risk factors* and to determine the person's overall health status. Table 3.2 lists common risk factors for the person undergoing surgery and the related nursing interventions and implications. For example, when a person is admitted for surgery on the right knee, it should be of concern to the nurse if this person has diabetes, smokes 25 cigarettes per day, has numbness in the right foot and takes insulin. This information should be incorporated into a nursing care plan, using appropriate nursing diagnoses and interventions to meet all of the person's needs and to assist them towards full postoperative recovery.

Risks are associated with all surgical interventions. For example, transporting the person to and from the operating suite requires assessment of their need for supplementary oxygen, intravenous therapy, cardiac monitoring and safety issues pertaining to the means of transport. Many people enter the operating suite highly anxious and may benefit from medication to help them relax prior to administration of anaesthesia. This can be discussed with the anaesthetist. Chemicals, electrical equipment and environmental hazards in the surgical area have the potential to cause harm and must be monitored and maintained carefully.

TABLE 3.2 Nursing implications for surgical risk factors

FACTOR	ASSOCIATED RISK	NURSING IMPLICATIONS
Advanced age	Older adults have age-related changes that affect physiological, cognitive and psychosocial responses to the stress of surgery; decreased tolerance of general anaesthesia and postoperative medications; and delayed wound healing.	Selected nursing interventions are summarised in Table 3.6.
Obesity	The obese person is at increased risk of delayed wound healing, wound dehiscence, infection, pneumonia, atelectasis, thrombophlebitis, arrhythmias and heart failure.	Promote weight reduction if time permits. Monitor closely for wound, pulmonary and cardiovascular complications postoperatively. Encourage coughing, turning, and diaphragmatic breathing exercises and early ambulation.
Malnutrition	Reserves may not be sufficient to allow the body to respond satisfactorily to the physical assault of surgery; organ failure and shock may result. Increased metabolic demands may result in poor wound healing and infection.	With the surgeon and dietitian, promote weight gain by providing a well-balanced diet high in kilojoules, protein and vitamin C. Administer nutritional supplements and, if indicated, total parenteral nutrition intravenously, and tube feedings as prescribed. Daily weights and kilojoule counts also may be ordered.
Dehydration/ electrolyte imbalance	Depending on the degree of dehydration and/or type of electrolyte imbalance, cardiac arrhythmia or heart failure may occur. Liver and renal failure may also result.	Administer intravenous fluids as ordered. Monitor fluid input and output and weight. Monitor for evidence of fluid and electrolyte imbalance (see Chapter 9). Closely monitor fluid intake (oral and parenteral) to prevent circulatory overload.
Renal and liver dysfunction	The person with renal or liver dysfunction may poorly tolerate general anaesthesia, have fluid/electrolyte and acid–base imbalances, decreased metabolism and excretion of drugs, increased risk of haemorrhage, and delayed wound healing.	Monitor for fluid volume overload, intake and output, and response to medication. Evaluate closely for drug side effects and evidence of acidosis or alkalosis.
Cardiovascular disorders	Presence of cardiovascular disease increases the risk of haemorrhage and shock, hypotension, thrombophlebitis, pulmonary embolism, stroke (especially in the older person) and fluid volume overload.	Diligently monitor vital signs, especially pulse rate, regularity and rhythm, and general condition of the person. Assess skin colour. Assess for chest pain, lung congestion and peripheral oedema. Observe for signs of hypoxia, and administer oxygen as ordered. Early postoperative ambulation and leg exercises reduce the risk of vascular problems such as thrombophlebitis and pulmonary embolism.
Respiratory disorders	Respiratory complications such as bronchitis, atelectasis and pneumonia are some of the most common and serious postoperative complications. Respiratory depression from general anaesthesia and acid–base imbalance may also occur. People with pulmonary disease are more at risk of developing these complications.	Closely monitor respirations, pulse and breath sounds. Also assess for hypoxia, dyspnoea, lung congestion and chest pain. Encourage coughing, turning and diaphragmatic breathing exercises and early postoperative ambulation. Encourage the person to stop smoking or at least to reduce the number of cigarettes smoked.
Diabetes mellitus	Diabetes causes an increased risk of fluctuating blood glucose levels, which can lead to life-threatening hypoglycaemia or ketoacidosis. Diabetes also increases the risk of cardiovascular disease, delayed wound healing and wound infection.	Monitor the person closely for signs and symptoms of hypoglycaemia and hyperglycaemia. Monitor blood glucose levels every 4 hours or as ordered. Administer insulin if prescribed. Encourage intake of food at the designated meal and snack times.
Alcoholism	The person may be malnourished and experience alcohol withdrawal and delirium tremens. More general anaesthesia may be required. Haemorrhage and delayed wound healing can result from liver damage and poor nutritional status.	Monitor closely for signs of alcohol withdrawal and delirium tremens. Encourage well-balanced diet. Monitor for wound complications. Administer supplementary nutrients parenterally as ordered.

TABLE 3.2 Nursing implications for surgical risk factors (continued)

FACTOR	ASSOCIATED RISK	NURSING IMPLICATIONS
Nicotine use	Cigarette smokers are at increased risk of respiratory complications such as pneumonia, atelectasis and bronchitis because of increased mucous secretions and a decreased ability to expel them.	Ideally, the person having surgery should stop smoking. Be supportive of them, and monitor closely for respiratory difficulties. Coughing, turning and diaphragmatic breathing exercises with early ambulation are very important. Increase daily fluid intake to 2500–3000 mL (unless contraindicated) to help liquefy respiratory secretions to aid expectoration. A nicotine patch may help them to tolerate withdrawal during the postoperative period.
Adolescence	Diversity in age and physical, cognitive and psychological maturation makes preparation for surgery vary in content and inclusion of significant others. Increased need for control, privacy and peer interaction poses special challenges in the acute care setting.	Adapt assessment and interventions to the developmental level of individual people, involving them in preparation and care to the extent possible. Allow for regressive and independent behaviour, including rejection of adult support.
Medications	Anaesthesia interaction with some medications can cause respiratory difficulties, hypotension and circulatory collapse. Other medications can produce side effects that may increase surgical risk.	Inform the anaesthetist of all prescribed or over-the-counter medications.
Anticoagulants (including aspirin)	May cause intraoperative and postoperative haemorrhage.	Monitor for bleeding. Assess prothrombin time (PT value) and partial thromboplastin time (PTT value).
Diuretics (particularly thiazides)	May lead to fluid and electrolyte imbalances, producing altered cardiovascular response and respiratory depression.	Monitor fluid input and output and electrolytes. Assess cardiovascular and respiratory status.
Antihypertensives (particularly phenothiazines)	Increase the hypotensive effects of anaesthesia.	Closely monitor blood pressure.
Antidepressants (particularly monoamine oxidase inhibitors)	Increase the hypotensive effects of anaesthesia.	Closely monitor blood pressure.
Antibiotics (particularly the 'mycin' group)	May cause apnoea and respiratory paralysis.	Monitor respirations.
Herbal supplements	Some may prolong the effects of anaesthesia. Others may increase the risks of bleeding or raise blood pressure.	Inquire about the use of herbs or other dietary supplements. These should be discontinued at least 2 weeks before surgery.
Temperature variations	Deviations from normothermia, either hypothermia or hyperthermia, may cause infection, cardiac morbidity, myocardial ischaemia, surgical bleeding, skin damage or discomfort for the person having surgery.	Monitor core temperatures and prevent chilling or overheating. Use warmed fluids. Remove wet drapes and ensure the person is clean and dry immediately post surgery.

Unfortunately, there is a risk of performing the wrong surgery on the wrong person: including wrong site, wrong procedure and/or wrong person. In 2012, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) (2012) issued a universal protocol mandating preoperatively: (1) verifying the procedure, (2) physically marking and initialling the site, and (3) taking a 'time out' before starting any procedure. The goal of the time out is to ensure the right procedure will be performed on the right person on the correct site with the necessary and correct healthcare professionals in attendance. A 'time out' is an intentional stoppage of the preparation for the operation, in the operating suite, before the person having surgery is anaesthetised. All participants are introduced and encouraged to ask any questions or express any concerns before the surgery is commenced to prevent adverse outcomes. The World Health Organization (WHO) and World Alliance for Patient Safety (2009) now recommends use of the Surgical Safety Checklist for this purpose. This checklist has been reported to significantly reduce surgical morbidity and mortality (de Vries et al., 2010), and a statement on the WHO Patient Safety (2009) webpage claims that half a million deaths per annum are potentially preventable if the checklist is implemented worldwide.

Another potential error is retained foreign bodies such as instruments, needles or sponges. To prevent accidental retention of foreign objects, standardised procedures for counting objects used in surgery must be consistently applied and

adhered to. Methodical wound exploration before closing the site, using x-ray-detectable materials in the wound, maintenance of optimal operating theatre environments to allow focused surgical performance and employing technological methods to ensure no unintended item remains are now the norm in surgical practice. Like wrong site injury, foreign body retainment is linked to poor communication among perioperative care providers and faulty processes of care in the operating theatre and perioperative period (Greenberg et al., 2007).

People having surgery may be using medications that increase the risk of complications associated with a surgical procedure, such as increasing the risk of bleeding. Over-the-counter medicines and herbal preparations, as well as prescription medications, may interact with drugs given during surgery, putting a person at increased risk (Collins, Oakey & Ramakrishnan, 2011; King et al., 2009). A complete history of the medications the person has been taking regularly is vital information. As part of the preoperative planning and teaching, early consideration of complementary and alternative medicine is also important. This information should be obtained in a non-judgmental manner, because a judgmental attitude could cause the person to withhold information.

Anticoagulant medications are frequently discontinued by the anaesthetist in the preoperative clinic prior to surgery to prevent excessive blood loss during surgery. These include aspirin and non-steroidal anti-inflammatory drugs. If laboratory tests of clotting/bleeding time, PT, PTT and INR (International Normalised Ratio) are elevated, the surgery may be cancelled. Guidelines for discontinuing use vary according to the particular medication; it is generally recommended that aspirin or products containing aspirin be discontinued 5 days or longer before surgery. Similarly, herbs or nutritional supplements that impair clotting should be discontinued 2 weeks prior to surgery (Collins, Oakey & Ramakrishnan, 2011; King et al., 2009). The most common self-prescribed medicines that may inhibit coagulation are vitamin E, garlic, ginkgo, ginseng, fish oil and chamomile. Many plants that contain coumarins have the potential to interact with warfarin and inhibit coagulation. Others inhibit platelet aggregation or prevent the conversion of fibrinogen to fibrin. All of these create a risk of bleeding.

People taking warfarin for the risk of blood clots due to atrial fibrillation will be counselled about the appropriate time to withdraw warfarin. If surgery is urgent due to trauma or sudden onset of morbidity, the impact of anticoagulants needs to be evaluated, with PT, PTT and INR results required before the operation and appropriate medication administered to promote clotting.

In addition to clotting impairment, excessive consumption of herbal medicines or dietary supplements can produce levels of chemicals that interact with conventional medications, exacerbating or impairing the intended effect (Collins, Oakey & Ramakrishnan, 2011). Anaesthetic drugs often decrease hepatic blood flow and interfere with the metabolism and elimination of medications. This increases the risk of adverse drug–herbal supplement interactions during surgery. Cardiovascular instability, impaired glucose control, increased metabolism of perioperative medication, and unpredictable response to anaesthesia are categories of adverse reactions of perioperative herbal use (King et al., 2009; Rowe & Baker, 2009).

In the perioperative period, hypothermia and hyperthermia are risks. Typically, surgical suites are maintained as cooler environments; however, research shows that normothermia (core body temperature in the range of 36.0˚C to 37.5˚C) in the person having surgery reduces the risk of infection, cardiac morbidity, myocardial ischaemia, surgical bleeding and personal discomfort (Burger & Fitzpatrick, 2009; Yang et al., 2012). Methods to minimise the risk of hypothermia include:

- Apply warm blankets on arrival in the surgical area and after sterile drapes are removed.
- Limit the amount of skin exposed during positioning and skin preparation.
- Limit the time of skin exposure between prepping and draping.
- Prevent surgical drapes from becoming wet.
- Adjust the room temperature for normothermia.
- Monitor the person's temperature to avoid overheating.
- Use heat-maintenance devices such as warming units, stockings, caps and leggings.
- Warm irrigation or infusion solutions as needed.
- Humidify the airway.
- Clean and dry the person immediately postoperatively.

An anaesthetised person loses heat intraoperatively and is unable to restore temperature through the normal mechanisms of shivering or muscle contractions (Burger & Fitzpatrick, 2009). Hyperthermia should also be avoided. Heating of fluids or use of heating units necessitates accurate measurement of the person's temperature. Body temperature is best evaluated through core temperature monitoring, which includes oesophageal or tympanic assessment.

Interpreting and responding to identified risk factors requires adequate clinical reasoning and interprofessional communication skills. It is important to bring concerns to the attention of the surgeon and/or anaesthetist prior to surgery, so that necessary modifications can be made for the person during the perioperative period.

CONSIDERATION FOR PRACTICE

Remind people with diabetes that the stress of surgery increases, rather than decreases, blood sugar. Coordinate insulin injections and/or hypoglycaemic medication with the person, surgeon and anaesthetist.

INTERPROFESSIONAL CARE

The person undergoing surgery receives care from a number of healthcare professionals. Surgeons, nurses, anaesthetists, anaesthetic technicians, venepuncturists, pathologists, x-ray technicians, physiotherapists and paramedical staff such as registration clerks and wardsmen/porters are often involved in securing the safety and health of people having surgery. Case managers, social workers and spiritual care providers are available based on the person's needs and preferences. This interprofessional approach focuses on maintaining the person in the best possible health status before, during and after surgery. For some major and frequently performed procedures, the interprofessional

team can develop and map a routine clinical process of recovery. This process is described in a document called a *clinical pathway* (or critical pathway, care path or map). These are multidisciplinary maps that document the various stages of recovery, and appropriate therapeutic responses by team members, in order to assist the person to arrive safely and successfully at the point of discharge. Any divergence from a stated pathway results in variances that must be carefully documented and appropriately managed by the interprofessional team.

Diagnostic tests

Thorough clinical preoperative assessments may be complemented by diagnostic tests if they are clinically indicated (Apfelbaum et al., 2012). Diagnostic tests performed prior to surgery provide baseline data or reveal problems that may place the person at additional risk during and after surgery. Because of the trend towards shortened hospital stays, many diagnostic studies and procedures are performed in a preadmission clinic (pre-op clinic) within a week prior to elective surgery as part of the preadmission assessment process.

Complete blood counts, electrolyte studies, coagulation studies and urinalysis are the most commonly performed preoperative laboratory tests. Table 3.3 discusses the significance and nursing implications of abnormal findings for these common tests. Additional diagnostic tests may be performed as the history and physical findings indicate. For example, if the person has a low haemoglobin and haematocrit, and significant blood loss during surgery is anticipated, then the surgeon may order a type and crossmatch of the person's blood for a possible transfusion.

In addition to laboratory tests, older people or those with risk factors related to heart and lung function typically have a chest x-ray. This radiological procedure provides baseline information about the size, shape and condition of the heart and lungs. Pulmonary complications such as lung disease, tuberculosis, calcification, infiltration or pneumonia may require that surgery be postponed to allow the person to undergo further evaluation or treatment. If findings are abnormal and the surgery cannot be postponed, information from the chest x-ray study can be used to determine the safest form of anaesthesia.

Another commonly performed preoperative diagnostic procedure is the electrocardiogram (ECG). This test is ordered routinely for people undergoing general anaesthesia (except for low-risk procedures) when they have cardiovascular disease or risk factors (Fleisher et al., 2014). The ECG provides data for the evaluation of either new or pre-existing cardiac conditions. The person's surgery may be cancelled or postponed if a life-threatening cardiac condition is discovered.

In addition to the chest x-ray study and ECG, other diagnostic tests may be performed preoperatively to gather further assessment data. For example, for people who have chronic obstructive pulmonary disease, lung function tests are often performed to determine the extent of respiratory dysfunction. This information guides the anaesthetist before and during surgery in choosing the type of anaesthetic to be used, and it guides the surgeon and nursing staff in the recovery phase.

The glomerular filtration rate is a rapid and useful indicator of renal function. A low serum creatinine is an indicator of good renal function, so the creatinine value must be confirmed in the urine; however, it is useful to know that normal values are lower in older adults due to decreased muscle mass. Older adults are especially susceptible to renal insufficiency, which puts them at risk of volume overload in the perioperative period and accumulation of metabolic by-products and medications dependent on renal clearance.

Pregnancy testing for women may also be useful prior to surgery, particularly for elective procedures.

TABLE 3.3 Laboratory tests for perioperative assessment

TEST	SIGNIFICANCE OF INCREASED VALUES	SIGNIFICANCE OF DECREASED VALUES	NURSING IMPLICATIONS
Haemoglobin (Hgb or Hb) and haematocrit (HCT)	Dehydration, excessive fluid plasma loss, polycythaemia vera	Fluid overload, excessive blood loss, anaemia	Monitor oxygenation, fluid input and output, and vital signs; assess for bleeding.
Glucose and haemoglobin-A (HbA1c)	Impaired glucose metabolism, stress or infection	Inadequate glucose intake in relation to insulin	If decreased, monitor for signs and symptoms of hypoglycaemia. Notify surgeon if < 48 mmol/mol or > 59 mmol/mol for diabetics and < 20 mmol/mol or > 42 mmol/mol for non-diabetics.
White blood cell (WBC) count	Infectious/inflammatory processes, leukaemia	Immune deficiencies	Monitor for signs of inflammation; monitor drainage, temperature and pulse. Use standard or transmission-based precautions.
Platelet count	Malignancies, polycythaemia vera	Clotting deficiency disorders, chemotherapy	If decreased, assess for bleeding at incision sites and drainage tubes, and assess for haematomas.
Carbon dioxide (CO_2)	Emphysema, chronic bronchitis, asthma, pneumonia, respiratory acidosis, vomiting, nasogastric (NG) suctioning	Metabolic acidosis, hyperventilation	Monitor respiratory status and arterial blood gases (ABGs).

(continued)

TABLE 3.3 Laboratory tests for perioperative assessment (continued)

TEST	SIGNIFICANCE OF INCREASED VALUES	SIGNIFICANCE OF DECREASED VALUES	NURSING IMPLICATIONS
Oxygen (O_2)	Can be excessive for exacerbated COPD patients or CO_2 retainers	May indicate respiratory depression associated with narcotic analgesia or physiological deterioration including haemorrhage	Monitor O_2 % using pulse oximetry and ABGs if respiratory status deteriorates.
Electrolytes:			
Potassium (K^+)	Kidney dysfunction, dehydration, suctioning	Side effects of diuretics, vomiting, NG suctioning	Monitor K^+ level, cardiac and neurological function, and preoperative diuretic therapy.
Sodium (Na^+)	Kidney dysfunction, normal saline-containing intravenous fluids	Side effects of diuretics, vomiting, NG suctioning	Monitor Na^+ level and fluid input and output; assess for peripheral oedema and effects of perioperative diuretic therapy.
Chloride (Cl^-)	Kidney dysfunction, dehydration, alkalosis	Side effects of diuretics, vomiting, NG suctioning	Monitor Cl^- level and fluid input and output; assess for peripheral oedema and perioperative diuretic therapy.
Prothrombin time (PT) and partial thromboplastin time (PTT)	Defect in mechanism for blood clotting, anticoagulant therapy (aspirin, heparin, warfarin), side effect of other drugs affecting clotting time	Hypercoagulability of the blood may lead to thrombus formation in the veins	If clotting time is elevated, monitor PT/PTT values. Assess for bleeding at incision site and drainage tubes and for haematomas. If clotting time is decreased, monitor for thrombus formation (pulmonary emboli, thrombophlebitis), and evaluate PT and PTT values.
Urinalysis	Varied	Varied	Used to detect abnormal substances (e.g. protein, glucose, red blood cells or bacteria) in the urine. Notify surgeon if abnormalities are detected.

Medications

The person having surgery receives medications before, during and after surgery to achieve specific therapeutic outcomes. Routine oral medications may be withheld during preoperative fasting periods, and the anaesthetist will normally specify medications that should be given, and those that should be withheld. People with diabetes require careful management of their medications during fasting so they don't experience extreme fluctuations in blood glucose levels and associated risks. The anaesthetist will often order pain medication, anticoagulants and antibiotics postoperatively if indicated. Generally, routine medication orders must be revised by the surgeon or anaesthetist when the person returns to the postsurgical care unit.

The person having surgery may be given preoperative medications 45 to 70 minutes before the scheduled surgery depending on medical orders. Any delay in administration should be reported promptly to the anaesthetist. Preoperative medications may also be given in the anaesthetic room to produce the desired effects.

An increasingly common strategy to prevent intense or lingering pain is the use of *pre-emptive analgesia*. Pre-emptive analgesia prevents sensitisation of the central and peripheral nervous system by painful stimuli, by blocking the pain pathways with local, regional or epidural analgesia prior to incision. Sensitisation to pain prolongs the painful experience; however, blocking the sensitisation throughout the perioperative period results in decreased pain in the postoperative period, shortened hospital stay, quicker return to self-care and decreased residual pain (Hariharan et al., 2009).

A combination of preoperative drugs may be ordered to achieve the desired outcomes with minimal side effects. Such outcomes include sedation, reducing anxiety, inducing amnesia to minimise unpleasant surgical memories, increasing comfort during preoperative procedures, reducing gastric acidity and volume, increasing gastric emptying, decreasing nausea and vomiting, and reducing the incidence of aspiration by drying oral and respiratory secretions.

Antibiotic prophylaxis is effective in the prevention of postoperative complications in many surgeries (Kwarteng, Ahluwalia & Osborne, 2008; Smaill & Gyte, 2010). In Australia, the Therapeutic Guidelines Limited (2014) publishes antibiotic guidelines and these are used to select the appropriate antibiotics, depending on the procedure to be performed. The timing of antibiotic prophylaxis is an important aspect of providing this preventive therapy (Kable, Gibberd & Spigelman, 2008a).

Thromboprophylaxis should also be provided for many procedures. People should be assessed to identify their risk factors for developing postoperative venous thrombosis. Deep vein thrombosis occurs in more than 20% of people who undergo major surgery, with an increased incidence following orthopaedic surgery (Joanna Briggs Institute (JBI), 2008). People having surgery who develop postoperative venous thrombus may complain of pain, tenderness, swelling, increased heat, changes to skin colouration or enlarged veins. Adverse outcomes may lead to pulmonary embolism, sudden death or post-thrombotic syndrome (JBI, 2008). Thromboprophylaxis includes pharmacological strategies such as the use of low molecular weight heparin (LMWH) preparations,

and a range of non-pharmacological strategies including thigh-length approved compression profile thromboembolic stockings, sequential compression devices and ambulation as soon as possible after surgery (JBI, 2008). The National Health and Medical Research Council (NHMRC) (2009) has published clinical practice guidelines for thromboprophylaxis in Australian hospitals.

Table 3.4 outlines some commonly prescribed preoperative medications.

Decisions made about which of the person's routine medications to administer prior to surgery when the person is required to be nil by mouth (NBM) are made in consultation with the surgeon and/or anaesthetist. Caution is required in relation to potential interactions between anaesthesia and medications and the effect on the person if drugs such as steroids, antiseizure medications and tranquillisers are discontinued abruptly. Generally, insulin is withheld when the person is NBM, but depending on the anticipated length of the surgery, the dosage may be adjusted for the previous evening as well as the morning of surgery. Under anaesthesia the signs and symptoms of hypoglycaemia (insulin reaction) are absent, so withholding insulin the morning of surgery when the person is NBM is advisable. Plasma glucose is monitored intermittently during surgery with the goal of maintaining a normal blood sugar level (see Table 3.2). People who ordinarily manage their diabetes mellitus with oral medications often experience hyperglycaemia perioperatively (Betts, Brink, Silink et al., 2009). It is common to manage hyperglycaemia with sliding-scale insulin. However, evidence-based practice now supports subcutaneous basal insulin administration for hyperglycaemic people to maintain glycaemia at 48–59 mmol/mol (previously measured in mmol/L in Australia) throughout the perioperative period. This practice is associated with better healing, fewer infections and shorter hospital stays (Betts et al., 2009).

Assessment of medications that the person normally uses is vital prior to anaesthesia. In addition to medications prescribed by a doctor, assessment should include over-the-counter preparations (including aspirin and illegal drugs) and herbal medications (see Table 3.2).

INTRAOPERATIVE MEDICATIONS **Anaesthesia** is used to produce unconsciousness, analgesia, reflex loss and muscle

TABLE 3.4 Preoperative medications

GENERIC	ACTION BY CATEGORY	NURSING IMPLICATIONS
Antibiotics	Prevent surgical site infections in orthopaedic and general surgeries and are associated with lower risk of mortality in elderly people	Correct timing is important for maximum effectiveness. Monitor people for reactions. Be aware of microbial resistance and responsible use of medicines.
Thromboprophylaxis: low molecular weight heparins (LMWHs)	Prevents formation of clots in the peripheral circulation which otherwise may result in deep venous thrombosis and possible emboli	Be aware of bruising and monitor bleeding.
Benzodiazepines (midazolam, diazepam, lorazepam)	Decrease anxiety and produce sedation to some extent. May induce amnesia	Monitor for respiratory depression, hypotension, drowsiness and lack of coordination.
Narcotic analgesics (morphine, fentanyl)	Decrease anxiety, provide analgesia	Monitor for respiratory depression and safety if ambulating. Anti-emetics may be needed.
Non-opioid analgesics	Provide mild to moderate analgesia. Single dose or 4-hourly administration over a short period. Sometimes referred to as NSAIDs, due to antipyretic and anti-inflammatory actions	Reassessment required if pain has not ceased. Assess for adverse effects. Cease 72 hours preoperatively and assess clotting times.
Antacids (sodium citrate)	Increase the pH and reduce volume of gastric fluid; used in people with gastro-oesophageal reflux disease and/or trauma	No significant factors in this setting.
H_2 receptor antagonists (cimetidine, famotidine, ranitidine)	Reduce gastric acid volume and concentration	Monitor for confusion and dizziness in older adults.
Gastric acid pump inhibitors (lansoprazole, pantoprazole, omeprazole)	Suppress gastric acid secretion	Monitor for dizziness and headache, rash or thirst.
Anti-emetics (metoclopramide, prochlorperazine, ondansetron)	Enhance gastric emptying. Often used with narcotic analgesics to alleviate side effects of nausea and vomiting	Monitor for sedation and extra-pyramidal reaction (involuntary movement, muscle tone changes and abnormal posture).
Anticholinergics (atropine sulfate, scopolamine)	Reduce oral and respiratory secretions to decrease risk of aspiration; decrease vomiting and laryngospasm	Monitor for confusion, restlessness and tachycardia. Prepare the person to expect a dry mouth.

relaxation during a surgical procedure. General anaesthesia produces these effects, whereas regional anaesthesia results in analgesia, reflex loss and muscle relaxation but does not cause the person to lose consciousness. An anaesthetist administers anaesthetics during the intraoperative phase of surgery.

General anaesthesia **General anaesthesia** is most commonly administered by inhalation and, to a lesser extent, by the intravenous route. It produces central nervous system depression. As a result, the person loses consciousness and does not perceive pain, skeletal muscles relax and reflexes diminish.

Advantages of general anaesthesia include rapid excretion of the anaesthetic agent and prompt reversal of its effects when desired. Additionally, general anaesthesia can be used with all age groups and any type of surgical procedure. It produces amnesia.

Disadvantages of general anaesthesia include risks associated with circulatory, respiratory, hepatic and renal side effects. People with serious respiratory or circulatory diseases, such as emphysema or congestive heart failure, are at greater risk of complications. People with renal or hepatic disease cannot metabolise and eliminate anaesthetics safely.

General anaesthesia is provided with inhalation agents or total intravenous anaesthesia (TIVA), also known as neuroleptanalgesia. People with a history of malignant hyperthermia (MH) avoid inhalational agents because they can trigger this complication (see Box 3.1). With the increase in ambulatory and minimally invasive surgeries, anaesthetics that enable shorter recovery phases are used, allowing a *fast-tracking* approach with a rapid recovery phase, and people who are managed this way will often bypass the PACU.

General anaesthesia comprises three distinct phases: induction, maintenance and emergence. During the induction phase, the person receives the anaesthetic agent intravenously or by inhalation. During this phase, airway patency is achieved and maintained with either endotracheal intubation or alternative devices including the laryngeal mask airway (LMA), oesophageal–tracheal Combitube, or lighted stylet or wand. These alternative methods of airway maintenance do not require direct visualisation of the vocal cords for placement, as is required for the endotracheal tube, but still maintain adequate ventilation. Intubation can be difficult with some people, and these alternative devices are an option to avoid other more invasive airway alternatives such as creation of a surgical airway with a cricothyroidotomy or tracheostomy.

The next phase of general anaesthesia is maintenance. During this period, the person is positioned, the skin is prepared and surgery is performed. The anaesthetist maintains the required depth of anaesthesia while constantly monitoring physiological parameters such as heart rate, blood pressure, respiratory rate, temperature, and oxygen and carbon dioxide levels. The final phase of anaesthesia is the person's emergence from this altered physiological state. As the anaesthetic agents are withdrawn or the effects reversed pharmacologically, the person begins to awaken. The endotracheal tube or laryngeal mask is removed (extubated) once the person is able to re-establish voluntary breathing. It is critical to ensure airway patency during this period, because extubation may cause bronchospasm or laryngospasm.

Regional anaesthesia **Regional anaesthesia** is a type of local anaesthesia in which medication instilled around the nerves blocks transmission of nerve impulses in a particular area. Regional anaesthesia produces analgesia, relaxation and reduced reflexes. The person is awake and conscious during the surgical procedure but does not perceive pain. Regional anaesthesia may be classified in several ways:

- Local nerve infiltration is achieved by injecting lignocaine around a local nerve to depress nerve sensation over a limited area of the body. This technique may be used when a skin or muscle biopsy is obtained or when a small wound is sutured.

BOX 3.1 Malignant hyperthermia

Malignant hyperthermia (MH) is a rare but serious reaction to volatile inhalational anaesthetic gases and succinylcholine, a depolarising neuromuscular blocker. The person who suffers this reaction manifests the following signs and symptoms: unexplained rise in end-tidal carbon dioxide that does not respond to ventilation, hyperthermia, tachypnoea, tachycardia and sustained skeletal muscle contraction. If unchecked the condition can progress to hyperkalaemia, myoglobinuria, disseminated intravascular coagulation, congestive heart failure, bowel ischaemia and compartment syndrome in the limbs. Dantrolene sodium is the drug that inhibits the muscular pathology and prevents death.

Because the condition is inherited, susceptibility testing is available but the testing is expensive and the most accurate test involves an invasive procedure. The 'gold standard' involves biopsy of thigh skeletal muscle tissue to determine sensitivity to caffeine and halothane (CHCT). Genetic testing is not as sensitive and reliable as CHCT but will be improved with the discovery of more causative mutations. People with muscle myopathies such as muscular dystrophy sometimes experience early signs of MH and respond well to dantrolene. The symptoms of MH may manifest with other pathologies, so it is important for people to know if they have a genetic susceptibility to MH, which could affect all members of their family.

MH can develop during an operation or when the person returns to the PACU. If the early symptoms of MH (e.g. escalating temperature, increased carbon dioxide production) are suspected, immediately administer 100% oxygen with a non-rebreather mask, stay with the person, ensure good IV access, and immediately call the anaesthetist who will order dantrolene which can be given as an IV bolus dose. Administration of dantrolene can be repeated until the signs and symptoms of MH diminish. Measures to decrease core body temperature should be started at once and continued until core temperature is 36.0°C. A urinary catheter should be placed to monitor urine output. Blood samples are taken and sent to pathology for testing. Blood gases should measure pH; and sodium bicarbonate is given to correct metabolic acidosis. Insulin may be ordered to decrease serum potassium. These people require critical care and are transferred to the intensive care unit for continued monitoring and doses of dantrolene every 4–6 hours.

- Nerve blocks are accomplished by injecting an anaesthetic agent at the nerve trunk to produce a lack of sensation over a specific area, such as an extremity.
- Epidural blocks are local anaesthetic agents injected into the epidural space, outside the dura mater of the spinal cord. This type of intraspinal anaesthesia provides effective pain relief for surgeries for people of all ages, with less risk of adverse effects than general anaesthesia. It is indicated for surgeries of the arms and shoulders, thorax, abdomen, pelvis and lower extremities. The epidural catheter is often left in place for pain relief in the postoperative period.
- Spinal anaesthesia is administered similarly to epidural except the anaesthetic medication is infused in a single injection. Spinal anaesthesia is effective for approximately 90 minutes. Surgeries of the lower abdomen, perineum and lower extremities are likely to use this type of regional anaesthesia. Any leakage of cerebrospinal fluid (CSF) into the epidural space may cause reduced CSF pressure and postoperative headaches. Treatment for this headache may include hydration, caffeine, analgesics or administration of an epidural blood patch. Hypotension is common with epidural and spinal anaesthesia. Monitor BP and, if critical hypotension occurs, alert the anaesthetist and expect to increase intravenous fluids and administer vasoactive medications.

CONSIDERATION FOR PRACTICE

The addition of adrenaline to lignocaine suppresses bleeding around the surgical site. Care must be taken to ensure that plain lignocaine is only ever used for end digit surgery (i.e. fingers, toes, tip of nose) to ensure that tissues remain perfused.

Conscious sedation An increasing number of surgical and diagnostic procedures are being performed using **conscious sedation**. This type of anaesthesia provides analgesia, amnesia and moderate sedation. The pharmacological effects are produced by administering a combination of intravenous medications with opioids (such as morphine sulfate and fentanyl) and/or sedatives (such as diazepam and midazolam). During conscious sedation the person is able to independently maintain an open airway. This allows them to respond to verbal and physical stimulation. Supervision by the surgeon or anaesthetist is always required, and staff must be prepared to initiate rescue if sedation becomes too deep. This type of sedation is increasingly being used in ambulatory care for procedures such as colonoscopy and cataract surgery.

Assessment prior to conscious sedation includes evaluating the person's physical status. People with compromised circulation or airway, a history of sleep apnoea or snoring, a history of problems with anaesthesia or analgesia, or medications that would potentially interact with conscious analgesia medications require more careful assessment. People who will be managed with conscious sedation should be appropriately fasted with a patent IV line in situ, and baseline vital signs are assessed and recorded prior to administration of the sedative. Equipment to rescue the person should be available if sedation becomes too deep. Monitor oxygen saturation, blood pressure, pulse, breathing and level of consciousness throughout the procedure.

Common adverse side effects include venous thrombosis, phlebitis, local irritation, confusion, drowsiness, hypotension and apnoea. Reversal agents (naloxone hydrochloride) are used as needed to enhance the safety of conscious sedation.

POSTOPERATIVE MEDICATIONS Management of acute postoperative pain by medication improves with greater understanding of pain physiology and the development of better methods to deliver adequate pain medication. For more information on pain management, see the 'Nursing care' section later in this chapter on managing acute postoperative pain, and also see Chapter 8.

Established, persistent, severe pain is more difficult to treat than recently commenced pain. Therefore, postoperative analgesics should be administered at regular intervals around the clock to maintain a therapeutic blood level. Administering analgesics on an 'as needed' (prn) basis lowers this therapeutic level and delays in medication administration further increase pain intensity. Therefore, regular rather than prn administration of analgesics is recommended in the first 36 to 48 hours postoperatively. People using patient-controlled analgesia (PCA) or patient-controlled epidural analgesia (PCEA) in the postoperative period need to be taught the importance of using the allowed dosages regularly to prevent increasing pain levels.

CONSIDERATION FOR PRACTICE

Nurses are responsible for assessing the pain level of people having surgery and administering pain medication. Work collaboratively with surgeons, anaesthetists and the pain team and people in the postoperative period to schedule postoperative analgesics, rather than rely on prn administration orders.

Non-steroidal anti-inflammatory drugs (NSAIDs) can be used to treat mild to moderate postoperative pain. This category of drugs should be given soon after surgery (orally, parenterally or rectally) along with opioids unless contraindicated. Although NSAIDs may not be sufficient to control pain completely, they allow lower doses of opioid analgesics and, therefore, fewer side effects. NSAIDs can usually be given safely to older people, but observe closely for side effects, particularly gastric and renal toxicity and bleeding. Be aware that NSAIDS are not comparable and their actions and side effects vary substantially, thus they cannot be used interchangeably.

Opioid analgesics, such as morphine, are considered to be most effective for managing moderate to severe postoperative pain. Opioid dosage requirements vary greatly from one person to another, so the dosage must be individually tailored. Later in the postoperative recovery period, opioid analgesics (oral or intramuscular) may be given prn. In this way, pain relief can be maintained while the potential for drug side effects is decreased.

Contrary to the belief of some healthcare professionals, physical dependence and tolerance to opioid analgesics is uncommon in short-term postoperative use. Additionally, opioid analgesics, when used to treat acute pain, rarely lead to psychological dependence and addiction. Acute pain can be appropriately treated initially with opioids, and subsequently with paracetamol as healing progresses. Management of

chronic or persistent pain, in contrast, evolves from paracetamol to opioids as tolerance develops or the condition worsens. The opioid-naive person will tolerate and achieve analgesia with a lower dose of opioid than the person who is opioid tolerant and uses opioids for persistent pain.

In the immediate postoperative period, older people benefit from the same protocol for morphine titration as younger people. Intravenous morphine may be initiated at a slightly reduced dose and then titrated to the same protocol as younger people. Morphine-related adverse effects such as nausea, vomiting, respiratory depression, urinary retention, pruritus and allergy or sedation are similar among age groups. However, older adults may require fewer opioids than younger people in the later postoperative period. PCEA may be more effective for older adults and is associated with earlier improved mental status and bowel activity (Rutledge, Caple & Pravikoff, 2011). Older people tend to be more sensitive to the analgesic effects of opioids, experiencing a higher peak effect with a longer duration of pain control.

Surgical environment

MEMBERS OF THE SURGICAL TEAM The intraoperative environment is complex and requires members of the surgical team to function as a coordinated unit. The surgeon, surgical assistant(s), perioperative nurse surgeon's assistant, anaesthetist, anaesthetic nurse or technician, instrument nurse (scrub) (see Figure 3.1) and circulating nurse (scout) constitute the main surgical team. Each member provides specialised skills and is essential to the successful outcome of the surgery. Risks to members of the surgical team from blood-borne pathogens or injury are minimised when the surgical team is well organised and prepared.

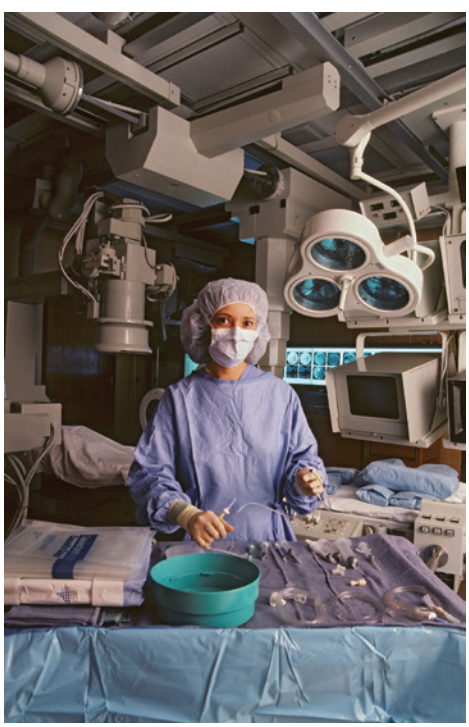


FIGURE 3.1 ■ An instrument nurse in the operating room

Source: ©Tom Tracy Photography/Alamy.

The surgeon is the medical officer who performs the operative procedure. As head of the surgical team, the surgeon is responsible for all medical actions and judgments.

The surgical assistant works closely with the surgeon in performing the operation. The number of assistants varies according to the complexity of the procedure. The assistant may be another surgeon, a nurse or other trained personnel. The assistant performs such duties as exposing the operative site, retracting nearby tissue, sponging and/or suctioning the wound, ligating bleeding vessels and suturing or helping suture the surgical wound.

The anaesthetist evaluates the person preoperatively, administers the anaesthesia and other required medications, transfuses blood or other blood products, infuses intravenous fluids, continuously monitors the person's physiological status, alerts the surgeon to developing problems and treats them as they arise, and supervises the person's recovery in the PACU.

The **circulating nurse** is a highly experienced nurse who coordinates and manages a wide range of activities before, during and after the surgical procedure. For example, the circulating nurse oversees the physical aspects of the operating room itself, including the equipment. The circulating nurse also assists with transferring and positioning the person, prepares the person's skin, ensures that no break in aseptic technique occurs, and records the surgical count of all accountable items, including sharps, sponges and instruments (ACORN, 2012). The handling of surgical instruments and associated accountable items must also comply with relevant state department of health policy directives. The circulating nurse assists all other team members, including the anaesthetist. Thorough documentation in the surgical area is essential, and the circulating nurse is responsible for documenting intraoperative nursing activities, medications, blood administration, placement of drains and catheters and length of the procedure. The circulating nurse also formulates a care plan based on physiological and psychosocial assessments of the person. Finally, the circulating nurse is at all times an advocate for the safety and well-being of the person having surgery.

The role of the **instrument nurse** primarily involves technical skills, manual dexterity and in-depth knowledge of the anatomical and mechanical aspects of a particular surgery. The instrument nurse handles sutures, instruments and other equipment immediately adjacent to the sterile field. The registered nurse ensures appropriate delegation and supervision of staff and participates in the surgical count (Australian College of Operating Room Nurses (ACORN), 2012).

The role of nurses in surgery continues to evolve to improve the care of people having surgery. Although not participating in the surgical procedure, PACU (recovery) nurses are part of the surgical team and are responsible for assessing, monitoring and implementing care of people during the recovery period from anaesthesia. In recent years, nurses have begun to specialise within the already specialised field of perioperative nursing and PACU nurses are now highly specialised care providers. Within the perioperative unit, specialty surgical teams are developing in response to the demands of increasingly complex technical surgeries. For example, a designated open heart surgical team may be responsible for all open cardiac cases and not be routinely involved with other procedures. The use of specialty

TRANSLATIONTO PRACTICE Evidence-based practice: assisting older adults to communicate postoperative pain

Nurses rely heavily on people's assessments of the pain they are experiencing. Pain is a subjective experience—a symptom rather than a sign. Rating of pain intensity by the person who has had surgery is the gold standard for knowing when to provide an intervention to decrease pain, and it is considered more accurate than nurses' evaluations of behavioural manifestations of pain. Older people who believe that healthcare providers know how to manage their pain are at risk of inadequately treated pain.

McDonald and colleagues (2005) used a program for coaching older adults about postoperative pain communication and management. In this study, older adults preparing for single-knee replacement surgery attended a preoperative joint replacement class where they learned about recovering from the surgery and pain management. Forty participants older than age 65 were randomly assigned either to the regular class, which included pain management information, or an intervention class, which included both information on pain management and skills to effectively communicate about pain.

Those in the communication skills class were taught to enhance pain communication with several strategies consistent with communication accommodation theory (CAT). CAT holds that people adjust their communication based on their own needs and the perceived behaviour of others. Skills to enhance communication include evaluating whether the other person (the nurse) has understood the message being given about pain, and is willing to include the person as a team member in controlling the pain. Language to describe pain intensity, location and sensation is modelled. People who have had surgery must report their pain because they are the experts about their own pain experience.

The groups were post-tested on postoperative days 1 and 2 and on days 1 and 7 after discharge, using the Brief Pain Inventory Short Form (BPI-SF). This instrument consists of 15 questions that measure pain severity, extent of interference with activities and pain relief in response to treatment. The group that received the communication skill information had significantly less pain interference with activity and greater pain relief with treatments on postoperative day 1 than the comparison group. On postoperative day 2, pain interference was similar for both groups and pain relief with treatment was greater for the comparison group. Pain severity scores were similar for both groups on postoperative days 1 and 2. There was no significant difference between the two groups in the post-discharge measures of any pain dimension.

IMPLICATIONS FOR NURSING

Findings from this study highlight the importance of coaching older adults to report their pain experience candidly, particularly in the immediate postoperative period. Establishing trust between the nurse and the person receiving care is critical to relieving pain. Coaching older people to describe their pain location, intensity and sensation gives them permission to communicate in a manner with which they may feel uncomfortable at first. Coaching is necessary to dispel myths about professional expertise and to allow personal control and independence so that they are willing to ask for pain medications. Exploring with older people their perception of pain, as well as the significance it has for recovery from illness, are necessary elements in providing adequate pain relief and restoring health.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. What physiological changes make pain management more difficult for an 80-year-old person following surgery than for a 30-year-old person?
2. An older person says: 'I deserved this pain, so I don't want to take anything to make it better.' What would be your response and why?
3. An Indigenous Australian man, aged 67, replies that 'Something doesn't feel right' when asked to rate his pain on a scale of 0 to 10. His pulse is increased and he is protective of his abdominal incision. What could you ask or do to accurately assess his pain?
4. If your grandfather or grandmother were having surgery tomorrow, what would you like them to be taught about pain management?
5. An independent, 85-year-old woman has a PCA pump for analgesia following major surgery. She continuously presses the pump button, but continues to complain of severe pain. What do you do now?
6. A female non-English-speaking Somalian refugee appears to be in pain following labiaplasty. What are the cultural issues associated with labiaplasty for these women? Would it be appropriate to ask her husband to act as a translator? How can you arrange an interpreter to assist this woman to communicate with you, so you can provide adequate pain relief for her?

surgical teams allows nurses to become highly skilled in a particular range of procedures and communications impacting on the care of people having surgery.

SURGICAL ATTIRE Strict dress codes are necessary to provide infection control within the operating theatre, to reduce cross-contamination between the surgery department and other hospital units or departments, and to promote the health and safety of people having surgery. Based on research and recommendations by hospital infection control authorities, guidelines for attire differ among surgical facilities. Following institutional guidelines, all personnel in the surgical department must be in proper surgical attire. The design and composition of the surgical attire minimises bacterial shedding, thus reducing wound contamination. The area in the surgical department is divided into *unrestricted*, *semi-restricted* and *restricted zones*. The unrestricted zones permit access by those in hospital uniforms or street clothes. These areas may also allow limited access for communicating with operating room personnel and handover of people having surgery.

CONSIDERATION FOR PRACTICE

Objects on the sterile drape are considered sterile. Remain a minimum of 30 cm away from draped tables and sterile fields to avoid contamination if you are not attired in sterile gown and gloves.

The semi-restricted zones require scrub attire, including a scrub suit, shoe covers and a cap or hood (see Figure 3.2). Hallways, work areas and storage areas are considered semi-restricted. Scrub attire is covered with sterile gowns in the restricted areas only if the person is 'scrubbed in' for the surgery. Only fabrics that are woven or disposable and will not harbour bacteria are allowed and all items of apparel must be covered by appropriate fabric. Nails that are chipped, varnished or artificial are not worn in surgery or anywhere the nurse will have direct contact with people at high risk. These nails are associated with glove tears and even after careful handwashing can harbour potential pathogens.

CONSIDERATION FOR PRACTICE

Staff in operating theatres must be knowledgeable about the nature of hazards within the perioperative environment. Hazards include a class A electrical area due to the quantity and type of equipment (including electrosurgical equipment) used in this environment; anaesthetic gases and other chemicals such as formaldehyde and fumes from bone cement and chemical sterilising agents, and surgical plume (smoke from electrosurgical and laser equipment); latex; sharps, including needle-stick injuries; laser and x-ray exposure; assembly and transport of theatre equipment; and biological hazards, including potential for exposure to blood and body fluids and possible sero-conversion for blood-borne viruses, including Creutzfeldt-Jakob disease (CJD).

Restricted zones are within operating rooms. Personnel wear masks, sterile gowns and gloves in addition to appropriate scrub attire if they are participating at the operating table. The outer sterile covering is changed between procedures or when it becomes soiled or wet.

FIGURE 3.2 ■ Surgical attire. Scrub attire includes scrub suit, shoe covers, and cap or hood to cover hair. Sterile attire includes scrub suit, shoe covers, and cap or hood, plus gown, gloves and mask

Source: ©Tyler Olson/Fotolia.com.

THE SURGICAL SCRUB The surgical scrub is performed to render hands and arms as clean as possible in preparation for a procedure. All personnel who participate directly in the procedure must perform a surgical scrub with an approved antimicrobial solution. Skin cannot be rendered sterile, but it can be considered 'surgically clean' following the scrub. The purposes of the surgical scrub are to:

- remove dirt, skin oils and transient microorganisms from hands and forearms
- increase safety for people having surgery by reducing microorganisms on surgical personnel
- leave an antimicrobial residue on the skin to inhibit growth of microbes for several hours.

Following the 3- to 5-minute surgical scrub (ACORN, 2012), hands and arms are dried with sterile towels.

Preparation of the person having surgery

Although much preparation has taken place prior to the transfer of the person to the surgical department, additional activities such as clipping of hair or shaving and positioning may be performed. The skin preparation, which usually includes cleansing the area with a prescribed antimicrobial agent, may have been performed either by the person or by nursing personnel before the transfer to the surgical department. Additional skin cleansing is performed in the surgical department to further decrease microorganisms on the skin and thereby reduce the possibility of wound infection.

The surgeon also may order preoperative hair removal to reduce surgical site infection; however, there is insufficient evidence to show that preoperative hair removal results in fewer surgical site infections than not removing the hair (JBI, 2007). The skin may be shaved or clipped in and around the proposed incision area (see Figure 3.3). Shaving may be completed preoperatively; however, it is best performed on the day of surgery (JBI, 2007) and is often performed within the operating theatre suite. The use of both clipping and depilatory creams results in fewer surgical site infections than shaving with a razor (JBI, 2007). The extent of hair removal for surgery varies. Generally, the area of hair removal is wider than the planned incision because of the possibility of unexpected extension of the incision. Disposable, sterile supplies are used, in accordance with aseptic techniques. However, the benefit of shaving the incisional site requires further research (JBI, 2007). Physical trauma to the shaved area can weaken the normal barrier that provides a defence against organisms, thus increasing the chance of wound infection. An altered body image also may result from the psychological trauma of a surgical shave, particularly if the shave involves the head or groin area.

Preparing the person for surgery also includes **positioning** them on the operating table. Figure 3.4 shows frequently used positions and describes corresponding surgical procedures and possible adverse effects. Positioning exposes the operative site in conjunction with access for anaesthesia administration. Careful and correct positioning is imperative to prevent injury to the person. Pressure, rubbing and/or shearing forces can cause injury to the tissue over bony prominences. If positioning causes normal joint range of motion to be exceeded, injury to muscles

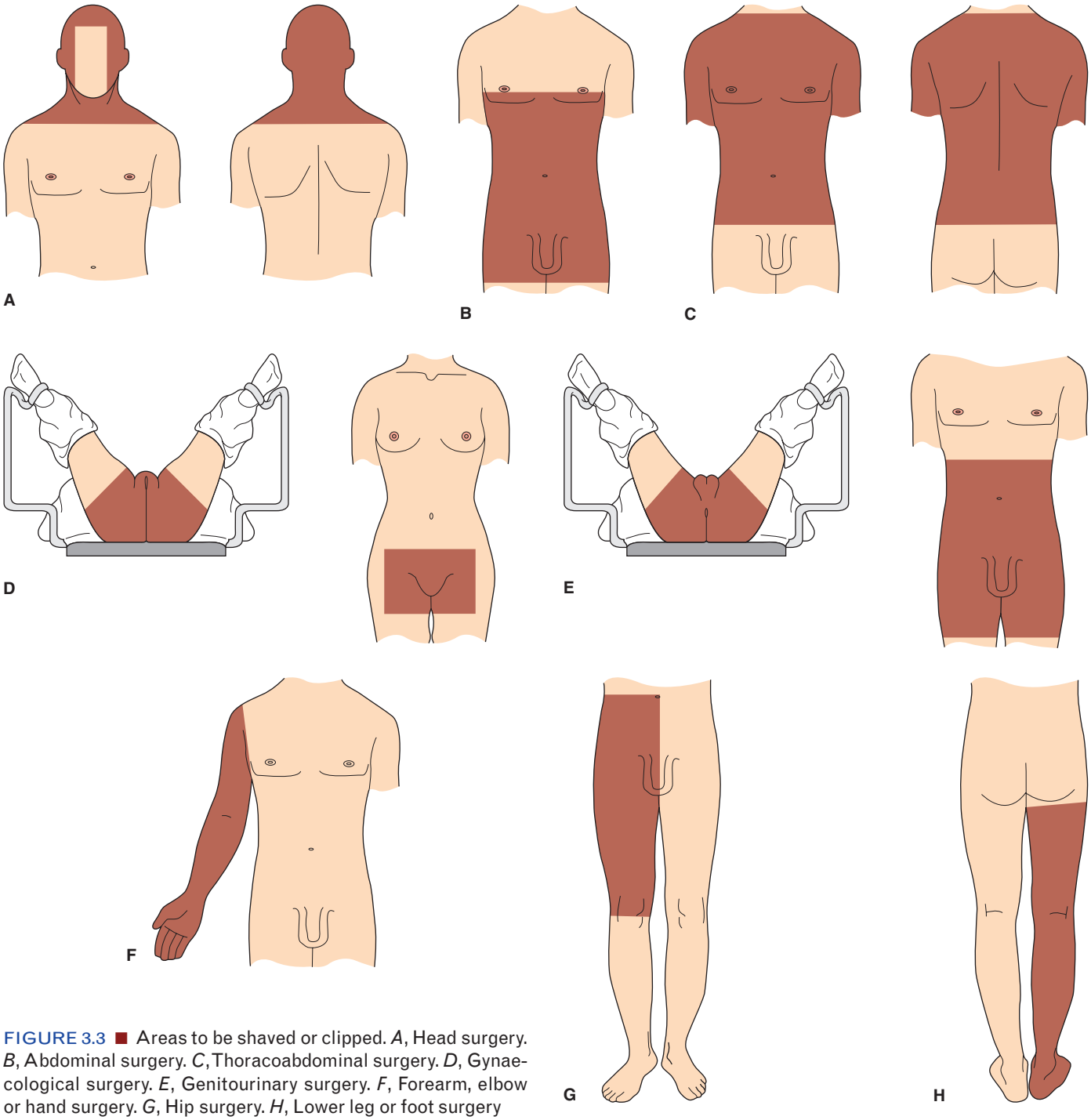


FIGURE 3.3 ■ Areas to be shaved or clipped. *A*, Head surgery. *B*, Abdominal surgery. *C*, Thoracoabdominal surgery. *D*, Gynaecological surgery. *E*, Genitourinary surgery. *F*, Forearm, elbow or hand surgery. *G*, Hip surgery. *H*, Lower leg or foot surgery

and joints can occur. Improper positioning also can lead to sensory and motor dysfunction, resulting in nerve damage. Pressure on peripheral blood vessels can decrease venous return to the heart and negatively affect the person's blood pressure. Additionally, oxygenation of the blood can be decreased if the person is not properly positioned to promote lung expansion.

As the anaesthetised person cannot respond to discomfort, it is the surgical team's responsibility to position them not only for the best surgical advantage but also for their safety and comfort. The circulating nurse refers to hospital policy, the surgeon's preference and the person's health history to ensure optimal positioning, and continuously assesses them during the intraoperative period.

INTRAOPERATIVE AWARENESS Prior to induction of anaesthesia, the circulating nurse establishes rapport with the person to assess their psychological status. After anaesthetic medications have been given, the person may appear oblivious to the surroundings; however, recall of intraoperative events has been reported. Although most people do not consciously

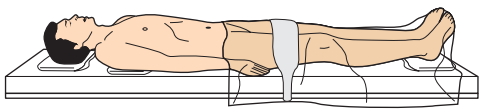
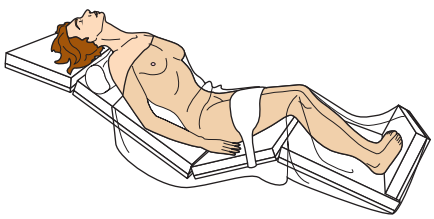
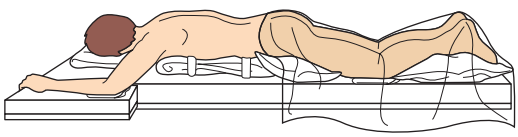
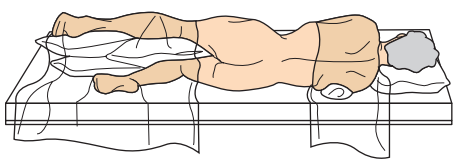
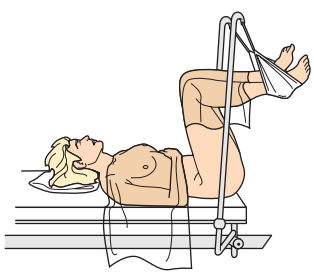
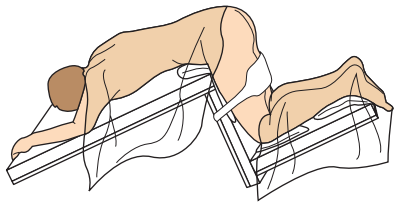
Position and use	Possible adverse effects and nursing interventions
(a) The *dorsal recumbent (*or *supine*) *position* is used for many abdominal surgeries (e.g. colostomy and herniorrhaphy), as well as for some thoracic surgeries (e.g. open heart surgery) and some surgeries on the extremities. 	This position may cause excessive pressure on posterior bony prominences, such as the back of the head, scapulae, sacrum and heels. Pad these areas with soft materials. To avoid compression of blood vessels and sluggish circulation, ensure that the knees are not flexed. Use trochanter rolls or other padding to avoid internal or external rotation of the hips and shoulders.
(b) The *semisitting position* is used for surgeries on the thyroid and neck areas. 	This position can lead to postural hypotension and venous pooling in the legs. It may promote skin breakdown on the buttocks. Sciatic nerve injury is possible. Assess for hypotension. Ensure that knees are not sharply flexed. Use soft padding to prevent nerve compression.
(c) The *prone position* is used for spinal fusions and removal of haemorrhoids. 	This position causes pressure on the face, knees, thighs, anterior ankles and toes. Pad bony prominences, and support the feet under the ankles. To promote optimum respiratory function, raise the person's chest and abdomen, and support with padding. Corneal abrasion could occur if the eyes are not closed or are insufficiently padded.
(d) The *lateral chest position* is used for some thoracic surgeries, as well as hip replacements. 	This position may cause excessive pressure on the bony prominences on the side on which the person is positioned. Ensure adequate padding and support, especially of the downside arm. The weight of the upper leg may cause peroneal nerve injury on the downside leg. Both legs must therefore be padded.
(e) The *lithotomy position* is used for gynaecological, perineal or rectal surgeries. 	This position causes an 18% decrease (from a standing position) in vital capacity of the lungs. Monitor respirations, and assess for hypoxia and dyspnoea. The lithotomy position can lead to joint damage, peroneal nerve damage and damage to peripheral blood vessels. To avoid injury, ensure adequate padding, and manipulate both legs into the stirrups simultaneously.
(f) The *jackknife position* is used for proctological surgeries, such as removal of haemorrhoids, and for some spinal surgeries. 	This position causes a 12% decrease (from a standing position) in vital capacity of the lungs. Monitor respirations, and assess for hypoxia and dyspnoea. In this position, the greatest pressure is felt at the bends in the table. Therefore, the person is supported with pads at the groin and knees, as well as at the ankles. Padding of the chest and knees helps prevent skin breakdown. Padding and proper positioning help prevent pressure on the ear, the neck, and the nerves of the upper arm.

FIGURE 3.4 ■ Common surgical positions

remember what happened or what was said, psychological trauma can result. Because loss of consciousness is gradual, conversations during surgery should be professional.

SPECIAL CONSIDERATIONS FOR THE OLDER ADULT Surgeries that last longer than two hours are associated with an increased risk of complications in older adults (Kable et al., 2008b). Because of cardiovascular and tissue changes that result from ageing, the older adult is more prone to hypotension, hypothermia and hypoxaemia resulting from anaesthesia and the cool temperature in the operating room.

Positioning may also cause complications in the older adult. Intraoperative positioning of arthritic joints can cause postoperative joint pain unrelated to the operative site. Extended duration of surgery may increase the chance of pressure areas. The older person is at increased risk of developing pressure areas because of decreased subcutaneous fat tissue and reduced peripheral circulation.

Finally, the older adult often has some degree of hearing and/or visual impairment. These impairments combined with a strange environment can make the operating room a frightening, disorienting place. By effectively communicating with the person, the nurse can provide support and reassurance to minimise these factors. To decrease confusion and assist in communication, hearing aids and glasses should be used when appropriate and possible.

Nutrition

Wound healing after surgery depends on adequate nutritional intake. During the immediate postoperative phase, dietary intake is often withheld until evidence of peristalsis is determined and the person can tolerate liquids without nausea and vomiting. While intravenous fluids maintain hydration and electrolyte balance, they do not provide nutrition. Some people believe that intravenous fluids are the same as intravenous 'feeding', but this is a myth. Unless balanced nutrition through gastrointestinal intake can be re-established within 3–4 days, parenteral hyperalimentation is critical for homeostasis and wound healing.

- Protein, kilojoules and vitamins are needed for wound healing and recovery from surgery.
- Low-fat, high-fibre diets are important for chronic cardiovascular fitness, but are contraindicated in the wound healing phase following surgery.
- Failure to use the gastrointestinal tract for more than 4–5 days allows the intestinal mucosa to atrophy, putting the person at risk of gastrointestinal tract haemorrhage and infection.

Fluid administered through peripheral veins must be isotonic, or only moderately hypertonic, to prevent sclerosing the small peripheral veins. Solutions of 10% dextrose are tolerable peripherally for a short time but do not provide adequate kilojoules for healing and maintenance. To provide adequate nutrition for people who have extended recovery periods without eating after surgery, central vein access must be established and parenteral nutrition administered. Education and counselling to support adequate nutritional intake should be ongoing throughout the preoperative and postoperative period.

Nursing care

The following section will discuss nursing care in each of the three phases of surgery. A case study at the end of the section follows one person through the postoperative experience, bringing this information together. Perioperative nursing diagnoses are provided in Table 3.5 to assist in identifying the needs of the person having surgery. This is not an exhaustive list, but it can serve as a guide in identifying possible nursing diagnoses.

TABLE 3.5 Examples of perioperative nursing diagnoses

PREOPERATIVE	INTRAOPERATIVE	POSTOPERATIVE
• Deficient knowledge	• Deficient knowledge	• Deficient knowledge
• Anxiety	• Anxiety	• Pain
• Fear	• Fear	• Ineffective breathing pattern
• Decisional conflict	• Ineffective airway clearance	• Ineffective airway clearance
• Ineffective coping	• Risk of aspiration	• Impaired skin integrity
• Ineffective sexuality patterns	• Decreased cardiac output	• Imbalanced nutrition
• Disturbed sleep pattern	• Hypothermia	• Ineffective sexuality patterns
• Disturbed thought processes	• Risk of infection	• Disturbed sleep pattern
• Interrupted family processes	• Disturbed thought processes	• Fatigue
• Spiritual distress	• Impaired gas exchange	• Urinary retention
	• Impaired urinary elimination	• Impaired urinary elimination
	• Deficient fluid volume	• Impaired adjustment
	• Excess fluid volume	• Disturbed body image
	• Impaired communication: verbal	• Impaired mobility: physical
		• Risk of activity intolerance
		• Risk of injury
		• Ineffective health maintenance
		• Deficient diversional activity
		• Social isolation
		• Spiritual distress

Preoperative nursing care

Each person's response to planned surgery varies greatly. When planning and implementing nursing care, consider individual psychological and physical differences, the type of surgery and the circumstances surrounding the need for surgery. A comprehensive nursing assessment is needed to determine the most appropriate care for each person undergoing surgery.

Before planning and implementing care for the person having surgery, gather assessment information by taking a nursing history and performing a physical examination. Use this information to establish baseline data, identify physical needs, determine teaching needs and psychological support for the person and their family, and prioritise nursing care. The type of surgical procedure directs the assessment and intervention planned by the nurse.

Surgery is a significant and stressful event. Regardless of the nature of the surgery (whether major or minor), the person and their family will be anxious. Some people seek care from a spiritual provider during this time. The degree of anxiety they will feel is not necessarily proportional to the magnitude of the surgical procedure. For example, a person scheduled to have a biopsy to rule out cancer, which is considered minor surgery, may be more anxious than a person undergoing gallbladder removal, which is considered major surgery.

The nurse's ability to listen actively to both verbal and non-verbal messages is imperative to establishing a trusting relationship with the person and their family. Therapeutic communication can help the person identify fears and concerns. The nurse can then plan nursing interventions and supportive care to reduce their anxiety level and assist them to cope successfully with the stressors encountered during the perioperative period.

Preoperative teaching

Teaching people is an essential nursing responsibility in the preoperative period. Education and emotional support have a positive effect on people's physical and psychological well-being, both before and after surgery. Previous research has determined that people who received preoperative education experienced less pain and anxiety, fewer complications, earlier discharge and increased satisfaction with their care, and returned to normal activities sooner. Positive outcomes may be attributed in part to the sense of control the person gains through the education they and their family receive.

Teaching should begin as soon as the person learns of the upcoming surgery. Teaching may begin in the surgeon's office or at the time of preadmission testing and assessment. Although education continues during postoperative care, most teaching is done before surgery because pain and the effects of anaesthesia can greatly diminish the person's ability to learn.

The amount of information desired varies for each individual. Therefore, the nurse should assess the person's need for and readiness to accept information. The teaching will be directed in part by the particular surgical procedure that is being performed and by the type of anaesthesia. The information in Box 3.2 is relevant to most people undergoing major surgery.

In addition to teaching the person and their family about measures that will decrease the risk of complications, provide other preoperative information to prepare them for surgery. This information should include the following:

- diagnostic tests—reasons and preparations
- arrival time for surgery
- preparations for surgery including: bowel preparation, skin preparation, indwelling catheter or bladder elimination, start of intravenous infusion, preoperative medication, handling of personal effects (rings, watch, money)
- sedative/hypnotic medication to be taken the night before surgery to promote rest and sleep
- education about whether to take medications on the morning of the surgery
- informed consent
- expected timetable for surgery and the recovery room
- method to inform family of progress throughout surgery
- transfer to the surgery department
- location of the surgical waiting room
- transfer to recovery room
- anticipated postoperative routine and devices or equipment (drains, tubes, equipment for IV infusions, oxygen or humidifying mask, dressings, splints, casts)
- plans for postoperative pain control
- if/when to commence fasting.

Be aware that dehydration, hypovolaemia and hypoglycaemia are recognised side effects of fasting. Thirst, worry and hunger are also reported by people having surgery and may be related to fasting. Fasting does not ensure that the stomach will be empty or that the gastric contents will be less acidic.

CONSIDERATION FOR PRACTICE

People about to undergo surgery may experience unnecessarily long preoperative fasts due to changes in surgery schedules and delays. How will this influence your management of people who are elderly, have diabetes mellitus or who may miss important medications as a result of these delays?

Preoperative preparation of people having surgery

A preoperative surgical checklist serves as a guide for preparing the person for surgery in most institutions. Complete the checklist before the person is transported to surgery. Nursing responsibilities on the day of surgery are as follows:

- Assist with bathing, grooming and changing into the operating room gown.
- Ensure that the person takes nothing by mouth. Provide additional teaching and reinforce prior teaching.
- Remove nail polish, lipstick and make-up to facilitate circulatory assessment during and after surgery.
- Ensure that identification and allergy bands are correct, legible and secure.
- Remove hair pins and jewellery; a wedding ring may be worn if it is taped to the finger.
- Complete skin or bowel preparation as ordered.
- Insert an indwelling catheter, intravenous line or nasogastric tube if ordered.
- Remove prostheses such as artificial eyes and contact lenses, and store them in a safe place. Note that

BOX 3.2 Preoperative teaching for people having surgery

Diaphragmatic breathing exercise

Diaphragmatic (abdominal) breathing exercises are taught to the person who is at risk of developing pulmonary complications, such as atelectasis or pneumonia. Risk factors for pulmonary complications include general anaesthesia, abdominal or thoracic surgery, history of smoking, chronic lung disease, obesity and advanced age.

In diaphragmatic breathing, the person inspires deeply while allowing the abdomen to expand outwards. On expiration, the abdomen contracts inwards as air from the lungs is expelled.

1. Explain to the person that the diaphragm is a muscle that makes up the floor of the thoracic cavity and assists in breathing. The purpose of diaphragmatic breathing is to promote lung expansion and ventilation and enhance blood oxygenation.
2. Position the person in a high or semi-Fowler's position (see figure below right).
3. Ask the person to place their hands lightly on their abdomen.
4. Instruct the person to breathe in deeply through their nose, allowing their chest and abdomen to expand.
5. Instruct the person to hold their breath for a count of five.
6. Tell the person to exhale completely through pursed (puckered) lips, allowing their chest and abdomen to deflate.
7. Instruct the person to repeat the exercise 5 times consecutively.

Encourage them to perform diaphragmatic breathing exercises every 1–2 waking hours, pre and postoperatively.

Coughing exercise

Coughing exercises are also taught to the person who is at risk of developing pulmonary complications. The purpose of coughing is to loosen, mobilise and remove pulmonary secretions. Splinting the incision decreases the physical and psychological discomfort associated with coughing.

1. Assist the person in following steps 1 to 4 for diaphragmatic breathing.
2. Ask them to splint the incision with hands or pillow (see figure below).
3. Tell them to take 3 deep breaths and then cough forcefully.
4. Instruct the person to repeat the exercise 5 times consecutively every 2 hours while awake, taking short rest periods between coughs, if necessary.
5. Provide fluids as appropriate following exercise.

Diaphragmatic breathing exercise

Splinting abdomen while coughing

(continued)

BOX 3.2 Preoperative teaching for people having surgery (continued)

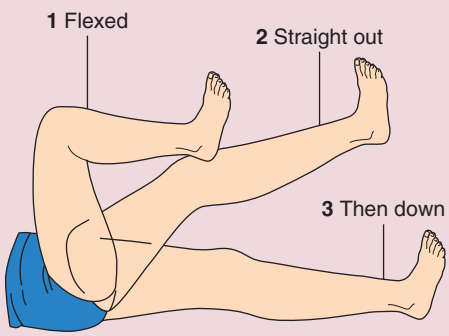
Leg, ankle and foot exercises

Leg exercises are taught to people who are at risk of developing thrombophlebitis (inflammation of a vein, which is associated with the formation of blood clots). Risk factors for developing thrombophlebitis include decreased mobility preoperatively and/or postoperatively, a history of difficulties with peripheral circulation, and cardiovascular, pelvic or lower extremity surgeries.

The purpose of leg exercises is to promote venous blood return from the extremities. As the leg muscles contract and relax, blood is pumped back to the heart, promoting cardiac output and reducing venous stasis. These exercises also maintain muscle tone and range of motion, which facilitate early ambulation.

Teach the person to perform the following exercises while lying in bed:

1. Muscle pumping exercise: contract and relax calf and thigh muscles at least 10 times consecutively.
2. Leg exercises:
   a. Bend the knee and raise it towards the chest (see figure below).
   b. Straighten out leg and hold for a few seconds before lowering the leg back to the bed.
   c. Repeat exercise 5 times consecutively prior to alternating to the other foot.
3. Ankle and foot exercises:
   a. Rotate both ankles by making complete circles, first to the right and then to the left (see figure below).
   b. Repeat 5 times and then relax.
   c. With feet together, point toes towards the head and then to the foot of the bed (see figure below).
   d. Repeat this pumping action 10 times and then relax.



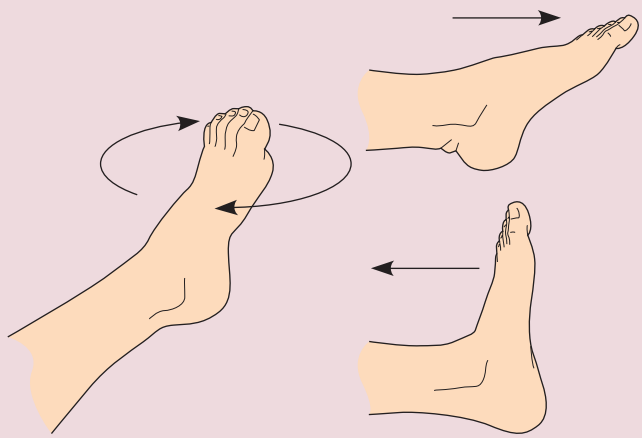
Leg exercises

Encourage the person to perform leg, ankle and foot exercises every 1–2 hours while awake, depending on their needs and ambulatory status.

Turning in bed

The person who is at risk of circulatory, respiratory or gastrointestinal dysfunction following surgery is taught to turn in bed. Although this may be a simple task prior to surgery, after surgery (particularly after abdominal surgery) they may find it a difficult procedure. To make the procedure more comfortable, they may need to splint the incision by using the hand placed on a small pillow or blanket. Additionally, they should be taught that analgesics can be given to ease postoperative discomfort involved with turning. Encourage them to turn every 2 hours while awake.

1. Tell them to grasp the side rail towards the direction to be turned, to rest the opposite foot on the mattress and to bend the knee.
2. Instruct them to roll over in one smooth motion by pulling on the side rail while pushing off with the bent knee.
3. Pillows may need to be positioned behind their back to help them maintain a side-lying position. Older people may also need padding over pressure points between the knees and ankles to decrease the chance of decubitus ulcer formation from pressure.



Ankle and foot exercises

the anaesthetist may request that dentures are worn to theatre and removed immediately prior to anaesthesia.

- Leave a hearing aid in place if the person cannot hear without it and notify the operating theatre nurse.
- Verify that the informed consent has been signed prior to administering preoperative medications.
- Weigh the person and record height and weight in the chart (for correct dosage of anaesthesia).
- Verify that all ordered diagnostic test reports are in the chart.
- Instruct the person to empty their bladder immediately before the preoperative medication is administered (unless an indwelling catheter is in place).
- Administer preoperative medication as scheduled (refer to 'Medications' earlier in the chapter and Table 3.4).
- Ensure the safety of the person once the medication has been given by placing them on bed rest with raised side rails and by placing the call switch within reach.
- Assess and record vital signs.
- Provide ongoing supportive care to the person and their family.
- Document all preoperative care in the appropriate location, such as the preoperative surgical checklist, the medication record and the narrative preoperative nursing notes.
- Verify with the surgical personnel the person's identity, and verify that all their information is documented appropriately.

- Assist with transfer from the bed to the theatre trolley if required.
- Prepare the person's room for postoperative care, including making the surgical bed and ensuring that the anticipated supplies and equipment are in the room.

Intraoperative nursing care

The intraoperative phase of surgery begins when the person enters the operating room and ends when they are transferred to the post anaesthesia care unit (PACU). Nursing care in this phase focuses on keeping them and the environment safe and providing physiological monitoring and psychological support.

Hazards associated with surgery can be minimised for people by use of a surgical safety checklist. The WHO (2009) has formed the World Alliance for Patient Safety that has developed a 'Safe Surgery Saves Lives' initiative. This program has produced a Surgical Safety Checklist that has been adopted in many countries including Australia.

A multisite study was conducted to evaluate the effectiveness of introducing this checklist and reported significant reductions in morbidity and mortality as a result of its implementation (Haynes et al., 2009). The checklist includes three phases: Sign in, Time out and Sign out, and requires all members of the surgical (and anaesthetic) team to contribute at critical junctures: before anaesthesia is administered, immediately before the incision and before the person is transported from the operating theatre. The implementation in Australia of a shorter version of this safe surgery protocol has been reported in a study by Healy, and compliance with this protocol is increasing (Healy, 2012).

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 5:
Patient Identification and Procedure Matching

'The intention of this standard is to correctly identify all patients whenever care is provided and correctly match patients to their intended treatment.' (ACSQHC, 2011, p. 40)

Implementing this standard is achieved by the establishment of explicit systems and processes that ensure routine use of at least three approved patient identifiers when providing care, therapy or services, and during transfer of care.

Caring for individuals having surgery will often require multiple occasions when routine checking of patient identification and procedure matching must be undertaken, and often involves several team members checking these details together. Some examples include during the process of documenting informed consent for the surgical procedure, during use of the preoperative surgical checklist in the ward before the person is transported to the operating theatre, during the transfer of care between ward staff and operating theatre staff, at the time when the person is checked into the anaesthetic room, during the use of the Surgical Safety Checklist in the operating theatre (in particular to check the correct operative site), during transfer to the PACU, and during transfer to the ward after surgery. In addition, this standard will apply to occasions of medication administration, infusion of intravenous fluids and blood products, and diagnostic testing. When these checks are conducted it is important that they are correctly documented. The documentation of these checking processes protects clinicians from making errors such as undertaking an incorrect procedure or at an incorrect surgical site, and reduces the risk of adverse events for people having surgery.

Source: © Australian Commission on Safety and Quality in Health Care.

Postoperative nursing care

Immediate postoperative care

Immediate postoperative care begins when the person has been transferred from the operating room to the PACU. The PACU nurse is part of the surgical team and monitors the person's vital signs and surgical site to determine the response to the surgical procedure and to detect significant changes. Assessing mental status and level of consciousness is another ongoing nursing responsibility, and the person may require repeated orientation to time, place and person. Emotional support also is essential, because the person is in a vulnerable and dependent position. Assessing and evaluating hydration status by monitoring intake and output is crucial to detecting cardiovascular or renal complications. In addition, the PACU nurse assesses the person's pain level. Careful administration of analgesics provides comfort without compounding the potential side effects from the anaesthesia.

Care when the person is stable

When awake and after being stabilised, the person is transferred to their hospital room. The PACU nurse communicates information about the person's condition and postoperative orders to the ward/unit nurse prior to their arrival. This prepares the ward/unit nurse for additional problems or needed equipment (see Box 3.3).

Immediate and continuing assessment is essential to detect and/or prevent complications. In documenting assessment findings, the nurse completes a flow record of the individual person's situation. Baseline data are obtained, documented and

BOX 3.3 Patient handover from the PACU nurse to the ward nurse

The quality of the patient handover from the PACU nurse to the ward nurse is a critical factor in patient safety. A systematic review by Wong, Yee and Turner (2008) identified that poor handover can result in discontinuity of care, the provision of inadequate or inaccurate care, adverse patient outcomes and legal claims of malpractice. A well-known definition of handover is 'the transfer of professional responsibility and accountability for some or all aspects of care for a patient, or group of patients, to another person or professional group on a temporary or permanent basis' (Australian Medical Association, 2007, p. 8). Because of the potential risk of patient harm, clinical handover is one of the Australian Commission on Quality and Safety in Healthcare's National Standards.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 6: Clinical Handover

Safe and effective clinical handover processes require standardised approaches that are fit for purpose and appropriate to the clinical context in which handover occurs. Attention to high-quality clinical handover enhances patient safety as critical information is more likely to be transferred and acted upon (ACSQHC, 2011).

Source: © Australian Commission on Safety and Quality in Health Care.

compared with preoperative data. A postoperative head-to-toe assessment includes, but may not be limited to, the following:

- general appearance
- vital signs
- level of consciousness
- emotional status
- skin colour and temperature
- discomfort/pain
- nausea/vomiting
- type of intravenous fluids and flow rate
- dressing site
- drainage on the dressing and/or bed linen
- urinary output (catheter or ability to urinate)
- ability to move all extremities.

The hospital policy or surgeon's orders dictate the frequency of follow-up assessments. After major surgery, the nurse generally assesses the person every 15 minutes during the first hour and, if they are stable, every 30 minutes for the next 2 hours, and then every hour during the subsequent 4 hours. Assessments are then carried out every 4 hours, subject to change according to the person's condition and the protocol for the particular surgical procedure. It is critical to inform the surgeon immediately if the assessment reveals any signs of impending shock or other life-threatening changes.

After carrying out the initial assessment and ensuring the person's safety by raising the side rails and placing the call switch within reach, the nurse reviews the surgeon's postoperative orders. These orders guide the nurse in the care of the postoperative person. For example, the orders specify activity level, diet, medications for pain and nausea, antibiotics, continuation of preoperative medications, frequency of vital sign assessments, administration of intravenous fluids, and laboratory tests such as haemoglobin and potassium level. In most institutions, orders written prior to surgery must be reordered following surgery because the person's condition is presumed to have changed.

Nursing care of common postoperative complications

Several factors place the person at risk of postoperative complications and adverse events (Kable, Gibberd & Spigelman, 2009). Nursing care before, during and after surgery is aimed at preventing and/or minimising the effects of these complications.

Preoperative care and teaching to decrease postoperative complications have been discussed previously. The following section addresses postoperative cardiovascular, respiratory and wound complications, and problems associated with elimination.

Cardiovascular complications

Common postoperative cardiovascular complications include shock, haemorrhage, deep venous thrombosis and pulmonary embolism.

SHOCK Shock is a life-threatening postoperative complication. It results from an insufficient blood flow to vital organs, an inability to use oxygen and nutrients, or the inability to rid tissues of waste material. Hypovolaemic shock, the most common type in the postoperative period, results from a decrease in circulating fluid volume. Decreased fluid volume develops with blood or plasma loss or, less commonly, from severe prolonged vomiting or diarrhoea. Symptoms vary according to the severity of the shock; the greater the loss of fluid volume, the more severe the symptoms. Chapter 10 provides a detailed discussion of nursing care of people with various types of shock.

HAEMORRHAGE Haemorrhage is an excessive loss of blood. A concealed haemorrhage occurs internally from a blood vessel that is no longer sutured or cauterised, or from a drainage tube that has eroded a blood vessel. An obvious haemorrhage occurs externally from a dislodged or ill-formed clot at the wound. Haemorrhage also may result from abnormalities in the blood's ability to clot; these abnormalities may result from a pathological condition, or they may be a side effect of medications.

Haemorrhage from a venous source oozes out quickly and is dark red, whereas an arterial haemorrhage is characterised by bright red spurts of blood pulsating with each heartbeat. Whether the haemorrhage is from a venous or an arterial source, hypovolaemic shock will occur if sufficient blood is lost from the circulation.

Common assessment findings with haemorrhage depend on the amount and rate of blood loss. Restlessness and anxiety are observed in the early stage of haemorrhage. Obvious bleeding will be present if the haemorrhage is external. The person will have symptoms characteristic of shock, such as hypotension and tachycardia (weak, thready pulse).

Care of the person who is haemorrhaging centres around stopping the bleeding and replenishing the circulating blood volume. Nursing care includes providing care for shock and one or more of the following:

- applying one or more sterile gauze pads and a firm pressure dressing to the area
- applying pressure with gloved hands (may be necessary for severe external bleeding)
- preparing the person and their family for emergency surgery (in severe situations when bleeding cannot be stopped).

DEEP VENOUS THROMBOSIS Deep venous thrombosis (DVT) is the formation of a thrombus (blood clot) in association with inflammation in deep veins. This complication most often occurs in the lower extremities of the person postoperatively. It may result from the combination of several factors, including trauma during surgery, pressure applied under the knees, sluggish blood flow during and after surgery and reduced mobility. People with a high risk of developing DVT include those who are over age 40 and who:

- have undergone orthopaedic surgery to lower extremities; urological, gynaecological or obstetric surgeries; or neurosurgery
- have a history of varicose veins
- have a history of thrombophlebitis or pulmonary emboli
- are obese
- have an infection
- have a malignancy.

Common assessment findings reveal pain or cramping in the involved calf or thigh. Redness, tenderness, warmth, discolouration of the skin and oedema (JBI, 2008) of the entire extremity may occur along with a slightly elevated temperature. The person may have a positive Homans' sign (pain in the calf on dorsiflexion of the affected foot). Suspected DVT can be confirmed by duplex Doppler scans; however, it is important to remember that many venous thrombi are asymptomatic.

Nursing care of the person with DVT focuses on preventing a portion of the clot from dislodging and becoming an embolus (travelling blood clot) circulating to the heart, brain or lungs; preventing other clots from forming; and supporting the person's own physiological mechanism for dissolving clots. Nursing care includes the following measures:

- Administer anticoagulants and analgesics as prescribed. (NSAIDs are not usually given in combination with anticoagulants, because doing so increases the anticoagulant effects.)
- Monitor pathology results for clotting times.
- Apply thigh-high graduated compression stockings or devices to stimulate venous return.
- Ensure that the affected area is not rubbed or massaged.
- Record bilateral calf or thigh circumferences every shift.
- Teach and support the person and their family about self-management.
- Assess colour and temperature of the involved extremity every shift.

PULMONARY EMBOLISM A pulmonary embolism is a dislodged blood clot or other substance that lodges in a pulmonary artery. For the postoperative person with DVT, the threat that a portion of the thrombus may dislodge from the vein wall and travel to the lung, heart or brain is a constant concern. Early detection of this potentially life-threatening complication depends on the nurse's astute, continuing assessment of the person postoperatively.

Common assessment findings of the person experiencing a pulmonary embolism include mild to moderate dyspnoea, chest pain, diaphoresis, anxiety, restlessness, rapid respirations and pulse, arrhythmias, cough and cyanosis. The severity of the symptoms is determined by the degree of pulmonary vascular blockage. Sudden death can occur if a major pulmonary artery becomes completely blocked.

Stabilising respiratory and cardiovascular functioning while preventing the formation of additional emboli is of utmost importance in the care of the person with a pulmonary embolism. Nursing care includes the following measures:

- Immediately notify the surgeon and/or anaesthetist.
- Frequently assess and record general condition and vital signs.
- Maintain the person on bed rest, and keep the head of the bed elevated.
- Provide oxygen as ordered and monitor pulse oximetry.
- Administer prescribed intravenous fluids to maintain fluid balance while preventing fluid overload.
- Administer prescribed anticoagulants.
- Maintain comfort by administering analgesics and sedatives. (Use caution to prevent respiratory depression.)
- Provide supportive measures for the person and their family.

Refer to Chapter 34 for a detailed discussion of pulmonary embolism.

Respiratory complications

Common postoperative respiratory complications include pneumonia and atelectasis.

PNEUMONIA Pneumonia is an inflammation of lung tissue. Inflammation is caused either by a microbial infection or by a foreign substance in the lung, which leads to an infection. Numerous factors may be involved in the development of pneumonia, including aspiration infection, retained pulmonary secretions, failure to cough deeply and impaired cough reflex, and decreased mobility.

Common assessment findings of the person with postoperative pneumonia are as follows:

- high fever
- rapid pulse and respirations
- chills (may be present initially)

- productive cough (may be present depending on the type of pneumonia)
- dyspnoea
- hypoxia
- chest pain
- pulmonary crackles and wheezes.

Treating the pulmonary infection, supporting the person's respiratory efforts, promoting lung expansion, and preventing the organisms' spread are the goals in the care of the person with pneumonia. Nursing care includes the following measures:

- Obtain sputum specimens for culture and sensitivity testing.
- Position the person with the head of the bed elevated.
- Encourage the person to turn, cough and perform deep-breathing exercises at least every 2 hours.
- Assist with incentive spirometry, intermittent positive pressure breathing (IPPB) and/or nebuliser treatments as ordered.
- Ambulate the person as their condition permits and as prescribed.
- Administer oxygen as ordered.
- Assess vital signs, breath sounds and general condition.
- Maintain hydration so that pulmonary secretions are easier for the person to expectorate.
- Administer antibiotics, expectorants, antipyretics and analgesics as ordered.
- Provide or assist with frequent oral hygiene.
- Prevent the spread of microorganisms by teaching proper disposal of tissues, covering mouth when coughing, and good handwashing technique.
- Provide supportive measures for the person and their family.

Chapter 34 provides a detailed discussion of pneumonia.

ATELECTASIS Atelectasis is an incomplete expansion or collapse of lung tissue resulting in inadequate ventilation and retention of pulmonary secretions. Common assessment findings include dyspnoea, hypoxia, diminished breath sounds over the affected area, anxiety, restlessness, crackles and cyanosis.

Promoting lung expansion and systemic oxygenation of tissues is a goal in the care of the person with atelectasis. Nursing care includes these tasks:

- Position the person with the head of bed elevated.
- Administer oxygen as prescribed.
- Encourage coughing, turning and deep breathing every 2 hours.
- Ambulate the person as their condition permits and as prescribed.
- Assist with incentive spirometry or other pulmonary exercises, such as inflating a balloon, as ordered.
- Administer analgesics as prescribed.
- Promote hydration.
- Provide supportive measures to the person and their family.

Wound complications

Discussion of the complications associated with surgical wounds follows an overview of wound healing, wound drainage and nursing care of wounds.

Wounds heal by either *primary*, *secondary* or *tertiary* intention (see Figure 3.5). Healing by primary intention takes place when the wound is uncomplicated and clean and has sustained little tissue loss. The edges of the incision are well approximated

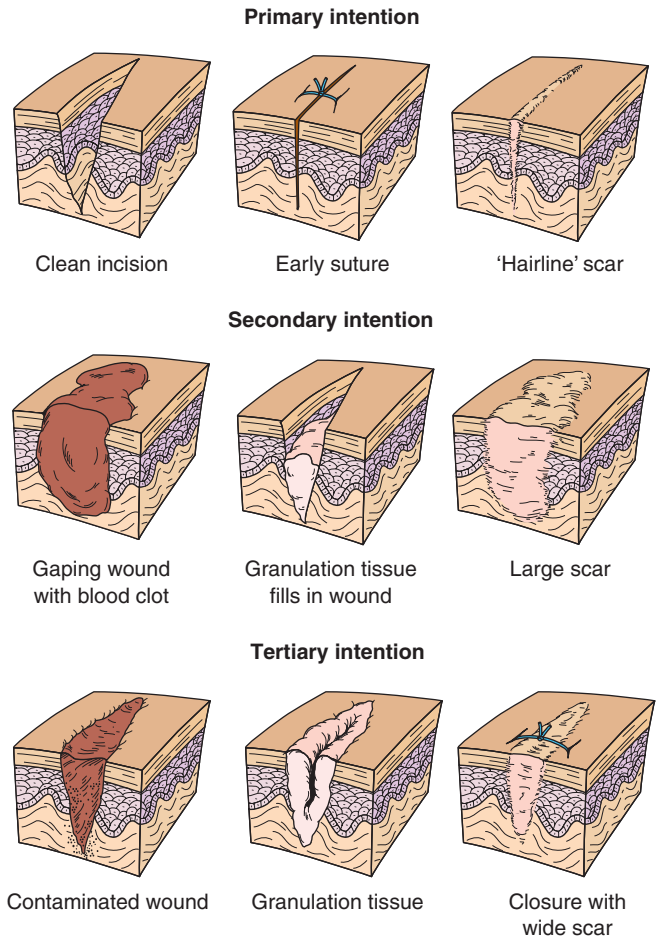


FIGURE 3.5 ■ Wound healing by primary, secondary and tertiary intention

(joined) with sutures, staples or glue for drain holes or superficial wounds. This type of surgical incision heals quickly and very little scarring is expected.

Secondary intention refers to the healing that occurs when the wound is large, gaping and irregular. Tissue loss prevents wound edges from approximating; therefore, granulation fills in the wound. This type of wound takes longer to heal, is more prone to infection, and develops more scar tissue.

If enough time passes before a wound is sutured, healing by tertiary intention occurs. Infection is more likely to take place. Because the wound edges are not approximated, tissue is regenerated by the granulation process. Closure of the wound results in a wide scar.

From the time the surgical incision is made until the wound is completely healed, all wounds progress through four stages of healing. However, healing time varies according to many factors, such as age, nutritional status, smoking, general health, and the type and location of the wound. Box 3.4 provides a summary of the stages of wound healing.

Wound drainage (exudate) results from the inflammatory process in the first two stages of wound healing. The drainage is from the rich blood supply that surrounds the wound tissue and is composed of escaped fluid and cells. The drainage is described as serous, sanguineous or purulent.

BOX 3.4 Stages of wound healing

- *Stage I: from surgery through to day 2.* Inflammatory process occurs to prepare the surrounding tissue for healing. Blood vessels constrict, and clotting occurs. Vasodilation and increased capillary permeability follows, bringing plasma, white blood cells and fibroplastin to the wound site. Epithelial cells begin to form and re-establish blood flow in the wound tissue. A mild temperature elevation is normal.
- *Stage II: day 3 through to day 14 following surgery.* Fewer white blood cells are present. Collagen tissue forms in the wound tissue. Granulation tissue, red with a rich blood supply, is established.
- *Stage III: day 15 to week 6 following surgery.* Collagen fibres continue to strengthen the wound. As the blood supply decreases, the scar tissue appears pink and somewhat raised.
- *Stage IV: several months to a year following surgery.* As the wound tissue constricts, the scar becomes flat, smaller and white.

- Serous drainage contains mostly the clear serous portion of the blood. The drainage appears clear or slightly yellow and is thin in consistency.
- Sanguineous drainage contains a combination of serum and red blood cells and has a thick, reddish appearance. This is the most common type of drainage from a non-complicated surgical wound.
- Purulent drainage is composed of white blood cells, tissue debris and bacteria. Purulent drainage is the result of infection and tends to be of a thicker consistency, with various colours specific to the type of organism. It also may have an unpleasant odour.

Box 3.5 describes and illustrates various types of wound drainage devices. These devices decrease pressure in the wound area by removing excess fluid, which promotes healing and decreases complications.

Nursing care of the person with a postoperative surgical wound focuses on prevention and monitoring for wound complications. The nurse assumes a leading role in supporting the wound healing process, providing emotional support to the person and teaching them wound care.

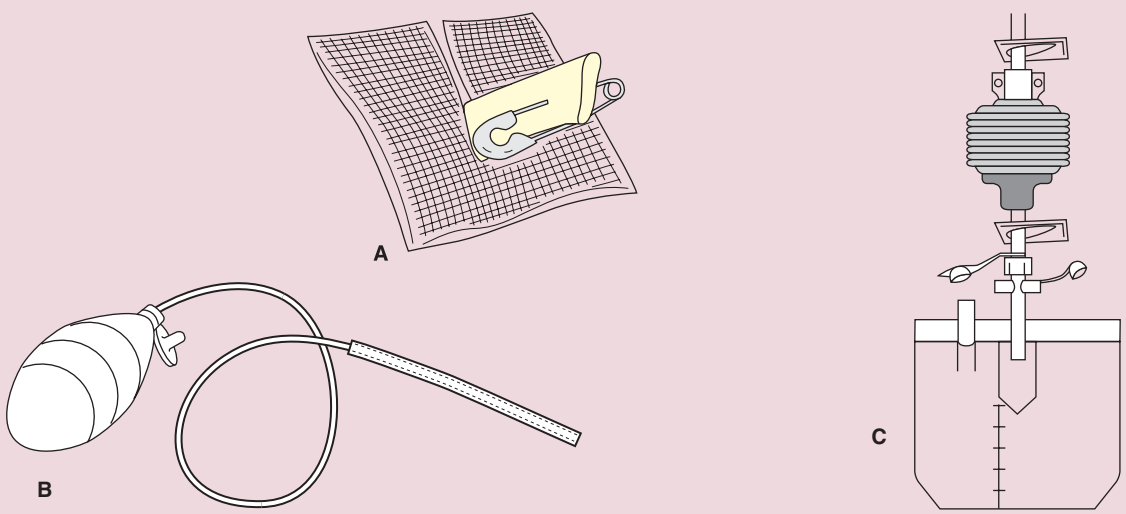
BOX 3.5 Wound drainage devices

A Penrose drain, used for passive wound drainage, promotes healing from the inside to the outside (see *Figure A* below). The use of the drain decreases the chance of abscess formation. The safety pin in the Penrose drain prevents the exposed end from slipping down into the wound, and facilitates x-ray detection if necessary. Wound care focuses on cleaning around the drain with a prescribed solution, such as sterile normal saline, and replacing the pre-cut gauze dressing as necessary to keep the surrounding skin dry and encourage further drainage. An absorbent dressing is placed over the drain and gauze (not shown).

Wound suction devices promote drainage of fluid from the incision site, decreasing pressure on healing tissues and reducing abscess formation. Shown are the Jackson-Pratt and Bellovac wound suction devices (see *Figures B* and *C* below).

The frequency with which the nurse empties the device depends on the time elapsed since surgery, type of surgery, amount of drainage and hospital policy. For example, immediately after surgery the nurse may empty the device every hour. With time, as drainage decreases, the device is emptied every 2 to 4 hours (per hospital policy). Amount, colour, consistency and odour of drainage are documented.

Usually, the drain is removed on the second to fourth day after surgery. Removal causes minor discomfort. The drain site is cleaned and a sterile dressing is applied.



Wound drainage devices. *A*, Penrose passive wound drainage device. *B*, Jackson-Pratt wound suction device. *C*, Bellovac wound suction device

Source: C © Wellspect Healthcare and Dentsply IH Pty Ltd (Australia).

Common assessment findings of an infected wound include pain; purulent, odorous discharge and redness; warmth; tenderness; and oedema around the edges of the incision. Additionally, the person may have a fever, chills and increased respiratory and pulse rates. Nursing care includes the following measures:

- Follow the Australian Wound Management Association's *Standards for Wound Management* (2011).
- Observe aseptic technique during dressing changes and handling of tubes and drains, including the 'Five Moments for Hand Hygiene' approach as recommended in Australia by Hand Hygiene Australia (ACSQHC, n.d.).
- Assess vital signs, especially temperature.
- Evaluate the characteristics of wound discharge (colour, odour and amount).
- Assess the condition of the incision (approximation of the edges, sutures, staples or drains).
- Clean, irrigate and pack the wound in the prescribed manner. Sterile normal saline is often prescribed.
- Maintain the person's hydration and nutritional status.
- Swab the wound for a microbial culture prior to beginning antibiotic therapy (if indicated).
- Administer antibiotics and antipyretics as prescribed.

Dehiscence is a separation in the layers of the incisional wound (see Figure 3.6A). Treatment depends on the extent of wound disruption. If the dehiscence is extensive, the incision must be resutured in surgery. **Evisceration** is the protrusion of body organs from a wound dehiscence (see Figure 3.6B). These serious complications may result from delayed wound healing or may occur immediately following surgery. They also may occur after forceful straining (coughing, sneezing or vomiting).

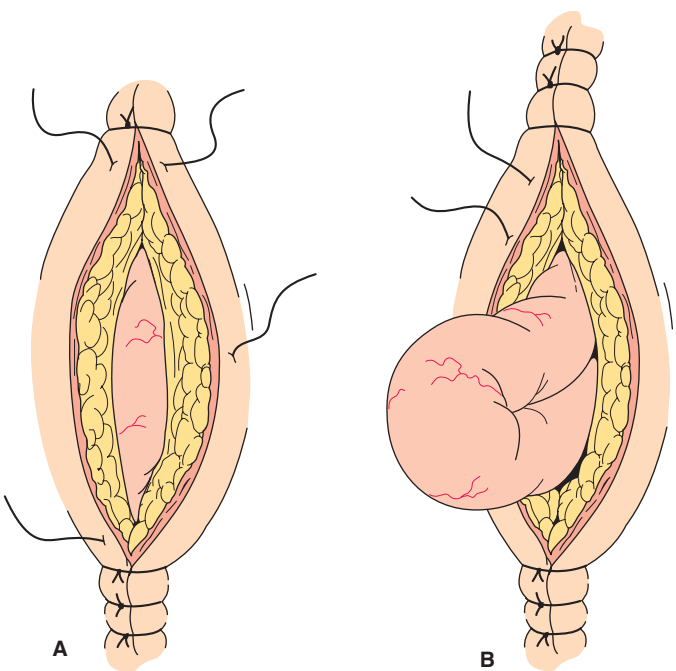


FIGURE 3.6 ■ Wound complications. *A*, Dehiscence is a disruption in the incision resulting in a separation of the layers of the wound. *B*, Evisceration is a protrusion of a body organ through a surgical incision

When dehiscence occurs, immediately cover the wound with a sterile dressing moistened with normal saline. Emergency surgery is performed to repair these conditions.

The nurse or medical officer may remove sutures or staples after the wound has healed sufficiently (usually 5 to 10 days after surgery). Removal is performed using aseptic technique. Additional support may be provided to the incision by applying strips of tape (or Steri-Strips) as directed by institutional policy or by the medical officer.

Complications associated with elimination

Common postoperative complications associated with elimination include urinary retention and altered bowel elimination. The inability to urinate with urinary retention may occur postoperatively as a result of the recumbent position, effects of anaesthesia and narcotics, inactivity, altered fluid balance, nervous tension or surgical manipulation in the pelvic area. Nursing care centres on promoting normal urinary elimination and includes the following measures:

- Assess for bladder distension if the person has not voided within 7 to 8 hours after surgery or is urinating small amounts frequently. Ultrasound assessment is often used for this purpose.
- Assess the amount of urine in the bladder with a portable ultrasound scanner. This non-invasive procedure provides information to prevent unnecessary catheterisation and decreases the potential for urinary tract infections and urethral trauma from repeated catheterisations.
- Monitor intake and output.
- Maintain intravenous infusion if fluids are prescribed.
- Increase daily oral fluid intake to 2500 to 3000 mL if the person's condition permits.
- Insert an intermittent or indwelling catheter if ordered.
- Promote normal urinary elimination by:
  a. Assisting and providing privacy when the person uses a bedpan.
  b. Helping the person to use the bedside commode or walk to the bathroom.
  c. Assisting males to stand to void.
  d. Pouring a measured amount of warm water over the perineal area (if urination occurs, subtract the amount of water from the total amount for an accurate output measurement).

Bowel elimination frequently is altered after abdominal or pelvic surgery and sometimes after other surgeries. Return to normal gastrointestinal function may be delayed by general anaesthesia, narcotic analgesia, decreased mobility or altered fluid and food intake during the perioperative period.

Nursing care centres on the return of normal bowel function and includes the following measures:

- Assess for the return of normal peristalsis:
  a. Auscultate bowel sounds every 8 hours while the person is awake.
  b. Assess the abdomen for distension. (A distended abdomen with absent or high-pitched bowel sounds may indicate paralytic ileus.)
  c. Determine whether the person is passing flatus.
  d. Monitor for passage of stool, including amount and consistency.

- Encourage early ambulation within prescribed limits.
- Facilitate a daily fluid intake of 2500 to 3000 mL (unless contraindicated).
- Provide privacy when the person is using the bedpan, bedside commode or bathroom.

If no bowel movement has occurred within 3 to 4 days after surgery, an aperient, suppository or an enema may be prescribed.

Special considerations for older adults

Physiological, cognitive and psychosocial changes associated with the ageing process place the older adult at increased risk of postoperative complications. These age-related changes with selected nursing interventions are summarised in Table 3.6. With an increasing population of older adults, particularly the very old, the nurse must be aware of these normal changes and modify nursing care accordingly in an effort to provide safe, supportive care.

Managing acute postoperative pain

Pain is expected after surgery (Layzell, 2008). It is neither realistic nor practical to eliminate postoperative pain completely. Nevertheless, the person having surgery should receive substantial relief from and control of this discomfort. Controlling postoperative pain not only promotes comfort but also facilitates coughing, turning, deep-breathing exercises, earlier ambulation and decreased length of hospitalisation, resulting in fewer postoperative complications and therefore reducing healthcare costs. Despite the apparent benefits and methods of effective pain control and improved understanding of pain physiology, many people do not receive adequate pain relief or control postoperatively.

TABLE 3.6 Nursing interventions for older people having surgery

SYSTEM	AGE-RELATED CHANGES	NURSING INTERVENTIONS
Body composition	Change in weight and fat distribution and hydration	Provide for warmth and frequent turning. Assess for dehydration and fluid imbalance. Provide comfort measures when NBM.
Integument	Diminished integrity secondary to loss of subcutaneous fat and decreased oil production and elasticity	Provide careful preoperative preparation, including hair removal, to avoid trauma. Position carefully to prevent pressure ulcers.
Sensory–perceptual	Decline in vision and hearing ability	Compensate for sensory deficits: speak low, not loud; minimise noise in environment; provide adequate room light; stay within the person's field of vision when speaking; encourage them to wear their hearing aid to the operating room.
Respiratory	Decreased efficiency of cough reflex and decreased aeration of lung fields. Reduced pulmonary functional reserve and lung function, with increased risk of infection and bronchospasm with airway obstruction	Maintain airway with positioning, provide oxygen as needed, maintain hydration and mobility. Teach and encourage coughing and diaphragmatic breathing exercises preoperatively and encourage smoking cessation. Constantly monitor lung sounds and respiratory status.
Cardiovascular	Less efficient, decreased cardiac reserve and adaptation to stress. Risk of arrhythmias and extreme blood pressure changes.	Monitor for hypotension and shock. Assess for thrombus formation, cardiac arrhythmias, peripheral pulses and oedema.
Gastrointestinal	Decline in gastric motility, risk of altered drug absorption, GORD, maldigestion and bowel elimination problems	Encourage intake of adequate fluids, nutritious meals, soft diet. Assist with feeding; monitor bowel function. Encourage mobility.
Genitourinary	Decreased renal function reserve, risk of nephrotoxic injury and adverse reactions to medications, risk of volume overload, dehydration and other electrolyte imbalances; loss of bladder control and urinary tract infections. Risk for falls.	Monitor fluid input and output and electrolyte levels. Assess for drug side effects. Assist with voiding as needed.
Musculoskeletal	Stiffness of joints; decrease in strength; brittleness of bones, intervertebral disc degeneration and joint erosion. Risk for falls.	Carefully position on operating table with bony prominences well padded to prevent pressure sores. Move carefully and gently. Provide effective pain management. Encourage mobilisation postoperatively. Prevent falls.
Cognition and nervous system	Decreased reaction time and reflexes and slow motor skills, deficits in balance and coordination, slowed cognitive processing; risk of delirium and altered mental status while in hospital. Risk for falls.	Provide ample time for making decisions. Implement safety measures. Talk to people respectfully as an adult. Orient frequently. Monitor for postoperative delirium. Provide effective pain management. Prevent falls.

Sources: C. M. Smith & V.T. Cotter (2012). *Age Related Changes in Health. Evidence-Based Geriatric Nursing Protocols for Best Practice* (4th ed.). New York: Springer Publishing Company; LLC. R. Griffiths, F. Beech, A. Brown, J. Dhesi, I. Foo, J. Goodall, W. Harrop-Griffiths, J. Jameson, N. Love, K. Pappenheim & S. White (2014). Guidelines: Peri-operative care of the elderly 2014. *Anaesthesia*, *69*(Suppl. 1), 81–98.

Managing acute postoperative pain is an important nursing role before, during and after surgery. Successful pain management involves the cooperative efforts of the person, anaesthetist and nurse (Layzell, 2008; McMain, 2010. The American Society of Anesthesiologists (2012) suggests six specific institutional policies for perioperative pain management. These include education and training for healthcare providers, monitoring of individual outcomes, documentation of monitoring activities, monitoring of outcomes at an institutional level, 24-hour availability of anaesthetists providing perioperative pain management and use of a dedicated Acute Pain Service. Preoperatively, the person should learn how much pain to anticipate and what methods are available to control pain. After discussing options with the person having surgery, healthcare providers must respect their personal preferences.

Postoperative medications were discussed earlier in the chapter. Various non-pharmacological approaches to pain management can also be used alone or in combination to control acute postoperative pain (ANZ College of Anaesthetists and Faculty of Pain Medicine, 2010). Music, distraction and imagery techniques and acupuncture can decrease mild pain and anxiety. Additional information on pain management techniques is found in Chapter 8.

The person's input and participation in assessing pain and pain relief is essential to a successful pain control regimen. For example, the person can rate the pain on a scale of 0 to 10 (where 0 signifies no pain and 10 signifies unbearable pain). Assess and document pain at scheduled intervals to determine the degree of pain control, to observe for drug side effects, and to assess the need for changes in the dosage and/or frequency of medication administration. When a range of dosage is ordered, carefully titrate opioid dosages based on individual assessments of need and response to therapy.

Community-based care

The postoperative phase does not end until the person has recovered completely from the surgical intervention. Thus the nurse plays a vital role as they near discharge. People who have surgery are usually discharged within a few days, and many complications can develop after discharge. As people prepare to recuperate at home, the nurse provides information and support to help them successfully meet self-care demands. All aspects of teaching should be accompanied by written guidelines, directions and information. This is particularly helpful when a large amount of unfamiliar, detailed information is presented. Because the hospital stay is often brief, an organised, coordinated effort to educate the person and their family should be made. Teaching needs vary, but the most common needs include:

- Wound care. Teaching is more effective if the nurse first demonstrates and explains the procedure for the person and their family or other caregiver. They should then participate in the care. To evaluate the effectiveness of the teaching, ask them to demonstrate the procedure in return. Ideally, teaching is carried out over several days, evaluated and periodically reinforced.
- Signs and symptoms of a wound infection. The person should be able to determine what is normal and what should be reported to their doctor.
- Method and frequency of taking own temperature.
- Limitations or restrictions that may be imposed on such activities as lifting, driving, bathing, sexual activity and other physical activities.
- Control of pain. If analgesics are prescribed, instruct the person in the dosage, frequency, purpose, common side effects and other side effects to report to the surgeon. Reinforce the use of relaxation, distraction, imagery or other pain control techniques that they have found useful in controlling their postoperative pain.
- Signs and symptoms of DVT and preventative strategies.
- Recommended strategies for rehabilitation and follow-up.

NURSING CARE PLAN A person having surgery

Martha Overbeck is a 74-year-old widow of German descent who lives alone in a retirement complex. She is active there, as well as in the Lutheran Church. She has been in good health and is independent, but she has become progressively less active as a result of arthritic pain and stiffness. Mrs Overbeck has degenerative joint changes that have particularly affected her right hip. On the recommendation of her surgeon and following a discussion with her friends, Mrs Overbeck has been admitted to the hospital for an elective right total hip arthroplasty. Her surgery has been scheduled for 8 am the following day.

Mrs Eva Jackson, a close friend and neighbour, accompanies Mrs Overbeck to the hospital. Mrs Overbeck explains that her friend will help in her home and assist her with the wound care and prescribed exercises.

ASSESSMENT

Gloria Nobis, RN, is assigned to Mrs Overbeck's care on return from surgery. Ms Nobis performs a complete physical assessment and determines that Mrs Overbeck is drowsy but oriented. Her skin is pale and slightly cool. Mrs Overbeck states that she is cold and requests additional covers. Ms Nobis places a warmed cotton blanket next to Mrs Overbeck's body, adds another blanket to her covers, and adjusts the room's thermostat to increase the room temperature. Mrs Overbeck states that she is in no pain and would like to sleep. She has even, unlaboured respirations and stable vital signs compared with preoperative readings.

Mrs Overbeck is NBM. An intravenous solution of dextrose and saline is infusing at 100 mL/h per infusion pump. No redness or oedema is noted at the infusion site. Ms Nobis notes that the antibiotic cephalothin is to be administered intravenously. Mrs Overbeck has a large secured dressing (Primapore) over her right upper lateral thigh and hip with no indications of ooze from the wound. Tubing protrudes from the distal end of the dressing and is attached to a passive suctioning device (Bellovac). Ms Nobis observes 50 mL of dark red drainage from the suctioning device and records the amount and characteristics on a wound assessment chart. Mrs Overbeck has an indwelling catheter in place with 250 mL of clear, light amber urine in the dependent gravity drainage bag.

NURSING CARE PLAN A person having surgery (continued)

When assessing Mrs Overbeck's lower extremities, Ms Nobis finds her feet slightly cool and pale with rapid capillary refill time bilaterally. Dorsalis pedis and posterior tibial pulses are strong and equal bilaterally. Ms Nobis notes slight pitting oedema in the right foot and ankle compared with the left extremity. She also notes sensation and ability to move both feet and toes, without numbness or tingling (paraesthesia).

Ms Nobis records these findings on the postoperative record. After ensuring that Mrs Overbeck is safely positioned and can reach her call switch, Ms Nobis gives Mrs Overbeck's friend a progress report. They then go into Mrs Overbeck's room.

DIAGNOSES

Ms Nobis makes the following postoperative nursing diagnoses for Mrs Overbeck.

- *Potential for infection:* right hip wound infection related to disruption of normal skin integrity by the surgical incision manifested by elevated temperature, redness, swelling, local pain, wound breakdown and wound exudate containing pus.
- *Injury* related to potential dislocation of right hip prosthesis secondary to hip arthroplasty manifested by acute severe pain, abnormal limb alignment and inability to mobilise.
- *Acute pain*, related to right hip incision and positioning of arthritic joints during surgery manifested by a high rating of pain on a pain scale, tachycardia, elevated blood pressure, dilated pupils, paleness, sweating, vasoconstriction, limited ability to move limb, restlessness, anxiety and shallow breathing.

PLANNING

Ms Nobis plans to use a clinical pathway to actively implement nursing strategies that will monitor the recovery of Mrs Overbeck, use evidence-based guidelines to prevent the development of potential postoperative complications, support her recovery and promote her comfort and progress during recovery. This will require using the clinical reasoning process to determine appropriate therapeutic responses, including the recognition of any indications of deterioration in physiological status. It will also include postoperative teaching to develop independence and motivate sustained self-management during hospitalisation and after discharge.

Expected outcomes

The expected outcomes established in the plan of care specify that Mrs Overbeck will:

- Regain skin integrity of the right hip incision without experiencing signs or symptoms of infection.
- Demonstrate (along with Mrs Jackson) proper aseptic technique while performing the dressing change.
- Verbalise signs and symptoms of infection to be reported to her surgeon.
- Describe measures to be taken to prevent dislocation of right hip prosthesis.
- Report control of pain at incision and in arthritic joints.
- Remain afebrile.

IMPLEMENTATION

Ms Nobis develops a care plan that includes the following interventions to assist Mrs Overbeck during her postoperative recovery.

- Provide prophylactic measures to minimise the risk of postoperative venous thromboembolism, including administration of low molecular weight heparin as ordered, early mobilisation, checking the correct application of graduated compression stockings, and/or sequential compression devices, assisting with exercises and monitoring Mrs Overbeck for potential manifestations of DVT.
- Use aseptic technique while changing dressing.
- Monitor temperature and pulse every 4 hours to assess for elevation.
- Assess wound every 8 hours for purulent drainage and odour. Assess edges of wound for approximation, oedema, redness or inflammation in excess of expected inflammatory response.
- Teach Mrs Overbeck and Mrs Jackson how to use aseptic technique while assessing the wound and performing the dressing change.
- Teach Mrs Overbeck and Mrs Jackson the signs and symptoms of infection and when to report findings to the surgeon.
- Review and discuss with Mrs Overbeck the written materials on hip arthroplasty.
- Convey empathetic understanding of Mrs Overbeck's incisional and arthritic joint pain.
- Provide analgesia every 4 hours (or as ordered) to maintain a therapeutic blood level.

EVALUATION

Throughout Mrs Overbeck's hospitalisation, Ms Nobis works with Mrs Overbeck and Mrs Jackson to ensure that Mrs Overbeck can care for herself after discharge from the hospital. Five days after her surgery, Mrs Overbeck is discharged with a well-approximated incision with no indications of an infection. Prior to discharge, Ms Nobis is confident that with Mrs Jackson's help, Mrs Overbeck can properly assess the incision. With minimal help, Mrs Overbeck is able to replace the dressing using aseptic technique. She can state the signs and symptoms of an infection, take her own oral temperature, and describe preventive measures to decrease the chances of dislocating her prosthetic hip. Because of her reduced mobility the past 5 days, Mrs Overbeck says she can tell the arthritis in her 'old bones' is 'acting up'. She reports a lessening of pain in her right hip following surgery and a pain that is different to her arthritic pain as experienced pre-surgery. Mrs Overbeck tells Ms Nobis she will be back the following winter to have her left hip replaced.

(continued)

NURSING CARE PLAN A person having surgery (continued)

CRITICAL THINKING IN THE NURSING PROCESS

1 Describe risk factors for Mrs Overbeck's safety; what changes in her home environment would you suggest to promote safety until she recovers more fully?
2 Why is Mrs Overbeck placed on the antibiotic cephalothin although she has no indications of an infection? What teaching would you do?
3 Mrs Overbeck's clotting time is slightly elevated as a result of an ordered anticoagulant. Why would this medication be ordered? Consider the person's age and the area of surgery.
4 Mrs Overbeck is 13.6 kg above her ideal weight and has osteoarthritis. Develop a care plan for the nursing diagnosis *Health Maintenance Problems* related to intake in excess of metabolic requirements and limited mobility.

REFLECTION ON THE NURSING PROCESS

1 Using the critical thinking process, identify what you have learned from this case study that you will apply to your clinical practice in the future.
2 Consider this learning in relation to the Nursing and Midwifery Board of Australia (NMBA) (2013) *National Competency Standards for the Registered Nurse (2006)*.
3 What teaching aids would be helpful to educate people recovering from hip arthroplasty surgery, during the postoperative period?

CHAPTER HIGHLIGHTS

- Surgery takes place in traditional and non-traditional settings with increasing use of minimally invasive procedures that expedite discharge, facilitate healing and increase satisfaction for people having surgery.
- Surgery is an invasive procedure, and legal guidelines must be followed to protect the person having surgery and the healthcare providers. The surgical team includes surgeons, anaesthetists, nurses and anaesthetic technicians; all are responsible for the safety of the person and the progression of the surgery.
- The focus on safety during surgery continues to increase, with attention directed to preventing wrong site/wrong patient operations occurring. Procedures are established to verify that the right person will have the correct surgery. A team approach to safety works best; each member of the team must feel accountable for the results of the surgery and entitled to share observations and concerns as the procedure progresses.
- Inpatients who have surgery have relatively short stays, which are best achieved by early ambulation, pain control and proper nutrition. Providing information for self-care is challenging with the shortened stays and rate of admissions and discharges. From the time of entry to the surgical setting, the person's discharge must be planned and prepared.
- Teaching people prior to and following surgery empowers them to achieve successful recovery, discharge and rehabilitation. Most of the care people receive during healing is provided either by themselves or a caregiver outside the healthcare environment. People having surgery and their families need to know appropriate assessments and interventions to monitor the healing process.
- Pain management is offered prior to, during and after surgery with methods designed to give the best therapeutic response. While acute pain related to the surgery occurs many people also experience persistent pain that affects their response to pain management therapies.
- Behaviours characteristic of older adults and ethnically diverse populations increase the need for individualised care. Assessment of physical and emotional status can be more difficult when people have hearing or visual impairments or when individuals speak and understand a foreign language. Surgery can be frightening to people and their families and they need reassurance and interventions to decrease pain, relieve anxiety and promote healing.
- Operating room and post anaesthesia nursing care are professional specialties that require unique orientation and education. These professionals make careful assessments of the risks each person faces and make plans to ensure safe, successful surgical outcomes. Special attention is focused on early recognition and treatment of postoperative complications associated with cardiopulmonary function, respiratory function, wound healing, elimination and pain.

CONCEPT CHECK

1 The nurse's primary responsibility relating to informed consent is:
1 defining the risks and benefits of the surgery
2 checking that the person's signature appears on the consent form and that the form is accurately completed
3 discussing alternative therapies with the person
4 advising the person and their family about what is needed for the diagnosis

2 Obtaining a preoperative blood pressure measurement serves the following purpose:
1 fulfils a legal requirement
2 provides information for the amount of anaesthetic required
3 prevents atelectasis
4 provides a baseline to compare with postoperative blood pressure levels

3 Non-steroidal anti-inflammatory drugs are given in the postoperative period to:
1 stimulate appetite
2 increase amnesia
3 potentiate analgesia
4 improve renal function

4 Discharge planning for a person following general surgery will include dietary management guidelines. Specifically, the person will eat a diet:
1 low in cholesterol, high in fat
2 high in protein, moderate in kilojoules
3 low in fat, high in fibre
4 without dairy products, but otherwise regular

5 In the immediate postoperative period for knee surgery, assessment distal to the site includes:
1 urinary pH
2 rebound tenderness
3 Chvostek's sign
4 neurovascular assessment

6 In the postoperative period, medications the person is prescribed prior to surgery must be:
1 continued after surgery
2 decreased by half for 36 hours
3 ordered anew prior to administration
4 withheld until evidence of anaesthesia is absent

7 The person with diabetes mellitus who is NBM prior to surgery:
1 has no risk of hyperglycaemia
2 should receive sliding-scale insulin prescriptions
3 will benefit from hypoglycaemia during anaesthesia
4 will fail to manifest signs of hypoglycaemia under anaesthesia

8 Acute pain management medications in the immediate postoperative period generally:
1 progress from NSAIDs to opioids
2 should include pre-emptive and multimodal analgesia
3 should be prn to promote control
4 induce a strong sedative effect to decrease the risk of nausea

9 Lengthy operative procedures can put the older person at risk of:
1 memory loss due to blood loss
2 hearing loss due to extended anaesthesia
3 weight loss due to lack of nutritional intake
4 pressure sores and joint pain from operative positioning

10 Hypothermia in the perioperative period:
1 decreases cardiac ischaemia
2 reduces the risk of wound infection
3 increases comfort and analgesia
4 requires interventions to prevent and relieve

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CHAPTER 4

NURSING CARE OF PEOPLE EXPERIENCING LOSS, GRIEF AND DEATH

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LEARNING OUTCOMES

- Differentiate the difference between loss, grief and mourning.
- Compare and contrast the theories of loss and grief.
- Explain factors affecting responses to loss.
- Describe person-centred care and the role of the nurse in end-of-life care.
- Discuss legal and ethical issues in end-of-life care.
- Describe palliative care philosophy and activities of a hospice.

CLINICAL COMPETENCIES

- Identify physiological changes in the dying person.
- Provide nursing interventions to promote a comfortable death.
- Provide person-centred care for individuals and families experiencing loss, grief or death.

KEY TERMS

LOSS, GRIEF AND MOURNING

Loss may be defined as an actual or potential situation in which a valued object, person, relationship, body part or emotion that was formerly present is lost or changed and can no longer be seen, felt, heard, known or experienced. A loss may be temporary or permanent, complete or partial, objectively verifiable or perceived, physical or symbolic. Only the person who experiences the loss can determine the meaning of the loss. Individuals experiencing health changes because of an eventually fatal condition may also feel a loss of trust in their body; this is particularly so for individuals diagnosed with cancer. Although the importance of loss varies for each person, people most commonly fear the losses listed in Box 4.1.

Loss always results in change. The stress associated with the loss may be the precipitating factor leading to physiological or psychological change in the person or family. The effective or ineffective resolution of feelings surrounding the loss determines the person's ability to deal with the resulting changes.

An **eventually fatal condition** is defined as:

> *any illness where it is expected that death will be a direct consequence of the specified illness. This definition is inclusive of illnesses of both a malignant and non-malignant nature. An eventually fatal condition might be expected to shorten an individual's life. This differs from chronic illness where, even though there may be significant impact on the person's abilities and quality of life, there is likely to be a less direct relationship between the illness and the person's death.* (Adapted from Palliative Care Australia, 2005, p. 11)

Grief is the emotional response to loss and its accompanying changes. Grief as a response to loss is a normal and an inevitable dimension of the human experience. The loss of a job, a role (e.g. the loss of the role of spouse, as occurs in divorce), a goal, body integrity, a loved one, or the impending loss of one's own life may trigger grief. Loss is also integral to death. Although death is a critical penultimate loss, losses that occur in any phase of the life cycle may produce grief responses as intensely painful as those observed in the death experience.

Grieving may be thought of as the internal process the person uses to work through the response to loss. **Mourning** describes the actions or expressions of the bereaved, including the symbols, clothing and ceremonies that make up the outward manifestations of grief. Both grieving and mourning are healthy responses to loss because they ultimately lead the person to invest energy in new relationships and to develop positive self-regard.

BOX 4.1 Types of losses

- Death
- Health
- Body part
- Social status
- Lifestyle
- Relationship (i.e. through breakup or divorce)
- Reproductive function
- Sexual function

A commonly used definition of **death** is an irreversible cessation of circulatory and respiratory functions or irreversible cessation of all functions of the entire brain, including the brainstem. With the current life support systems available, the most often used criterion for determining death is whole-brain death (permanent irreversible cessation of the functioning of all areas of the brain). The criteria for brain death are listed in Chapter 10.

Although death is an inevitable part of life, the acknowledgement of death as part of the process of an eventually fatal condition is often immensely difficult for the person and their loved ones. Death may be accidental (such as from trauma), intentional (suicide) or come at the end of a long and painful struggle with an eventually fatal condition such as cancer or motor neuron disease.

THEORIES OF LOSS AND GRIEF

Nurses often care for individuals exhibiting responses typical of various stages of the grieving process. Highly individual in quality and duration, the grief process may range from discomforting to debilitating, and it may last a day or a lifetime, depending on what the loss means to the person experiencing it. Although each person experiences loss in a different manner, knowledge of some of the major theories of loss and grief can give the nurse a framework for holistic, person-centred care of the individual and family anticipating or experiencing a loss. Table 4.1 summarises the theories discussed.

Freud: psychoanalytic theory

Freud (1917/1957) discussed grief and mourning as reactions to loss. Freud described the process of mourning as one in which the person gradually withdraws attachment from the lost object or person. He observed that with normal grieving,

TABLE 4.1 Summary of theories of loss

THEORIST	DYNAMICS
Freud (1917/1957)	Grief and mourning are reactions to loss. Grieving is the inner labour of mourning a loss. Inability to grieve a loss results in depression.
Bowlby (1973, 1980)	The successful grieving process initiated by a loss or separation during childhood ends with feelings of emancipation from the lost person or object.
Engel (1964)	After the person perceives and evaluates the loss, they adapt to it. Shock and disbelief, developing awareness and restitution occur during the first year following the loss; in the months following, the person puts the lost relationship into perspective.
Kübler-Ross (1969)	Five stages define the response to loss: denial, anger, bargaining, depression and acceptance. Stages are not necessarily sequential.
Caplan (1990)	Periods of psychological crisis are precipitated by hazardous circumstances; successful resolution of grief involves feelings of hope and engaging in activities of ordinary living.

this withdrawal of attachment is followed by a readiness to make new attachments. In comparing melancholia (prolonged gloominess, depression) with the 'normal' emotions of grief and its expression in mourning, Freud observed that the 'work of mourning' is a non-pathological condition that reaches a state of completion after a period of inner labour.

Bowlby: protest, despair and detachment

Bowlby (1973, 1980) believed that the grieving process initiated by a loss or separation from a loved object or person successfully ends when the grieving person experiences feelings of emancipation from the lost object or person. He divided the grieving process into three phases and identified behaviour characteristics of each phase.

1. *Protest*. The protest phase is marked by a lack of acceptance of the loss. All energy is directed towards protesting the loss. The person experiences feelings of anger towards self and others, and feelings of ambivalence towards the lost object or person. Crying and angry behaviour characterise this phase.
2. *Despair*. The person's behaviour becomes disorganised. Despair mounts as efforts to deny the loss compete with acceptance of permanent loss. Crying and sadness, coupled with a desire for the lost object or person to return, result in disorganised thoughts as the person recognises the reality of the loss.
3. *Detachment*. As the person realises the permanence of the loss and gradually relinquishes attachment to the lost object, a reinvestment of energy occurs. Both the positive and negative aspects of the relationship are remembered. Expressions of hopefulness and readiness to move forward are characteristic of this phase.

Engel: acute grief, restitution and long-term grief

Engel (1964) related the grief process to other methods of coping with stress: after the person perceives and evaluates the loss (the stressful event), they adapt to it. Engel's recognition of the impact of cognitive factors on the grieving process is an important contribution to an understanding of grieving.

Engel described three main stages in the grief process: an acute stage, a restitution stage and a long-term stage. The acute stage is initiated by shock and disbelief and is manifested by denial, which may help the person to cope with their overwhelming pain. As the shock and disbelief begin to fade, the loss becomes a reality, and pain, anguish, anger, guilt and blame surface. Culturally patterned behaviour, such as maintaining a stoic pose in public or weeping openly, characterise this phase.

The acute stage is followed by a stage of restitution, in which the mourning is institutionalised. Friends and family gather to support the grieving person through rituals dictated by the culture. The mourner continues to feel a painful void and is preoccupied with thoughts of the loss. The bereaved may join a support group or seek other social support for coping with the loss. This stage lasts until the point at which the bereaved begins to come to terms with the loss and their interest in people and activities is renewed.

Kübler-Ross: stages of coping with loss

Kübler-Ross's (1969) research on death and dying provided a framework for gaining insight about the stages of coping with an impending or actual loss. According to Kübler-Ross, not all people dealing with a loss go through these stages, and those who do may not experience the stages in the sequence described. In identifying the stages of death and dying, Kübler-Ross (1978) repeatedly stressed the danger of prematurely labelling a 'stage' and emphasised that her goal was to describe her observations of how people come to terms with situations of loss.

Some or all of the following reactions may occur during the grieving process and may reappear as the person experiences the loss:

- *Denial*. A person may react with shock and disbelief after receiving word of an actual or potential loss. After receiving a life-limiting diagnosis, notification of a death or other serious loss, people may make such statements as 'This can't be happening to me' or 'This can't be true'.
- *Anger*. In the anger stage, the person resists the loss. The anger is often directed towards family and healthcare providers in the biomedical model.
- *Bargaining*. The bargaining stage serves as an attempt to postpone the reality of the loss. The person makes a secret bargain with a higher power, expressing a willingness to do anything to postpone the loss or change the prognosis.
- *Depression*. The person enters a stage of depression as the full impact of the actual or perceived loss is realised. The person prepares for the impending loss by working through the struggle of separation. While grieving over 'what cannot be', the person may either talk freely about the loss or withdraw from others.
- *Acceptance*. The person begins to come to terms with the loss and resumes activities with an air of hopefulness for the future. Some dying people, but not all, reach a stage of acceptance in which they may appear to be almost devoid of emotion. The struggle is past, and the emotional pain is gone.

Caplan: stress and loss

Caplan's (1990) theory of stress and its relationship to loss is useful in understanding the grief process. He expanded the focus of the grief process to include not only bereavement but also other episodes of stress that people experience, such as the stress that can result from surgery or childbirth. Caplan described three factors that influence the person's ability to deal with a loss:

1. the psychic pain of the broken bond and the agony of coming to terms with the loss
2. living without the assets and guidance of the lost person or resource
3. the reduced cognitive and problem-solving effectiveness associated with the distressing emotional arousal.

He believed these factors might cause distress for a year or more following the loss.

Caplan described the process of building new attachments to replace those that have been lost. This process involves two elements: a feeling of hope and the assumption of regular activity as a form of participating in ordinary living.

FACTORS AFFECTING RESPONSES TO LOSS

A variety of factors affect a person's responses to loss. These include age, social support, families, cultural and spiritual practices, and rituals of mourning.

Age

The understanding of and reaction to loss is influenced by the age of the person experiencing the loss. In general, as people experience life transitions, their ability to understand and accept the losses associated with the transitions increases. From the age of three years, the development of the concept of death as a loss proceeds rapidly. However, from the toddler to older adolescents, individuals may experience difficulties expressing their emotional responses. Therefore, it is important for the nurse to encourage the family and carers to assist the child to understand that the source of their response may be their reaction to the grief or loss (Crowe, 2004).

Social support

Grieving is painful and lonely. One's social support system is important because of its potentially positive influence on the successful resolution of grief. Some losses may lead to social isolation, placing the individual at high risk of dysfunctional grief reactions. Characteristic factors that can interfere with successful grieving include the following:

- perceived inability to share the loss
- lack of social recognition of the loss
- ambivalent relationships prior to the loss
- extreme traumatic circumstances of the loss.

The relocation of work or home, a relationship breakdown or even the death of a pet can cause a person to feel extremely isolated, yet the person experiencing these types of losses does not ordinarily receive the same social support offered to the person mourning the death of a loved one. A woman having an abortion or giving up a child for adoption seldom receives the same social support as a mother who has lost a child at birth. It is especially important, therefore, for the nurse *not* to place a value on the individual's loss when assessing the need for support.

The painful nature of grief can cause the individual to withdraw from a previously established social support system, thereby increasing the feelings of loneliness caused by the loss. A recently widowed woman, for example, may refuse invitations involving married couples with whom she had socialised while her husband was alive. The individual's needs for social interaction, however, remain similar to those established before the loss.

Families

A well-functioning family usually rallies after the initial shock and disbelief and provides support for each other during all phases of the grieving process. After a loss, the functional family is able to shift roles, levels of responsibility and ways of communicating.

The family may have negative as well as positive effects. For example, the dying person may request that someone the family perceives as an outsider be near, and the family may respond with anger to the perceived 'intrusion'. Similarly, certain family members may express hurt feelings or anger if the individual is unresponsive to other family members. Well-meaning family members also may try to shield the individual from the pain of grieving. It is rare for the family and the dying person to experience anger, denial and acceptance in unison. While one member is in denial, another may be angry because 'not enough is being done'.

Cultural and spiritual practices

Spirituality is at the core of human existence, integrating and transcending the physical, emotional, intellectual and social dimensions (Reed, 1996). It involves a person's beliefs and values and cannot be viewed in isolation from their culture and background (Abbas & Panjwani, 2008). When confronted by a life-limiting illness, people often ask questions of themselves, and others, as to what their life has meant, why this illness has affected them and what will happen to them when they die. Spirituality can provide structure to a person's experience, giving a sense of coherence, and can assist with coping and providing feelings of wellbeing (Rumbold, 2003; Vivat, 2008).

Many individuals have beliefs about their life, including principles and values that they have lived by, their personal philosophy and the goals they have pursued in life. These beliefs may be questioned as the individual responds to actual or perceived losses experienced in their life. If unresolved, these concerns and perceptions can lead to spiritual or existential distress, causing a sense of hopelessness, anxiety and depression. When spiritual distress is resolved, people can die more peacefully.

For some people, spirituality is about religion, while for others it is about making sense of their existence. Spirituality is a way of being and often involves multiple practices/multiple forms of understanding experiences; it is, therefore, not necessarily the same for everyone. Abbs' fluid description captures the discursive context of spirituality:

> *. . . an intrinsic part of human existence—it can be experienced in particular moments of relationship, of heightened perception, and of high creativity; in states of trance, self transcendence and spontaneous enthusiasm, as in those unexpected moments of timelessness when the sheer miracle of consciousness quietly reveals itself. It can also be located in moments of acute personal crises and near breakdown; in moments of abandonment and anxiety.* (Abbs, 1995, p. 28)

Furthermore, Abbas and Panjwani (2008) point out that failure to provide spiritual care means failure to provide holistic care. The care of a dying person needs to consider more than attention to physical needs; it must also take into account a holistic approach involving a spiritual dimension of care (Abbas & Panjwani, 2008; Amoah, 2011; Penman, Oliver & Harrington, 2013; Puchalski, 2012). van der Riet (1999), in her research

FIGURE 4.1 ■ Nurses play an important role in caring for people who are experiencing an eventually fatal condition

involving the ways in which people undergoing treatment for cancer experienced massage and visualisation, found that people may not use the term 'spirituality'; however, they may use metaphors of spirituality to mean peace, centredness, comfort, healing, wellbeing, intimacy and reconnection with their bodies. Some of these metaphors included 'floating on a cloud' and 'I felt at one'. For them, spirituality involved an awareness of their bodies as new and no longer diseased with cancer.

Nurses play an important role in caring for people who are experiencing an eventually fatal condition (see Figure 4.1), as it is often difficult for them to maintain hope and a sense that their lives have had meaning. Spirituality is an important part of nursing practice. To assist with the person's spiritual needs, nurses can help them, through sensitive and empathetic responding, to accept the uncertainty that comes with their illness and possible death. Respect for the cultural and spiritual beliefs and practices of people and their families are paramount in caring for dying people. Context and environment are both an important part of a person's experience of spirituality (Billock, 2009). People who are religious need opportunities for prayer, devotions and religious rituals. Those without religious beliefs may find solace and achieve a sense of spirituality from meditation, guided imagery, music or art.

It is essential that the nurse has the ability to reflect on their own practice and actions that support the spirituality of the people they care for. For the nurse the concept of spirituality may be captured by the sense of seeing people at peace, or expressing that they feel balanced and connected again. Spirituality also encapsulates the sense of the nurse connecting with a person and having an understanding of their suffering and of being privileged to see their courage and strength. It could be sharing an intimate moment with a person.

Rituals of mourning

Through participation in ceremonies such as baptisms, confirmations and weddings, people joyously celebrate progression to a new stage of life and loss of a former way of being. The funeral ceremony may serve many purposes in meeting the needs of the bereaved, as people gather to share their loss. Through the funeral preparation and ceremony, people symbolically express triumph over death and deny the fear of death. Culture is the primary factor that dictates the rituals of mourning. It is important for health professionals to understand and respect the diversity that exists in our society in relation to grief and loss. The following section is intended to guide health professionals in developing an understanding of the diverse cultures within our society.

Death and dying in Indigenous Australian culture

There are no set rules that define how Indigenous Australians react to the loss of a loved one. However, many of the customs observed during funerals in the past are still evident in contemporary Indigenous society. The health professional should take into account the impact of colonisation when addressing the needs of Indigenous Australians. Christianity, assimilation and other values and beliefs adopted from colonial society have affected how Indigenous Australians now treat death and dying. Indigenous Australian culture is an evolving social phenomenon bringing change and diversity.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 2:
Partnering with Consumers

'The intention of this standard is to create a health service that is responsive to patient, carer and consumer input and needs.' (ACSQHC, 2011, p. 22)

Implementation of this standard is ensured through strong governance structures that reflect the importance of the cultural diversity of a population. Partnering with consumers supports the active participation of all groups in the evaluation and improvement of an organisation, especially those groups who may not always participate in providing feedback.

Patients who are dying and their grieving families are extremely vulnerable and need to feel safe and supported by compassionate and professional care. Culturally competent partnerships between health systems, services, patients, families, carers and consumers are important in ensuring a sense of dignity and safety in the vulnerable state of grief and loss. Systems that support consumers as partners in planning, designing and evaluating care will strengthen the delivery of culturally competent care, ensuring that with ongoing support patients and their families will adapt to loss.

Source: © Australian Commission on Safety and Quality in Health Care.

The following information reflects some of the history, customs and values of Indigenous Australians during times of grief, bereavement and funerals, and suggests avenues of understanding and approach for health professionals providing care for Indigenous Australians.

POST-TRAUMATIC STRESS SYNDROME Colonisation has had a major transgenerational impact on the psyche of many Indigenous Australians. The clashes between Indigenous Australians and Europeans included massacres, poisoning, dispossession, displacement and alienation, and there were many deaths and funerals (Reynolds, 1989). The socio-historical impact on contemporary Indigenous Australians should be considered when dealing with issues of death and dying.

GRIEF AND BEREAVEMENT FOR INDIGENOUS AUSTRALIANS The perspectives that were too often portrayed in the pages of Australian colonial ethnography texts ignored some of the fundamental issues that are critical to the provision of adequate support and comfort to Indigenous people experiencing grief and bereavement following the loss of loved ones. There are commonalities in the ways in which different Indigenous groups cope with death, but these were not explored in these texts and need to be addressed.

- *Grief counselling*. The family, extended family and friends gather around the deceased person as a collective group. This practice allows the grieving party space, connection and, most importantly, an opportunity to provide support to each other in a time of grief, loss and crisis. This support operates as a mechanism whereby the members of the group perform the roles of grief counsellor, bereavement counsellor and priest, therefore facilitating the healing process.
- *Barriers*. One of the major problems that confront Indigenous people and health professionals can sometimes be found in the rigid hospital policies that are embedded in administrative bureaucracy. In the event that a person dies in hospital, there may be policies that restrict visitation. For example, in some hospitals restrictions may apply that allow no more than two or three people to be with the deceased body at any one time and there may also be policies that only permit visitors between certain hours. These rules are impediments to the essence of healing in Indigenous Australian culture. They stop critical connections, respect and space, and deny the opportunity for the enactment of the healing process. Large numbers of Indigenous people faced with institutional barriers to this healing process may end up gathering inside and outside hospital areas where they can group together.
- *Space*. Indigenous people need sufficient space to gather together to facilitate the grieving and bereavement process where they can offer each other support, respect and counselling (see Figure 4.2).
- *Respect*. Health professionals should listen and understand the importance of this connection with regard to the grieving and healing process.
- *Time*. The grieving and bereavement process may take days or even weeks. It is important to respect this process and to provide space for this length of time. For example, in the early 20th century if an Indigenous woman lost and buried her child she grieved at the gravesite until new grass began shooting where her child was buried. During this period of around two weeks other members of the community brought food and water to support the grieving mother.
- *Cultural safety*. Health professionals create a culturally safe environment through awareness and consideration of the Indigenous person's cultural identity and acknowledgement of their difference within a Western-orientated healthcare system. The Nursing Council of New Zealand (2002, p. 9) defines unsafe cultural practice as any actions that 'diminish, demean or disempower the cultural identity of an individual'. Listening in a non-judgmental way to Indigenous people is a critical skill in acquiring cultural safety. As a nurse, culturally safe practice means you need to be able to recognise and understand the dynamics of cultural, personal and professional power (Richardson & Carryer, 2005).

Notifying the designated Indigenous liaison coordinator attached to the hospital about the death is critical, as this is often a link to the deceased person and their family.

The following quote provides some insight into the meaning of death, and the connection to the land, for the Indigenous person: 'Tree the same as me, When he get old he'll die. He'll be dead and burn. He'll leave his ashes behind. Tree becomes earth.' (Neidjie, 1985).

The important thing for health professionals to understand when dealing with death and dying is to treat people as they find them and to be mindful that there will be diversity and cultural variance which may be contrary to their own practices and beliefs. If Indigenous Australians are able to facilitate their own healing mechanisms and engage in mutual respect, connection and group support, then these practices will often minimise the need for ongoing support or bereavement counselling.

FIGURE 4.2 ■ Indigenous Australians need sufficient space to gather together to facilitate the grieving and bereavement process where they can offer each other support, respect and counselling

Source: © Dave Hunt/AAP Image.

Cultural diversity in the care of individuals with an eventually fatal condition

The 'Focus on Cultural Diversity' box below provides some insights into the practices of major cultural groups present in Australia. This is not a prescription for dealing with individuals within these cultures with an eventually fatal condition, but rather a guide. Rituals and practices within society are fluid and diverse and are affected by changes within the society, and community and family groups. Nurses should focus on the individual and provide respect and care in response to their particular needs.

END-OF-LIFE CARE

Providing quality care to individuals at the **end-of-life** (the final weeks of life when death is imminent) is a priority of the Australian Government. In 2000 the National Palliative Care Program was established and has since funded many initiatives that aim to improve both access to and quality of palliative care services across all Australian states and territories (Department of Health and Ageing, 2009). These initiatives are supported by Palliative Care Australia (2005), which has published *Standards for Providing Quality Palliative Care for All Australians*. Selected standards are:

- Care, decision making and care planning are each based on a respect for the uniqueness of the person, their caregiver/s and family. Their needs and wishes are acknowledged and guide decision making and care planning.
- Ongoing and comprehensive assessment and care planning are undertaken to meet the needs and wishes of the person, their caregiver/s and family.
- Care is coordinated to minimise the burden on the person, their caregiver/s and family.
- The primary caregiver/s is/are provided with information, support and guidance about their role according to their needs and wishes.
- The unique needs of dying people are considered, their comfort maximised and their dignity preserved.
- Staff and volunteers reflect on practice, and initiate and maintain effective self-care strategies (Palliative Care Australia, 2005).

FOCUS ON CULTURAL DIVERSITY Cultural aspects of eventually fatal condition care

Culture	Nursing interventions
Arabic	Arabic-speaking people tend to come from the Middle East and north-east Africa and follow the Muslim culture. In Muslim culture the body of the deceased is handled as little as possible, and preferably by those of the same sex and from a Muslim culture. The body of the deceased should be handed over as soon as possible to the Muslim community so washing and burial rituals can be undertaken.
Chinese	Ensure the head of the family is present when illness is discussed. The person may not want to discuss approaching death. Special amulets or cloths may be brought from home. Family members may prefer to bathe the body after death.
Greek	In the past, the family would control information related to an individual's eventually fatal condition and not provide the person with that information. However, this approach has changed over time, although the family is still significantly involved in care and decision making. Home is a significant place and is the preferred care environment. Nurses should offer to contact the family priest who may give communion, rather than 'last rites', as this has a more positive connotation.
Italian	The family is usually involved when an eventually fatal condition is discussed and may wish to shelter the person from discussions of diagnosis and prognosis. Home is the preferred place for care. Prayer may be an important part of end-of-life care and it is important to offer attendance by a priest.
Korean	Ensure the eldest son in the family is involved in discussions concerning an individual's eventual fatal condition. The hospital tends to be the preferred place to die, especially if there are young children living at home. Many Koreans follow a Buddhist or Confucianist doctrine of religion. The family may have sought help from a herbal doctor called a '*Hanui*'. The body of the deceased is usually left overnight.
Polish	Family is a significant part of healthcare. However, not all individuals have family present in Australia. Home is the preferred place for care. However, hospital and hospice environments are accepted places for care. Many are involved in the Catholic Church, and respect for the church and a belief in God is a significant aspect of life.
Vietnamese	Ensure the head of the family is involved in discussions concerning an eventually fatal condition with the person. You may find that the entire family will make end-of-life decisions, often with assistance from spiritual leaders such as a priest or monk. Individuals often prefer to die at home. The family should have extra time with the body, and vocal expressions of grief are not uncommon. After the person has died, the family might request to open all the windows and outside doors of the room to allow the person's spirit to leave their body.

Source: Adapted from *Multicultural palliative care guidelines* by A. Taylor & M. Box (1999). Canberra: Department of Health and Ageing.

Nursing considerations for end-of-life care

Nurses care for the dying person in many environments, including general and acute hospital settings such as intensive care and emergency departments, residential aged care facilities, hospices and the home. (See 'Translation to practice: Evidence-based practice for end-of-life care' for research on intensive care nurses' experiences with end-of-life care.) Regardless of the setting, the individual's wishes about death should be respected.

Legal and ethical issues

Decision making and consent are issues that are often debated strongly when considering end-of-life care. The legal and ethical issues surrounding advance directives, enduring power of attorney, do-not-resuscitate (DNR) directives, euthanasia and quality of life are especially important to nurses in upholding each person's specific care requests. Types of advance directives are shown in Box 4.2.

Settings for end-of-life care

Settings and services for the provision of end-of-life care range from residential aged care facilities, hospitals and hospices to the person's own home. **Palliative care** is a term used within Australia to describe a philosophy of care provided for individuals at the end-of-life or with an eventually fatal condition. In the literature, the term '**hospice care**' may be used to refer to the philosophy of care; however, in Australia, the term 'hospice' more often refers to a setting where palliative care is provided. The philosophy of palliative care is outlined in this section, followed by a brief description of the settings in which this philosophy may be integrated into care.

Philosophy of palliative care

Palliative care is a philosophy of care, rather than a program of care. It is holistic, comprehensive and coordinated care for individuals with an eventually fatal condition, provided where possible in a setting of the person's choice (Department of Health and Ageing, 2000). Palliative care reaffirms the right of every person and family to fully participate in the final stages of life. Palliative care is provided by nurses, doctors, allied health professionals and volunteers. It is based on a philosophy of dying being a normal part of living, and on ensuring the individual is provided with comfort and dignity, encompassing physical, psychological, social, emotional and spiritual aspects of the dying experience (Department of Health and Ageing, 2000). The philosophy of palliative care may be provided in any setting, including those outlined below.

HOME/RESIDENTIAL AGED CARE FACILITY/ COMMUNITY LIVING An individual often prefers to die at their permanent living environment, which may include a private home or residential aged care facility. Community or hospital/hospice outreach program-based nurses working within the palliative care philosophy are usually the main care providers for individuals in this setting. It is essential that care provided to individuals in this setting includes ongoing comprehensive assessment involving the person, the family and the support services that are available, including general medical and palliative care specialist teams.

BOX 4.2 Types of advance directives

Advance directives, sometimes referred to as 'living wills', are legal documents that allow a person to plan healthcare or provide insights into treatment preferences in the event of incapacity. Advance directives may be formally written legal documents, including living wills or enduring powers of attorney, or informal orally communicated requests expressed to loved ones, carers or health professionals. Advocating for the person's treatment preferences is an aspect of care that many nurses will face, and the nurse must always ensure that the person is aware of the preferences stated in their directives and that they remain current. The legislation across Australia concerning advance directives varies, so it is essential that the nurse is aware of the approach taken by the state or territory in which they are practising.

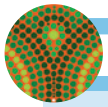
Advanced care planning has evolved in the face of limitations associated with advance directives. An advanced care plan should support the advance directive, in that it further develops documented communications of the person's preferences as to future care and/or the withdrawal of care and treatment.

An **enduring power of attorney** is a document that can delegate the authority to make health, financial and/or legal decisions on a person's behalf. It must be in writing and must state that the designated person is authorised to make healthcare decisions.

A **do-not-resuscitate (DNR) directive** or *not for resuscitation (NFR) order* is written by the doctor for the individual for whom it is believed that cardiopulmonary resuscitation (CPR) would be of no benefit. This order is usually based on the wishes of the individual and family that no CPR be performed for respiratory or cardiac arrest. However, this directive does not extend to all treatment and care, and the nurse must ensure that other treatments should be offered and discussed with the person if applicable. The nurse must ensure that current, clear verbal and written communication of the directive is established within the multidisciplinary team, the person and the family.

Euthanasia (from the Greek for painless, easy, gentle or good death) is now a commonly used term to signify a killing prompted by humanitarian motives. There are many arguments and ethical debates for and against euthanasia, and nurses have often found themselves at the centre of the debate. As a result, nurses have pushed for the development of appropriate guidelines and procedures for DNR directives. Within Australia it is illegal to assist in an individual's suicide or to perform euthanasia.

Source: Based on *Bioethics: A nursing perspective (5th ed.)* by M. Johnstone (2011). Sydney: Elsevier.



TRANSLATION TO PRACTICE Evidence-based practice for end-of-life care

End-of-life care is emerging as a major issue in Australia. In a submission to the National Health and Hospitals Reform Commission, Palliative Care Australia has rightly pointed out that end-of-life care is everyone's affair. Further to its submission, many people in acute care are not receiving adequate end-of-life care. Good palliative care at the end of life should be consistent across all settings and for all people with a life-limiting illness. Sadly this is not the case.

Although many individuals die in hospital, little research has been done about deaths in acute care settings, such as intensive care (ICU), coronary care and critical care units. Although people are admitted to ICUs to gain access to advanced life-saving technologies, it is a reality that some die in these settings (Kirchhoff et al., 2000). Most nurses working in ICU acknowledge that it is not the most desirable setting for providing end-of-life care for an individual and their family (Kirchhoff et al., 2000; Sorensen & Iedema, 2007). However, research is beginning to address ways that nurses and the nursing profession can move forward to improve end-of-life care in ICUs (Kirchhoff et al., 2000; Sorensen & Iedema, 2007; Truog et al., 2008).

IMPLICATIONS FOR NURSING

Kirchhoff et al. (2000) advocate that, regardless of the setting for end-of-life care, nurses need to ensure that individuals are as free from pain as possible, and that their comfort and dignity are maintained. In addition, family members should be given time to begin to accept the dying process. This may be facilitated by having the family member involved in care and providing the time and space for family rituals and saying goodbye. Sorensen and Iedema (2007) focus on the importance of patient advocacy in end-of-life care in ICU and encourage nurses working in ICU to develop end-of-life care knowledge and formal assessments and to engage in open discussions with a multidisciplinary approach to advocate for the most appropriate care for the individual.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. What environmental differences are present in an ICU that might make quality end-of-life care more difficult for nurses?
2. The family of a person in the ICU says to you, 'My son is going to die, isn't he?' What would you need to know before responding? How would you respond?
3. A person in the ICU who previously had been improving suddenly dies. Staff are saddened and several are in tears. A more experienced nurse says, 'Oh, you just have to go on. There's nothing else to do.' Do you agree? Why or why not?

Sources: Data from Intensive care nurses' experiences with end-of-life care by K. Kirchhoff, V. Spuhler, L. Walker et al. (2000). *American Journal of Critical Care*, *9*(1), 35–42; R. Sorensen & R. Iedema (2007). Advocacy at end-of-life. Research design: An ethnographic study of an ICU. *International Journal of Nursing Studies*, *44,* 1343–1353; R. D. Truog, M. L. Campbell, J. R. Curtis, C. E. Haas, J. M. Luce, G. D. Rubenfeld, C. H. Rushton & D. C. Kaufman (2008). Recommendations for end-of-life care in the intensive care unit: A consensus statement by the American College of Critical Care Medicine. *Critical Care Med*, *36*(3), 953–96; Palliative Care Australia (2012). Inquiry into Palliative Care in Australia—Submission to the Australian Senate Standing Committee on Community Affairs. Deakin West, ACT: Author.

HOSPICE/INPATIENT PALLIATIVE CARE BED The hospice setting is not restricted to the end of life, and is often used earlier in the disease experience. Individuals may seek admission to a hospice for assessment, symptom management, respite or care at the end of life. The hospice environment may be separate from or connected to a hospital setting and is focused on the relief of physical, emotional and spiritual distress for individuals who have an eventually fatal condition. Its goal is to prevent and relieve suffering by early assessment and treatment of pain and other physical, psychosocial and spiritual needs to improve the person's quality of life.

The team providing care to individuals in this setting has a well-defined palliative care philosophy and usually includes doctors, nurses, allied health professionals (including social workers, occupational therapists and dietitians), pastoral care, community liaison nurses and volunteers. If it is the individual's wish to return to their home environment, this is supported by the team with continued assessment and involvement of a community outreach hospice/palliative care team. The expected outcomes of care are directed by interventions to manage current manifestations of the illness and to prevent new manifestations from occurring.

ACUTE HOSPITAL SETTING Individuals with an eventually fatal condition who are receiving palliative care are often nursed within acute care settings. It is a significant factor of the National Palliative Care Strategy (Department of Health and Ageing, 2010) that nurses acknowledge that individuals with an eventually fatal condition often move between hospice/palliative environments and the acute care setting. In recognition of this, nurses need to develop further knowledge of the palliative care philosophy in order to facilitate and support person's individual care needs.

Symptom management in the dying person

Death is a highly individualised process and may occur rapidly or slowly. Physiological changes are a part of the dying process. Although each person responds differently, certain manifestations are common in the dying process, regardless of the trauma or disease process that is causing death. The discussion that follows includes treatments and related nursing care.

Pain

Pain is a common problem for individuals at the end of life. It is acknowledged that about two-thirds of people with cancer will experience pain that requires intervention (Quigley, 2005). Pain is what individuals often say they fear the most. Pain is a subjective experience influenced by the person's

emotions, previous experiences with pain, and family and culture. Unfortunately, pain may be under-treated at the end of life because doctors and nurses fear they will cause addiction, respiratory depression or sedation (Quigley, 2005). There is also the concern that the use of adequate pain relief may be seen as contributing to euthanasia rather than a person's comfort. However, nearly all pain at the end of life can be managed without causing addiction or respiratory depression. It is of utmost importance to keep the individual comfortable through general comfort measures (see Box 4.3) and by administering prescribed medications for pain, neuropathic pain (which is rarely relieved by opioids), seizures and/or anxiety. Thse pathophysiology, treatment and nursing care of people experiencing pain are fully described in Chapter 8.

CONSIDERATION FOR PRACTICE

There is no maximum allowable dose at end of life for opioids such as morphine sulfate; the dose should be increased by the palliative care medical specialist/doctor to whatever is necessary to relieve pain. Pethidine is not useful for chronic pain because it has a short half-life and a toxic metabolite that can cause irritability and seizures (Tierney, McPhee & Papadakis, 2004).

BOX 4.3 Providing comfort for the person nearing death

- Maintain clean skin and bed linens.
- Use a slide sheet to turn the person as often as required to keep them comfortable.
- Position the person to promote comfort and protect bony areas with padding. Reposition the person and raise the head of the bed if fluids accumulate in the upper airways and back of the throat.
- Use incontinence pads or insert an indwelling catheter (only if ordered) for urinary incontinence.
- Use gentle massage to reduce anxiety, improve circulation and shift oedema.
- Provide small, frequent sips of fluids or ice chips.
- Provide oral care, using a soft moist brush or moistened swabs. Do not use glycerine swabs as they dry the mouth out.
- Clean secretions from the eyes and nose.
- Administer prescribed pain medications as needed to maintain comfort.

Dyspnoea

Dyspnoea is the symptom of breathlessness. Although it may be experienced by some individuals with lung disease throughout their illness trajectory, it becomes more common as death nears. Dyspnoea is a subjective experience, and individuals often report feelings of suffocation, shortness of breath or tightness in the chest, which increase their anxiety and distress.

As death nears, respirations often become fast or slow, shallow and laboured. The person may have apnoea or Cheyne-Stokes respirations (regular periods of deep, rapid breathing followed by no breaths for 5 to 30 seconds). Secretions often gather at the back of the throat, which may cause an audible sound as the person breathes through them (Nuccio & Nuccio, 2009). This can be very distressing for family members and they should be reassured that these symptoms are normal for a person close to death. Fluid may accumulate in the lungs causing rales and rhonchi, especially in people who are well hydrated and are having difficulty swallowing or coughing. These sounds are not usually distressing for the person, but they may be treated by changing their position, and administering oxygen and opioids (to improve respirations and decrease anxiety). Anticholergic drugs provided early in care, prior to the build-up of secretions, can be helpful for people at the end of life (Nuccio & Nuccio, 2009).

Note that oxygen and suctioning are only temporary measures (especially with suctioning) and may even be traumatic for the person. Nursing care that may improve respiration includes keeping the head of the bed elevated and regular position changes. Morphine is the medication of choice for palliative treatment of dyspnoea. For individuals experiencing dyspnoea earlier in their illness, the breeze from a fan may provide some benefit.

Nausea, anorexia and dehydration

Typically, people at the end stage of life will be drinking and eating very little, if at all. This reduction in food and fluid intake may occur for a number of reasons, including symptoms of nausea and vomiting, dysphagia (difficulty swallowing) or as a result of fatigue. It may also be related to a reduced level of consciousness or a bowel obstruction, or simply anorexia (the absence of appetite) (van der Riet, 2009).

Nausea, with or without vomiting, is a common problem experienced by dying people. Nausea and vomiting may be caused by reduced gastric emptying, constipation, bowel obstruction, a side effect of morphine, uraemia or hypercalcaemia. The following list includes anti-emetic medications that may be used for nausea and vomiting at the end stage of life: prochlorperazine (Phenergan) or odansetron (Zofran), metoclopramide (Maxalon), levomepramazine, methotrimeprazine (Nozinan), cyclizine and haloperidol (Serenace). As with all medications, those used as anti-emetics may cause further complications or compound present health issues, such as increased constipation or drowsiness. It is essential that the nurse conduct a comprehensive assessment prior to and following the initiation of any medication regimens ordered.

Anorexia is another issue faced by many at the end of life. However, anorexia may be a protective mechanism by the body. The breakdown of body fats results in ketosis, which leads to a sense of wellbeing and helps decrease pain. This is one reason that parenteral or enteral feeding is not advocated at the end of life as it does not improve symptoms or prolong life, and may actually cause discomfort (Good et al., 2011).

Anorexia and a decrease in food and fluid intake are normal in the dying person; however, the family may view the practice of not enforcing fluids and food as 'giving up'. They may feel that their loved one is being abandoned if nurses and doctors are not intervening by providing intravenous fluids (van der Riet et al., 2008). All people at the end of life should be offered food and drink, but intravenous fluids are not routinely given (van der Riet, Brooks & Ashby, 2006).

Dehydration in the person nearing death primarily causes discomfort from dry mouth and thirst. The person should be given small sips of water, or an atomiser can be used to spray the inside of the mouth. Mouth care should be provided at least every 2 hours and more often if the person is breathing through their mouth. Good mouth care should involve moistening swabs with water (not glycerine swabs, as they dry mucous membranes), an oral gel or oral spray. It is important to avoid any drying agents such as lollies (sugar-free are fine), coffee and high-sugar drinks. When cleaning the person's teeth, there is no need to rinse the toothpaste. Mouthwashes may be used if tolerated and might include the following: salt and soda bicarbonate, Difflam and chlorhexidine.

Families need to be aware that dehydration is less of a problem than over-hydration. Forcing fluids or initiating intravenous fluids for hydration may in turn have adverse affects such as pulmonary oedema (increased fluid in the lungs), peripheral oedema, ascites, excessive wound or fistula drainage, and increased gastrointestinal secretions leading to vomiting.

CONSIDERATION FOR PRACTICE

Intravenous tubing may affect communication and physical contact between an individual who is dying and their loved ones. In a study by van der Riet et al. (2008), nurses reported that having tubes or lines in place, such as intravenous lines, affected the intimacy and the relationship between family members and the person.

Altered levels of consciousness

Neurological dysfunction may result from any or all of the following: decreased cerebral perfusion, hypoxaemia, metabolic acidosis, sepsis, an accumulation of toxins from liver and renal failure, the effects of medications and disease-related factors. These changes may result in a decreased level of consciousness or agitated delirium (Bush et al., 2014). People with terminal delirium may be confused, restless or agitated. Moaning, groaning and grimacing often accompany the agitation and may be misinterpreted as pain. The level of consciousness often decreases to the point where the person cannot be aroused. Although decreased consciousness and agitation are both normal states at the end of life, they are very distressing to the individual's family.

If possible, treatment for confusion or agitation is based on its cause—for example, pain or dyspnoea. Other medications include low doses of neuroleptics, tranquillisers or anti-anxiety medications. A person near death often has altered cerebral function, so the nurse must stand near the bedside and speak clearly. Hearing is believed to be the last sense a dying person loses, so the nurse should never whisper or engage in conversation with the family as if the person were not there.

Hypotension

As death nears, cardiac output decreases, as does intravascular blood volume. As a result, blood pressure gradually decreases and the pulse is often rapid and irregular. The extremities are cooler, and cyanosis is present in nail beds, skin and lips. The skin on the legs and in dependent areas may become mottled in colour. Renal perfusion decreases and the kidneys cease to function. Urinary output is scanty.

Complementary therapies: their use in end-of-life care

Complementary therapy is a broad term that recognises the link between the individual's mind, body and soul as fundamental to their health and wellbeing (Harris, Nagy & Vardaxis, 2006). Individuals may investigate complementary therapies and trial or extensively use therapies that are not prescribed by a medical practitioner.

Complementary therapies at the end of life have the potential to promote relaxation and comfort. The most frequently used complementary therapies for people requiring palliative care include aromatherapy, meditation, visualisation and massage (van der Riet, 2011). Nowadays in a hospital setting it is not uncommon to see some family members or carers practising therapies such as Reiki or therapeutic touch on a dying person. Reiki has been described as an art in which the practitioner transfers life energy between the practitioner and person in a compassionate way to bring balance, strength and harmony to both body and mind (Bossi, Ott & DeCristofare, 2008). For dying patients Reiki has the benefit of an improved sense of wellbeing and a positive sense of spiritual renewal (Viliotti, 2013). Throughout the increasing trajectory of an illness it is important for the nurse to adequately assess, inform, educate and support the person through the process of investigation, trial and use of these therapies. Complementary therapies may contribute to a nurse's self-care strategies, especially modalities such as yoga and meditation.

Support for the person and family

As the person's condition deteriorates, the nurse's knowledge of the person and family guides the care provided. It may be necessary to provide opportunities for individuals to express personal preferences about where they want to die and about funeral and burial arrangements. If the family feels that this is morbid, the nurse may explain that it helps the person to keep a sense of control as they approach death.

People who are dying need the opportunity to say goodbye to others. The nurse encourages and supports the person and family as they terminate relationships as a necessary part of the grief process. The nurse acknowledges that termination is painful and, if the person or family desires, stays with them during this time. Family members are often afraid to be present at the moment of death, yet dying alone is the greatest fear expressed by people at end of life.

Death

The nurse may also fear being present at the moment of the person's death. Research indicates that nurses and other health professionals frequently have difficulty in dealing with death and dying (Mallory, 2003; Melo & Oliver, 2011) In fact, Kübler-Ross (1969) noted that the nurse's fear of death frequently interferes with their ability to provide support for the dying person and family. Thoughts such as 'Please, God, don't let him die on my shift' are common, and they express the nurse's emotional turmoil in dealing with the task. Nurses who have worked through their own feelings about death and dying with effective self-care strategies are more at ease in assisting the dying person towards a peaceful death.

After the death, the nurse should be attentive to the needs of the family and significant others and acknowledge that grief reactions and needs vary for individuals. The nurse should allow the bereaved to express their sorrow, anger or guilt, which may help them resolve their grief. By accepting variations in the expression of grief, the nurse supports the family's grief reactions and helps prevent dysfunctional grieving. Dysfunctional grieving is an extended and unsuccessful resolution of grief.

Resolution of grief begins with acceptance of the loss. The nurse can encourage this by respecting the bereaved person's needs by maintaining open, honest dialogue and by providing the family with the opportunity to view, touch, hold, wash, kiss or visit with the person's body as long as is needed. As family members realise the finality of the death, they are often comforted by the presence of the nurse who cared for the person during the final days. Open and honest communication can ensure that the nurse is aware if their presence is required or not.

Care after a person dies

The nurse documents the time of death (which is required for the death certificate and all official records), notifies the doctor and notifies and supports the family. Once a person dies it is important that all infusion pumps and pressure mattresses are turned off. Experiencing machine alarms or noticing a deceased loved one's body move (from the rise and fall of a pressure mattress) can cause unnecessary distress for the bereaved person.

If the person dies at home, death must be pronounced before the body is removed from the home. Jewellery may be removed from the deceased and given to the family unless they ask that it be left on. Nurses should document the removal of valuables or jewellery and record who takes possession of the items, especially if the death occurs in a hospital or residential aged care facility. All tubing may be removed from the deceased unless otherwise indicated, such as in cases requiring a coroner's inquest. The body of the deceased is kept in place until the family is ready and gives permission for the body to be removed; this may be minutes, hours or days.

Acknowledgement of death in a hospital setting

Part of the philosophy of palliative care is the acknowledgement that death is a normal part of the life cycle. However, in hospitals or residential aged care facility settings, a person's death is not always acknowledged or discussed with the other residents of the facility/setting. Nurses in acute settings must acknowledge that a person may witness the death of another person and may need an opportunity to share their experience and express their feelings. There is a changing culture in Australian residential aged care facilities, where the deceased person's life may be celebrated with the other residents, allowing them to gain closure and acknowledgement of the death of a fellow resident.

Nurses' grief

The nurse who has developed a close relationship with the person who has died may experience strong feelings of grief. Sharing grief with the family after the death of a loved one helps both the nurse and family to cope with their feelings about the loss. Taking time to grieve after the person's death provides a release that can help prevent 'blunting' of feelings, a problem often experienced by nurses who care for individuals with an eventually fatal condition.

CONSIDERATION FOR PRACTICE

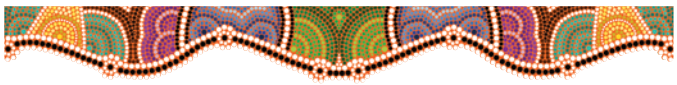
Crying with families, at one time considered unprofessional, is now recognised as simply an expression of empathy and caring.

Nurses working with individuals who are critically ill or have an eventually fatal condition should be aware that witnessing a person's death and the family's grief may reactivate feelings about some unresolved grief in their own lives. In these cases, nurses may need to reflect on their responses to their own losses. Also, nurses who work with individuals who are dying need support from peers and other professionals to work through the often overwhelming feelings that result from dealing with death, grief and loss.

INTERPROFESSIONAL CARE

Interventions for loss and grief may be planned and implemented by any or all members of the healthcare team. Nurses and social workers provide interventions to help individuals or families adapt to a loss. They also make referrals to mental health professionals (grief counsellors, social services), support groups, chaplains/pastoral care or legal agencies.

Grieving individuals may enter the healthcare system with significant somatic symptoms. Somatic symptoms are physical symptoms caused by the distress of grief. In some cases, the symptoms of grief and loss are overlooked until the person reaches a crisis state requiring psychiatric medical intervention. Collaborative care by the doctor and the nurse early in the normal grieving process can help the person achieve an early and effective resolution of grief and avoid physical or psychiatric health problems.



Nursing care

Nurses practising in all types of settings care for individuals who are in various stages of the grieving process. Like pain, grief is highly individual. The grief process may range from uncomfortable to debilitating, and it may last for a day or a lifetime, depending on what the loss means to the person experiencing it. The following section provides nurses with insights into the assessment and exploration of grief and loss for individuals for whom they care. It is acknowledged that this information is not exhaustive. Nurses should always maintain open communication with the person, significant others and multidisciplinary team; this will ensure individualised assessment and enquiry that has the person's needs as the main focus.

Health promotion

In planning a grief assessment of an individual, acknowledgement that the experience of loss can be associated with a range of factors is essential. It is also important that the nurse considers the individual's response, which may vary greatly from others' responses. In an era of short acute care hospitalisation, nurses may feel that an elaborate grief assessment is impossible or, at the least, impractical. However, research and clinical experience suggest that individuals who delay the grieving process after a loss are prone to have health problems that may last a lifetime.

Assessment

Grief reactions are personal and individual. However, knowledge of the expected physical reactions to loss provides the nurse with a basis for identifying reactions that require further assessment. To assess the extent of somatic distress—that is, physical symptoms caused by distress—the nurse observes for changes in sensory processes and asks questions about the individual's sleeping and eating patterns, activities of daily living, general health status and pain.

Physical assessment

Individuals may experience one or more predictable somatic symptoms as they become aware of a loss. Gastrointestinal symptoms occur frequently and may include indigestion, nausea or vomiting, anorexia, weight gain or loss, constipation or diarrhoea. The shock and disbelief that accompany a loss may cause shortness of breath, a choking sensation, hyperventilation or loss of strength. Some individuals also report insomnia, preoccupation with sleep, fatigue and decreased or increased activity level.

Crying and sadness are observed during normal grief states. Crying may make the individual feel exhausted and interfere with carrying out activities of daily living. However, a person who is unable to cry may have difficulty completing the mourning process. If the person does not express feelings of grief, somatic symptoms may increase. If a person is experiencing pain, it is imperative that their concerns be assessed, especially if the person has cancer or another painful illness. Knowledge of pain theories and pain assessment can help the nurse assess the need for pain medication (see Chapter 8).

Reactions to loss are not always obvious. For example, in individuals who experience an illness following a serious loss, assessment may reveal somatic complaints related to the grief state as well as the illness. When a person who has been healthy begins to develop patterns of increased illness, the nurse should be aware that this may signal dysfunctional grieving. This is especially common in the loss and grieving associated with a change in body image. In addition to making a physical assessment, assess the person's perception of the alteration in body image. The loss of a body part, weight gain or loss, and scars from surgery or trauma can be difficult for the person to accept. Some individuals may grieve hair loss that accompanies chemotherapy used in cancer treatment.

Spiritual assessment

Spiritual beliefs and practices greatly influence people's reaction to loss, and it is important for nurses to use sensitivity and skill when assessing a person's spiritual needs. Nurses need to establish rapport and trust with the person before they assess their spirituality. Nurses do not have a right to ask deeply personal questions about spirituality unless they have established trust and a connection with the person. The word 'spirituality' is loaded and can have many different interpretations. As Johnson (2005) and Roy (2011) remind us, spirituality has an elusive and perplexing quality. Some people might find the word 'spiritual' quite confronting because of possible judgments and may use metaphors such as 'peace' to describe their sense of spirituality.

Assessing the person's spiritual life and its significance to the person and family helps identify spiritual support systems. However, assessment needs to be explored within the context of the individual and their culture. Areas that might be explored include beliefs and meaning, experience and emotion, ritual and practice, courage and hope (Swinton, 2006).

Psychosocial assessment

When working through the grief process, individuals can be overwhelmed by the fears associated with the loss and the changes it will produce. Individuals diagnosed with cancer can also experience a lack of trust in their body, especially if their diagnosis was not prompted by specific symptoms. The individual responding to an actual or perceived loss commonly expresses anxiety (fear of the unknown). An extreme level of anxiety can threaten the individual's wellbeing. Assessment includes helping individuals openly acknowledge their fears. Some individuals may fear the feelings they experience while proceeding through the grief process more than the loss itself. The most common fear expressed by individuals facing a loss is that of losing self-control.

Focusing on the meaning of the loss to the person is more important than attempting to place the person in a sequence or phase of grief. The degree of caring and sensitivity shown when asking questions that consider the meaning of the loss influences the amount of information the person will be willing to reveal. Asking questions such as 'Why do you feel this way?' or 'What does this loss mean to you?' is less helpful than making a statement such as 'This must be difficult for you.'

MEETING INDIVIDUALISED NEEDS Teaching suggestions for individuals experiencing a loss

- Encourage both children and adults to discuss expected or impending loss and to express their feelings.
- Encourage problem-solving skills: define what possible changes and problems may be related to the predicted loss, develop potential strategies for dealing with problems, list pros and cons of each strategy and decide which strategies might be most useful to try first to solve potential problems associated with loss.
- Talk to individuals and families about how to support a person who is dealing with an impending loss.
- Explain what to expect with a loss: sadness, fear, rejection, anger, guilt, loneliness.
- Develop strategies for goal setting and future-orientated planning.
- Discuss signs of grief resolution:
  - Individuals and families need to understand that the acute stage of grief has no set timeline and, as discussed previously, grief reactions may vary for each individual.
  - Explain that 'triggers' such as photographs, events, songs or memories and especially the anniversary of the loss may potentially cause painful 'waves' of grief.

The latter more effectively conveys empathy and a genuine interest in hearing how the person feels about the loss.

Awareness of the altered sensorium observed during the stage of shock and disbelief provides parameters for assessment. The nurse may note in the individual feelings of numbness, unreality, emotional distance, intense preoccupation with the loss, helplessness, loneliness and disorganisation. As awareness of the loss begins to develop, preoccupation with the person who has died or the object that has been lost may increase, and self-accusation and ambivalence towards the person or object may follow.

Exploring grief

Anticipatory grieving, chronic sorrow and **death anxiety** are issues that individuals experiencing loss and grief, as well as the person who is nearing death, may experience. Nurses may find that the following information will assist in the assessment and exploration of anticipatory grieving, chronic sorrow and death anxiety.

Anticipatory grieving

Anticipatory grieving is a combination of intellectual and emotional responses and behaviours by which people adjust their self-concept in the face of a potential loss. Anticipatory grieving may be a response to one's own future death; to potential loss of body parts or functions; to potential loss of a significant person, animal or possession; or to potential loss of a social role. Nursing enquiries are designed to assist with grief resolution.

- Assess for factors causing or contributing to the grief. Ask about support systems, how many losses have occurred, relationship with the lost person, significance of the body part and previous experiences with loss and grief. *Grief and mourning occur when a person experiences any type of loss.*
- Use open-ended questions to encourage the person to share concerns and the possible effect on the family. *Grief resolution cannot occur until the individual acknowledges the loss.*
- Promote a trusting nurse–patient relationship: allow enough time for communications; speak clearly, simply and concisely; listen; be honest in responses to questions; do not give unrealistic hope; offer support; and demonstrate respect for the person's age, culture, religion, ethnicity and values. *An effective nurse–patient relationship begins with acceptance of the individual's feelings, attitude and values related to the loss. If the person is ready to talk, listening and being present are the most appropriate interventions.*
- Discuss with the person and their family the stages of grief. *This helps them to be aware of their emotions in each stage and reassures them that their reactions are normal.*
- Provide time for decision making. *In periods of stress, people may need extra time to make informed decisions.*
- Provide information about appropriate resources, including support from family, friends and support groups, community resources and legal/financial aids. *Support from others decreases feelings of loneliness and isolation and facilitates grief work.*

Chronic sorrow

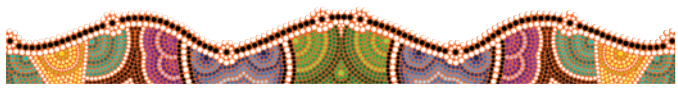
Chronic sorrow is a cyclical, recurring and potentially progressive pattern of pervasive sadness experienced in response to continual loss, throughout the trajectory of an illness or disability. It is triggered by situations that bring to mind the person's losses, disappointments or fears. It may be experienced by a person, parent or caregiver, or a person with a chronic illness or a disability.

- Explain the difference between chronic sorrow and chronic grieving. *Grieving is time limited and ends in adaptation to the loss. Chronic sorrow may vary in intensity, but it persists as long as the person with the disability or chronic sorrow condition lives.*
- Encourage verbalisation of feelings about the loss and about the personal relevance of changes to hopes for the future. *Expressing feelings is normal and necessary to decrease the emotional pain.*
- Help identify triggers that intensify the sorrow, such as birthdays, anniversaries and holidays. *When triggers have been identified, role-playing may make the events less painful.*
- Refer to appropriate community support groups. *Participating in support groups with others experiencing grief is helpful in coping with loss.*
- *Encourage use of personal, family, significant other and spiritual support systems to facilitate coping with loss.*

Death anxiety

Death anxiety is worry or fear related to death or dying. It may be present in individuals who have an acute life-threatening illness, who have an eventually fatal condition, who have experienced the death of a family member or friend, or who have experienced multiple deaths in the same family.

- Explore the person's knowledge of the situation. For example, ask, 'What has your doctor told you about your condition?' *This informs you of the person's knowledge base about the condition and about their ability to make informed decisions.*
- Ask them to identify their specific fears about death. *This provides data about any unrealistic expectations or misperceptions.*
- Ask the person to identify needed help. *This determines whether available resources are adequate.*
- Encourage independence and control in decisions about treatment and care. *This promotes self-esteem, decreases feelings of powerlessness and allows the person to retain dignity in dying.*
- Facilitate access to culturally appropriate spiritual rituals and practices. *This provides spiritual comfort.*
- Explain advance directives and assist with them if necessary. *Advance directives help ensure that the individual's wishes for end-of-life care are carried out.*
- Encourage life review and reminiscence. *Life review is self-affirming.*
- Encourage activities that may bring comfort such as listening to music, aromatherapy, massage or relaxation exercises. *These activities decrease anxiety.*
- Suggest keeping a journal or leaving a written legacy. *A written document provides continuing support to others after death.*



NURSING CARE PLAN A person experiencing loss and grief

Mrs Sandra Bell is an 87-year-old widow recently admitted to a residential aged care facility following her husband Allan's death 12 months ago. Mr Bell had suffered from dementia, and his wife was his carer until 2 years prior to his death when he had been admitted to a dementia unit of the local rural hospital. Mrs Bell visited her husband regularly even when he could no longer recognise her. She has lived all her life in a country town and had established a strong network of friends through membership of the golf club and local services club. However, her social contacts with friends have diminished because of their poor health. Mrs Bell has a daughter and son who both live in the city. Although they keep in regular contact by telephone, they are only able to visit on special family occasions.

Knowing the strain of maintaining a home and losing contact with close friends, Mrs Bell's family felt it would be best for her to move to the city to be closer to her children and grandchildren. Mrs Bell sold her home and entered a residential aged care facility in the city, to be closer to her family.

After several visits, Mrs Bell's daughter notices that her mother has become less social, not wanting to mix with the other residents or participate in community activities, and is more and more upset about her situation. Mrs Bell complains that the other residents are unfriendly and that she misses her home and friends. She does not like the community dining room and says that her back and hips are painful. Mrs Bell also tells her daughter she is not sleeping and is constipated. Her daughter speaks to the nurse and is told that her mother is very quiet, does not mix well with the other residents, seems resentful when approached by the staff and does not volunteer that she is unwell. The nurse reports to Mrs Bell that during her admission to the aged care facility her medical tests showed arthritis but no other pathological disorder.

ASSESSMENT

On assessment, Mrs Bell says, 'I'm a sick woman and no one will listen to me! I can't walk, I'm so weak, my head hurts and I'm always sick in my stomach. I haven't had a bowel movement in a week and I never sleep more than three hours a night.' Physical assessment findings include swollen knees and ankles, with limited mobility of the lower extremities.

DIAGNOSES

- *Grieving* related to death of husband, lost contact with friends, and recent move from a country town to a residential aged care facility in the city.
- *Disturbed sleep pattern* related to grieving.
- *Constipation* related to inactivity.

PLANNING

- A multidisciplinary team planning meeting was organised by the Nursing Unit Manager (NUM) to review and identify Mrs Bell's issues. The following diagnoses were identified, along with expected outcomes, implementation and evaluation.

Expected outcomes

- Engage in normal grief work: work through grief process, discuss reality of losses, use non-destructive coping mechanisms, and discuss positive and negative aspects of the loss.
- Experience adequate and restful sleep.
- Have a bowel movement with soft formed stools at least every other day.

IMPLEMENTATION

- Promote trust: show empathy and caring, demonstrate respect for her culture and values, offer support and reassurance, be honest and engage in active listening.
- Assist in identifying her feelings: anger, fear, loneliness, guilt, isolation.
- Explore previous losses and the ways in which she has coped.
- Encourage review of her relationship with her deceased husband.
- Reinforce expressions of behaviours associated with normal grieving.
- Encourage participation in usual spiritual practices.
- Encourage participation in a grief group that meets at the facility.
- Consult with the physical and recreational therapist to help the nursing staff provide afternoon activities tailored to Mrs Bell's interests.
- Provide measures that assist in bowel evacuation: Encourage exercise as tolerated, including walks. Offer

(continued)

NURSING CARE PLAN A person experiencing loss and grief (continued)

fluids foods that stimulate bowel movement (e.g. fresh fruit). Offer privacy: close the door, ensuring that the buzzer is within reach and do not interrupt.
- Provide measures to assist sleep (e.g. offer warm milk drink with honey, chamomile tea, quiet environment). Offer gentle back massage, use of aromatherapy oils such as lavender.
- Administer a mild laxative and/or stool softener, if necessary, but discontinue as soon as possible.

EVALUATION

After 4 weeks at the residential aged care facility, Mrs Bell states, 'I don't feel any better, but I know I have to accept my situation.' Although Mrs Bell says she doesn't feel better, she is walking the length of the hall, sleeping better and having regular bowel movements. She is also less withdrawn and has openly discussed her feelings related to her husband's death and the loss of her social contacts. She has attended the grief group once and has attended pastoral care services on Sunday for the past 2 weeks. Her children visit her each Saturday and take her in a wheelchair to the shopping centre.

CRITICAL THINKING QUESTIONS

1. What common physical manifestations of grief did Mrs Bell experience?
2. Outline what you have learned from Mrs Bell's case study/story.
3. What communication strategies and education would you use if Mrs Bell told you she does not want any help and that she just wants to be left alone to die?

REFLECTION ON THE NURSING PRACTICE

1. How would you respond to Mrs Bell if she says to you, 'Please help me die'?
2. How might you broach the topic of advance directives with a person? Do you feel comfortable about talking to people about advanced care planning?

CHAPTER HIGHLIGHTS

- Grief is the emotional response to a loss, experienced by a person as grieving. Bereavement, a form of depression accompanied by anxiety, is a common response to loss of a loved one by death. Death, although inevitable, is an immensely difficult loss.
- There are many different theories of how individuals respond to loss, and grief. These theories are useful when providing nursing care to individuals and their families.
- A person's response to loss is influenced by age, social support, family members, cultural and spiritual beliefs, and rituals of mourning. Nurses need to assess the way in which they respond to loss, to better care for people.
- Legal and ethical issues involved in end-of-life care include advance directives, advanced care planning, enduring power of attorney, do-not-resuscitate directives and euthanasia.
- Palliative care is a philosophy of care that supports a dignified and peaceful death when individuals and their families are faced with limited life expectancy. Palliative care is focused on the relief of physical, mental and spiritual distress for people with an eventually fatal condition.
- To provide knowledgeable and compassionate care at the end of life, nurses must recognise symptoms that may be present as the individual nears death, support the person and their family, provide care to the individual and family immediately after death, and resolve their own grief.
- Nursing care of individuals experiencing an actual or potential loss includes accurate individualised physical, spiritual and psychosocial assessment, and awareness of responses of anticipatory grieving, chronic sorrow and/or death anxiety.

CONCEPT CHECK

1 Which of the following statements best describes loss?
   1 It is determined by one's cultural values.
   2 It is largely dependent on support of family and friends.
   3 It can be determined only by the person who experiences it.
   4 It is the same as grief and mourning.

2 Kübler-Ross believed that one usually first responds to a situation of loss with:
   1 anger
   2 bargaining
   3 depression
   4 denial

3 What is an important factor in the successful resolution of grief?
   1 social isolation
   2 support systems
   3 triggers of grief
   4 loss acknowledgement

4 What is the primary factor that dictates the rituals of mourning?
   1 culture
   2 age
   3 gender
   4 religion

5 What document expresses a person's wishes in relation to the planning of treatment in the event of their inability to communicate their wishes because of an eventually fatal condition?
   1 enduring power of attorney
   2 advanced care planning
   3 do-not-resuscitate directive
   4 living will

6 Which of the following statements is true of a hospice?
   1 A hospice is a special place of care.
   2 A hospice care is a lifelong type of care.
   3 A hospice is a model of care rather than a place of care.
   4 A hospice is designed for individuals with serious chronic illness.

7 A person nearing death requests that no medication be given that would cause a loss of consciousness, including pain medication. What would a nurse do to provide the best end-of-life care in this situation?
   1 Give the medication; comfort is the highest priority.
   2 Give half the ordered dose to provide compassionate care.
   3 Discuss this with family members and follow their wishes.
   4 Respect the person's wishes and withhold pain medications.

8 Which of the senses is believed to be the last one lost as a person nears death?
1 hearing
2 vision
3 touch
4 smell

9 Which of the following statements best describes the treatment of pain at the end of life?
1 As an individual nears death, no pain is perceived and no medications are necessary.
2 It is important to withhold pain medications if the individual has respiratory changes.
3 There is no maximum allowable dose for opioids during end-of-life care.
4 Nurses should not administer opioids to the dying person.

10 A woman, recently widowed, tells the nurse, 'I just can't even get out of bed in the mornings anymore.' What response would be most helpful in resolving her grief?
1 'I don't know why you feel that way.'
2 'This must be a difficult time for you.'
3 'Why do you think you feel this way?'
4 'After you get up, you will feel better.'

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CHAPTER 5

NURSING CARE OF PEOPLE WITH PROBLEMS OF SUBSTANCE MISUSE

ANNA TRELOAR, TERESA STONE

KEY TERMS

LEARNING OUTCOMES

- Distinguish between substance use disorders and substance-induced disorders.
- Explain risk factors associated with substance misuse.
- Classify major addictive substances and explain effects of addictive substances on physiological, cognitive, psychological and social wellbeing.
- Support interprofessional care in the provision of individualised care for people living with substance use problems, including diagnostic tests, emergency care for overdose and treatment of withdrawal.
- Develop awareness of current policy, both local and global, and future directions in relation to alcohol and drug use treatment and primary prevention measures.
- Recognise signs and symptoms of potential substance use disorders in co-workers and understand professional responsibilities in relation to this.

CLINICAL COMPETENCIES

- Assess health status of people living with substance use disorders.
- Monitor, document and report physical manifestations of substance abuse.
- Assess for signs of withdrawal and monitor for complications.
- Use evidence-based research to plan and implement nursing care for people experiencing withdrawal symptoms.
- Implement individualised nursing interventions for people living with substance use disorders.
- Provide skilled nursing care in a range of clinical settings during the period of substance withdrawal.
- Collaborate with other disciplines when caring for people with substance use disorders.
- Educate people about stress management, coping skills, nutrition, relapse prevention and healthy lifestyle choices.

Substance use, misuse and dependence are widespread in Australia today. There is a common belief that legal drugs do less damage than illegal drugs, but as you read this chapter you will see that this is not the case. People who misuse substances may believe that they cannot change their pattern of consumption; many people do not recognise that their use has become problematic or, if they do, do not know how to find help. Their families also may require assistance and education.

Consider this family:

Sue (39) is in the kitchen preparing the evening meal. She finds her job very stressful on top of family responsibilities. She takes a couple of pills from the packet on the shelf. She used to take alprazolam, but that's hard to get now. She makes do with what she can get from all the doctors she visits regularly, and with what friends give her.

Her husband Joe (43) is still in the pub. He works hard on the building site and doesn't see why he shouldn't relax at the end of the day. It's his shout. He is not sure how many schooners he has had tonight—same as other days most likely. But how many is that? He's often very late for dinner at home now, and there is not much cash in his wallet.

Joe's elder son, Dale (19), is just home from TAFE and wishing he had never ever been introduced to ice (crystal methamphetamine). He is behind in his course, he is running out of money, and lately he has had the strange feeling that people are looking at him and talking and laughing about him.

Upstairs in his room Joe's younger son, Milton (15), is rolling a joint (marijuana cigarette). He wants to be a pilot, or a soccer star, or play guitar in a band. He is in love with Rosie next door. But she doesn't know and wouldn't care anyway. There's nobody to talk to. Angrily, he lights up.

Nobody in this family realises that their substance use has become problematic, but all are misusing a drug—whether the drug is legal, illegal or a legal substance being illegally used.

SUBSTANCE-RELATED DISORDERS

Substance use and psychiatric diagnoses are categorised in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.) (DSM-5-TM) published by the American Psychiatric Association (APA) (2013). The APA currently divides **substance-related disorders** into two groups: substance use disorders (e.g. alcohol use disorder, opioid use disorder) and substance-induced disorders (e.g. substance-induced delirium, substance-induced psychotic disorder). Physiological dependence, including tolerance and withdrawal, is a normal response to repeated doses of many prescribed medications including opioids, sedatives and stimulants; people should not be diagnosed with a substance use disorder solely on the basis of this (APA, 2013). Substance use disorders are seen as a pathological pattern of behaviours related to substance use (APA, 2013). Diagnostic criteria are described in more detail in Table 5.1.

A mild disorder is suggested by two or three of the points in Table 5.1, a moderate disorder by four or five, and a severe disorder by six or more.

APA's DSM-5-TM criteria (discussed in more depth in Chapter 50) deal with behavioural aspects and maladaptive patterns of substance use, emphasising the physical symptoms of **tolerance** and **withdrawal**. Tolerance occurs when the initial dose of a substance loses its effectiveness over time. As tolerance increases, higher and higher doses are needed to obtain the desired effect.

TABLE 5.1 Substance use disorders

1. Substance is taken in larger amounts or for longer periods than is intended.
2. Unsuccessful or persistent desire to cut down or control substance use.
3. More time occupied in getting, taking and recovering from the substance.
4. Intense craving.
5. Failure to fulfil important obligations.
6. Continued use of substance despite persistent problems.
7. Important activities given up or reduced because of use.
8. Continued use in hazardous situations.
9. Continued use despite knowledge of physical or psychological problems caused by use.
10. Presence of tolerance to the drug.
11. Presence of withdrawal symptoms.

Source: Adapted from American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.) (DSM-5-TM). Washington, DC: Author.

Ceasing to take a drug on which a person is physically dependent can produce **withdrawal symptoms** within hours—an uncomfortable state lasting several days, manifested by a range of signs and symptoms depending on which drug is involved.

In any 12-month period, 5.1% of Australians aged 16–85 years suffer a substance use disorder, men at twice the rate of women (7.0% compared with 3.3%). Higher rates prevail in lower age groups. Alcohol use disorders are more prevalent than all other substances (Australian Bureau of Statistics, 2008).

Substance use problems frequently coexist with other physical and mental health conditions such as anxiety and depressive disorders. More than 90% of people who commit suicide have a depressive or substance abuse disorder (National Institute of Mental Health, 2004). The human tendency to seek pleasure and avoid stress and pain is partially responsible for substance abuse. Currently available data suggest that ethanol increases opioid neurotransmission, this activation being part of the mechanism responsible for its reinforcing effect (Oswald & Wand, 2004). Most studies have focused on the role of dopamine D1 and D2 receptors in sustaining the addictive nature of drugs, but recently the dopamine D3 receptor has also appeared to be involved in drug-seeking behaviour (Heidbreder et al., 2004). Alcohol also binds directly to the receptors for acetylcholine, serotonin and gamma-aminobutyric acid (GABA) and the NMDA (*N*-methyl-D-aspartate) receptors for glutamate (Dubuc, 2002). The following link will show you in detail how drugs and alcohol interact: http://thebrain.mcgill.ca/flash/i/i_03/i_03_m/i_03_m_par/i_03_m_par_alcool.html#drogues.

The reinforcing properties of alcohol and other drugs can create a pleasurable experience, and reduce the intensity of an unpleasant one.

Dependence on psychoactive substances is a chronic relapsing brain disease. Repeated use of psychoactive substances tends to hijack the reward centre of the brain, which has strong neuronal pathways to the prefrontal regions that regulate behaviour, decision making and inhibition control. Neuroadaptation associated with **substance dependence** triggers strong urges and cravings to use drugs, thus contributing to the relapsing nature of the condition: stress, withdrawal symptoms and

drug cues can all be triggers to relapse. These neurophysiological aspects of substance dependence currently form an area of intense research interest (Baler & Volkow, 2006).

Substance craving may be heightened by the **kindling** effect—long-term changes in brain neurotransmission occurring after repeated detoxifications which increase neuron sensitivity, and are believed to intensify obsessive thoughts or cravings. Eventually the brain responds spontaneously in a dysfunctional manner even when the substance is no longer used (Breese, Overstreet & Knapp, 2005). This phenomenon may explain the tendency for subsequent episodes of withdrawal to worsen progressively.

Co-occurring disorders refers to substance abuse or dependence and a psychiatric disorder coexisting in one individual, a condition also described as co-morbidity, **dual diagnosis** or dual disorder. Increasing numbers of people experience co-morbid mental health and drug and alcohol problems and disorders, which adds complexity to assessment, diagnosis, treatment and recovery, and is known to increase the risk of relapse (Government Department of Human Services, Victoria, 2007), and of HIV infection, medical complications and premature death (NSW Health, 2008b). A recent large-scale study of Australians living with psychosis found that alcohol and illicit drug use was very common (51% were diagnosed with alcohol dependence, 51% with cannabis misuse or dependence, and 32% with other illicit misuse or dependence), and that there has been a substantial increase in substance use among Australians living with psychosis since the last large-scale study in 1997–1998 (Moore et al., 2012). One disorder can be an indication of another: alcohol dependence and major depression commonly occur together. Gilman and Abraham (2001) suggested that each of these illnesses poses a significant risk of development of the other within 12 months; a depressed person may use alcohol in self-medication, and an alcohol-dependent person may become depressed. Virgo et al. (2001) found that those dually diagnosed were more likely to be younger, male, in less stable accommodation, unemployed, and to have more than one psychiatric diagnosis and personality disorder. They tended also to have more crises and to pose greater risk to themselves and others. (Table 5.2 lists terminology associated with substance abuse.)

TABLE 5.2 Terminology associated with substance abuse

TERM	DEFINITION
Abstinence	Voluntarily refraining from drugs or alcohol
Addiction	A disease process characterised by the continued use of a specific chemical substance despite physical, psychological or social harm (formerly used interchangeably with substance dependence; dependence is the preferred term)
Co-dependence	Maladaptive behaviour by a partner of a person misusing substances tending to perpetuate the alcohol or drug dependence
Co-occurring disorders	Concurrent diagnosis of a substance use disorder and a psychiatric disorder. One can precede and cause the other, as in the relationship between alcoholism and depression
Cross-tolerance	Tolerance of one drug confers tolerance of another
Delirium tremens	A medical emergency usually occurring 3 to 5 days after the start of alcohol withdrawal and lasting 2 to 3 days; characterised by paranoia, disorientation, delusions, visual hallucinations, elevated vital signs, vomiting, diarrhoea and diaphoresis
Detoxification	The process of helping an addicted individual safely through withdrawal
Dual diagnosis	The coexistence of substance abuse/dependence and a psychiatric disorder in one individual (used interchangeably with dual disorder and co-occurring disorders)
Harm reduction	A strategy recommended by NSW Health (2008b) which emphasises the use of realistic and practical approaches to harm reduction, focusing on reducing the negative effects of drug and alcohol use
Kindling	Brain sensitisation to events such as stress, trauma or the effects of substance use
Korsakoff's psychosis	Secondary dementia caused by thiamine (B_1) deficiency which may be associated with chronic alcoholism; characterised by progressive cognitive deterioration, confabulation, peripheral neuropathy and myopathy
Physical dependence	A state in which withdrawal syndrome will occur if drug use is discontinued
Polysubstance abuse	Simultaneous use of many substances
Problematic drug and alcohol use	Patterns of drug and alcohol use warranting intervention (NSW Health, 2008b)
Psychological dependence	An intensive subjective need for a particular psychoactive drug
Risk factor	Any attribute, characteristic or exposure that increases the likelihood of developing a disease or condition; may include behavioural, environmental, biomedical or genetic factors.
Tolerance	State in which a particular dose elicits a smaller response than formerly. With increased tolerance, higher and higher doses are needed to obtain the desired response
Wernicke's encephalopathy	Caused by thiamine (B_1) deficiency, characterised by nystagmus, ptosis, ataxia, confusion, coma and possible death; thiamine deficiency is common in chronic alcoholism
Withdrawal syndrome	Constellation of signs and symptoms occurring in physically dependent individuals when they discontinue drug use

RISK FACTORS

Various **risk factors** help explain why one person becomes addicted while another does not. Genetic, biological, psychological and sociocultural factors can influence dependence on or abuse of a substance.

- *Genetic factors* include an apparent hereditary factor, especially with alcohol use and dependence. Up to 50% of vulnerability to addiction can be attributed to genetic factors (Dick & Agrawa, 2008). Women generally drink less alcohol than men do, and are less likely to have characteristics associated with heavy drinking such as aggressiveness, drinking to reduce distress and antisocial tendencies (Nolen-Hoeksema, 2004); however, women are more vulnerable than males to the acute and long-term effects of alcohol abuse.
- *Biological factors* were first identified by Jellinek (1946) in his disease model of alcoholism; he hypothesised that addiction to alcohol might have a biochemical basis and noted specific phases of the disease. Expanding on Jellinek's early work, researchers have implicated low levels of dopamine and serotonin in the development of alcohol dependence (Czermak et al., 2004; Nellissery et al., 2003). Dopamine and dopamine receptor sites are intricately involved in the complex workings between the nervous system and substances of abuse; an impact of any drug on the biochemical mechanism of the brain must occur at a receptor site or at a number of them (see Figure 5.1).

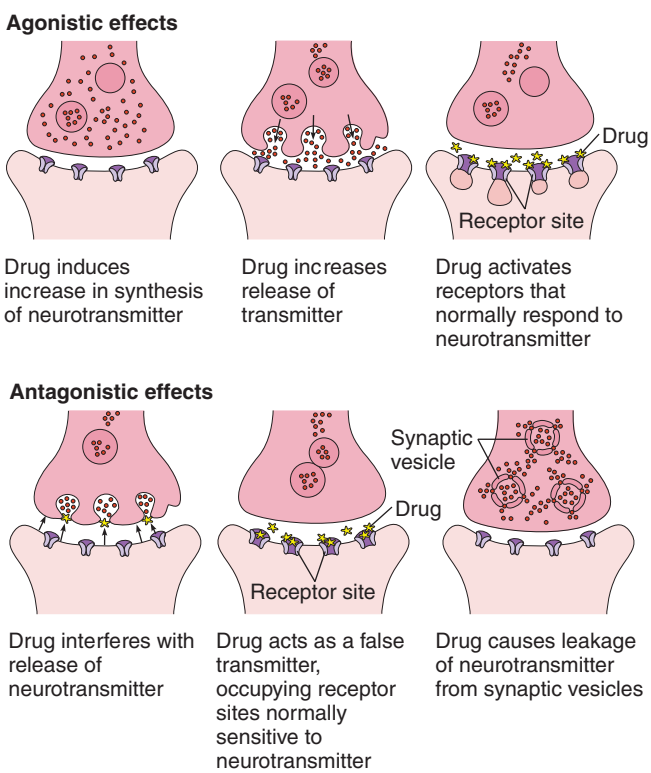


FIGURE 5.1 ■ Action of abusive substances at brain receptor sites

Most substances of abuse either mimic or block the brain's most important neurotransmitters at their respective receptor sites; for example, heroin and other opioids mimic natural opiate-like neurotransmitters such as endorphins, encephalins and dynorphins. In contrast, cocaine and other stimulants block the re-uptake of dopamine, serotonin and norepinephrine (noradrenaline) (Stuart & Laraia, 2005).

- *Psychological factors* attempt to explain substance abuse through a combination of psychoanalytical, behavioural and family system theories. Psychoanalytical theorists may view substance abuse as a fixation at the oral stage of development, whereas behavioural theorists see addiction as a learned, maladaptive behaviour. Family system theory focuses on the pattern of family relationships through several generations. Although no addictive personality type has been identified, substance-dependent people seem to exhibit several common factors: many substance abusers have experienced sexual or physical abuse in childhood and, as a result, have low self-esteem and difficulty in expressing emotions; higher levels of distress have been associated in young adults with hazardous or harmful alcohol or illicit drug consumption (Australian Institute of Health and Welfare (AIHW), 2014; Caldwell et al., 2002). A link also exists between substance abuse and psychological disorders such as depression, anxiety, and antisocial and dependent personalities; substance users who are depressed or anxious are less likely to embrace treatment for drug or alcohol dependence. The habit of using a substance becomes a form of self-medication to cope with day-to-day problems, and over time develops into dependence.
- *Sociocultural factors* may influence individuals' decisions as to when and how they use which substances. Environmental factors, including socioeconomic disadvantage and peer group pressure as well as cultural norms about drug and alcohol, influence patterns of use (de Crespigny et al., 2003). Little firm evidence concerning culturally and linguistically diverse people living in Australia indicates any variables associated with problematic drug and alcohol use (NSW Health, 2008b). Compared with other ethnic groups, Asians report the lowest prevalence of family history of alcoholism (Ebberhart et al., 2003). While Indigenous Australians have been shown to be less likely than the non-Indigenous to consume alcohol, those who do are more likely to drink at risky and high-risk levels, and are therefore more likely to experience the adverse physical and social effects of heavy alcohol consumption (Ministerial Council on Drug Strategy, 2006).

Many factors contribute to substance use and to the risk of substance abuse and dependence; no single element can cause one individual to develop a pattern of drug use while another does not. Some people experiment, some use recreationally, and a few become dependent. Risk factors can only help identify those who are most vulnerable. Thorough assessment is necessary in order to understand the whole person and plan appropriate interventions.

Substance users come from all ages and social, economic and cultural groups, with no particular personality styles or traits. Young people by nature are often risk takers, their

FOCUS ON CULTURAL DIVERSITY Substance use and special groups

Ethnic identity plays a unique role in substance use; adolescents in particular are influenced by cultural norms and practices, in addition to other environmental and biological factors. Positive ethnic identity (i.e. strong affiliation, attachment and pride) may 'protect' adolescents and help them form resistant behaviours (Marsiglia et al., 2004). However, introduction to and immersion in Australian culture may contribute to the initiation and maintenance of drug and alcohol abuse (NSW Health, 2008b).

High levels of problematic drug and alcohol use among Indigenous Australians are both the cause and effect of much suffering (Ministerial Council on Drug Strategy, 2006, p. 2), and are linked to the effects of dispossession and dislocation from traditional lands. Indigenous Australians are at increased risk of violence, depression, anxiety, attempted suicide, poor nutrition and medical complications (NSW Health, 2008b). In order to develop a culturally appropriate treatment plan, nurses need to consider the community and to consult with the person living with substance use disorder, the family, senior community members and specialist Indigenous health workers. Factors identified as contributing to the success of drug and alcohol health interventions include Indigenous community control, clearly defined management structures and procedures, trained staff and effective staff development programs, multi-strategy and collaborative approaches, adequate funding, and clearly defined, realistic objectives for provision of appropriate services to meet community needs (Gray, Saggers & Sputore, 2000).

In New Zealand, Kypri (2003) observed different patterns of drinking among Maori and non-Maori people: while similar total volumes of alcohol were consumed, non-Maori drank more frequently but, on average, 40% less alcohol per drinking occasion. The alcohol-related mortality of Maori is four times that of non-Maori, and years of life lost due to alcohol more than double (Ministry of Agriculture and Forestry, 2005).

Critical thinking in person-centred care

1 You are a school nurse in a community with a large population of Indigenous people. Alcohol consumption and binge drinking among the high school students are an increasing problem, and the head teacher has asked you for ideas to deal with it. How would you respond?
2 You are caring in the emergency department for a 23-year-old Asian female, brought in by her boyfriend. He tells you they had been at a party where she consumed alcohol for the first time. She is weak, her face is flushed and she is vomiting violently. What would you do?

experimental or social drug use arising in response to a desire to test social boundaries and cultural norms as part of their path to independence and adulthood. The majority mature out of this stage of their lives and become responsible and productive adults, but the age of initiation into alcohol or other drug use has emerged as a risk factor for developing a substance use disorder (King et al., 2013).

A variety of factors can lead to continued problematic use of alcohol and/or other drugs, resulting in substance abuse and substance dependence; these include a history of abuse or trauma, and mental health problems such as antisocial personality disorder, depression, anxiety disorders, low self-esteem and social anxiety. Alarmingly, prescription medication misuse is on the rise (AIHW, 2015).

ADDICTIVE SUBSTANCES AND THEIR EFFECTS

Caffeine

Caffeine is a central nervous system stimulant, which increases the heart rate and acts as a diuretic. Although commonly consumed daily in soft drinks, energy drinks, coffee, tea, chocolate, guarana, and some prescription and over-the-counter medicines, an excessive amount can cause negative physiological effects, especially cardiac-related risks. Approximately 300 mg per day is safe for most people, but more than 600 mg is considered excessive and is not recommended; it may cause insomnia, anxiety, depression and stomach upsets (Kneisl & Trigoboff, 2013; NSW Health, 2007). An average cup of instant coffee may contain 60–100 mg of caffeine; an espresso coffee, 90–200 mg; tea, 30–100 mg; cola drinks, 30–50 mg per 250 mL serve; and chocolate, 20–60 mg per 200 g bar (NSW Health, 2007).

People with a history of cardiac disease are advised to reduce caffeine intake or eliminate it altogether: large quantities can cause higher total cholesterol levels. Abrupt withdrawal is likely to cause headaches, irritability and generalised feelings of tiredness in a caffeine-addicted person.

Nicotine

Nicotine is found in tobacco (cigarettes and cigars) and enters the system via the lungs and oral mucous membranes; in low doses it stimulates nicotinic receptors in the brain to release noradrenaline and adrenaline, causing vasoconstriction. As a result, the heart rate accelerates and the force of ventricular contractions increases. Gastrointestinal (GI) effects include an increase in gastric acid secretion, tone and motility of GI smooth muscle, and promotion of vomiting. Nicotine acts on the central nervous system (CNS) as a stimulant, binding to acetylcholine receptors in the brain and causing the release of dopamine and noradrenaline. Initially, nicotine increases respiration, mental alertness and cognitive ability, but eventually it depresses these responses (Kneisl & Trigoboff, 2013).

The difficulty of quitting smoking is thought to be caused by dopamine release, which in turn reinforces the addictive craving for more. Nicotine in cigarettes has a half-life of 1 to 4 hours, so withdrawal symptoms become evident within a few hours after the last cigarette; withdrawal peaks during days 2 and 3, and tends to resolve at about 2 to 4 weeks. According

to DSM-5-TM (APA, 2013), nicotine withdrawal can be diagnosed if four or more of the following symptoms are present within the first 24 hours of nicotine reduction or cessation:

- depressed mood
- insomnia
- irritability, frustration or anger
- anxiety
- difficulty concentrating
- restlessness
- increased appetite.

Cessation can pose a problem for hospitalised smokers now that health facilities are smoke-free (see Table 5.3).

Tobacco smoking is the single most preventable cause of ill health and death, being a major risk factor for coronary heart disease, stroke, peripheral vascular disease, cancer and a variety of other diseases and conditions. However, smoking rates in Australia are falling (AIHW, 2014). Almost one-third of persistent smokers have reduced their daily consumption, and one-quarter tried unsuccessfully to give up the habit. Rates were higher in remote areas and for those of the lowest socioeconomic status. People who have never smoked (10.6 million) and ex-smokers (4.4 million) far exceed the number of smokers (3.3 million) aged 14 years and older. The age group 12–17 years was the only one in which girls were more likely than boys to smoke daily (3.2% versus 1.8%) (AIHW, 2011).

For women, unique health concerns arise: second-hand effects from smoking have been demonstrated, especially on the foetus during pregnancy, leading to increased risks such as low birth weight, spontaneous abortion, perinatal mortality and sudden infant death.

Cannabis

Cannabis is the general name given to the psychoactive substances found in the marijuana plant, *cannabis sativa*, the main active constituent being delta-9-tetra-hydrocannabinol (THC). The psychoactive properties are mostly of a CNS depressant nature; in high doses hallucinogenic properties can occur, affecting some of the senses—for example, distortion of lights, sounds and music—but full hallucinations are rare (NSW Health, 2008a).

TABLE 5.3 Guide for the management of nicotine-dependent inpatients

1. IDENTIFY EVERY TOBACCO USER ON ADMISSION

Use Substance Use History form, or include smoking status on existing admission forms
Ex-smokers—encourage continuing abstinence
Daily/occasional smokers—follow steps 2 to 5

2. MANAGE INPATIENT NICOTINE DEPENDENCE

Inform people of the policy for smoking in your facility and specify contra-indications to their treatment regimen if they leave the ward/facility to smoke
Discuss options for management of nicotine dependence while in hospital, such as:

- abstinence
- abstinence supported by nicotine replacement therapy (NRT), unless contra-indicated
- smoking offsite in outdoor designated areas, if available

If a person has a history of mental health problems, consult treating clinician—adjustment of medications may be necessary.

3. PRESCRIBE NICOTINE THERAPY

Arrange prescriptions for NRT (with the person's consent)
Record:

- type (patch/inhaler/gum) and dose on medication chart
- 'Nicotine dependent' in person's notes

4. MONITOR PERSON'S WITHDRAWAL SYMPTOMS

If a person is still experiencing withdrawal symptoms:

- review NRT dose/product (person may benefit from combination therapy)

5. DISCHARGE

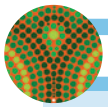
Ask all smokers: 'Do you plan to smoke when you go home?'
'Yes.'

- Encourage future quit attempt: 'The best thing you can do for your health is to stop smoking. When you're ready, phone the Quitline or talk to your doctor.'

'No.'

- Arrange 3-day post-discharge NRT
- Include treatment summary in discharge plan
- Advise person to seek cessation support from GP/pharmacist/Quitline 137 848
- Provide brochures on smoking cessation.

Source: Adapted from NSW Health (2015). *Managing nicotine dependence: A guide for NSW Health staff.* Reproduced with the permission of NSW Health.



TRANSLATION TO PRACTICE Evidence-based practice for smoking cessation in hospital

Despite its well-publicised deleterious health effects, and legally restricted access to cigarettes, tobacco was still responsible for the greatest disease burden in Australia in 2003 (7.8% of the total burden) (AIHW, 2007). Smoking has been banned from shops, malls, hospitals, office buildings and restaurants. Admission to hospital provides an excellent opportunity for nurses to assist people to stop smoking. It may be easier to quit in an environment where smoking is restricted or prohibited, and those facing the risks associated with surgery may be more open to cessation efforts.

In a review of the literature Rigotti et al. (2005) found that high-intensity behavioural interventions which included at least 1 month of follow-up contact were effective in helping people in hospital care to quit smoking; healthcare professionals, especially nurses, can be very effective in smoking cessation efforts, and evidence-based brief intervention guidelines are available (NSW Health, 2005).

Silagy et al. (2005) found that nicotine replacement therapy (NRT), available as chewing gum, skin patches, inhalers and lozenges, increases the likelihood of success in quitting smoking, with or without additional counselling; its purpose is to reduce symptoms of withdrawal from tobacco products by replacing nicotine in the blood. Rice and Stead (2005) were of the same opinion and stressed the potential benefits of nursing interventions. Effective strategies, which nurses should incorporate as part of their standard practice, include asking people about their tobacco use, counselling those who want to quit, encouraging and reinforcing cessation efforts, and early follow-up with those who succeed.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. You are caring for a 55-year-old man, recently hospitalised for acute angina, who wants information about the best way to stop smoking. What would you do?
2. Why do you think nurses and other healthcare professionals should (or should not) quit smoking?
3. A 12-year-old girl in your care tells you she has occasionally smoked cigarettes and believes it increases her popularity among older friends. She admits knowing smoking is supposed to be bad for you but doesn't see the harm in a few cigarettes every day. How would you respond?

THC activates specific cannabinoid receptors in the brain. Evidence indicates effects similar to those of opioids and cocaine, producing a pleasurable sensation, probably through release of endogenous opioids and then dopamine (Kneisl & Trigoboff, 2013). Adverse effects include paranoia, increased appetite, anxiety, depression and sedation. Cannabis can cause drug-induced psychosis (but usually only in people with a genetic predisposition or susceptibility); this can be difficult to distinguish from other psychotic illnesses, such as schizophrenia. Marijuana use can also trigger relapse in people living with schizophrenia. Drug-induced psychoses tend to resolve relatively quickly (within a week) with cessation of drug use (NSW Health, 2008a).

Marijuana/cannabis is the illicit drug most used across all Australian age groups with about 1.9 million people having used the drug in the previous 12 months, an increase from 1.6 million in 2007 (AIHW, 2011). A World Health Organization (WHO) study found cannabis use in the United States and New Zealand (both 42%) to be far higher than in any other country surveyed (Degenhardt et al., 2008).

Cannabis withdrawal is marked by three or more of the following:

- sleep disturbance (insomnia and vivid dreams)
- reduced appetite or weight loss
- irritability, anger or aggression
- anxiety or nervousness
- restlessness
- depressed mood
- at least one of abdominal pain, shakiness, sweating, fever, chills or headaches (APA, 2013).

The major part of the management approach for cannabis withdrawal should be supportive counselling, provision of accurate information about signs and symptoms, and relapse prevention planning. Medications may be appropriate for some people; most commonly prescribed are benzodiazepines, to be used with discretion and in the short term.

Alcohol

Worldwide, an astonishing 3.3 million people died in 2012 (5.9% of all deaths) from the harmful use of alcohol (WHO, 2014). **Alcohol** is a CNS depressant and acts on the main inhibitory neurotransmitter, GABA. It is the most commonly used drug in Australia and New Zealand because of its broad social acceptance and entrenchment in cultural norms; however, it can cause a wide range of harm (Shield, Parry & Rehm, 2013).

In 2010, most people aged 14 years or more (80.5%) had consumed a full serve of alcohol (AIHW, 2011) and in 2011–2012 nearly one in five adults drank more than two standard drinks per day, men being three times as likely as females to drink at risky levels (AIHW, 2014). The main psychoactive ingredient in alcohol is ethyl alcohol, which contributes to changes in mood, thinking and behaviour.

Alcohol is rapidly absorbed from the small bowel (about 80%) and stomach (about 20%). It reaches the brain within 5 minutes of ingestion, with blood plasma concentrations peaking at between 30 and 90 minutes. Alcohol readily crosses the blood–brain barrier and placenta; 95% is metabolised by the liver into carbon dioxide and water, and 1–5% is excreted unchanged in saliva, urine, faeces and sweat (National Centre for Education and Training on Addiction (NCETA), 2004). Generally, one standard alcoholic drink takes approximately 1 hour to metabolise; low doses induce a decrease in inhibitions, a feeling of comfort and relaxation, flushing of the skin, and mild impairment of thinking and judgment. With rising blood alcohol levels, speech becomes slurred and is accompanied by increasing loss of motor control, including development of ataxia. At higher levels

memory is affected, with potential for blackouts; the person becomes stuporous and unable to be aroused, and coma and death can occur (NSW Health, 2008a). Blood alcohol levels (BALs) are highly predictive of CNS effects: euphoria, reduced inhibitions, impaired judgment and increased confidence are seen at 0.05%; toxic levels in excess of 0.5% can cause coma, respiratory depression, peripheral collapse and death (Kneisl & Trigoboff, 2013).

The 2010 National Drug Strategy Household Survey (AIHW, 2011) showed that one in five Australians aged 14 or older, far more males than females, were consuming alcohol at a level that put them at risk of short- or long-term harm from drinking.

Moderate use of certain types of alcohol can have positive physiological effects in reducing coronary artery disease and protecting against stroke, but in excess it can diminish the ability to function and lead ultimately to life-threatening conditions. Alcohol abuse can cause severe neurological and psychiatric disorders, and damage to the liver can progress from fatty liver to other diseases such as hepatitis; chronic alcoholism is the major cause of fatal cirrhosis. Damaging effects on many other systems include myocardial disease, erosive gastritis, acute and chronic pancreatitis, sexual dysfunction and an increased risk of breast cancer.

Malnutrition, another serious complication, can result in neurological impairments; in particular, thiamine (B_1) deficiency is thought to cause the severe cognitive impairment which is a principal feature of **Wernicke's encephalopathy** and **Korsakoff's psychosis** observed in people with chronic alcohol dependence (Stuart & Laraia, 2005). Sometimes considered to be two distinctive disorders, these are actually different phases of the same disease, commonly called Wernicke–Korsakoff syndrome: Wernicke's encephalopathy indicates the 'acute' stage of the illness and Korsakoff's psychosis indicates the 'chronic' stage.

Although alcohol is a CNS depressant, it actually disrupts and alters the sleep cycle, decreasing sleep quality, intensifying obstructive apnoea and reducing total sleeping time. Heavy drinkers have a higher mortality rate and many fatalities result from alcohol-related accidents.

Chronic consumption of alcohol produces tolerance. Cross-tolerance occurs when alcohol and other CNS depressants such as benzodiazepines and opioids are used in combination; taken together they have a mutually enhancing effect, thereby increasing the possibility of respiratory depression and death (Australian Government Department of Health and Ageing, 2009).

WITHDRAWAL Abrupt withdrawal of alcohol causes overexcitation of the brain because previously inhibited receptors are no longer inhibited (Carlson et al., 2012); this manifests clinically as anxiety, tachycardia, hypertension, diaphoresis, nausea, vomiting, tremors, sleeplessness and irritability.

The onset of alcohol withdrawal usually occurs 6–24 hours after the last drink. Heavily dependent people can begin to develop signs and symptoms of withdrawal even before the blood alcohol level reaches zero. Consumption of other CNS depressants—for example, benzodiazepines—may delay the emergence of the syndrome.

Mild withdrawal syndrome episodes generally settle after 24–48 hours, and the majority of uncomplicated cases within 72 hours; occasionally, more severe cases may continue for up to 10 days (NSW Health, 2008a).

Complicated alcohol withdrawal carries a significant risk of further morbidity and is potentially life threatening. Proper assessment and monitoring procedures, in combination with early and adequate institution of the necessary medical treatment, should prevent the development of complicated withdrawal syndrome in a hospital setting.

Withdrawal scales provide an objective method of assessing a person at risk, and the course of the syndrome. The most validated and widely used scale is the Clinical Institute Withdrawal Assessment for Alcohol—revised version (CIWA-AR) (Sullivan et al., 1989).

- Withdrawal scales do not diagnose withdrawal; they monitor the severity of the range of signs and symptoms of those experiencing or at risk of developing alcohol withdrawal.
- Start the monitoring as soon as possible after admission to hospital.
- Re-evaluate regularly.
- Explain why monitoring is necessary and what might be expected.
- Report promptly any changes in the person's condition.
- Don't assume that the condition is just alcohol withdrawal; assess for other potential factors and if concerned seek further advice.
- Supportive care alone is often effective in minor alcohol withdrawal.

Risk factors for severe alcohol withdrawal include:

- previous episodes of alcohol withdrawal
- previous seizures during withdrawal
- history of delirium tremens
- CIWA-AR score above 10
- advanced age
- co-morbidities
- detectable blood alcohol level on admission
- high daily intake of alcohol
- abnormal liver function
- prior benzodiazepine use
- male sex
- previous blackouts
- concomitant use of CNS depressants or illicit substances (Carlson et al., 2012; Maldonado et al., 2014).

TREATMENT

- Early and adequate pharmacological intervention can prevent development of a complicated alcohol withdrawal syndrome carrying significant risk of added morbidity.
- Caution is required in treating elderly or respiratorily compromised people.
- Diazepam, a long-acting benzodiazepine, is widely recognised as the pharmaceutical agent of choice. Contraindications to diazepam include respiratory failure, significant liver impairment, and possible head injury or cerebrovascular accident. In these situations, specialist consultation is essential (NSW Health, 2008c).

Best practice principles of nursing care for treatment are outlined in Box 5.1.

Screening in primary care settings, such as general practices, community health centres and multipurpose health facilities, for identification of people drinking at risky or harmful

BOX 5.1 Principles of nursing care in relation to alcohol

- Routinely take an alcohol use history from all admissions.
- Consider the possibility of alcohol withdrawal if clinical symptoms are occurring in a patient, as some patients may minimise how much alcohol they consume when initially asked.
- Identify those at risk of alcohol withdrawal (for men, daily consumption of 80–100 grams or 8–10 standard drinks, and for women 80 grams or 8 standard drinks).
- Monitor those at risk by means of an alcohol withdrawal scale.
- Start monitoring for alcohol withdrawal as soon as possible; if the person is intoxicated on admission, as soon as the BAL goes below 0.10%.
- Seek medical advice and, if treatment for withdrawal is indicated, commence it early.
- Clearly explain all aspects of care.
- Provide a quiet, softly lit, pleasant environment.
- Give thiamine.
- Monitor hydration.

levels, or who may be dependent, is a useful initiative; early intervention prevents delay until irreparable health damage or psychosocial consequences have occurred (Australian Government Department of Health and Ageing, 2009). A range of validated screening instruments/questionnaires available for use includes the AUDIT, the MAST and the CAGE.

Biological markers useful in assessing alcohol consumption include serum GGT (gamma-glutamyl transferase), AST (aspartate aminotransferase), ALT (alanine aminotransferase), HDL-cholesterol, uric acid, mean corpuscular volume and carbohydrate-deficient transferrin (CDT). Serum GGT, a liver enzyme, is the most useful of the current available tests (Australian Government Department of Health and Ageing, 2009).

Benzodiazepines

Benzodiazepines, minor tranquillisers belonging to the sedative–hypnotic group of drugs, have a CNS depressant effect through action at the GABA receptor sites. Widely prescribed for sedation such as premedication for hospital procedures for their muscle relaxant and amnestic properties, for relief of anxiety (anxiolytic) and as anticonvulsants, benzodiazepines also negatively affect some areas of performance, including memory, motor skills and reaction times (NCETA, 2004), and have been linked to increased falls in the elderly. Often referred to as 'benzos', they are also widely used for illicit or non-prescribed purposes. Benzodiazepines were first marketed in 1959 as a safe alternative to barbiturates, and in the 1970s and 1980s became the most commonly prescribed class of drugs. With increasing doses the level of sedation progresses, extending to unconsciousness. The respiratory depression caused by benzodiazepines is minimal, but combination with other **central nervous system depressants** (e.g. alcohol and opioids) may have life-threatening consequences.

WITHDRAWAL The rate of developing dependence varies: after 3 to 12 months' usage, 10% to 20% of users will become dependent, rising to 20% to 45% when duration of use exceeds 12 months. Heavy users can experience such medical complications of withdrawal as seizures, hallucinations and delirium if dosage is reduced too rapidly; gradual reduction is therefore recommended (NSW Health, 2008c). Depending on the half-life of the particular benzodiazepine, onset of withdrawal occurs 2 to 5 days after stopping or significantly reducing the usual intake; the peak is usually reached on days 7 to 10, and most signs and symptoms usually abate by the end of the second or third week (NSW Health, 2008c). Table 5.4 gives the absorption rates and half-lives of common benzodiazepines.

CONSIDERATION FOR PRACTICE

People in hospital for other reasons may undergo withdrawal from regular, long-term use of even low doses of benzodiazepines—particularly the elderly, who may develop delirium.

- Usage even at low doses should not be stopped abruptly because of the risk, particularly for the sick and the elderly, of precipitating withdrawal.
- Routinely take a history of benzodiazepine use at admission, or as soon as practicable.
- Heavy users or polydrug users should be stabilised before admission on a long-acting preparation (preferably diazepam) at a dose about 40–50% of their regular intake (or 80 mg/day, whichever is lower) (NSW Health, 2008c).
- A hospital admission, during an acute illness, may not be the appropriate time to undertake an elective withdrawal; this may best be managed in an outpatient setting.

Psychostimulants

Psychostimulants, or CNS stimulants, comprising a diverse group of natural and synthetic drugs with a wide range of actions and effects, include:

- amphetamine-type substances, including amphetamine, methamphetamine and dexamphetamine (speed, crystal, crystal meth, ice)
- synthetic amphetamine derivatives, such as methylenedioxymethamphetamine (MDMA—ecstasy), paramethoxyamphetamine (PMA), methylenedioxy-amphetamine (MDA)
- methylphenidates—Ritalin®
- cocaine
- phentermine (Duramine®) and diethylpropion (Tenuate®), prescribed appetite suppressants
- ephedrine and pseudoephedrine, contained in various prescribed cold and flu preparations (adapted from Hulse, White & Cape, 2002).

Psychostimulants are structurally related to the naturally occurring neurotransmitters dopamine, serotonin, adrenaline and noradrenaline. CNS or psychoactive effects include euphoria, increased alertness, increased talkativeness, sense of wellbeing, increased energy and confidence, improved mental and physical performance, loss of appetite and insomnia. Physiological effects include increased blood pressure, tachycardia or irregular heartbeat and increased temperature (Hulse et al., 2002).

TABLE 5.4 Absorption rates, half-lives and equivalent daily doses of common benzodiazepines*

GENERIC NAME	TRADE NAME	TIME TO PEAK CONCENTRATION	ELIMINATION HALF-LIFE†	EQUIVALENT DOSE‡
Diazepam	Antenex Ducene Ranzepam Valium Valpam	30–90 min	Biphasic: rapid phase half-life, 3 hours; elimination half-life, 20–48 hours	5 mg
Alprazolam	Alprax Xanax Kalma	1 hour	6–25 hours	0.5–1.0 mg
Bromazepam	Lexotan	0.5–4 hours	20 hours	3–6 mg
Clobazam	Frisium	1–4 hours	17–49 hours	10 mg
Clonazepam	Paxam Rivotril	2–3 hours	22–54 hours	0.5 mg
Flunitrazepam	Hypnodorm	1–2 hours	20–30 hours	1–2 mg
Lorazepam	Ativan	2 hours	12–16 hours	1 mg
Nitrazepam	Alodorm Mogadon	2 hours	16–48 hours	2.5–5 mg
Oxazepam	Alepam Murelax Serepax	2–3 hours	4–15 hours	15–30 mg
Temazepam	Euhypnos Normison Temaze Temtabs	30–60 min after tablets, 2 hours after capsules	5–15 hours	10–20 mg
Triazolam	Halcion	1–3 hours	Biphasic: rapid phase half-life, 2.5–3.5 hours; elimination half-life, 6–9 hours	0.25 mg
Zolpidem (not a benzodiazepine)	Stilnox	0.5–3 hours	2.5 hours	Not known

* Based on manufacturer's product information.

† Elimination half-life: time for the plasma drug concentration to decrease by 50%.

‡ Equivalent dose: approximate dose equivalent to diazepam 5 mg.

Source: NSW Health (2008c). *Drug and alcohol withdrawal: Clinical practice guidelines: NSW*. Sydney: NSW Health. © New South Wales Ministry of Health for and on behalf of the Crown in right of the State of New South Wales.

Amphetamines, MDMA (ecstasy) and cocaine are types of psychostimulants more commonly available in Australia.

Amphetamines

The term **'amphetamine'** includes three types: amphetamine, dexamphetamine and methamphetamine and it is available in five forms:

1. *Powder* ('speed', 'goey', 'whiz'): texture ranges from fine to crystallised or coarse powder, coloured white, yellow, brown, orange or pink. Powder is usually snorted or injected but is sometimes mixed in drinks.
2. *Pills*: methamphetamine often appears in pill form, frequently sold as MDMA (ecstasy); manufacturers may mix it with ketamine or gamma hydroxybutyrate (GHB) to produce MDMA-like hallucinogens (McKetin, McLaren & Kelly, 2005).
3. *Base* ('paste', 'pure', 'wax'): oily, waxy or pasty substance with a sticky, damp or gluggy texture. Usually swallowed, smoked or snorted, but may require mixing with a dry powder; can be injected if heated, but has the potential to damage veins.
4. *Liquid* ('ox blood'): high-purity freebase, more pure than speed and normally swallowed or smoked.
5. *Crystal methamphetamine* ('crystal', 'crystal meth', 'ice'): appears as crystals or coarse crystalline powder, white or translucent in colour. The most pure and potent form of methamphetamine, it is usually smoked in a glass pipe or injected, but can be snorted or swallowed. Ice use in Australia, as a proportion of stimulant use, has more than doubled from 22% in 2010 to 50% in 2013 (AIHW, 2015).

MDMA

MDMA (methylene-dioxymethamphetamine), known as 'ecstasy', 'eccies', 'dingers', appears most often in tablet form, but it can be crushed then snorted, and occasionally injected.

Cocaine

Extracted from the coca leaf and imported into Australia, **cocaine** is a white, odourless, crystalline powder and is snorted, injected or swallowed. Crack cocaine, which is smoked, has been reported in Australia but is rare. Cocaine is less available and more expensive than amphetamines.

Stimulant drugs are used by a diverse group of people from a wide variety of socio-demographic backgrounds. Long-distance transport drivers and shift workers use them for their wakefulness-promoting effects, nightclub and dance/music festival patrons for increased energy, and others for potential weight loss, sexual enhancement, and to self-medicate otherwise untreated attention deficit and hyperactivity disorders. The range of users extends from younger age groups experimenting with drugs to polydrug users and long-term injectors.

Drug users will often present to hospitals and other health-care facilities for matters unrelated to their drug use, which they may be reluctant to disclose. It is important to note that they may not regard their drug use as a problem, so it is necessary to take a good history and interact in a non-judgmental way.

Psychostimulant intoxication

Because of burgeoning use in Australia, presentations to general hospital emergency departments and psychiatric emergency centres by people experiencing methamphetamine intoxication have increased; symptoms include agitation, aggression, paranoia, bruxism (teeth grinding and jaw clenching), psychosis and confusion. The primary goal of treatment is to ensure person and staff safety. Sedation is the principal method, the main medications employed being benzodiazepines, anti-psychotics, and even short-acting anaesthetic agents.

WITHDRAWAL Psychostimulant drug withdrawal is not a medically dangerous condition, and generally involves a self-limiting 2- to 5-day period of lethargy, dysphoria, irritability, anxiety, somnolence and increased appetite. The extent of signs and symptoms and the duration of the withdrawal syndrome will be influenced by which drug is involved, the dose, duration of use, and health and personality of the user. Withdrawal typically occurs in three phases, as illustrated in Table 5.5.

TREATMENT Treatment is mostly supportive: understanding, patience, encouragement and positive affirmation. Medical treatment might involve judicious short-term use of benzodiazepines to alleviate some of the anxiety and agitation and help with disturbed sleep. If protracted low mood or more severe depression exists or psychotic features do not resolve within a couple of days, further assessment will be required. Medical complications, usually resulting from injecting drug use, may need further investigation and treatment. A primary aim of health services contact is to educate people about the risks of continued use, discuss harm minimisation options, offer referral or arrange engagement in some sort of relapse prevention support. The medical and psychiatric complications of stimulant use are listed in Table 5.6.

TABLE 5.5 Phases of psychostimulant withdrawal

PHASE	TIME SINCE LAST STIMULANT USE	COMMON SIGNS AND SYMPTOMS
Crash	*Amphetamines:* typically commences 12–24 hours after last amphetamine use, and subsides by days 2–4. *Cocaine:* occurs within hours of last use, with short duration (up to 48 hours). Some people do not report a significant crash on stopping cocaine.	Exhaustion, fatigue Sleep disturbances (typically increased sleep, although insomnia or restless sleep may occur) Mood disturbances—typically flat mood or dysphoria: may be associated with anxiety or agitation Low cravings Generalised aches and pains
Withdrawal	*Amphetamines:* typically commences 2–4 days after last use, peaks in severity over 7–10 days, and then subsides over 2–4 weeks. *Cocaine:* typically commences 1–2 days after last use, peaks in severity over 4–7 days, then subsides over 1–2 weeks.	Strong cravings Fluctuating mood and energy levels, alternating between irritability, restlessness, anxiety and agitation Fatigue, lacking energy, anhedonia Disturbed sleep, including vivid dreams, insomnia General aches and pains, headaches Muscle tension Increased appetite Poor concentration and attention Disturbances of thought (e.g. paranoid ideation, strange beliefs) and perception (misperceptions, hallucinations) can re-emerge during withdrawal phase after having been masked during crash
Extinction	Weeks to months	Gradual resumption of normal mood with episodic fluctuations in mood and energy levels, alternating between irritability, restlessness, anxiety, agitation and fatigue Fatigue, lacking energy and anhedonia Episodic cravings Disturbed sleep

Source: NSW Health (2008a). *Clinical guidelines for nursing and midwifery practice in NSW: Identifying and responding to drug and alcohol issues*. Sydney: NSW Health. © New South Wales Ministry of Health for and on behalf of the Crown in right of the State of New South Wales.

TABLE 5.6 Medical and psychiatric complications of stimulant use

SYSTEM	COMPLICATIONS
Cardiovascular	Arrhythmias: tachycardia, bradycardia, ventricular tachycardia. Hypertension: may lead to cerebrovascular accidents. Spasm of arteries: leading to myocardial infarcts or cerebrovascular accidents. (Myocardial infarcts can occur during first weeks of withdrawal.) Cardiomyopathy and congestive heart failure.
Neurological	Seizures: clonic convulsions. Cerebrovascular accident: including brain haemorrhages, infarcts and ischaemic episodes. Neuropsychological changes: deficits in attention, concentration, memory and new learning skills. Movement disorders: e.g. tics, disturbed gait, stereotyped repetitive movements, choreiform movements.
Psychiatric	May mimic any psychiatric disorder. More commonly: • depression, with changes in mood and affect, sleep, activity • paranoia, ranging from hypervigilance to paranoid psychosis • anxiety and aggression, ranging from irritability and agitation to panic attacks or violence (more common in amphetamine and methamphetamine users) • delirium, with clouding of consciousness, disorientation, confusion • psychosis, characterised by paranoia and anxiety, impaired reality testing with loss of insight and delusions (e.g. ideas of reference, persecutory delusions) and perceptual disturbances (including misperceptions and visual, auditory or tactile (formication) hallucinations).
Respiratory	Smoking of cocaine and amphetamines can result in chronic lung damage (including pneumonia, pulmonary oedema, bronchitis).
Sexuality	Short-term stimulant use is often associated with increased sexual drive and performance. However, chronic use can lead to difficulties achieving orgasm, altered menstruation (oligomenorrhea, amenorrhoea) and galactorrhoea in women, and reduced libido, impotence and gynaecomastia in men.
Hyperpyrexia	Extremely elevated body temperature, which can contribute to seizures, cardiac arrhythmias and death. Rhabdomyolysis can also occur, resulting in acute renal and hepatic failure, disseminated intravascular coagulation and death.
Pregnancy	Stimulant use during pregnancy is associated with higher rates of obstetric complications (spontaneous abortion, miscarriage and placental abruption), and harm to the foetus.
Other	Weight loss (chronic loss of appetite and increased metabolism). Skin lesions and abscesses, due to adulterants, particularly in injectors.

Source: NSW Health (2008c). *Drug and alcohol withdrawal: Clinical practice guidelines: NSW.* Sydney: NSW Health. © New South Wales Ministry of Health for and on behalf of the Crown in right of the State of New South Wales.

Although over the past decade the number of psychostimulant users in Australia and worldwide has increased, not many specialist treatment programs are available; withdrawal management and follow-up support are the mainstays of treatment. Interventions such as relapse prevention counselling, including coping with cravings and learning refusal skills (Lee et al., 2007), self-help groups and harm-reduction programs should be encouraged. Motivational interviewing and cognitive behavioural therapy are the most empirically supported approaches to drug counselling, and a validated counselling manual has been developed for regular amphetamine users (Baker et al., 2003).

Opioids

Opioids such as morphine (Kapanol, Anamorph, MS Contin), pethidine, codeine, methadone (Physeptone, Biodone), hydromorphone (Dilaudid, Jurnista) buprenorphine (Subutex, Suboxone), oxycodone (Oxycontin, Endone), fentanyl, and heroin (diacetylmorphine) are narcotic analgesics. While opioids have many therapeutic uses, they also have abuse potential. Heroin has historically been the primary illicitly used opioid, but over the past decade its availability in Australia (and worldwide) has fluctuated, and abuse of pharmaceutical opioids has significantly increased. Narcotic analgesics are a pain reliever derived from natural or synthetic opioids; interacting with the endogenous opioid receptors, they produce a depressant effect on the CNS causing drowsiness, reduced pain perception and euphoria, the last effect being highly reinforcing.

Prolonged use of opioid drugs leads to the development of tolerance—progressing to euphoria, respiratory depression, analgesia and nausea—and physical dependence. Tolerance to the analgesic effects of opioids manifests itself in lowering the pain threshold: apparently mild pain may be perceived as more severe, which may inadvertently be misinterpreted by the treating nurse as drug-seeking behaviour rather than inadequately relieved pain.

Withdrawal

Unlike alcohol withdrawal, the syndrome associated with cessation of opioid use is not medically serious except in pregnant women. Opioid withdrawal is potentially life threatening for the foetus, as is withdrawal precipitated by naltrexone (an

opioid antagonist). Opioid withdrawal can be viewed as a flu-like syndrome with major and minor symptoms:

Minor

- restlessness/agitation
- lacrimation
- rhinorrhoea
- perspiration/hot and cold flushes
- yawning
- insomnia
- mydriasis (dilated pupils)
- piloerection (goose flesh).

Major

- muscle aches
- joint pain/back pain/pain in long bones
- abdominal cramps
- nausea and vomiting
- diarrhoea
- drug-seeking behaviour.

Psychological/emotional aspects

- emotional and teary
- scattered thoughts
- drug cravings.

Treatment

Opioid-dependent people are often admitted to hospitals and develop unplanned withdrawal. While the syndrome is not medically dangerous, the symptoms can cause considerable discomfort and may lead to resumption of use to avoid or abate the symptoms. Early discharge and thus poor intervention outcomes are probable results.

An opioid withdrawal scale is helpful in monitoring the progress of the syndrome (Wesson & Ling, 2003). Another method of assessment is that withdrawal signs and symptoms will usually begin around the time of the user's expected next dose. The short half-life of heroin produces onset at 6 to 24 hours after last use, and duration is 5 to 10 days. Withdrawal from methadone, which has a longer half-life, occurs 24 to 48 hours after the last dose and lasts for 10 to 20 days.

The requirement in Australia is for a medical practitioner to seek specific approval to prescribe or supply a drug of addiction (listed in Schedule 8 of the Poisons Standard by the Australian Government Department of Health Therapeutic Goods Administration, 2015) to anyone who is known to be, or suspected of being, a drug-dependent person. This does not apply in hospital settings, where these drugs may, without approval, be prescribed for such people for up to 14 days. The policy provides for the management of opioid-dependent people who have been admitted to a hospital (public or private), whether or not they are on an opioid substitution program.

Methadone maintenance is the recommended treatment for pregnant women who are opioid dependent, and breastfeeding is encouraged; linking with specialist antenatal services is also important (NSW Health, 2008a). If anyone on an opioid substitution program is admitted to a hospital, the program should be continued unless medically contra-indicated. Details of the type of substitution medication, current dose, when last administered, and whether any 'take-away' doses have been dispensed all need to be confirmed with the person's prescriber, clinic, dispensing pharmacy, or the relevant state regulatory body, and the prescriber, clinic or pharmacy must be notified on discharge.

People who have been treated for opioid withdrawal should be advised of the increased risk of overdose if they resume using, because they have lost their tolerance; even lesser amounts than those previously used could result in overdose.

Treatment services suitable for an opioid-dependent person include detoxification, residential rehabilitation, outpatient counselling, opioid substitution treatment with methadone or buprenorphine, 12-step self-help programs and naltrexone maintenance. Because of the unique pharmaceutical properties of buprenorphine, which is the principal treatment, specialist advice is recommended for the management of opioid withdrawal undertaken in a general hospital setting. Different types of treatment are more or less appropriate at different stages of dependence; referring the person to a specialist drug and alcohol treatment service may be appropriate to aid exploration of these options.

Hallucinogens

Hallucinogens work on the brain to cause hallucinations, which can affect any of the senses—visual (sight), auditory (hearing), olfactory (smell), gustatory (taste) and tactile (touch)—and distort thoughts and moods. Sometimes called 'psychedelic drugs', they include naturally occurring and synthetic (man-made) compounds.

Hallucinogenic drugs include:

- d-lysergic acid diethylamide (LSD)—'trips', 'acid'
- psilocybin—'magic mushrooms'
- anticholinergics—datura or angel's trumpet
- dimethyltryptamine (DMT)
- phenethylamines—mescaline
- phencyclidine (PCP)—angel dust.

Some drugs, such as cannabis and ecstasy, can cause hallucinogen-like effects in high doses; normally taken orally, on an irregular basis.

Harm (especially behavioural and psychiatric consequences) is more likely to arise from acute drug effects than regular or dependent patterns of use. They are typically used experimentally, occasionally or irregularly, so withdrawal treatment is not required; few people present to treatment centres because of hallucinogen use.

Immediate effects can include:

- disturbances of the senses (sight, hearing, touch, taste or smell); these can be full hallucinations (i.e. have no basis in reality) or illusions (i.e. distortions of reality)
- a sense of increased clarity or sharpness
- altered perceptions (space becomes distorted, changed sense of time)
- changed and intense thoughts
- emotional swings
- dizziness
- weakness
- numbness
- nausea
- dilated pupils
- raised blood pressure, pulse and body temperature
- tremor
- ataxia

- agitation
- anxiety/panic attacks
- psychosis.

Death from overdose of hallucinogens is rare.

One of the few known long-term effects of hallucinogen use is 'flashbacks'—times when feelings similar to those caused by the drug arise intermittently some days, weeks or years after last use. Flashbacks can be precipitated by other drug use, stress, anxiety and fatigue. Other effects include difficulties with memory and concentration, and precipitation or worsening of mental health problems (NSW Health, 2008b).

Treatment

Management of hallucinogen intoxication involves ensuring the safety of the person and others; disturbed thinking and moods may result in agitation and erratic behaviour. Helpful interpersonal techniques include reality orientation, reassurance, and a calm and confident manner. Medical management of acute reaction to hallucinogen ingestion usually includes administration of benzodiazepines, clonidine and antipsychotic medications (Hulse, White & Cape, 2002).

Solvents

Solvents produce a CNS depressant effect: they comprise a range of products producing vapours to be inhaled through the nose or mouth, which may cause feelings of intoxication and lead to an altered state of consciousness. They can be sprayed into a plastic bag ('bagging') or soaked on a cloth or sleeve ('huffing'), or inhaled directly from the container or a drink bottle ('sniffing'). They include gases (e.g. nitrous oxide) and highly volatile compounds or mixtures (petrol, chrome-based paints, glues, aerosol spray cans, butane gas and paint thinners).

Solvents are rapidly absorbed from the lungs, so small amounts quickly take effect. Brain damage or 'sudden sniffing death' can occur the first, tenth or hundredth time of use; another peril is the wide assortment of organic solvents available to and potentially inhaled by young children.

Effects usually clear within a few hours. A sustained effect can be achieved by repeated use; high doses can result in coma and death. Toxicity varies greatly, depending on the substance. Generally, signs include cardiac arrhythmia, hypoxia and neurological impairment. Solvent and alcohol intoxication are similar; short-term effects include euphoria, excitation, disinhibition, slurred speech, ataxia, drowsiness, dizziness, nausea, vomiting, headaches, disorientation and sensory distortions.

Withdrawal

Withdrawal symptoms are not common, and are generally mild:

- anxiety
- depression
- anorexia
- irritation
- tremors
- headache
- nausea
- dizziness.

Confusion and hallucinations can occur after chronic solvent use (NSW Health, 2008b).

OTHER DRUGS

Ketamine

Ketamine (ketamine hydrochloride) is a CNS depressant, best described as a dissociative anaesthetic agent. It is one of the newer drugs to migrate into the illicit-drug-using world, and was developed in the 1970s as a medical anaesthetic for both humans and animals. Illicitly, ketamine can be used as the primary drug, but is also mixed with drugs such as methamphetamine and ecstasy to mimic or supplement their effects. It is pharmaceutically produced in liquid form, but converted to powder and tablet forms for illicit purposes. Commonly swallowed, snorted, smoked or injected, it has a rapid onset but short duration of action (1–6 hours). The half-life is 3 hours (Hulse, White & Cape, 2002) and it can be 24 to 48 hours before the user feels completely 'normal' again.

Ketamine's effects appear to be subjective, depending on the user and the setting. Rarely in Australia do people need treatment because of ketamine, which tends to be used experimentally or socially. Whether it will become a bigger problem is not clear.

At low doses ketamine can produce a state that has some stimulant effects and other sedating effects resembling alcohol intoxication.

At higher doses the effects can include:

- drowsiness
- increased perception
- apathy
- dissociative 'out of body' sensations (flying or floating, detachment from the immediate environment, near-death experiences)
- feelings of paralysis
- analgesia or a reduced response to pain
- hallucinations
- disorganised thoughts
- personality changes
- euphoria
- convulsions
- confusion and disorientation
- risk of respiratory failure
- loss of consciousness.

Larger doses can induce emesis, seizures and respiratory depression. Doses of 1 gram or more can cause death. Ketamine is extremely dangerous when combined with other CNS depressants such as alcohol, benzodiazepines or GHB because of the potentiating effects, whereby each drug increases the effect of the other drug.

Effects of longer-term use can include:

- weight loss and anorexia
- possible memory and concentration difficulties
- mental health disturbances (e.g. anxiety)
- development of tolerance
- possible dependence.

Withdrawal

A withdrawal syndrome involving fear, tremors, facial twitches and craving can occur after cessation of long-term daily use (NSW Health, 2008c).

Gamma hydroxybutyrate (GHB)

Gamma hydroxybutyrate is another CNS depressant and, although it has some mixed properties, it is essentially a dissociative anaesthetic agent; another of the newer drugs diverted to illicit use. Occurring naturally in the brain, GHB was first manufactured and studied in the 1960s, and used in several countries as a general anaesthetic. However, it was withdrawn from use in most countries, including Australia, because of unwanted side effects such as vomiting and seizures.

GHB has a narrow dosage range between stages of a therapeutic effect, the desired effect if used illicitly, and overdose. Like most drugs, it can have a potentiating effect in combination with drugs of the same or similar class. GHB comes in the form of liquid, capsules, powder or crystals.

The effects of GHB are experienced usually within 15 minutes of use, and last for approximately 3 hours.

Effects of lower amounts may include:

- a sense of wellbeing
- relaxation
- drowsiness
- induced sleep
- nausea
- increased confidence, reduced inhibitions
- dizziness
- headache
- increased sociability
- enhanced sense of touch.

There is a narrow threshold between the desired effect and coma. Effects of an overdose can include:

- confusion
- agitation
- extreme drowsiness/grogginess
- hallucinations
- difficulty focusing eyes
- vomiting
- stiffening of muscles
- impaired movement and speech
- convulsions
- unconsciousness/abrupt short-term coma
- respiratory collapse
- amnesia (afterwards)
- death.

GHB is a difficult substance to detect or measure in body fluids and no rapid assay urine test is available. Obtaining verbal drug use history is the best method for assessing recent GHB use.

Withdrawal

Abrupt cessation may cause a user who has developed tolerance to and dependence on GHB to suffer withdrawal symptoms, which usually start about 12 hours after the last dose and can continue for approximately 2 weeks (NCETA, 2004).

Withdrawal symptoms may include:

- confusion/delirium
- agitation
- anxiety
- paranoia
- tremor
- diaphoresis
- muscle cramps
- hallucinations
- tachycardia
- disturbed sleeping patterns
- bowel and bladder incontinence.

Management of GHB withdrawal may require use of both short- and long-acting benzodiazepines. Some users may require further sedation with a short-acting anaesthetic agent (e.g. propofol) (NSW Health, 2008c).

New psychoactive substances

The National Drug and Alcohol Research Centre at the University of NSW reports regularly on new psychoactive substances (NPSs) such as synthetic cannabis. Patients may not know exactly what they have taken; nurses need to be aware of NPSs.

INTERPROFESSIONAL CARE

Successful treatment of substance abuse and dependence requires the skills of an interprofessional team specialising in the psychiatric aspects of such disorders, in effective clinical partnership with professionals working in other settings (NSW Health, 2008b).

Psychosocial interventions for which there is the strongest evidence and professional consensus include assessment and brief intervention, motivational interviewing, cognitive behaviour therapy, psychodynamic therapy, dialectical behaviour therapy, self-help approaches and continuing care (NSW Health, 2008b). Therapies may include detoxification, pharmacotherapy, drug antagonists, drug agonists, anti-craving medication, aversion agents, group and/or individual psychotherapy, psychotropic medications, cognitive–behavioural strategies, family counselling and self-help groups.

Substance users can be treated in either inpatient or outpatient settings. Substance overdose can be a life-threatening condition requiring emergency hospitalisation for medical stabilisation before implementation of any of the recommended interventions. Several diagnostic tests provide valuable information about the person's physical condition and set the course for treatment.

Diagnostic tests

Blood and urine are the body fluids most often tested for drug content; saliva, perspiration and hair also may be tested. A breathalyser provides the simplest method of detecting blood alcohol content; more invasive procedures such as serum drug levels to treat drug overdoses or complications are useful in the emergency department and other hospital settings. Urine drug screens (UDS) may be a rapid assay test via 'dip-stick' products, or sent away for laboratory analysis; UDS is non-invasive, and is the preferred method for detecting substances in the body. Companies often require a precautionary UDS of prospective employees, some amateur and professional athletes have to submit to random drug testing, and determination of drug use arises in the legal system in connection with criminal activity and family court proceedings. The period in which drugs can be found in blood and urine varies according to dosage and the metabolic properties of the drug; traces may disappear within 24

hours, or still be detectable 30 days later. The psychoactive substance found in marijuana, THC, is stored in fatty tissues (especially the brain and reproductive system), and can be detected in the body for up to 6 weeks (Kneisl & Trigoboff, 2013).

Knowledge of the blood alcohol level is helpful in ascertaining levels of intoxication and tolerance, and whether recent drinking has been accurately reported. At 0.10% (after five to six drinks in 1 to 2 hours), voluntary motor action becomes clumsy, and reaction time is impaired to a degree that varies with gender, weight and food ingestion. Intoxication will occur more rapidly in small women drinking alcohol on an empty stomach than in large males who have eaten a full meal. At 0.20% (after 10 to 12 drinks in 2 to 4 hours), function of the motor area in the brain is depressed, causing staggering and ataxia (Kneisl & Trigoboff, 2013). A level above 0.10% without associated behavioural symptoms indicates the presence of tolerance; high tolerance is a sign of physical dependence.

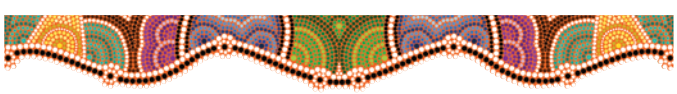
Assessing for withdrawal symptoms is important when the BAL is high and the person shows significant tolerance: medication for treatment of withdrawal from alcohol is usually not started until the BAL is below a set norm (usually 0.10%) unless withdrawal symptoms become severe. BAL may be repeated several times, several hours apart, to assess the body's metabolism of alcohol and determine when it is safe to administer medication to minimise the withdrawal symptoms.

Emergency care for overdose

People who have overdosed on any substance constitute a serious medical emergency. They may become severely sedated and difficult to arouse, requiring determined efforts to maintain wakefulness, but stupor and coma may nevertheless occur. Respiratory depression may require mechanical ventilation, while a seizure is another serious complication needing emergency treatment. Methamphetamine overdose can cause psychosis, arrhythmias, stroke and cerebral haemorrhage (Lee et al., 2007).

Cases of intentional overdose necessitate constant monitoring for further signs of suicidal ideation—never leave an actively suicidal person alone.

It is important not to assume the level of sedation to be due solely to intoxication if there is any possibility of a concomitant or contributing factor; conditions which can mimic aspects of intoxication include post-ictal states, hypoglycaemia, head injury, hepatic failure, stroke and other cerebrovascular events, infection, respiratory failure and hypoxia (NCETA, 2004).



Nursing care

Nurses and midwives will encounter people with problems of substance misuse in all healthcare settings—hospital emergency departments, medical and surgical units, mental health, inpatient wards, outpatient clinics, maternity wards, home visiting, community health departments, pain clinics, ambulatory care centres and in specific drug and alcohol treatment programs.

Substance users are a diverse group of people with differing needs and backgrounds; they are often subject to discrimination and prejudices similar to those directed at other minority groups, which can effect further marginalisation. These negative attitudes are often formed and fuelled by fear, ignorance and inaccurate information. If judged in a negative manner by nurses and other healthcare workers, drug users are likely to be less comfortable in accessing healthcare, thereby potentially worsening their condition and prognosis. Nurses need to view drug and alcohol misuse as a health problem, not a moral one. Forming a therapeutic relationship and delivering person-centred care will increase the likelihood of useful health interventions; nurses command a high level of community support and trust, and are therefore in an ideal position to positively influence outcomes for people with these health problems (NCETA, 2004).

Drug and alcohol use assessment

Drug and alcohol use assessment should be a routine part of everyone's care. It comprises a comprehensive history of use of all types of drugs: route of administration; frequency, amount and duration of use, and the time and amount of last dose; likelihood of substance intoxication; potential risk of withdrawal; possible mental health co-morbidities; physical and psychosocial issues; child protection concerns; and harm minimisation strategies.

Nursing interventions

A substantial body of evidence confirms the effectiveness of interventions and benefits of treatment that nurses can provide for people presenting with substance use problems: screening, assessment, provision of information and advice, opportunistic interventions and withdrawal management; counselling, including motivational interviewing and relapse prevention; referral to clinicians with specialist skills in drug and alcohol; and follow-up monitoring and care coordination. These interventions have been shown to be effective in specialist and non-specialist settings.

Opportunistic intervention

'Opportunistic intervention' describes taking advantage of an opening or chance to discuss a health-related concern which might not be the primary reason for presentation to the health facility: essentially, dealing with problems of substance use at a critical time for the person admitted. For example, opportunistically during routine contacts (perhaps at needle and syringe exchanges), staff should provide all drug users with information and advice about reducing exposure to blood-borne viruses, including reduction of sexual and injection risk behaviours (National Institute for Health and Clinical Excellence, 2007). A discussion about consumption of alcohol above the recommended safe levels with a person admitted to a gastroenterology ward for a bleeding gastric ulcer is another example.

Key strategies include interacting with people in a non-judgmental way, clearly identifying the link between their substance use and the negative health or lifestyle consequences; providing information; and offering further assistance or referral.

Opportunistic interventions need not be extensive, and can be executed in as little as 5 minutes. Australian research (Heather et al., 1996) has shown that brief counselling provided on a general hospital medical ward directed at men's heavy drinking

resulted in reduction of their alcohol consumption after discharge. Pregnancy and the perinatal phase are appropriate times to discuss drug and/or alcohol problems and ascertain whether further assistance or treatment might be beneficial. While opportunistic interventions can be undertaken at any stage of substance using, they are most appropriate in the earlier stages. Heavily substance-dependent people, people with co-morbid mental health issues, or those who feel powerless to change should be referred for specialist treatment (NSW Health, 2008b).

Psychosocial interventions

Conveying respect and providing non-judgmental care are the basis of all psychological interventions. The efficacy of several psychosocial interventions has been proven by research: these include motivational interviewing, problem solving, goal setting and relapse prevention (including identifying triggers and distinguishing a lapse from a relapse), which are briefly outlined here.

Stages of change model

The stages of change model can assist nurses both to understand and to build a rapport with people, and is the basis for many of the psychological approaches employed. DiClemente (2005) posits the five stages of change summarised in Table 5.7: precontemplation, contemplation, preparation, action and maintenance. Research has shown this model underpins all types of change related to behaviours such as smoking, drinking and using drugs. Relapse also is seen as a part of the circle—it is normal for people to 'travel around' the circle before reaching a stable change.

Motivational interviewing

This therapeutic approach is intended as a means for clinicians to help people to work towards change, and to deal with their fluctuation between opposing behaviours and thoughts (Miller & Rollnick, 1991). It uses an approach that matches the current stage of change and assists movement through the stages towards successful sustained change (Miller, 1995); it replaces previous direct and often confrontational approaches. Box 5.2 explains the principles of this approach.

Problem solving

The ability to respond effectively to problems is associated with improved treatment outcomes (NCETA Consortium, 2004). Nurses should support the development of problem-solving skills, including techniques such as problem analysis, verbal instruction, written information and skill rehearsal.

Goal setting

Goal setting gives people a direction, and provides a standard by which progress can be reviewed together with evidence of improvement. Goals should be practical, realistic and linked with

TABLE 5.7 Stages of change model

STAGE	DESCRIPTION	STRATEGIES AND CONSIDERATIONS
Precontemplation	The person is unaware, or barely aware, that there is a problem; the cons of giving up outweigh the pros, and there is no intent to change substance using in the foreseeable future. There may also be a lack of hope because of previous failures.	Prescriptive advice can be counterproductive, creating resistance, and arousing a range of defence mechanisms which prevent people from hearing/understanding the need for change. Provide information and feedback to raise problem awareness and the possibility of change, and raise doubt to increase the perception of the risks and problems of their current behaviour.
Contemplation	The existence of a problem is acknowledged, and the person may exhibit visible signs of distress. At this stage people are open to information and education, are contemplating change but are not quite ready, and are considering the positives and negatives of giving up.	Using the person's language and goals, examine the reasons for both changing and not changing, and tip the balance in favour of change.
Preparation	The person is beginning to set goals and make plans, and strategies are developed.	Suggest choices and probe the person's thinking about options.
Action	Behavioural change has clearly begun, and significant efforts have been made to stop using alcohol or drugs. The action stage on average lasts 6 months.	Give support to take steps towards change. Raise awareness of the psychological, cognitive, behavioural and emotional events which can work against best efforts, and plan strategies to overcome potential triggers.
Relapse	Alcohol or drugs are used again.	Treat this as a learning opportunity—a chance to ascertain which strategies and which part of the plan did not work. Help the person to identify and use strategies to prevent relapse, but to prepare for and expect it. Urge them not to give up, and to continue on the wheel of change.
Maintenance	Continued abstention from alcohol or drugs, and intent to sustain and strengthen improvements made. It can take a few years to feel 'secure'.	

Source: Based on Blueprint to build strong foundations for change by C. DiClemente (2005). *Addiction today*. Retrieved from www.addictiontoday.org/addictiontoday/2010/08/stages-of-change-carlo-diclemente.html.

BOX 5.2 Principles of motivational interviewing

1. Express empathy
Understanding the cycle of change can help the nurse to empathise, communicate a sense of respect, and give direction to intervention strategies (Miller, 1995); an empathic therapeutic style is associated with greater long-term behaviour change.

2. Develop discrepancy
Motivation for change occurs when people perceive a discrepancy between where they are and where they want to be. The object of this approach is to enhance and focus attention on such discrepancies with regard to alcohol and drug use (Miller, 1995); at this stage the nurse may discuss concerns about the person's current level of use, what is positive about such use and what is causing problems (NSW Health, 2008b).

3. Roll with resistance and avoid argumentation
New perspectives may be raised but not imposed: arguing is counterproductive and can create defensiveness. The simplest response to resistance is non-resistance; repeating the person's statement in a neutral form acknowledges and validates it, and can elicit an opposite reaction.

4. Support self-efficacy
Self-efficacy is the belief that one can adopt a behaviour or accomplish a particular task; it is important because belief in the possibility of change is an effective motivator. The nurse facilitates informed choice of goal and method of treatment, and communicates confidence and optimism (NCETA Consortium, 2004).

treatment plans derived directly from results of assessment and person-centred collaboration; the plans should contain strategies for achieving these goals and, where appropriate, include parents, partners, families and friends (Marsh & Willis, 2007).

Relapse prevention

Relapse prevention and management encompass cognitive behavioural strategies which provide people with the skills and confidence to avoid or to deal with any setbacks; thus nurses should explore their triggers with them—what situations, moods, thoughts or events might lead to a high risk of relapse (Marsh & Willis, 2007).

Health promotion and global policy on primary prevention measures for substance abuse

Member states of the United Nations General Assembly, increasingly concerned about threats posed by the world drug problem, have called for the elimination or significant reduction of drug supply and demand by 2019 (United Nations Office on Drugs and Crime, 2012). Controversially others, citing the failure of the so-called 'war on drugs' and its effects on the murder rate ('Eight of the world's most violent countries are in Latin America or the Caribbean, the source of most of the world's cocaine and heroin'), have called for an end to prohibition (*Economist*, 2012). Supporters of this viewpoint note that because the trade is illegal the 'multi-billion dollar profits' go to criminal entities; this gives them enormous power and in some cases funds terrorism (*Economist*, 2012).

The World Health Organization (2010), acknowledging the desirability of global measures to reduce alcohol misuse and recognising its links with socioeconomic status, has drawn up a list of areas for action. These include drink-driving policies and countermeasures, the availability, marketing and pricing of alcoholic beverages, and reducing the negative consequences of drinking and alcohol intoxication.

Health promotion

Health promotion efforts are aimed at preventing drug use among children and adolescents and reducing the risks among adults. Teenagers are a vulnerable population, often succumbing to peer pressure; therefore, adolescence is the most common phase for the first experience with drugs (Stuart & Laraia, 2005). Healthy lifestyles, parental support, stress management, good nutrition and information about ways to cope with peer pressure are important topics for the nurse to cover in school prevention programs.

Nurses should advise adults about the physiological or physical effects of substances on the body, and provide information about healthy coping mechanisms and relaxation and stress reduction techniques to decrease the risks of substance abuse.

Harm minimisation is the key philosophy and basis for government policy in the management of drug- and alcohol-related issues. This philosophy accepts that drug and alcohol use exists, is likely to continue, and is widespread across all levels of the Australian and international communities; it does not preclude abstinence and is interrelated with harm (demand and supply) reduction. Harm minimisation describes reducing the impact of drug- and/or alcohol-related harm to individuals and the community through a range of cost-effective public health policies, strategies and practices. Because of their numbers, roles, knowledge and skills, nurses and midwives are particularly well placed to identify risks of harm associated with drug and alcohol use, and can apply a range of harm reduction strategies and interventions (NSW Health, 2008c).

Assessment

A comprehensive approach to the assessment of substance use is essential to ensure adequate and appropriate intervention. Three important areas to assess are people's history of substance use, their medical and psychiatric history, and the presence of psychosocial concerns.

Ask questions in a non-threatening, matter-of-fact manner, as if you are having a casual conversation. Questions can be phrased so as not to imply judgment or disapproval: for example, a non-threatening question such as, 'How much alcohol do you drink?' is preferable to the judgmental question, 'You don't drink too much alcohol, do you?' Open-ended questions that elicit more than a simple 'yes' or 'no' answer help to determine the direction of future counselling. Examples of open-ended questions are

BOX 5.3 Examples of open-ended questions for assessment

- On average, how many days per week do you drink alcohol or use drugs?
- On a typical day when you use drugs or alcohol, how many hits or drinks do you have?
- What is the greatest number of drinks you have had at any one time during the past month?
- What drug(s) did you take before coming to the hospital or clinic?
- How long have you been using the substances?
- How often and how much do you usually use?
- What kind of problems has substance use caused for you, your family, friends, finances and health?

provided in Box 5.3. Use of therapeutic communication techniques helps to establish trust prior to the assessment process.

A diverse range of medications is available for use in the management of substance abuse or dependence and substance withdrawal (see Table 5.8). The most appropriate medication for use in any particular situation will depend on which substance has been abused, the level of use, whether the treatment is for withdrawal management or as relapse prevention, and the physical and mental health of the person.

History of past substance use

A thorough substance use history is necessary to ascertain the possibility of tolerance, physical dependence, or withdrawal syndrome; the following questions are helpful in eliciting a pattern of substance use behaviour:

- How many substances have been used simultaneously (**polysubstance abuse**—simultaneous use of many substances)?
- When was the first use of the substance(s), and thereafter how often and how much?
- Is there a history of blackouts, delirium or seizures?
- Is there a history of withdrawal syndrome, overdoses and complications from previous substance use?
- Has the person ever been treated in an alcohol or drug abuse clinic?
- Has the person ever been arrested for driving under the influence, or charged with any criminal offence committed while using drugs or alcohol?
- Is there a family history of drug or alcohol use?

Medical and psychiatric history

A personal medical history is an essential area for assessment, and should include the existence of any concomitant physical or mental conditions (e.g. HIV, hepatitis, cirrhosis, oesophageal varices, pancreatitis, gastritis, Wernicke–Korsakoff syndrome, depression or anxiety). Ask about prescribed and

NURSING CARE OF THE OLDER ADULT Alcohol use disorders in the older adult

Alcohol use disorders in older people are common and are associated with considerable morbidity and numerous physical problems. Alcohol abuse affects alertness, judgment, coordination and reaction time, increasing the risk of falls and injuries, which can rob older people of their independence. Alcohol negatively interacts with the natural ageing process to increase the risk of hypertension, cardiac arrhythmias, cancers, gastrointestinal problems, cognitive deficits, bone loss and emotional challenges, most notably depression (Stevenson, 2005). Older adults (especially women) are also more likely to use prescription or over-the-counter medicines, which can be harmful when mixed with alcohol and/or illicit drugs (Lantz, 2005; Simoni-Wastila & Strickler, 2004).

Although substance abuse and dependence are not as common in the elderly, they are under-detected and misdiagnosed for a number of reasons: people in this age group are less likely to disclose their problems, and screening and diagnostic instruments may not uncover misuse (O'Connell & Chin, 2009). Substance abuse problems can also be difficult to detect because many of the symptoms (e.g. insomnia, depression, loss of memory, anxiety, musculo-skeletal pain) may be confused with conditions commonly seen in the elderly (Lantz, 2005), and may be thought of as 'part of the ageing process'. Alcohol and drug abuse can also make other medical problems hard to diagnose—for example, by dulling a pain sensation that might warn of a heart attack.

Clinical staff may view alcohol use disorders as being understandable in the context of poor health and changing life circumstances—this can lead to therapeutic pessimism when confronted with such problems (O'Connell & Chin, 2009). However, the absolute number of elderly people with substance use disorders is increasing in proportion with the ageing population (Blow & Barry, 2014): by 2020 the number in need of substance abuse treatment is predicted to increase from 1.7 million in 2001 to 4.4 million (Gfroerer et al., 2003). Therefore, health services need to improve provision of age-appropriate screening, treatment methods and services (O'Connell & Chin, 2009). Because depression and alcohol abuse are the disorders most frequently found in completed suicides, it is important that nurses routinely screen older adults for both substance abuse and mental disorders.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. You are caring for an 85-year-old man who tells you that his wife died 6 months ago, and he drinks only to dull the pain of her loss. How would you respond?
2. What do you think is the reason for the frequent failure of nurses and other healthcare professionals to recognise and treat substance abuse problems in older adults?
3. You are caring for an older adult who denies that alcohol has become a serious problem, even after three episodes of hospitalisation in 6 months because of accidents, falls and blackouts. What would you do?

TABLE 5.8 Drugs used in the treatment of substance withdrawal/abuse

DRUG	DOSE	PURPOSE
BENZODIAZEPINES		
1. Midazolam	Individualised, IMI, IV	Diminishes anxiety and has anticonvulsant qualities to provide safe withdrawal. May be ordered q4h or prn to manage adverse effects from withdrawal, then dose is tapered to zero.
2. Diazepam (Valium)	4–120 mg/day	
3. Oxazepam (Serepax)	30–120 mg/day	
4. Lorazepam (Ativan)	2–6 mg/day	
VITAMINS		
1. Thiamine (vitamin B_1)	100 mg/day	Prevents Wernicke's encephalopathy
2. Folic acid	1 mg/day	Corrects vitamin deficiency caused by heavy long-term alcohol abuse
3. Multivitamins	1 tab/cap daily	
ANTICONVULSANTS		
1. Phenytoin (Dilantin)	Individualised as per bodyweight	Seizure prophylaxis
2. Magnesium sulfate	1 g q6h	Reduces post-withdrawal seizures
ABSTINENCE MEDICATIONS		
1. Disulfiram (Antabuse)	250 mg/day	Prevents breakdown of alcohol
2. Naltrexone (ReVia)	50 mg/day	Diminishes cravings for alcohol and opioids
3. Acamprosate (Campral)	133.3–199.8 mg/day	Decreases alcohol craving
ANTIDEPRESSANTS		
1. Fluoxetine (Prozac)	20–80 mg/day	Enhances and stabilises mood and diminishes anxiety
2. Sertraline (Zoloft)	50–200 mg/day	Enhances and stabilises mood and diminishes anxiety
SYMPTOMATIC TREATMENT FOR INDIVIDUAL WITHDRAWAL SYMPTOMS		
Metoclopramide (Maxolon)	30 mg/day	Nausea and vomiting
Prochlorperazine (Stemetil)	10–30 mg/day	Nausea and vomiting
Paracetamol	Up to 4 g/day	Headache, generalised aches and pains
Ibuprofen (or other non-steroidal anti-inflammatory agents)	Up to 1.6 g/day	Headache, generalised aches and pains
Kaomagma		Diarrhoea
Hyoscine butylbromide (Buscopan)	80 mg/day	GIT spasm
OPIOID AGONISTS		
Methadone	Individualised	Opioid substitution agent for maintenance treatment
Buprenorphine	Individualised	Opioid substitution agent for maintenance treatment or opioid withdrawal management
Buprenorphine-naloxone	Individual dosage up to 32 mg/day	Opioid substitution agent for maintenance treatment

GIT = gastrointestinal tract; IMI = intramuscular injection; IV = intravenous; q4h (q6h) = every 4 (6) hours; prn = as needed.

over-the-counter medications as well as any allergies or sensitivity to drugs. Also significant is the current mental status, and a brief overview is required:

- Is there a history of abuse (physical or sexual) or family violence?
- Any history of suicide attempts?
- Current suicidal or homicidal ideation?

PSYCHOSOCIAL ISSUES Information about the person's level of stress and other psychosocial concerns can assist in the assessment of substance use problems:

- Has substance use affected the person's ability to hold a job?
- Has substance use affected relationships with spouse, family, friends or co-workers?
- How does the person usually cope with stress?
- Does the person have a support system that helps in times of need?
- How does the person spend leisure time?

SCREENING TOOLS Several screening tools are available which may help the nurse to determine the degree of severity of substance abuse or dependence (see Figure 5.2). These provide a non-judgmental, brief and easy method to ascertain patterns of substance abuse behaviours:

- The Alcohol Use Disorders Identification Test (AUDIT), the 10-question tool most commonly used in Australia for screening for excessive drinking, was developed by the

Brief MAST **Scoring Yes to 3 or more indicates alcoholism**

1. Do you feel you are a normal drinker?
2. Do friends or relatives think you are a normal drinker?
3. Have you ever attended a meeting of Alcoholics Anonymous?
4. Have you ever been in trouble at work because of drinking?
5. Have you ever lost friends or girlfriends/boyfriends because of drinking?
6. Have you ever neglected your obligations, your family, or your work for 2 or more days in a row because of your drinking?
7. Have you ever had delirium tremens (DTs), severe shaking, or heard voices or seen things that were not there after heavy drinking?
8. Have you ever gone to anyone for help about your drinking?
9. Have you ever been in a hospital because of your drinking?
10. Have you ever been arrested for drunken driving or other drunken behaviour?

B-DAST **The following questions concern information about your involvement with drugs *not including alcoholic beverages* during the past 12 months.**

In the statements, 'drug abuse' refers to (1) the use of prescribed or over-the-counter drugs in excess of the directions and (2) any non-medical use of drugs. The various classes of drugs may include cannabis, solvents, anti-anxiety drugs, sedative-hypnotics, cocaine, stimulants, hallucinogens, and narcotics. Remember *do not include alcoholic beverages*.

Have you used drugs other than those required for medical purposes?	Yes ___	No ___
Do you abuse more than one drug at a time?	Yes ___	No ___
Are you always able to stop using drugs when you want to?	Yes ___	No ___
Have you had 'blackouts' or 'flashbacks' as a result of drug use?	Yes ___	No ___
Do you ever feel bad about your drug abuse?	Yes ___	No ___
Does your spouse (or parents) ever complain about your involvement with drugs?	Yes ___	No ___
Have you neglected your family because of your use of drugs?	Yes ___	No ___
Have you engaged in illegal activities in order to obtain drugs?	Yes ___	No ___
Have you ever experienced withdrawal symptoms (felt sick) when you stopped taking drugs?	Yes ___	No ___
Have you had medical problems as a result of your drug use (e.g. memory loss, hepatitis, convulsions, bleeding, etc.)?	Yes ___	No ___

Scoring: one positive response warrants further evaluation.

FIGURE 5.2 ■ Screening tools for alcohol and drug abuse

Sources: Adapted from The brief MAST: A shortened version of the Michigan Alcohol Screening Test by A. D. Porkorny, B. A. Miller & H. B. Kaplan (1972). *American Journal of Psychiatry*, *129*, 342–345. Copyright 1972 by the American Psychiatric Association; and *Brief Drug Abuse Screening Test* (B-DAST) (p. 363) by H. A. Skinner (1982). Langford Lance, England: Elsevier Science Ltd. Copyright 1982.

World Health Organization. Particularly suitable for primary healthcare settings, it has been used in several different countries with diverse cultural groups. A score of 8 to 10 is associated with harmful or hazardous drinking. As a general guide, a score of 13 or more is likely to indicate alcohol dependence.

- Other screening tools, less often used in Australia, include the MAST and the CAGE.

WITHDRAWAL ASSESSMENT TOOLS Nurses working in medical–surgical, mental health and special substance abuse units routinely care for people experiencing acute alcohol or opiate withdrawal. Several assessment tools are available to determine the severity of withdrawal symptoms and indicate the need for pharmacological treatment to manage them; for example:

- The Clinical Institute Withdrawal Assessment of Alcohol—Revised (CIWA-Ar) (Sullivan et al., 1989) (see Figure 5.3) is used widely in clinical and research settings for initial assessment and ongoing monitoring of alcohol withdrawal signs and symptoms. The CIWA-Ar scale is a validated 10-item assessment tool to monitor and help guide treatment for people undergoing alcohol withdrawal. It screens for several symptoms (e.g. high blood pressure, rapid pulse, tremors, anxiety, agitation, hallucinations and sweating); the resulting score directs administration of benzodiazepines or other drugs to relieve associated symptoms of withdrawal and prevent seizures. A score of 8 points or fewer corresponds with mild withdrawal symptoms; 9 to 15 points indicate moderate withdrawal; 15 or greater denote severe withdrawal and an increased risk of delirium tremens and seizures.
- The Clinical Opiate Withdrawal Scale (COWS) (Wesson & Ling, 2003) rates 11 common signs or symptoms of opiate withdrawal. The total score assesses the intensity

Clinical Institute Withdrawal Assessment of Alcohol Scale, Revised (CIWA-Ar)

Patient:_____ **Date:** _____ **Time:** _____ (24 hour clock, midnight = 00:00)

Pulse or heart rate, taken for one minute:_____ **Blood pressure:**_____

NAUSEA AND VOMITING -- Ask 'Do you feel sick to your stomach? Have you vomited?' Observation.
0 no nausea and no vomiting
1 mild nausea with no vomiting
2
3
4 intermittent nausea with dry heaves
5
6
7 constant nausea, frequent dry heaves and vomiting

TACTILE DISTURBANCES -- Ask 'Have you any itching, pins and needles sensations, any burning, any numbness, or do you feel bugs crawling on or under your skin?' Observation.
0 none
1 very mild itching, pins and needles, burning or numbness
2 mild itching, pins and needles, burning or numbness
3 moderate itching, pins and needles, burning or numbness
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

TREMOR -- Arms extended and fingers spread apart. Observation.
0 no tremor
1 not visible, but can be felt fingertip to fingertip
2
3
4 moderate, with patient's arms extended
5
6
7 severe, even with arms not extended

AUDITORY DISTURBANCES -- Ask 'Are you more aware of sounds around you? Are they harsh? Do they frighten you? Are you hearing anything that is disturbing to you? Are you hearing things you know are not there?' Observation.
0 not present
1 very mild harshness or ability to frighten
2 mild harshness or ability to frighten
3 moderate harshness or ability to frighten
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

PAROXYSMAL SWEATS -- Observation.
0 no sweat visible
1 barely perceptible sweating, palms moist
2
3
4 beads of sweat obvious on forehead
5
6
7 drenching sweats

VISUAL DISTURBANCES -- Ask 'Does the light appear to be too bright? Is its color different? Does it hurt your eyes? Are you seeing anything that is disturbing to you? Are you seeing things you know are not there?' Observation.
0 not present
1 very mild sensitivity
2 mild sensitivity
3 moderate sensitivity
4 moderately severe hallucinations
5 severe hallucinations
6 extremely severe hallucinations
7 continuous hallucinations

ANXIETY -- Ask 'Do you feel nervous?' Observation.
0 no anxiety, at ease
1 mildly anxious
2
3
4 moderately anxious, or guarded, so anxiety is inferred
5
6
7 equivalent to acute panic states as seen in severe delirium or acute schizophrenic reactions

HEADACHE, FULLNESS IN HEAD -- Ask 'Does your head feel different? Does it feel like there is a band around your head?' Do not rate for dizziness or lightheadedness. Otherwise, rate severity.
0 not present
1 very mild
2 mild
3 moderate
4 moderately severe
5 severe
6 very severe
7 extremely severe

AGITATION -- Observation.
0 normal activity
1 somewhat more than normal activity
2
3
4 moderately fidgety and restless
5
6
7 paces back and forth during most of the interview, or constantly thrashes about

ORIENTATION AND CLOUDING OF SENSORIUM -- Ask 'What day is this? Where are you? Who am I?'
0 oriented and can do serial additions
1 cannot do serial additions or is uncertain about date
2 disoriented for date by no more than 2 calendar days
3 disoriented for date by more than 2 calendar days
4 disoriented for place/or person

Total **CIWA-Ar** Score _____
Rater's Initials _____
Maximum Possible Score 67

*The **CIWA-Ar** is not* copyrighted and may be reproduced freely. This assessment for monitoring withdrawal symptoms requires approximately 5 minutes to administer. The maximum score is 67 (see instrument). Patients scoring less than 10 do not usually need additional medication for withdrawal.

Sullivan, J.T.; Sykora, K.; Schneiderman, J.; Naranjo, C.A.; and Sellers, E.M. Assessment of alcohol withdrawal: The revised Clinical Institute Withdrawal Assessment for Alcohol scale (**CIWA-Ar**). *British Journal of Addiction* 84:1353-1357, 1989.

FIGURE 5.3 ■ Assessment tool for alcohol withdrawal

Source: From Assessment of alcohol withdrawal: The revised Clinical Institute Withdrawal Assessment for Alcohol scale (CIWA-Ar) by J.T. Sullivan, K. Sykora, J. Schneiderman, C. A. Naranjo & E. M. Sellers (1989). *British Journal of Addiction*, *84*, 1353–1357.

of opiate withdrawal and determines the extent of physical dependence on opioids: a score of 12 or less indicates mild symptoms or none; 13 or more, moderate to severe.

Nursing interventions

Primary nursing diagnoses and interventions for people with substance abuse problems are listed below, together with implications for nursing care in acute and home care settings. See the 'Nursing care plan' box for more information about withdrawal from alcohol.

Risk of injury and risk of violence

- Assess level of disorientation to determine specific risks to safety. *Knowledge of level of cognitive functioning is essential to the development of an appropriate plan of care.*
- Obtain a drug history as well as urine and blood samples for laboratory analysis of substance content. *Subjective history is often not accurate and knowledge regarding substance use is important for accurate assessment.*
- Place the person in a quiet, private room to decrease excessive stimuli, but do not leave alone if excessive hyperactivity or suicidal ideation is present. *Excessive stimuli increase agitation.*
- Frequently orient the person to reality and the environment, ensuring that potentially harmful objects are stored out of reach. *People's disorientation and confusion may cause harm to themselves or others.*
- Monitor vital signs every 15 minutes until stable, and assess for signs of intoxication or withdrawal. *The most reliable information about withdrawal symptoms is vital signs; they provide information about the need for medication during detoxification.*

Ineffective denial

- Be genuine, honest and respectful. Keep all promises and convey an attitude of acceptance. *The development of a non-judgmental, therapeutic relationship is essential to gain trust.*
- Identify maladaptive behaviours or situations that have occurred in the person's life and discuss how the use of substances might have been a contributing factor. *The first step in combating denial is for the person to recognise the relationship between substance use and personal problems.*
- Encourage participation in therapeutic group activities such as co-occurring disorder or Alcoholics Anonymous (AA) meetings with people who are having or have had similar problems. *Peer feedback is often more accepted than feedback from authority figures.*

Ineffective coping

- Establish a trusting relationship. *Trust is essential to the therapeutic relationship*.
- Set limits on so-called manipulative behaviour and maintain consistency in responses. It is more helpful to think of this behaviour as care-eliciting to avoid the judgmental overtones of the word 'manipulative'. *The person may not be able to set their own limits, and needs to begin to accept responsibility without being manipulative.*
- Encourage the person to verbalise feelings, fears or anxieties, and make observations or statements acknowledging their validity. *Verbalisation of feelings helps to develop insight into behaviours and long-standing problems.*
- Explore methods of dealing with stressful situations other than resorting to substance use. Provide encouragement for changing to a healthier lifestyle. *Teach healthy coping mechanisms (e.g. physical exercise, progressive muscle relaxation, deep-breathing exercises, meditation and mindfulness).*

Imbalanced nutrition: less than body requirements

- Administer prescribed vitamins and dietary supplements. *Vitamin B_1 is necessary to prevent complications from chronic alcoholism such as Wernicke's syndrome.*
- Monitor pathology results (e.g. total albumin, complete blood count, urinalysis, electrolytes and liver enzymes) and report significant changes. *Pathology results provide necessary information to determine the extent of malnourishment.*
- Collaborate with the dietitian to determine the number of kilojoules needed for adequate nutrition and realistic weight gain. Document intake, output and kilojoule count. Weigh daily if condition warrants. *Weight loss or gain is significant assessment information for development of an appropriate plan of care.*
- Emphasise the importance of a balanced diet and relate this to the physical effects of malnutrition on body systems.

Chronic or situational low self-esteem

- Spend time with the person and convey an attitude of acceptance. Encourage the person to accept responsibility for their own behaviours and feelings. *An attitude of acceptance enhances self-worth.*
- Encourage the person to focus on strengths and accomplishments rather than weaknesses and failures. *Minimise attention to negative ruminations.*
- Encourage participation in therapeutic group activities and offer recognition and positive feedback for actual achievements. *Success and recognition increase self-esteem.*
- Teach assertiveness techniques and effective communication techniques such as using 'I feel' rather than 'You make me feel' statements. *Previous patterns of communication may have been aggressive and accusatory, causing barriers to interpersonal relationships.*

Deficient knowledge

- Assess level of knowledge and readiness to learn the effects of drugs and alcohol on the body. *Baseline assessment is required to develop appropriate teaching material.*
- Develop a teaching plan that includes measurable objectives. Include significant others, if possible. *Lifestyle changes often affect all family members.*

- Begin with simple concepts and progress to more complex issues. Use interactive teaching strategies and written materials at the appropriate educational level. Include information on physiological effects of substances, the propensity for physical and psychological dependence, and the risks to a foetus if pregnant. *Active participation and handouts enhance retention of important concepts.*

Disturbed sensory perceptions

- Observe for withdrawal symptoms and watch closely for seizures. Monitor vital signs. Provide adequate nutrition and hydration. *These actions provide supportive physical care during detoxification.*
- Frequently assess level of orientation. Orient and reassure if hallucinations, delusions or illusions are being experienced.

NURSING CARE PLAN A person experiencing withdrawal from alcohol

George Russell, aged 58, fell at home and broke his right wrist. His wife took him to the emergency department (ED) where an open reduction internal fixation (ORIF) was performed. Extensive anaesthesia was required so he was admitted to the postoperative unit for observation.

He has a ruddy complexion and looks older than his stated age. He discloses that he lost his factory job two years ago and did only odd jobs until being hired last week by a local assembly plant. His father was recovering from alcoholism and his 30-year-old son has in the past been treated for alcohol misuse. Mr Russell believes alcoholism runs in the family, but feels that his drinking is under control; however, he cannot remember the events leading up to his fall, or how he might have broken his wrist.

ASSESSMENT

During the nursing assessment, Mr Russell is hesitant in providing information and refuses to make eye contact. The ED nurse had detected alcohol on his breath, and a pre-surgery assessment had registered his blood alcohol level as 0.40%. His vital signs are within the upper limits of normal, but he is confused and disoriented, with slurred speech and a slight tremor of the hands; he is 1.83 m tall and weighs 63 kg; his total albumin is 2.9 mg and he has elevated liver enzymes. His wife says that he rarely eats the meals she prepares; he is usually drinking and has no appetite for food.

DIAGNOSES

- *Ineffective individual coping* related to possible hereditary factor and personal vulnerability.
- *Risk of injury* related to aggressive behaviour, unsteady gait and impaired motor responses.
- *Ineffective denial* related to inability to recognise maladaptive behaviours caused by substance use.
- *Imbalanced nutrition*: *less than body requirements* related to anorexia manifested by decreased weight and low serum protein levels.

PLANNING

- Establish trusting relationship with Mr Russell and spend time with him discussing his feelings, fears and anxieties.
- Consult with a doctor regarding a schedule for medications during detoxification.
- Consult with a dietitian to determine a suitable diet to provide adequate nutrition and realistic weight gain. Consult with doctor to begin vitamin B_1 (thiamine) and dietary supplements.

Expected outcomes

- He will identify three adaptive coping mechanisms he can use as alternatives to alcohol in response to stress.
- He will verbalise the negative effects of alcohol and agree to seek professional help with his drinking.
- He will be free of injury as evidenced by steady gait and absence of subsequent falls.
- He will gain 0.45 kg per week without evidence of increased fluid retention. Serum albumin levels will return to normal range.

IMPLEMENTATION

- Observe for signs of withdrawal syndrome; medicate according to regimen. Provide softly lit, comfortable, supportive, safe environment.
- Document intake and output.
- Explain the effects of alcohol abuse on the body and emphasise that prognosis is closely associated with abstinence.
- Teach three relaxation techniques that Mr Russell feels are useful.
- Provide information about self-help groups and, if he is receptive, a list of meeting times and phone numbers.

EVALUATION

Mr Russell was discharged from the postoperative unit without complications. He successfully underwent detoxification and contacted the Employee Assistance Program at his new place of employment. He was on medical leave while his wrist completely healed, and now attends Alcoholics Anonymous meetings 5 days a week. He reports that he enjoys taking long walks with his wife in the warm weather, and his appetite has returned. He has gained 2 kg in the past 6 weeks and feels physically better than he has in many years.

CRITICAL THINKING IN THE NURSING PROCESS

1. Explain why it would be important during the initial nursing assessment to include questions about Mr Russell's medication history and his use of additional medications.
2. Mr Russell asks you to explain the risks of taking disulfiram (Antabuse). What should you tell him?
3. Develop a care plan for Mr Russell for the identified problem of *Imbalanced nutrition: less than body requirements*. Why is this necessary?

REFLECTION ON THE NURSING PROCESS

1. Mr Russell tells you he believes that alcoholism 'runs in the family'. What response do you make?
2. Mrs Russell tells you she noticed that her husband increased his alcohol consumption after his father, to whom he was very close, died suddenly. She asks, 'Could it have been the grief?'. What can you say to her?

- Explain all interventions before approaching a person withdrawing from alcohol. Avoid loud noises and talk softly. Decrease external stimuli by dimming lights. *Excessive stimuli increase agitation.*
- Administer prn medications according to detoxification schedule. *Benzodiazepines help to minimise the discomfort of the withdrawal symptoms.*

Disturbed thought processes

- Use simple, step-by-step instructions and face-to-face interaction when communicating. *A person withdrawing from alcohol may be confused or disoriented.*
- Express reasonable doubt if the person relays suspicious or paranoid beliefs. Reinforce accurate perception of people or situations. *It is important to communicate that you do not share the false belief as reality, without devaluing the emotional experience of the person.*
- Do not argue with delusions or hallucinations. Convey acceptance that the person believes a situation to be true (their perception), but that the nurse does not see or hear what is not there. *Arguing or denying the belief serves no useful purpose, because delusions are not eliminated.*
- Talk to a person withdrawing from alcohol about real events and real people. Respond to feelings and reassure them that they are safe from harm. Discussions that focus on the delusions may aggravate the condition. *Verbalisation of feelings in a non-threatening environment may help develop insight.*

Community-based care

The community provides many options for treating substance abuse, comprising a mixture of individual, group and family therapy. Specific treatment options include outpatient drug and alcohol counselling through facilities such as community health centres, outpatient detoxification, outreach services, day programs, youth-specific services and self-help groups: for example, Alcoholics Anonymous and Narcotics Anonymous. Community-based care allows people to deal with problems relating to such matters as relationships or parenting, employment, and training and education while still receiving support for substance use recovery. The range of options will depend on the particular mix of services available in each locality.

Teaching the substance user and family covers:

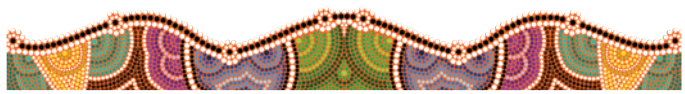
- the negative effects of substance abuse, including physical and psychological complications
- the signs of relapse, and the importance of after-care programs and self-help groups in preventing it
- information about specific medications that help to reduce the craving for alcohol (naltrexone) and maintain abstinence (disulfiram [Antabuse]), including the potential side effects, possible drug interactions, and any special precautions to be taken (e.g. avoiding over-the-counter medications such as cough syrup, which may have alcohol content)
- ways to manage stress, including techniques such as progressive muscle relaxation, abdominal breathing, mindfulness, meditation and effective coping skills.

In addition, suggest the following resources:

- Alcoholics Anonymous, Narcotics Anonymous and other self-help groups
- employee assistance programs
- individual, group and/or family counselling
- community rehabilitation programs.

CONSIDERATION FOR PRACTICE

People are at highest risk of relapse within the first few months after stopping the abused substance. Intense triggers and cravings are a normal, although difficult, feature of the earlier stages of recovery. An acronym which can assist in recognising behaviours that lead to relapse is HALT: hungry, angry, lonely and tired. Nurses should emphasise the importance in preventing relapse of a balanced diet, adequate sleep, healthy recreation activities and a caring support system.



IMPAIRED NURSES

Healthcare providers are as susceptible as anyone else to developing substance abuse; by the very nature of their roles, dentists, pharmacists, physicians and nurses are at higher risk than other professionals because of their high degree of accessibility to opioids (Trinkoff et al., 2000). One study exploring family history of alcohol and drug use in healthcare professionals found that nurses reported a higher prevalence of alcoholism than did dentists and physicians (Kenna & Wood, 2005). No significant differences, however, were found in healthcare professionals' drinking levels. Nurses experience many pressures in the workplace and have easy access to drugs; two factors—stronger religious practices and treatment for depressive symptoms—were associated with reduced substance abuse. Substance abuse and dependence can lead to impaired professional practice; nurses must act responsibly when co-workers display signs of substance abuse. Warning signs of impaired nurses in the workplace are listed in Table 5.9.

Impairment panels

The rules of the Nursing and Midwifery Board of Australia and the Nursing Council of New Zealand ensure that nurses and midwives whose ability to practise is impaired, due to factors such as mental or physical illness, or alcohol or drug use, are brought to the attention of the regulating body and assisted to overcome their problems before those problems develop to such an extent that the public are placed at risk. Programs generally provide a confidential and non-disciplinary alternative for handling impairment and the opportunity for nurses to agree to conditions being placed on their registration to enable them to continue to practise (Nurses and Midwives Board NSW, 2009).

TABLE 5.9 Warning signs of impaired nurses in the workplace

AT-RISK SITUATIONS	OBSERVABLE WARNING SIGNS
Easy access to prescription drugs	Inaccurate narcotic counts or frequent missing drugs People complain of ineffective pain control, deny receiving pain medication Excessive 'wasting' of drugs Volunteering to give medications to people Frequent trips to the bathroom
Role strain	Frequent tardiness or absenteeism, especially before and after scheduled days off Haphazard or inaccurate documentation Errors of judgment in nursing care Unorganised, erratic behaviour; unkempt appearance
Depression	Irritability, inability to focus or concentrate Abrupt mood swings Isolating self, taking long breaks Apathetic, depressed, lethargic Unexplained absences from assigned unit
Signs of alcohol or drug use	Smell of alcohol on breath Excessive use of perfumes, mouthwash or mints Slurred speech, flushed face, reddened eyes, unsteady gait
Signs of withdrawal	Tremors, restlessness, sweating Watery eyes, runny nose, stomach aches

CHAPTER HIGHLIGHTS

- Substance misuse is the unsanctioned use of any chemical despite adverse effects on the individual's physical, psychological, interpersonal or social health.
- Substance dependence occurs when control over the chemical substance is lost and the individual must use increasing amounts to produce the desired effect (tolerance), and must use the substance to avoid or relieve uncomfortable symptoms (withdrawal).
- Combinations of genetic, biological, psychological and sociocultural factors contribute to substance misuse or dependence. Addictive behaviour has been linked to biochemical changes in dopamine and serotonin brain levels, as well as heredity, ethnic differences and peer pressure. Thorough assessment of individual risk factors is necessary in order to plan and deliver appropriate nursing interventions.
- Adolescents are particularly influenced by society and peers to use substances: predominantly tobacco, alcohol and illicit drugs. A positive ethnic identity and family environment act as 'protective' deterrents for substance use.
- Substance misusers have common characteristics including risk-taking behaviour, low tolerance for frustration or pain, compulsive preoccupation with the substance, anxiety, anger and low self-esteem. Stress management, anger control, social support and counselling are helpful management strategies.
- While alcohol is the most commonly used and misused substance, polysubstance misuse frequently occurs: substances such as marijuana, cocaine and methamphetamine are often used in conjunction with alcohol. Prescription anti-anxiety agents have been misused in the past, and there is a growing trend towards prescription narcotic analgesic abuse.
- Nurses are susceptible to substance misuse because of pressures in the workplace and easy access to drugs. In order to avoid impaired professional practice, nurses need to assess their response to stress and seek early treatment for depressive symptoms.

CONCEPT CHECK

1 In which stage of change are people not considering a change in their drinking or drug-taking behaviour and frequently described as being 'in denial'?
   1 contemplation
   2 precontemplation
   3 preparation
   4 relapse

2 Which of the following are the *most* appropriate questions to ask in interviewing a person who you suspect has been abusing alcohol?
   1 'Typically, on how many days a week do you drink alcohol?'
   2 'Have you been drinking lately?'
   3 'You don't drink too much alcohol, do you?'
   4 'Has your drinking caused a lot of problems in your personal relationships?'

3 What is the rationale behind ordering thiamine (vitamin B_1) for a person with a history of chronic alcoholism?
1 to prevent acute pancreatitis
2 to prevent cirrhosis of the liver
3 to prevent hepatic encephalopathy
4 to prevent Wernicke's encephalopathy

4 Which of the following substances present the highest medical danger during withdrawal?
1 CNS stimulants and amphetamines
2 opioids and marijuana
3 alcohol and benzodiazepines
4 amphetamines and hallucinogens

5 What is the rationale for prescribing disulfiram (Antabuse) for someone with alcohol abuse problems?
1 to decrease the discomfort of withdrawal symptoms
2 to decrease the pleasant, reinforcing effects of alcohol
3 to prevent the breakdown of alcohol, thereby inhibiting impulsive drinking
4 to block the signs and symptoms of alcohol withdrawal

6 Which of the following is NOT a warning sign of substance abuse by a nurse?
1 impaired motor coordination, slurred speech, bloodshot eyes
2 unkempt appearance, disorganised, erratic behaviour
3 patients consistently report effective pain control
4 frequent absenteeism or tardiness, unexplained absences from the unit

7 Which of the following statements is FALSE?
1 Smoking is the leading known cause of preventable death and disease among women.
2 Smoking rates for women have steadily declined since the 1950s.
3 Women who smoke during pregnancy have a higher risk of spontaneous abortions.
4 Women who smoke have an increased risk of stroke and heart disease.

8 Which statement illustrates an understanding of quality use of prescribed benzodiazepines?
1 'I can take as many as I need to feel better.'
2 'It's okay to give some to my daughter if she is stressing about exams.'
3 'My new pills must be safe because my GP prescribed them.'
4 'I know why my pills were prescribed, how they work, possible side-effects and how to take them correctly.'

9 Which of the following is a realistic goal for people with a substance abuse problem?
1 They will identify ways to deal with stressful situations instead of resorting to substance use.
2 They will refrain from using substances until craving for the substance has been eliminated.
3 They will focus on negative aspects of past behaviours and interpersonal relationships.
4 They will be able to use alcohol or drugs in moderation.

10 All of the following are TRUE except:
1 People may use or abuse alcohol and other drugs because of an underlying and undiagnosed mental health problem.
2 Many drugs both legal and illegal can cause symptoms which suggest mental illness.
3 People who have become dependent on substances cannot ever change their behaviour.
4 MAST, B-DAST, COWS, AUDIT, CAGE and CIWA-Ar are all assessment tools used in alcohol and other drug work.

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CHAPTER 6

NURSING CARE OF PEOPLE IN THE EMERGENCY DEPARTMENT OR EXPERIENCING DISASTERS

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LEARNING OUTCOMES

- Outline the role of the emergency department within the Australian healthcare system.
- Discuss the aims and purpose of the triage system.
- Outline the range of assessments conducted in the emergency department, including primary and secondary survey.
- Outline the processes of disaster planning, response and mitigation.
- Explain the scope of nursing practice in the emergency department.
- Define the meaning, types and classifications of disasters.
- Describe the common types of injuries or symptoms that are associated with a disaster.
- Identify ways that nurses are able to provide care to people with special considerations during a disaster.

CLINICAL COMPETENCIES

- Demonstrate a structured approach to assessment using a primary/secondary survey.
- Assess health status of people who have experienced unexpected health breakdown.
- Use evidence-based research to plan and implement nursing care for people with injuries suffered as a result of a disaster.
- Using assessment skills, determine priority nursing diagnoses, and implement and evaluate individualised nursing interventions for people experiencing disasters.
- Provide skilled nursing care to treat disaster-related injuries.
- Integrate interprofessional care with an understanding of local, state and federal systems of disaster response.
- Evaluate and revise plan of care and interventions based on the person's condition, environmental factors and resources to promote, maintain or restore functional health status to people who have sustained injuries.

KEY TERMS

The emergency department (ED) is a common pathway for admission to hospital. Accidents, sudden illness and exacerbation of chronic conditions are just a few of the reasons people present to an ED. The ED is unique in the hospital setting as, in reality, there are no limits on the type and number of patient presentations. People being cared for in the ED are usually considered unstable until they have a definite diagnosis.

Although all nurses will be expected to care for victims of disasters, whether they work in acute care settings, ambulatory sites, long-term care facilities or at home in their communities, the ED is usually where people experiencing disasters receive initial care. There is no way to know where or when a disaster may strike. Because of this, nurses must be prepared to assist individuals, families, friends, healthcare workers, first responders and communities in their recovery from disastrous events.

There are a number of basic competencies that nurses should be cognisant of related to disaster preparedness and management of people in the ED. This chapter provides an introduction to the concepts important to understanding the role and function of emergency nurses, both within the ED setting and in the disaster setting.

EMERGENCY NURSING IN AUSTRALIA

The ED is a unique and unpredictable practice setting, defined as 'a dedicated area in a hospital that is organised and administered to provide a high standard of emergency care to those in the community who perceive the need for or are in need of acute or urgent care including hospital admission' (Australian College for Emergency Medicine (ACEM), 2009, p. 2). In Australia and New Zealand, all residents are entitled to present to public hospital EDs for free emergency healthcare, and 40% of all hospital admissions come through the ED (Department of Health (Vic.), 2011).

The provision of quality emergency nursing care requires an extensive knowledge of body systems, disease processes, age groups, specialty populations, health promotion and education. Emergency nurses must be able to apply their knowledge and skills to care for people of all ages and populations, with a diversity of undiagnosed illnesses and injuries and a multitude of different presentations in clinical areas ranging from minor injury clinics through to the resuscitation cubicles and triage (College of Emergency Nursing (CENA), 2009; 2013).

Emergency nurses must also be skilled at assessment, triage, emergency preparedness, resuscitation and crisis intervention (CENA, 2013). The busy and variable nature of the ED means that communication and teamwork are also essential emergency nursing skills.

Communication

Emergency nurses play a fundamental role in the assessment, management and disposition (transfer to a hospital ward or discharge home) of people within the ED. Effective assessment and management of people in the ED is reliant on clear communication. **Communication** is the exchange of information between two or more people, groups or entities. It involves verbal and written exchanges, as well as body language, attitude and tone (Nadzam, 2009). Emergency nurses need to communicate with people in the ED and their families, nursing colleagues, and medical allied health and support staff. Appropriate communication facilitates safe and timely interventions, and appropriate dispatch of people from the ED to hospital wards or the community.

Visits to the ED are usually episodic and often distressing. Emergency nurses can support people by providing them with information and explanations regarding processes, health conditions and plans of care. Clear communication also allows the emergency nurse to provide education for the person on health promotion, illness prevention and access to relevant community services. 'Effective communication, which is timely, accurate, complete, unambiguous, and understood by the recipient, reduces errors and results in improved patient safety' (Victorian Quality Council, 2010, p. 3), as well as increasing patient satisfaction (Saunders, 2005). However, when effective communication is absent, care of the person is compromised: it has been reported that 20% of sentinel events in Australia were the result of communication failures (Victorian Quality Council, 2010), and that poor communication can increase ED length of stay (Sprivulis et al., 2006).

Communication between emergency nurses and other health professionals must be based on current, concise and timely information. The emergency nurse should be able to distinguish between urgent and non-urgent problems, and convey this information clearly. Emergency nurses should ensure they have assessed the person and read their notes, and should have the person's health history available when communicating with other members of the healthcare team (Curtis et al., 2009).

ISBAR (Identify, Situation, Background, Assessment, Request) is a tool that is used to help provide standardised structure to verbal communication both in the ED and in other clinical contexts to ensure timely, relevant and structured clinical handover, in line with the National Safety and Quality Health Service Standards (Australian Commission on Safety and Quality in Healthcare (ACSQH), 2012). ISBAR helps to prioritise information for both the transmitter (speaker) and the receiver. It decreases the chance of forgetting relevant information and helps to decrease assumptions by making the purpose of the communication obvious at the outset (Marshall et al., 2009). Table 6.1 provides examples of information that should be communicated within each step of the tool.

The triage system in the emergency department

In 2013–2014 there were 9.2 million presentations to Australian public hospital EDs (Australian Institute of Health and Welfare (AIHW), 2014, p.11), and 609 000 presentations to New Zealand EDs in 2011–2012 (New Zealand Ministry of Health, 2013). In light of the number and variety of people presenting to Australasian emergency departments, it is important that there are both appropriately skilled staff to care for people, and a system in place to prioritise care and ensure the most effective utilisation of resources.

Triage is the process by which all people presenting to an ED for care are assessed and their care prioritised according to

TABLE 6.1 ISBAR

	SEQUENCE	INFORMATION TO BE COMMUNICATED
I	Identify	Identify yourself—name, position, location. Identify the person you are speaking about.
S	Situation	Explain WHY you are calling. Provide the person's age, gender, current status (stable, unstable).
B	Background	Give the relevant details such as presenting problems and clinical history. Include aspects of history, examination, investigations and management where relevant.
A	Assessment	Outline your assessment of the person. State what you think is going on. Give your interpretation of the situation. Don't leave the receiver to guess what you are thinking—tell them. Stating the obvious is helpful here.
R	Request	State what you want from them. Be clear about what you are requesting (e.g. review, transfer) and how urgently your request should be addressed.

Source: Adapted from S. Marshall et al. (2009). The teaching of a structured tool improves the clarity and content of interprofessional clinical communication. *Quality and Safety in Health Care*, *18*, 137–140.

actual or potential severity of illness or injury; it is the first point of clinical contact for every person who presents to the ED in need of healthcare (ACEM, 2012; CENA, 2012). In Australasia, triage is a nursing responsibility, and ACEM guidelines indicate that EDs must have 'a nurse at triage . . . at all times' (2009, p. 2). The triage nurse must perform a rapid assessment on each person who presents to the ED, and, commensurate with their findings, allocate each person a triage category based on the Australasian Triage Scale (ATS) (ACEM, 2012; CENA, 2009). The ATS is a five-level priority scale which determines the time and sequence in which people receive care (CENA, 2012). As illustrated in Table 6.2, each triage category equates to a maximum time a person can wait before being reviewed by a medical officer. As such, the ATS categories indicate the level of clinical urgency for each person (ACEM, 2012), and ensure that resources are allocated according to need, resulting in the most critically ill people receiving the greatest resource allocation. ATS categories should be applied in response to the question: 'This patient should wait for medical assessment and treatment no longer than . . . [e.g. 10 minutes if an ATS category 2]' (ACEM, 2000, p. 1).

Triage is an autonomous role which should be undertaken by an experienced and appropriately prepared emergency nurse (CENA, 2009). The triage nurse has many responsibilities, including patient assessment and allocation of an ATS category, initiation of nursing interventions to improve patient outcomes (e.g. first aid), reassessment and management of people in the waiting room, and provision of education to people in the ED and their family members (CENA, 2009).

Triage decisions clearly affect a person's safety in determining the order in which people are seen. The Emergency Triage Education Kit (ETEK) was developed by the Australian government to provide consistent education to triage nurses (Department of Health and Ageing, 2007), thereby supporting consistency in triage decision making. Consistency in triage decision making means that a person should receive the same triage category regardless of the ED to which they present, or how busy the ED is when they arrive. Importantly, triage consistency ensures the appropriate utilisation of finite ED resources and optimal service delivery (CENA, 2009).

TABLE 6.2 Australasian Triage Scale

ATS CATEGORY	TREATMENT ACUITY (MAXIMUM WAITING TIME)	PERFORMANCE INDICATOR THRESHOLD
ATS 1	Immediate	100%
ATS 2	10 minutes	80%
ATS 3	30 minutes	75%
ATS 4	60 minutes	70%
ATS 5	120 minutes	70%

Source: Policy on the Australasian Triage Scale (2000). Melbourne: Australasian College for Emergency Medicine. Reproduced by permission of the Australasian College for Emergency Medicine.

Disaster triage

Civilian disasters also require a triage system to support the allocation of resources: however, this triage system is quite different from the ATS. While the ATS aims to ensure that the sickest people receive the most resources, disaster triage aims to 'do the most for the most' (Mackway-Jones, 2011, p. 91). In disaster settings, decisions are made to allocate resources only to those who are likely to survive, rather than allocate valuable resources such as blood products or a surgeon's time on those unlikely to survive. In disaster triage, the focus is not what is best for individuals, but what is best for the community.

As resources are often limited, disaster triage relies upon the use of colours to reflect triage categories. Table 6.3 illustrates the disaster triage system used in Australasia.

Patient assessment in the emergency department

Primary survey

As people presenting to the ED are largely an undiagnosed population, assessment is focused on recognition of symptoms rather than disease processes. Assessment in emergency nursing is systematic,

TABLE 6.3 Disaster triage system, by category

TRIAGE CATEGORY	LEVEL OF URGENCY	DESCRIPTION	COLOUR
T1	Immediate priority	Casualties who require immediate life-saving procedures	Red
T2	Urgent priority	Casualties who require surgical or medical intervention within 2–4 hours	Yellow
T3	Delayed priority	Less serious cases whose treatment can safely be delayed beyond 4 hours	Green
T4	Expectant priority	Casualties whose condition is so severe that they cannot survive despite the best available care and whose treatment would divert medical resources from salvageable people who may then be compromised	Blue
Dead	N/A	Deceased casualties	White or black

Source: Adapted from K. Mackway-Jones (ed.) (2011). *Emergency triage*, (Ch. 15). Manchester: Wiley-Blackwell.

and is guided by the *primary survey*, *secondary survey* and a *focused assessment*. The National Safety and Quality Health Service Standards identify the importance of recognition and response to clinical deterioration (ACSQH, 2012). Failure to identify that a person is deteriorating can have serious consequences, and this structured approach to assessment reduces the risk that signs or symptoms of illness are missed. The **primary survey** identifies life-threatening illnesses or conditions, and follows the ABCDE mnemonic (see Table 6.4) (Higginson, Jones & Davies, 2011). The ABCDE mnemonic is designed to aid the nurse in identifying actual or potential threats to airway, breathing, circulation, disability (i.e. neurological status) and exposure. Life-threatening illness or injury must be treated prior to continuing with the assessment (Resuscitation Council (UK), 2005). For example, if the airway is not patent, the nurse should intervene to ensure airway patency before moving on to assess breathing. It is also important that these findings are reported to senior nursing and medical staff.

Airway assessment is the first step in the primary survey. The ED nurse will assess the airway for patency. Common causes of airway obstruction include foreign bodies, vomit or the person's tongue (Higginson, Jones, & Davies, 2010). Signs of obstructed airway include audible stridor, gurgling, wheezing or snoring (Higginson et al., 2011), a hoarse voice or drooling. If a person is talking, it can be concluded that their airway is patent. If a person has an obstructed airway, the nurse should call for help, then intervene by performing a head tilt/chin lift or jaw thrust (Australian Resuscitation Council, 2010). The head tilt/chin lift can move the tongue from the pharynx, thus opening the airway (Higginson et al., 2011).

TABLE 6.4 Primary survey mnemonic

PRIMARY SURVEY MNEMONIC		EXAMPLES OF DOCUMENTATION
A	Airway	Patent/stridor/drooling etc.
B	Breathing	Respiration rate, work of breathing, SpO_2
C	Circulation	Heart rate, strength and regularity, blood pressure, skin colour and temperature
D	Disability	Glasgow Coma Scale, temperature, blood sugar level
E	Exposure	Head to toe identifying abnormalities

If cervical injury is suspected, a jaw lift is preferred, as there is less risk of cervical spine movement (Higginson et al., 2011). The use of suction can also remove many obstructions if performed correctly (Higginson et al., 2011).

When the airway is patent, the nurse can assess breathing. This will include looking and feeling for rise and fall of the chest, and listening for air entry. The ED nurse will observe the work of breathing, including the use of accessory muscles during respiration, patient positioning, respiratory rate and chest symmetry. If the person is breathing, the ED nurse may decide to initiate some oxygen therapy (Higginson et al., 2011) (e.g. to improve oxygenation in response to observed increased work of breathing or tachypnoea), before moving on to assess circulation.

Assessing circulation entails feeling for rate, strength and regularity of pulse, checking blood pressure, and looking for sources of uncontrolled haemorrhage. If circulation is intact, the ED nurse can assess for disability. This includes assessment of neurological status using the Glasgow Coma Scale (GCS) (see Chapter 40 for a description of the GCS), and assessment of pain. Pain can be assessed using a numerical rating scale (e.g. 0–10) or a visual analogue scale (horizontal or vertical lines on a continuum of increasing pain). Refer to Chapter 8 for more information about pain assessment.

In the person with a GCS less than 13 (or two points less than their normal), the emergency nurse may also measure the person's blood glucose level (BGL). Alterations to BGL can affect neurological status, with some people presenting unconscious or with signs similar to those of a stroke. The nurse can check for exposure by removing the person's clothing and performing a thorough examination. If appropriate, the nurse may insert a urinary catheter to facilitate measuring response to fluid resuscitation. The primary survey is completed with an assessment of temperature and the initiation of treatments to minimise hypothermia.

Secondary survey

After the primary survey is complete, and any life-threatening findings addressed, the secondary survey can commence. The **secondary survey** is a brief but thorough head-to-toe examination which aims to detect all signs and symptoms of illness or injury, with an emphasis on the need for continued reassessment of the person and evaluation of their response to interventions (CENA, 2011). The head-to-toe examination should include visual inspection, palpation and auscultation of the person's

anterior and posterior body surfaces, looking for abnormalities. Many actions occur simultaneously during the assessment and, while performing the head-to-toe physical assessment, the emergency nurse should also be gathering the person's health history.

Focused assessment

Following the primary and secondary survey, the emergency nurse should undertake a focused assessment of the body system or region of concern, as identified by the signs and symptoms or mechanism of injury (Curtis et al., 2009). The **focused assessment** should help to identify specific issues and guide treatment. For example, a person who presents with a painful wrist following a fall would be assessed for threats to ABCDE, have a secondary assessment, including collection of health history, followed by a focused assessment of the distal limb (Curtis et al., 2009). A mental health assessment may be undertaken as part of the focused assessment if relevant (see Chapter 50).

Once the person has been thoroughly assessed, any concerns—sometimes referred to as 'red flags' (Curtis et al., 2009)—must be identified, reported (using ISBAR) and documented. Abnormal vital signs, symptoms of a time-critical illness (e.g. acute myocardial infarction (AMI), stroke or envenomation), or a high-risk medical history (e.g. renal failure in a person with confusion), are all concerns which may be of threat to the person's life or limb.

HEALTH HISTORY The health history can be collected by asking questions of the person admitted to the ED and their carer/next of kin during the physical assessment. The health history should include the presenting problem and associated symptoms, the mechanism of injury (i.e. how the injury occurred), if relevant, current treatment for the presenting problem, relevant past medical and surgical history, medications, known allergies, immunisation status, family history and a social history (CENA, 2011; Curtis et al., 2009).

INVESTIGATIONS Investigations assist in diagnosis of illness and injury, and subsequent development of a definitive plan of care for the person. Investigations can assist in the identification of people who are sick or have complex needs. It is important that emergency nurses understand the rationale for ordering specific investigations and the significance of each result, so that they understand the rationale for further management of the person. Many EDs in Australia and internationally (Innes et al., 2015) have introduced protocols to allow emergency nurses to initiate investigations such as ordering pathology and distal limb x-rays. Nurse-initiated investigations have been shown to reduce length of stay in the ED (Innes et al., 2015).

REASSESSMENT Following each intervention, an important responsibility of the emergency nurse is the reassessment of the person (Curtis et al., 2009). The purpose of reassessment is two-fold: (i) to ascertain if the intervention was effective, and (ii) to establish if there are any undesired effects from the intervention. For example, a person who is hypotensive may be given a fluid bolus to improve blood pressure. Following the fluid bolus, the emergency nurse should reassess the person's heart rate and blood pressure to determine whether the intervention has achieved its purpose. The emergency nurse should also consider the undesired effect of a fluid bolus in some people (fluid overload), and assess for signs of this (i.e. assess work of breathing and listen to breath sounds).

DOCUMENTATION It is important in all areas of healthcare provision that each person's assessment findings and interventions are documented, and this is also true in the ED. Australasian EDs are moving towards a paperless environment, and for many emergency nurses documentation from triage through to discharge is all computer-based. In the ED, nursing documentation will include the person's health history, nursing assessment and vital signs, interventions (including drugs and fluids administered), reassessment, and reporting of findings (CENA, 2011).

The scope of nursing practice in the emergency department

The scope of practice for Australasian emergency nurses is broad and highly specialised. Unlike nurses in most ward settings, emergency nurses care for people across the lifespan, delivering newborns, managing unwell children and adults, and providing end-of-life care. The problems that people may present with also vary, from those with minor injuries to the critically ill. As a result, Australasian emergency nurses need to have excellent critical thinking and clinical reasoning skills, as well as an extensive clinical skill set. Emergency nurses need to consolidate the person's health history with assessment findings to determine appropriate nursing interventions and the degree of urgency with which interventions must occur.

Emergency nursing skills in Australasian EDs include the ability to provide simple first aid at triage; insert IV cannulae and draw blood; select and apply appropriate wound dressings; apply plaster backslabs and splints to support injured limbs; close simple wounds using sutures; and provide appropriate oxygen delivery to people who are short of breath. As well as these clinical skills, emergency nurses have many advanced practice roles, including the collection and systematic interpretation of 12-lead electrocardiograms, haemodynamic monitoring and management of the intubated person. Emergency nurses require a practical understanding of ventilation, with studies revealing that Australian emergency nurses are responsible for the initial and ongoing selection of ventilator settings using both non-invasive positive pressure ventilation and mechanical ventilation (Rose & Gerdtz, 2009a; 2009b), for both adult and paediatric patients.

As they are likely to care for people with a variety of illnesses, it is essential that emergency nurses possess considerable pharmacological knowledge. Pain is the most common presenting complaint in the ED, and many Australian EDs have introduced policies allowing emergency nurses to initiate oral analgesia (Shaban et al., 2012), including Schedule 8 drugs, for people in pain.

Nurses have a central role in the timely discharge of people from the ED. Consistent triage decisions, nurse-initiated analgesia, timely assessment, nurse-initiated investigations, and recognition and reporting of abnormal findings all contribute to a rapid ED journey and achieving the national throughput targets. Communication of each person's requirements and plans of

care facilitates the timely allocation of a hospital ward bed, or the arrival of family to transport the person home.

Emergency Nurse Practitioners

Many Australian EDs also have Nurse Practitioners working in the ED team. Nurse Practitioners have completed formal education beyond that of the Registered Nurse, allowing them to work in an advanced practice role, making independent and collaborative decisions about people's healthcare needs. The scope of the Nurse Practitioner includes ordering investigations, diagnosis of illness, prescribing medications and referral of people to specialists as required (CENA, 2006; Middleton et al., 2011; Nursing and Midwifery Board of Australia (NMBA), 2014).

People with unique needs in the emergency department

Emergency departments cater for a broad range of people, all with unique healthcare needs. However, some groups are recognised as collectively having specific needs that impact on clinical presentation, resource allocation, clinician experience and education requirements, and their equipment and environmental needs. These groups include those in the community who are more vulnerable, such as children, the elderly, people with an aural or visual impairment, people with a mental illness and victims of violence. Other people with unique needs include obstetric patients, Aboriginal and Torres Strait Islander and Maori people, and people who are culturally and linguistically diverse. Emergency nurses are encouraged to consider the unique health needs of all people presenting to the ED and to address each person's needs in a culturally safe way.

DISASTER NURSING

Definition

The World Health Organization (2015a) defines a **disaster** as:

1. A serious disruption of the functioning of a community or a society causing widespread human, material, economic or environmental losses which exceed the ability of the affected community or society to cope using its own resources (International Strategy for Disaster Reduction (ISDR)).
2. [A] Situation or event, which overwhelms local capacity, necessitating a request to national or international level for external assistance (Center for Research on the Epidemiology of Disasters (CRED)).
3. A term describing an event that can be defined spatially and geographically, but that demands observation to produce evidence. It implies the interaction of an external stressor with a human community and it carries the implicit concept of non-manageability. The term is used in the entire range of risk-reduction activities, but it is possibly the least appropriate for response.

For a disaster to be declared, CRED (2015) states that at least one of the following must be fulfilled:

- ten or more people reported killed
- one hundred or more people reported affected
- declaration of a state of emergency
- call for international assistance.

Emergency Management Australia's *Emergency Glossary* (1998) states that in Australia a disaster is recorded when at least one of the following occurs:

- three or more deaths
- 20 injuries or illnesses
- significant damage to property, infrastructure, agriculture or the environment; or disruption to essential services, commerce or industry at an estimated total cost of A$10 million or more at the time the event occurred.

Types of disasters

In the past, disasters were simply categorised as **natural** or **man-made** (technological), accidental or intentional. As there are more events than fit these definitions, the categories for disasters have been significantly expanded (see Table 6.5). Acts of terrorism are not contained within the new classification.

TABLE 6.5 Classification of disasters

DISASTER GROUP	SUBGROUP	MAIN TYPE
Natural	Geophysical	Earthquake
		Mass movement
		Volcanic activity
	Meteorological	Extreme temperature
		Fog
		Storm
	Hydrological	Flood
		Landslide
		Wave action
	Climatological	Drought
		Glacial lake outburst
		Wildfire
	Biological	Epidemic
		Insect infestation
		Animal accident
	Extraterrestrial	Impact
		Space weather
Technological	Industrial accident	Chemical spill
		Collapse
		Explosion
		Fire
		Gas leak
		Poisoning
		Radiation
		Other
	Transport accident	Air
		Road
		Rail
		Water
	Miscellaneous accident	Collapse
		Explosion
		Fire
		Other

Source: EM-DAT (2015c).

In recent history the Asia–Pacific basin has been affected by a number of significant natural disasters, resulting in 119 disaster events, 6050 deaths and 79.6 million people affected. In Australia in 2015 the Western Australian wildfire (see Figure 6.1) and the New South Wales storm event (see Figure 6.2) resulted in significant losses. The storm resulted in three deaths and 5890 people affected and the wildfires resulted in one death, 450 people affected and 29 homes lost (EM-DAT, 2015b).

Disasters can be unintentional, such as a campfire that has been left unattended and creates a massive bushfire. This is an example of an accidental man-made disaster. Intentional disasters occur when harm and/or destruction is the primary aim of the perpetrators. Most disasters result in the destruction of property, loss of life and/or injury to person.

The potential for a disaster exists at any time when hazardous materials are involved as, by their nature, they pose a potential risk to life, health or property if they are released, due to their chemical, biological or physical nature. The hazard exists during any stage of use, from the production and storage of these substances to their transportation, use or disposal. Hazardous materials are often involved in acts of terrorism.

FIGURE 6.1 ■ Western Australian wildfire 2015

Source: © Sam Edmonds/Corbis.

FIGURE 6.2 ■ New South Wales storm event 2015

Source: andesign101/123rf.

Terrorism

Terrorism is not a modern concept; the earliest recorded acts of terrorism are seen throughout the New Testament of the Holy Bible where the Zealots of Judea waged a campaign against the Romans to free Judea from foreign rule. In Australia **terrorism**. is defined in the Commonwealth *Criminal Code Act 1995*, Part 5.3, Division 100:

> *Terrorist act means an action or threat of action where:*
>
> *a. The action is done or the threat is made with the intention of advancing a political, religious or ideological cause; and*
> *b. The action is done or the threat is made with the intention of:*
> *i. Coercing, or influencing by intimidation, the government of the Commonwealth or a State, Territory or foreign country, or of part of a State, Territory or foreign country; or*
> *ii. Intimidating the public or a section of the public.*
>
> *A terrorist group commits acts of violence to:*
>
> - *produce widespread fear*
> - *obtain worldwide, national or local recognition for their cause by attracting the attention of the media*
> - *harass, weaken or embarrass government security forces so that the government overreacts and appears repressive*
> - *steal or extort money and equipment, especially weapons and ammunition vital to the operation of their group*
> - *destroy facilities or disrupt lines of communication in order to create doubt that the government can provide for and protect its citizens*
> - *discourage foreign investments, tourism or assistance programs that can affect the target country's economy and support of the government in power*
> - *influence governmental decisions, legislation or other critical decisions*
> - *free prisoners*
> - *satisfy vengeance*
> - *turn the tide in a guerrilla war by forcing government security forces to concentrate their efforts in urban areas. This allows the terrorist group to establish itself among the local populace in rural areas.* (Terrorism Research, 2012, para. 5)

Terrorists use both conventional (e.g. bombs, guns) and non-conventional (e.g. chemical, biological and nuclear) means to achieve their end. A terrorist strike is not usually predictable. Government counter-terrorism efforts use surveillance with the aim of preventing or minimising danger to the public; however, there have been situations when surveillance information was not analysed correctly or not passed on to the relevant authorities. Surveillance relates to the collection and analysis of data to determine a change or significant trend in a group (Bradt, Bartley, Hibble, & Varshney, 2015). The goal of the surveillance system is to determine the status of the public's health and detect any sudden change in that status.

Healthcare providers have become a necessary component of terrorism surveillance, especially with biologically based agents. A disaster preparedness plan that outlines the protocol and procedures to be taken with a suspected bioterrorism attack, as well as the response to mass casualty incidents,

should be established in every healthcare facility. Most countries have a central or federal disaster plan; for example, COMDISPLAN 2014 (Australia) and National Civil Defence Emergency Management Plan Order 2015 (New Zealand).

Common disaster-related injuries and related symptoms

Natural disasters

Tsunami can bring water waves travelling more than 800 kph and *cyclones* have winds of up to 200 kph and flooding rains. Most deaths from tsunami and cyclones occur as a result of drowning, collapsed buildings or flying debris which becomes lethal in high winds (Queensland Disaster Management, 2012).

Thunderstorms bring the risk of a lightning strike. The short duration of a lightning strike results in a very short flow of current internally, despite the high voltage of lightning. However, the longer the duration of contact with high-voltage current, the greater the potential for tissue destruction. The greatest conductors of electrical current in the body are the nerves, muscles and blood vessels, which have a high electrolyte and water content. High resistors to electric current are bones, tendons and fat, due to their tendency to heat up and coagulate instead of transmitting current. Much of the energy current may be dissipated at the skin surface, resulting in significant surface burns (Talley & O'Connor, 2013; Thomson & Howard, 2013).

Floods, most commonly caused by heavy rainfall in Australia, can spread over thousands of square kilometres causing injury and destruction. Most flood-related deaths result when people attempt to drive, walk, swim or play in flood waters as the depth and current are easily misjudged.

Bushfires (or wildfires) are a common occurrence in Australia and, with a combination of high temperatures, high winds and dry but plentiful vegetation, can be lethal. The warning system includes a 'Catastrophic alert' category (Code Red) (Australian Government Bureau of Meteorology, 2015). Fire-related injuries include burns, smoke inhalation and toxic fume poisoning, the last being the most common.

Earthquakes have a high incidence of mortality and morbidity due to the multiple injury modalities, as seen in Christchurch in February 2011. The most common health effects experienced by victims of earthquakes include stress-related symptoms; wounds; bone, joint and muscle injuries; burns from explosions; clean-up injuries; gastrointestinal and respiratory problems; aggravation of chronic illnesses; obstetric complications; and death (Lu-Ping et al., 2012). Countries such as New Zealand and Japan are prone to earthquakes as they are situated on the boundaries of two constantly moving tectonic plates which move, causing frequent quakes. The incidence of earthquakes in Australia is low, as it is sitting on a single tectonic plate.

Technological injuries

Explosive or blast injuries are the result of explosive munitions, often involving car or package bombs. Care for people injured by blast injuries typically focuses on abdominal and lung injuries, penetrating wounds, traumatic amputations and burns. The level of injury depends on how close the victim was to the epicentre of the blast. More complex are injuries from dirty bombs, which contain a conventional explosive packed with radioactive waste by-products. When detonated, deadly radioactive particles are released into the environment, spreading in the wind like a dust cloud. In this way, dirty bombs reach far wider areas than the initial explosion (Gaillard, Regenstreif & Fanton, 2014) and cause long-term effects as a result of radiation exposure changing cellular DNA. These changes can result in either cell/organ death or malignant changes within an organ.

Thermal burns are the most common mechanism resulting in injury and death associated with nuclear detonation. Thermal burn injuries can be severe and are treated like any other burn. Radiation suppresses the immune system, so special care must be taken to reduce the potential infection often associated with full-thickness burns (Christensen et al., 2014). More information on burn care can be found in Chapter 16.

DISASTER PLANNING, RESPONSE AND MITIGATION

Disaster planning and risk reduction in Australia is informed by the World Health Organization (WHO) Sendai Framework for Disaster Risk Reduction 2015–2030, the outcomes of which are stated as:

> *The substantial reduction of disaster risk and losses in lives, livelihoods and health and in the economic, physical, social, cultural and environmental assets of persons, businesses, communities and countries.* (WHO, 2015b, p. 12)

Disaster preparedness has been a priority issue for the Australian government. There are federal and state disaster plans which involve defence forces, public health services, State Emergency Services, police, and fire and ambulance services. The main federal coordinating bodies are the Emergency Management Australia (EMA) and the Australian Disaster Information Network (ADIN). There are disaster plans in each Australian state and the Commonwealth Government Disaster Response Plan (COMDISPLAN) (Emergency Management Australia (EMA), 2014) is the framework for states and territories to request Commonwealth assistance arising from any type of emergency.

EMA has created detailed manuals containing information about emergency management, such as *Aspects of Chemical, Biological and Radiological Hazards* (EMA, 2000) and *Flood Preparedness* (Australian Attorney-General's Department, 2009). These guides are a resource for individuals and families to form a disaster plan.

In addition, the handbook *Disaster Health* (Australian Emergency Management Institute, 2011) is another invaluable source for Australasian nurses.

Stages and phases of a disaster

Warfield (2015) outlines the *goals of disaster management* as follows:

1. Reduce, or avoid, losses from hazards.
2. Assure prompt assistance to victims.
3. Achieve rapid and effective recovery.

There are four disaster management phases to achieve the above goals. The length of each phase greatly depends on the severity of the disaster.

1. *Mitigation*: reducing the effects of disaster
2. *Preparedness*: preparing and planning a response in advance
3. *Response*: putting the plans into action so that hazards are minimised and the needs of the victims met
4. *Recovery*: restoration of the community to a pre-disaster functional level.

Mitigation occurs when actions are taken that eliminate or reduce the chance of a disaster happening or reduce the effects of an unavoidable disaster. Information such as countermeasures and emergency risk are critical for mitigation to be successful. Appropriate building codes, vulnerability analyses and public education all contribute to mitigation.

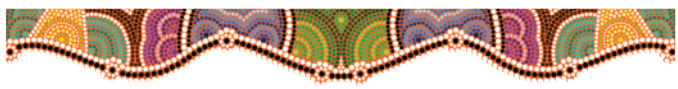
Preparedness is having a comprehensive disaster plan in place that coordinates the efforts of public and private organisations such as the State Emergency Services, the military and all levels of government. Each organisation has specific roles and is able to mobilise quickly and effectively. This level of readiness enables a community to respond to any emergency situation.

The plan will be based on familiarity with possible disaster agents based on previous experiences, as well as experiences of others from various regions and countries. It is imperative that all people and agencies who may be involved in the disaster response be involved in the planning. In this way, information is shared and representatives from each agency explain and offer their respective resources and expertise and note deficiencies in the plan. Planning committees will exist on all levels—federal, regional, state, local and individual agency. Nurses participate in this facet of disaster planning by having a nurse representative on the planning committee at least at the agency level.

Response to disasters happens in the emergency stage and after the disaster event has occurred. The purpose of the emergency response is to act to maintain life, maintain health and evaluate and respond to the psychological needs of the affected community. The community has been rapidly assessed for damage, and the types and extent of injuries suffered, as well as the immediate needs of the community, have been determined. Hospital disaster planners must plan for the possibility that the next disaster may involve the hospital. The hospital's response may include the evacuation of people receiving care as well as relocating and operating from an independent facility. Surge capacity is the healthcare system's ability to rapidly expand beyond normal services to meet the increased demand for qualified personnel, medical care and public health in the event of a large-scale disaster. The aims of the Australian Government Department of Health and Ageing (2011) *National Health Emergency Response Arrangements* (2011) are to:

- outline the strategic authorities, responsibilities, arrangements and the mechanisms that enable a coordinated national health sector response to emergencies of national consequence;
- inform and guide a coordinated Australian health sector response to, and recovery from, emergencies of national consequence; and
- provide a strategic planning framework for guidance to the future revisions of existing health sector emergency plans.

Recovery is the final phase and, as the word implies, it is the period when the emergency is under control and the community starts to rebuild. In this phase the resources established in the disaster plan may be put into action. The recovery depends on the ability of the resources to meet the needs of the community affected. In many disasters the available resources need to be supplemented by government and aid agencies. There is no distinct point at which immediate relief changes into recovery and then into long-term sustainable development. There is no set period for the recovery phase; this phase depends on the type of disaster. It can take years for a community to recover; some never return to their pre disaster state.



Nursing care

The role of the nurse in a disaster

Disaster nursing is a relatively new discipline internationally; however, it should be remembered that nurses have been on the front line of wars and disasters for over a century. The International Red Cross has deployed nurses to conflict zones since 1919. The World Association for Disaster and Emergency Medicine (WADEM), an international multidisciplinary organisation, established a nursing chapter in 2004. The role of nurses in a disaster situation in Australia is not clearly defined but depends greatly on a number of variables, including the nature of the disaster; the number of victims and severity of injuries; the location of the disaster and of the nurse; and the availability of supplies, rescue and command personnel, and other necessary resources. The nurse must be able to perform under stressful conditions but will not be expected to endanger self, other nurses or other rescuers. It is important to remember that during a disaster access to normal services such as transport, communication, interpreters, food and water, and even medical care is going to be disrupted and at times not available. In addition, your colleagues may not react or respond to the disaster as you might expect.

The emergency nurse may be involved in first-line response (i.e. attending the disaster site) or be responsible for individuals attending the ED for stabilisation. All Australian hospitals have a disaster plan and increasingly these are put to the test.

Nurses may have to assume expanded roles in making decisions for the most appropriate treatment of casualties. Discussions should take place among doctors, nurses and policy makers regarding the necessity of nurses' expanded roles in crisis situations. Additionally, healthcare personnel should receive specialised training in order to be safe and competent practitioners of the expanded duties. This training must be practised and updated and those participating in the training

BOX 6.1 Key triage points to remember in mass casualty management

- Use a triage system that is easy to learn, easy to implement in stressful conditions and does not require advanced diagnostic skills yet allows for basic interventions.
- Use the Incident Management System on every incident and wear personnel identification vests.
- Get accurate preliminary and final headcounts and relay this information to the incident commander.
- Use some type of visual colour-coded identification system to indicate each person's priority.
- Do not fall into the trap of using your time by providing one-to-one care.
- Re-triage individuals frequently, at the incident, on arrival at the treatment area and periodically thereafter.
- Make certain the 'walking-wounded' are gathered and treated.
- Pre-plan for potential incidents that may occur.
- Be aware that emergency responders may be potential targets.
- Practise, practise, practise.

Source: Adapted from Prehospital triage by M.R. Streger (1998) in *Emergency Medical Services, 21*(6), 23–27, 45.

must be tracked and notified of additional requirements as necessary. Nurses take on a variety of roles based on their expertise and the needs of the victims. Nurses will be expected to follow the emergency preparedness plans outlined in their communities and in their agencies of employment. It is critical that nurses work within their scope of practice, as victims of a disaster will present with a variety of individualised needs including anxiety, impaired verbal communication, ineffective or impaired coping, fear, post-trauma syndrome, powerlessness, injury and trauma.

Mass casualty management

During a disaster, nurses may be expected to triage victims. The nature of triage can change depending on the type of disaster and the number of victims; however, the system described in Table 6.3 is initially used. When the number of casualties is around 100, then **reverse triage** may be instigated; that is, those with the best chance of survival are transported first (Lerner et al., 2015). Many emergency personnel will share the difficulty of making these decisions at disaster sites when the first inclination might be to rescue the most severely injured. Box 6.1 shows key triage points to remember.

Isolation and personal protective equipment

People suspected of having a highly contagious disease, such as H2N2 influenza, will need to be isolated to prevent spread of the disease to others. Airborne protection can be achieved with negative-pressure ventilation. All people entering the room should wear personal respiratory protective devices capable of filtering submicron particles. Decontamination of the air may be achieved through ventilation and supplemented by ultraviolet light (Heymann, 2004).

Gas masks are used in a broad range of military, industrial and emergency situations to protect the user from hazardous dust, gas or other aerosols. Biological contaminants that are spread through aerosolised droplets create a threat to those not wearing personal protective equipment (PPE). A gas mask may be considered a high-performance respirator and is usually equipped with both eye protection and air supply protection or treatment. Protective clothing is made to guard against mild irritants and even serious lethal materials. Some protective suits are disposable, intended for one use only. Others are durable, multilayered fabrics that are completely impermeable and are reusable. Each state disaster plan has issued guidelines to inform healthcare workers and first responders about the correct level of PPE for various situations (Australian Radiation Protection and Nuclear Safety Agency, 2014).

The recommendations by the US Environmental Protection Agency (EPA) (2015) for personal protection clothing are listed in Box 6.2.

In addition to the isolation of individuals, special air handling systems are used in the isolation rooms to prevent the spread of the contaminated droplets into the general hospital air vents. Many hospitals have the capability to shut off airflow in contaminated areas to prevent the spread of contaminants to other 'clean' areas of the hospital. The heating, ventilation, air conditioning and refrigeration (HVAC) systems are closely monitored and can be shut down in designated areas to avoid air intake from the outside as well, especially in cases of outdoor environmental contamination.

Recording victim data

Each health area has a trauma sheet to record data about victims of disasters. The categories on the data sheet include demographics, circumstances of the injury, injury conditions, and disposition and details of the conditions. The completion of this form will be initiated by the triage nurse and completed by the nurse who implements the treatment or transfers the injured person to another unit/department.

Psychosocial needs

The importance of mental health services for victims, the public, first responders and healthcare workers cannot be overstated in both emergency and disaster situations. People react to disasters in a variety of ways, both physically and behaviourally. Their reactions depend on the severity of their injuries, real and perceived threat and their proximity to the area of direct impact. The closer the person is to the area of impact and the longer the exposure, the greater the likelihood of a more severe reaction to the event.

BOX 6.2 EPA levels of protective clothing

Level A protection is required when the greatest potential for exposure to hazards exists and when the greatest level of skin, respiratory and eye protection is required. Examples of Level A clothing and equipment include:

- positive-pressure, full-face-piece self-contained breathing apparatus (SCBA) or positive-pressure supplied-air respirator with escape SCBA
- totally encapsulated chemical- and vapour-protective suit
- inner and outer chemical-resistant gloves
- disposable protective suit, gloves and boots.

Level B protection is required under circumstances requiring the highest level of respiratory protection, with lesser level of skin protection. Examples of Level B protection include:

- positive-pressure, full-face-piece self-contained breathing apparatus (SCBA) or positive-pressure supplied-air respirator with escape SCBA
- inner and outer chemical-resistant gloves
- face shield
- hooded chemical-resistant clothing
- coveralls
- outer chemical-resistant boots.

Level C protection is required when the concentration and type of airborne substances is known and the criteria for using air-purifying respirators is met. Typical Level C equipment includes:

- full-face air-purifying respirators
- inner and outer chemical-resistant gloves
- hard hat
- escape mask
- disposable chemical-resistant outer boots.

Level D protection is the minimum protection required. Level D protection may be sufficient when no contaminants are present or work operations preclude splashes, immersion or the potential for unexpected inhalation or contact with hazardous levels of chemicals. Appropriate Level D protective equipment may include:

- gloves
- coveralls
- safety glasses
- face shield
- chemical-resistant, steel-toe boots or shoes.

Source: US Environmental Protection Agency (EPA) (2015). *Personal Protection Clothing.* www2.epa.gov/emergency-response/personal-protective-equipment.

Table 6.6 summarises the normal initial responses aimed at survival (Murray, Zentner & Yakimo, 2008; Selye, 1965; 1980; Wilson & Raphael, 2013).

Special considerations

Disasters are not selective in their victims; anyone in the target zone will be affected: young, old impaired or healthy. As in most emergency situations the very young and old are likely to be more vulnerable. Issues such as limited mobility and access to resources can impact on the younger and older person's ability to react to the crisis. The notion of community becomes important. Community knowledge, such as knowing where older people or young families live in your area, can help first responders to assist these victims and evacuate them to safety. When injured, the older person may have fewer physiological reserves due to the ageing process, and their chances of full recovery are reduced in accordance with the amount of time it takes to be rescued. The very young are dependent for all basic needs and have limited physiological reserves. If separated from their carer they risk further harm. Therefore, restoring a carer's role is critical.

TABLE 6.6 Responses to stress: General Adaptation Syndrome (GAS) and levels of anxiety

GAS STAGE	PHYSICAL RESPONSE	BEHAVIOURS RELATED TO ANXIETY
Alarm stage	Pupils dilate; blurred vision. Hearing sharper or diminished.	Misinterpret stimuli. Confusion. Poor concentration. Selective inattention. Need for assistance.
Severe anxiety or panic	Stronger, faster heart rate and respirations. Palpitations, arrhythmias, elevated blood pressure.	Feeling of impending doom. Terror. Fearful. Agitation Irritability. Demanding. Impulsive. Paraesthesias.
	Muscle tone increased. Headaches.	Muscle tension. Excitable, restless movements. Tremors. Rigidity. Weakness.
	Basal metabolism rate increased. Body temperature elevated. Perspiration. Altered glucose, protein and lipid metabolism. Increased startle response.	Insomnia. Urgency of speech and movement. Fatigue. Dehydration. Weight loss. Appetite changes. Smooth muscle of gastrointestinal and urinary tracts less motile, interfering with digestion and elimination of wastes.
	Hypoglycaemia from glycogenolysis due to high energy demands.	Blood glucose increase. Appetite changes. Dehydration. Fatigue. Poor concentration.
	Increased blood clotting and suppressed immune response if stage persists.	Blood stasis; thrombus formation. Resistance to infection and disease reduced.

Source: Based on K. S. Quigley (2010). General adaptation syndrome. *Corsini Encyclopedia of Psychology*, 1.

Each age group will have unique reactions to a disaster. Teenagers may take unnecessary risks such as crossing a flooding river—these behaviours result in an increase in the death and injury toll.

PEOPLE WITH MOBILITY AND SENSORY DEFICITS The Australian Institute of Health and Welfare estimates that one in four Australians has some form of activity limitation due to a chronic condition. Many people require the use of assistive technology devices to accommodate mobility and other impairments. Careful planning must be in place in order to provide necessary support to this group during and after a disaster. Volunteers or staff can assist with relocation to a safe room or shelter when required; however, relocation can be problematic when access to this group of people is limited by the disaster. These individuals or their caregivers must provide input to service personnel to determine what kind of support services would be necessary in an emergency or disaster.

NON-ENGLISH-SPEAKING PEOPLE Providing information in a variety of languages is not unusual in Australian culture; however, information regarding actions to be taken during a disaster is often provided in English only. In addition, one cannot assume that people are literate in their own language. It is ideal to obtain the assistance of an interpreter who can translate information for those who don't speak English. Communication aids can be prepared in advance of disasters to be used during emergencies. The use of visual aids is very helpful.

IMMUNOCOMPROMISED PEOPLE The immunocompromised population is at greater risk of complications and death than the general population. Bottled water should be ready so the immunocompromised person can avoid drinking water of questionable purity. It is safest for this population of individuals to consume processed or tinned foods if they can be heated to the proper temperatures.

SPIRITUAL CONSIDERATIONS Religion tends to be a source of comfort for those who are experiencing the threat of loss of life, property or way of living. Churches, mosques, synagogues and religious leaders become active in supporting their congregations in times of disaster. Religious leaders should be actively involved in community planning for disaster preparedness, especially if certain religious considerations should be strictly followed where possible. In times of uncertainty many individuals turn to their religion for answers and support. Access to normal spiritual activities and support may be interrupted and in some cases it falls to the nurse to provide spiritual care. The nurse does not need to believe in the same way the victim does; however, assisting them to carry out a religious ritual will assist in that person's recovery or journey to death.

Community-based care

Nurses are invaluable in disaster relief efforts, whether they are answering questions from their neighbours regarding the water supply in their communities or assisting in complex care using their advanced practice knowledge in a hospital.

Nurses have a responsibility to the public to maintain competence in nursing practice and to be cognisant of evolving threats to the public's health. Nurses must learn about anticipated and unexpected disasters, as well as their vital roles in emergency preparedness and response. Nurses will play a role in disaster response whether they work in acute care settings, long-term care, ambulatory care or in the community or at home.

Teaching about disaster preparedness and appropriate actions is important in all communities. The Australian government has become more active in educating the public at all stages of a disaster, as seen in the guides mentioned earlier. Disaster preparedness and response procedures should be practised on a regular basis prior to an emergency. A multitude of communication means are available through technological support systems, such as internal phones and handheld communication devices. Written and visual cue boards are often more useful because they are not reliant on power. However, these methods of communication have been challenged by recent disasters in Japan and the Black Saturday fires in Victoria, but when services are limited adaptation will occur.

NURSING CARE PLAN A person with spinal injuries from being blown off a roof during a storm

Michael Vaughan, a 29-year-old carpenter, is married to Jacqui, 27; they have an 18-month-old daughter Grace. The couple has just purchased an older house in the city that needs a great deal of work. Michael is fit and well and cannot remember the last time he needed to have medical assistance. During a heavy rainstorm with high winds, the roof started leaking. To protect his house Michael climbed onto the roof to seal the leak. He did not secure himself on the roof and a gust of wind blew him to the ground, a fall of approximately 6 metres. He was transported to hospital by ambulance on a spine board with a (hard) cervical collar fitted. At the hospital's ED he was given a triage category of 2 because of the potential spinal injuries and height of his fall.

ASSESSMENT

Lisa Smith, RN, obtains a nursing assessment, as follows:

Primary survey

D(anger): Michael's cervical spine is secured and the spinal board is maintaining spinal alignment

R(esponse): Michael is alert and oriented to his surroundings

NURSING CARE PLAN A person with spinal injuries from being blown off a roof during a storm (continued)

A(irway): Michael is able to talk articulately and without distress—airway patent
B(reathing): Michael has equal air entry on both sides, chest rising is equal, R 26, SaO_2 96% on room air
C(irculation): Michael is pink and well perfused. All major pulses present, strong and equal. Right dorsalis pedis pulse is weak and thready. Capillary return is < 2 secs except on the right leg below the knee. HR 122, BP 110/60
D(isability): GCS 15. Non-visual pain scale 8

Secondary survey

Head: Conscious and alert, no lacerations on head—Cx collar in place.
Torso: Multiple bruises appearing on the right side, no tenderness over liver, spleen, kidneys, bowel sounds present and normal, no evidence of deformity to the chest cavity. Back examination by medical staff (log rolled) shows bruising on right side, no palpable deformity of spine or spinous processes.
Limbs: Arms normal in appearance and range of motion, no pain on palpation, no deformity seen. Bruising on right shoulder. Left leg and foot normal with no deformity or pain. Right leg is pale, cool, reduced sensation to touch, unable to move without severe pain.

Michael does not accept that he needed to take precautions when attempting to fix the roof or that calling the SES may have been a better option.

Focused assessment (spine and right leg)

X-rays rule out any spinal injury or concerns about the cervical spine.

Examination of the right leg shows bruising on anterior surface. Slight deformity 10° laterally. Gentle palpation reveals disruption mid-shaft tibia and fibula.

A peripheral IV is initiated with continuous fluids, and IV antibiotics are ordered every 8 hours. Michael is now fasting.

DIAGNOSES

- *Acute pain* related to right leg injury evidenced by pain score of 8.
- *Impaired circulation* to right leg related to displaced and separated fracture mid-shaft tibia and fibula evidenced by changes in colour, warmth, sensation and movement (CWSM).
- *Risk of hypovolaemia* due to internal haemorrhage related to leg fractures.

Expected outcomes

- Pain relieved within 15 minutes with narcotic IVI.
- Return of circulation to right lower leg—surgical intervention required.
- Mobility restored—surgical intervention required.
- Remains normovolaemic.
- Skin remains intact.
- Michael understands his role in this situation and does not take any further risks.

PLANNING

- Monitor Michael's heart rate and rhythm, blood pressure and respiratory rate every half hour.
- Arrange orthopaedic review for management of fractured tibia and fibula.
- Assess Michael's signs and symptoms of pain and administer pain relief as required and prescribed.
- Observe for internal bleeding.
- Maintain hydration: monitor intake and output.
- Inspect posterior skin every 4 hours, document skin condition, report changes.
- Arrange education in regard to home safety.

Expected outcomes

- Pain relieved within 15 minutes with narcotic IVI.
- Return of circulation to right lower leg—surgical intervention required.
- Mobility restored—surgical intervention required.
- Remains normovolaemic.
- Skin remains intact.
- Michael understands his role in this situation and does not take any further risks.

IMPLEMENTATION

- Assess and record Michael's heart rate and rhythm, blood pressure and respiratory rate every half hour.
- Administer IVI narcotics as per pain score and medication orders.
- Monitor and record pain scores every 30 minutes.
- Prepare Michael for surgery.
- Monitor CWSM every 15 minutes until surgery then 30/60 for 4 hours, 1/24 for 24 hours.
- Post-operative rehabilitation to restore pre-incident mobility.
- Provide education in regard to home safety.

EVALUATION

Michael was transferred to surgery for internal fixation of right tibia and fibula. He was admitted to the orthopaedic unit for 7 days then discharged home in a POP cast and non-weight-bearing on crutches.

CRITICAL THINKING IN THE NURSING PROCESS

1. Explain Michael's initial observations in relation to his pain and injury.
2. What post-surgical observations would indicate neurovascular compromise and what are the appropriate nursing interventions?
3. What are the major physiological considerations for Michael?
4. What could impede healing in Michael's situation?

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study.
2. How will you apply the systematic assessment process described in this case study in your future nursing practice?
3. What cues were important for the critical decision making?
4. How did you determine priority?

CHAPTER HIGHLIGHTS

- Triage in the ED is the process of sorting people using medical acuity as its basis.
- Emergency assessment consists of three stages: primary assessment, secondary assessment and a focused assessment.
- ED nursing interventions are focused on stabilisation and disposition.
- Disasters require extraordinary efforts beyond those needed to respond to everyday emergencies.
- Reverse triage is used in mass casualty events instead of traditional triage in order to do the greatest good for the greatest number.
- Nurses will be actively engaged in assessing the physical as well as the mental needs of victims, their families, first responders and other healthcare personnel.
- Nurses are actively involved in disaster mitigation, planning and response efforts by learning and practising their communities' and agencies' disaster preparedness systems.

CONCEPT CHECK

1 The primary aim of the Australasian Triage Scale is to:
   1 ensure appropriate allocation of funding to the ED
   2 ensure that ED care is delivered according to time of arrival to the ED
   3 ensure that ED care is delivered according to clinical need
   4 prevent ED overcrowding

2 The primary function of the ED triage nurse is to:
   1 provide first aid to people presenting to the ED
   2 ensure that ED care is delivered according to time of arrival to the ED
   3 assess and prioritise patient care
   4 provide education and redirect people to appropriate community services

3 During the primary assessment of a collapsed victim, the nurse determines that the person has a patent airway. The next assessment the nurse makes includes:
   1 the level of consciousness
   2 observation for external bleeding
   3 the status of the person's respiration
   4 the rate and character of carotid or femoral pulses

4 A man is brought into the ED via ambulance following a high-speed motor vehicle accident. He is unconscious, and bleeding profusely from the arm. In planning care for this man, the nurse gives the highest priority to the goal of:
   1 maintaining the person's airway
   2 assisting the person's breathing
   3 controlling the bleeding
   4 maintaining the person's fluid volume

5 The key difference between emergencies and disasters is that:
   1 emergencies are controlled
   2 disasters result from man-made errors
   3 emergencies can typically be handled by available emergency services
   4 disasters typically involve the local emergency services and no other agencies

6 The purpose of reverse triage is to:
   1 save scarce resources for future use
   2 test first responders on their triage classification categories
   3 save those people who are in the most critical condition
   4 do the greatest good for the greatest number with limited resources

7 Which of the following is true about personal protective equipment (PPE)?
   1 PPE protects by creating a barrier against hazards
   2 eye, face, head, foot and hand protection are addressed in PPE programs
   3 PPE should reduce the likelihood of occupational injury and/or illness
   4 healthcare workers do not need to wear PPE if they follow strict handwashing protocol and universal precautions

8 The goals of disaster management are:
   1 preparedness, response and recovery
   2 mitigation, response and recovery
   3 reduce hazards, prompt assistance, effective recovery
   4 mitigation, reduce hazards, prompt assistance, effective recovery

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UNIT 2 BUILDING CLINICAL COMPETENCE

Alterations in patterns of health

CLINICAL SCENARIO

You have been assigned to care for the following four people for the 0700 shift in an emergency department (ED). Significant data obtained during report are as follows:

- Peter Black is a 46-year-old who was admitted to the ED 2 hours ago after being thrown 20 metres during a cyclone. When last taken, his vital signs were T 37.6°C, P 86, R 24 and BP 140/86. He had multiple abrasions and lacerations that were sutured in the emergency room. He is now complaining of numbness in both legs.
- Alice Jones is a 19-year-old woman with a history of asthma. She has not been using her preventer, and came to the ED 4 hours ago complaining of shortness of breath. She has been having salbutamol nebulisers every 30 minutes. Her vital signs are T 37.2°C, P 90, R 26, BP 134/88, SpO_2 95% on room air.
- John Linner, aged 67, was brought in by his family 3 hours ago. He is in the terminal stages of colon cancer, and the family are no longer able to care for him at home. Vital signs are T 36°C, P 54, R 10, BP 88/68. The family is requesting that a nurse check on Mr Linner because they feel that death is imminent.
- Paul Gregs, aged 47, was brought in by police after he was seen standing in the middle of the road directing traffic. He has a history of alcohol abuse, and on admission his alcohol level was 0.45. Current vital signs are T 36.7°C, P 110, R 30, BP 168/94. He is confused, agitated and diaphoretic.

Critical thinking questions

1 In what order would you review these people after handover?

1.
2.
3.
4.

2 What top two priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Peter Black		
Alice Jones		
John Linner		
Paul Gregs		

3 After assessing Mr Black, which nursing intervention should the nurse perform first?

1. Call the doctor to report the numbness in his legs.
2. Have Mr Black perform active exercises to prevent thrombosis in his legs.
3. Ambulate Mr Black in the hall to promote circulation to his legs.
4. Medicate Mr Black for pain so he can move his legs better.

4 Outline the elements of the primary and secondary survey, and describe how you would apply them in relation to Peter Black.

5 What are the changes that occur in the airway with asthma?

1. Oedema, bronchoconstriction, increased mucus production
2. Oedema, bronchoconstriction, decreased mucus production
3. Oedema, bronchodilation, decreased mucus production
4. Oedema, bronchodilation, increased mucus production

6 Describe the focused respiratory assessment you would undertake on Alice Jones.

7 Prioritise four nursing interventions for Alice Jones.

1.
2.
3.
4.

8 With a history of alcoholism for 5 years, what is a priority nursing intervention in the plan of care for Mr Gregs?

1. Identify maladaptive behaviours that may contribute to the alcoholism.
2. Encourage participation in therapeutic group activities.
3. Teach the effects of alcohol on the body.
4. Use a respectful, non-judgmental approach to gain trust.

9 A prescription for naltrexone is given to Mr Gregs. He voices understanding of how to take the medication when he states:

1. 'I must avoid all forms of alcohol and narcotics while taking this medication.'
2. 'It is all right to take over-the-counter cold medications if I catch a cold.'
3. 'This medication will keep me from having withdrawal symptoms.'
4. 'I can get physically ill if I drink alcohol while taking this medication.'

10 When Mr Gregs was admitted to the ED, which laboratory studies would you expect to have taken? (Select all that apply.)

1. bilirubin
2. serum electrolytes
3. ALK
4. complete blood cell count
5. AST
6. WCC

11 To prepare the family for Mr Linner's death, the nurse institutes the following interventions:

1. Teach the stages of coping with the loss of their family member.
2. Explain the physical symptoms they may see as death approaches.
3. Discuss funeral and burial arrangements with the family.
4. Refer family to appropriate support groups to assist in dealing with death.

12 When preparing for the role the nurse will play in disaster relief, the nurse must first:

1. be able to apply basic first aid skills
2. be aware of decontamination procedures
3. serve on disaster preparedness committees
4. know how to take care of himself or herself

CASE STUDY

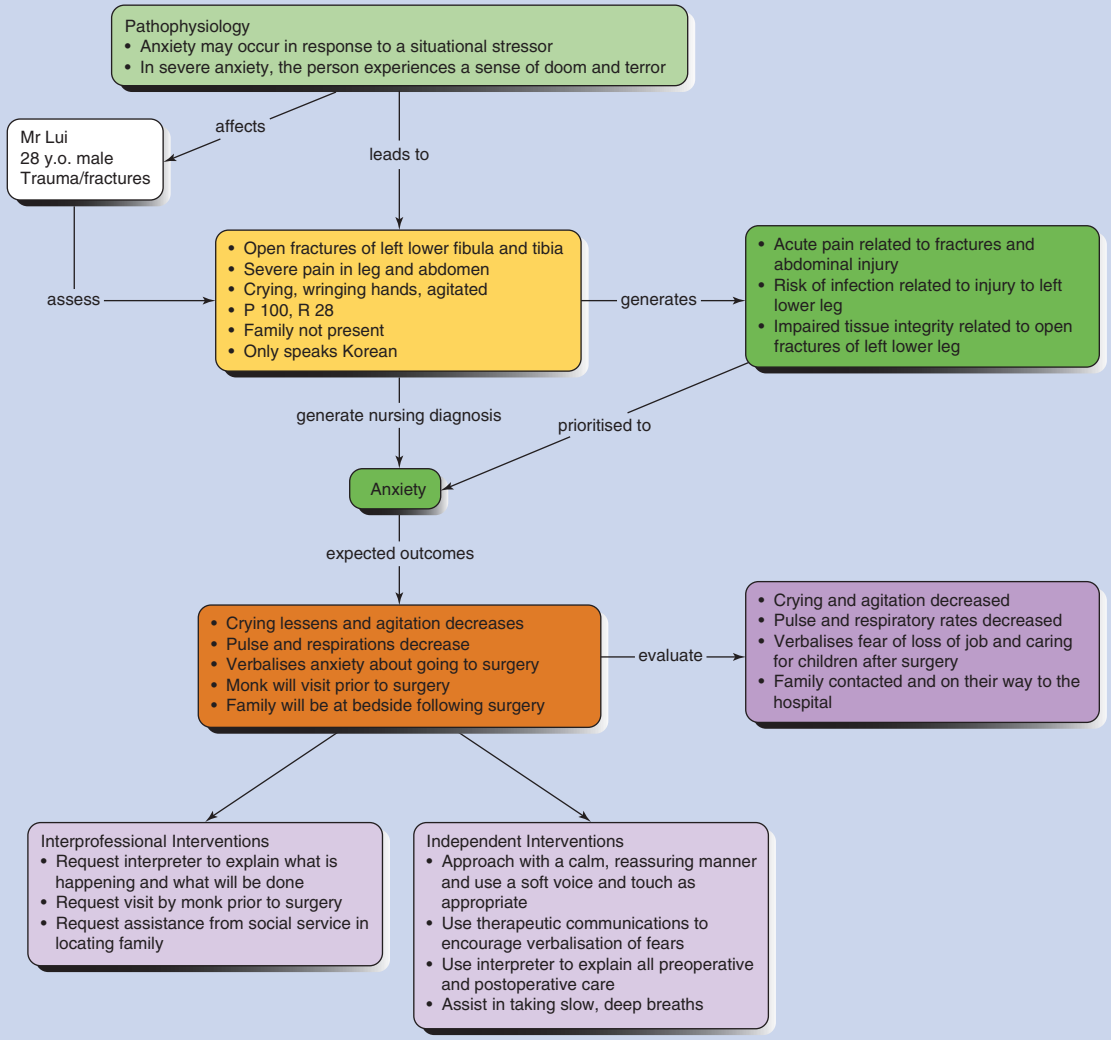
Mr Kim Lui is admitted to the emergency department with open left fractures of the tibia and fibula, left upper quadrant pain and reddened areas across the left shoulder, neck and chest. According to the paramedics, he was involved in a multiple-car crash on a freeway. On initial assessment he is found to be a non-English-speaking Korean, 28 years of age, is 1.8 m in height and weighs 54 kg. His vital signs areT 37°C, P 100, R 28 and shallow, BP 150/86. He indicates his pain scale level as 9 out of 10, even after being medicated with morphine in the ambulance.

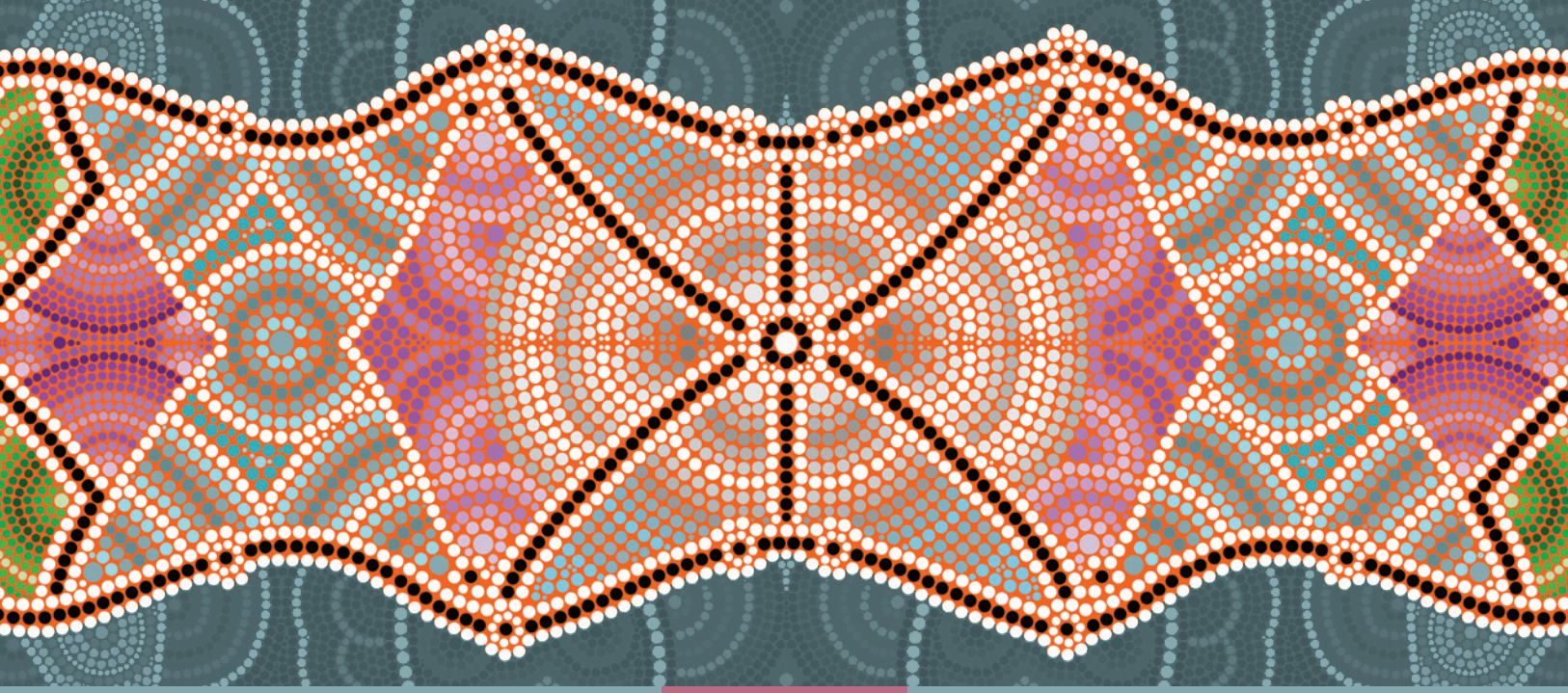
After chest, abdominal and leg x-rays, Mr Lui is diagnosed with crushing injury and comminuted fractures of the left tibia and fibula, a haematoma on his spleen and bruising across the shoulder, neck and chest due to the seat belt. With the use of a translator, the doctor explains that he will have to have surgery to repair his fractures.

Blood is taken for the following pathology tests: full blood count (FBC) with differential, electrolytes, prothrombin time (PT), partial thromboplastin time (PTT) and blood gases. A urine specimen is sent to the laboratory for a urinalysis. Preoperative preparation is completed. The nurse tries to answer any questions Mr Lui has regarding the surgery and what will happen in the postoperative period. The nurse attempts to contact family members and a monk to see Mr Lui before he goes to surgery. At the ordered time, the nurse administers prescribed preoperative medication and Mr Lui is sent to the operating room.

Due to difficulty understanding the English language, pain and the need for surgery, Mr Lui is very anxious. The nursing diagnosis of *Anxiety* is appropriate for guiding preoperative nursing care. Anxiety is an uneasy feeling of not knowing what is going to happen. The pathophysiology of anxiety is anticipation of danger or a threat to health status that leads to a 'fight-or-flight' response from the sympathetic nervous system. Manifestations of anxiety are restlessness, tachycardia, rapid breathing, facial flushing, increased perspiration, weakness, tremors, and impaired attention and concentration. Complications of anxiety are nausea, vomiting, diarrhoea, loss of appetite, insomnia, immobility and powerlessness that can lead to panic or phobias.

Based on Mr Lui's medical diagnosis and treatment plan, anxiety is identified as the priority nursing diagnosis at this time.





UNIT 3

PATHOPHYSIOLOGY AND PATTERNS OF HEALTH

CHAPTER 7

GENETIC IMPLICATIONS OF ADULT HEALTH NURSING

KAMAREE BERRY

KEY TERMS

LEARNING OUTCOMES

- Discuss the role of genetic concepts in health promotion and health maintenance.
- Apply knowledge of the principles of genetic transmission and risk factors for genetic disorders to nursing practice.
- Describe the significance of providing education about genetics and counselling follow-up in an ethically justifiable manner.
- Identify the implications of genetic advances for nursing practice with particular attention to spiritual, cultural, ethical, legal and social issues.
- Identify the significance of recent advances in human genetics in terms of healthcare delivery.

CLINICAL COMPETENCIES

- Integrate genetic physical assessment and the use of a pedigree family history into nursing care according to the NMBA professional standards (2016) required of the RN.
- Identify people or families with actual or potential genetic problems and initiate referrals to appropriate professionals according to the relevant nurses' scope of practice and NMBA professional standards (2016) for the RN.
- Prepare people and their families for a genetic evaluation and facilitate the genetic counselling process according to the relevant nurses' scope of practice.
- Integrate genetic concepts into education for people and their families.

OVERVIEW OF GENETICS AND GENOMICS

Increasingly nurses and midwives encounter people with conditions related to genetics and thus they need the knowledge, skills and attitudes to effectively care for these people in culturally safe and ethically justifiable ways. The completion of the Human Genome Project (HGP) (see Box 7.1) has accelerated the incorporation of genetics into mainstream medical, midwifery and nursing practice. The HGP changed not only the way disease treatment is approached but, more importantly, how nurses and midwives look at health promotion and health maintenance. Thanks to the HGP we now have a much better idea of the contribution of genetics to illness and wellness, targeted drug therapy (**pharmacogenetics**) and the development of genetic tests that identify those at risk of specific illnesses. We know now that most illnesses have a genetic component. We know that myocardial infarction, some forms of cancer, mental illness and diabetes, as well as addiction and Alzheimer's disease are all caused by the interaction between at least one gene and complex environmental and social factors.

The explosion of knowledge about genetics and genomics brings significant nursing, medical, ethical and legal concerns, which all health professionals must consider. With the ability to predict the risk of developing many conditions comes a better understanding of how genetic and environmental and lifestyle factors interact to produce disease. In addition, we have the capacity to tailor treatments based on an understanding of the genetic basis of disease in the individual, and in all likelihood this will reduce the burden of these conditions on society and individuals. For example, population-based screening for colorectal cancer guidelines recommends that screening begin at age 50. It is now possible to adjust the guidelines to take into account genomic risk factors such as having a first-degree relative (parent, sibling or child) who developed cancer at a younger age, or people with multiple affected first-degree relatives so that screening begins at an earlier age.

People and populations are increasingly offered predictive tests and carrier tests to determine if they are at risk of conditions such as Alzheimer's disease (AD), which is a major cause of disability in the older adult. Most AD cases are late in onset and are undoubtedly influenced by a combination of genetic and environmental factors. Inheritance plays a role in approximately 80% of cases (Rao, Degnan & Levy, 2014) and has a risk association with the APOE 4 allele. The social and ethical implications of this knowledge are enormous. Would you want to know if you are at risk of Alzheimer's disease? Would you tell your life insurer? If you knew that you are at a genetic risk of heart disease, would you be more likely to change your lifestyle? Should testing for Alzheimer's disease or Down syndrome be compulsory? If the tests are 'positive', should an affected foetus be aborted by state order because of the financial costs of lifelong care? These questions show the financial price we pay as individuals and members of society for information about our future health risks, as well as the social consequences, including unfair discrimination with regard to life insurance and employment.

DNA is at the centre of the state of our health (see Figure 7.1). We know that wellness and good health are associated with properly structured and functioning genes. If they are not functioning properly, ill health or an increased risk of disease can result. This includes not only the well-known genetic disorders and problems, but also complex conditions such as heart disease, stroke, diabetes and several kinds of cancer. The knowledge gained from human genome research has and will have a profound impact on the prevention, diagnosis, prediction and treatment of genetic disorders and complex diseases.

BOX 7.1 Human Genome Project

The Human Genome Project (HGP) is one of the great accomplishments in medical history. Funded in the United States by the National Institutes of Health and the Department of Energy in 1990, the mission of the now completed HGP was to map the complexities of chromosomes and the genes within them, and how they affect human health.

The ultimate goal was to sequence the human genome and identify all human genes. The completion of a high-quality reference sequence was announced in April 2003, marking 50 years since the publication in the journal *Nature* of the letter by James Watson and Francis Crick describing DNA's double-helix structure. Information obtained through the sequencing of the human genome has had a tremendous impact on finding the genes associated with human illness and health. Now that the HGP has been completed, research is being directed towards understanding the complex functions of cellular regulation, human variation, and the interplay of genes and environment, and how all the cell organelles, genes and proteins work together in life's functions (USDOE Genome Programs, 2008).

INTEGRATING GENETICS AND GENOMICS INTO NURSING PRACTICE

Genetics generally focuses on one gene, whereas **genomics** involves multiple genes as well as interacting factors such as environmental conditions and cultural and social influences, all of which affect the 'expression' or triggering of individual or groups of genes. Genetics and genomics have revolutionised how people perceive themselves, as well as their health status and their health potential. Therefore, nurses and midwives must integrate new genetic knowledge into practice. In 2012, the American Nurses Association (ANA) and International Society of Nurses in Genetics (ISONG) published the *Statement on the Scope and Standards of Genetics Clinical Nursing Practice* defining 38 competencies under seven major categories (ANA/ISONG, 2012): (1) risk assessment and interpretation, (2) genetic education, counselling, testing and results interpretation, (3) clinical management, (4) ethical, legal and social implications, (5) professional role, (6) leadership, and (7) research.

Kirk, Tonkin and Skirton (2011) published, for the National Genetics Education and Development Centre in the UK, a wide-ranging review and revised competence-based framework, including learning outcomes and practice indicators for nurses and midwives. While the Australian standards for the RN (Nursing and Midwifery Board of Australia (NMBA), 2010) do not specifically refer to genetics, Standard 5.1 states that nurses 'use a

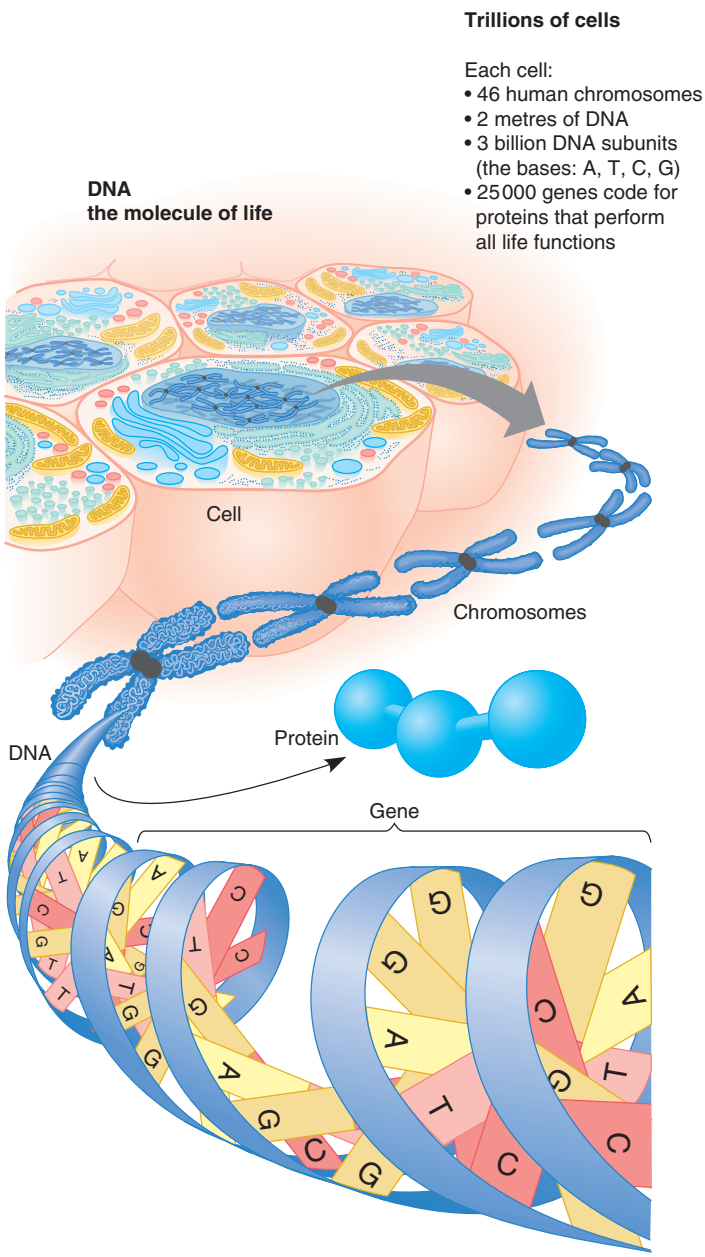


FIGURE 7.1 ■ Each cell nucleus throughout the body contains the genes, DNA and chromosomes that make up the majority of an individual's genome. The remaining portion of the human genome is in the mitochondria

relevant evidence-based assessment framework to collect data about the physical, socio-cultural and mental health of the individual/group' which of course would include a family history. Therefore, both the UK and US frameworks and competency standards have relevance for Australian nurses and midwives.

FOUNDATIONS OF GENETICS

Nurses need to have foundational knowledge of the cell, DNA, cell division, chromosomes and genes in order to deliver competent care and to enable people requiring assessment, support and referral to fully benefit from advances in genetics (see Box 7.2).

BOX 7.2 Using the people-first approach

Nurses must incorporate a 'people-first' philosophy and use genetic terminology that is sensitive to the maintenance of an individual's positive self-image. This can be accomplished by using terms such as 'unaltered' or 'wild-type' gene instead of 'normal' gene and 'altered gene'; 'altered, disease-producing gene' or 'gene alteration' instead of the terms 'mutated' or 'abnormal' gene when communicating genetic concerns.

wild-type = normal = expected = unaltered

versus

mutated = abnormal = defective = unexpected = altered

The cell is the basic unit of life and the working unit of all living systems. Life starts as a single cell, but the developed human body is made up of trillions of cells. These cells share common features, such as a nucleus that contains 46 chromosomes and organelles such as mitochondria. There are many different types of 'specialised' cells that function differently depending on their location. For example, pancreatic cells have a very different function from that of nerve cells.

All human cells, except mature red blood cells, contain a complete set of deoxyribonucleic acid (DNA) molecules. DNA molecules consist of long sequences of nucleotides or bases represented by the letters A, G, T and C. The order of these bases gives the exact instructions for the functioning of that particular cell. Writing the correct order of the bases using the above abbreviations represents the sequence of the bases in DNA. The entire DNA in a human cell is referred to as the **human genome**, or the complete set of inheritance for an individual. The human genome includes the DNA in the cell nucleus as well as the DNA found in the mitochondria, which will be discussed later in this section. Each person's genome is unique. Identical (monozygotic) twins are the exception because they develop from only one fertilised ovum and share identical DNA.

The cell nucleus contains about 183 cm of DNA that is tightly wound and packaged into 23 pairs of **chromosomes**, making a complete set of 46 chromosomes. The structure and number of chromosomes can be shown by a karyotype, or picture, of an individual's chromosomes (see Figure 7.2). There are two copies of each chromosome. One copy, or half of the complete set of these 46 chromosomes, is inherited from the mother and the other copy, or the other half of the 46 chromosomes, is inherited from the father. For example, an individual will have two of chromosome 1, one inherited from her mother and one inherited from her father. These two copies or pairs of inherited chromosomes are called **homologous chromosomes**. Chromosomes are numbered according to size, with chromosome 1 being the largest and chromosome 22 being the smallest. The first 22 pairs of chromosomes, known as **autosomes**, are alike in males and females. The 23rd pair, the **sex chromosomes**, determines an individual's gender. A female has two copies of the X chromosomes (one copy inherited from each parent) and a male has one X chromosome (inherited from his mother) and one Y chromosome (inherited from his father).

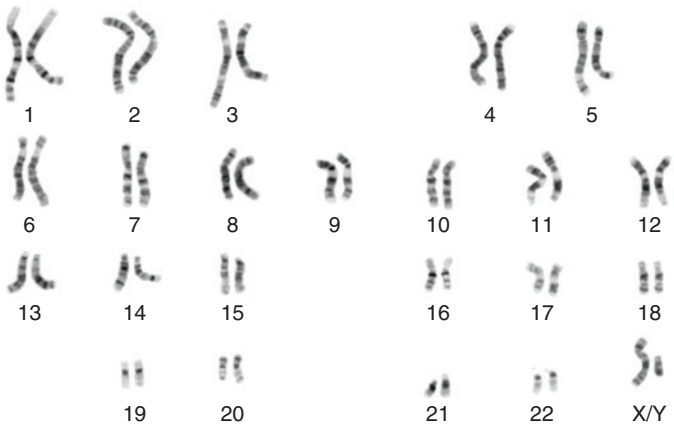


FIGURE 7.2 ■ A karyotype is a picture of an individual's chromosomes. It shows the chromosomal structure and number of the 22 pairs of autosomes and the sex chromosomes

Source: © Medical-On-Line/Alamy.

Cell division

Mitosis and meiosis are the two types of cell division in human cells. **Mitosis** is the process of making new cells and takes place in the **somatic**, or tissue, **cells** of the body. Cell division through mitosis heals wounds and replaces cells lost daily on skin surfaces and in the lining of gastrointestinal and respiratory tracts. In addition, mitosis is responsible for development. The mitotic activity of the zygote and its daughter cells is the foundation for a human's growth and development. The zygote undergoes mitosis to form a multicellular embryo, then foetus, then infant. Cell division through mitosis results in two cells, called *daughter cells,* that are genetically identical to the original cell, or *mother cell*, and each other.

Meiosis is also known as the reduction division of the cell. Meiosis occurs only in the sex cells of the testes and ovaries and results in the formation of the sperm and oocyte (gametes). Meiosis is very similar to mitosis in that it is a form of cell division; however, through a series of complex mechanisms, the amount of genetic material is reduced by half (23 chromosomes). This is very important because when the two sex cells combine during fertilisation, the total number of chromosomes (46) is present in the offspring's cells. The purpose of meiosis is to produce gametes, to reduce the number of chromosomes by half and to make new combinations of genetic material from crossing over and independent assortment processes, which allow diversity in the human population.

Chromosomal alterations

Alterations in chromosomes often occur during cell division (meiosis or mitosis) and are classified as either alterations in the number of chromosomes or structural alterations. They involve either part of or the whole chromosome. The clinical consequences of number and structural changes in the chromosomes in an individual vary depending on the amount and type of DNA affected by the alterations.

Alterations in chromosome number

An increase or decrease in chromosomal numbers can occur during meiosis or mitosis (see Box 7.3). Alterations often occur during meiosis because meiosis is a highly specific and complex process and each new daughter cell must contain exactly one chromosome from each homologous pair of chromosomes. During meiosis, the paired chromosomes may fail to separate, resulting in daughter cells with either two copies or no copies of that chromosome. This is known as **nondisjunction**.

Nondisjunction creates an egg or sperm cell with either two copies or no copies of a particular chromosome. When these egg or sperm cells are fertilised by a normal gamete that contains 23 copies of all of the chromosomes, a zygote that is monosomic (one member of the chromosome pair is missing) or trisomic (having three chromosomes instead of the usual two) results. These circumstances produce such conditions as Turner's syndrome (**monosomy**) or Down syndrome (**trisomy** 21). As you have already read, normally each cell has 23 pairs of chromosomes, making a total of 46 chromosomes. One of these pairs, the sex chromosomes, determines the gender of the foetus (46XY for a boy and 46XX for a girl). In **Turner's syndrome** (affects about 1:2000 girls) there will usually be only one X chromosome or portion of one missing in all or some of the cells. The reason for this total or partial loss of the chromosome usually cannot be found, but the loss occurs soon after conception. Sometimes there may be abnormalities in the X chromosome in only some cells in the body. This is referred to as mosaic Turner's syndrome and there may be few or no symptoms and fertility may not be affected. Parents need to be reassured that there was nothing either parent could have done to prevent this from happening; it is a biological accident for which no one is responsible. Girls with Turner's syndrome are at risk of impairments in the cognitive, behavioural and social domains—learning disabilities, particularly with regard to spatial perception, visual–motor integration, mathematics, memory, the ability to formulate goals and plan action sequences to attain them, and attention span. To complicate matters, a female with 45X Turner's syndrome may manifest an X-linked recessive disorder, such as haemophilia, because she has only one X chromosome.

The missing genes cause significant problems for the girl, but the main ones are:

- short stature (average height: 143 cm)
- lack of secondary sexual characteristics (failure to develop at puberty)
- infertility.

BOX 7.3 Variations in chromosomal number

Aneuploidy—the condition when extra or missing chromosomes exist; if there is an addition or deletion of chromosome number and the individual lives, physical abnormalities and/or mental retardation are common.

Monosomy—the loss of a single chromosome from a pair; i.e. Turner's syndrome (45XO).

Trisomy—the gain of a single chromosome, making a total of three copies of a certain chromosome; i.e. trisomy 21 or Down syndrome.

Euploidy—the presence of the normal number of 46 chromosomes.

Polyploidy—the condition when more than two pairs of all the chromosomes are present.

Klinefelter's syndrome, also known as the XXY condition, describes males who have an extra X chromosome in most of their cells. Klinefelter's syndrome is named after Dr Henry Klinefelter. Boys with Klinefelter's syndrome are born with at least one extra X chromosome (47 chromosomes in each cell, rather than the normal number of 46). Klinefelter's syndrome is also called 47XXY syndrome. It is thought to be caused by an error within the fertilised egg or the dividing cells as the baby develops. The presence of the Y chromosome ensures male sexual characteristics but, because the testicles are underdeveloped, there may not be enough testosterone production. This is why the penis and testicles are smaller than average, and why most men with Klinefelter's syndrome are infertile. Some researchers suspect that advanced maternal age may be a risk factor.

Down syndrome is named after Dr John Langdon Down who first identified the syndrome in 1866. Trisomy 21 (Down syndrome) is caused by an extra copy of chromosome 21. An obsolete term that should never be used to describe someone with Down syndrome is *mongolism*. The extra chromosome causes certain physical characteristics and affects intellectual development. In Australia, about 95% of all pregnant women consent to have a screening test to determine if the foetus is at risk of Down syndrome. About 1:20 (5%) women will be notified that the foetus is at increased risk. Only further testing will show which babies do have the problem. It is important to remember that even if the test shows that the baby is at low risk of Down syndrome the child may be born with other complications. Testing does NOT ensure the 'perfect baby'. Down syndrome occurs in all races and cultures. Approximately 1 in every 800 babies is born with Down syndrome. However, the actual rate is probably much higher than this because many women will choose to terminate their pregnancy.

Alterations in chromosome structure

Alterations in chromosome structure include inversions, deletions and duplications, and translocations. In a chromosomal inversion a segment of a chromosome is reversed, changing the DNA sequence for that portion of the chromosome. It occurs when a chromosome breaks in two places and the piece between the breaks turns upside down and reattaches within the same chromosome. The clinical consequences of an inversion depend on how much chromosomal material is involved, where the inversion occurs and what type of inversion is present.

A chromosomal alteration that includes a missing (deletion) or additional (duplication) whole chromosome or segment of a chromosome is an unbalanced rearrangement. An unbalanced rearrangement can result in missing genes, confusing directions from the genes, or too much gene product, which often results in a condition that is not compatible with life, or in altered physical and/or mental development. An example is cri du chat syndrome (intellectual disability, crying that sounds like a cat mewing and low-set ears). Cri du chat syndrome is an abnormality resulting in the deletion of a large part of the short arm of chromosome 5. **Translocation** (chromosomal reshuffling) occurs when a segment of a chromosome transfers or moves and attaches itself to another chromosome. An example is the reciprocal translocation that is found in 95% of people with chronic myelogenous leukaemia (CML). The contributing translocation occurs between chromosomes 9 and 22. The translocation results in a shortened chromosome 22, an observation first described by Nowell and Hungerford in 1960 and subsequently termed the Philadelphia (Ph1) chromosome after the city where it was discovered. The translocation (two chromosomes break, then parts from each chromosome switch places) relocates an oncogene called Abl from the long arm of chromosome 9 to the long arm of chromosome 22. As a result, a new, abnormal gene called BCR produces Bcr-Abl tyrosine kinase, an abnormal protein that causes too many stem cells to develop into white blood cells (granulocytes or blasts). The exact cause of CML is unknown, though it is now known how the disease develops from genetic changes in myeloid cells.

Unlike the translocation responsible for Down syndrome, which occurs in the germ cells, the translocation responsible for CML occurs in somatic cells and therefore is not inheritable (National Cancer Institute, 2009; Nussbaum et al., 2007). Thus environmental factors account for only a small number of CML cases and so family history does not appear to play a role in the development of CML.

Genes

Nurses must also have knowledge of genes—what they are, the role genes play in homeostasis, as well as the consequences of gene alterations. How these gene alterations are inherited is also important for nursing and midwifery interventions and teaching the person who is at risk of, or who has, a known gene (DNA-based) condition. Knowledge of the function and inheritance of genes is implicit in health promotion as well as health maintenance of the person and their family.

A **gene** is a small portion (segment) of the nucleotide (base) sequence of a chromosome DNA molecule that can be identified as having a particular function or characteristic. These segments of DNA within each gene have specific directions for the functioning of the gene. This specific sequence of nucleotides (the genes and the variations therein) is referred to as the individual's **genotype**. Each chromosome contains numerous genes arranged in a linear order. Researchers currently believe there are about 20 000 to 30 000 genes in the human genome (Jameson & Kopp, 2012; Lister Hill National Center for Biomedical Communications, 2014). The number of genes present on each chromosome varies. Chromosome 1 is the largest chromosome and has the largest number of genes, with 2968. The Y chromosome has the smallest number of genes, with 231 (Human Genome Project (HGP), 2008a).

All genes come in pairs because chromosomes come in pairs. The only exception to all genes being paired are the genes on the sex chromosomes (X and Y) present in males. All genes have a specific location on a specific chromosome. This is known as the **genetic locus**. For example, one of the many genes located on chromosome 19 is a gene for eye colour. There may be slight variations or different forms of a gene—for instance, green versus blue eye colour—and these different forms or versions of genes are called **alleles**. When an individual has two identical forms (alleles) of a gene, they are said to be **homozygous** (homo = same). If an individual has two different forms (alleles) of the gene, they are said to be **heterozygous** (hetero = different). Genes can be described as *altered* or *mutated*, when a change has taken place, or *expressed*, when the gene has an impact on the outward

appearance of an individual and/or the functioning of cells. The observable, outward expression of an individual's entire physical, biochemical and physiological make-up, as determined by their genotype (alleles) and environmental factors, is referred to as **phenotype**. Phenotype may be expressed or observed as curly or straight hair or the presentation of signs and symptoms of a disease.

Function and distribution of genes

Although the function of more than 50% of the genes in the human genome is still unknown, we do know that about 2% of the genes give directions to parts of the cell for how to make proteins, what type of proteins to make and how much of a protein to make (HGP, 2008a). These protein-directing genes are very important to life and functioning as a human being because proteins are very specialised and perform a variety of functions within the cell. These functions include transmitting messages between cells, fighting infection, directing genes to turn 'on' or 'off' and forming structures, as well as sensing light, taste and smell (Jegalian, 2000). Some gene activities change from moment to moment in response to tens of thousands of intra- and extracellular environmental signals (USDOE Genome Programs, 2008). An example of this is the feedback mechanism that stimulates a cell to produce insulin after eating a candy bar. After eating, a gene on chromosome 11 directs pancreatic cells to produce, modify and secrete insulin. Although the gene for producing insulin is present in all nucleated cells of the body, it is only functional in insulin-secreting pancreatic cells (Guttmacher et al., 2004).

Mitochondrial genes

Chromosomes in the cell nucleus are not the only site where genes reside. Several dozen that are involved in energy metabolism are located in the cell mitochondria (the 'powerhouse' of the cell). **Mitochondria** are concerned with energy production and metabolism. Some cells contain more mitochondria than others, but each mitochondrion contains its own copies of DNA, identified as mitochondrial DNA (mtDNA). Because ova have many mitochondria and sperm do not (most mitochondria are located in the tail of the sperm that detaches after fertilisation), mtDNA is primarily inherited from the mother. Therefore, mitochondrial genes and any diseases due to DNA alterations on those genes are transmitted through the mother in a matrilineal pattern. This pattern of inheritance is very different from the pattern of inheritance of genes found in the nucleus of the cell (Guttmacher et al., 2004). Thus, an affected female will pass the mtDNA mutation to all of her children; however, an affected male will not pass the mtDNA mutation to any of his children (John et al., 2010; Nussbaum et al., 2007). Signs and symptoms of conditions occurring as a result of mitochondrial gene alterations primarily involve high-energy tissues and organs such as skeletal muscles, liver, kidney, brain and nerve cells, ears, eyes, endocrine system and heart muscle. Symptoms develop over years as unhealthy or dying cells are not replaced. Hypertrophic cardiomyopathy, heart block, seizures and deafness are also associated with mtDNA gene alterations (John et al., 2010; Nussbaum et al., 2007). An Italian research team has found that chronic kidney disease (CKD) is possibly linked to oxidative stress caused by dysregulation of the genes that control mitochondria (Granata et al., 2009).

Gene alterations and disease

A protein will malfunction and in many cases cause disease if any kind of alteration (mutation or change) is present in the order of the DNA sequence within a gene. These gene alterations can be inherited from one or either parent or they can be acquired. *Mutations* inherited from a parent (hereditary mutations) are also known as *germline mutations* because the mutation exists in the reproductive sperm or ova of the parent. Consequently, the DNA in every cell of that offspring will have the gene alteration and also can be inherited from generation to generation.

The second kind of gene alteration is an *acquired mutation* or *somatic mutation*. These alterations occur in the DNA of cells of the individual throughout their life. They can result from errors during cell division (mitosis) or from environmental influences such as radiation or toxins (National Institutes of Health (NIH) and National Cancer Institute (NCI), 1995).

Today, we know that gene alterations are responsible for approximately 6000 hereditary diseases. However, different gene alterations within a particular gene can result in a wide variety of signs and symptoms. Since it occurs in one of the first 22 pairs of chromosomes, the cystic fibrosis (CF) defect is autosomal. It is not sex-linked, so the disease can occur in either gender. For example, the CFTR gene for cystic fibrosis is a very large gene located on chromosome 7. More than 1500 different mutations of this gene have been reported to cause cystic fibrosis (Ferraguti et al., 2011). The area of the CFTR gene that controls mucus production can have more than 300 different gene alterations, resulting in a variety of symptoms ranging from mild, to severe, or no symptoms at all (NIH/NCI, 1995). Gene alterations, not the genes themselves, cause genetic diseases and conditions. Since CF is a recessive trait, an affected individual must receive two defective genes in order to be born with CF. As with all chromosome pairs, one is inherited from the mother and one is inherited from the father. This means that both parents must carry the cystic fibrosis trait or have CF themselves in order to have a child with CF. People who are carriers have only one defective gene. They will not have CF and will not have any symptoms. On average about 1 in 25 Australians and New Zealanders are genetic carriers for CF but they are more likely to be a carrier if they are of Northern European descent (including the UK). If both parents are carriers there is a 1 in 4 chance that each parent will pass on their defective gene, meaning their baby will also have CF. Interestingly, other factors probably influence the course of CF. For example, changes in genes other than CFTR might explain why some people with the disease are more severely affected than others. So far none of these factors have been identified.

All babies in Australia are screened for CF shortly after birth:

- Genetic testing may be available to determine if a person is a carrier of the faulty CFTR gene. The screening test may be offered pre-pregnancy and in pregnancy when there is a family history of CF or a blood relative is a genetic carrier for CF.
- Genetic screening may also be available as part of pre-pregnancy planning for those people with a high chance of being a genetic carrier for CF based on their family history. The screening will only pick up those who are carriers of one of the more common changes in the CFTR gene.

Other situations where gene alterations cause illness and disease are through gene interaction with the environment. These genes and conditions are referred to as multifactorial (Jegalian, 2000). Alterations in regulatory genes may also occur. Regulatory genes play a part in maintaining homeostasis or normal functioning. A regulatory gene mutation might lead to the loss of expression of a gene, to unexpected expression in a tissue in which it is usually silent, or a change in the time when a gene is usually expressed. An example of a regulatory gene mutation associated with disease includes the insulin gene region that increases the risk of type 1 diabetes (Guttmacher et al., 2004). Researchers have identified a gene, KIAA0350, that increases the risk of getting type 1 diabetes (insulin-dependent diabetes) (Hakonarson et al., 2007). This finding does not mean that researchers know how to prevent diabetes in those who have these genetic mutations. Diabetes is a complex disorder and the risk of developing it is known to be affected by environmental factors and at least four other genes.

Gene alterations that decrease risk of disease

Although it is common to associate gene mutations with disease, it is important to remember that gene mutations can also be helpful and decrease the risk of disease. Gene alterations and genetic variations may also have a protective role in the expression of diseases. A common example is the protective value of the gene alteration that causes sickle cell disease. Those individuals with this gene alteration have protection against malaria. Another, less common example of a 'protective' gene alteration is the one on the receptor gene named CCR5. This mutation consists of a deletion within the DNA sequence. Persons who are homozygous for the CCR5 mutation (have two copies of the altered gene) are almost completely resistant to infection with HIV type I, and those who are heterozygous for the deletion (have one copy of the altered gene) progress much more slowly from the stage of HIV infection to AIDS (Guttmacher et al., 2004). As genomic research continues, more and more of these types of beneficial gene alterations will be identified.

Single nucleotide polymorphisms

Single nucleotide polymorphisms, or SNPs ('snips'), are one-letter (base pair) variations in the DNA sequence that occur in more than 1% of the population. In all people, 99.9% of the DNA is identical; SNPs are responsible for differences among individuals. **Polymorphisms** are DNA sequences that have many forms but give the genetic 'directions' for the same thing. Most of these differences have no effect on the individual. Some cause subtle differences in numerous characteristics in appearance such as widow's peak, tongue rolling and attached ear lobes. Other SNPs, however, affect an individual's risk of certain diseases and have a major impact on how the individual responds to environmental factors such as toxins, microbes and medications. Biological markers are important for the construction of chromosome maps and are easily tracked, stable segments of DNA. Scientists are mapping these areas of SNPs in order to move to the next step of identifying the multiple genes that are associated with diseases that are not caused by single-gene alterations—those complex diseases caused by multiple genes such as cancer, cardiovascular disease, some forms of mental illness and diabetes (HGP, 2008c; Jegalian, 2000). More recently, an international collaboration has linked common SNPs on chromosome 5 to five different types of cancer while at the same time conferring protection against melanoma. Of note, four of the five cancers have a strong environmental contribution to risk (smoking for lung and bladder cancer, UV light for skin cancer, and HPV infection for cervical cancers) (Stefanson & Thorunn Rafnar, 2009).

PRINCIPLES OF INHERITANCE

Knowledge of inheritance allows nurses and midwives not only to offer and reinforce genetic information to people and their families, but also to assist them in managing their care and in making reproductive decisions. The basic underlying principles of inheritance that health professionals can apply to inheritance risk assessment and teaching include: (1) all genes are paired, (2) only one gene of each pair is transmitted (passed on) to an offspring, and (3) one copy of each gene in the offspring comes from the mother and the other copy comes from the father. Understanding the Mendelian patterns of inheritance is made easier by relating these principles.

Mendelian patterns of inheritance

Conditions that are caused by a mutation or alteration of a single gene are known as *monogenic* or *single-gene disorders*. There are more than 6000 known single-gene disorders occurring in about 1 in 200 births (HGP, 2008a). The most common gene alterations that result in genetic disorders are categorised into Mendelian inheritance patterns because they are predictably passed on from generation to generation following Mendel's laws of inheritance. These single-gene mutations follow an autosomal dominant, autosomal recessive, **X-linked recessive** or **X-linked dominant** inheritance pattern. The first three of these patterns are the most common. Modes of transmission or inheritance for thousands of conditions resulting from monogenic alterations have been identified (*Online Mendelian inheritance in man*, 2003).

Recessive versus dominant disorders

The distinction between recessive and dominant phenotypes or disease presence (expression) lies in the amount of gene product (usually proteins) from the unaltered (**wild-type** or normal) **gene**. When the individual is heterozygous (has one unaltered gene and one altered gene), the altered gene as well as the disease is classified as recessive if half of the product produced from the unaltered gene is enough to maintain homeostasis and perform the expected function. Therefore, two altered genes must be present to cause a diseased state. If the altered gene causes disease even though the unaltered gene is producing the gene product, then the altered gene as well as the disease is classified as dominant (Nussbaum et al., 2007).

Autosomal dominant conditions

Autosomal dominant (AD) conditions are the result of an altered gene on any of the 22 autosomes or non-sex chromosomes (see Figure 7.3). More than half of the known Mendelian

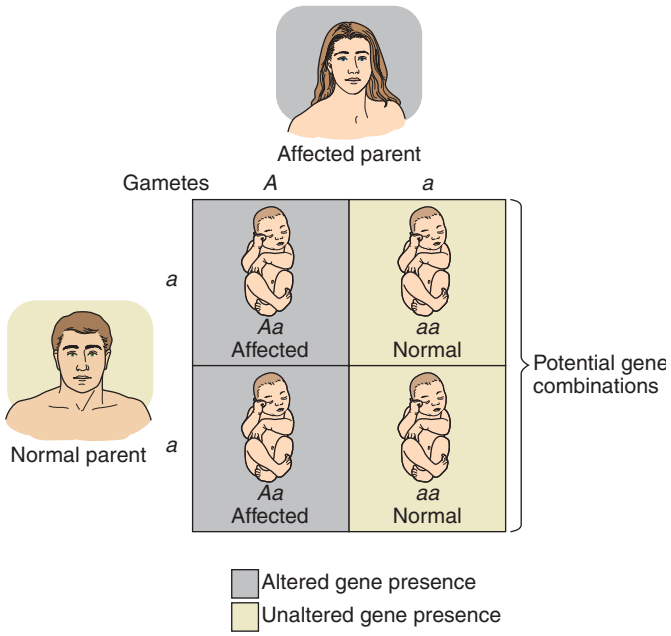


FIGURE 7.3 ■ This Punnett square shows potential gene combinations (genotypes) and resulting phenotypes of children from parent genotypes with an autosomal dominant altered gene. Phenotypes are expressed (affected) when a male or female has one copy of the gene alteration

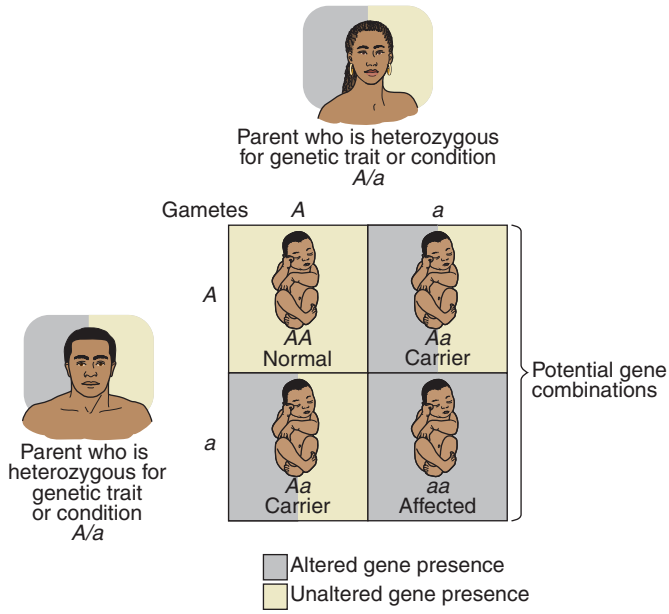


FIGURE 7.4 ■ This Punnett square shows potential gene combinations (genotypes) and resulting phenotypes of children from parent genotypes with an autosomal recessive altered gene. Phenotypes are expressed (affected) when a male or female has two copies of the gene alteration

conditions are autosomal dominant. In AD conditions, disease occurs in spite of the fact that there exists one unaltered or normal gene. Also, homozygous dominant conditions are generally much more severe than heterozygous dominant conditions and are often lethal. Because homozygous dominant conditions are usually lethal and would result from *both parents being affected*, the nurse or midwife should consider an individual exhibiting an autosomal dominant condition as heterozygous. See Box 7.4 for characteristics of an AD pattern of inheritance.

BOX 7.4 Autosomal dominant Mendelian inheritance characteristics

(*Examples:* neurofibromatosis, breast and ovarian cancer, autosomal dominant polycystic kidney disease, Marfan's syndrome, Huntington's disease, familial hypercholesterolaemia.)

When the health professional gathers a family history, they should assess for any of the following characteristics of autosomal dominant inheritance:

1. Both males and females are affected.
2. Males and females are usually affected in equal numbers.
3. An affected child will have an affected parent and/or all generations will have an affected individual (appearing as a vertical pattern of affected individuals on the family pedigree).
4. Unaffected children of an affected parent will have unaffected offspring.
5. A significant proportion of isolated cases are due to a new mutation.

Autosomal recessive conditions

A gene or genetic condition is considered recessive when two copies of altered genes are needed to express the condition—for example, cystic fibrosis. Autosomal recessive (AR) conditions are the result of an altered gene on any of the 22 autosomes or non-sex chromosomes (see Figure 7.4). An individual with a recessive condition has inherited one altered gene from their mother and one from their father. In most cases, neither of the parents is affected and, therefore, each of the parents must have a single gene alteration on one chromosome of a pair and the normal, wild-type or unaltered form of the gene on the other chromosome. These parents would be known as **carriers** of the condition and they do not usually exhibit any signs and symptoms of the condition. Because the gene alteration occurs on a non-sex chromosome, both males and females have an equal chance of inheriting the altered gene from their parent. Generally, conditions that are autosomal recessive are more severe and have an earlier onset than conditions with other patterns of inheritance. Most inborn errors in metabolism or metabolic conditions are autosomal recessive. Many are enzyme defects and the functioning of the unaltered gene is sufficient to provide normal functioning in the person who is heterozygous or the carrier of one copy of the altered gene (Lashley, 2005). See Box 7.5 for characteristics of an AR pattern of inheritance.

X-linked recessive conditions

X-linked conditions are the result of an altered gene on the X chromosome. Unlike the autosomes, the sex chromosome, X, is unevenly distributed to males and females. The female has two X chromosomes and the male has only one. Thus, the family history and pattern of inheritance has a characteristic distribution pattern among the males and females in the family (see Figure 7.5).

BOX 7.5 Autosomal recessive Mendelian inheritance characteristics

(*Examples*: haemochromatosis type 1, cystic fibrosis, phenylketonuria, sickle cell anaemia.)

When the health professional gathers a family history, they should assess for any of the following characteristics of autosomal recessive inheritance:

1. Both males and females are affected.
2. Males and females are usually affected in equal numbers.
3. An affected child will have an unaffected parent but may have affected siblings (appearing as a horizontal pattern of affected individuals on the family pedigree).
4. The condition may appear to skip a generation.
5. The parents of the affected child may be consanguineous (close blood relatives).
6. The family may be descendants of a certain ethnic group that is known to have a more frequent occurrence of a certain genetic condition.

Because the male has only one copy of any gene on the X chromosome, it becomes the only copy available to give direction for those particular functions of these genes regardless of whether it is considered dominant or recessive in the female. Thus, if any of these genes are altered, an unaltered counterpart is not present to 'override' the altered functioning gene.

The consequences of the altered gene on an X chromosome will be expressed in all males. Females, on the other hand, will have two copies and the unaltered gene generally compensates for the altered gene, making the female a carrier. The male receives his X chromosome from his mother and his Y chromosome from his father. The female offspring receives an X chromosome from each of her parents. Thus, all affected males will pass on the altered X chromosome to all of his daughters who will be carriers of the altered gene. A male can never transmit an altered gene on the X chromosome to his sons because the male will transmit only the Y chromosome to his sons. Because of these transmission patterns, the most commonly occurring transmission of an X-linked condition is through a female who is a carrier of an altered gene. See Box 7.6 for characteristics of an X-linked recessive pattern of inheritance.

X-linked dominant conditions

X-linked dominant conditions also exist but are very rare. The inheritance pattern for X-linked inheritance differs from autosomal inheritance only because the X chromosome has no homologous chromosome in the male; the male has an X and a Y chromosome. For an X-linked dominant disorder, if the father carries the abnormal X gene, all of his daughters will inherit the disease but no sons will have the disease. If the mother carries the abnormal X gene, 50% of all her children (daughters and sons) will inherit the disease tendency. The other children inherit the normal copy of the chromosome. If a male is affected, the condition is severe and often lethal.

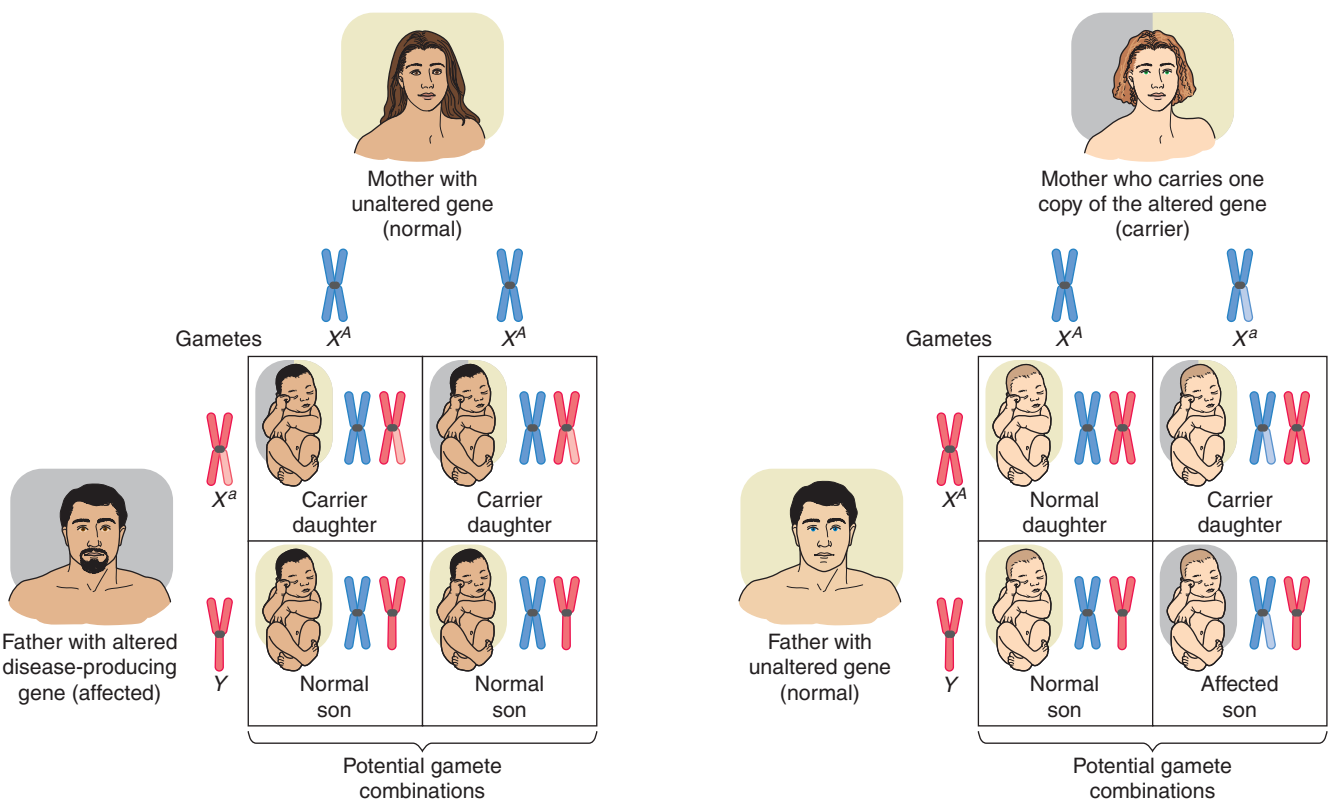


FIGURE 7.5 ■ These Punnett squares show potential gene combinations (genotypes) and resulting phenotypes of children from different parent genotypes with an X-linked recessive altered gene. Phenotypes are expressed (affected) in a male with only one copy of the gene alteration and in a female with two copies of the altered gene

BOX 7.6 X-linked recessive Mendelian inheritance characteristics

(*Examples:* haemophilia A; Duchenne muscular dystrophy.)
When gathering a family history, the health professional should assess for any of the following characteristics of X-linked recessive inheritance:

1. More males will be affected than females; rarely seen in females.
2. An affected male will have all carrier daughters.
3. There is no male-to-male inheritance.
4. Affected males are related by carrier females.
5. Females may report varying milder symptoms of the condition.
6. A new sporadic case could be due to a new mutation.

A family history of multiple male miscarriages may be a sign of an X-linked dominant condition. X-linked dominant diseases are very uncommon, but some inherited forms of rickets are transmitted in this manner. Very few genes have been discovered on the Y chromosome.

Variability in classic Mendelian patterns of inheritance

Along with understanding the classic Mendelian inheritance patterns, several other concepts are also important for families to understand when health professionals are assisting people with, or at risk of inheriting, a genetic disorder. These include the following exceptions or variations to the traditional Mendelian patterns of inheritance.

Penetrance

Penetrance is the probability that a gene will be expressed phenotypically. It is an 'all or none' concept, in that either the gene will be expressed (even if mildly expressed) or it will not be expressed at all. Penetrance can be measured in the following way. In a certain group of individuals with the same genotype, what percentage of them will exhibit at least some signs and/or symptoms of the condition? If the number is less than 100%, then that condition is said to show reduced penetrance. For example, the gene alterations that cause achondroplasia exhibit 100% penetrance and all individuals with one copy of the gene alteration will exhibit signs and symptoms of the disease (Daly et al., 2010; Nussbaum et al., 2007).

New mutation

When there is no previous history of a condition, including even subtle signs and symptoms of the disease in any other immediate or distant family member, the disease may be caused by a spontaneous new mutation. This case is usually called *de novo* mutation. New mutations of a gene are most frequently seen in autosomal dominant conditions because one copy of an altered gene is all that is necessary to elicit a state of altered health. Autosomal dominant diseases known to have high mutation rates include neurofibromatosis, achondroplasia (dwarfism) and Marfan's syndrome. New mutations are also possible in autosomal recessive diseases, although rarely expressed because two altered genes are necessary for signs and symptoms to appear. Finally, new mutations are often seen in X-linked recessive disorders, such as haemophilia A, since a male with just one altered gene will demonstrate the disease.

Anticipation

Anticipation is said to occur when successive generations of a family exhibit more severe signs and symptoms of certain diseases and the disease often has an earlier onset. An example is myotonic dystrophy type 1, an autosomal dominant condition characterised by a range of signs and symptoms including myotonia, muscle weakness, cataracts and cardiac arrhythmias. The congenital form is severe, causing intellectual disability, and may be life threatening. Most children with this congenital form of myotonic dystrophy have a mildly affected mother who may not even be aware she has the disease (Nussbaum et al., 2007). The severity of the condition, as well as the age of onset, is determined by the number of trinucleotide repeats. Trinucleotide repeats are short DNA sequences such as the CTG base sequence of the gene DMPK that are repeated to more than 2000 times, resulting in alterations in the protein products produced by the gene and varying signs and symptoms.

Variable expressivity

Expressivity is used to describe the severity of the **gene expression** of the phenotype. When people with the same genetic make-up (genotype) exhibit signs and/or symptoms with varying degrees of severity the phenotype is described as *achondroplasia* exhibit 100% penetrance (Nussbaum et al., 2007).

Neurofibromatosis type 1 (NF1) is a common autosomal dominant disorder which displays considerable inter- and intra-familial variability in phenotypic expression. Although neurofibromatosis has 100% penetrance, variable expressivity can occur within family members with each family member exhibiting a variety of signs and/or symptoms.

Multifactorial (polygenic or complex) disorders

Many birth defects such as cleft lip and palate, as well as many adult-onset conditions such as cancer, mental illnesses, asthma, diabetes, obesity, heart disease and Alzheimer's disease, have a multifactorial cause. **Multifactorial** conditions occur as a result of several gene (polygenic) variations, lifestyle and environmental influences that work together. The polygenic concept is illustrated with the multiple genes involved in an individual's susceptibility for breast cancer. These genes have been identified on chromosomes 6, 11, 13, 14, 15, 17 and 22. Exactly which genes interrelate and how many environmental influences are enough to cause the presentation of many of the common complex diseases or conditions is not known. It is now known that the birth defect spina bifida is caused by the action of several genes, but its prevalence also depends on the amount of folate in the diet. Hypertension is influenced by a number of genes, but also by obesity. The precise causes of multiple sclerosis (MS) are largely unknown. MS is most likely a multifactorial condition involving interaction between genetic, lifestyle and environmental factors. It is well documented that MS also runs in some

families. There are probably a number of genes that cause susceptibility to MS as well as affecting the severity and progression of the disease. The major contributing genes for many common complex conditions have now been identified.

Multifactorial conditions accumulate in families, but these conditions do not follow the characteristic Mendelian pattern of inheritance seen with single-gene conditions. Inheritable recurrence risks vary in multifactorial conditions. With information gathered from a family history, basic occurrence risks can be assessed for an individual. For instance, premature death in a first-degree relative, two affected first-degree relatives, and two second-degree maternal or paternal relatives with at least one individual having premature onset of the disease are all considered high inheritance risks. Moderate risks include an individual having a first-degree relative with late or unknown disease onset or two second-degree relatives from the same lineage with late or unknown disease onset. An individual having no affected relatives or a negative family history, or only one affected second-degree relative from one or both sides of the pedigree, is considered average or general population risk (Scheuner et al., 1997).

Recurrence risks refer to whether or not a condition will occur again in subsequent pregnancies. Because a Mendelian pattern is not present, statistical percentages can be used to represent the chance that parents have a condition that will occur in another child. The risk of recurrence is higher when more than one family member is affected. The recurrence risk after the first affected child is 4%, whereas the recurrence risk after a second affected child increases to 10%. It is also known that the recurrence risk increases with an increase in severity of the defect.

INTERPROFESSIONAL CARE

Many health professionals work together in the screening, diagnosis, identification, prediction and treatment of genetic disorders. The goals of collaborative care are early diagnosis through testing and assessment, prediction and development of an effective treatment plan, psychosocial support to enhance decision making and coping, and referral to a genetic specialist as needed.

Genetic testing

Genetic testing may be used for a person's clinical management, for making personal decisions or for assisting in reproductive choices. A genetic test is very different from other types of clinical tests. Genetic tests involve the analysis of DNA, RNA, chromosomes and serum levels of specific enzymes or metabolites. Enzymes and metabolites are part of the protein products that genes produce. DNA, RNA and/or chromosomes are unique for each individual and the results have personal, social, financial and legal implications. Some genetic tests are diagnostic, while others are predictive or inform the individual of an increased risk of acquiring a disease or condition. A 'positive' genetic test may indicate that an asymptomatic individual will develop a genetic condition, but a prediction of the onset or severity of the condition cannot be made.

Complications also arise in a person's understanding because a negative test result cannot guarantee that the disease or condition might not develop in the future, often because environmental influences cannot be measured or controlled. Also, the genetic test may have only been able to detect the most common gene mutations and not all of the disease-producing gene alterations are known or available for inclusion in clinical testing. People may learn that they will develop a genetic condition such as Huntington's disease, for which there is no treatment. People may find out through genetic testing that they are a carrier and they have unknowingly passed the altered disease-producing gene on to their children.

Finally, the implications of genetic test results are far reaching. While confidentiality and autonomy for the individuals are always foremost for the nurse, the implications for the person's children, grandchildren, siblings and other extended family members who share a percentage of the same genes can be life altering. This information may be very confusing and very different from how they have perceived healthcare in the past and how they perceived the implications of a simple 'blood test'.

TYPES OF GENETIC TESTS Nurses and midwives should understand that genetic tests can be classified into two categories: screening and diagnostic. A positive screening genetic test result notifies the person of an increased risk or probability but must always be confirmed by diagnostic testing. Screening genetic tests are most commonly completed in prenatal, newborn and carrier circumstances. In contrast, a diagnostic test can definitively validate or eliminate a genetic disorder in the symptomatic person and then direct clinical management. Box 7.7 lists some of the positive and negative aspects of genetic testing.

BOX 7.7 Positive and negative outcomes related to genetic testing

Benefits of genetic testing

Provides for:

- Early screening and preventive measures
- Future planning and life preparation
- Lifestyle adaptations
- Decreased confusion and anxiety
- Psychological stress relief
- Reproductive choices
- Informed extended family members
- Early medical and/or surgical intervention
- Cost of medical follow-up reduced (if negative result).

Possible negative outcomes of genetic testing

- Survivor guilt
- Loss of identity
- No treatment may exist
- Employability and insurability affected
- Confusion about accessing healthcare and resources
- Risk of invasion of confidentiality and privacy
- Social stigmatisation.

Source: Data from Secretary's Advisory Committee on Genetic Testing (SACGT), *National Institutes of Health, 2000.*

Several categories of genetic tests included as subcategories of screening and diagnostic genetic tests follow:

- *Newborn screening*. In Australia, the Guthrie test is offered to mothers of all newborn infants and provides a means to identify children who have an increased risk of developing more than 30 genetic diseases such as phenylketonuria, CF and hypothyroidism. Some researchers are lobbying for newborns to be tested for fragile X syndrome, which is the most common cause of inherited intellectual disability, even though in Australia it affects only about 1 in 3600 males and between 1 in 4000 and 1 in 6000 females. The syndrome is caused by a genetic abnormality on the X chromosome, where a single sequence of three DNA base pairs is repeated many times, with devastating consequences.
- *Carrier testing* is completed on asymptomatic individuals who may be carriers of one copy of a gene alteration that can be transmitted to future children in an autosomal recessive or X-linked pattern of inheritance. This may be part of a couple's premarriage or preconception planning if they belong to a particular ethnic group with known risks of developing genetic disorders such as sickle cell anaemia and haemophilia. It may be necessary to determine the exact gene mutation from an affected family member prior to carrier testing. This is often completed through lineage analysis. In Australia, the haemoglobinopathies are the only group of conditions for which population screening is widely offered and which is government funded. The thalassaemias are the most common single gene disorders in the world's population and are a common cause of hereditary anaemia. Recent immigration to Australia, especially from South-East Asia, has introduced large numbers of people from areas where alpha-thalassaemia is common. Screening programs, particularly antenatal testing, are used to detect the carrier state for alpha-thalassaemia as well as the Hb variants in the homozygous form, or in combination with alpha-thalassaemia, which may cause severe disease. In some Australian states there are also population screening programs for cystic fibrosis and autosomal recessive conditions more common in Ashkenazi (Eastern European) Jewish individuals; this screening is generally offered on a user-pays basis.
- *Preimplantation genetic diagnosis (PGD).* Parents with a family history of a serious or fatal genetic condition now have the option of combining IVF and genetic testing, in a technique known as preimplantation genetic diagnosis (PGD). Couples using PGD first need to use IVF procedures to generate embryos. A single cell can then be removed from the early embryo without damaging it. This cell can be tested to see if it carries the genetic defect that causes the condition. Only embryos that do not carry the defective gene are implanted in the mother. Some couples have used PGD on embryos to determine if they can provide a bone marrow transplant for a sick sibling. The bone marrow cells for the sick sibling are taken from the umbilical cord blood of the new baby. Using this process of tissue typing, these babies are sometimes called 'saviour siblings', as they have the potential to save their brother or sister's life.
- Regulations regarding this use of PGD testing vary from country to country. In Australia, some states have PGD regulations and others do not. (See www.nscc.edu.au/public-education_what-cells_cord.aspx for a discussion of the social and ethical implications of this form of genetic testing.)
- *Predictive genetic testing* is usually made available to the asymptomatic individual and includes both predispositional and presymptomatic testing. A positive predispositional testing result will indicate there is an increased risk that the individual might eventually develop the disease. Common examples include breast cancer and hereditary non-polyposis colorectal cancer. A presymptomatic test is performed when development of the disease is certain if the gene alteration is present. The Australian Breast Cancer Network (2013) position statement explains that an extensive family history of breast cancer is a known predictive risk factor for the disease. A strong family history can indicate an inherited predisposition through the presence of a germline mutation in genes associated with breast cancer. A germline mutation is a mutation that occurs in the genetic material of the egg or sperm and can be passed on at conception. Inherited gene mutations account for 5% to 10% of all breast cancer. Some research suggests that BRCA1 and BRCA2 are tumour suppressor genes; that is, genes whose loss of function can lead to neoplastic growth. However, other studies point to the role of BRCA1 and BRCA2 in DNA repair, where inadequate repair may cause additional mutations and ultimately cancer.
- The estimated frequency of BRCA1 or BRCA2 gene mutation carriers in the general population is 1 per 1000 people. The frequency in specific population groups has also been estimated: for those of Ashkenazi Jewish descent, the rate of BRCA1 or BRCA2 gene mutation carriers is 1 per 50 to 100 people.
- Women with inherited mutations in genes such as BRCA1 and BRCA2 have a potentially high risk of developing breast cancer and for developing the disease at an earlier age. Those carrying mutations are also at increased risk of cancers of the ovary and fallopian tube (and perhaps other cancers). It is important to note that the risk of developing breast cancer never reaches 100%, which means that those with inherited gene mutations have a genetic predisposition to the disease but are not certain to develop it.
- Predictive genetic testing is medically indicated when the seriousness and mortality of the disease can be reduced with knowledge of the gene alteration. Life planning and lifestyle choices can be influenced by predictive testing. In Australia, premiums for private health insurance are not based on risk assessment according to the individual's present or past health or their family history. Premiums for life insurance products, which include cover for life, disability and trauma, and business and bank loans, are calculated according to the present and past health of the applicant and any genetic information, including their family history or any genetic test result (underwritten). The Investment and Financial Services Association Ltd (IFSA) has a policy on genetic testing and life insurance which states that no applicant will be required to undergo a predictive genetic test. Under Australian law, an application for a life insurance product is required to disclose any health or genetic information known to the applicant (Otlowski et al., 2007).
- Other uses of genetic testing include organ transplantation tissue typing and pharmacogenetic testing, which involves

predicting or studying the person's response to particular medications (Javitt, Stanley & Hudson, 2004). Predictive tests and carrier tests are now being offered to determine if people are at risk of conditions such as Alzheimer's disease (AD). The precise causation of AD is not fully understood. However, some cases of early-onset AD are caused by a number of genetic mutations on chromosomes 21, 14 and 1. Scientists have recently discovered that the mutations seen in early-onset AD are not involved in this form of disease. Dozens of studies have confirmed that the APOE-D4 allele increases the risk of developing AD. In September 2009, researchers identified three genes (CLU, PICALM and CR1) that significantly increase the risk of someone developing AD. Three of the genes have roles in protecting the brain from damage. Changes in the genes may remove this protection or may even turn them into 'killers' (van Es & van den Berg (2009).

DIAGNOSING CHROMOSOMAL ALTERATIONS Microscopic examination of chromosomes through a karyotype can reveal chromosomal alterations such as chromosomal additions, deletions, gross breaks, and rearrangements or rejoinings (translocations) (USDOE Genome Programs, 2008). Among other things, these chromosomal alterations are responsible for many forms of cancer and, more importantly, particular tumour types. Chromosomal diagnostic examination can be accomplished with a simple blood sample and skin or buccal cell sampling. A karyotype is completed in a cytogenetics laboratory. Chromosomes can be identified by their size and unique light and dark banding patterns. The pairs of autosomal chromosomes are arranged from 1 to 22 according to each chromosome's size, unique banding patterns and centromere position. The sex chromosomes complete the picture, with the X chromosome(s) first, then the Y chromosome (if present). The karyotype shows all of the chromosome pairs lined up and positioned on a piece of paper, allowing for visual chromosomal analysis. (See Figure 7.2 earlier in this chapter.) The final report contains numerical data that includes the total number of chromosomes present. If there is an additional or deleted chromosome, it is identified with a plus (+) or minus (–) symbol. For example, the male individual with 47, XY, +18 has 47 chromosomes (instead of the expected number of 46) that include an additional chromosome 18. Guidelines for writing results of karyotyping are determined by the International System of Human Cytogenetic Nomenclature (ISCN). These guidelines allow for use of a standardised universal language by cytogenetic laboratories and in medical publications.

DIAGNOSING GENE ALTERATIONS With the rapidly expanding advances in technology and the identification of genes in the human genome, the availability of genetic, DNA (gene)-based tests has grown tremendously. Currently, more than 1000 genetic tests are available with more becoming available each day (USDOE Genome Programs, 2008). **DNA-based tests** involve new, sophisticated technology that permits the examination of the DNA itself. Genetic testing that is DNA-based can be obtained from blood, bone marrow, amniotic fluid, fibroblast cells of the skin, or buccal cells from the mouth. Genetic testing includes different types of DNA-based tests. The appropriate genetic tests may be testing for a specific mutation. This would be used if a family member was known to have a genetic condition and could therefore be tested for that particular gene alteration. Another way to examine DNA is by running a panel of mutations. This is done when there are a specific number of identified genes that the majority of individuals with a genetic condition have—for instance, a panel of the three mutations on the BRCA1 gene that are common in the Ashkenazi Jewish population. A third type of DNA-based test is a complete gene sequence (GeneTests, 2004).

Quality and accuracy of genetic tests

Genetic nurses have expressed concerns that genetic tests are becoming available too quickly, with no regulation of the companies that are offering genetic tests. The quality, accuracy and reliability of genetic test results are not measured against any common standard. Individuals often make hard and irrevocable life-altering decisions after receiving test results, so accuracy and reliability are essential. Also, in most cases, minimal or no education is provided for the individual who is undergoing testing; nor is there any quality counselling or follow-up after the results are given to the individual. Genetic tests are often offered by laboratories before the tests have been proven safe, effective and practical. Because the majority of genetic conditions are rare, there is often only one laboratory offering the genetic test that is needed. Recently, concerns have been voiced about 'direct-to-consumer' genetic testing. Genetic tests are being offered at 'walk-in' locations and also via the internet. Individuals can receive results of genetic tests in private without a physician's order and fear of discrimination, but also without education or knowledge of the implications of the test results.

Concerns also exist related to test validity, test sensitivity and specificity, the quality of the laboratory performing the test, and the competence of the person's healthcare provider to interpret the test results. **Test sensitivity** refers to how specifically the test identifies (positive test result) individuals who are affected and/or who have the disease phenotype. A test with a high degree of sensitivity has very few false negatives and many true positives. **Test specificity** refers to how specifically the test does not identify (negative test result) individuals who are unaffected or do not have the disease phenotype. A test with a high degree of specificity has very few false positives (SACGT, 2000). The laboratory selected for the genetic test should have a CLIA88 (Clinical Laboratories Improvement Amendments of 1988) certification (Javitt, Stanley & Hudson, 2004).

Nursing care

The role of the nurse in genetic testing

With knowledge of available genetic tests and the many implications related to genetic testing, the nurse can assist people as they weigh choices regarding genetic testing. As consumers of multimedia, people often have unreliable sources for information related to genetic testing. They may form many misconceptions about the types of genetic tests available and what information these different types of genetic tests are able and not able to provide. Nurses involved in education about genetic testing must include education for the individual and the

family. Communication with the person about genetic testing should include an assessment of the positive and negative outcomes of the test. Are there existing treatments for the condition being tested? Psychological issues should also be emphasised. Who will be affected by the test results? Will the test results be shared with extended family members?

The nurse is responsible for alerting people to their right to make an informed decision prior to any genetic testing, with consideration of the special circumstances arising from the family, culture and community life. All genetic testing should be voluntary and it is the nurse's responsibility to ensure that the consent process includes discussion of the risks and benefits of the test, including any physical harm as well as potential psychological and societal injury by stigmatisation, discrimination and emotional stress (Beskow et al., 2001; International Society of Nurses in Genetics, 2005).

Above all, nurses have a responsibility to fully educate people about the multiple issues related to genetic testing. People should engage in genetic testing with full knowledge and confidentiality, and autonomously. Informed consent may be given verbally, although some laboratories require written consent. Prior to the testing, the people should have an idea of the probability of a positive or negative result, if one can be determined by the person's or family history (GeneTests, 2004). To deliver the expected standard of care, it is imperative that the nurse includes these issues when developing an educational plan for the person anticipating the use of genetic testing and as part of the informed consent process.

Ensuring confidentiality and privacy for genetic testing

Although confidentiality and privacy are integral parts of delivery of care for all nurses, this issue is of even more concern as it relates to genetic information. Results of genetic tests can be far reaching and can affect employment and insurance options. Will the results affect the person's ability to obtain and/or maintain insurance coverage? Can an employer refuse to hire or promote an individual because of genetic testing results? Can genetic information be released to the courts, the armed forces, schools or adoption agencies? Would a person with a known gene alteration for Huntington's disease be offered a scholarship to the best university? There is debate over whether genetic privacy is different from medical privacy. Nurses should inform people of their rights and responsibilities regarding who will have access to the genetic test results. Those providing the genetic tests must provide the person with assurance that the results will be handled confidentially and that there will be no access to the genetic information by a third party without written permission of the individual being tested.

Results of genetic tests should only be communicated directly to the individual who gave the consent. No outside governmental, employment or insurance organisations should ever have access to genetic test results without the written permission of individuals. People should confirm how they will receive the test results. They should ask who will have access to the test results and what will happen to the DNA sample after the test is completed. In the majority of cases, results of genetic tests should not be shared with extended family members without written permission. Healthcare providers are legally liable to maintain that confidence. Exceptions to the individual's privacy may be made only when the individual refuses to inform extended family members when a very high probability of irreversible harm exists for an extended family member, and informing the family member can prevent the harm (Badzek et al., 2013; National Human Genome Research Institute, 2008). Every effort should be made to educate the individual about the benefits of informing extended family members if applicable. Genetic testing should ideally be accompanied by pre-test and post-test counselling by genetic specialists or by another knowledgeable healthcare provider.

Psychosocial issues

Although family and individual anxiety may be decreased with a negative test result, potential problems do exist and the nurse or midwife must be prepared to address them. Concerns about carrier status may interfere with development of intimacy and interpersonal relationships. Non-paternity may be revealed through genetic testing. For example, the parents of a child born with an autosomal recessive condition will be considered carriers of the altered gene the majority of the time. To counsel the parents about future pregnancies, the parents would be tested to confirm their genotype and non-paternity may become an issue. A positive test result may lead to feelings of unworthiness, confusion, anger, depression and self-image disturbance. Survivor guilt may affect adults with negative results if their siblings are positive. The individual carrying a gene alteration for a late-onset disease may have an increased tendency for risky behaviours and may choose not to be a positive member of society. Relatives of an individual affected with a genetic disorder may be very frightened when they realise what their own future might be (SACGT, 2000). The individual who has inherited an altered disease-producing gene may foster deep resentment towards the parent who carries the altered gene. Parents and older generations may feel tremendous guilt for passing the altered gene to their children and grandchildren (Wertz, Famos & Reilly, 1994).

Economic issues

The nurse should consider the cost of genetic tests, which can range from hundreds to thousands of dollars, depending on the size of the gene being tested. In Australia, many of the tests are sent to overseas organisations for analysis.

Genetic tests differ from routine medical tests in many ways. The risks and benefits of genetic testing are numerous and complicated. Nurses have an obligation to maintain their knowledge regarding genetic testing, to advocate for the person, and to maintain ethical standards of care. Above all, nurses and midwives must be able to recognise the limits of their expertise and how to refer a person to genetic specialists and additional resources.

Assessment

Health promotion and health maintenance

Health promotion and health maintenance of the person are viewed as the foundations of all nursing and midwifery care. However, most individuals do not know their complete genetic make-up. Some know they carry an altered gene that causes a specific disease, but the majority of individuals do not know with certainty what their future health status will be. With no

sure knowledge of genetic make-up or whether a certain alteration in health status will occur (e.g. heart disease), healthy lifestyles are not always a priority. Imagine, then, if people knew their statistical risks for developing or inheriting disease by having complete access to the types of genes in their cells? Health promotion and health maintenance teaching and nursing interventions would be based on specific genes. The nurse could provide important, life-saving nutritional information to people based on their specific risks, and each individual then might be more inclined to maintain a proper diet, give up their sedentary lifestyle, increase exercise and decrease fast-food intake. Personal lifestyle choices would become more personal and monitoring health might take on a new meaning.

With knowledge of genetic conditions, the nurse can ensure health teaching and early detection of complications from genetic conditions with emphasis on primary and secondary care interventions. For example:

- A woman with a strong family history and/or mutations in the BRCA1 and BRCA2 tumour suppressor genes should begin monthly self breast exams and have screening clinical breast exams and mammographies at an earlier age than the general population.
- A man with a strong family history and/or mutations in the BRCA1 and BRCA2 tumour suppressor genes should report any mass, tenderness or swelling in the breast tissue and maintain early screening for prostate cancer.
- Aggressive colonoscopy screening every 1 to 2 years beginning at age 25 is important for the individual with a positive family history and/or mutations in the MLH1/MSH2 gene, which increases the risk of hereditary nonpolyposis colorectal cancer.

People receiving early intervention and health-promotion-focused care can live longer and with a much better quality of life than those who do not. The nurse must be able to identify both community-based and genetic-based resources that are available to assist the person with strategies to support both health promotion and health maintenance activities.

By simply integrating into practice the genetic aspects of assessment, observation and history gathering, the nurse can improve the standard of care delivered and have a very positive impact on the person. The nurse does not need to be a genetic expert, but with heightened awareness, appropriate inquiries and referrals to genetic specialists can be completed.

Health history

Nurses can improve the standard of nursing care and have a positive impact on people by integrating genetic concepts into their existing practice of inspection, observation and history gathering. Nurses should be able to recognise genetic features of physical assessment, basic patterns of inheritance and predisposition to development of disease. As health professionals integrate genetic concepts into their delivery of care, appropriate inquiries and referrals to genetic specialists can be completed.

Although family history has long been a part of nursing assessments, the relative importance of this assessment piece has recently increased as our knowledge of the interaction of genes and the environment has expanded. In phenotypically 'healthy' individuals, an accurate and complete family history can identify a single-gene (Mendelian) disorder or a mitochondrial, multifactorial or chromosomal inheritance pattern as well as guide the prevention, diagnosis and treatment of common complex diseases such as cardiovascular disease and cancer. A family history illustrates the interaction of genes and the environment for an individual and consequently provides a basis for individualised disease prevention (Guttmacher et al., 2004). Although an individual's inheritance risks from their own genotype are non-modifiable, knowledge of an individual's increased risk of chronic disease can influence lifestyle choices, clinical management, and sometimes risk reduction and prevention of the disease. Knowledge of a family history can also guide diagnostic workups and clinical treatment (Guttmacher et al., 2004).

Pedigrees

A nurse should know how to take a family history, record the history in a pedigree and think 'genetically'. A pedigree is a pictorial representation or diagram of the medical history of a family. Multiple symbols are utilised to present this picture (see Figure 7.6) and the finished pedigree presents a family's medical data and biological relationship information at a glance (see Figure 7.7). A pedigree provides the nurse, genetic counsellor or geneticist with a clear, visual representation of relationships of affected individuals to the immediate and extended family. It can identify other individuals in the family who might benefit from a genetic consultation. It can also

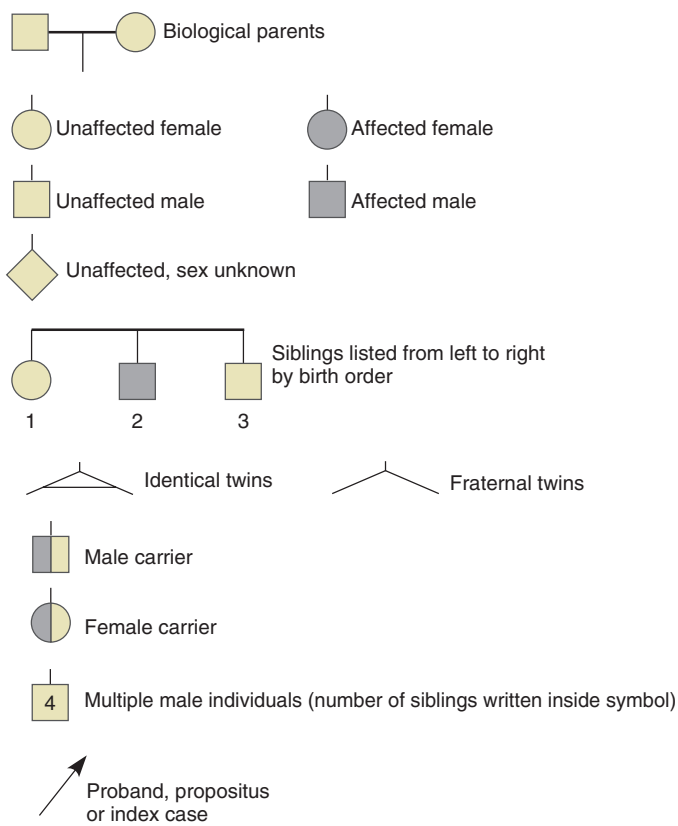


FIGURE 7.6 ■ Selected standardised symbols for use in drawing a pedigree

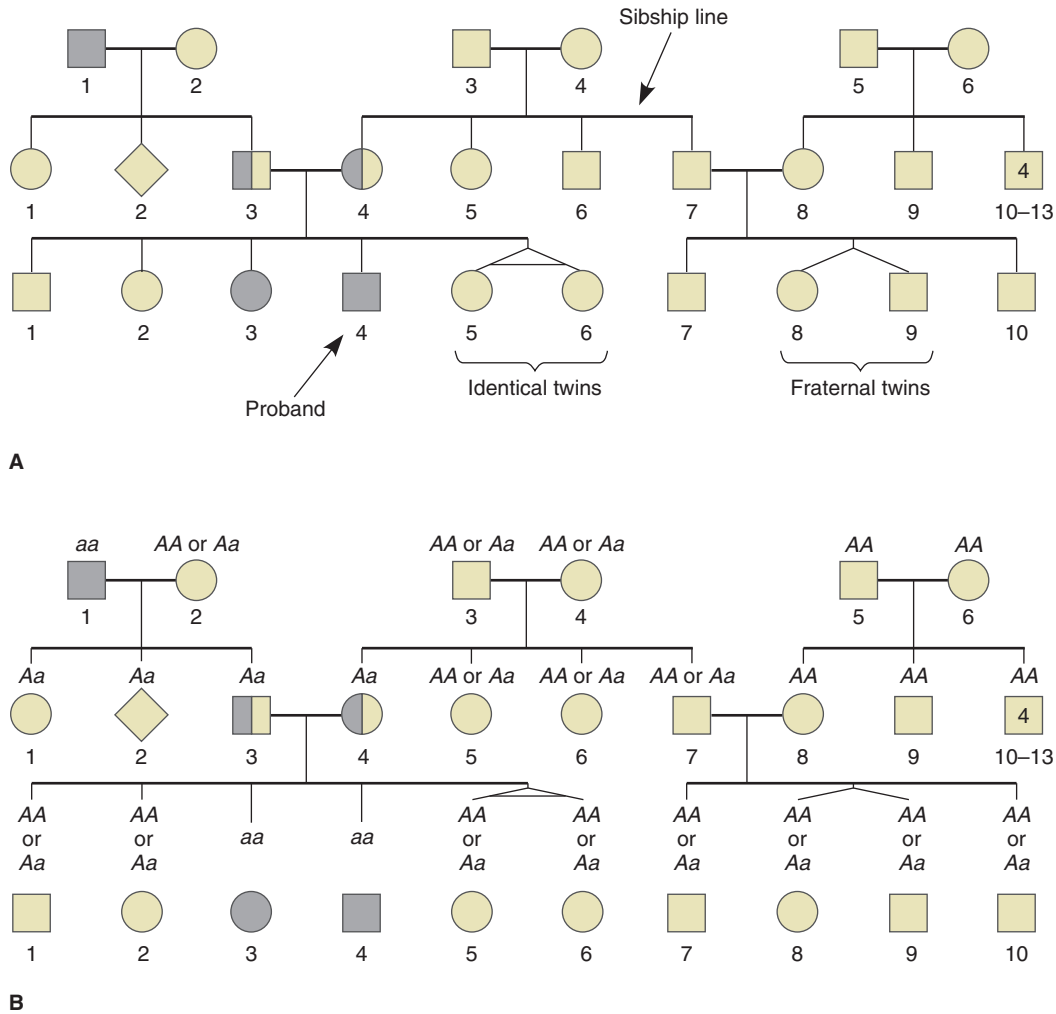


FIGURE 7.7 ■ Sample three-generation pedigree. *A*, A representative pedigree for a single character or genetic condition through three generations. *B*, The most probable genotypes of each individual in the pedigree for an autosomal recessive condition, represented by AA, Aa, or aa

Source: Concepts of genetics by W. S. Klug, M. R. Cummings, C. A. Spencer & M. A. Palladino (11th ed.), p. 60, Figure 3.13. Copyright © 2012. Printed and electronically reproduced by permission of Pearson Education, Inc., Upper Saddle River, New Jersey.

identify a single-gene alteration pattern of inheritance or a cluster of multifactorial conditions, and a referral and/or reproductive risk teaching for the individual and family can result. A family's learning can be enhanced by the visual teaching contribution a pedigree can provide and which may also clarify any inheritance misunderstandings or misconceptions. Box 7.8 lists steps for creating a family pedigree. If correctly and fully completed, a pedigree allows all healthcare professionals working with the individual or family to quickly see what history and background information has been collected (see Box 7.9).

It is important to gather a three-generation family pedigree even if the nurse believes this is a first occasion of the condition within a family (see Figure 7.7). A condition without any identifiable inheritance pattern on the pedigree may be due to a new mutation or variable expressivity. Throughout the process of gathering family history assessment data, the nurse must remember family confidentiality at all times: all information related to a pedigree is confidential information. The history may reveal sensitive details that include infertility problems, elective termination of pregnancies or non-paternity. This information may not even be known by a current partner or immediate and extended family members. Other sensitive issues include pregnancies conceived by technology, a history of suicides, drug or alcohol abuse, and same-sex relationships. Box 7.10 lists ethical implications of genetic information.

Challenges inherent in recalling the family history include the person's inability to remember any conditions that may have been surgically repaired and then forgotten, or reporting conditions that may have been attributed incorrectly to other causes. Also, the family history may contain information previously unknown to extended family members. Reproductive decisions may have been made that were against the family's religious or cultural beliefs. Both immediate and extended

BOX 7.8 Steps in drawing a pedigree

I. How to:
   1. Work in pencil.
      a. Family historians often remember additional relatives and details only after questioning is almost completed.

II. Organisation:
   1. Begin recording data in the middle of the sheet of paper.
      a. Allow enough room for both the maternal and paternal sides of the family.
   2. Use only standard pedigree symbols.
      a. For example, males are represented by squares and females by a circle.
   3. The male individual in a couple is placed on the left of the relationship line and the paternal side of the family also goes on the left-hand side of the paper.

III. Determining family relationships:
   1. The clinician should determine the relationships within the family by asking questions such as:
      - Do you have a partner or are you married?
      - How many biological brothers and sisters do you have?
      - How many children do you have?
      - Do all the children have the same biological father?
      - Do all the children share the same mother and father?
   2. Referral to 'the baby's father or mother' can be helpful until a relationship or marriage is established between the parents.
   3. Referral to a 'union' if marriage does not exist can also help communication.
   4. Determine if each individual is married, has children, signs and symptoms, etc., before continuing on to the next individual.
   5. Always ask if there is any chance the mother could be related to the father or if any other parents in the family are blood relatives.
      - To determine consanguinity.

IV. Who should or should not be included:
   1. To ensure accuracy, the pedigree should include the parents, offspring, siblings, aunts, uncles, grandparents and first cousins of the individual seeking counselling.
   2. Detailed information about the spouses of the proband's family can be omitted unless there is a history of some kind of disorder or condition. The proband is the first clinically affected family member diagnosed with a genetic disorder. For example, a baby born with Down syndrome may be the proband.
   3. Eliminating persons or any information that do not contribute any valuable information can help keep the pedigree small and more manageable.

V. Recording the family history:
   1. Determine the approximate size of the family.
   2. Record the family's ethnic background at the top of the page.
   3. The initial drawing should begin with the proband, or the person who is affected with the genetic condition.
      a. This is usually the reason someone is seeking a genetic referral.
   4. The proband is marked with an arrow on the pedigree.
   5. Draw and mark the symbols for the brothers and sisters of the proband. Draw the relationship line, the line of descent, marriage or union line, and symbols for the parents of the proband.
      a. Repeat this step for any children of the proband or children of the proband's brothers and sisters.
   6. Children resulting from a mating (siblings) should be recorded in descending order of their birth, with the oldest sibling on the left.
   7. Continue with symbols for all immediate relatives drawn previously and then draw and mark symbols for paternal grandparents and indicated relatives followed by the same for the maternal grandparents and relatives.
   8. A legend key should contain all of the correct symbols for each indicated disease.
   9. Record the age of onset of common and complex diseases and/or conditions such as coronary heart disease; diabetes mellitus; hypertension; colon, breast, ovarian or endometrial cancer; and stroke.
   10. The pedigree should include at least three generations.
      a. Generations are symbolised by Roman numerals along the left-hand side of the paper with the first generation marker, I, at the top.
      b. Each person in the generation should follow an imaginary horizontal line from left to right.
   11. The names of each individual (maiden names in case of married women) and their dates of birth should be included along with half-siblings, pregnancy losses, stillbirths, previous marriages and adopted children.

VI. Other:
   1. Consanguinity may be suspected if the historian repeatedly gives the same last name on both sides of the family.
      a. Consanguinity can be confirmed by asking if any relatives in the family have ever had a child together.

VII. Completing the pedigree:
   1. When completed, the pedigree should be dated and signed with the name, credentials and position of the person drawing it.

Source: Data from *The practical guide to the genetic family history* by R. L. Bennett (1999). New York: Wiley-Liss.

BOX 7.9 Specific facts and health information to include in a pedigree

- Age/birth date or year of birth
- Age of death (year, if known)
- Cause of death
- Age at diagnosis
- Full siblings versus half- or step-siblings
- Pregnancy with gestational age (last menstrual period (LMP)) or estimated date of delivery (EDD)
- Infertility versus no children by choice
- Pregnancy complications with gestational ages noted (e.g. 6 wks, 32 wks)
- Miscarriage (spontaneous abortion (SAB))
- Stillbirth (SB)
- Pregnancy termination (TOP)
- Relevant health information (e.g. height and weight)
- Affected/unaffected status—define shading of symbols in a legend key
- Ethnic background
- Consanguinity
- Date pedigree taken or updated
- Name of person who took pedigree and credentials
- Key or legend

family members may be unaware of these 'family skeletons' and the person may be very reluctant to reveal this information (Bennett, 1999; Bowers, 2002).

Genetic physical assessment

An assessment of newborns and children with a genetics focus on minor and major anomalies is essential. A minor anomaly or malformation is an unusual or morphological feature that in itself is of no serious medical or cosmetic concern to the individual or family. A major anomaly is a serious structural defect at birth that may interfere with normal functioning of body systems and may lead to a lifelong disability or even an early death (Aase, 1992).

Some children grow into adults whose physical features may or may not have been recognised as potential links to genetic conditions. Anomalies that exist after a person is beyond expected developmental and cognitive milestones may go unnoticed, but they may still be important. The nurse can pick up cues to genetic problems by inspecting the person and other family members. If practical, nurses should ask to look at family photographs and examine them for common dysmorphic features and family traits. Subjective assessment data can also be valuable. A person's complaints of fatigue and joint pain may indicate the onset of hereditary haemochromatosis. A physical assessment that includes a genetics focus is important for nurses when caring for people of any age. An undiagnosed genetic condition may also have implications for reproductive decisions the person makes. However, genetic assessments and genetic referrals are important throughout the lifespan.

Nursing diagnosis and interventions

Nurses are responsible for comprehensively delivering the correct standard of care to people, but at the same time being aware of the limitations of their own knowledge and expertise. In addition to the continuous integration of genetic aspects into nurses' assessments of family history and physical assessment, nurses and midwives are also responsible for carrying out interventions that include initiating referrals to genetic specialists and delivering care to the individual or family in any of the following ways. Nursing diagnoses to consider include:

- *Anticipatory grieving*
- *Anxiety*
- *Disturbed body image*
- *Ineffective coping*
- *Decisional conflict*
- *Interrupted family processes*
- *Ineffective health maintenance*
- *Deficient knowledge*
- *Powerlessness*
- *Spiritual distress*.

Genetic referrals and counselling

After gathering assessment data that incorporates genetic concepts, the nurse and/or midwife is able to initiate a referral to genetic specialists if there are indicators for a genetic referral (see Box 7.11). The nurse and/or midwife should provide the person with information about the advantages of a referral to genetic specialists and the disadvantages of not following through with the referral. The nurse should inform the person that a genetic referral can provide information and answer many questions they may have concerning genetic health. Questions regarding the conditions, inheritance, availability of treatment, as well as economic, insurance and future implications, can be addressed.

People who are concerned about genetic disease may benefit from a genetic consultation whether or not genetic testing is available for that condition. Many people seek information and coping strategies as much as they do test results. Referral of a person with a suspected genetic problem to a geneticist or genetic clinic is an expected nursing responsibility in the same way as are referrals to a dietitian or a social worker. When in doubt, the nurse should contact a genetic counsellor or geneticist to discuss concerns.

PREPARATION FOR GENETIC REFERRALS AND GENETIC COUNSELLING Not knowing what to expect from a genetic referral is common, and fear of the unknown may cause anxiety for the person. To facilitate the referral to a genetic specialist, the clinical nurse should educate the person so that they know what to expect during as well as after a genetic evaluation.

Usually, before the first genetic evaluation visit the person will be contacted to provide a detailed medical and family history and to make an appointment for genetic consultation. The person should be prepared to give as exact a family history as possible so that a detailed three-generation pedigree can be constructed. The person should be informed that a genetic consultation usually lasts several hours. During the appointment, a genetic clinical nurse, genetic counsellor and/or physician will perform an initial interview, and a geneticist will examine the person in order to establish an accurate

BOX 7.10 Ethical implications of genetic information

Clinicians must consider the enormity of the ethical issues facing all families who have knowledge of their genetic make-up. The ethical issues a nurse may have to discuss with the person are numerous. A few of the issues are listed below.

Access to information

- Who should have access to personal genetic information and how will it be used?
- Do insurers, employers, courts, police force, schools, universities, adoption agencies and the armed forces have a right to access this information?

Self-perception

- How does personal genetic information affect an individual's perception of self?
- How does personal genetic information affect society's perceptions of that individual?
- How does personal genetic information affect an individual's cultural identity?
- How is self-identity and self-worth affected by a confirmed genetic risk or condition?

Family roles and relationships

- Should an individual be tested for an autosomal dominant condition if the siblings and/or parents are opposed to knowing if they, themselves, have the altered gene?
- Should potential mates have genetic information?
- Should two people with increased genetic risk be prohibited from having children?
- Should a child be tested?
- Should the father be told if genetic testing and/or genetic counselling reveals non-paternity?
- Should adoption records contain a complete genetic history of the biological parents?
- Is there an obligation to tell other family members if an altered gene that demands a change in lifestyle (nutrition, exercise, smoking, etc.) is diagnosed?
- Is there an obligation to tell other family members if an altered gene that causes early debilitation and/or death is diagnosed?

Informed consent

- Are all individuals receiving true informed consent and do they understand all of the consequences of agreeing to even a simple blood test in the doctor's office that may reveal a diagnosis or increased risk of a genetic condition?

Health and life insurance

- Should insurance companies have access to genetic test results?
- Should medical insurance costs be higher for persons with a known gene disease-producing alteration?
- Should medical insurance costs be higher for persons with a known increased risk of disease because of any gene alteration?
- Should medical insurance costs be higher for persons with known increased risk of disease because of any gene alteration if they make unhealthy lifestyle choices and do nothing to lower their risk?
- Should the individual be covered by medical insurance at all?
- Should individuals pay higher costs if they have children?
- Should individuals be required to have a large life insurance policy to financially protect their families?

Financial

- Should the child be eligible for government grants or any scholarship money?
- Should society be expected to financially support children through government programs or private insurance?
- What is the motivation to save money for the future?

Employment

- Should an employer have access to an individual's genetic profile?
- Should a young adult be hired even though they will burden the company with multiple sick days, higher insurance financial support, etc.?
- Should the individual receive promotional opportunities and increased job responsibilities if the employer knows there will be a large number of lost work days?
- Will the individual's productivity be affected by the genetic condition?

diagnosis. Tests may be ordered. These may include chromosome (cytogenetic) analysis, DNA-based testing, x-rays, biopsy, biochemical tests and linkage studies (Lashley, 2005). After the exam and the completion of any applicable testing, the geneticist and/or genetic counsellor will discuss the findings with the person and make recommendations. The discussion will include the natural history of the condition, the inheritance patterns, the current preventive or treatment options, and the risks to the person and/or family. The visit will also include opportunities for questions and answers, as well as the assessment and evaluation of the person's understanding. It is typical for the information retention of a person facing a new genetic diagnosis to be very low. This makes it imperative for the clinician to take advantage of opportunities to reinforce genetic concepts at a later time when the person is ready.

As the visit concludes, the person can expect appropriate referrals to be made, discussion of available services or research studies, and possible scheduling of a follow-up visit. A summary of the information is usually sent to the person, and their healthcare provider will receive a report if requested by the person.

Genetic healthcare providers present the person with information to promote informed decisions. They are also sensitive to the importance of protecting the individual's autonomy. A challenge during any visit to a genetic

BOX 7.11 Adult indicators for a referral to a genetic specialist

Adult history assessment data

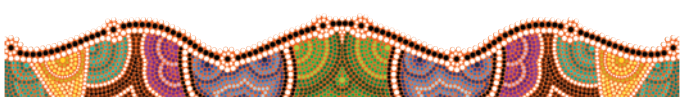
- Several closely related individuals affected with the same or related conditions:
  - Breast and ovarian cancer
  - Colon and endometrial cancer
  - Diabetes
  - Hypertension
  - Coronary heart disease
  - Thyroid cancer
  - Colon polyps
- A common disorder with earlier age of onset than typical (increase concern if it occurs in more than one family member):
  - Breast cancer: < 45–50 years of age or premenopausal
  - Colon cancer: < 45–50 years of age
  - Prostate cancer: < 45–60 years of age
  - Vision loss: < 55 years of age
  - Hearing loss: < 50–60 years of age
  - Dementia: < 60 years of age
  - Heart disease: < 40–60 years of age
  - Stroke: < 60 years of age
- A sudden or unexpected death in someone who 'seemed' healthy:
  - Renal disease
  - Asthma
  - Suicides

An individual with:

- Two or more conditions
- A medical condition and dysmorphic features
- Developmental delay with dysmorphic features and/or physical birth anomalies
- Learning disabilities
- Behavioural problems
- Unexplained:
  - Movement disorders
  - Seizures
  - Hypotonia
  - Ataxia
  - Infertility
- Disproportionate tall or short stature
- Proportionate short stature with dysmorphic features
- Atypical sexual development
- Premature ovarian failure

Source: American Medical Association (2004). *Family medical history in disease prevention.* Retrieved from www.ama-assn.org/resources/doc/genetics/family_history02.pdf. American Medical Association. Copyright 2005.

specialist is providing non-directive counselling. People should be permitted to make decisions that are not influenced by any biases or values from the nurse, counsellor or geneticist. Many people are accustomed to practitioners and nurses providing direction and guidance in their decision making so they may be very uncomfortable with the non-directional approach of the nurse. They may believe that the nurse or healthcare provider is withholding very bad news. The nurse should discuss the positives and negatives of each decision and present as many options as possible through the use of therapeutic listening and communication skills (Cunniff, 2001).



EDUCATION

The nurse and/or midwife must be aware of available genetic resources and participate in educating the person on genetic disorders as well as health promotion and prevention. Informing people of what to expect from a genetic referral, as well as clarifying and/or reinforcing information obtained during a genetic referral or from genetic test results, is also important.

Prior to teaching, the clinician assesses the person's cultural and religious beliefs. Are the gene alterations viewed as uncontrollable and believed to be occurring secondary to cultural belief, such as a stranger looking at the person? Or, are the gene alterations considered a 'punishment'? A person's readiness to learn can be influenced by cultural or religious beliefs and values. Obtaining educational materials in the person's native language will also help facilitate the teaching–learning experience.

The clinician must be aware of common inheritance misconceptions such as a person's belief that, with a 25% recurrence risk, after one child is affected, the next three children will be unaffected, or with a 50% recurrence risk, every other child will be affected. The recurrence risk *for each pregnancy* should be continually stressed by the nurse. People often believe that a family member has inherited the genetic condition because they look like or 'take after' a relative with a genetic condition. When new gene alterations or mutations are discussed, people will often exhibit surprise because no one else in the family has the condition, so they perceive that the trait or condition cannot possibly be inherited (Bennett, 1999). Helping people to understand these genetic concepts is fundamental to delivering the required standard of genetic nursing care.

Psychosocial care

To meet the person's psychosocial needs, the nurse should identify the person's expectations and needs, as well as their cultural and spiritual values and belief system. From where does the person receive strength? Denial of the genetic diagnosis is common and nurses must be aware of the person's state of acceptance. Individuals often will not believe that a chronic genetic condition exists. Nurses must also provide care to help alleviate any anxiety and/or guilt in the person. Anxiety about the unknown is common when awaiting diagnosis or test results, but individuals also experience anxiety from not understanding the future implications of a confirmed genetic disease. Guilt may be associated with knowledge of the existence of a genetic condition in a family. The nurse must support people as they contemplate telling extended family members, friends and neighbours about a confirmed diagnosis. People often do not want to tell extended family members until they are ready. The nurse should encourage open discussion and the expression of fears and concerns. Feelings of guilt and shame are very common as a person deals with the loss of the expectation and dream of a healthy, productive life. Reinforce that genetic alterations are caused by changes within a gene and not by superstitions related to sin or other cultural beliefs. However, it is important to remember that everyone has superstitions or beliefs and the nurse must remain non-judgmental. As mothers, fathers, siblings and extended family members provide continuous care for the person with a genetic condition, depression can result. Depression also can occur in the individual with the chronic condition. The clinician must maintain awareness of the possibility of depression and be proactive in obtaining support for the individual or family.

The nurse also is responsible for assessing the person's coping mechanisms as well as available family, spiritual, cultural and community support systems. Genetic conditions can cause a permanent strain on family dynamics and relationships. The counsellor may need to help the person reaffirm their own self-worth and value (Lashley, 2005). If seen in an academic setting, people may feel they are part of a 'production line' even though they are present for a very private problem (Cunniff, 2001). It is important to be sensitive to these perceptions, provide open communication and encourage discussion of feelings. Growth and development, and meeting adult developmental milestones, can be altered by actual or potential genetic disorders. Especially unique is the potential or actual inheritance of a late-onset condition such as Huntington's disease. The person with this altered gene may not meet any of the developmental tasks in moving through adulthood. Should the person get married, attend university, save money or worry about future? The nurse must identify the impact of genetic knowledge on activities of daily living but also on movement through developmental milestones. Both individual and family strengths need to be identified.

Working in collaboration with the other members of the healthcare team, nurses can refer the person to a support group. However, it is important to have the person's permission if the nurse is providing their name and contact information to a support group.

Another key role for the team is to help people with the often difficult task of communicating genetic information such as inheritance patterns to extended family members. Cultural values of autonomy and privacy are affected when a person must consider whether to communicate genetic information to extended family members who may also carry the altered gene. The history of a genetic alteration that may or may not cause disease can be extensive within a family, affecting multiple family members. Family members often have difficulty understanding that some genetic conditions have variable expressivity. Members of the extended family often are shocked and feel a profound sense of guilt that they are the one who has carried the gene alteration that caused their loved one to have a genetic condition.

Careful self-assessment of feelings is essential for every member of the team. Nurses play an important role in advocating for people and support their decisions even if the decisions contradict their own ideals and morals. Coping with genetic revelations and making genetic-related treatment decisions are difficult activities for everyone. Team members must remember that people will need resources and support and also help in gathering information about reproductive options.

Evaluation

Expected outcomes of delivering healthcare with a genetic focus include:

- The person will make informed and voluntary decisions related to genetic health issues.
- The person will accurately identify:
  - basic genetic concepts and simple inheritance risk probabilities
  - what to expect from a genetic referral
  - the influence of genetic factors in health promotion and health maintenance
  - differences between medical and genetic tests
  - social, legal and ethical issues related to genetic testing.

VISIONS FOR THE FUTURE

Nurses are often the primary caregivers to whom people turn for information, guidance and clarification of ideas. Their role is essential not only in providing direct nursing care but as a member of the community. As more information about the genetic revolution becomes available to consumers—in areas such as pharmacogenomics, gene transfer, ethics, genetic engineering and stem cell research—the role of nurses not only remains vital but will also grow enormously. Clinicians should remain educated, informed, knowledgeable and ready to discuss trends and changes with individuals and their families.

CHAPTER HIGHLIGHTS

- Nurses and midwives are responsible for basic genetic knowledge and for delivering the expected standard of genetic nursing care.
- When cell division does not occur as expected, chromosomal alterations on the autosomes or sex chromosomes can result.
- Chromosomal alterations can be seen in a human karyotype.
- Protein-directing genes are very important to life and functioning as a human being because proteins are very specialised and perform a variety of functions within the cell.
- Different forms of genes are alleles.
- A person may be identified as heterozygous or homozygous for a single gene.
- Some gene alterations cause disease and some are protective from disease.
- Mitochondrial gene alterations are inherited from the mother and primarily involve high-energy organs such as skeletal muscles, brain and heart muscle.
- Multifactorial inheritance does not follow Mendelian inheritance patterns.
- Genetic healthcare providers present the person and their family with information to promote informed decisions.
- Many types of genetic tests are available and they differ from routine medical tests.
- All genetic tests have special considerations related to social, financial, ethical and legal implications.
- Basic genetic nursing care involves family risk assessment through a detailed family history, drawing a three-generation pedigree and integrating genetic concepts into a physical assessment.
- Care should be taken in initiating a referral to a genetic specialist.
- Knowledge of the principles of inheritance allows the clinician not only to offer and reinforce genetic information to individuals and their families, but also to assist them in managing their care and in making reproductive decisions.
- Genetic concepts can be applied to health promotion and health maintenance.
- The nurse must be aware of the social, ethical, cultural and spiritual issues related to the delivery of healthcare.

CONCEPT CHECK

1 A person you are caring for is discussing the inheritance of an autosomal dominant trait. He has the condition and his wife does not. They have one child without the condition. The nurse would be correct in explaining to the person that he is most likely which genotype?
1 FF
2 Ff
3 ff
4 X_fY

2 A male diagnosed with Fabry disease is admitted to the unit. Which statement made by the person would indicate to the nurse that he understands Mendelian inheritance concepts? 'I have the disease because . . .' (Select all that apply.)
1 'my mother had Fabry disease and my father did not'
2 'my father's mother had Fabry disease'
3 'my grandmother's brother had Fabry disease'
4 'my father has Fabry disease'

3 The nurse is providing information regarding genetic testing to a couple who believe they are carriers of an autosomal recessive gene alteration. Which statement is appropriate?
1 'If both of you are carriers, all of your sons will be affected and all of your daughters will be carriers.'
2 'Chromosomal studies will reveal if you are actually a carrier.'
3 'Newborn screening will reveal if your child is affected.'
4 'During the genetic evaluation, you will be asked to provide at least a three-generation family history.'

4 The nurse knows that which assessment data obtained during a family history may suggest a genetic condition or inherited susceptibility to a common disease? (Select all that apply.)
1 breast cancer at age 33
2 a sibling who died unexpectedly while playing basketball at age 66
3 colon polyps in four third-degree relatives
4 a brother's unexplained infertility

5 When analysing a family pedigree, the nurse/midwife notes the pedigree demonstrates that successive generations contain affected individuals, both males and females are affected, and there is no father-to-offspring inheritance. What is the most likely pattern of inheritance?
1 autosomal dominant
2 autosomal recessive
3 X-linked recessive
4 multifactorial
5 mitochondrial

6 When developing a teaching plan, which statement is a correct rationale regarding the health promotion and health maintenance benefits from an assessment of family history? (Select all that apply.)
1 Clinical treatment options can be more focused.
2 Prophylactic treatments can be started early.
3 Specific diet, exercise regimen and genotype can be determined.
4 Single-gene alteration can be diagnosed.

7 The nurse is recording a family pedigree. Which would be correct to include in drawing the pedigree?
1 Detailed information is important for all persons recorded on the pedigree.
2 The maternal side of the family should be placed on the left of the page.
3 The proband is marked with an arrow and a 'P'.
4 Two generations should be recorded and labelled with Roman numerals.

8 The clinician would consider which assessment finding(s) as minor anomalies? (Select all that apply.)
1 café au lait spots
2 ear pits
3 atrial septal defect (ASD)
4 hypertelorism

9 Which are appropriate concepts for the clinician to include when developing a teaching plan for the individual prior to genetic testing? (Select all that apply.)
1 Predispositional genetic tests are medically indicated when the seriousness and mortality of the disease can be reduced with knowledge of the gene alteration.
2 To meet quality assurance, laboratories should hold a CLIA88 certification.
3 A mutation panel contains the most common gene alterations, but may not include all of the disease-causing mutations.
4 Family members affected by genetic test results have a legal right to the test results.

10 The person asks the nurse if a genetic referral is necessary. Which information would be appropriate for the nurse to provide? Most likely genetic specialists will: (Select all that apply.)
1 provide direction for important decision making
2 complete chromosomal studies
3 ask to see photographs of relatives
4 provide information about the natural history of the condition

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CHAPTER 8

NURSING CARE OF PEOPLE IN PAIN

ADAM BURSTON, FLORAIDH CORFEE

KEY TERMS

LEARNING OUTCOMES

- Describe the neurophysiology and theories of pain.
- Compare and contrast definitions and characteristics of acute and chronic pain.
- Discuss factors affecting individualised responses to pain.
- Clarify myths and misconceptions about pain.
- Introduce interprofessional care for the person in pain, and discuss pharmacological and non-pharmacological treatment alternatives.
- Describe a framework for providing individualised nursing care for the person experiencing pain.

CLINICAL COMPETENCIES

- Assess pain intensity; quality; location; pattern; intensifiers; nullifiers; side effects of analgesics; effect on physical, psychological and social function, and on mood; and support for managing pain.
- Determine the person's desire and preference for pain management.
- Intervene with pharmacological and non-pharmacological methodologies. Administer medications knowledgeably and safely.
- Teach the person, their family and significant others about effective pain control.
- Evaluate effectiveness of pain relief; retreat or adjust doses of medication; and intervene as necessary.
- Revise plan of care according to the person's response to interventions and need for control.

The International Association for the Study of Pain (IASP) defines pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' (ANZCA, 2010). Pain is a subjective response, not always measureable or observable, to both physical and psychological stressors which all people experience at some point during their lives. Although pain is usually experienced as uncomfortable and unwelcome, it also serves a protective role, and may warn of potential health-threatening conditions. For this reason, pain needs to be assessed and considered an essential vital sign.

Each individual pain event is a distinct personal experience. It is influenced by physiological, cognitive, psychological, sociocultural and spiritual factors. Pain is the symptom most associated with describing oneself as ill, and it is the most common reason for seeking healthcare (Merskey, 2014).

However, the inability to communicate pain does not imply the person does not need suitable pain-relieving treatment. The clinician must be vigilant in the assessment and management of any person's pain (ANZCA, 2010). Linguistic and cultural familiarity do not guarantee effective communication of the complexities of pain (Hadjistavropoulos et al., 2011).

NEUROPHYSIOLOGY AND THEORIES OF PAIN

Neurophysiology

The peripheral nervous system is composed of two types of neurons: sensory and motor. Pain is perceived through the sensory neurons and responded to through the motor neurons. Connections or synapses occur within the spinal cord and again within the central nervous system (CNS), where cognitive analysis of the painful stimulus leads to a response.

Nerve receptors of pain are called **nociceptors** (see Figure 8.1). These are the nerve receptors that are sensitive to pain (noxious stimuli) and give an immediate response when stimulated. They are located at the ends of small afferent neurons and are woven throughout all tissues of the body, except the brain. Nociceptors are especially numerous in the skin and muscles. Pain occurs when biological, mechanical, thermal, electrical or chemical factors stimulate nociceptor activity to the spinal cord. The intensity and duration of the stimuli determines the sensation.

Reactions are caused either by persistent mechanical, chemical or thermal stimuli that create a cascade of chemical mediators which can act directly on the cell or via a messenger system

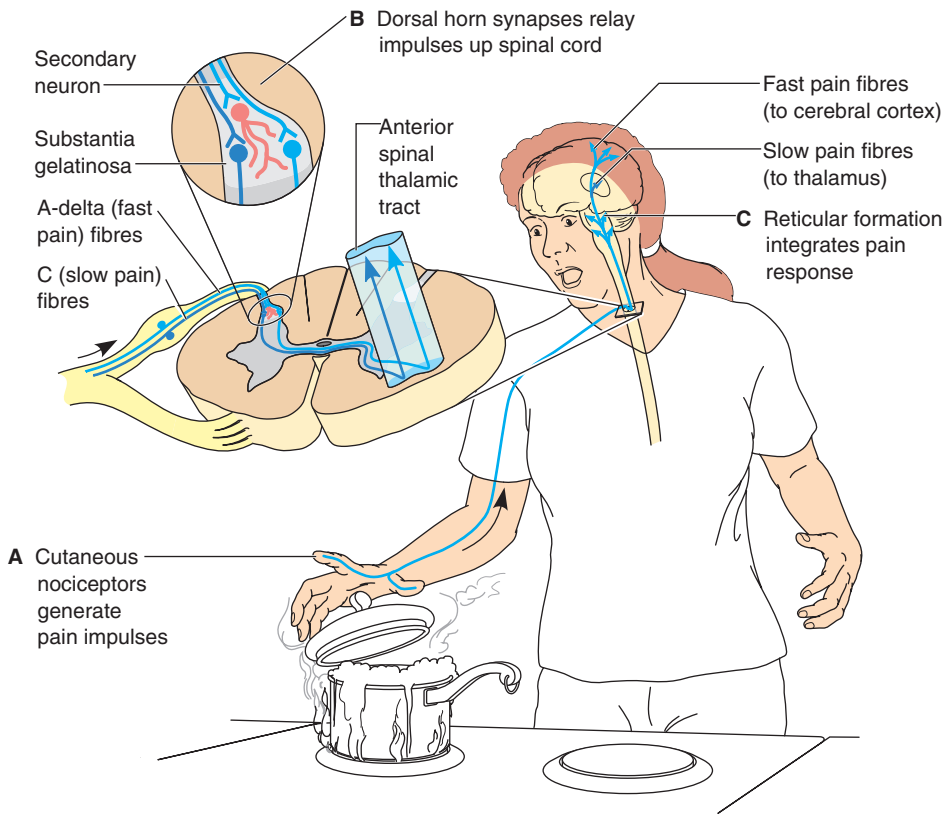


FIGURE 8.1 ■ *A*, Cutaneous nociceptors generate pain impulses that travel via A-delta and C fibres to the spinal cord's dorsal horn. *B*, Secondary neurons in the dorsal horn pass impulses across the spinal cord to the anterior spinothalamic tract. *C*, Slow pain impulses ascend to the thalamus, while fast pain impulses ascend to the cerebral cortex. The reticular formation in the brainstem integrates the emotional, cognitive and autonomic responses to pain

to activate **nociception**. Bradykinin, a polypeptide element of the kinin protein system, is a pain-producing chemical; other biochemical sources of pain include prostaglandins, substance P, histamine, leukotriene B, hydrogen ions and serotonin (ANZCA, 2010; Crisp, Taylor, Douglas & Rebeiro, 2012). These biochemicals are thought to bind to nociceptors in response to noxious stimuli, causing the nociceptors to initiate pain impulses.

Ascending pain pathways

The ascending pathways of pain are illustrated in Figure 8.1 and are summarised as follows:

1. Pain is perceived by the nociceptors in the periphery of the body—for example, in the skin or viscera. Cutaneous pain is transmitted through two types of nerve fibres that transmit signals:
   - A-delta fibres are medium-diameter, myelinated and respond primarily to high-intensity mechanical or heat stimuli, causing sharp, stabbing-like pain.
   - Smaller C nerve fibres are small-diameter, unmyelinated, and respond to lower-intensity thermal, chemical or mechanical cold stimuli, resulting in a dull and aching type of pain. The pain from deep body structures (such as muscles and viscera) is primarily transmitted by C fibres and is commonly associated with persistent pain.

   Both A-delta and C fibres are involved in most injuries. For example, if a person bangs their elbow, A-delta fibres transmit this pain stimulus within 0.1 second. The person feels this pain as a sharp, localised, smarting sensation. One or more seconds after the blow, the person experiences a duller, aching, diffuse sensation of pain impulses carried by the C fibres.
2. Second-order sensory neurons transmit the impulses from the afferent neurons (A-delta fibres and C fibres) through the dorsal horn of the spinal cord, where they synapse in the substantia gelatinosa. This first synapse is important for modulation input in the CNS. The impulses then cross over to the anterior and lateral spinothalamic tracts.
3. The impulses of the second-order neurons ascend via the anterior and lateral spinothalamic tracts and pass through the medulla and midbrain to the thalamus.
4. In the thalamus and cerebral cortex, the pain impulses are perceived, described, localised and interpreted, and a response is formulated. A noxious impulse becomes pain when the sensation reaches conscious levels and is perceived and evaluated by the person experiencing the sensation.

Some pain impulses ascend along the paleospinothalamic tract in the medial section of the spinal cord. These impulses enter the reticular formation and the limbic systems, which integrate emotional and cognitive responses to pain. Interconnections in the autonomic nervous system may also cause an autonomic response to the pain. In addition, deep nociceptors often converge on the same spinal neuron, resulting in pain that is experienced in a part of the body other than its origin.

Inhibitory mechanisms

Efferent fibres run from the reticular formation and midbrain to the substantia gelatinosa in the dorsal horns of the spinal cord. Along these fibres, pain may be inhibited or modulated. The analgesia system is a group of midbrain neurons that transmits impulses to the pons and medulla, which in turn stimulate a pain inhibitory centre in the dorsal horns of the spinal cord. The exact nature of this inhibitory mechanism is unknown.

The most clearly defined chemical inhibitory mechanism is fuelled by endorphins (endogenous morphines) which are naturally occurring opioid peptides present in neurons in the brain, spinal cord and gastrointestinal tract. Endorphins in the brain are released in response to afferent noxious stimuli, whereas endorphins in the spinal cord are released in response to efferent impulses. Endorphins work by binding with opiate receptors on the neurons to inhibit pain impulse transmission (see Figure 8.2).

Pain theories

Several theories attempt to explain the response to pain and the diversity of human experiences with pain. Specificity and pattern theories describe nerve impulses of varying intensity terminating in pain centres in the forebrain. These theories provide explanations of the neurophysiological basis of pain.

The gate-control theory

In 1965, Melzack and Wall postulated the gate-control theory (Perl, 2011). A gating mechanism exists at the spinal cord level where nerve transmission may be blocked by competing

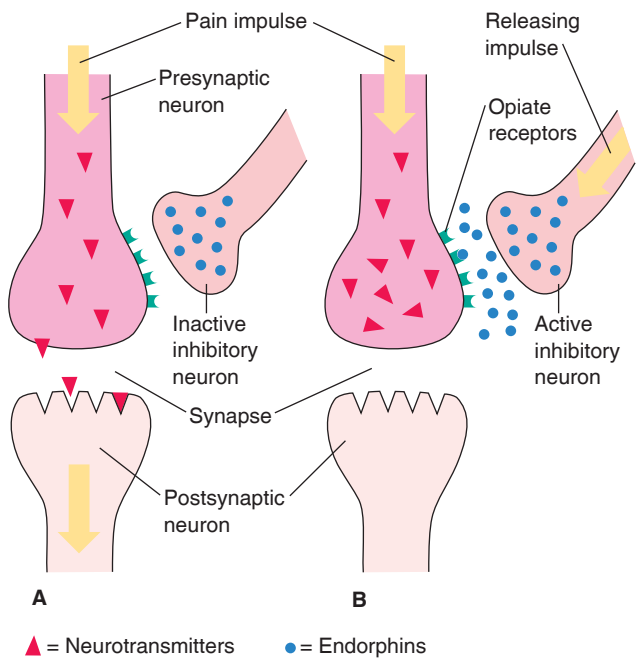


FIGURE 8.2 ■ *A*, Pain impulse causes presynaptic neuron to release burst of neurotransmitters across synapse. These bind to postsynaptic neuron and propagate impulse. *B*, Inhibitory neuron releases endorphins, which bind to presynaptic opiate receptors. Neuro-transmitter release is inhibited and pain impulse is interrupted

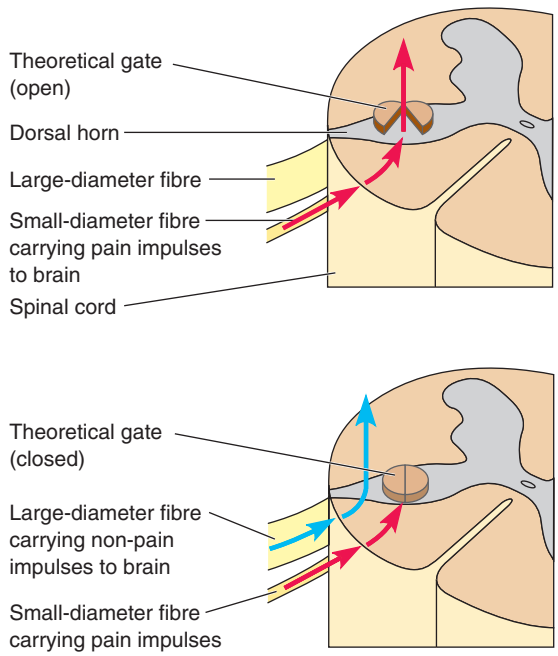


FIGURE 8.3 ■ The spinal cord component of the gate-control theory. Pain transmission by small-diameter fibres is blocked when large-diameter fibres carrying touch impulses dominate, closing the gate in the substantia gelatinosa

impulses. This explains the ability of even low-intensity stimulation such as light brushing of the skin to successfully block the experience of pain. Pain perception results from the interaction of two systems: the substantia gelatinosa in the dorsal horns of the spinal cord (see Figure 8.3), which regulates impulses entering or leaving the spinal cord; and an inhibitory system within the brainstem.

A-delta and C fibres in the spinal cord carry fast and slow pain impulses, while large-diameter A-beta fibres carry impulses for tactile stimulation from the skin. In the substantia gelatinosa, these impulses encounter a 'gate' thought to be opened and closed by the domination of either the large-diameter or the small-diameter fibres. If impulses along the small-diameter pain fibres outnumber impulses along the large-diameter touch fibres, the gate is open, and pain impulses travel unimpeded to the brain. If impulses from the touch fibres predominate, they will close the gate, and the pain impulses will be 'turned away' at the gate. This explains why light stimulation such as massaging a stubbed toe can reduce the intensity and duration of the pain.

The second system described by gate-control theory is the inhibitory system located in the brainstem. It is believed that cells in the midbrain, activated by a variety of stimuli such as opiates, psychological factors, or even simply the presence of pain itself, signal receptors in the medulla which in turn stimulate nerve fibres in the spinal cord to block the transmission of impulses from pain fibres. Ongoing research demonstrates that the control and modulation of pain is much more complex than the description supplied by gate-control theory, which served as a basis for further research about pain-modulating systems. Ongoing theory development of the neuromatrix integrates cultural, genetic, attention, expectation, personality and stress factors with basic neurophysiological function (Perl, 2011). This neuromatrix is particularly useful to understand chronic pain and phantom limb pain, considering there is no defined relationship between tissue injury and the pain experience.

Central sensitisation

Another pain theory that is quite significant in clinical terms describes the effect of sensitising the central and peripheral nervous system to painful stimuli. Central sensitisation manifests as pain hypersensitivity. This theory suggests painful signals create a cascade of changes in the nervous system, which in turn increase the responsiveness of the peripheral and central neurons. These changes, in turn, amplify light touch and pressure, causing enhanced after sensations, increased temporal summation and response to future signals (IASP, 2010). Studies of infants undergoing painful procedures show that those who received analgesia experienced reduced sensitivity to future painful events, while those who did not receive analgesia experienced greater sensitivity (Shen & El-Chaar, 2015). Sensitisation occurs from nociceptive barrage as well as inflammation following an injury or incision. In adults this theory indicates the value of preventing sensitisation as well as treating perceived pain with multimodal pain therapy.

DEFINITIONS AND CHARACTERISTICS OF PAIN

Acute pain

Acute pain has a sudden onset, is usually temporary, is localised and is the common, everyday pain that most people know. Pain that lasts for less than 3 months and has an identified cause is classified as acute pain (Hague & Shenker, 2014). The onset is usually immediate, most often resulting from tissue injury from trauma, surgery or inflammation. The pain is often sharp and localised, although it may radiate. Acute pain warns of actual or potential injury to tissues. As a stressor, it initiates the fight-or-flight autonomic stress response. Characteristic physical responses include tachycardia, rapid and shallow respirations, increased blood pressure, dilated pupils, sweating, pallor and alterations to blood sugar levels. The three major types of acute pain are as follows:

1. *Somatic pain* arises from nerve receptors originating in the skin or close to the surface of the body. Somatic pain may be either sharp and well localised, or dull and diffuse. It is often accompanied by nausea and vomiting.
2. *Visceral pain* arises from body organs, and is dull and poorly localised because of the low number of nociceptors. The viscera are sensitive to stretching, inflammation and ischaemia, but relatively insensitive to cutting and temperature extremes. Visceral pain is associated with nausea and vomiting, hypotension and restlessness. It often radiates or is referred, that is perceived at a location other than that of the painful stimulus. It may be described as cramping, intermittent pain or colicky pain.

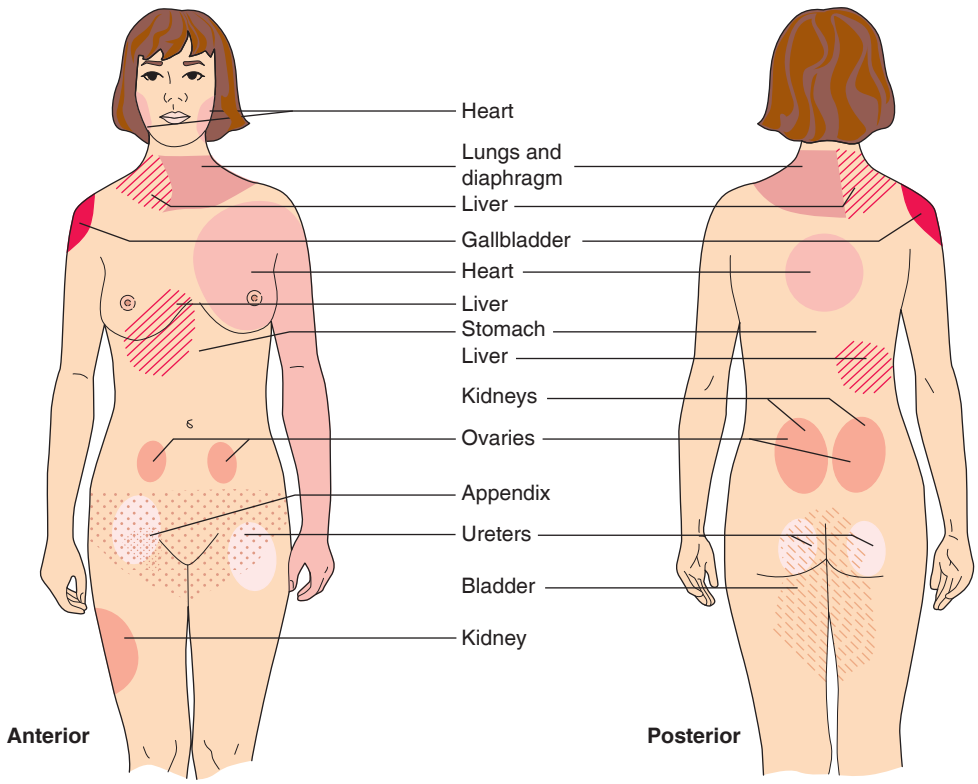


FIGURE 8.4 ■ Referred pain is the result of the convergence of sensory nerves from certain areas of the body before they enter the brain for interpretation. For example, a toothache may be felt in the ear, pain from inflammation of the diaphragm may be felt in the shoulder, and pain from ischaemia of the heart muscle (angina) may be felt in the left arm

3. *Referred pain* is perceived in an area distant from the site of the stimuli and commonly occurs with visceral pain. Visceral fibres synapse at the level of the spinal cord, close to fibres innervating other subcutaneous tissue areas of the body (see Figure 8.4). Pain in a spinal nerve may be felt over the skin in any body area innervated by sensory neurons that share that same spinal nerve route. Body areas defined by spinal nerve routes are called dermatomes (see Chapter 40).

Persistent (chronic) pain

Persistent (chronic) pain is ongoing and prolonged pain. It is not always associated with an identifiable cause but often arises from an acute situation such as post trauma, herpes zoster, acute back pain and postoperative surgical pain.

Predictive factors for chronic post-surgical pain include preoperative, intraoperative and postoperative factors:

- Preoperative considerations include pain, moderate to severe lasting for more than one month, repeat surgery, psychological vulnerability (e.g. catastrophising), preoperative anxiety.
- Intraoperative considerations include a surgical approach with risk of nerve damage.
- Postoperative factors include pain (acute, moderate to severe), radiation to the area, depression, neuroticism and anxiety (adapted from ANZCA, 2010, p. 11).

Neurological changes that can occur with persistent pain are changes in perception. Physical changes that result are loss of muscle mass, deconditioning, postural changes, alterations in appetite and weight, constipation and sleep disturbances. Persistent pain is complex and is poorly understood. It is suggested that when persistent pain is present it can develop to an individual disease process because of the secondary changes that occur in the body (Cousins, 2012).

The 'persistent (chronic) pain syndrome' refers to the unspecific behaviours that can occur with persistent pain. Often a cycle of persistent pain and disability causes physical deconditioning, drug tolerance, reduced activity, passive treatments, distorted beliefs and social stresses such as financial pressures, altered gender roles and the destruction of intimate relationships. From a psychological perspective the person with persistent pain experiences anxiety, anger, hopelessness and frustration, which can result in the person becoming depressed, withdrawn and irritable (Hill, 2014). Although persistent pain may range from mild to severe, its unrelenting presence often results in the pain itself becoming a pathological process requiring intervention.

Breakthrough pain

Breakthrough and incident pain relates to how pain progresses through time. **Breakthrough pain** occurs between doses of analgesia; it can be prevented by giving breakthrough analgesia more

frequently, increasing the dose of the analgesia, or increasing the slow (continuous) release medication. **Incident pain** occurs when procedures, dressings or activity increase the pain experience. Incident pain can often be predicted and analgesia should be available and given prior to the activity commencing.

Neuropathic pain

Neuropathic pain may be acute or chronic resulting from injury or disease that affects the peripheral or central nervous systems. Acute neuropathic pain may result from lesions or entrapment of nerves (Brown et al., 2015; Taverner, 2014). Neuropathic pain can be caused by numerous factors such as trauma, surgery, inflammation, toxicity, and immunological and vascular changes affecting either the central nervous or the peripheral nervous systems of the body. Examples of neuropathic pain arising from the CNS are post-stroke pain and spinal cord injury. Peripheral causes of neuropathic pain are diabetic neuropathy, HIV-related neuropathies and tumours invading the nerve area and surgery.

Common surgeries that have a higher incidence postoperatively of persistent neuropathic pain are amputation (phantom limb pain), thoracotomy, mastectomy, hernia repair and cholecystectomy (ANZCA, 2010). The person with neuropathic pain will often state their pain is burning, shooting or electric; they might comment on numbness in and around the area or the sensation of pins and needles. Diagnosis is usually made with a detailed history, personal description and pain assessment.

- *Complex regional pain syndrome* (*CRPS*) is neuropathic pain that results from nerve damage from either major or minor trauma (Marinus et al., 2011). CRPS is recognised as a persistent pain condition in which functional restoration by constant rehabilitation is the main goal (Pollard, 2013). Common features of CRPS are continuous, severe pain (usually burning or electric), trophic changes (hair and nails alter in growth), vasospasm changes (vasodilation causing the limb to become hot, red and swollen, followed by vasoconstriction) and limb immobility causing muscle wasting. It is common for CRPS to develop from minor injuries and research suggests that the pathophysiology is characterised by an 'aberrant host response to tissue injury' (Marinus et al., 2011, p. 637).
- *Neuralgias* are painful conditions that result from damage to a peripheral nerve caused by infection or disease. Postherpetic neuralgia (following shingles) is an example occurring in 50% of those who are over 50 years and 75% of those who are over 75 years. Trigeminal neuralgia can occur as an acute exacerbation of neuropathic pain. Most causes are idiopathic and the main treatment is with carbamazapine (ANZCA, 2010).

Phantom pain

Phantom pain is a common condition among amputees. Phantom pain commonly resembles pre-injury pain (if it was present) and is exacerbated by stump problems, ill-fitting prostheses and back pain. Phantom pain also occurs in other parts of the body after surgical removal (e.g. mastectomy, tongue). Phantom pain is *not* phantom sensation which resolves over time as the sensation retreats into the stump and is a contributory mechanism to phantom pain development (ANZCA, 2010).

Central pain

Central pain is related to a lesion in the brain or spinal cord that may spontaneously produce a high-frequency burst of impulses from the ascending spinothalamocortical pathways; their relays or end stations in the brain or spinal cord create a sensation of pain. A vascular lesion, tumour, trauma or inflammation may also cause central pain. Thalamic pain is most common, severe, spontaneous and often continuous. Hyperaesthesia (an abnormal sensitivity to touch, pain or other sensory stimuli) may occur on the side of the body opposite the lesion in the thalamus. The perception of body position and movement may also be lost.

Cancer (palliative) pain

Cancer (palliative) pain is a common condition of those with advanced cancer. Cancer pain is often persistent, arising from a number of factors (e.g. the disease process, the prescribed treatment, resultant disability and subsequent co-morbidity problems). Cancer pain can be a challenge to manage as curative opportunities have been ceased and death is inevitable. Cancer pain is often a mixture of nociceptive and neuropathic, having acute and persistent features with problems of breakthrough and incident pain.

FACTORS AFFECTING RESPONSES TO PAIN

Physical response to pain involves specific and often predictable neurological changes. In fact, everyone has the same pain *threshold* and perceives pain stimuli at the same stimulus intensity. For example, heat is perceived as painful at 44ºC to 46ºC, the range at which it begins to damage tissue. What varies is *tolerance*, which is based on perception of, and reaction to, pain. When a person is described as highly sensitive to pain, this is a reference to their **pain tolerance**, which is the amount of pain a person can endure before outwardly responding to it. The ability to tolerate pain may be decreased by repeated episodes of pain, fatigue, anger, anxiety and sleep deprivation. The use of such practices such as heat/cold, position, relaxation, distraction, hypnosis, spiritual practices, medications and alcohol may increase pain tolerance. The individualised response to pain is shaped by multiple and interacting factors, including sociocultural influences, emotional status, past experiences with pain, the source and meaning of the pain, the person's knowledge base and their age.

Sociocultural influences

The findings of research so far suggest that cultural influences should be an important consideration when assessing and treating pain. A person's response to pain is strongly influenced by family, community and culture. Sociocultural influences affect the way in which a person tolerates pain, interprets the meaning of pain, and reacts verbally and non-verbally to the pain. For example, if a person has a cultural background where males should not cry and must tolerate pain stoically, rather than expressing his discomfort a male person may appear withdrawn and refuse pain medication. On the other hand, when open and intense emotional expression is culturally acceptable, a person may demonstrate their discomfort clearly and be comfortable requesting pain medication.

Cultural standards also influence an individual regarding how much pain to tolerate, what types of pain to report, who to report the pain to, and what kind of treatment to seek. For example, a person from a Western culture may value 'being a good patient', which may cause them to avoid 'complaining' about their pain, whereas a person from another culture may value seeking information about pain, which may cause them to discuss their pain often and in detail. However, behaviours vary greatly within a culture and from generation to generation. The nurse should approach each person as an individual, observing them carefully, taking the time to ask questions, and avoiding making assumptions.

The nurse also brings with them a set of personal sociocultural values, beliefs and experiences about pain. If these values, beliefs and experiences differ from those of the person, the assessment and management of pain may be based on the values of the nurse rather than on the needs of the person. It is not uncommon to hear staff compare their own personal experiences of pain with the person receiving care (e.g. 'When I had my appendix removed I had two paracetamol and I was fine; this person doesn't need a PCA [patient-controlled analgesia] machine.').

Psychological status

The person's psychological status influences the perception of pain. Remember the IASP's definition of pain as a *sensory and emotional experience*; therefore, how an individual feels psychologically and emotionally will influence their response to pain. Pain sensations may be blocked by intense concentration (e.g. during sports activities) or may be increased by anxiety or fear. Pain is often increased when it occurs in conjunction with other illnesses or physical discomforts such as nausea or vomiting. The presence or absence of support people or caregivers who genuinely care about pain management may alter emotional status and the perception of pain.

Anxiety and depression may increase the perception of pain, and pain may in turn cause anxiety. Depression is clearly linked to pain and is generally associated with higher pain intensity (ANZCA, 2010). In addition, the muscle tension commonly found with anxiety can create its own source of pain. This association explains why non-pharmacological interventions such as relaxation or guided imagery are helpful in relieving or decreasing pain. Fatigue, lack of sleep and depression also are related to pain experiences. Pain interferes with a person's ability to fall asleep and stay asleep, and thus induces fatigue. In turn, fatigue can lower pain tolerance.

Past experiences with pain

Previous experiences with pain are likely to influence the person's response to a current pain episode. If supportive adults responded to childhood experiences with pain appropriately, the adult usually will have a healthy attitude to pain. If, however, the person's pain was responded to with exaggerated emotions or neglectful indifference, that person's future responses to pain may be exaggerated or denied.

The responses of healthcare providers to the person in pain can influence their response during the next pain episode. If providers respond to pain with effective strategies and a caring attitude, the person will remain more comfortable during any subsequent pain episode, and anxiety will be avoided. If, however, the pain is not adequately relieved, or if the person feels that empathetic care was not given, anxiety about the next pain episode sets up the person for a more complex and therefore more painful event.

Source and meaning

The meaning associated with the pain influences the experience of pain. For example, the pain of labour to deliver a baby is experienced differently from the pain following removal of a major organ for cancer. Because pain is the major signal for health problems, it is strongly linked to all associated meanings of health problems, such as disability, loss of role and death. For this reason, it is important to explain the aetiology and prognosis of pain to the person receiving care.

Knowledge

A lack of understanding of the source, outcome and meaning of pain can contribute negatively to the pain experience. The nurse needs to assess the person's readiness to learn, use methods of teaching that are effective for the person and family, and evaluate learning carefully. Teaching must include the process of the pain, its predictable course (if possible), and the proposed plan of care. In addition, nurses should discuss strategies for managing pain and encourage the person to communicate preferences for pain relief. Involving the patient's significant others regarding the presence of pain and how they can help promote effective relief assist in a holistic approach to pain management.

Age

Age influences a person's perception and expression of pain, and of the physical changes that will influence drug requirements (see Table 8.1). Dangerous misconceptions exist regarding the management of pain in older adults.

TABLE 8.1 Physical changes related to ageing and their influence on drug requirements

PHYSICAL CHANGE	EFFECT	OUTCOME
Cardiac output	Drug concentrations after bolus	Require smaller bolus dose
Muscle mass	Change in drug distribution	Potential for unwanted side effects
Liver function	Influences drug elimination	Drug accumulation
Renal function	Influences drug excretion	Drug accumulation
Cerebral blood flow	Alters response to analgesia	Unwanted and unexpected side effects can occur

Sources: Australian and New Zealand College of Anaesthetists and Faculty of Pain Medicine (2010). *Acute pain management: Scientific evidence* (3rd ed.). Melbourne: Australian and New Zealand College of Anaesthetists; Prowse, M. (2005). Postoperative pain in older people: A review of the literature. *Journal of Clinical Nursing, 16,* 84–97.

Misconceptions regarding ageing

Denny and Guido (2012) suggest there are several myths about the older adult and the process of ageing.

1. *Pain is expected in the older adult.* While the occurrence of pain is common in the older adult, it is often not recognised, or is undertreated (Tracy & Morrison, 2013); however, it is still an indicator of an underlying clinical issue. Often believing that pain is a part of growing older, the person may ignore pain or self-medicate with over-the-counter medications. Individuals in this age group may fail to acknowledge pain, believing that it is inevitable or fearing dependency if they alarm their loved ones (Gammons & Caswell, 2014).
2. *Pain perception decreases with age.* Age does not change the perception of acute pain. There is no evidence that nociception is altered by age. Stotts et al. (2007) investigated procedural pain in the hospitalised adult, and found that the younger group (18–64 years) and the older group (65+ years) experienced the same amount of procedural pain during wound care, wound drain removal, tracheal suctioning, turning, femoral sheath removal and central line insertion. However, it was interesting to note that the procedural *distress* was greater in the younger than the older patient (Stotts et al., 2007).
3. *When older adults report pain, they are attention seeking.* This is highly unlikely. Do not forget that some older people suffer mute myocardial infarction and experience painless peritonitis, pancreatitis and cholecystitis (Tracy & Morrison, 2013). The elderly are at a greater risk of mortality and morbidity if pain is not relieved.
4. *Opiates are dangerous in older adults.* When opiates are prescribed the factors identified in Table 8.1 need to be considered.
5. *Opiate use causes addiction in older adults.* Older adults may hesitate to ask for pain medicine because they fear addiction and loss of independence (Brown et al., 2015).

FAST FACTS

- Older people have the highest rate of illness and surgical procedures associated with pain; they also have the highest rate of complications associated with surgical interventions.
- Persistent pain is common in older adults. For those over 70 years of age, 50% of those living in the community and 80% of those in residential care suffer persistent pain.
- Musculoskeletal pain affecting major joints and back, or neuropathic pain from diabetic neuropathy and post-herpetic neuralgia have an increased prevalence in the ageing population.
- Concurrent illnesses are common in the elderly, making clinical presentation complex and sometimes difficult.
- Age and cognitive impairment enhance the risk of poor pain control which will influence the individual's quality of life.

Sources: Australian and New Zealand College of Anaesthetists and Faculty of Pain Medicine (2010). *Acute pain management: Scientific evidence* (3rd ed.). Melbourne: Australian and New Zealand College of Anaesthetists; Prowse, M. (2005). Postoperative pain in older people: A review of the literature. *Journal of Clinical Nursing*, *16*, 84–97.

Pain assessment in older adults may be difficult when the person is cognitively impaired and has difficulty communicating descriptions of pain. When discussing pain with the elderly person it is often better to use the terms they use when self-reporting pain, such as 'aching' or 'soreness'. However, some adults with cognitive impairments are able to describe current, usual or worse pain when a standardised pain scale is used, such as the verbal descriptor of 'mild, moderate or severe'.

A study of pain and associated behavioural and psychiatric symptoms of 2282 aged care residents was undertaken by Tosato et al. (2012). The study showed pain resulted in significant and positively associated socially inappropriate behaviours such as wandering, resistance to care, abnormal thought processes and delusions. Treatment of pain needs to be an integral component of behavioural management in the aged care resident with dementia. Predictive pain tools such as the Abbey scale need to be utilised and others developed to accurately record the pain of the non-verbal person (Ersek, Polissar & Neradilek, 2011; Liu, Briggs & Closs, 2010).

Delirium in the person who is acutely ill or has dementia (occurring in approximately 30% of adults over the age of 85) is a barrier to assessing pain (Alzheimer's Australia, 2015). Recommended methods for pain assessment include: (1) behavioural observations for which no tool has been validated; (2) documenting baseline behaviours and activity patterns and monitoring changes that might indicate the need for further pain assessment; and (3) conducting an empirical analgesia trial.

Pain in the paediatric population

'Children must be assumed to experience pain from birth onwards' (American Medical Association, 2010). In the past, infants and young children have been assumed to have a lack of pain sensation; however, it is now known that due to a more robust inflammatory response and lack of central inhibitory influence, their response to pain may be greater than that of adults.

Olmstead, Scott and Austin (2010) assert pain is often undermanaged in children due to:

- lack of assessment and reassessment of pain
- misunderstanding of how to quantify a subjective experience
- lack of knowledge in pain treatment
- the notion that addressing pain in children takes too much time
- fear of adverse effects of analgesic medications, including respiratory depression and addiction.

It is recommended that health professionals should anticipate painful experiences and monitor the condition of the child, and provide adequate information about what to expect and appropriate measures to reduce the distress to children and parents (Kozlowski & Monitto, 2013).

Optimal pain control can be achieved using a range of techniques. These may range from deep sedation or anaesthesia to cognitive behavioural strategies such as imagery and relaxation (see page 165). Whatever strategy is used, a quiet environment with calm parents and clear and confident instruction will assist the paediatric person experiencing pain (Truba & Hoyle, 2014).

Pain and cultural diversity

Research into pain and cultural diversity is needed to produce and refine multilingual pain-scoring charts. The nurse must try to be familiar with ethnic and cultural diversity in pain expression and management, and respect cultural differences. Pain behaviours are not an objective indicator of the amount of pain present for any individual person. An accurate history is needed and the use of a professional interpreter rather than family members is preferable to ensure accurate translation.

Patient-controlled analgesia devices may be used to provide efficient and effective analgesia (ANZCA, 2010). Nurses may underestimate the pain of those from a different cultural background or having a different language. In an American study, Asian patients received 24% lower doses of analgesics postoperatively than Caucasian people; however, both groups used similar amounts of opioid analgesia when using a PCA (Brown et al., 2015). A thorough explanation of the PCA is needed, through an interpreter, to ensure this concept is understood and this form of analgesia is utilised.

Pain and Indigenous Australians

Significant disparities exist between the life expectancy at birth of the general Australian population and that of Aboriginal and Torres Strait Islander Australians.

> *In 2005–2007, life expectancy at birth for Aboriginal and Torres Strait Islander males was 67.2 years, 11.5 years less than that for non-Indigenous males (78.7 years). For Aboriginal and Torres Strait Islander females, life expectancy at birth was 9.7 years less than for non-Indigenous females (72.9 years and 82.6 years respectively). The lower life expectancy for Aboriginal and Torres Strait Islander Australians can be attributed to a higher infant mortality rate, and a higher incidence of diseases such as diabetes mellitus, respiratory disorders, ear disease, eye disorders and some cancers, among Aboriginal and Torres Strait Islander peoples.* (Australian Bureau of Statistics, 2010)

Indigenous Australians are therefore considered to be a high-risk group in relation to healthcare. Variations in pain behaviours, verbal and non-verbal communication, and the accuracy of standard pain assessment tools have been identified as specific challenges (Fenwick, 2006). A culturally safe assessment of the person is required, with sensitivity to pain tolerance, language barriers and tendencies to display a stoic attitude to pain demonstrated. Where possible, nurses should use interpreters, Indigenous health workers or liaison officers. Note that, like other high-risk patient groups, Indigenous Australians often have multiple co-morbidities, which may influence the types of analgesics that may be prescribed.

MYTHS AND MISCONCEPTIONS ABOUT PAIN

Myths and misconceptions about pain and its management are common in both healthcare providers and the general population. Some of the most common misconceptions are:

- *Pain is a result, not a cause.* According to the traditional view of pain, it is only a symptom of a condition. However, it is now recognised that unrelieved or poorly relieved pain sets up further responses such as central sensitisation, amplifying pain sensations and delaying rehabilitation.
- *Persistent pain is really a masked form of depression.* Serotonin plays a chemical role in pain transmission and is also the major modulator of depression. Therefore, pain and depression are chemically related, not mutually exclusive. It is common to find them coexisting.
- *Opioid medication is too risky to be used to treat persistent pain.* This common misconception often deprives the person experiencing pain of the most effective source of pain relief. It is true that other methods should be tried first; however, if they prove ineffective, opioids should be considered as an appropriate alternative.
- *It is best to wait until the person has pain before giving medication.* It is now widely accepted that anticipating pain has a noticeable effect on the amount of pain a person experiences. Offering pain relief before a pain event is well on its way can lessen the pain. Remember that agony is harder to manage.
- *Many people lie about the existence or severity of their pain.* The most reliable source of pain severity is the person's score, as they will rarely lie about their pain.
- *Postoperative pain is best treated with intramuscular injections.* The most commonly used postoperative pain relief for many years was morphine or pethidine given intramuscularly. However, both have adverse effects, such as late-onset respiratory depression, they are painful to give and they can irritate the tissues (causing tissue abscess). Pethidine is short acting and also produces norpethidine, a CNS stimulant that can cause seizures. Most experts now do not recommend pethidine to manage postoperative pain (Cohen & Schecter, 2005).
- *Pain relief interferes with diagnosis*. Pain is the single most common reason that people present to an emergency department (ED).
- A common misconception is that analgesia given prior to medical assessment will mask the pathology and therefore diagnosis. In the case of abdominal pain, research shows that pain relief will not interfere with the diagnostic process in adults and children (ANZCA, 2010). Despite a prevailing attitude that pain management is an essential part of quality medical care, pain management in the ED is difficult because of the short-term associations with the person, increased vigilance against drug abuse and the myth that diagnosis is impaired by pain relief. Nevertheless, 60% of people presenting to Australian EDs with abdominal pain are satisfied with their analgesia on discharge (ANZCA, 2010).

INTERPROFESSIONAL CARE AND PHARMACOLOGICAL/NON-PHARMACOLOGICAL TREATMENT ALTERNATIVES

Effective analgesia relief results from collaboration among healthcare providers, particularly nurses, as it is the nursing staff that most closely cares for the person 24 hours a day, especially in an acute situation. For those with more persistent pain problems, there are pain clinics staffed by teams of healthcare professionals who use a multidisciplinary approach to manage persistent pain. Therapies may include traditional pharmacological agents as well

as psychotherapy, biofeedback, hypnosis, acupuncture, massage and other treatments. Hospices for palliation provide a multifaceted approach to pain management (see Chapter 4).

Non-pharmacological strategies to manage pain

KNOWLEDGE AND INFORMATION Knowledge and information will assist the person in managing their pain. Give direct, clear, concise information that the person understands, but do not make them more anxious and distressed. A clear, concise plan of action that has been discussed with the person will assist in effective pain management.

RELAXATION Relaxation involves learning activities that deeply relax the body and mind. Relaxation distracts the person, lessens the effects of stress from pain, increases pain tolerance, increases the effectiveness of other analgesic measures and increases perception of pain control. In addition, by teaching the person relaxation techniques, the nurse acknowledges the person's pain and provides reassurance that the person will receive help in managing the pain (Rejeh et al., 2013). Examples of relaxation activities include:

- *Diaphragmatic breathing* can relax muscles, improve oxygen levels and provide a feeling of release from tension. The technique for diaphragmatic breathing is described and illustrated in Chapter 3.
- *Progressive muscle relaxation* may be used alone or in conjunction with deep breathing to help manage pain. The person should be taught to tighten one group of muscles (such as those of the face), hold the tension for a few seconds, and then relax the muscle group completely. The person should repeat these actions for all parts of the body.
- *Guided imagery*, also called *creative visualisation*, is the use of the imaginative power of the mind to create a scene or sensory experience that relaxes the muscles and moves the attention of the mind away from the pain experience. To use guided imagery, the person must be able to concentrate, use their imagination and follow directions. The nurse can facilitate this technique by asking the person for some descriptions of what they find most relaxing. The nurse then speaks to the person in a calming, soothing voice about those places or situations. Audio recordings are available to assist with guided imagery.
- *Meditation* is a process whereby the person empties the mind of all sensory data and, typically, concentrates on a single object, word or idea. This activity produces a deeply relaxed state in which oxygen consumption decreases, muscles relax and endorphins are produced. At its deepest level, the meditative state may resemble a trance. Many books and CD/DVD recordings are available commercially.

DISTRACTION Distraction involves the redirection of the person's attention away from the pain and on to something that the person finds more pleasant. Examples of distracting activities are practising focused breathing, and listening to or doing some form of rhythmic activity to music. For example, the person using recorded music for distraction may sing along with the song, or tap out the rhythm with their fingers or foot. Full participation in the music is key to analgesia.

Distraction may also involve the individual participating in an activity that promotes pleasure and stimulates laughter, as laughing for 20 minutes or more is known to produce an increase in endorphins that may continue to give pain relief even after the person stops laughing.

BIOFEEDBACK Biofeedback is an electronic method of measuring physiological responses, such as brain waves, muscle contraction and skin temperature, and then 'feeding' this information back to the person. Most biofeedback units consist of electrodes placed on the skin and an amplification unit that transforms data into visual cues, such as coloured lights. The person thus learns to recognise stress-related responses and to replace them with relaxation responses. Eventually, the person learns to repeat independently those actions that produce the desired brain wave effect.

Relaxation helps avoid the anxiety that often accompanies and complicates pain. Additionally, biofeedback gives the person a measure of control over the response to pain.

HYPNOSIS Hypnosis is a trance state in which the mind becomes extremely suggestible. To achieve hypnosis, the person sits or lies down in a dimly lit, quiet room. The therapist suggests that the person relax and fix attention on an object. The therapist then repeats, in a calming, soothing voice, simple phrases such as instructions to relax and listen to the therapist's voice. Eventually the person hears only the therapist's voice; during this state, the therapist may then make suggestions to encourage pain relief.

Manual therapies

PHYSIOTHERAPY Physiotherapy can offer a range of pain management strategies—for example, graded reactivation programs, therapeutic ultrasound and exercises to increase strength and flexibility to promote pre-injury functional status.

MASSAGE Massage has been shown to assist in post-surgical abdominal pain and thoracic surgery. Pain scores are not necessarily lowered, but there is a reduction in the unpleasantness of pain (ANZCA, 2010).

HEAT AND COLD THERAPY Heat and cold therapy are commonly used as strategies to relieve pain; however, evidence regarding their effectiveness is inconsistent.

ACUPUNCTURE Acupuncture is an ancient Chinese system involving the stimulation of certain specific points on the body to enhance the flow of vital energy (chi) along pathways called meridians. Acupuncture points can be stimulated by the insertion and withdrawing of needles, the application of heat, massage, laser, electrical stimulation or a combination of these methods. Only care providers with special training can use this method. Acupuncture is becoming a more widely accepted therapy, especially for the treatment of pain.

TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION A transcutaneous electrical nerve stimulation (TENS) unit consists of a low-voltage transmitter connected by wires to electrodes placed by the person as directed by the physical therapist (see Figure 8.5). The person experiences a gentle tapping or vibrating sensation over the electrodes. The person can adjust the voltage to achieve maximum pain relief. It is believed that TENS electrodes stimulate the large-diameter A-delta fibres activating inhibitory networks in the dorsal horn, which reduces the nociceptive transmission of the C fibres.

FIGURE 8.5 ■ The TENS unit is believed to assist in pain management in the ways described in the gate-control theory. Electrodes that deliver low-voltage electrical stimuli are placed directly on the person over painful areas

Source: © Rob Byron/Shutterstock.com.

A TENS unit is most commonly used to relieve persistent benign pain. Thorough education is essential, including an explanation of the manufacturer's directions, instructions on where to place the electrodes and the importance of placing the electrodes on clean, unbroken skin. The clinical use of TENS has been extensively studied (Johnson, 2014). TENS offers several advantages: avoidance of drug side effects, person control and good interaction with other therapies. Disadvantages are its cost and the need for initial expert training. When choosing a TENS machine make sure it is portable, has adjustable amplitude, can be managed by the user and has built-in output short-circuit protection. TENS should not be used by people with cardiac pacemakers and implanted defibrillators.

HYPERBARIC OXYGEN THERAPY This therapy is defined as the intermittent inhalation of 100% oxygen in a hyperbaric chamber at a pressure higher than 1 absolute atmosphere (1 ATA = 760 mmHg, the normal atmospheric pressure at sea level) (Yildez, Uzun & Kiralp, 2006).

This method is increasingly being tried and researched to manage neuropathic pain as well as headaches, cancer pain and postoperative pain. Initial animal studies relating to inflammation pain have been positive with hyperbaric oxygen therapy successfully relieving neuropathic pain for an extended period (Thompson et al., 2010).

Pharmacological strategies for managing pain

MEDICATION Medications are the most common approach to pain management. A variety of drugs with many kinds of delivery systems are available. These drugs include simple analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), opioids, antidepressants and local anaesthetic agents. In addition to administering the prescribed medications, the nurse may act independently in choosing the dosage and timing. The nurse is also responsible for assessing the side effects of the medications, evaluating their effectiveness, and providing education. The nurse's roles in pain relief are those of advocate, educator and direct caregiver.

The World Health Organization (WHO) 'ladder of analgesia' effectively guides the use of medications (WHO, 1986/1990) (see Figure 8.6). Analgesics are used progressively until pain is reduced or relieved, reflecting the interactive nature of these types of medications. Initially a simple analgesic (e.g. paracetamol) is used. A person will then progress to NSAIDs and then

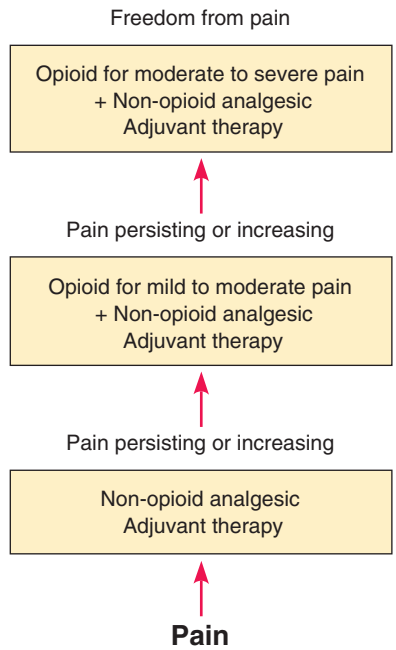


FIGURE 8.6 ■ The WHO analgesic ladder illustrates the process for selection of analgesic medications for pain management

Source: World Health Organization (1990). *Cancer pain relief and palliative care* (Technical Report Series, no. 804). Geneva: WHO. Reprinted by permission.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 4:
Medication Safety

'This standard is concerned with the systems and strategies to ensure clinicians safely prescribe, dispense and administer appropriate medicines to informed patients.' (Australian Commission on Safety and Quality in Healthcare (ACSQHC), 2011, p. 3)

Medication safety is supported by a range of initiatives across the interprofessional teams, including the implementation of a National Inpatient Medication Chart (NIMC), safe labelling practice, quality education and training, and accurate reconciliation and administration of medications (ACSQHC, 2011). Nurses play an integral role in the safe administration of medicines to an informed patient.

Source: © Australian Commission on Safety and Quality in Health Care.

BOX 8.1 Terms associated with pain medications (usually opioids)

- **Addiction**: a disease characterised by aberrant drug-seeking behaviour that includes compulsive use of a substance, cravings and loss of control despite the negative biopsychosocial consequences.
- *Substance abuse disorder*: the use of any chemical substance for other than a medical purpose that causes a disintegration of the person and their life commitments.
- *Physical drug dependence*: a physiological need for a substance, that results in physical withdrawal symptoms if it is not supplied or the amount is reduced suddenly. Common withdrawal symptoms include agitation, insomnia, yawning, tachycardia, sneezing and diarrhoea.
- *Psychological drug dependence*: a psychological need for a substance. If the substance is not supplied, psychological withdrawal symptoms occur, including anxiety and irritability.
- *Drug tolerance*: the process by which the body requires a progressively greater amount of a drug to achieve the same results.
- *Equianalgesic*: having the same analgesic effect when administered to the same individual. Drug dosages are equianalgesic if they have the same effect as morphine sulfate 10 mg administered parenterally.
- *Pseudoaddiction*: behaviours resembling drug seeking; often as a result of receiving inadequate analgesia.

to opioid medications. Box 8.1 describes terms associated with pain medication.

Simple analgesics Simple **analgesics** such as paracetamol produce analgesia and reduce fever. The exact mechanism of action is uncertain, but it is used to treat mild to moderate pain. It is absorbed rapidly in the small intestine after oral administration, having an effect in approximately 30 minutes. Paracetamol can be given rectally, though absorption is erratic; there is also an intravenous preparation giving analgesia in approximately 10 minutes. Paracetamol should be given with caution to children, and those who are underweight or have liver or renal dysfunction.

NSAIDs NSAIDs (non-steroidal anti-inflammatory drugs) act on peripheral nerve endings and minimise pain by interfering with prostaglandin synthesis. These medications provide analgesic effects by reducing inflammation and by perhaps blocking the generation of noxious impulses. Examples include aspirin, diclofenac and ibuprofen. The NSAIDs have anti-inflammatory, analgesic and antipyretic actions. NSAIDs are useful (providing renal function is adequate) for mild to moderate pain and continue to be effective when combined with opioids for moderate to severe pain. The cyclooxygenase-2 (COX-2) selective NSAIDs (celecoxib, parecoxib) are newer formulations; although less harmful to gastric mucosa, their usage has been questioned because of cardiac-associated adverse reactions and care must always be taken when prescribing regarding the person's renal function.

NSAIDs have minimal side effects if used on a short-term basis, but side effects can and do occur when used over a long period for persistent pain problems.

Tramadol Tramadol is a centrally acting synthetic analgesic that is used for moderate to severe pain. Tramadol has a three-way action, having some opioid activity and also inhibiting the re-uptake of noradrenaline and serotonin. Because it is not completely an opioid, tramadol has been classed as a Schedule 4 medication. Its analgesic effect is comparable to that of codeine. It can be used for moderate to severe pain, though it is not as powerful as morphine. Tramadol is contraindicated in those with epilepsy, as seizures have been reported, but it causes less respiratory depression, gastric stasis and constipation than the opioids (Martinez, Guichard & Fletcher, 2015). Tramadol is available in oral (immediate and slow release) and intravenous preparations.

Opioids Opioids are derivatives of the opium plant. ('Opioid' is the preferred term as 'narcotic' has negative connotations.) These medications (and their synthetic forms) are the pharmacological treatment of choice for moderate to severe pain. Examples are morphine, codeine and fentanyl. Opioid analgesics produce analgesia by binding to opioid receptors both within and outside the CNS. A common myth among healthcare professionals is that using opioids for analgesia poses a real threat of addiction. When opioids are used as recommended, there is little to no risk of addiction (see Box 8.2). Nursing implications for opioids are found in the 'Medication administration' box below.

A summary of the common opioids, their preparations and the brand names available in Australia is shown in Table 8.2, and general adverse effects of opioids are listed in Box 8.3.

ADJUVANTS There are a number of drugs that are often used in pain management that assist with analgesia or control specific symptoms. These drugs are often referred to as the adjuvants or co-analgesics.

Antidepressants Antidepressants within the tricyclic chemical group act on the production and retention of serotonin in the CNS, thus inhibiting pain sensation. They also promote normal sleeping patterns, further alleviating the suffering of the person in pain. They are useful with neuropathic pain. Common drugs used in this class are amitriptyline, nortriptyline and doxepin.

Anticonvulsant medications Anticonvulsant medications such as gabapentin (Neurontin), pregabalin (Lyrica) and carbamazepine (Tegretol) are useful with neuropathic pain, including phantom limb pain, shingles (herpes zoster), migraine headaches and diabetic neuropathic pain. These drugs reduce pain and sleep disruption. Although these drugs are primarily used to treat epilepsy (seizures), they are also used to treat nerve pain conditions. Clinical studies have shown many anticonvulsant drugs to be effective.

Ketamine Ketamine is a dissociative anaesthetic and has the action of being an NMDA (N-methyl-D-asparate) antagonist. Tissue damage that causes continual nociception or neuropathic

BOX 8.2 Pain management and drug abuse history

The person with a substance abuse disorder often experiences sub-therapeutic dosing of opioid medications for pain. When providers suspect or learn of drug abuse, they tend to order lower doses than they would for a person of similar age and weight. Despite significant data showing very little addiction as the result of treating pain with adequate pain relief, prescribers still tend to under-treat pain in the person with a substance abuse disorder. When the person with a substance abuse disorder has an acute injury they usually need greater doses of pain medication because of the tolerance they have developed from repeated exposure to opioids and other drugs. The person with chronic cancer or non-cancer pain can display opioid tolerance due to perioperative opioid administration, particularly opioids of high potency (ANZCA, 2010).

Cook, Sefcik & Stetina (2004) reported on a study comparing analgesic prescribing practices for those in acute pain with a history of drug abuse with those in acute pain who had not abused drugs. It was found that prescribing practices were significantly different for the two groups. All were prescribed the same selection of medication and dosages; however, those with a history of drug abuse were prescribed significantly more non-opioid analgesics. Furthermore, there was no significant difference in the dosages prescribed for the two groups, despite the risk of greater tolerance to opiates by the heroin-addicted person. The prescribers did not take into account heroin-dependent tolerance to opioid medications and the increased need for analgesia among those with a history of drug abuse. It was also pointed out that opioid analgesics were rationed in an effort to wean the person from their addiction. This indicates how uninformed the prescribers were regarding the needs of a heroin-dependent person and their increased tolerance to opioids. The findings of this study have been supported by more recent research by Blay et al. (2012) who looked at pain management practices with patients who had a history of substance abuse, in the Australian context.

During the acute stress of injury or infection, withholding pain medication is an added stressor. Nurses attempting to advocate for the comfort needs of an addicted person may encounter resistance around this issue. This creates a potential ethical as well as a professional dilemma. Therefore, where possible, use a pain service to assist in a person's analgesic management.

Providing analgesics for a person with pain who has a history of or ongoing substance abuse is challenging. It is important to communicate clearly all information about medications and accessibility to the providers. Giving appropriate analgesic relief will help prevent the person sourcing medications illegally. Dose escalation may be monitored with careful assessment and random urine screens if requested by medical staff.

When the person is discharged from hospital care, consideration must be taken to exposing the community to opioid medications. If diversion of the drug or inappropriate use of the prescribed medication is suspected, then opioids can be prescribed with restrictions (e.g. collecting limited amounts at one time, or only after review by a medical practitioner). Detoxification is a matter for the person to determine when they are over their crisis.

TABLE 8.2 Common opioids available in Australia

OPIOID	PREPARATION	BRAND NAME
Morphine	Oral syrup (immediate release)	Ordine
	Oral tablet (immediate release)	Anamorph/Severdol
	Oral tablet/capsule (sustained release)	MSContin/Kapanol
	Injection (immediate release)	Morphine sulfate/Tartrate
Oxycodone	Oral tablet/capsule/elixir (immediate release)	Endone, Oxycodone, OxyNorm
		Oxycontin
	Oral tablet (sustained release)	Prolodone
	Suppository (absorption uncertain)	Oxycodone
	Injection (immediate release)	
Fentanyl	Lozenge (immediate release)	Atiq
	Transdermal patch (sustained release)	Durogesic
	Injection (immediate release)	Sublimaze
Hydromorphone	Oral liquid/tablet/injection (immediate release)	Dilaudid
	Oral (sustained release)	Junista
Methadone (absorption independently variable)	Oral syrup	Physeptone syrup/Biodone Forte
	Oral tablets	Physeptone
	Injection	Physeptone
Codeine	Oral tablet/linctus (immediate release)	Codeine phosphate
Pethidine	Injection (immediate release)	Pethidine

BOX 8.3 General adverse effects of opioids

Neurological symptoms are often dose-dependent, and may include sedation, dysphoria, confusion, dizziness, mental cloudiness, euphoria, miosis, muscle rigidity and seizures. It is usually advised that people do not drive, work heavy machinery or make critical decisions when initially taking these medications.

Respiratory symptoms are often dose-related, and may include cough suppression, respiratory depression, bronchospasm and asthma.

Cardiovascular symptoms may include bradycardia, hypotension and vasodilation.

Gastrointestinal symptoms may include nausea, vomiting, constipation, loss of appetite and biliary colic.

Dermatological symptoms may include itch, sweating, flushing and rash.

Urinary symptoms may include urinary retention, and changes in bladder and sphincter tone.

Neuroendocrine symptoms tend to develop after long-term use.They include a reduction in some hypothalamic-releasing hormones, which affects the gonads, adrenal cortex and endorphins.

Opioid rotation is common, especially for persistent pain as different opioids act on different receptors. Therefore, often a specialist will rotate the person on to a different opioid in the hope of producing more effective pain relief at a reduced dose. Sometimes equianalgesic tables are used to aid this rotation process, but these must be used with caution as each individual has different tolerances and preferences.

pain activates NMDA which subsequently produces sensitisation of the central nervous system. Low-dose ketamine calms down this NMDA reaction, and is used clinically for the person with persistent pain, opioid tolerance, substance abuse issues or a neuropathic pain state.

Nitrous oxide Nitrous oxide is useful for women in labour and for some dressings and procedures that are painful. Despite its analgesic and sedative effects, it has minimal cardiovascular or respiratory depressive effects. Neurological and bone marrow problems can develop with regular use, so relevant system testing should occur at regular intervals. To help prevent neurological problems, methionine, folic or folinic acid, and vitamin B12 should be prescribed (ANZCA, 2010).

Local anaesthetics Local anaesthetics block the initiation and transmission of nerve impulses in a local area, thus also blocking pain sensations. Common examples of these drugs are lignocaine, bupivacaine and ropivacaine.

Local anaesthetics can be delivered by a variety of methods—for example, a single-shot nerve injection, as wound infiltration, or via topical application. All have been shown to aid analgesia. Delivery can also be made directly to the sheath of a nerve through a peripheral nerve catheter offering a continuous nerve blockade when the catheter is connected to an ongoing local anaesthetic infusion. When this latter method is used, staff must be competent in the delivery system and the hospital should have standard protocols and policies to manage this technique. Side effects can occur from receiving local anaesthetics, such as trauma to the anaesthetised area and local anaesthetic toxicity from accidental intravascular injection.

Bisphosphonates Bisphosphonates are medications that target malignant tumours growing in bone. Referred to as osseous metastases, these expanding, painful tumours impair function. Bisphosphonates stabilise bone, slowing or preventing the development of tumours, and have a pain-relieving benefit which exceeds that of steroids and NSAIDs. Disodium pamidronate (Aredia) is effective with breast cancer metastases and multiple myeloma. A newer generation bisphosphonate, zoledronic acid (Aclasta), is useful with bony metastases secondary to lung, prostate, renal cell and other solid tumours (Mathew & Brufsky, 2015).

Radiopharmaceuticals Radiopharmaceuticals are unsealed substances produced in a nuclear reactor that emit a beta particle or an electron. The radioactivity is particularly damaging to malignant cells and is a safe and effective treatment for bony metastases. Radiopharmaceuticals are particularly useful in the management of those with prostate cancer and painful osteoblastic metastases confirmed on bone scan. This treatment coupled with external beam radiation therapy (EBRT) has been shown to provide symptomatic relief in 80% of this group (Goyal & Antonarakis, 2012). Negative effects may include severe renal dysfunction or severe bone marrow depression.

Treatments may be given intravenously or orally. Care of the person treated with radiopharmaceuticals must follow radiation precautions, disposing of all body fluids quickly and thoroughly. Urine excretion is essentially completed in the first 6 hours.

Duration of action

Each pharmacological agent has a unique absorption and duration of action. Remember that no drug will have a totally predictable course of action, because each person absorbs, metabolises and excretes medications at different dosage levels. The only way to obtain reliable data about the effectiveness of the medication for the individual person is to assess how that person responds. Therefore, the best choice is to individualise the dosing schedule.

There are two major descriptors of dosing schedules. The first type is for medications prescribed ‘on a regular basis’, such as paracetamol, NSAIDs and slow-release opioid preparations. These medications are usually given regularly if the person experiences constant pain predictably during a 24-hour period. The second type of dosing schedule is ‘on an as-needed basis’ (prn)—meaning *pro re nata* (Latin for ‘as circumstances may require’)—whereby immediate-release opioid medications are commonly used. Note that prn medication should be administered:

- as soon as the pain begins or prior to onset
- when increased pain is anticipated, such as when an activity is planned (e.g. a dressing), or when the regular prescribed medication does not cover all the pain. This is often the case in treating cancer pain as breakthrough medications may be required.

Giving analgesics before the pain occurs or increases gives the person confidence in the certainty of pain relief and thereby

MEDICATION ADMINISTRATION Opioid analgesics

Opioid analgesics are used to treat severe pain. The drugs in this category include morphine, codeine, opium derivatives, and synthetic substances. Morphine and codeine are pure chemical substances isolated from opium. These drugs decrease the awareness of the sensation of pain by binding to opiate receptors in the brain and spinal cord. It is also believed that they diminish the transmission of pain impulses by altering cell membrane permeability to sodium and by affecting the release of neurotransmitters for efferent nerves sensitive to noxious stimuli. People can develop a tolerance to opioids and they cause psychological and physical dependence. For the person who has had an acute painful assault and is now requiring slow-release preparations in the short term, ensure that there is a strategy to decrease their opioid consumption. For example, the person is given a written reduction plan to follow, a written reduction regimen is sent to their general practitioner, or the person is sent to a specialised pain medicine clinic to assist with opioid reduction.

NURSING RESPONSIBILITIES

- Opioids are regulated by individual state laws; the nurse and witness must record the date, time, person's name, type and amount of the drug used, and sign the entry in the Schedule 8 book (commonly known as the DD—Dangerous Drugs—Book). A witness must check the drug is given to the correct person. If the drug is disposed of after it is signed out, this must be recorded and witnessed in the Schedule 8 book.
- Keep an opioid antagonist, such as naloxone, immediately available to treat respiratory depression.
- Assess allergies or adverse effects from any opioids previously experienced by the person.
- Pethidine is associated with CNS toxicity and thus involves significant risk; use is discouraged.
- Assess for any respiratory disease, such as asthma or sleep apnoea, that might increase the risk of respiratory depression.
- Assess the characteristics of the pain and the effectiveness of drugs that have been previously used to treat pain.
- Take and record baseline vital signs before administering the drug.
- Administer the drugs following established guidelines.
- Monitor vital signs, level of consciousness, papillary response, nausea, bowel function, urinary function and analgesic effectiveness.
- Use non-invasive methods of pain management and multimodal analgesics in conjunction with opioid medications.
- Provide for person safety.

HEALTH EDUCATION FOR THE PERSON WITH PAIN, THEIR FAMILY AND SIGNIFICANT OTHERS

- The use of opioids to treat severe pain is unlikely to cause addiction.
- Do not drink alcohol.
- Do not take over-the-counter medications unless approved by the healthcare provider.
- Increase intake of fluids and fibre in the diet to prevent constipation.
- The drugs often cause dizziness, drowsiness and impaired thinking; use caution when driving or making critical decisions.

avoids some of the untoward effects of pain. The benefits of a preventive approach can be summarised as follows:

- The person may spend less time in pain.
- Frequent analgesic administration may allow for smaller doses and less analgesic administration.
- Smaller doses will in turn mean fewer side effects.
- The person's fear and anxiety about the return of pain will decrease.
- The person will probably be more physically active and avoid the difficulties caused by immobility.
- The swift and effective management of acute pain can prevent persistent pain states occurring.

The side effects of a drug can become difficult to manage if the dosage is too high, and the person may suffer unnecessary pain because of reluctance to endure side effects. The best formula for adequate dosage is a balance between effective analgesia and minimal side effects. Within prescribed limits, the nurse can choose the most appropriate dose according to the person's response. It is also the role of the nurse to inform the physician and request a review of their analgesics if the prescribed dosage does not meet the person's needs.

Routes of administration

ORAL ROUTE The oral route is the simplest route for both person and nurse. Special nursing care is still required, because some medications must be given with food, some are irritating to the gastrointestinal system, and some people may have trouble swallowing pills. Liquids, elixirs, capsules, soluble preparations and slow-release formulations are available for special applications.

RECTAL ROUTE The rectal route is helpful for those who are unable to swallow; however, absorption is unpredictable. The rectal route is contraindicated in those with diarrhoea, those with neutropenia and those who have had rectal surgery.

TRANSDERMAL MEDICATION The **transdermal** or 'patch' form of medication is increasingly being used because it is simple, painless and delivers a continuous level of medication (see Figure 8.7). Transdermal medications are easy to store and apply. Reapplication depends on the type of patch used (every 72 hours for fentanyl patches or 7 days for buprenorphine). Additional short-acting medication is often needed for breakthrough pain. Over-dosage can occur; therefore, it is important to start with a low-dose patch and **titrate** (which means to increase or decrease the dose in small increments) to the effective level. Again, these patches are good for those who are not eating or drinking; however, for those who sweat profusely or have a fever or inflammation of the skin, expect an increased absorption rate. Exercise and use of electric blankets or heating pads may also accelerate absorption of the medication and cause respiratory depression. (Deaths have occurred in the United States because of this problem.)

FIGURE 8.7 ■ The transdermal patch administers medication in predictable doses

To apply a medication transdermally, apply to a hairless area of skin that is not irritated or broken. The area of skin needs to be cleaned and then the patch applied. Apply the patch immediately upon opening the package, and ensure that the contact is complete, especially around the edges. The effectiveness of a patch is dependent on its absorption, and the next patch should always be applied to a different site. When first applying a transdermal medication, expect 12 to 24 hours (for fentanyl) and up to 3 days (for buprenorphine) until a therapeutic level is absorbed; also, when discontinuing expect a similar decline in level because of the medication reservoir in the skin. If the person complains of severe light-headedness, is pale, sweaty and weak, the medical officer needs to be informed immediately and the patch removed.

INTRAMUSCULAR INJECTION Intramuscular injection was once the most popular route for pain medication administration. Its disadvantages include uneven absorption from the muscle (resulting in delayed respiratory depression), discomfort on administration, and the time needed to prepare and administer the medication. It is also recognised that the quality of analgesia is inferior to that of intravenous patient-controlled analgesia.

INTRAVENOUS ROUTE The intravenous (IV) route provides the most rapid onset, usually ranging from 1 to 15 minutes. Medication can be given by drip, bolus or person-controlled analgesia (PCA)—a pump with a control mechanism that affords the person self-management of pain (see Figure 8.8). The advantages of PCA are dose precision, timeliness and convenience. The person does not have to wait for a nurse to assess the need for pain medication, then procure and deliver the analgesia (Hicks, Hernandez & Wanzer, 2012). Respiratory depression and sedation are minimised when plasma levels of opioids are steady (Lehne, 2012). Several drugs are available for this route. The disadvantages are the nursing care needed for any intravenous line, the potential for infection, the cost of disposable supplies and ensuring that only the actual patient presses the PCA button. For this reason the PCA method of administration requires careful education coupled with close and attentive monitoring.

SUBCUTANEOUS ROUTE The subcutaneous (SC) route is accepted; its disadvantages are similar to those of the intramuscular route.

EPIDURAL ROUTE The epidural route is invasive and requires more extensive nursing care, but may provide better analgesia and postoperative recovery than intravenous delivery. When opioids are inserted into the epidural space they are usually combined with local anaesthetics. This mixture can be given as an infusion or as a 'top-up'. When this combination of drugs is used the person experiences better analgesia, earlier bowel recovery, earlier mobility and a shorter length of hospital stay than with the intravenous route (ANZCA, 2010). See below for nursing implications for the person receiving epidural analgesia.

INTRATHECAL ROUTE The intrathecal route places drugs (usually opioids, local anaesthetics or both) into the cerebrospinal fluid. When medication is inserted into the intrathecal space it is usually as a single injection, and much smaller doses are required compared to the epidural route.

REGIONAL ANALGESIA Regional analgesia is commonly known as 'a nerve block'. Nerve blocks use local anaesthetics (sometimes in combination with a steroidal drug, depending on

FIGURE 8.8 ■ PCA units allow the person to self-manage severe pain. The units may be portable or mounted on intravenous poles

Source: A, © Roy Ramsey/Pearson Education.

the situation) injected by a physician into or near a nerve, usually in an area between the nociceptor and the dorsal root. In the postoperative situation, nerve blocks can be either a single-shot injection or run as a continuous peripheral nerve block infusion (CPNB). When a CPNB is ordered, strict protocols, procedures and standards must be adhered to so as to ensure safety.

In the persistent-pain setting the procedure may be performed to determine the precise location of the source of the pain: pain relief indicates that the injection site is the source of the pain. Temporary nerve blocks may give the person enough relief to:

- develop a more hopeful attitude that pain relief is possible
- allow local procedures to be performed without causing discomfort
- exercise and move the affected part.

If the nerve block is successful, a permanent neurolytic agent can be used to give long-term analgesia.

SURGERY As an analgesic measure, surgery is usually performed only after all other methods have failed. Those who need this measure must understand possible risks such as reduced motor function or incontinences. Surgical procedures used to relieve pain are shown in Figure 8.9 and include the following:

- A *rhizotomy* is destruction of the dorsal spinal roots. It is most often performed to relieve back/neck pain. A rhizotomy can be performed by a spinal or neurosurgeon: the nerve fibres are surgically severed. Rhizotomies may also be performed by a pain specialist who injects a chemical, or uses a radiofrequency current or cryotherapy, to selectively destroy painful fibres. These types of rhizotomy are called percutaneous and usually offer temporary pain relief.
- A *sympathectomy* involves blocking the ganglia of sympathetic nerves, usually in the lumbar region, often with local anaesthetic or surgery. The sympathetic nerves play an important role in producing and transmitting the sensation of pain.
- A *cordotomy* is an incision into the anterolateral tracts of the spinal cord to interrupt the transmission of pain. Because it is difficult to isolate the nerves responsible for upper body pain, this surgery is most often performed for pain in the abdominal region and legs, including severe pain from terminal cancer. A percutaneous cordotomy produces lesions of the anterolateral surface of the spinal cord by means of a radiofrequency current.
- A *neurectomy* is the removal of a nerve. It is sometimes used for pain relief. A peripheral neurectomy is the severing of a nerve at any point distal to the spinal cord.

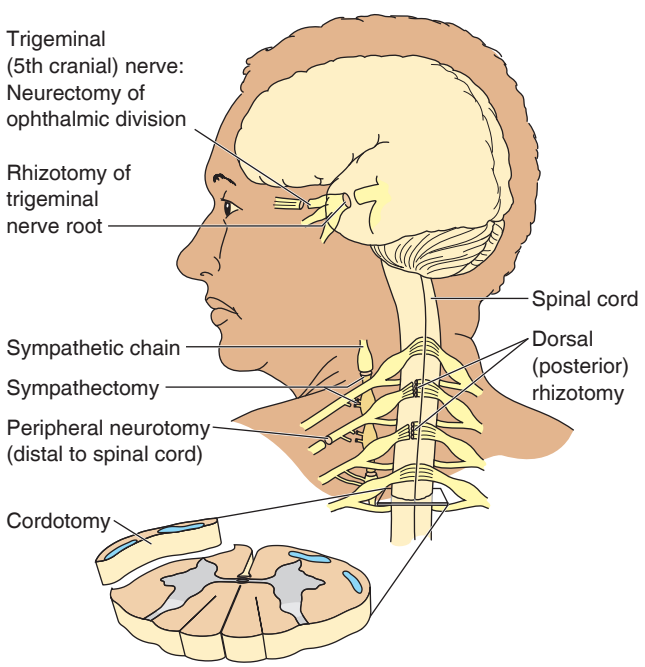


FIGURE 8.9 ■ Surgical procedures are used to treat severe pain that does not respond to other types of management. They include cordotomy, neurectomy, sympathectomy and rhizotomy

Nursing care

Assessment

Nursing assessment of the person with pain presents perhaps more of a challenge than almost any other type of illness or injury because of the subjectivity of pain and the effects it has on the individual.

A comprehensive approach to pain assessment is essential to ensure adequate and appropriate interventions. The five assessment areas are: (1) health history, (2) physiological response, (3) examination, (4) behavioural response, and (5) response to treatment.

1. Health history

Before commencing an in-depth assessment, discuss:

- The definition of the word 'pain' to ensure that the person and the provider are communicating on the same level. It is often helpful to use the person's own words when describing the pain. For example, the person may talk about discomfort as opposed to pain.
- Explain that the report of pain is important for promoting recovery, not just for achieving temporary comfort.
- Ask the person to establish a comfort-function goal. This is a level of pain that does not interfere with or prevent the performance of essential activities of recovery or living.

The person's history of the pain can be assessed by using the PQRST technique (Gregory, 2014):

- What precipitated (triggered, stimulated, makes it worse) the pain? What palliates (what makes it better, has anything relieved) the pain? What is the pattern of the pain?
- What are the quality and quantity of the pain? Is it sharp, stabbing, aching, burning, stinging, deep, crushing, vice-like, gnawing? A reliable indicator of the presence and degree of pain is the person's own statement about the pain. The McGill Pain Questionnaire is a useful tool in assessing the person's subjective experience of the pain. It asks the person to locate the pain, to describe the quality of the pain, to indicate how the pain changes with time and to rate the intensity of the pain (see Figure 8.10). Several tools are available to assess specific pain qualities (Hjermstad et al., 2011; Lin et al., 2011).

NURSING CARE PLAN A person receiving intraspinal analgesia

Intraspinal analgesia is used to manage acute postoperative, persistent and intractable cancer pain. The intraspinal route may be either intrathecal or epidural. Putting opioids into these spaces directly affects the opiate receptors in the dorsal horn of the spinal cord; the opioids are also absorbed systemically and affect the brain. This method provides complete pain relief but has some potentially dangerous side effects.

PROCEDURE

The physician places a needle and/or a catheter into the intrathecal or epidural space. Tubing may be attached to an infusion pump, and the prescribed medication is administered.

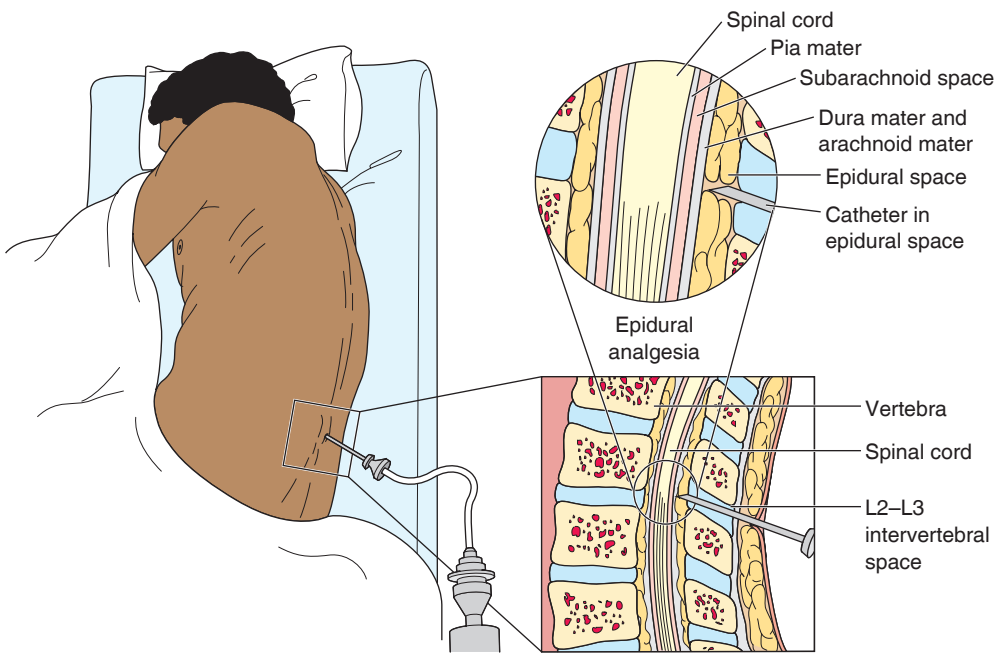
NURSING CARE

- Monitor vital signs closely. The exact methods will be determined by each individual hospital, but will most likely be similar to: monitor vital signs every 5 minutes for the first half-hour and then hourly for every hour for the first 24 hours. There is a risk of respiratory depression, which may not manifest itself for several hours.
- Ensure that naloxone, an opioid antagonist, is immediately available to reverse respiratory depression.
- Monitor the effectiveness of pain management.
- Monitor motor function (Bromage score) at least hourly in the first 6 hours post initial injection; if motor function has not returned after 3 hours, contact the anaesthetist immediately.
- Monitor fluid balance. Intraspinal opioids and local anaesthetics may alter the micturition reflex if the technique affects the lumbar dermatomes, causing urinary retention and necessitating the insertion of a urinary catheter.
- Use sterile technique to care for the catheter.

There are potential problems and contraindications when using intrathecal and epidural analgesia. For example:

- untrained staff
- refusal of treatment
- catheter placement issues (sepsis, infected injuries, spinal surgery or injury, catheter migration, disconnection or leakage at the site)
- hypovolaemia
- hypotension
- coagulation problems (including anticoagulant medications)
- potential for a dural puncture, epidural haematoma and abscess.

There are also several side effects of intrathecal and epidural analgesia, especially when opioids are used—for example, respiratory depression, sedation, nausea, vomiting, pruritis and urinary retention.



Placement of the catheter in the epidural space

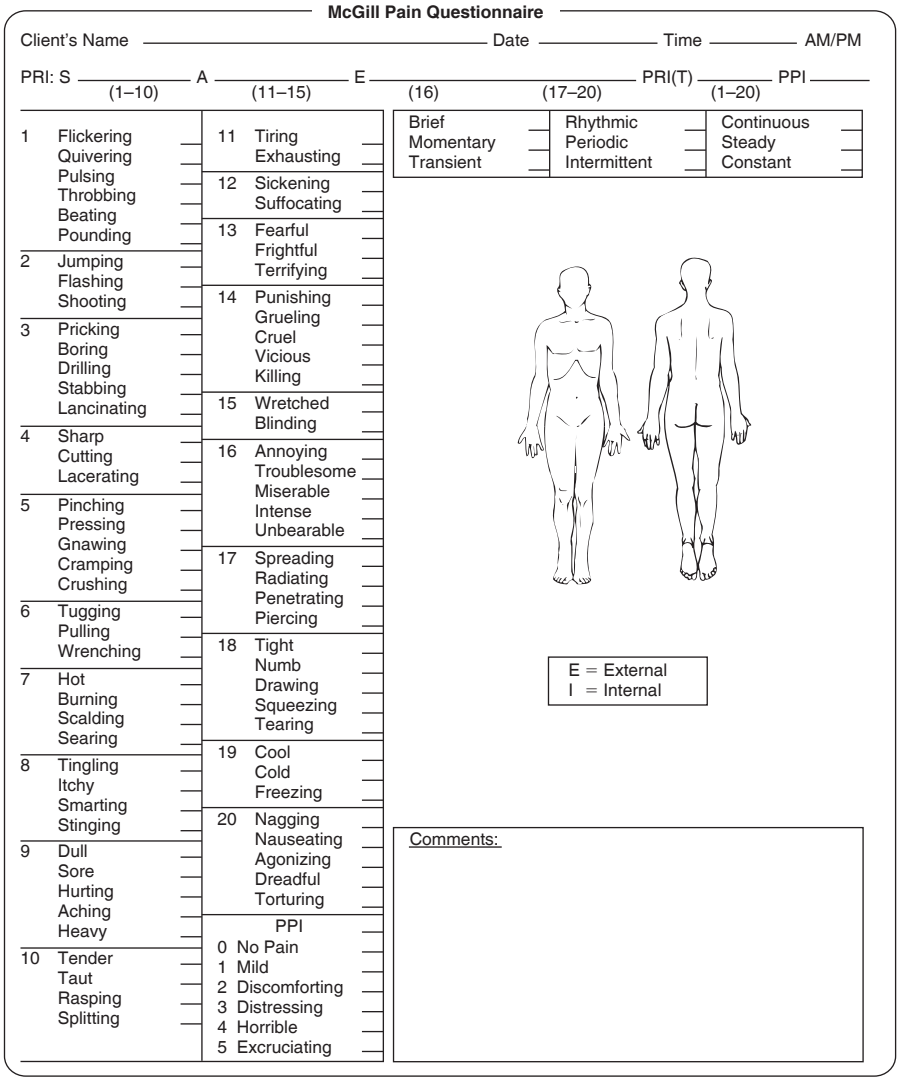


FIGURE 8.10 ■ The McGill Pain Questionnaire

Source: R. Melzack, (1975) The McGill Pain Questionnaire: Major properties and scoring methods. *Pain*, *1*(3), 277. © International Association for the Study of Pain.

- What is the region (location) of the pain? Does the pain radiate to other areas of the body?
- What is the severity of the pain? The most common method to assess the severity of pain is a pain rating scale. Several scales are illustrated in Figure 8.11.
- If you are caring for the person with an established persistent pain condition in an acute care setting, remember to ask what their normal pain score is when they are managing their life in the community. It is not unusual for those with a persistent pain condition to give higher pain scores.
- For the person who does not understand English or numerals, a scale using colours (e.g. light blue for no pain through to bright red for worst possible pain) or pictures may be helpful. Often pain assessment is made while a person is sedentary. In this state the person may experience less pain than when active and falsely estimate tolerable pain ratings. Provide guidelines for setting goals.
- What is the timing of the pain? When does it begin, how long does it last (continuous or intermittent), and how is it related to other events in the person's life?

The following nursing interventions will help the nurse assess for pain using a pain rating scale:

- Ensure consistent communication. Explain the specific pain rating scale used. If a word descriptor scale is used, verify that the person can read the language being used. If a numerical scale is used, be sure the person can count to 10. If the person is not able to report pain because of communication difficulties, intubation, emotional disturbances or cognitive impairments, monitor the manifestations of pain by taking vital signs, assessing skin temperature and moisture, observing pupils, observing facial expressions, position in bed, guarding of body parts and restlessness. Autonomic responses to pain may result in increased blood pressure, tachycardia, rapid respirations and perspiration and dilated pupils. Other responses to pain include grimacing, clenching the hands, muscle rigidity, guarding, restlessness and nausea. The person with chronic pain may have an unexpressive or tired face.
- Be sure the person is able to report pain. Researchers have found that nursing home residents with cognitive impairments can validly self-report pain (Chatterjee, 2012).

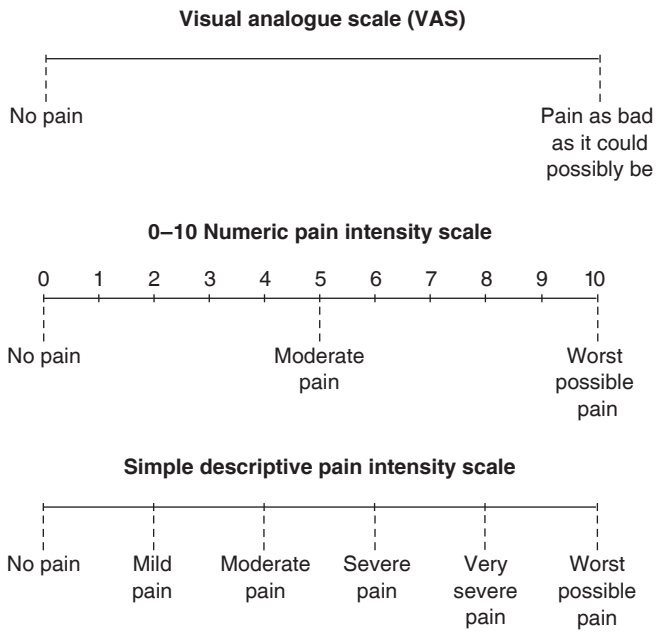


FIGURE 8.11 ■ Examples of commonly used pain scales

The following nursing interventions will help the nurse assess for pain using other measures:

- Look for indicators of pain, such as grimacing, restlessness, rubbing, stillness, verbal or non-verbal vocalisations, and holding on to an object tightly (Chatterjee, 2012).
- Pain has been shown to increase adverse behaviours such as wandering, carer resistance, socially inappropriate behaviours and delusional thoughts. Ensure pain is considered as part of the assessment of all aged care residents (Tosato et al., 2012).
- Ask a family member or caregiver, if they suspect the person has pain, to serve as a proxy pain rating.

2. Physiological response

When the body experiences acute pain, the stress response is initiated. This metabolic response releases neuroendocrine hormones systemically and a local release of cytokines at the injury site. Surgical stimuli that cause pain can influence the sympathetic efferent nerves, causing an increase in myocardial oxygen demand, influencing heart rate and blood pressure. Gastric motility is often reduced, so the potential for an ileus can develop. Injury/surgery to the upper abdominal or the thoracic area can cause difficulty in coughing, which can result in reduced functional residual capacity. If this persists, then ventilation perfusion problems can occur.

For those suffering with persistent pain, loss of muscle mass and physical deconditioning can occur. As these symptoms continue, movement restrictions develop, and insomnia, fatigue and a change in appetite may occur.

3. Examination

Examination of the person should include observation, auscultation, palpation and percussion of the sensitive areas. The physician will also undertake a physical assessment that does not cause any undue pain. Discuss the person's normal ability to perform activities of daily living and how this acute or chronic condition has affected this. Observe for pain behaviours and non-verbal clues such as facial expression and body language. Assess the effect the pain has on the person's sleep pattern. Note any pre-existing medical or pain conditions that could influence the person's current condition. Investigations ordered by the medical staff should assist in confirming clinical impressions.

4. Behavioural response

It is essential to assess the person's response to any treatment given for pain. If you give a medication for pain relief, go back and *assess* its effectiveness, especially with respect to functional abilities. The person's satisfaction must also be noted in response to their pain treatments, whether a treatment helped, had no effect on, or increased their pain. Meticulous documentation is critical as future pain regimens may be based on this.

5. Responses to treatment

Some behaviours are so typical of people in pain that the behaviours are referred to as pain behaviours. They include bracing or guarding the painful part, taking medication, crying, moaning, grimacing, withdrawing from activity and socialisation, becoming immobile, talking about pain, holding the painful area, breathing with increased effort, exhibiting a sad facial expression and being restless.

Behavioural responses to pain may or may not coincide with the person's report of pain and are not reliable cues to the pain experience. For example, one person may rate pain at an 8 on a 0–10 scale (where 0 = no pain and 10 = worst imaginable) while laughing or walking down the hall, while another may deny pain completely but be reluctant to move and grimace or moan. Denial of pain may be for a variety of reasons, including fear of injections, fear of drug addiction, misinterpretation of terms (the person may not think that aching, soreness or discomfort qualify as pain), or the belief that healthcare providers alone decide when a person is experiencing pain. Some may deny pain as part of an attempt to deny that there is something wrong with them. Others, by contrast, may think that 'as-needed' medications will be given only if their pain rating is high. Discrepancies between the person's report of pain and behavioural responses may be influenced by certain factors—for example, sociocultural factors, emotional state, knowledge, past experiences and age, and relaxation or distraction techniques.

SELF-MANAGEMENT OF PAIN The person's attempts to manage pain are useful additions to the assessment database. This information is individualised and specific to each person; it includes many factors such as culture, age and knowledge. Obtain detailed descriptions of actions the person or significant others used, and when and how these measures were applied, and how well they worked.

CONSIDERATION FOR PRACTICE

Do not assume that the older person or the person with a cognitive impairment is not having pain or is unable to identify its intensity.

Nursing diagnoses and interventions

Acute or persistent pain

Assess the characteristics of the pain by asking the person to:

- Point to the pain location, or mark the pain location on a figure drawing. *Pain location provides information about the aetiology of the pain and the type of pain being experienced.*
- Rate the intensity of the pain at rest and during movement by using a pain scale (1 to 10, with 10 being the worst pain ever experienced), a visual analogue scale (a scale on which pain is marked on a continuum from no pain to severe pain), or with word descriptors (such as the McGill Pain Questionnaire, and mild, moderate or severe). Use the same scale with each assessment. *The intensity of pain is a subjective experience. The perception of the intensity of pain is affected by the person's degree of concentration or distraction, state of consciousness and expectations.*
- Describe the quality of the pain. For example, say to the person: 'Describe what your pain feels like.' If necessary, provide word descriptors for the person to select from. *Descriptive terms provide insight into the nature and perception of the pain. In addition, the location and type of pain (e.g. acute versus persistent) affect the quality.*
- Describe the pattern of the pain, including time of onset, duration, persistence and times without pain. It is also important to ask whether the pain is worse at regular times of the day and whether it has any relationship to activity. *The pattern of pain provides clues about cause and location.*
- Describe any precipitating or relieving factors. *Precipitating factors include sleep deficits, anxiety, temperature extremes, excessive noise, anxiety, fear, depression and activity.*
- Describe the meaning of the pain, including its effects on lifestyle, self-concept, roles and relationships. *The person with acute pain may believe the pain is a normal response to injury, or that it signals serious illness and death. Pain is a stressor that may affect the person's ability to cope effectively. The person with persistent pain often has concerns about addiction to pain medication, costs, social interactions, sexual activities and relationships with significant others.*
- Monitor manifestations of pain by assessing skin temperature and moisture; observing pupils; observing facial expressions, position in bed and guarding of body parts; and noting restlessness. *Autonomic responses to pain may result in increased blood pressure, tachycardia, rapid respirations, perspiration and dilated pupils. Other responses to pain include grimacing, clenching the hands, muscle rigidity, guarding, restlessness and nausea. The person with persistent pain may have an unexpressive, flat affect with a tired facial appearance. (Remember, if the person has persistent pain, vital signs may not be increased.)*
- Communicate belief in the person's pain by verbally acknowledging the presence of the pain, listen carefully to the description of pain, and act to help the person manage the pain. *Because pain is a personal, subjective experience, the nurse must convey belief in the person's pain. By doing so, the nurse reduces the person's anxiety and thereby lessens their pain.* See the 'Translation to practice' box below.
- Provide optimal pain relief with prescribed analgesics, determining the preferred route of administration. Provide pain-relieving measures for severe pain on a regular around-the-clock basis or by self-administration (such as with a PCA pump). *The person is part of the decision-making process and can exert some control over the situation by choosing the administration route. Analgesics are usually most effective when they are administered before pain occurs or becomes severe. Around-the-clock administration has been proven to provide better pain management for both acute and persistent pain. Do not crush or break, or allow the person to chew, controlled-release oral preparations; a dose meant to be slowly absorbed that is absorbed rapidly may lead to a toxic overdose and death.*
- Evaluate and monitor the effects of analgesics and other pain-relieving measures and teach family members or significant others to be alert for adverse reactions to pain medications. Sedation, constipation, nausea and dizziness are common side effects. *Excessive sedation can progress to significant respiratory depression. Oxygen saturation should be checked regularly. Protocols will vary within your institution. Prevent falls that may result from sedation or dizziness. If the person has symptoms of excessive opioid dosage, antidotes are available. Naloxone is used for opioid overdose. Titrate naloxone slowly. Never push an entire dose all at once. Administer only enough naloxone to eliminate adverse effects such as respiratory depression or excessive sedation. If excessive naloxone is administered, the person may experience acute withdrawal and will have no pain relief. It may take considerable time to re-establish a therapeutic comfort level.*
- Determine the level of sedation the person will tolerate. *For those with persistent pain or cancer pain who need high doses of opioids, sedation may interfere with quality of life and neither the person nor the family want them to be sedated.* Several classes of drugs can be used to counteract sedation. They are usually given in the morning so that they will not interfere with night sleep. Amphetamines, especially methylphenidate (Ritalin), is the most commonly used; modafinil (Modavigil) has been used for several years; and donepezil (Aricept), which is used for the symptoms of Alzheimer's disease, reduces sedation and fatigue (Caraceni et al., 2012).
- Teach the person and family non-pharmacological methods of pain management, such as relaxation, distraction and cutaneous stimulation. *These techniques are especially useful when used in conjunction with pain medications and may also be useful in managing persistent pain.*
- Provide comfort measures, such as changing position, massage, oral care, skin care and changing bed linen. *Basic comfort measures for personal cleanliness, skin care and mobility promote physical and psychosocial wellbeing, lessening the perception of pain.*
- Provide person and family education, and make referrals if necessary to assist with coping, such as financial support services and home care. *The person (and family) with pain requires information about medications, non-invasive techniques for pain management and sources of assistance with home-based care. The person with acute*

TRANSLATIONTO PRACTICE Evidence-based practice for the person experiencing pain

Poor pain management is common. Despite having a wealth of evidence-based practice available there is a gap between research and clinical practice. Observational studies of pain management are valuable because they are time-sensitive. Relying on nurses' self-report of pain management introduces bias and loses currency. In a study by Manias, Bucknall & Botti (2005), nurses were observed in 2-hour periods. Examination of the collected observations by independent analysts revealed six categories of response to a person's pain. The pain was:

1. responded to effectively
2. prioritised as less important than completing medication administration, assessing vital signs, taking telephone calls or changing dressings
3. ignored because cues were missed
4. treated as part of the medication administration regimen and given or withheld according to schedule
5. prevented through comfort measures, medicating before pain was present or was going to occur as with dressing changes, teaching the importance of early communication about pain
6. was only addressed reactively, after the painful experience.

Because pain management is important, many hospitals employ acute, palliative and persistent pain services, with more attention being encouraged regarding listening to each person's self-reporting of pain. Communication among nurses, physicians and the person experiencing pain is key to pain relief. Knowledge of the pharmacodynamics and pharmacokinetics of analgesics needs to be taught and updated regularly. Environmental distractions and interruptions are associated with less attention to pain management. Few conditions have higher priority for the person than pain relief, but the study revealed that nurses accept pain as a normal component of the postoperative surgery experience. Administrators need to be aware that competing responsibilities affect nurses' ability to provide effective pain management. These responsibilities include documentation, admitting new people, and completing discharge education and arrangements. Pain management is an important component of professional nursing—nurses should be supported in their efforts to address pain with compassion and efficiency.

CRITICAL THINKING IN PERSON-CENTRED CARE

1 Reflect on your own experiences with pain. Evaluate how these experiences help or hinder your assessments and interventions for a person in pain.
2 You are caring for a young man who has multiple injuries from a motorcycle accident. He tells you his pain is so bad that he just wants to die. Describe and explain how you would respond, and why.
3 You are caring for an 80-year-old man with diabetes who has had his left foot amputated for gangrene. He is restless and moaning. Another nurse tells you to give only one-half of the ordered dose of opioids because 'he is old and there is a danger of respiratory depression'. Assess this nurse's suggested intervention and explain how you would handle this situation.
4 'Nurses tend to underestimate and under-medicate pain.' Discuss this statement using recent published research.

pain requires information about the expected course of pain resolution.

Community-based care

Educate the person, family and significant others regarding:

- specific drugs to be taken, including the frequency, potential side effects, possible drug interactions and any special precautions to be taken (such as taking with food or avoiding alcohol)
- how to take or administer the drugs (see Table 8.3)
- the importance of taking pain medications before the pain becomes severe
- an explanation that the risk of addiction to pain medications is very small when they are used appropriately for pain relief
- the importance of scheduling periods of rest and sleep.

In addition, suggest the following resources:

- pain clinics
- community support groups
- International Association for the Study of Pain (IASP)
- Australian Pain Society (APS)
- Cancer Council Australia.

TABLE 8.3 Providing long-term analgesia at home

ROUTE	DRUG	NURSING IMPLICATIONS
Oral	Oxycodone	Available in a slow-release formulation for 12-hour dosing (Oxycontin) and as fast-acting formulations (Oxynorm or Oxycodone elixir) for breakthrough pain.
Oral	Morphine	Formulated as slow-release particles in a capsule (Kapanol). If the person cannot swallow the capsule, it may be sprinkled over food or given by gastric tube. Morphine can also be given as a slow-release tablet (MsContin) or as immediate-release preparations either as a tablet (Anamorph) or capsule (Sevredol) or an elixir (Ordine).
Transdermal	Fentanyl	A patch absorbed slowly through the skin (Durogesic); allows 72-hour dose schedule. Takes up to 12 hours to achieve therapeutic level; when discontinued, therapeutic effect will decay slowly. A lozenge formulation fentanyl citrate (Actiq) absorbed through the buccal cavity can be used to treat breakthrough cancer pain in opioid-tolerant people.

NURSING CARE PLAN A person with persistent pain

Susan Akers, aged 37, is currently being seen at an outpatient clinic for persistent non-malignant pain. She works at a local paper factory. She has a 3-year history of neck and shoulder pain that usually is accompanied by headaches. She believes the pain is related to lifting objects at work, but it is now precipitated by activities of daily living. Susan is absent from work approximately three times a month and states that the absences are due to her pain and headaches. She has been seeking care in the local emergency department on an average of twice monthly for injections for pain. She does not regularly use medications but does take Panadeine Forte and Valium as needed (usually 3 or 4 times a day). Susan is divorced and has two children. She states that she has several friends in the area, but her parents and siblings live interstate.

ASSESSMENT

During the nursing history, Susan rates her pain during an acute episode as a 7 on a 1 to 10 scale. She states that lifting objects and moving her hands and arms above shoulder level causes sharp pain. The pain never really goes away, but it does decrease with upper extremity rest. She says that when she lifts a lot at work, she has difficulty sleeping that night. She takes two Panadeine Forte tablets every 4 hours when the pain is severe, but does not get complete relief. The medical officer diagnoses muscular pain.

DIAGNOSIS

- *Persistent pain* related to muscle inflammation resulting from repetitive movement and lifting of heavy objects manifested by pain on movement that lessens at rest and in response to analgesia.

PLANNING

After negotiating expected outcomes and intervention techniques, opioid analgesia and distraction techniques are to be used during the acute phase of management. Non-opioid analgesia is to be used after the acute phase and review of the work environment is to be undertaken.

Expected outcomes

- Return for follow-up visits with a journal of activities and pain experiences.
- After 3 to 5 days on regularly scheduled doses of pain medication, report a decrease in the level of pain from 7 to 3 or 4 on a 1 to 10 scale.
- Decreased number of absences from work.
- Modify activities at work and at home, especially when pain is intense.

IMPLEMENTATION

- Encourage discussion of pain and acknowledge belief in Susan's report of pain.
- Consult with a physician for a non-opioid analgesic with a minimum of side effects, and instruct in maintaining regular dosing schedules.
- For episodes of acute pain, take opioid analgesics as soon as the pain begins and every 4 hours, while continuing the dosage of non-opioid analgesic.
- Teach one relaxation technique that is personally useful.
- Explore distraction techniques such as listening to music, watching comedies or reading.
- Provide clinic phone number and instruct to call if pain is unrelieved with narcotic and non-opioid analgesics.

EVALUATION

Susan returns for scheduled follow-up visits with a completed journal of her activities and associated pain. She reports that taking oral opioid analgesics has relieved her pain and that within 3 weeks non-opioid analgesics have brought her pain under control. She also reports that her supervisor has reassigned her to a position that requires no lifting. She now rates her pain at 2 or 3 on a 1 to 10 scale. She has missed only 1 day of work in the last 3 months and reports that her children and friends have helped with her household tasks when she has requested they do so.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe three factors that support the statement 'Pain is a personal experience.'
2. Susan asks you how often she should take her pain medications. Do you tell her to (a) take them on a regular basis, or (b) wait until she experiences pain? Which action would you choose, and why?
3. Susan is at risk of constipation. Why, and what information would you provide?

REFLECTION ON THE NURSING PROCESS

1. What have you learned from the case study that you will apply to your clinical practice?
2. If someone is non-compliant with the negotiated care plan and returns to the pain clinic with continued discomfort, consider some of the reasons this non-compliance may have occurred.

CHAPTER HIGHLIGHTS

- Pain is perceived in the central nervous system. Opioids, and other analgesics, block the perception of pain; NSAIDs and most non-pharmacological interventions block or decrease the transmission of pain from the periphery to the CNS. Measures to block the sensitisation of pain-transmitting fibres are being used prior to painful procedures and incision in surgery.
- There are many types of pain, and treatment varies according to the type and combination of types. Acute pain usually decreases as healing progresses; persistent pain has acute exacerbations and compounds acute pain. Breakthrough pain is recognised as an increase in pain intensity that occurs when the peak and duration of medications are reached. Incident pain occurs in relation to a change or increase in activity. Central pain results from CNS lesions. Phantom pain occurs after amputation, seeming to originate in the missing body part.
- Ethnicity (genetics and culture) and gender impact on pain perception and behaviour. Behavioural assessment of pain intensity is less accurate than the person's report of pain intensity.

- Myths and misconceptions exist about pain and its management; the person's perceived cause of pain and the best self-care method to relieve pain provide pertinent assessment information.
- People respond to pain on the basis of their emotional state, past experiences with pain, and the meaning of the painful experience. The person with a malignant diagnosis may have an interpretation of pain that is significantly different from the person with benign disease.
- Older adults perceive pain as intensely as younger adults. The amount of opioids they may need is variable, as they may have difficulty metabolising or eliminating medications.
- Assertively assess pain in the older person. Older adults may hesitate to report pain for fear of losing independence or being considered a nuisance. Cognitive impairment following cardiovascular accident, dementia or delirium makes self-report of pain less available. Behavioural scales for assessing pain are useful when the person cannot give self-reports.
- Addiction is a neurophysiological disease. The person with addiction to opioids may need greater doses of opioids to control pain because of the tolerance they may have developed through usage.
- Emergency department physicians and nurses are constrained in the prescription of opioids by the risk of overmedicating the person who is drug-seeking and by the myth that pain management interferes with diagnosis.
- Pain management includes assessment, intervention and evaluation. It is important to verify that interventions have been effective. If not, interventions must be identified that bring pain down to a level of intensity with which the person feels satisfied.

CONCEPT CHECK

1 Your neighbour has had lower back pain for 9 months. How would this pain be categorised?
   1 acute pain
   2 persistent pain
   3 referred pain
   4 somatic pain

2 Which of the following statements is a misconception about pain?
   1 It is best to wait until a person has pain before giving medication.
   2 Anxiety can cause pain and pain can cause anxiety.
   3 Pethidine is no longer recommended for postoperative pain.
   4 The rationale for use of a TENS unit is supported by the gate-control theory.

3 You are taking a health history for a person who has taken a NSAID for several years. What would be an appropriate question to ask?
   1 'Do you understand what this drug could do to you?'
   2 'Have you ever vomited blood or had very dark stools?'
   3 'Do you know that you may become addicted to this drug?'
   4 'Have you noticed any problems with your breathing?'

4 You are replacing a transdermal pain medication. Where on the body would you place it?
   1 on one side of the buttocks
   2 below the navel, midline on the abdomen
   3 on the anterior thigh
   4 on the upper torso

5 Which of the following statements would be most useful in determining the quality of a person's pain?
   1 'Tell me where you hurt.'
   2 'Rate your pain on a scale of 0 to 10.'
   3 'Describe what your pain feels like.'
   4 'Tell me how this pain affects your sleep.'

6 The person has orders for intravenous patient-controlled analgesia (PCA). The following principles are true, except:
   1 basal doses are continuous
   2 overdose cannot occur
   3 a 10-minute lockout each hour allows six bolus doses
   4 unused bolus doses cannot accumulate

7 The most common side effects of opioid analgesics are:
   1 anuria, diplopia and cough
   2 constipation, nausea and sedation
   3 pruritus, constipation and hallucinations
   4 dysphagia, fever and gastritis

8 The preferred route of opioid administration for persistent pain is:
   1 transdermal
   2 oral
   3 intravenous
   4 rectal

9 The equivalent dose of an oral drug compared to the intravenous preparation of the same drug:
   1 is equal dosage
   2 is twice the intravenous dose
   3 varies according to the medication
   4 is one-half the intravenous dose

10 People treated for persistent pain may need additional pain management strategies for:
   1 breakthrough pain
   2 acute pain
   3 end-of-dose pain
   4 all of the above

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CHAPTER 9

NURSING CARE OF PEOPLE WITH ALTERED FLUID, ELECTROLYTE AND ACID–BASE BALANCE

KAMAREE BERRY

KEY TERMS

LEARNING OUTCOMES

- Describe the functions and regulatory mechanisms that maintain water and electrolyte balance in the body.
- Compare and contrast the causes, effects and care of the person with fluid volume or electrolyte imbalance.
- Explain the pathophysiology and manifestations of imbalances of sodium, potassium, calcium, magnesium and phosphorus.
- Describe the causes and effects of acid–base imbalances.

CLINICAL COMPETENCIES

- Assess and monitor the person's fluid, electrolyte and acid–base balance.
- Administer fluids and medications knowledgeably and safely.
- Determine priority nursing diagnoses, based on assessment data, to select and implement individualised nursing interventions.
- Deliver pertinent information to the person and their family about diet and medications used to restore, promote and maintain fluid, electrolyte and acid–base balance.
- Integrate interprofessional care into care of a person with altered fluid, electrolyte and acid–base balance.

Changes in the normal distribution and composition of body fluids often occur in response to illness and trauma. These changes affect fluid balance of the intracellular and extracellular compartments of the body, the concentration of electrolytes within fluid compartments and the body's hydrogen ion concentration (pH). Normal physiological processes depend on a relatively stable state in the internal environment of the body. The fluid volume, electrolyte composition and pH of both intracellular and extracellular spaces must remain constant within a relatively narrow range to maintain health and life.

Homeostasis is the body's tendency to maintain a state of physiological balance in the presence of constantly changing conditions. Homeostasis is necessary if the body is to function optimally at a cellular level and as a total organism. Homeostasis depends on multiple factors in both the external and internal environments, such as available oxygen in the air and nutrients in food, as well as normal body temperature, respiration and digestive processes. The normal volume, composition, distribution and pH of body fluids reflect a state of homeostasis.

Changes in the normal volume of fluids, their composition, distribution, and relative acidity or alkalinity have the potential to disrupt most functional health patterns. Imbalances of fluids, electrolytes and pH affect the ability to maintain activities of daily living (the Activity–Exercise Pattern), think clearly (the Cognitive–Perceptual Pattern) and engage in self-care (the Health Perception–Health Management Pattern). Conversely, alterations in a number of health patterns affect the ability to maintain homeostasis. Alterations in the Nutritional–Metabolic Pattern affect the ability to consume adequate food and fluids. Disruptions of the Elimination Pattern may lead to retention or loss of excess amounts of fluids and electrolytes. Disrupted heart or respiratory function, which falls within the Activity–Exercise Pattern, has the potential to affect fluid, electrolyte and acid–base balance.

The goal in managing fluid, electrolyte and acid–base imbalances is to re-establish and maintain a normal balance. Nursing care includes identifying and assessing a person who is likely to develop imbalances, monitoring the person for early manifestations, and implementing collaborative and nursing interventions to prevent or correct imbalances. Effective nursing interventions require an understanding both of the multiple processes that maintain fluid, electrolyte and acid–base balance, and of the causes and treatment of imbalances that occur.

Mechanisms that maintain normal fluid and electrolyte balance are discussed first, followed by sections on fluid imbalances and electrolyte imbalances. Discussion of normal acid–base balance precedes discussion of acid–base imbalances. Case studies related to selected fluid, electrolyte and acid–base disorders are found throughout the chapter.

OVERVIEW OF NORMAL FLUID AND ELECTROLYTE BALANCE

Fluid and electrolyte balance in the body involves regulatory mechanisms that maintain the composition, distribution and movement of fluids and electrolytes. This section provides an overview of fluid and electrolyte balance in the body. It is followed by discussion of fluid volume and electrolyte balance disorders.

Body fluid composition

Body fluid is composed of water and various dissolved substances (solutes).

Water

Water is the primary component of body fluids. It functions in several ways to maintain normal cellular function. Water provides a medium for the transport and exchange of nutrients and other substances such as oxygen, carbon dioxide and metabolic wastes to and from cells; provides a medium for metabolic reactions within cells; assists in regulating body temperature through the evaporation of perspiration; provides form for body structure and acts as a shock absorber; provides insulation and acts as a lubricant.

Total body water constitutes about 60% of total body weight, but this amount varies with age, gender and the amount of body fat. Total body water decreases with ageing; in the older adult, body water may decrease to 45% to 50% of total body weight (Marieb & Hoehn, 2011). Adipose tissue contains comparatively little water. In the person who is obese, the proportion of water to total body weight is less than in the person of average weight; in a person who is very thin, the proportion of water to total body weight is greater than in the person of average weight. Adult females have a greater ratio of fat to lean tissue mass than adult males; therefore, they have a lower percentage of body water content.

To maintain normal fluid balance, body water intake and output should be approximately equal. The average fluid intake and output usually is about 2500 mL over a 24-hour period. Food and fluids consumed provide the majority of water gain; carbohydrate metabolism and other metabolic processes produce an additional small amount.

Urine production and excretion account for most water loss. The average daily urine output is 1200 to 1500 mL in adults. At least 400 mL of highly concentrated urine per day is required to excrete metabolic wastes produced by the body (Marieb & Hoehn, 2011). *Insensible* water loss (which normally cannot be measured) occurs through the skin, lungs and faeces. These losses, while normally small, can increase significantly during exercise, when environmental temperatures are high, and during illness that increases the respiratory rate, perspiration or gastrointestinal (GI) losses (particularly diarrhoea). Table 9.1 shows the sources of fluid gain and loss.

Electrolytes

Body fluids contain both water molecules and chemical compounds. These chemical compounds can either remain intact in solution or separate (dissociate) into discrete particles. **Electrolytes** are substances that dissociate in solution to form charged particles called ions. *Cations* are positively charged electrolytes; *anions* are negatively charged electrolytes. For example, sodium chloride (NaCl) in solution dissociates into a sodium ion, a cation carrying a positive charge (Na^+), and a chloride ion, an anion carrying a

TABLE 9.1 Balanced fluid gain and loss for an adult

	SOURCE	AMOUNT (mL)
Gain	Fluids taken orally	1200
	Water in food	1000
	Water as by-product of food metabolism	300
	↓	
	Total	**2500**
	↑	
Loss	Urine	1500
	Faeces	200
	Perspiration	300
	Respiration	500

negative charge (Cl^-). Electrolytes may be *univalent*, with only one unit of electrical charge, such as sodium (Na^+) and chloride (Cl^-); or they may be *divalent*, carrying two units of electrical charge, such as magnesium (Mg^{2+}) and phosphate (HPO_4^{2-}).

Electrolytes have many functions, including assisting in regulating water balance; regulating and maintaining acid–base balance; and contributing to enzyme reactions essential for neuromuscular activity.

The concentration of electrolytes in body fluids generally is measured in milliequivalents per litre of water (mEq/L). A *milliequivalent* is a measure of the chemical combining power of the ion. For example, 100 mEq of sodium (Na^+) can combine with 100 mEq of chloride (Cl^-) to form sodium chloride (NaCl). Sodium, potassium and chloride usually are measured in milliequivalents. In some cases, the amount of an electrolyte in body fluid may be measured by weight in milligrams per 100 mL (1 decilitre, dL) of water (mg/dL). Calcium, magnesium and phosphorus often are measured by weight in milligrams per decilitre. Other laboratories use the International System of Measurements, or SI units.

Body fluid distribution

Body fluid is classified by its location inside or outside cells. *Intracellular fluid* (ICF) is found within cells. It accounts for approximately 40% of total body weight (see Figure 9.1) and is essential for normal cell function, providing a medium for metabolic processes. *Extracellular fluid* (ECF) is located outside cells and accounts for approximately 20% of total body weight. ECF is classified by location:

- Interstitial fluid is located in the spaces between most of the cells of the body. It accounts for approximately 15% of total body weight.
- Intravascular fluid, called *plasma*, is contained within the arteries, veins and capillaries. It accounts for approximately 5% of total body weight.
- Transcellular fluid includes urine, digestive secretions and perspiration, as well as cerebrospinal, pleural, synovial, intraocular, gonadal and pericardial fluids.

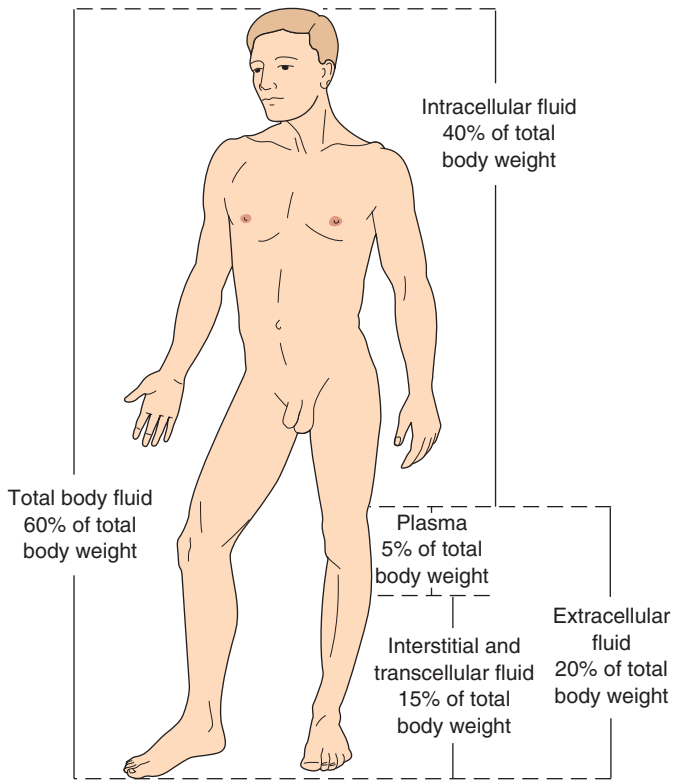


FIGURE 9.1 ■ The major fluid compartments of the body

A trace amount of water is found in bone, cartilage and other dense connective tissues; this water is not exchangeable with other body fluids.

ECF is the transport medium that carries oxygen and nutrients to, and waste products from, the cells. For example, plasma transports oxygen from the lungs and glucose from the digestive system to the tissues. These solutes diffuse through the capillary wall into the interstitial space and from there across the cell membrane into the cells. Waste products of metabolism (e.g. carbon dioxide and hydrogen ions) diffuse from the intracellular space into the interstitial space and from there into plasma via the capillary walls. Plasma then transports these waste products to the lungs and kidneys for elimination.

Although the overall concentration of solutes in ICF and ECF is nearly identical, the concentration of specific electrolytes differs significantly between these compartments, as shown in Figure 9.2. ICF contains high concentrations of potassium (K^+), magnesium (Mg^{2+}) and phosphate (PO_4^{2-}), as well as other solutes such as glucose and oxygen. Sodium (Na^+), chloride (Cl^-) and bicarbonate (HCO_3^-) are the principal extracellular electrolytes. The high sodium concentration in ECF is essential to regulating body fluid volume. The concentration of potassium in ECF is low. There is a minimal difference in electrolyte concentration between plasma and interstitial fluid. Normal values for electrolytes in plasma are shown in Table 9.2.

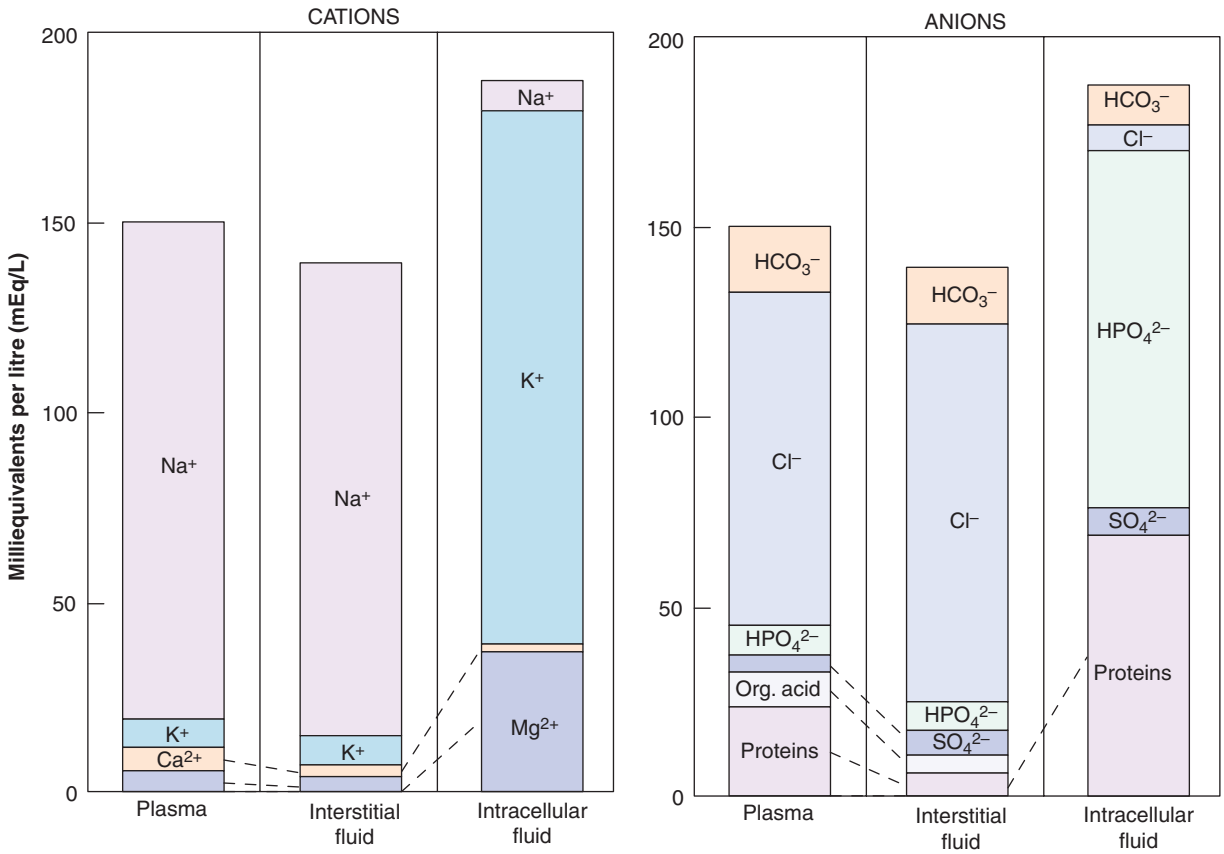


FIGURE 9.2 ■ Electrolyte composition (cations and anions) of body fluid compartments

Source: Fundamentals of anatomy and physiology (4th ed.) by F. Martini (1998). Figure 27.2, p. 1009. Reprinted and electronically produced by permission of Pearson Education, Inc., Upper Saddle River, NJ.

The body fluid compartments are separated by several types of membranes:

- Cell membranes separate interstitial fluid from intracellular fluid.
- Capillary membranes separate plasma from interstitial fluid.
- Epithelial membranes separate transcellular fluid from interstitial fluid and plasma. These membranes include the mucosa of the stomach, intestines and gallbladder; the pleural, peritoneal and synovial membranes; and the tubules of the kidney.

A cell membrane consists of layers of lipid and protein molecules. The layering of these molecules controls the passage of fluid and solutes between the cell and interstitial fluid. The cell membrane is selectively permeable; that is, it allows the passage of water, oxygen, carbon dioxide and small water-soluble

TABLE 9.2 Normal values for electrolytes and serum osmolality

SERUM COMPONENT	VALUES	
ELECTROLYTES	CONVENTIONAL UNITS	SI UNITS
Sodium (Na^+)	135–145 mEq/L	135–145 mmol/L
Chloride (Cl^-)	95–105 mEq/L	95–105 mmol/L
Bicarbonate (HCO_3^-)	22–30 mEq/L	22–30 mmol/L
Calcium (Ca^{2+}) (total)	9–11 mg/dL	2.3–2.8 mmol/L
Potassium (K^+)	3.5–5.3 mEq/L	3.5–5.0 mmol/L
Phosphate/inorganic phosphorus (PO_4^{2-})	1.7–2.6 mEq/L (2.5–4.5 mg/dL)	0.8–1.5 mmol/L
Magnesium (Mg^{2+})	1.5–2.5 mg/dL (1.8–3.0 mEq/L)	0.8–1.3 mmol/L
Serum osmolality	280–300 mOsm/kg	275–295 mmol/kg

molecules, but bars proteins and other intracellular colloids. The capillary membrane separating the plasma from the interstitial space is made of squamous epithelial cells. Pores in the membrane allow solute molecules (such as glucose and sodium), dissolved gases and water to cross the membrane. Minute amounts of albumin and other proteins can also pass through the pores of a capillary membrane, but normally plasma proteins stay in the intravascular compartment.

Body fluid movement

Four chemical and physiological processes control the movement of fluid, electrolytes and other molecules across membranes between the intracellular and interstitial spaces and the interstitial space and plasma. These processes are osmosis, diffusion, filtration and active transport.

OSMOSIS The process by which water moves across a selectively permeable membrane from an area of lower solute concentration to an area of higher solute concentration is called **osmosis** (see Figure 9.3). A *selectively permeable membrane* allows water molecules to cross but is relatively impermeable to dissolved substances (solutes). Osmosis continues until the solute concentration on both sides of the membrane is equal. For example, if pure water and a sodium chloride solution are separated by a selectively permeable membrane, then water molecules will move across the membrane to the sodium chloride solution. Osmosis is the primary process that controls body fluid movement between the ICF and ECF compartments.

Osmolarity and osmolality The concentration of a solution may be expressed as the osmolarity or osmolality of the solution. *Osmolarity* refers to the quantity of solutes per litre of solution (by volume); it is reported in milliosmoles per litre (mOsm/L) in a solution. *Osmolality* refers to the quantity of solutes per kilogram of water (by weight); it is reported in milliosmoles per kilogram (mOsm/kg). Because osmotic activity in the body is regulated by the number of active particles (solutes) per kilogram of water, osmolality is used to describe the concentration of body fluids. The normal osmolality of both ICF and ECF ranges between 275 and 295 mOsm/kg. The osmolality of the ECF depends chiefly on sodium concentration. Serum osmolality may be estimated by doubling the serum sodium concentration (approximately 142 mEq/L). Glucose and urea contribute to the osmolality of ECF, although to a lesser extent than sodium.

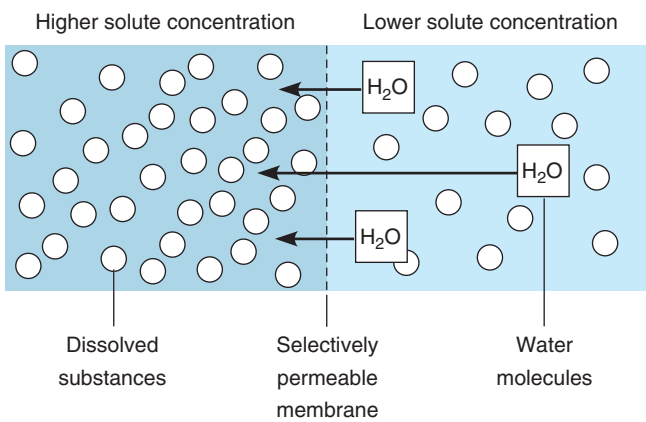


FIGURE 9.3 ■ Osmosis. Water molecules move through a selectively permeable membrane from an area of low solute concentration to an area of high solute concentration

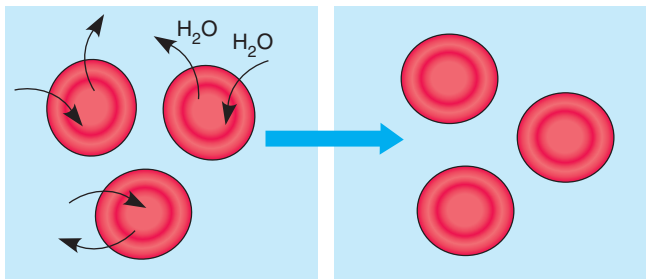
Osmotic pressure and tonicity The power of a solution to draw water across a membrane is known as the *osmotic pressure* of the solution. The composition of interstitial fluid and intravascular plasma is essentially the same except for a higher concentration of proteins in the plasma. These proteins (especially albumin) exert colloid osmotic pressure (also called oncotic pressure), pulling fluid from the interstitial space into the intravascular compartment. Because the osmolality of intravascular and interstitial fluid is essentially identical, the osmotic activity of plasma proteins is important in maintaining fluid balance between the interstitial and intravascular spaces, helping to hold water within the vascular system.

Tonicity refers to the effect a solution's osmotic pressure has on water movement across the cell membrane of cells within that solution. *Isotonic* solutions have the same concentration of solutes as plasma. Cells placed in an isotonic solution will neither shrink nor swell because there is no net gain or loss of water within the cell and no change in cell volume (see Figure 9.4A). Normal saline (0.9% sodium chloride solution) is an example of an isotonic solution.

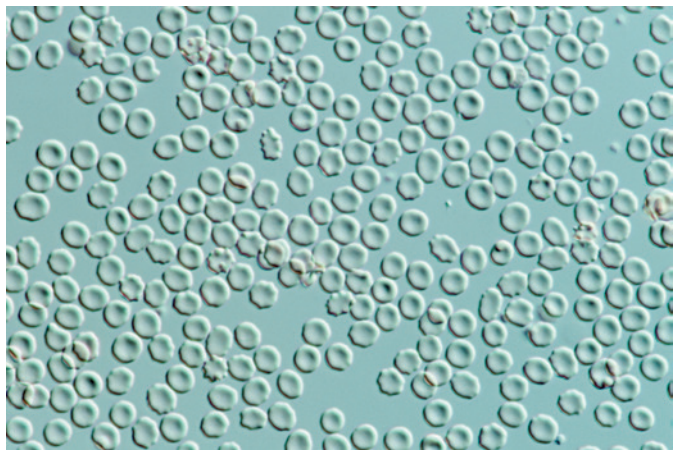
Hypertonic solutions have a greater concentration of solutes than plasma. In their presence, water is drawn out of a cell, causing it to shrink (see Figure 9.4B). A 3% sodium chloride solution is hypertonic. *Hypotonic* solutions (such as 0.45% sodium chloride) have a lower solute concentration than plasma (see Figure 9.4C). When red blood cells are placed in a hypotonic solution, water moves into the cells, causing them to swell and rupture (*haemolyse*).

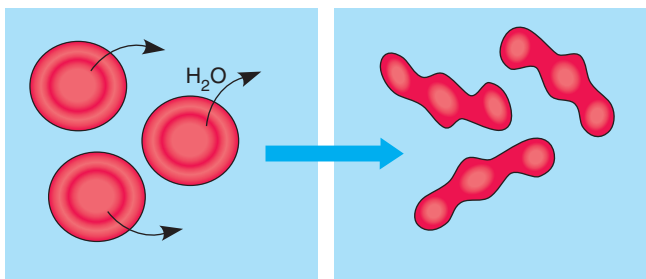
The concepts of osmotic draw and tonicity are important in understanding the pathophysiological changes that occur with fluid and electrolyte imbalances, as well as treatment measures. For example, an increased sodium concentration of extracellular fluid pulls water from the ICF compartment into the ECF compartment, causing cells to shrink. In this case, administering a hypotonic intravenous solution to reduce the sodium concentration and osmolality of ECF will facilitate water movement back into the cells.

DIFFUSION The process by which solute molecules move from an area of high solute concentration to an area of low solute concentration to become evenly distributed is called **diffusion** (see Figure 9.5). The two types of diffusion are simple and facilitated diffusion. *Simple diffusion* occurs by the random movement of particles through a solution. Water, carbon dioxide, oxygen and solutes move between plasma and the interstitial space by simple diffusion through the capillary membrane. Water and solutes move into the cell by passing through protein channels or by dissolving in the lipid cell membrane. *Facilitated diffusion*, also

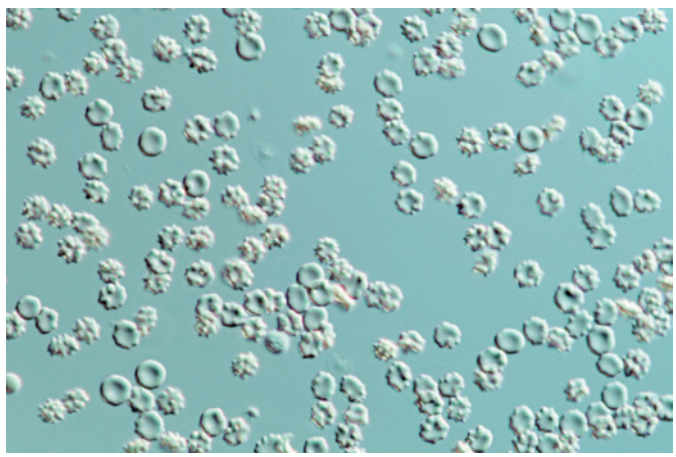


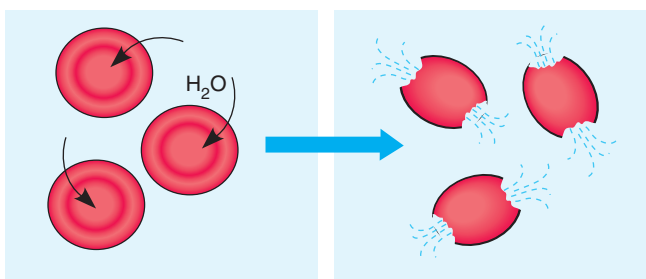
A Isotonic solution





B Hypertonic solution





C Hypotonic solution

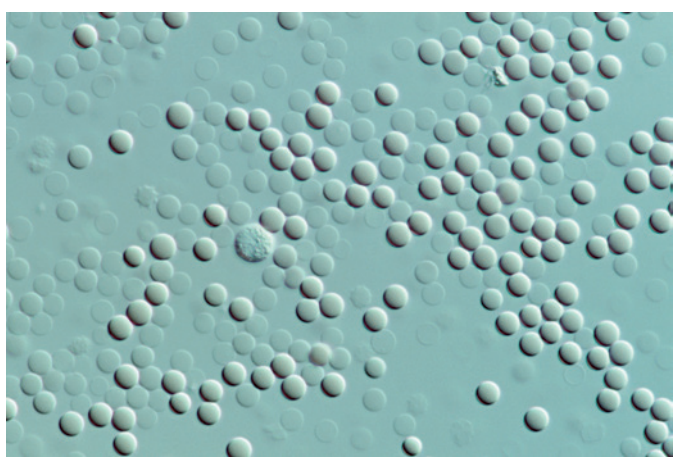


FIGURE 9.4 ■ The effect of tonicity on red blood cells. *A*, In an isotonic solution, red blood cells neither gain nor lose water, retaining their normal biconcave shape. *B*, In a hypertonic solution, cells lose water and shrink in size. *C*, In a hypotonic solution, cells absorb water and may burst (haemolysis)

Source: Photos © Herve Conge, ISM/Science Photo Library.

called carrier-mediated diffusion, allows large water-soluble molecules, such as glucose and amino acids, to diffuse across cell membranes. Proteins embedded in the cell membrane function as *carriers*, helping large molecules cross the membrane.

The rate of diffusion is influenced by a number of factors, such as the concentration of solute and the availability of carrier proteins in the cell membrane. The effect of both simple and facilitated diffusion is to establish equal concentrations of the molecules on both sides of a membrane.

FILTRATION The process by which water and dissolved substances (solutes) move from an area of high hydrostatic pressure to an area of low hydrostatic pressure is called **filtration**. This usually occurs across capillary membranes. *Hydrostatic*

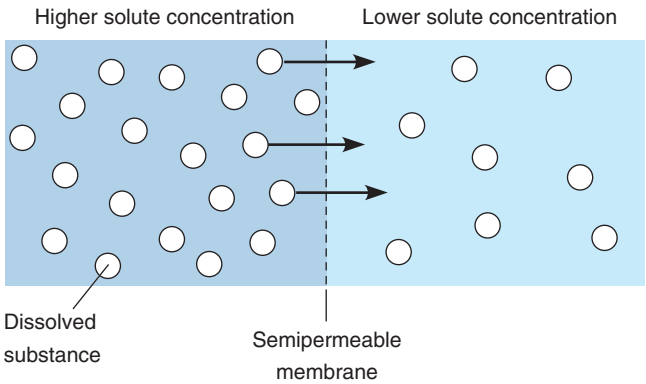


FIGURE 9.5 ■ Diffusion. Solute molecules move through a semipermeable membrane from an area of high solute concentration to an area of low solute concentration

pressure is created by the pumping action of the heart and gravity against the capillary wall. Filtration occurs in the glomerulus of the kidneys, as well as at the arterial end of capillaries.

A balance of hydrostatic (filtration) pressure and osmotic pressure regulates the movement of water between the intravascular and interstitial spaces in the capillary beds of the body. Hydrostatic pressure within the arterial end of the capillary pushes water into the interstitial space. Hydrostatic pressure within the interstitial space opposes this movement to some degree. At the venous end of the capillary, the osmotic force of plasma proteins draws fluid back into the capillary (see Figure 9.6).

ACTIVE TRANSPORT **Active transport** allows molecules to move across cell membranes and epithelial membranes against a concentration gradient. This movement requires energy (adenosine triphosphate, or ATP) and a carrier mechanism to maintain a higher concentration of a substance on one side of the membrane than on the other. The sodium–potassium pump is an important example of active transport (see Figure 9.7). High concentrations of potassium in intracellular fluids and of sodium in extracellular fluids are maintained because cells actively transport potassium from interstitial fluid (where the concentration of potassium is about 5 mEq/L) into intracellular fluid (where the potassium concentration is about 150 mEq/L).

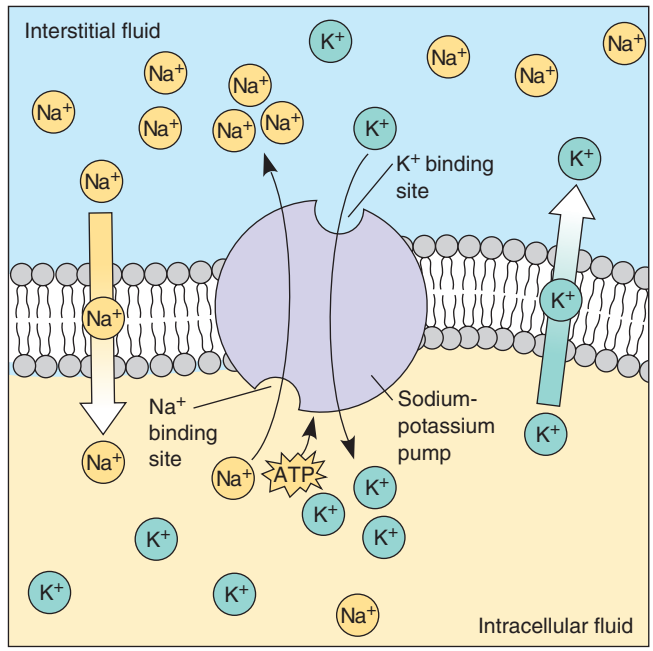


FIGURE 9.7 ■ The sodium–potassium pump. Sodium and potassium ions are moved across the cell membranes against their concentration gradients. This active transport process is fuelled by energy from ATP

Body fluid regulation

Homeostasis requires several regulatory mechanisms and processes to maintain the balance between fluid intake and excretion. These include thirst, the kidneys, the renin–angiotensin–aldosterone mechanism, antidiuretic hormone and atrial natriuretic peptide. These mechanisms affect the volume, distribution and composition of body fluids.

Thirst

Thirst is the primary regulator of water intake. Thirst plays an important role in maintaining fluid balance and preventing dehydration. The thirst centre, located in the brain, is stimulated when the blood volume drops because of water losses or when serum osmolality (solute concentration) increases (see Figure 9.8).

The thirst mechanism is highly effective in regulating extracellular sodium levels. Increased sodium in ECF increases

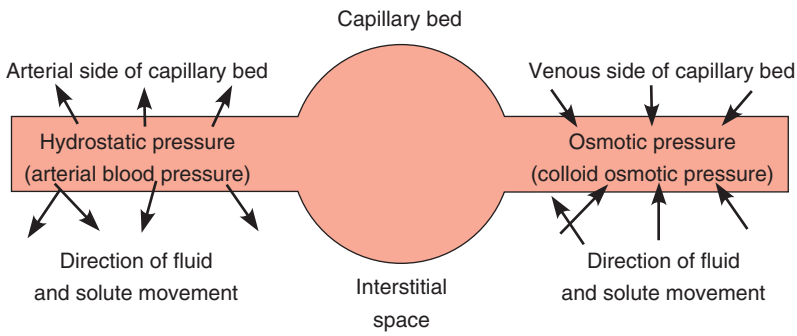


FIGURE 9.6 ■ Fluid balance between the intravascular and interstitial spaces is maintained in the capillary beds by a balance of filtration at the arterial end and osmotic draw at the venous end

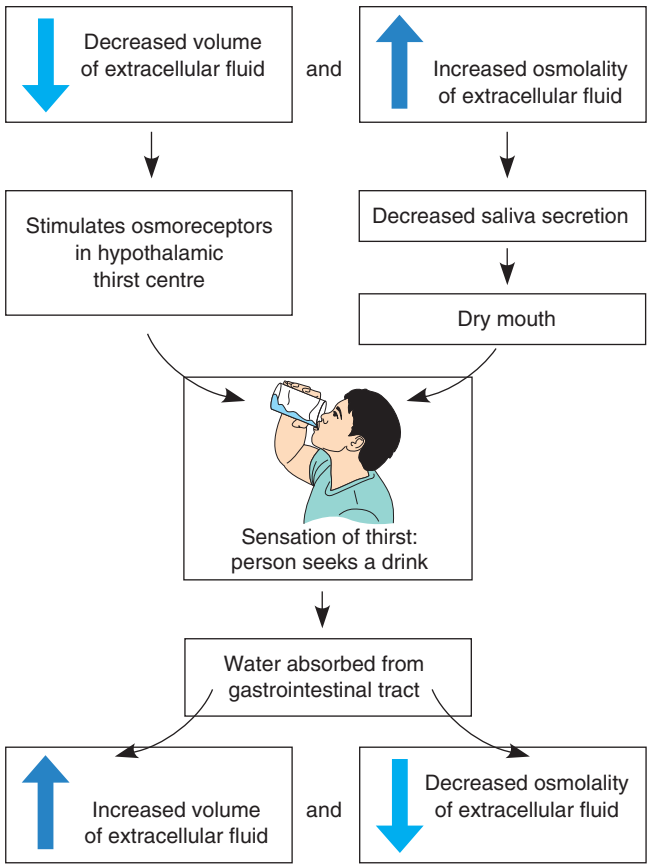


FIGURE 9.8 ■ Factors stimulating water intake through the thirst mechanism

serum osmolality, stimulating the thirst centre. Fluid intake in turn reduces the sodium concentration of ECF and lowers serum osmolality. Conversely, a drop in serum sodium and low serum osmolality inhibit the thirst centre.

CONSIDERATION FOR PRACTICE

The thirst mechanism declines with ageing, making older adults more vulnerable to dehydration and hyperosmolality (high serum osmolality). People with an altered level of consciousness or who are unable to respond to thirst are also at risk.

Kidneys

The kidneys are primarily responsible for regulating fluid volume and electrolyte balance in the body. They regulate the volume and osmolality of body fluids by controlling the excretion of water and electrolytes. In adults, about 170 L of plasma are filtered through the glomeruli every day. By selectively reabsorbing water and electrolytes, the kidneys maintain the volume and osmolality of body fluids. About 99% of the glomerular filtrate is reabsorbed, and only about 1500 mL of urine is produced over a 24-hour period.

Renin–angiotensin–aldosterone system

The renin–angiotensin–aldosterone system works to maintain intravascular fluid balance and blood pressure. A decrease in blood flow or blood pressure to the kidneys stimulates specialised receptors in the juxtaglomerular cells of the nephrons to produce renin, an enzyme. Renin converts angiotensinogen (a plasma protein) in the circulating blood into angiotensin I. Angiotensin I travels through the bloodstream to the lungs, where it is converted to angiotensin II by angiotensin-converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor; it raises the blood pressure. It also stimulates the thirst mechanism to promote fluid intake and acts directly on the kidneys, causing them to retain sodium and water. Angiotensin II stimulates the adrenal cortex to release aldosterone. Aldosterone promotes sodium and water retention in the distal nephron of the kidney, restoring blood volume (see Figure 9.9).

Antidiuretic hormone

Antidiuretic hormone (ADH), released by the posterior pituitary gland, regulates water excretion from the kidneys. Osmoreceptors in the hypothalamus respond to increases in serum osmolality and decreases in blood volume, stimulating ADH production and release. ADH acts on the distal tubules of the kidney, making them more permeable to water and thus increasing water reabsorption. With increased water reabsorption, urine output falls, blood volume is restored and serum osmolality drops as the water dilutes body fluids (see Figure 9.10).

In addition to decreased blood volume and increased serum osmolality, increased amounts of ADH are released in response to stress, pain, surgery and anaesthesia, some medications such as morphine and barbiturates, and mechanical ventilation. Its

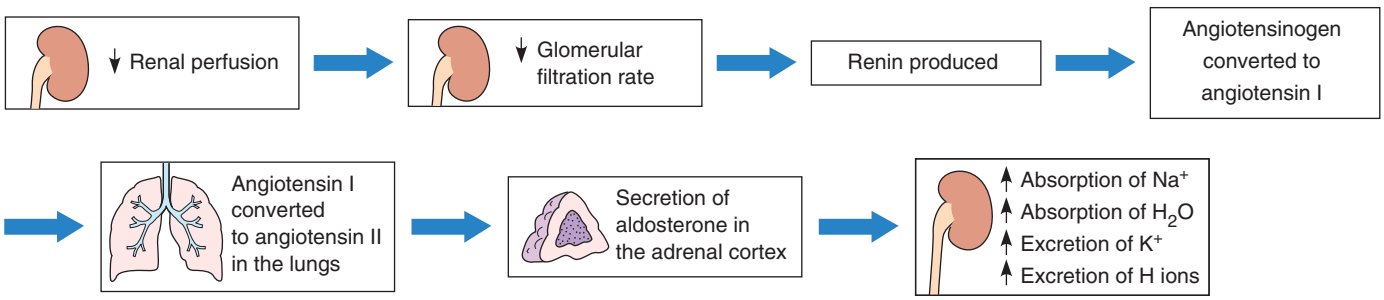


FIGURE 9.9 ■ The renin–angiotensin–aldosterone system. Decreased blood volume and renal perfusion set off a chain of reactions, leading to release of aldosterone from the adrenal cortex. Increased levels of aldosterone regulate serum K^+ and Na^+, blood pressure and water balance through effects on the kidney tubules

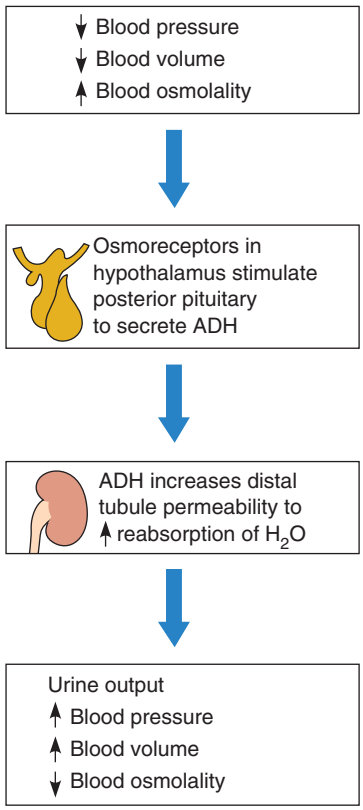


FIGURE 9.10 ■ Antidiuretic hormone (ADH) release and effect. Increased serum osmolality or a fall in blood volume stimulates the release of ADH from the posterior pituitary. ADH increases the permeability of distal tubules, promoting water reabsorption

release is inhibited by ethanol, medications such as phenytoin, as well as increased circulating blood volume and decreased serum osmolality.

Two disorders of ADH production illustrate the effect of ADH on water balance and urine output. First, diabetes insipidus is a condition characterised by deficient ADH production. The lack of ADH causes the distal tubules and collecting ducts of the kidney to be impermeable to water, so little water is reabsorbed into the bloodstream. As a result, copious, very dilute urine is excreted. Water loss leads to increased concentration of the plasma or increased serum osmolality. ADH is not released in response to the serum hyperosmolality, but the thirst mechanism is stimulated and the person drinks additional fluids, maintaining high urine output. In the other condition, the syndrome of inappropriate ADH secretion (SIADH), excess ADH is released. Increased water reabsorption causes increased fluid volume and scant, concentrated urine output. These diseases of the pituitary gland are discussed in Chapter 18.

Atrial natriuretic peptide

Atrial natriuretic peptide (ANP) is a hormone released by atrial muscle cells in response to distension from fluid overload. ANP affects several body systems, including the cardiovascular, renal, neural, gastrointestinal and endocrine systems, but it primarily affects the renin–angiotensin–aldosterone system. ANP opposes this system by inhibiting renin secretion and blocking the secretion and sodium-retaining effects of aldosterone. As a result, ANP promotes sodium wasting and diuresis (increased urine output) and causes vasodilation.

FLUID AND ELECTROLYTE IMBALANCES

THE PERSON WITH FLUID VOLUME DEFICIT

Fluid volume deficit (FVD) is a decrease in intravascular, interstitial and/or intracellular fluid in the body. Fluid volume deficits may be due to excessive fluid losses, insufficient fluid intake, or failure of regulatory mechanisms and fluid shifts within the body. FVD is a relatively common problem that may exist alone or in combination with other electrolyte or acid–base imbalances. The term **dehydration** refers to loss of water alone, even though it often is used interchangeably with fluid volume deficit.

Pathophysiology

The most common cause of fluid volume deficit is excessive loss of gastrointestinal fluids from vomiting, diarrhoea, gastrointestinal suctioning, intestinal fistulas and intestinal drainage. Other causes of fluid losses include excessive renal losses of water and sodium from diuretic therapy, renal disorders or endocrine disorders, inability to swallow fluids, oral trauma, water and sodium losses during sweating from excessive exercise or increased environmental temperature, haemorrhage and chronic abuse of laxatives and/or enemas. Older adults in particular are at risk of fluid volume deficit (see the 'Nursing care of the older adult' box below).

Fluid volume deficit can develop slowly or rapidly, depending on the type of fluid loss. Loss of extracellular fluid volume can lead to *hypovolaemia*, decreased circulating blood volume. Electrolytes often are lost along with fluid, resulting in an *isotonic fluid volume deficit*. When both water and electrolytes are lost, the serum sodium level remains normal, although levels of other electrolytes such as potassium may fall. Fluid is drawn into the vascular compartment from the interstitial spaces as the body attempts to maintain tissue perfusion. This eventually depletes fluid in the intracellular compartment as well.

Hypovolaemia stimulates regulatory mechanisms to maintain circulation. The sympathetic nervous system is stimulated, as is the thirst mechanism. ADH and aldosterone are released, prompting sodium and water retention by the kidneys. Severe fluid loss can lead to cardiovascular collapse.

NURSING CARE OF THE OLDER ADULT Fluid volume deficit

Changes in the normal ageing process affect homeostasis in several ways. In older adults, the percentage of total body water is about 10% lower than in younger or middle-aged adults, and thus they have less body reserve. Lean muscle mass is lower in older adults, and the percentage of body fat is higher; as a result, water accounts for about 50% of the total body weight (TBW) of an older man and about 45% of the TBW of an older woman. Sodium and water regulation become less efficient with ageing. Renal blood flow and glomerular filtration decline with ageing; the kidneys are less able to effectively concentrate the urine and conserve sodium and water. The perception of thirst decreases, interfering with the thirst mechanism. Consequently, the older adult may become dehydrated without being aware of the need to increase fluid intake.

Undetected fever in older adults can increase the total body need for water with every degree of temperature. Dehydration can cause a fever and further compound dehydration in the older adult. Older adults who have self-care deficits, or who are confused, depressed, tube fed, on bed rest or taking medications (such as sedatives, tranquillisers, diuretics and laxatives) are at greatest risk for fluid volume imbalance. Older adults without air-conditioning are at risk during extremely hot weather. In addition, functional changes and illnesses can affect fluid balance. For example, fear of incontinence can lead to self-limiting of fluid intake; physical disabilities associated with age-related illnesses, such as arthritis or stroke, may limit access to fluids; and cognitive impairments can interfere with recognition of thirst and the ability to respond to it.

Manifestations of fluid volume deficit may be more difficult to recognise in the older adult. A change in mental status, memory or attention may be an early manifestation. Skin turgor is less reliable as an indicator of dehydration, although assessing turgor over the sternum or on the inner aspect of the thigh may be more effective. Dry oral mucous membranes, increased tongue furrows, subnormal temperature, tachycardia and a pinched facial expression are also indicative of dehydration. Orthostatic vital signs may not demonstrate typical changes in the dehydrated older adult.

Two other types of fluid volume deficit—hypovolaemic fluid volume deficit and hypertonic fluid volume deficit—are discussed as effects of sodium imbalance in that section of this chapter.

Third spacing

Third spacing is a shift of fluid from the vascular space into an area where it is not available to support normal physiological processes. Fluid may be sequestered in the abdomen or bowel, or in such other actual or potential body spaces as the pleural or peritoneal spaces. Fluid may also become trapped within soft tissues following trauma or burns. The trapped fluid is unavailable to support cardiovascular or renal function; therefore, it represents a volume loss.

Increased vascular permeability or decreased protein levels can trigger third spacing (Longo et al., 2012). Stress hormones released in response to tissue trauma or sepsis (catecholamines in particular) promote redistribution of blood to vital organs (heart and brain). Renal blood flow falls, stimulating the renin–angiotensin–aldosterone system. This promotes sodium and water retention to maintain intravascular volume. The blood vessel and tissue damage caused by surgery stimulates the release of inflammatory mediators such as histamine and prostaglandins. These substances lead to local vasodilation and increased capillary permeability, allowing fluid to accumulate in interstitial tissues.

Assessing the extent of FVD resulting from third spacing is difficult. It may not be reflected by changes in weight or intake-and-output records and it may not become apparent until after organ malfunction occurs (Metheny, 2012). Delays in recognition and treatment can lead to irreversible shock and multiorgan system failure (Perrin, 2012).

Manifestations

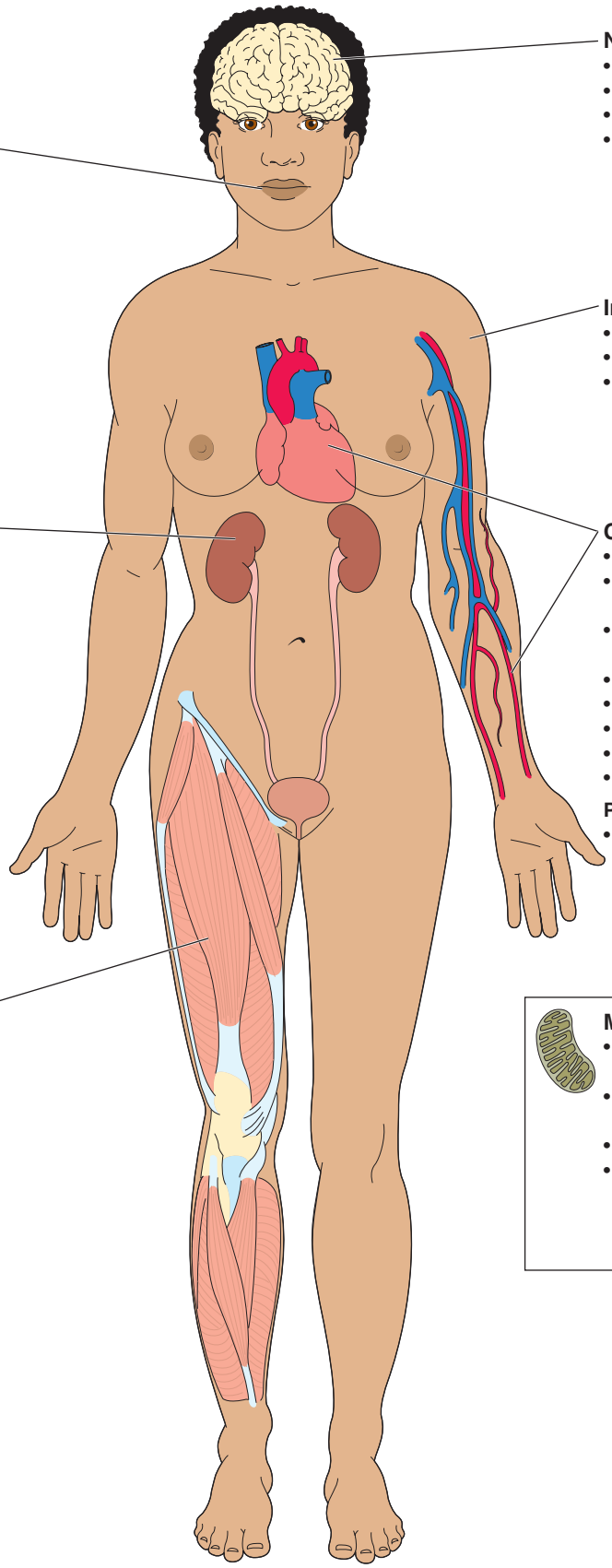
With a rapid fluid loss (such as with haemorrhage or uncontrolled vomiting), manifestations of hypovolaemia develop rapidly. When the loss of fluid occurs more gradually, the person's fluid volume may be very low before symptoms develop. The *multisystem effects of fluid volume deficit* are illustrated on the following page.

Rapid weight loss is a good indicator of fluid volume deficit. Each litre of body fluid weighs about 1 kg. The severity of the fluid volume deficit can be estimated by the percentage of rapid weight loss: a loss of 2% to 5% of total body weight represents a mild FVD; 6% to 9%, moderate FVD; and 10% or greater, severe FVD (Metheny, 2012).

Loss of interstitial fluid causes skin turgor to diminish. When pinched, the skin of a person with FVD remains elevated. Loss of skin elasticity with ageing makes this assessment finding less accurate in older adults. Tongue turgor is not generally affected by age; therefore, assessing the size, dryness and longitudinal furrows of the tongue may be a more accurate indicator of FVD.

Postural or orthostatic hypotension is a sign of hypovolaemia. A drop of more than 15 mmHg in systolic blood pressure when changing from a lying to a standing position often indicates loss of intravascular volume. Venous pressure falls as well, causing flat neck veins, even when the person is recumbent. Loss of intravascular fluid causes the haematocrit to increase. Compensatory mechanisms to conserve water and sodium and maintain circulation account for many of the manifestations of fluid volume deficit, such as tachycardia; pale, cool skin (vasoconstriction); and decreased urine output. The specific gravity of urine increases as water is reabsorbed in the tubules. Table 9.3 compares assessment findings for fluid deficit and fluid excess.

MULTISYSTEM EFFECTS OF FLUID VOLUME DEFICIT (FVD)



Neurological
- Altered mental status
- Anxiety, restlessness
- Diminished alertness/cognition
- Possible coma (severe FVD)

Mucous membranes
- Dry; may be sticky
- ↓ tongue size, longitudinal furrows ↑

Integumentary
- Diminished skin turgor
- Dry skin
- Pale, cool extremities

Urinary
- ↓ urine output
- Oliguria (severe FVD)
- ↑ urine-specific gravity

Cardiovascular
- Tachycardia
- Orthostatic hypotension (moderate FVD)
- Falling systolic/diastolic pressure (severe FVD)
- Flat neck veins
- ↓ venous filling
- ↓ pulse volume
- ↓ capillary refill
- ↑ haematocrit

Potential complication
- Hypovolaemic shock

Musculoskeletal
- Fatigue

Metabolic processes
- ↓ body temperature (isotonic FVD)
- ↑ body temperature (dehydration)
- Thirst
- Weight loss
  - 2–5% mild FVD
  - 6–9% moderate FVD
  - >10% severe FVD

TABLE 9.3 Comparison of assessment findings in a person with fluid imbalance

ASSESSMENT	FLUID DEFICIT	FLUID EXCESS
Blood pressure	Decreased systolic Postural hypotension	Increased
Heart rate	Increased	Increased
Pulse amplitude	Decreased	Increased
Respirations	Normal	Moist crackles Wheezes
Jugular vein	Flat	Distended
Oedema	Rare	Dependent
Skin turgor	Loose, poor turgor	Taut
Output	Low, concentrated	May be low or normal
Urine-specific gravity	High	Low
Weight	Loss	Gain

INTERPROFESSIONAL CARE

The primary goals of care related to fluid volume deficit are to prevent deficits in people at risk and to correct deficits and their underlying causes. Depending on the acuity of the imbalance, treatment may include replacement of fluids and electrolytes by the intravenous, oral or enteral routes. When possible, the oral or enteral route is preferred for administering fluids. In acute situations, however, intravenous fluid administration is necessary.

Diagnosis

Laboratory and diagnostic tests may be ordered when fluid volume deficit is suspected. Such tests measure:

- *Serum electrolytes*. In an isotonic fluid deficit, sodium levels are within normal limits; when the loss is water only, sodium levels are high. Decreases in potassium are common.
- *Serum osmolality*. Measurement of serum osmolality helps to differentiate isotonic fluid loss from water loss. With water loss, osmolality is high; it may be within normal limits with an isotonic fluid loss.
- *Haemoglobin and haematocrit*. The haematocrit often is elevated due to loss of intravascular volume and haemoconcentration.
- *Urine-specific gravity and osmolality*. As the kidneys conserve water, both the specific gravity and osmolality of urine increase.
- *Haemodynamic pressures*. The mean arterial pressure (MAP), *central venous pressure* (CVP), right atrial pressure (RAP) and pulmonary artery wedge pressure (PAWP) are decreased in severe FVD (Perrin, 2012). The technique for measuring CVP is outlined in Box 9.1.

Fluid management

Oral rehydration is the safest and most effective treatment for fluid volume deficit in alert people who are able to take oral fluids. Adults require a minimum of 1500 mL of fluid per day or approximately 30 mL per kg of body weight (ideal body weight is used to calculate fluid requirements for the person who is obese) for maintenance. Fluids are replaced gradually, particularly in older adults, to prevent rapid rehydration of the cells. In general, fluid deficits are replaced at a rate of approximately 30% to 50% of the deficit per 24 hours.

For mild fluid deficits in which the loss of electrolytes has been minimal (e.g. moderate exercise in warm weather), water alone may be used for fluid replacement. When the fluid deficit is more severe and when electrolytes have also been lost (e.g. FVD due to vomiting and/or diarrhoea, strenuous exercise for longer than an hour or two), a carbohydrate/electrolyte solution such as a sports drink, ginger ale or a rehydrating solution is more appropriate. These solutions provide sodium, potassium, chloride and kilojoules to help meet metabolic needs.

INTRAVENOUS THERAPY When the fluid deficit is severe or the person is unable to ingest fluids, the intravenous route is used to administer replacement fluids. Table 9.4 describes the types, tonicity and uses of commonly administered intravenous fluids. Isotonic electrolyte solutions (0.9% NaCl or Ringer's solution) are used to expand plasma volume in a hypotensive person or to replace abnormal losses, which are usually isotonic in nature. Normal saline (0.9% NaCl) tends to remain in the vascular compartment, increasing blood volume. When administered rapidly, however, this solution can precipitate acid–base imbalances, so balanced electrolyte solutions such as lactated Ringer's solution are preferred to expand plasma volume.

Five per cent dextrose in water (D_5W) or 0.45% NaCl (one-half normal saline or 1/2 NS) is given to provide water to treat total body water deficits. D_5W is isotonic (similar in tonicity to the plasma) when administered and thus does not provoke haemolysis of red blood cells. The dextrose is metabolised to carbon dioxide and water, leaving free water available for tissue needs. Hypotonic saline solution (0.45% NaCl with or without added electrolytes) or 5% dextrose in 0.45% sodium chloride (D_5 1/2 NS) are used as maintenance solutions (Metheny, 2012). These solutions provide additional electrolytes such as potassium, a buffer (lactate or acetate) as needed and water. When dextrose is added, they also provide a minimal number of kilojoules.

FLUID CHALLENGE A fluid challenge, the rapid administration of a designated amount of intravenous fluid, may be performed to evaluate fluid volume when urine output is low and cardiac or renal function is questionable. A fluid challenge

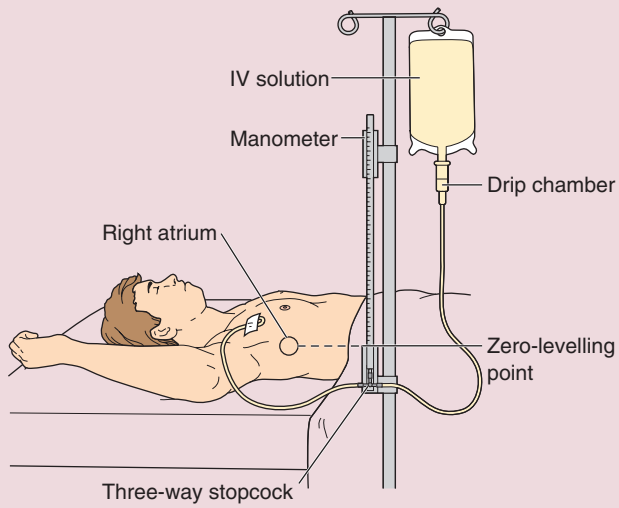
BOX 9.1 Measuring central venous pressure with a manometer

CVP is a haemodynamic monitoring method for evaluating fluid volume status. It measures mean right atrial pressure by means of a catheter. The CVP catheter is inserted by a doctor, most often at the person's bedside, into the antecubital, internal jugular or subclavian vein. Either a haemodynamic monitoring system (see Chapter 29) or a manual system may be used to measure the CVP. Nursing responsibilities in measuring CVP are as follows:

1. Explain to the person and their family what is being done.
2. Prior to the first measurement, take baseline vital signs and measure the level of the right atrium on the person's thorax. This is usually at the fourth intercostal space on the lateral chest wall, midway between the anterior and posterior chest. This site, called the *phlebostatic axis*, is marked and used as the reference point for all measurements.
3. If possible, place the bed in the same position for each reading, usually with the person supine and the head of the bed flat. Elevating the head of the bed to as much as 60 degrees usually does not affect the accuracy of the CVP reading in a person who is haemodynamically stable (Urden, Stacy & Lough, 2009).
4. Use a carpenter's level to check the level of the measuring device to make sure the transducer or the 0 on the manometer is level with the phlebostatic axis (see figure).
5. Remove any air bubbles in the line.
6. If using a manometer, turn the stopcock so that fluid flows into the manometer, filling it a few centimetres above the expected reading. Then turn the stopcock to open the line between the manometer and the person. The fluid level will fall and then reach a point at which it fluctuates with the person's respirations. This point is recorded as the CVP.
7. After the measurement is taken, turn the stopcock so that the fluid can again flow from the fluid source to the person.

Normal values

When CVP is measured using a manometer, normal values range from 2 to 8 cm water. With a haemodynamic monitoring system, the normal CVP range is 2 to 5 mmHg. A low CVP indicates inadequate venous return from fluid deficit and hypovolaemia or due to peripheral vasodilation. A high CVP indicates fluid overload, cardiac problems that decrease cardiac contractility or pulmonary disorders that increase pulmonary vascular resistance.



helps prevent fluid volume overload resulting from intravenous fluid therapy when cardiac or renal function is compromised. Nursing responsibilities for a fluid challenge are as follows:

- Obtain and document baseline vital signs, breath sounds, urine output and mental status.
- Administer (by IV infusion) an initial fluid volume of 200 to 300 mL over 5 to 10 minutes.
- Re-evaluate baseline data at the end of the 5- or 10-minute infusion period.
- Administer additional fluid until a specified volume is infused or the desired haemodynamic parameters are achieved.

Nursing care

Nurses are responsible for identifying if a person is at risk of fluid volume deficit, initiating and carrying out measures to prevent and treat fluid volume deficit, and monitoring the effects of therapy.

Health promotion

Health promotion activities focus on teaching people to prevent fluid volume deficit. Discuss the importance of maintaining adequate fluid intake, particularly when exercising and during hot weather. Advise people to use commercial sports drinks to replace both water and electrolytes when exercising during warm weather. Instruct people to maintain fluid intake when ill, particularly during periods of fever or when diarrhoea is a problem.

Discuss the increased risk of fluid volume deficit with older adults and provide information about prevention. Teach older adults (and their caregivers) that thirst decreases with ageing and urge them to maintain a regular fluid intake of about 1500 mL per day, regardless of perception of thirst.

Carefully monitor a person at risk of abnormal fluid losses through routes such as vomiting, diarrhoea, nasogastric suction, increased urine output, fever or wounds. Monitor fluid intake in a person with a decreased level of consciousness, disorientation, nausea and anorexia, and physical limitations.

Assessment

Collect assessment data through the health history interview and physical examination.

TABLE 9.4 Commonly administered intravenous fluids

	FLUID AND TONICITY	USES
Dextrose in water solutions	5% dextrose in water (D_5W)	Replaces water losses
	Isotonic	Provides free water necessary for cellular rehydration
		Lowers serum sodium in hypernatraemia
	10% dextrose in water ($D_{10}W$)	Provides free water
	Hypertonic	Provides nutrition (supplies 340 kcal/L)
	20% dextrose in water ($D_{20}W$)	Supplies 680 kcal/L
	Hypertonic	May cause diuresis
	50% dextrose in water ($D_{50}W$)	Supplies 1700 kcal/L
	Hypertonic	Used to correct hypoglycaemia
Saline solutions	0.45% sodium chloride	Provides free water to replace hypotonic fluid losses
	Hypotonic	Maintains levels of plasma sodium and chloride
	0.9% sodium chloride	Expands intravascular volume
	Isotonic	Replaces water lost from extracellular fluid
		Used with blood transfusions
		Replaces large sodium losses (as from burns)
	3% sodium chloride	Corrects serious sodium depletion
	Hypertonic	
Combined dextrose and saline solution	5% dextrose and 0.45% sodium chloride	Provides free water
	Isotonic	Provides sodium chloride
		Maintenance fluid of choice if there are no electrolyte imbalances
Multiple electrolyte solutions	Hartmann's solution	Expands the intracellular fluid
	Isotonic (electrolyte concentrations of sodium, potassium, chloride and calcium are similar to plasma levels)	Replaces extracellular fluid losses
	Lactated Ringer's solution Isotonic (similar in composition of electrolytes to plasma but does not contain magnesium)	Replaces fluid losses from burns and the lower gastrointestinal tract Fluid of choice for acute blood loss

- *Health history*: risk factors such as medications, acute or chronic renal or endocrine disease; precipitating factors such as hot weather, extensive exercise, lack of access to fluids, recent illness (especially if accompanied by fever, vomiting and/or diarrhoea); onset and duration of symptoms.
- *Physical assessment*: weight; vital signs including orthostatic blood pressure and pulse; peripheral pulses and capillary refill; jugular neck vein distension; skin colour, temperature, turgor; level of consciousness and mentation; urine output. See Box 9.2 for physical assessment changes in the older adult.
- *Diagnostic tests*: serum osmolality and electrolytes, haemoglobin and haematocrit (expect values to fall with rehydration), urine-specific gravity and osmolality, central venous pressure readings.

BOX 9.2 Assessing older adults: fluid volume deficit

With ageing, the elasticity of skin decreases. As a result, turgor diminishes, even in the well-hydrated older adult. This makes skin turgor less reliable when assessing for fluid volume deficit. In addition, some older adults experience postural hypotension, even when well hydrated. Allow the older adult to stand quietly for a full minute before rechecking blood pressure and pulse when measuring orthostatic vital signs.

Nursing diagnoses and interventions

The focus for nursing diagnoses and interventions for the person with *Fluid volume deficit* is on managing the effects of the deficit and preventing complications.

Deficient fluid volume

A person with a fluid volume deficit due to abnormal losses, inadequate intake or impaired fluid regulation requires close monitoring as well as immediate and ongoing fluid replacement.

- Assess intake and output accurately, monitoring fluid balance. In acute situations, hourly intake and output may be indicated. *Urine output should be 30 to 60 mL per hour (unless renal failure is present). Urine output of less than 30 mL per hour indicates inadequate renal perfusion and an increased risk of acute renal failure and inadequate tissue perfusion* (Perrin, 2012).

CONSIDERATION FOR PRACTICE

Report a urine output of less than 30 mL per hour to the attending doctor.

- Assess vital signs, CVP and peripheral pulse volume at least every 4 hours. *Hypotension, tachycardia, low CVP and weak, easily obliterated peripheral pulses indicate hypovolaemia.*
- Weigh daily under standard conditions (time of day, clothing and scale). *In most instances (except third spacing), changes in weight accurately reflect fluid balance.* (See the 'Translation to practice' box below.)
- Administer and monitor the intake of oral fluids as prescribed. Identify beverage preferences and provide these on a schedule. *Oral fluid replacement is preferred when the person is able to drink and retain fluids.*
- Administer IV as prescribed using an electronic infusion pump. Monitor for indicators of fluid overload if rapid fluid replacement is ordered: dyspnoea, tachypnoea, tachycardia, increased CVP, jugular vein distension and oedema. *Rapid fluid replacement may lead to hypervolaemia, resulting in pulmonary oedema and cardiac failure, particularly in the person with compromised cardiac and renal function.*
- Monitor laboratory values: electrolytes, serum osmolality and haematocrit. *Rehydration may lead to changes in serum electrolytes, osmolality and haematocrit. In some cases, electrolyte replacement may be necessary during rehydration.*

Ineffective tissue perfusion

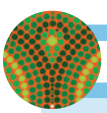
A fluid volume deficit can lead to decreased perfusion of renal, cerebral and peripheral tissues. Inadequate renal perfusion can lead to acute renal failure. Decreased cerebral perfusion leads to changes in mental status and cognitive function, causing restlessness, anxiety, agitation, excitability, confusion, vertigo, fainting and weakness.

- Monitor for changes in level of consciousness and mental status. *Restlessness, anxiety, confusion and agitation may indicate inadequate cerebral blood flow and circulatory collapse.*
- Monitor serum urea and creatinine and cardiac enzymes, reporting elevated levels to the physician. *Elevated levels may indicate impaired renal function or cardiac perfusion related to circulatory failure.*
- Turn at least every 2 hours. Provide good skin care and monitor for evidence of skin or tissue breakdown. *Impaired circulation to peripheral tissues increases the risk of skin breakdown. Turn frequently to relieve pressure over bony prominences. Keep skin clean, dry and moisturised to help maintain integrity.*

Risk of injury

The person with fluid volume deficit is at risk of injury because of dizziness and loss of balance resulting from decreased cerebral perfusion secondary to hypovolaemia.

- Institute safety precautions, including keeping the bed in a low position, using side rails as needed, and slowly raising



TRANSLATION TO PRACTICE Evidence-based practice for determining fluid needs for the person in long-term care

Residents of long-term care facilities are at significant risk of developing fluid volume deficit. Most are elderly, many have some degree of dementia, and a significant number are dependent on caregivers to provide fluids. Dehydration, when it occurs, can be a sentinel health event leading to serious and potentially life-threatening secondary problems (Gaspar, 2011).

Various standards for determining the amount of fluid a resident requires have been developed. These standards vary in complexity from a simple 30 mL fluid per kilogram of body weight to a formula that uses body surface area to determine fluid needs. A retrospective study by Gaspar (2011) compared four different formulas, ultimately recommending a formula based on the height and weight of the resident to determine fluid intake.

IMPLICATIONS FOR NURSING

As noted at the beginning of this chapter, the percentage of total body water varies with age and the amount of lean body tissue to adipose tissue. Likewise, fluid requirements of residents in long-term care facilities vary, necessitating attention to the needs of the individual. Furthermore, caregivers are more likely to attend to an individualised plan for a resident's fluid intake than to a generalised recommendation to 'push fluids'. This plan should include not only the target amount of daily fluid intake but also residents' preferences for the type, temperature and timing of fluid intake.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Why are older adults more vulnerable to dehydration and fluid volume deficit than younger adults?
2. Identify factors in long-term care settings that increase the risk for fluid volume deficit. Consider the setting, residents and caregivers.
3. Develop a teaching plan about resident fluid intake for caregivers in a long-term care facility.

the person from supine to sitting or sitting to standing position. Using safety precautions and allowing time for the blood pressure to adjust to position changes will reduce the risk of injury.

- Teach the person and their family members how to reduce orthostatic hypotension:
  a. Move from one position to another in stages; for example, raise the head of the bed before sitting up, and sit for a few minutes before standing.
  b. Avoid prolonged standing.
  c. Rest in a recliner rather than in bed during the day.
  d. Use assistive devices to pick up objects from the floor, rather than stooping.

Teaching measures to reduce orthostatic hypotension reduces the person's risk of injury. Prolonged bed rest increases skeletal muscle weakness and decreases venous tone, contributing to postural hypotension. Prolonged standing allows blood to pool in the legs, reducing venous return and cardiac output.

Community-based care

Depending on the severity of the fluid volume deficit, the person may be managed in the home or residential facility or may be admitted to an acute care facility. Assess the person's understanding of the cause of the deficit and the fluids necessary for providing replacement. Address the following topics when preparing the person and their family for home care:

- The importance of maintaining adequate fluid intake (at least 1500 mL per day; more if extra fluid is being lost through perspiration, fever or diarrhoea).
- Manifestations of fluid imbalance and how to monitor fluid balance.
- How to prevent fluid deficit:
  - Avoid exercising during extreme heat.
  - Increase fluid intake during hot weather.
  - If vomiting, take small frequent amounts of ice chips or clear liquids, such as weak tea, flat cola or ginger ale.
  - Reduce intake of coffee, tea and alcohol, which increase urine output and can cause fluid loss.
- Replacement of fluids lost through diarrhoea with fruit juices or bouillon, rather than large amounts of tap water.
- Alternative sources of fluid (such as gelatin, frozen juices or ice-cream) for effective replacement of lost fluids.

FLUID VOLUME EXCESS

Fluid volume excess (FVE) results when both water and sodium are retained in the body. Fluid volume excess may be caused by fluid overload (excess water and sodium intake) or by impairment of the mechanisms that maintain homeostasis. The excess fluid can lead to excess intravascular fluid (hypervolaemia) and excess interstitial fluid (**oedema**).

Pathophysiology

Fluid volume excess usually results from conditions that cause retention of both sodium and water. These conditions include heart failure, cirrhosis of the liver, renal failure, adrenal gland disorders, corticosteroid administration and stress conditions causing the release of ADH and aldosterone. Other causes include an excessive intake of sodium-containing foods, drugs that cause sodium retention and the administration of excess amounts of sodium-containing intravenous fluids (such as 0.9% NaCl or Ringer's solution). This *iatrogenic* (induced by the effects of treatment) cause of fluid volume excess primarily affects a person with impaired regulatory mechanisms.

In fluid volume excess, both water and sodium are gained in about the same proportions as normally exists in extracellular fluid. The total body sodium content is increased, which in turn causes an increase in total body water. Because the increase in sodium and water is isotonic, the serum sodium and osmolality remain normal and the excess fluid remains in the extracellular space.

Stress responses activated before, during and immediately after surgery commonly lead to increased ADH and aldosterone levels, leading to sodium and water retention. In the immediate postoperative period, however, this additional fluid tends to be sequestered in interstitial tissues and is unavailable to support cardiovascular and renal function (see the 'Third spacing' section earlier in this chapter.) This sequestered fluid is reabsorbed into the circulation within about 48 to 72 hours after surgery. Although it is then normally eliminated through a process of diuresis, a person with heart or kidney failure is at risk of developing fluid overload.

Manifestations and complications

Excess extracellular fluid leads to hypervolaemia and circulatory overload. Excess fluid in the interstitial space causes peripheral or generalised oedema. Manifestations of fluid volume excess with related pathophysiology are described in Table 9.5.

Heart failure is not only a potential cause of fluid volume excess, but also a potential complication of the condition if the heart is unable to increase its workload to handle the excess blood volume. Severe fluid overload and heart failure can lead to pulmonary oedema, a medical emergency. See Chapter 30 for more information about heart failure and pulmonary oedema.

INTERPROFESSIONAL CARE

Managing fluid volume excess focuses on prevention in a person at risk, treating its manifestations and correcting the underlying cause. Management includes limiting sodium and water intake and administering diuretics.

Diagnosis

The following laboratory tests may be ordered.

- *Serum electrolytes* and *serum osmolality* are measured. Serum sodium and osmolality usually remain within normal limits.
- *Serum haematocrit* and *haemoglobin* often are decreased due to plasma dilution from excess extracellular fluid.

TABLE 9.5 Manifestations of fluid volume excess

MANIFESTATIONS	RELATED PATHOPHYSIOLOGY
Peripheral oedema, or if severe, **anasarca** (severe, generalised oedema)	Excess fluid in the interstitial spaces, usually resulting from conditions that cause retention of both sodium and water (e.g. heart failure, renal failure and stress responses causing the release of ADH and aldosterone, such as surgery)
Full bounding pulse, distended neck and peripheral veins, increased central venous and right atrial pressures, cough, **dyspnoea** (laboured or difficult breathing), **orthopnoea** (difficult breathing when supine)	Circulatory overload from increased water and sodium retention
Dyspnoea at rest	Mobilisation (reabsorption) of fluid from peripheral tissues increases circulatory fluid volume
Tachycardia and hypertension	Increased circulatory fluid volume
Reduced oxygen saturation	As fluid increases in the interstitial spaces and alveoli, gas exchange is impaired, leading to hypoxia and hypercapnia
Moist crackles on auscultation of the lungs, pulmonary oedema	Excess fluid in pulmonary interstitial spaces and alveoli
Increased urine output (**polyuria**)	Increased circulatory volume and increased perfusion of the renal arteries increases amount of filtrate produced in glomerulus
Ascites (excess fluid in the peritoneal cavity)	Increased filtration pressure due to hypervolaemia
Decreased haematocrit and BUN	Dilutional effect of increased circulatory volume
Altered mental status and anxiety	Pressure on the cerebral cortex from cerebral hypertension and oedema causes decreased oxygenation (hypoxia) of neurons
Pulmonary oedema	Elevation of left-sided filling pressures from increased circulatory volume and heart failure increase pressures in pulmonary vascular system

- Additional tests of *renal* and *liver function* (such as serum urea and creatinine and liver enzymes) may be ordered to help determine the cause of fluid volume excess if it is unclear.

Medications

Diuretics are commonly used to treat fluid volume excess. They inhibit sodium and water reabsorption, increasing urine output. The three major classes of diuretics, each of which acts on a different part of the kidney tubule, are as follows:

1. Loop diuretics act in the ascending loop of Henle.
2. Thiazide-type diuretics act on the distal convoluted tubule.
3. Potassium-sparing diuretics affect the distal nephron.

The nursing implications for diuretics are outlined in the 'Medication administration' box below.

Treatments

FLUID MANAGEMENT Fluid intake may be restricted in a person who has fluid volume excess. The amount of fluid allowed per day is prescribed by the primary attending doctor. All fluid intake must be calculated, including meals and that used to administer medications orally or intravenously. Box 9.3 provides guidelines for a person with a fluid restriction.

DIETARY MANAGEMENT Because sodium retention is a primary cause of fluid volume excess, a sodium-restricted diet is often prescribed. This is particularly important for people with a history of heart or renal failure (Baraz et al., 2010; Son et al., 2011). Australian adults typically consume 2.5 to 3 g of sodium every day; recommended sodium intake is 500 to 2300 mg per day (Food Standards Australia New Zealand, 2015). The primary dietary sources of sodium are the salt shaker, processed foods and foods themselves (see Box 9.4).

A mild sodium restriction can be achieved by instructing the person and primary food preparer in the household to reduce the amount of salt in recipes by half, avoid using the salt shaker during meals and avoid foods that contain high levels of sodium (either naturally or because of processing).

BOX 9.3 Fluid restriction guidelines

- Subtract requisite fluids (e.g. ordered IV fluids, fluid used to dilute IV medications) from total daily allowance.
- Divide remaining fluid allowance:
  - day shift: 50% of total
  - evening shift: 25% to 33% of total
  - night shift: remainder.
- Explain the fluid restriction to the person and family members.
- Identify preferred fluids and intake pattern.
- Place allowed amounts of fluid in small glasses (gives perception of a full glass).
- Offer ice chips (when melted, ice chips are approximately half the frozen volume).
- Provide frequent mouth care.
- Provide sugarless chewing gum (if allowed) to reduce thirst sensation.

MEDICATION ADMINISTRATION Diuretics for fluid volume excess

Diuretics increase urinary excretion of water and sodium. They are categorised into three major groups: loop diuretics, thiazide and thiazide-like diuretics, and potassium-sparing diuretics. Diuretics are used to enhance renal function and to treat vascular fluid overload and oedema. Common side effects include orthostatic hypotension, dehydration, electrolyte imbalance and possible hyperglycaemia. Diuretics should be used with caution in the older adult. Examples of each major type follow.

LOOP DIURETICS

Frusemide (Lasix)
Ethacrynic acid
Bumetanide

Loop diuretics inhibit sodium and chloride reabsorption in the ascending loop of Henle. (See Chapter 27 for the anatomy of the kidneys.) As a result, loop diuretics promote the excretion of sodium, chloride, potassium and water.

THIAZIDE AND THIAZIDE-LIKE DIURETICS

Bendroflumethiazide
Chlorothiazide
Hydrochlorothiazide
Chlorthalidone
Indapamide

Thiazide and thiazide-like diuretics promote the excretion of sodium, chloride, potassium and water by decreasing absorption in the distal tubule.

POTASSIUM-SPARING DIURETICS

Spironolactone (Aldactone)
Amiloride HCl (Midamor)

Potassium-sparing diuretics promote excretion of sodium and water by inhibiting sodium–potassium exchange in the distal tubule.

HEALTH EDUCATION FOR THE PERSON AND THEIR FAMILY

- The drugs will increase the amount and frequency of urination.
- The drugs must be taken even when you feel well.
- Take the drugs in the morning and afternoon to avoid having to get up at night to urinate.
- Change position slowly to avoid dizziness.
- Report the following to your attending doctor: dizziness; trouble breathing; or swelling of face, hands or feet.
- Weigh yourself every day and report sudden gains or losses.
- Avoid using the salt shaker when eating.
- If the drug increases potassium loss, eat foods high in potassium, such as orange juice and bananas.
- Do not use salt substitutes if you are taking a potassium-sparing diuretic.

BOX 9.4 Foods high in sodium

Processed meat and fish
- Bacon
- Sausage
- Luncheon meat and other cold cuts
- Smoked fish

Selected dairy products
- Buttermilk
- Cottage cheese
- Cheeses
- Ice-cream

Processed grains
- Cracker biscuits
- Most dry cereals

Most canned goods
- Meats
- Vegetables
- Soups

Snack foods
- Salted popcorn
- Nuts
- Potato chips/pretzels
- Gelatin desserts

Condiments and food additives
- Barbecue sauce
- Saccharin
- Pickles
- Chilli sauce
- Soy sauce
- Meat tenderisers
- Salted margarine
- Worcestershire sauce
- Salad dressings

Naturally high in sodium
- Brains
- Oysters
- Kidneys
- Prawns
- Dried fruit
- Crab
- Spinach
- Lobster
- Carrots

In moderate and severely sodium-restricted diets, salt is avoided altogether, as are all foods containing significant amounts of sodium.

Nursing care

Nursing care focuses on preventing fluid volume excess in a person at risk and on managing problems resulting from its effects. See 'Nursing care plan: A person with fluid volume excess' below.

Health promotion

Health promotion related to fluid volume excess focuses on teaching preventive measures to a person who are at risk (e.g. people who have heart or kidney failure). Discuss the relationship between sodium intake and water retention. Provide guidelines for a low-sodium diet and teach the person to carefully read food labels to identify 'hidden' sodium, particularly in processed foods. Instruct the person at risk to weigh themselves on a regular basis, using the same scales, and to notify their primary attending doctor if they gain more than 2 kg in a week or less. Carefully monitor the person receiving intravenous fluids for signs of hypervolaemia. Reduce the flow rate and promptly report manifestations of fluid overload to the physician.

NURSING CARE PLAN A person with fluid volume excess

Dorothy Smith is a 45-year-old Aboriginal woman hospitalised with acute renal failure that developed as a result of acute glomerulonephritis. She is expected to recover, but she has very little urine output. Ms Smith is a single mother of two teenage sons. Until her illness, she was active in caring for her family, her career as a primary school teacher's aide and community activities.

ASSESSMENT

Mike Penning, Ms Smith's nurse, notes that she is in the oliguric phase of acute renal failure and that her urine output for the previous 24 hours is 250 mL; this low output has been constant for the past 8 days. She gained 0.45 kg in the past 24 hours. Laboratory test results from that morning are sodium, 155 mEq/L (normal 135 to 145 mEq/L); potassium, 5.3 mEq/L (normal 3.5 to 5.0 mEq/L); calcium, 7.6 mg/dL (normal 8.0 to 10.5 mg/dL) and urine-specific gravity 1.008 (normal 1.010 to 1.030). Ms Smith's serum creatinine and urea are high; however, her ABGs are within normal limits.

In his assessment of Ms Smith, Mike notes the following:

- BP 160/92; P 102, with obvious neck vein distension; R 28, with crackles and wheezes; head of bed elevated 30 degrees; T 37.0°C.
- Periorbital and sacral oedema present; 3+ pitting bilateral pedal oedema; skin cool, pale and shiny.
- Alert, oriented; responds appropriately to questions.
- States she is thirsty, slightly nauseated and extremely tired.

Ms Smith is receiving intravenous frusemide and is on a 24-hour fluid restriction of 500 mL plus the previous day's urine output to manage her fluid volume excess.

DIAGNOSES

- *Excess fluid volume* related to acute renal failure
- *Risk of impaired skin integrity* related to fluid retention and oedema
- *Risk of impaired gas exchange* related to pulmonary congestion
- *Activity intolerance* related to fluid volume excess, fatigue and weakness

PLANNING

- Advise the person that they need to be weighed twice daily, at 0600 and 1800 hours to monitor fluid balance daily.
- Explain to the person that the nurses will be monitoring vital signs and SaO_2 every 4 hours and the reasons for monitoring vital signs.
- Explain the purpose for the person being placed on restricted fluids and suggested ways to maintain the fluid restriction.
- Explain to the person that all fluids consumed and all urine output will need to be measured and documented on a fluid balance chart.
- Instruct and educate the person on the importance of oral hygiene and the effectiveness of moistened oral applicators to prevent mouth dryness.
- Advise the person of the importance to sit out of bed on a chair three times a day and of the necessity to call for assistance when ambulating or if dyspnoea is increasing.
- Explain the importance of keeping the head of the bed elevated 30 to 40 degrees and the importance of not staying in one position for a prolonged period of time.
- Attend to pressure area care as required to maintain skin integrity.

Expected outcomes

- Regain fluid balance, as evidenced by weight loss, decreasing oedema and normal vital signs.
- Experience decreased dyspnoea.
- Maintain intact skin and mucous membranes.
- Increase activity levels as prescribed.

IMPLEMENTATION

- Weigh the person at 0600 and 1800 daily and record the findings.
- Document and review the vital signs, fluid balance chart 4 hourly.
- Obtain, measure and document urine-specific gravity every 8 hours.
- Discuss and consult with the person about her compliance with the prescribed fluid restriction.
- Consult with the person about her ability to move and inspect her skin for any signs of pressure areas.
- Consult with the person and identify her ability to maintain oral hygiene. Provide extra assistance if required to maintain oral care every 2 to 4 hours.
- Consult with the person and identify if the elevation of the head of bed is reducing the dyspnoea.
- Observe and monitor the person's ability to sit out of bed and ambulate safely without increasing shortness of breath and fatigue.

EVALUATION

At the end of the shift, Mike evaluates the effectiveness of the plan of care and continues all diagnoses and interventions. Ms Smith has gained no weight and her urinary output during this shift is 170 mL. Her urine-specific gravity remains at 1.008. Her vital signs are unchanged, but her crackles and wheezes have decreased slightly. Her skin and mucous membranes are intact. Ms Smith tolerated the bedside chair without dyspnoea or fatigue.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the pathophysiological basis for Ms Smith's increased respiratory rate, blood pressure and pulse?
2. Explain how elevating the head of the bed 30 to 40 degrees facilitates respirations.
3. Suppose Ms Smith says, 'I would really like to have all my fluids at once instead of spreading them out.' How would you reply and why?
4. Outline a plan for teaching Ms Smith about diuretics.

REFLECTION ON THE NURSING PROCESS

1. As the Registered Nurse, how do you know if the education pertaining to fluid restriction has been effective?
2. Outline what you have learned from this case study that you will apply to your future practice.

Assessment

Collect assessment data through the health history interview and physical examination.

- *Health history*: risk factors such as medications, heart failure, acute or chronic renal or endocrine disease; precipitating factors such as a recent illness, change in diet or change in medications; recent weight gain; complaints of persistent cough, shortness of breath, swelling of feet and ankles, or difficulty sleeping when lying down.
- *Physical assessment*: weight; vital signs; peripheral pulses and capillary refill; jugular neck vein distension; oedema; lung sounds (crackles or wheezes), dyspnoea, cough and sputum; urine output; mental status.
- *Diagnostic tests*: monitor serum electrolytes and osmolality, haemoglobin and haematocrit, urine-specific gravity.

Nursing diagnoses and interventions

Nursing diagnoses and interventions for the person with fluid volume excess focus on the multisystem effects of the fluid overload.

Excess fluid volume

Nursing care for the person with excess fluid volume includes collaborative interventions such as administering diuretics and maintaining a fluid restriction, as well as monitoring the status and effects of the excess fluid volume. This is particularly critical in older adults because of the age-related decline in cardiac and renal compensatory responses.

- Assess vital signs, heart sounds, CVP and volume of peripheral arteries. *Hypervolaemia can cause hypertension, bounding peripheral pulses, a third heart sound (S_3) due to the volume of blood flow through the heart and high CVP readings.*
- Assess for the presence and extent of oedema, particularly in the lower extremities, the back, and sacral and periorbital areas. *Initially, oedema affects the dependent portions of the body—the lower extremities of the ambulatory person and the sacrum of the bedridden person. Periorbital oedema indicates more generalised oedema.*

CONSIDERATION FOR PRACTICE

Assess urine output hourly. Maintain accurate intake and output records. Note urine output of less than 30 mL per hour or a positive fluid balance on 24-hour total intake and output calculations. Heart failure and inadequate renal perfusion may result in decreased urine output and fluid retention.

- Obtain daily weights at the same time of day, using approximately the same clothing and balanced scales. *Daily weights are one of the most important gauges of fluid balance. Acute weight gain or loss represents fluid gain or loss. Weight gain of 2 kg is equivalent to 2 L of fluid gain.*
- Administer oral fluids cautiously, adhering to any prescribed fluid restriction. Discuss the restriction with the person and significant others, including the total volume allowed, the rationale, and the importance of reporting all fluid taken. *All sources of fluid intake, including ice chips, are recorded to avoid excess fluid intake.*
- Provide oral hygiene at least every 2 hours. *Oral hygiene contributes to the comfort of the person and keeps mucous membranes intact; it also helps to relieve thirst if fluids are restricted.*
- Teach the person and significant others about the sodium-restricted diet and emphasise the importance of checking before bringing foods to the person on this diet. *Excess sodium promotes water retention; a sodium-restricted diet is ordered to reduce water gain.*
- Administer prescribed diuretics as ordered, monitoring the person's response to therapy. *Loop or high-ceiling diuretics such as frusemide can lead to rapid fluid loss and signs of hypovolaemia and electrolyte imbalance.*
- Promptly report significant changes in serum electrolytes or osmolality or abnormal results of tests done to determine contributing factors to the fluid volume excess. *Gradual correction of serum electrolytes and osmolality is expected; however, aggressive diuretic therapy can lead to over-correction.*

Risk of impaired skin integrity

Tissue oedema decreases oxygen and nutrient delivery to the skin and subcutaneous tissues, increasing the risk of injury.

- Frequently assess skin, particularly in pressure areas and over bony prominences. *Skin breakdown can progress rapidly when circulation is impaired.*
- Reposition the person at least every 2 hours. Provide skin care with each position change. *Frequent position changes minimise tissue pressure and promote blood flow to tissues.*
- Provide an egg-crate mattress or alternating pressure mattress, foot cradle, heel protectors and other devices to reduce pressure on tissues. *These devices, which distribute pressure away from bony prominences, reduce the risk of skin breakdown.*

Risk of impaired gas exchange

With fluid volume excess, gas exchange may be impaired by oedema of pulmonary interstitial tissues. Acute pulmonary oedema is a serious and potentially life-threatening complication of pulmonary congestion.

- Auscultate lungs for presence or worsening of crackles and wheezes; auscultate heart for extra heart sounds. *Crackles and wheezes indicate pulmonary congestion and oedema. A gallop rhythm (S_3) may indicate diastolic overloading of the ventricles secondary to fluid volume excess.*
- Place in Fowler's position if dyspnoea or orthopnoea is present. *Fowler's position improves lung expansion by decreasing the pressure of abdominal contents on the diaphragm.*
- Monitor oxygen saturation levels and **arterial blood gases (ABGs)** for evidence of impaired gas exchange ($SaO_2 < 92\%$ to 95%; $PaO_2 < 80$ mmHg). Administer oxygen as indicated. *Oedema of interstitial lung tissues can interfere with gas exchange and delivery to body tissues. Supplemental oxygen promotes gas exchange across the alveolar–capillary membrane, improving tissue oxygenation.*

Community-based care

Teaching for home care focuses on managing the underlying cause of fluid volume excess and preventing future episodes of excess fluid volume. Address the following topics when preparing the person and the family for home care:

- Signs and symptoms of excess fluid and when to contact the attending doctor.
- Prescribed medications: when and how to take, intended and adverse effects, what to report to attending doctor.
- Recommended or prescribed diet; ways to reduce sodium intake; how to read food labels for salt and sodium content; use of salt substitutes, if allowed.
- If restricted, the amount and type of fluids to take each day; how to balance intake over 24 hours.
- Monitoring weight; changes reported to attending doctor.
- Ways to decrease dependent oedema:
  a. Change position frequently.
  b. Avoid restrictive clothing.
  c. Avoid crossing the legs when sitting.
  d. Wear support stockings or hose.
  e. Elevate feet and legs when sitting.
- How to protect oedematous skin from injury:
  a. Do not walk barefoot.
  b. Buy good-fitting shoes; shop in the afternoon when feet are more likely to be swollen.
- Using additional pillows or a recliner to sleep, to relieve orthopnoea.

BOX 9.5 Teaching people to reduce sodium intake

- Reducing sodium intake will help the body excrete excess sodium and water.
- The body needs less than one-tenth of a teaspoon of salt per day.
- Approximately one-third of sodium intake comes from salt added to foods during cooking and at the table; one-quarter to one-third comes from processed foods; and the rest comes from food and water naturally high in sodium.
- Sodium compounds are used in foods as preservatives, leavening agents and flavour enhancers.
- Many non-prescription drugs (such as analgesics, cough medicine, laxatives and antacids), as well as toothpastes and mouthwashes, contain high amounts of sodium.
- Low-sodium salt substitutes are not really sodium-free and may contain half as much sodium as regular salt.
- Use salt substitutes sparingly; larger amounts often taste bitter instead of salty.
- The preference for salt will eventually diminish.
- Salt, monosodium glutamate, baking soda and baking powder contain substantial amounts of sodium.
- Read labels.
- In place of salt or salt substitutes, use herbs, spices, lemon juice, vinegar and wine as flavouring when cooking.

SODIUM IMBALANCE

Sodium is the most plentiful electrolyte in ECF, with normal serum sodium levels ranging from 135 to 145 mEq/L. Sodium is the primary regulator of the volume, osmolality and distribution of ECF. It is also important for maintaining neuromuscular activity. Because of the close interrelationship between sodium and water balance, disorders of fluid volume and sodium balance often occur together. Sodium imbalances affect the osmolality of ECF and water distribution between the fluid compartments. When sodium levels are low (hyponatraemia), water is drawn into the cells of the body, causing them to swell. In contrast, high levels of sodium in ECF (hypernatraemia) draw water out of body cells, causing them to shrink. See Box 9.5 for information on teaching the person to help reduce sodium intake.

Overview of normal sodium balance

Most of the body's sodium comes from dietary intake. Other sources of sodium include prescription drugs and certain self-prescribed remedies. Sodium is primarily excreted by the kidneys. A small amount is excreted through the skin and the gastrointestinal (GI) tract.

The kidney is the primary regulator of sodium balance in the body. The kidney excretes or conserves sodium in response to changes in vascular volume. A fall in blood volume prompts several mechanisms that lead to sodium and water retention:

- The renin–angiotensin–aldosterone system (see Figure 9.9) is stimulated. Angiotensin II prompts the renal tubules to reabsorb sodium. It also causes vasoconstriction, slowing blood flow through the kidney and reducing glomerular filtration. This further reduces the amount of sodium excreted. Angiotensin II promotes the release of aldosterone from the adrenal cortex. In the presence of aldosterone, more sodium is reabsorbed in the cortical collecting tubules of the kidney and more potassium is eliminated in the urine.
- ADH is released from the posterior pituitary (see Figure 9.10). ADH promotes sodium and water reabsorption in the distal tubules of the kidney, reducing urine output and expanding blood volume.

By contrast, when blood volume expands, sodium and water elimination by the kidneys increases.

- The **glomerular filtration rate** (the rate at which plasma is filtered through the glomeruli of the kidney) increases, allowing more water and sodium to be filtered and excreted.
- ANP is released by cells in the atria of the heart. ANP increases sodium excretion by the kidneys.
- ADH release from the pituitary gland is inhibited. In the absence of ADH, the distal tubule is relatively impermeable to water and sodium, allowing more to be excreted in the urine. Table 9.6 summarises the causes and effects of sodium imbalances.

TABLE 9.6 Causes and manifestations of sodium imbalances

IMBALANCE	POSSIBLE CAUSES	MANIFESTATIONS
Hyponatraemia Serum sodium < 135 mEq/L Critical value < 120 mEq/L *Other lab values* Serum osmolality < 280 mOsm/kg Critical value < 250 mOsm/kg	• Excess sodium loss through kidneys, GI tract or skin • Water gains related to renal disease, heart failure or cirrhosis of the liver • SIADH • Excessive hypotonic IV fluids	• Anorexia, nausea, vomiting, abdominal cramping and diarrhoea • Headache • Altered mental status • Muscle cramps, weakness and tremors • Seizures and coma
Hypernatraemia Serum sodium > 145 mEq/L Critical value > 160 mEq/L *Other lab values* Serum osmolality > 295 mOsm/kg Critical value > 325 mOsm/kg	• Altered thirst • Inability to respond to thirst sensation or obtain water • Profuse sweating • Diarrhoea • Diabetes insipidus • Oral electrolyte solutions or hyperosmolar tube-feeding formulas • Excess IV fluids such as normal saline, 3% or 5% sodium chloride or sodium bicarbonate	• Thirst • Increased temperature • Dry, sticky mucous membranes • Restlessness • Weakness • Altered mental status • Decreasing level of consciousness • Muscle twitching • Seizures

The person with hyponatraemia

Hyponatraemia is a serum sodium level of less than 135 mEq/L. Hyponatraemia usually results from a loss of sodium from the body, but it may also be caused by water gains that dilute ECF.

Pathophysiology

Excess sodium loss can occur through the kidneys, GI tract or skin. Diuretic medications, kidney diseases or adrenal insufficiency with impaired aldosterone and cortisol production can lead to excessive sodium excretion in urine. Vomiting, diarrhoea and gastrointestinal suction are common causes of excess sodium loss through the GI tract. Neurological conditions such as stroke, cerebral haemorrhage, trauma or surgery can cause cerebral salt wasting (Tocco, 2010). Sodium may also be lost when gastrointestinal tubes are irrigated with water instead of saline or when repeated tap-water enemas are administered (Porth & Matfin, 2009). Excessive sweating or loss of skin surface (as with an extensive burn) can also cause excessive sodium loss.

Water gains that can lead to hyponatraemia may occur with:

- systemic diseases such as heart failure, renal failure or cirrhosis of the liver
- syndrome of inappropriate secretion of antidiuretic hormone (SIADH), in which water excretion is impaired
- excessive administration of hypotonic intravenous fluids.

Hyponatraemia causes a drop in serum osmolality. Water shifts from ECF into the intracellular space, causing cells to swell and reducing the osmolality of intracellular fluid. Many of the manifestations of hyponatraemia can be attributed to cellular oedema and hypo-osmolality.

Manifestations

The manifestations of hyponatraemia depend on the rapidity of onset, the severity and the cause of the imbalance. If the condition develops slowly, manifestations are usually not experienced until the serum sodium levels reach 125 mEq/L. In addition, the manifestations of hyponatraemia vary depending on extracellular fluid volume. Early manifestations of hyponatraemia include muscle cramps, weakness and fatigue from its effects on muscle cells. Gastrointestinal function is affected, causing anorexia, nausea and vomiting, abdominal cramping and diarrhoea.

As sodium levels continue to decrease, the brain and nervous system are affected by cellular oedema. Neurological manifestations progress rapidly when the serum sodium level falls below 120 mEq/L and include headache, depression, dulled sensorium, personality changes, irritability, lethargy, hyperreflexia, muscle twitching and tremors. If serum sodium falls to very low levels, convulsions and coma are likely to occur. When hyponatraemia is associated with decreased ECF volume, the manifestations are those of hypovolaemia (*hypotonic dehydration*). In hyponatraemia associated with fluid volume excess, manifestations include those of hypervolaemia.

INTERPROFESSIONAL CARE

Interprofessional management of hyponatraemia focuses on restoring normal blood volume and serum sodium levels.

Diagnosis

The following laboratory tests may be ordered:

- *Serum sodium* and *osmolality* are decreased in hyponatraemia (serum sodium < 135 mEq/L; serum osmolality < 275 mOsm/kg).
- A *24-hour urine specimen* is obtained to evaluate sodium excretion. In conditions associated with normal or increased extracellular volume (such as SIADH), urinary sodium is increased; in conditions resulting from losses of isotonic fluids (e.g. sweating, diarrhoea, vomiting and third-space fluid accumulation), by contrast, urinary sodium is decreased.

Medications

When both sodium and water have been lost (hyponatraemia with hypovolaemia), sodium-containing fluids are given to replace both water and sodium. These fluids may be given by mouth, nasogastric tube or intravenously. Isotonic Ringer's solution or isotonic saline (0.9% NaCl) solution may be administered. Cautious administration of intravenous 3% or 5% NaCl

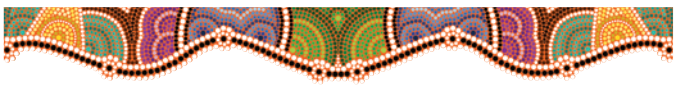
solution may be necessary in a person who has very low plasma sodium levels (110 to 115 mEq/L).

Loop diuretics are administered to a person who has hyponatraemia with normal or excess ECF volume. Loop diuretics promote an isotonic diuresis and fluid volume loss without hyponatraemia. Thiazide diuretics are avoided because they cause a relatively greater sodium loss in relation to water loss.

In addition, drugs to treat the underlying cause of hyponatraemia may be administered.

Fluid and dietary management

If hyponatraemia is mild, increasing the intake of foods high in sodium may restore normal sodium balance. Fluids often are restricted to help reduce ECF volume and correct hyponatraemia.



Nursing care

Nursing care of the person with hyponatraemia focuses on identifying the person at risk and managing problems resulting from the systemic effects of the disorder.

Health promotion

People at risk of mild hyponatraemia include those who participate in activities that increase fluid loss through excessive perspiration (diaphoresis) and then replace those losses by drinking large amounts of water. This includes athletes, people who do heavy labour in high environmental temperatures and older adults living in non-air-conditioned settings during hot weather. Teach the following to people who are at risk:

- manifestations of mild hyponatraemia, including nausea, abdominal cramps and muscle weakness
- the importance of drinking liquids containing sodium and other electrolytes at frequent intervals when perspiring heavily, when environmental temperatures are high and/or if watery diarrhoea persists for several days.

Assessment

Assessment data related to hyponatraemia include the following:

- *Health history*: current manifestations, including nausea and vomiting, abdominal discomfort, muscle weakness, headache and other symptoms; duration of symptoms and any precipitating factors such as heavy perspiration, vomiting or diarrhoea; chronic diseases such as heart or renal failure, cirrhosis of the liver or endocrine disorders; current medications.
- *Physical assessment*: mental status and level of consciousness; vital signs, including orthostatic vitals and peripheral pulses; presence of oedema or weight gain.
- *Diagnostic tests*: serum sodium and osmolality; serum potassium.

Nursing diagnoses and interventions

Risk of imbalanced fluid volume

Because of its role in maintaining fluid balance, sodium imbalances often are accompanied by water imbalances. In addition, treatment of hyponatraemia can affect the person's fluid balance.

- Monitor intake and output, weigh daily and calculate 24-hour fluid balance. *Fluid excess or deficit may occur with hyponatraemia.*

CONSIDERATION FOR PRACTICE

Carefully monitor the person receiving sodium-containing intravenous solutions for signs of hypervolaemia (increased blood pressure and CVP, tachypnoea, tachycardia, gallop rhythm S3 and/or S4 heart sounds, shortness of breath, crackles). Hypertonic saline solutions can lead to hypervolaemia, particularly in the person with cardiovascular or renal disease.

- Use an intravenous flow control device to administer hypertonic saline (3% and 5% NaCl) solutions; carefully monitor flow rate and response. *Hypertonic solutions can increase the risk of pulmonary and cerebral oedema due to water retention. Careful monitoring is vital to prevent these complications and possible permanent damage.*
- If fluids are restricted, explain the reason for the restriction, the amount of fluid allowed and how to calculate fluid intake. *Teaching increases the person's sense of control and compliance.*

For additional nursing interventions that may apply to the person with hyponatraemia, review the discussions of fluid volume deficit and fluid volume excess.

Risk of ineffective cerebral tissue perfusion

The person with severe hyponatraemia experiences fluid shifts that cause an increase in intracellular fluid volume. This can cause brain cells to swell, increasing pressure within the cranial vault.

- Monitor serum electrolytes and serum osmolality and report abnormal results. *As serum sodium levels fall, the manifestations and neurological effects of hyponatraemia become increasingly severe.*
- Assess for neurological changes, such as lethargy, altered level of consciousness, confusion and convulsions. Monitor mental status and orientation. Compare baseline data with continuing assessments. *Serum sodium levels of 115 to 120 mEq/L can cause headache, lethargy and decreased responsiveness; sodium levels less than 110 to 115 mEq/L may cause seizures and coma.*
- Assess muscle strength and tone and deep tendon reflexes. *Increasing muscle weakness and decreased deep tendon reflexes are manifestations of increasing hyponatraemia.*

CONSIDERATION FOR PRACTICE

Maintain a quiet environment and institute seizure precautions in the person with severe hyponatraemia. Severe hyponatraemia can lead to seizures and it is evidenced that a quiet environment reduces neurological stimulation. It is pertinent to maintain the person's safety and reduce the risk of injury from seizures by using precautions, such as ensuring that side rails are up, lowering the height of the bed and having airway equipment readily available.

Community-based care

Teaching for home care focuses on the underlying cause of the sodium deficit and often on prevention. Teach the person who

has experienced hyponatraemia and those who are at risk of developing hyponatraemia about the following:

- manifestations of mild and more severe hyponatraemia to report to the primary attending doctor
- the importance of regular serum electrolyte monitoring if taking a potent diuretic or on a low-sodium diet
- types of foods and fluids to replace sodium orally if dietary sodium is not restricted
- older adults' increased risk for hyponatraemia from the effects of medications and potential fluid imbalances.

The person with hypernatraemia

Hypernatraemia is a serum sodium level greater than 145 mEq/L. It may develop when sodium is gained in excess of water or when water is lost in excess of sodium. Either fluid volume deficit or fluid volume excess often accompanies hypernatraemia. Older adults with diminished thirst or who have limited access to water are at particular risk for hypernatraemia (Mount, 2012).

Pathophysiology

Two regulatory mechanisms protect the body from hypernatraemia: (1) excess sodium in ECF stimulates the release of ADH so more water is retained by the kidneys, and (2) the thirst mechanism is stimulated to increase the intake of water (Metheny, 2012). These two factors increase extracellular water, diluting the excess sodium and restoring normal levels. Because of the effectiveness of these mechanisms, hypernatraemia almost never occurs in the person who has an intact thirst mechanism and access to water.

Water deprivation is a cause of hypernatraemia in the person who is unable to respond to thirst due to altered mental status or physical disability. Excess water loss may occur with watery diarrhoea or increased insensible losses (due to fever, hyperventilation, excessive perspiration or massive burns). Unless water is adequately replaced, the person with diabetes insipidus (see Chapter 18) also may develop hypernatraemia. Excess sodium intake can result from ingestion of excess salt or hypertonic intravenous solutions. People who experience near-drowning in seawater are at risk of hypernatraemia, as are people with heat stroke.

Manifestations

Hypernatraemia (also known as *hypertonic dehydration*) causes hyperosmolality of ECF. As a result, water is drawn out of cells, leading to cellular dehydration. The most serious effects of cellular dehydration are seen in the brain. As brain cells contract, neurological manifestations develop. The brain itself shrinks, causing mechanical traction on cerebral vessels. These vessels may tear and bleed. Although the brain rapidly adapts to hyperosmolality to minimise the water loss, acute hypernatraemia can cause widespread cerebral vascular bleeding (Grossman & Porth, 2014; Metheny, 2010).

Thirst is the first manifestation of hypernatraemia. If thirst is not relieved, the primary manifestations relate to altered neurological function (see Table 9.6). Initial lethargy, weakness and irritability can progress to seizures, coma and death in severe hypernatraemia. Both the severity of the sodium excess and the rapidity of its onset affect the manifestations of hypernatraemia.

INTERPROFESSIONAL CARE

Treatment of hypernatraemia depends on its cause. Hypernatraemia is corrected slowly (over a 48-hour period) to avoid development of cerebral oedema secondary to a shift of water into the brain cells.

Diagnosis

The following laboratory and diagnostic tests may be ordered:

- *Serum sodium levels* are greater than 145 mEq/L in hypernatraemia.
- *Serum osmolality* is greater than 295 mOsm/kg in hypernatraemia.
- The *water deprivation test* may be conducted to identify diabetes insipidus. Water and all other fluids are withheld for a specified period of time. During this time, urine specimens are obtained for osmolality and specific gravity. No change in these values supports the diagnosis of diabetes insipidus.

Medications

The principal treatment for hypernatraemia is oral or intravenous water replacement. Hypotonic intravenous fluids such as 0.45% NaCl solution or 5% dextrose in water (which is isotonic when administered, but provides pure water when the glucose is metabolised) may be administered to correct the water deficit. Diuretics may also be given to increase sodium excretion (Lukitsch, 2012).

Nursing care

The primary focus of nursing care related to hypernatraemia is prevention. Measures to prevent hypernatraemia include identifying risk factors, teaching the person and caregivers, monitoring laboratory test results and working with the interprofessional team to reduce the potential for hypernatraemia.

Health promotion

Education pertaining to hypernatremia is required for the person at risk of hypernatraemia, as well as their attending doctors, and is essential to prevent this electrolyte disorder. Instruct caregivers of debilitated people who are unable to perceive or respond to thirst to offer fluids at regular intervals. If the person is unable to maintain adequate fluid intake, contact the medical staff about an alternative route for fluid intake (e.g. a feeding tube). Teach attending doctors the importance of providing adequate water for the person receiving tube feedings (many of which are hypertonic).

Assessment

Assessment data related to hypernatraemia include the following:

- *Health history*: duration of symptoms and any precipitating factors such as water deprivation, increased water loss due

to heavy perspiration, temperature or rapid breathing, diarrhoea, excess salt intake or diabetes insipidus; current medications; perception of thirst.
- *Physical assessment*: vital signs including temperature; mucous membranes; altered mental status or level of consciousness; manifestations of fluid volume excess or fluid volume deficit.
- *Diagnostic tests*: monitor serum sodium and osmolality, serum potassium.

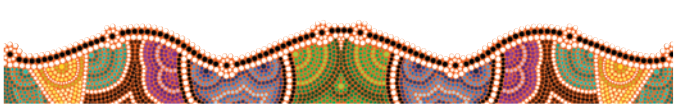
Nursing diagnoses and interventions

Risk of injury

Mental status and brain function may be affected by hypernatraemia itself or by rapid correction of the condition that leads to cerebral oedema. In either case, closely monitor the person and take precautions to reduce risk of injury.
- Monitor and maintain fluid replacement to within the prescribed limits. Monitor serum sodium levels and osmolality; report rapid changes to the attending doctor. *Rapid water replacement or rapid changes in serum sodium or osmolality can cause fluid shifts within the brain, increasing the risk of bleeding or cerebral oedema.*
- Monitor neurological function, including mental status, level of consciousness and other manifestations such as headache, nausea, vomiting, elevated blood pressure and decreased pulse rate. *Both hypernatraemia and rapid correction of hypernatraemia affect the brain and brain function. Careful monitoring is vital to detect changes in mental status that may indicate cerebral bleeding or oedema.*
- Institute safety precautions as necessary: keep the bed in its lowest position, side rails up and padded, and an airway at the bedside. *The person with a sodium disorder is at risk of injury due to seizure activity and changes in mental status.*
- Keep clocks, calendars and familiar objects at the bedside. Orient to time, place and circumstances as needed. Allow significant others to remain with the person as much as possible. *An unfamiliar environment and altered thought processes can further increase the person's risk of injury. Significant others provide a sense of security and reduce the person's anxiety.*

Community-based care

When preparing the person who has experienced hypernatraemia for home care, discuss the following topics:
- the importance of responding to thirst and consuming adequate fluids (if the person is dependent on a caregiver, stress to the caregiver the importance of regularly offering fluids)
- if prescribed, guidelines for following a low-sodium diet (see Box 9.5)
- use and effects (intended and unintended) of any prescribed diuretic or other medication
- the importance of following a schedule for regular monitoring of serum electrolyte levels and reporting manifestations of imbalance to the attending doctor.



POTASSIUM IMBALANCE

Potassium, the primary intracellular cation, plays a vital role in cell metabolism and cardiac and neuromuscular function. The normal serum (ECF) potassium level is 3.5 to 5.0 mEq/L.

Overview of normal potassium balance

Most potassium in the body is found within the cells (ICF), which have a concentration of 140 to 150 mEq/L. This significant difference in the potassium concentrations of ICF and ECF helps maintain the resting membrane potential of nerve and muscle cells; either a deficit or an excess of potassium can adversely affect neuromuscular and cardiac function. The higher intracellular potassium concentration is maintained by the sodium–potassium pump.

To maintain its balance, potassium must be replaced daily. Normally, potassium is supplied in food. Virtually all foods contain potassium, although some foods and fluids are richer sources of this element than others (see Box 9.6).

The kidneys eliminate potassium very efficiently; even when potassium intake is stopped, the kidneys continue to excrete it. Because the kidneys do not conserve potassium well, significant amounts may be lost through this route. However, because the kidneys are the principal organs involved in the elimination of potassium, renal failure can lead to potentially serious elevations of serum potassium.

Aldosterone helps regulate potassium elimination by the kidneys. An increased potassium concentration in ECF stimulates aldosterone production by the adrenal gland. The kidneys respond to aldosterone by increasing potassium excretion. Changes in aldosterone secretion can profoundly affect the serum potassium level.

Normally only small amounts of potassium are lost in the faeces, but substantial amounts may be lost from the gastrointestinal tract with diarrhoea or through drainage from an ileostomy (a permanent opening into the small bowel).

Potassium constantly shifts into and out of the cells. This movement between ICF and ECF can significantly affect the

BOX 9.6 Foods high in potassium

Fruits
- Apricots
- Avocados
- Bananas
- Dates
- Oranges
- Raisins

Vegetables and vegetable juices
- Carrots
- Cauliflower
- Mushrooms
- Peas
- Potatoes
- Spinach
- Tomatoes

Meats and fish
- Beef
- Chicken
- Kidney
- Liver
- Lobster
- Pork
- Salmon
- Tuna
- Turkey

Milk products
- Buttermilk
- Chocolate milk
- Evaporated milk
- Low-fat yoghurt
- Milk

TABLE 9.7 Causes and manifestations of potassium imbalances

IMBALANCE	CAUSES	MANIFESTATIONS
Hypokalaemia Serum potassium < 3.5 mEq/L Critical value < 2.5 mEq/L	• Excess GI losses: vomiting, diarrhoea, ileostomy drainage • Renal losses: diuretics, hyperaldosteronism • Inadequate intake • Shift into cells: alkalosis, rapid tissue repair	Cardiovascular • Arrhythmias • ECG changes Gastrointestinal • Nausea and vomiting • Anorexia • Decreased bowel sounds • Ileus Musculoskeletal • Muscle weakness • Leg cramps
Hyperkalaemia Serum potassium > 5.0 mEq/L Critical value > 6.5 mEq/L	• Renal failure • Potassium-sparing diuretics • Adrenal insufficiency • Excess potassium intake (e.g. excess potassium replacement) • Aged blood • Shift out of cells: cell and tissue damage, acidosis	Cardiovascular • Tall, peaked T waves, widened QRS • Arrhythmias • Cardiac arrest Gastrointestinal • Nausea and vomiting • Abdominal cramping • Diarrhoea Neuromuscular • Muscle weakness • Paraesthesias • Flaccid paralysis

serum potassium level. For example, potassium shifts into or out of the cells in response to changes in hydrogen ion concentration (pH, discussed later in this chapter) as the body strives to maintain a stable acid–base balance.

The significant difference between intracellular and extracellular potassium concentrations is vital to the resting membrane potential of cells. Resting membrane potential, in turn, is necessary for transmitting nerve impulses. Potassium imbalances affect transmission and conduction of nerve impulses, maintenance of normal cardiac rhythms and contraction of skeletal and smooth muscle (McCance & Huether, 2014).

As the primary intracellular cation, potassium plays a major role in regulating the osmolality of ICF and is involved in metabolic processes. Potassium is necessary for the storage of glycogen in skeletal muscle cells. Table 9.7 summarises the causes and manifestations of potassium imbalances.

The person with hypokalaemia

Hypokalaemia is an abnormally low serum potassium (less than 3.5 mEq/L). It usually results from excess potassium loss, although the hospitalised person may be at risk of hypokalaemia because of inadequate potassium intake.

Pathophysiology

Excess potassium may be lost through the kidneys or the GI tract. These losses deplete total potassium stores in the body.

- Excess potassium loss through the kidneys often is secondary to drugs such as potassium-wasting diuretics, corticosteroids, amphotericin B and large doses of some antibiotics. Hyperaldosteronism, a condition in which the adrenal glands secrete excess aldosterone, also causes excess elimination of potassium through the kidneys. Glucosuria and osmotic diuresis (e.g. associated with diabetes mellitus) also cause potassium wasting through the kidneys (Metheny, 2012).
- Gastrointestinal losses of potassium result from severe vomiting, gastric suction, or loss of intestinal fluids through diarrhoea or ileostomy drainage.

Potassium intake may be inadequate in the person who is unable or unwilling to eat for prolonged periods. The person who is hospitalised is at risk, especially when on extended parenteral fluid therapy with solutions that do not contain potassium. The person with anorexia nervosa or alcoholism may develop hypokalaemia due to both inadequate intake and loss of potassium through vomiting, diarrhoea, or laxative or diuretic use.

A *relative* loss of potassium occurs when potassium shifts from ECF into the cells. This usually is due to loss of hydrogen ions and alkalosis, although it also may occur during periods of rapid tissue repair (e.g. following a burn or trauma), in the presence of excess insulin (insulin promotes potassium entry into skeletal muscle and liver cells), during acute stress or because of hypothermia. In these instances, the total body stores of potassium remain adequate.

Manifestations

Hypokalaemia affects the transmission of nerve impulses, interfering with the contractility of smooth, skeletal and cardiac muscle, as well as the regulation and transmission of cardiac impulses.

- Characteristic electrocardiogram (ECG) changes of hypokalaemia include flattened or inverted T waves, the development of U waves and a depressed ST segment (see Figure 9.11). The most serious cardiac effect is an increased risk of atrial and ventricular arrhythmias (abnormal rhythms). Hypokalaemia increases the risk of digitalis toxicity in the person receiving this drug used to treat heart failure (see Chapter 30).

- Hypokalaemia affects both the resting membrane potential and intracellular enzymes in skeletal and smooth muscle cells. This causes skeletal muscle weakness and slowed peristalsis of the GI tract. Muscles of the lower extremities are affected first, then the trunk and upper extremities. This effect of hypokalaemia is magnified when serum calcium levels are above normal.
- Carbohydrate metabolism is affected by hypokalaemia. Insulin secretion is suppressed, as is the synthesis of glycogen in skeletal muscle and the liver.

Hypokalaemia also can affect kidney function, particularly the ability to concentrate urine. Severe hypokalaemia can lead to rhabdomyolysis, a condition in which muscle fibres disintegrate, releasing myoglobin to be excreted in the urine.

Manifestations of hypokalaemia are more pronounced when potassium losses occur acutely. When hypokalaemia develops gradually, potassium shifts out of the cells, helping maintain the ratio of intracellular to extracellular potassium. As a result, the neuromuscular manifestations of hypokalaemia are less severe. The *multisystem effects of hypokalaemia* are summarised on the following page.

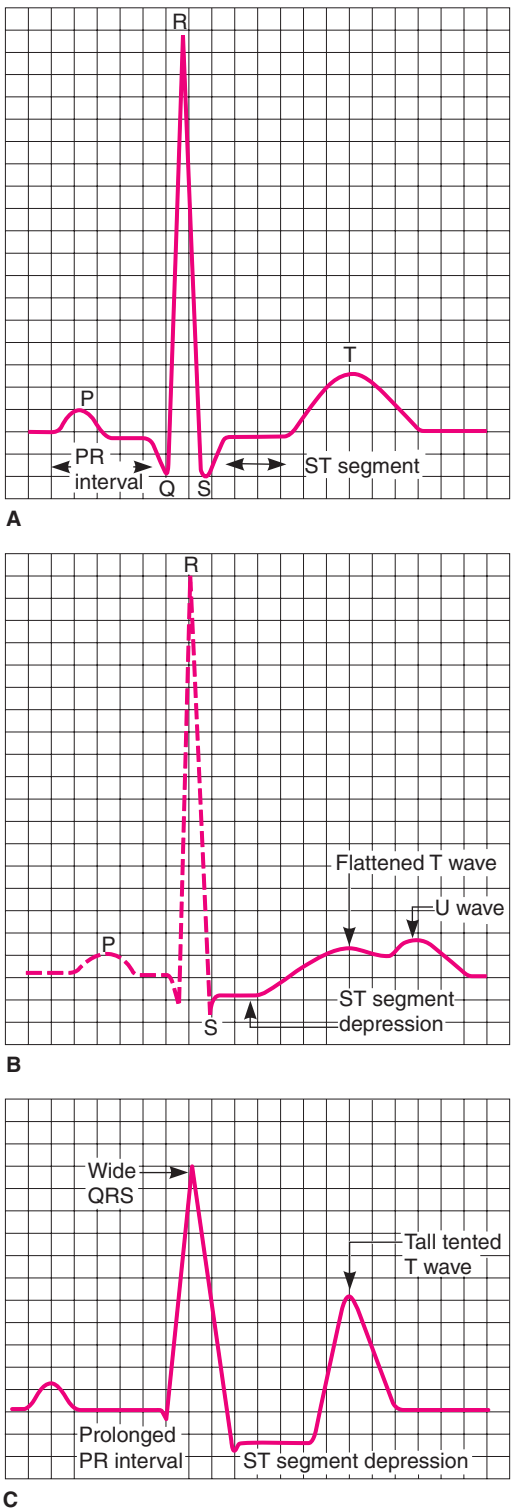


FIGURE 9.11 ■ The effects of changes in potassium levels on the electrocardiogram (ECG). *A*, Normal ECG; *B*, ECG in hypokalaemia; *C*, ECG in hyperkalaemia

INTERPROFESSIONAL CARE

The management of hypokalaemia focuses on prevention and treatment of a deficiency.

Diagnosis

The following laboratory and diagnostic tests may be ordered:

- *Serum potassium* (K^+) is used to monitor potassium levels in the person who is at risk of, or who is being treated for, hypokalaemia. A serum K^+ of 3.0 to 3.5 mEq/L is considered mild hypokalaemia. Moderate hypokalaemia is defined as a serum K^+ of 2.5 to 3.0 mEq/L and severe hypokalaemia as a serum K^+ of less than 2.5 mEq/L (Metheny, 2012).
- *Arterial blood gases (ABGs)* are measured to determine acid–base status. An increased pH (alkalosis) often is associated with hypokalaemia. (See Table 9.11 later in this chapter for normal ABG values.)
- *Renal function studies*, such as *serum urea and creatinine,* may be ordered to evaluate for potential causes or effects of hypokalaemia.
- *ECG recordings* are obtained to evaluate the effects of hypokalaemia on the cardiac conduction system.

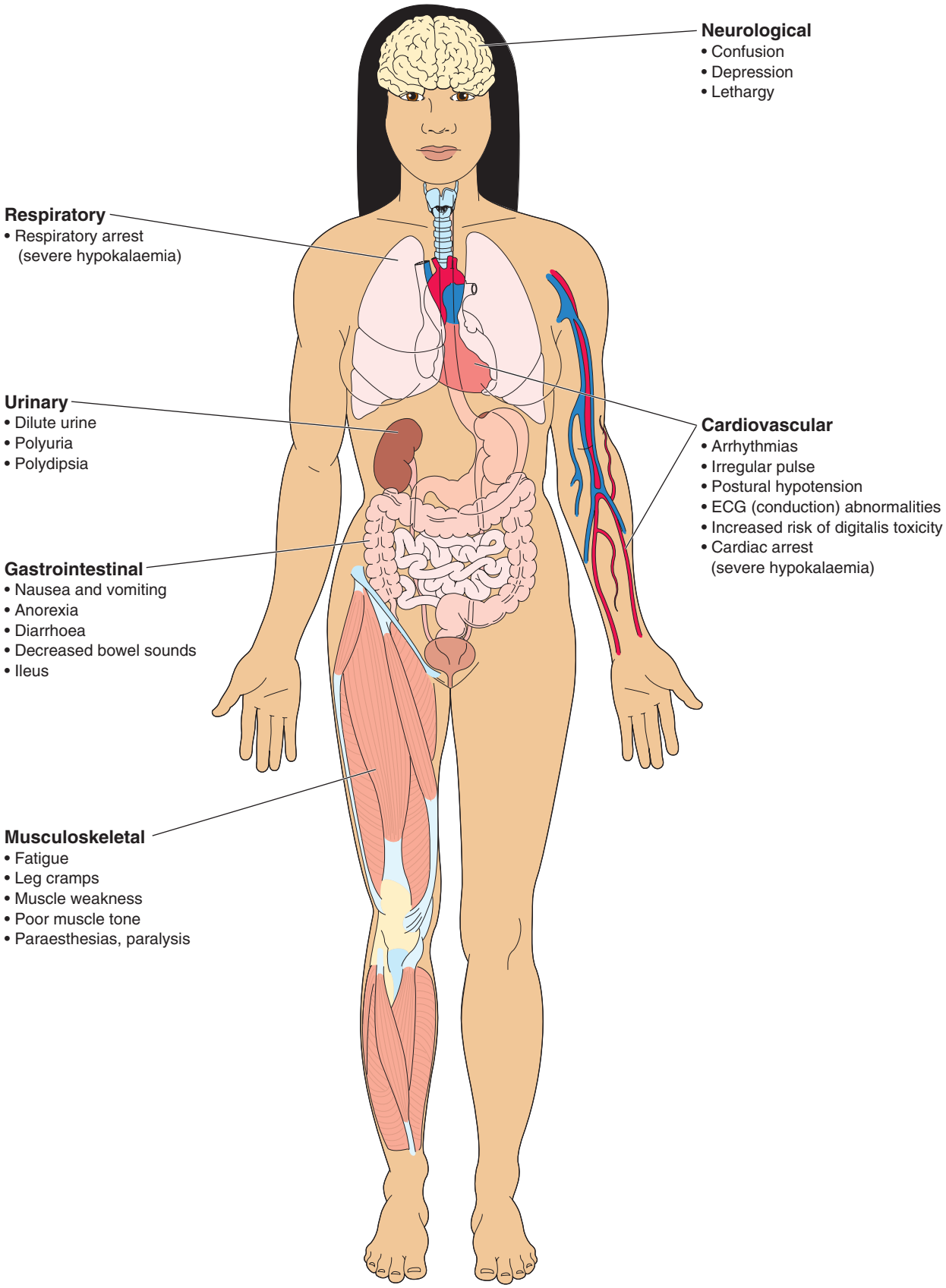
Medications

Oral and/or parenteral potassium supplements are given to prevent and, as needed, treat hypokalaemia. To prevent hypokalaemia in the person taking nothing by mouth, potassium chloride is added to IV fluids. The dose used to treat hypokalaemia includes the daily maintenance requirement, replacement of ongoing losses (e.g. gastric suction) and additional potassium to correct the existing deficit. Several days of therapy may be required. Commonly prescribed potassium supplements, their actions and nursing implications are described in the 'Medication administration' box below.

Nutrition

A diet high in potassium-rich foods is recommended for the person at risk of developing hypokalaemia or to supplement drug therapy (see Box 9.6).

MULTISYSTEM EFFECTS OF HYPOKALAEMIA



MEDICATION ADMINISTRATION Hypokalaemia

POTASSIUM SOURCES

Potassium acetate
Potassium bicarbonate
Potassium citrate
Potassium chloride
Potassium gluconate

Potassium is rapidly absorbed from the gastrointestinal tract; potassium chloride is the agent of choice, because low chloride often accompanies low potassium. Potassium is used to prevent and/or treat hypokalaemia (e.g. with parenteral nutrition and potassium-wasting diuretics and prophylactically after major surgery).

Nursing responsibilities

- When giving oral forms of potassium:
  a. Dilute or dissolve effervescent, soluble or liquid potassium in fruit or vegetable juice or cold water.
  b. Chill to increase palatability.
  c. Give with food to minimise GI effects.
- When giving parenteral forms of potassium:
  a. Check correct infusion rate and administer slowly.
  b. Check appropriate administration mode and use as an additive (usually NOT administered IV push and NOT added to fluids already hanging).
  c. Do *not* administer undiluted.
  d. Assess injection site frequently for signs of pain and inflammation.
  e. Use an infusion control device.
- Assess for abdominal pain, distension, gastrointestinal bleeding; if present, do not administer medication. Notify the attending doctor.
- Monitor fluid intake and output.
- Assess for manifestations of hyperkalaemia: weakness, feeling of heaviness in legs, mental confusion, hypotension, cardiac arrhythmias, changes in ECG, increased serum potassium levels.

Health education for the person and their family

- Do not take potassium supplements if you are also taking a potassium-sparing diuretic.
- When parenteral potassium is discontinued, eat potassium-rich foods.
- Do not chew enteric-coated tablets or allow them to dissolve in the mouth, as this may affect the potency and action of the medications.
- Take potassium supplements with meals.
- Do not use salt substitutes when taking potassium (most salt substitutes are potassium-based).

Nursing care

See below for 'Nursing care plan: A person with hypokalaemia'.

Health promotion

When providing general health education, discuss the use of balanced electrolyte solutions (e.g. sports drinks) to replace abnormal fluid losses (excess perspiration, vomiting or severe diarrhoea). Discuss the necessity of preventing hypokalaemia with the at-risk person. Provide diet teaching and refer the person with anorexia nervosa for counselling. Stress the potassium-losing effects of taking diuretics and using laxatives to enhance weight loss. Discuss the potassium-wasting effects of most diuretics with the person taking these drugs and encourage a diet rich in high-potassium foods, as well as regular monitoring of serum potassium levels.

Assessment

Assessment data related to hypokalaemia include the following:

- *Health history*: current manifestations, including anorexia, nausea and vomiting, abdominal discomfort, muscle weakness or cramping, and other symptoms; duration of symptoms and any precipitating factors such as diuretic use, prolonged vomiting or diarrhoea; chronic diseases such as diabetes, hyperaldosteronism or Cushing's syndrome; current medications.
- *Physical assessment*: mental status; vital signs, including orthostatic vitals, apical and peripheral pulses; bowel sounds, abdominal distension; muscle strength and tone.
- *Diagnostic tests*: serum electrolytes, K^+, Na^+ and Ca^{2+} in particular, arterial pH and other ABG results, renal function tests (urea and creatinine), ECG changes.

Nursing diagnoses and interventions

The effects of hypokalaemia on cardiac impulse transmission and cardiac and skeletal muscle function are the highest priority nursing care focus.

Decreased cardiac output

Hypokalaemia affects the strength of cardiac contractions and can lead to arrhythmias that further impair cardiac output. Hypokalaemia also alters the response to cardiac drugs, such as digitalis and the anti-arrhythmics.

- Monitor serum potassium levels, particularly in the person at risk of hypokalaemia (those with excess losses due to drug therapy, gastrointestinal losses or who are unable to consume a normal diet). Report abnormal levels to the attending doctor. *Potassium must be replaced daily, because the body is unable to conserve it. Either lack of intake or abnormal losses of potassium in the urine or gastric fluids can lead to hypokalaemia.*
- Monitor vital signs, including orthostatic vitals and peripheral pulses. *As cardiac output falls, the pulse becomes weak and thready. Orthostatic hypotension may be noted with decreased cardiac output.*
- Monitor the person taking digitalis for toxicity. Monitor response to antiarrhythmic drugs. *Hypokalaemia potentiates digitalis effects and increases resistance to certain anti-arrhythmics.*

NURSING CARE PLAN A person with hypokalaemia

Rose Ortiz is a 72-year-old widow who lives alone, although close to her daughter's home. Ms Ortiz has mild heart failure and is being treated with digoxin (Lanoxin) 0.125 mg, frusemide (Lasix) 40 mg PO daily and a mildly restricted sodium diet (2 g daily). For the last several weeks, Ms Ortiz has complained that she feels weak and sometimes faint, lightheaded and dizzy. Serum electrolyte tests ordered by her physician reveal a potassium level of 2.4 mEq/L. Potassium chloride solution (K Ciel 20 mEq/15 mL) PO twice daily is prescribed and Ms Ortiz is referred to Nancy Walters, RN, for follow-up care.

ASSESSMENT

Ms Ortiz's health history reveals that she has rigidly adhered to her sodium-restricted diet and has been compliant in taking her prescribed medications, with the exception of occasionally taking an additional 'water pill' when her ankles swell. She takes a laxative every evening to ensure a daily bowel movement. Ms Ortiz states that she is reluctant to take the potassium chloride the doctor has ordered because her neighbour complains that his potassium supplement upsets his stomach. Physical assessment findings include T 36.8°C, P 70, R 20 and BP 138/84. Muscle strength in her upper extremities is normal and equal; lower extremity strength is weak but equal. No sensory deficits are apparent.

DIAGNOSES

- *Risk of injury* related to muscle weakness.
- *Risk of ineffective health maintenance* related to lack of knowledge about how diuretic therapy and laxative use affect potassium levels.

PLANNING

- Advise the person about the potential hazards of negotiating stairs.
- Highlight awareness of potential medication side effects and adverse effects and explain how taking additional tablets may have contributed to hypokalaemia.
- Consider alternative measures to prevent constipation without using laxatives on a regular basis (e.g. high-fibre diet, adequate fluid intake).
- Explain purpose of the prescribed potassium and its role in reversing muscle weakness.
- Instruct and educate the person of the importance of taking the medication (potassium supplement) after breakfast and supper. Advise to call if gastric irritation occurs.
- Discuss dietary sources of potassium and provide a list of potassium-rich foods.

Expected outcomes

- Maintain potassium level within normal limits (3.5 to 5.0 mEq/L).
- Regain normal muscle strength.
- Remain free of injury.
- Verbalise understanding of the effects of diuretic therapy and laxatives on potassium levels.
- Identify measures to avoid gastrointestinal irritation when taking oral potassium.
- Identify potassium-rich foods.

IMPLEMENTATION

- To observe and monitor Ms Ortiz's ability to safely ambulate up and down stairs.
- Discuss and consult with Ms Ortiz if there have been any heart palpations, dizziness which could be a result of side effects of frusemide.
- Discuss and consult with Mrs Ortiz about her dietary intake with a focus on high fibre and fluids.
- Discuss with Mrs Ortiz her compliance with medication administration after food, advising report of any gastric irritation.
- Review Ms Ortiz's dietary intake, identifying that she is eating potassium-rich food sources.

EVALUATION

On a follow-up visit 1 week later, Ms Ortiz states that her muscle weakness, dizziness and other symptoms have resolved. She is taking the prescribed drugs as directed and is using laxatives only 2 or 3 times a week. Ms Ortiz reports that she has increased her intake of both potassium-rich foods and fluids and high-fibre foods. Her potassium level is within normal limits.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the pathophysiological basis for Ms Ortiz's muscle weakness and dizziness?
2. How might the chronic overuse of laxatives contribute to hypokalaemia?
3. Describe the interaction of digitalis, diuretics and potassium.
4. Develop a plan of care for Ms Ortiz for the nursing diagnosis of *Perceived constipation*.

REFLECTION ON THE NURSING PROCESS

1. As the Registered Nurse, how do you know that education pertaining to medication compliance has been effective?
2. What communication and education strategies could you use when providing care for a person with hypokalaemia?

CONSIDERATION FOR PRACTICE

Place the person with severe hypokalaemia on a cardiac monitor. Closely monitor cardiac rhythm and observe for characteristic ECG changes of hypokalaemia (ST segment depression, flattened T waves and U waves). Report rhythm changes immediately and treat as indicated. Severe hypokalaemia can cause life-threatening arrhythmias.

- Dilute intravenous potassium and administer using an electronic infusion device. In general, potassium is given no faster than 10 to 20 mEq/hour. Closely monitor intravenous flow rate and response to potassium replacement. *Rapid potassium administration is dangerous and can lead to hyperkalaemia and cardiac arrest.*

CONSIDERATION FOR PRACTICE

Never administer undiluted potassium directly into the vein.

Activity intolerance

Muscle cramping and weakness are common early manifestations of hypokalaemia. The lower extremities are usually affected initially. This muscle weakness can cause the person to fatigue easily, particularly with activity.

- Monitor skeletal muscle strength and tone, which are affected by moderate hypokalaemia. *Increasing weakness, paraesthesias or paralysis of muscles or progression of affected muscles to include the upper extremities or trunk can indicate a further drop in serum potassium levels.*
- Monitor respiratory rate, depth and effort; heart rate and rhythm; and blood pressure at rest and following activity. *Tachypnoea, dyspnoea, tachycardia and/or a change in blood pressure may indicate decreasing ability to tolerate activities. Report changes to the attending doctor.*
- Assist with self-care activities as needed. *Increasing muscle weakness can lead to fatigue and affect the ability to meet self-care needs.*

Risk of imbalanced fluid volume

- Maintain accurate intake and output records. *Gastrointestinal fluid losses can lead to significant potassium losses.*
- Monitor bowel sounds and abdominal distension. *Hypokalaemia affects smooth muscle function and can lead to slowed peristalsis and paralytic ileus.*

Acute pain

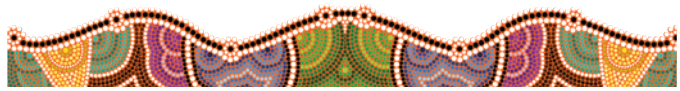
Discomfort is common when intravenous potassium chloride at a concentration of more than 40 mEq/L is given into a peripheral vein.

- When possible, administer intravenous KCl through a central line. *The rapid blood flow through central veins dilutes the KCl solution, decreasing discomfort.*
- Spread the total daily dose of KCl over 24 hours to minimise the concentration of intravenous solutions. *High concentrations of KCl are irritating to vein walls, particularly if inflammation is present.*
- Discuss with the physician the use of a small amount of lidocaine prior to or with the infusion. *Both a lidocaine bolus given at the infusion site and a small amount of lidocaine in the intravenous infusion have been shown to at least partially relieve discomfort associated with concentrated potassium solutions* (Metheny, 2012).

Community-based care

The focus in preparing the person with or at risk of hypokalaemia is prevention. Discharge planning focuses on teaching self-care practices. Include the following topics when preparing the person and family for home care:

- recommended diet, including a list of potassium-rich foods
- prescribed medications and potassium supplements, their use, and desired and unintended effects
- using salt substitutes (if recommended) to increase potassium intake; avoiding substitutes if taking a potassium supplement or potassium-sparing diuretic
- manifestations of potassium imbalance (hypokalaemia or hyperkalaemia) to report to the attending doctor
- recommendations for monitoring serum potassium levels
- if taking digitalis, manifestations of digitalis toxicity to report to the attending doctor
- managing gastrointestinal disorders that cause potassium loss (vomiting, diarrhoea, ileostomy drainage) to prevent hypokalaemia.



The person with hyperkalaemia

Hyperkalaemia is an abnormally high serum potassium level (greater than 5 mEq/L). Hyperkalaemia can result from inadequate excretion of potassium, excessively high intake of potassium, or a shift of potassium from the intracellular to the extracellular space. *Pseudohyperkalaemia* (an erroneously high serum potassium reading) can occur if the blood sample haemolyses, releasing potassium from blood cells, before it is analysed. Hyperkalaemia affects neuromuscular and cardiac function.

Pathophysiology

Impaired renal excretion of potassium is a primary cause of hyperkalaemia. Untreated renal failure, adrenal insufficiency (e.g. Addison's disease or inadequate aldosterone production) and medications (such as potassium-sparing diuretics, the antimicrobial drug trimethoprim and some NSAIDs) impair potassium excretion by the kidneys.

In the person with a normal renal excretion of potassium, excess oral potassium (e.g. by supplement or use of salt substitutes) rarely leads to hyperkalaemia. Rapid intravenous administration of potassium or transfusion of aged blood can lead to hyperkalaemia. A shift of potassium ions from the intracellular space can occur in acidosis, with severe tissue trauma, during chemotherapy and due to starvation. In acidosis, excess hydrogen ions enter the cells, displacing potassium and causing it to shift into the extracellular space. The extent of this shift is greater with metabolic acidosis than with respiratory acidosis (see 'Acid–base disorders' later in this chapter).

Hyperkalaemia alters the cell membrane potential, affecting the heart, skeletal muscle function and the GI tract. The most harmful consequence of hyperkalaemia is its effect on cardiac function. The cardiac conduction system is affected first, with slowing of the heart rate, possible heart blocks and prolonged depolarisation. ECG changes include peaked T waves, a prolonged PR interval and widening of the QRS complex (see Figure 9.11). Ventricular arrhythmias develop and cardiac arrest may occur. Severe hyperkalaemia decreases the strength of myocardial contractions.

Skeletal muscles become weak and paralysis may occur with very high serum potassium levels. Hyperkalaemia causes smooth muscle hyperactivity, leading to gastrointestinal disturbances.

The seriousness of hyperkalaemia is based on the serum potassium (K^+) level and ECG changes.

- *Mild hyperkalaemia:* serum K^+ between 5 and 6.5 mEq/L; ECG changes limited to peaked T wave.
- *Moderate hyperkalaemia:* serum K^+ between 6.5 and 8 mEq/L; ECG changes limited to peaked T wave.
- *Severe hyperkalaemia:* serum K^+ greater than 8 mEq/L; ECG shows absent P waves and widened QRS pattern.

Manifestations

The manifestations of hyperkalaemia result from its effects on the heart, skeletal and smooth muscles. Early manifestations include diarrhoea, colic (abdominal cramping), anxiety, paraesthesias, irritability, and muscle tremors and twitching. As serum potassium levels increase, muscle weakness develops, progressing to flaccid paralysis. The lower extremities are affected first, progressing to the trunk and upper extremities. The heart rate may be slow (bradycardia) and irregular. The ECG shows T-wave changes and, at high serum potassium levels, widening of the QRS complex and absence of P waves.

INTERPROFESSIONAL CARE

The management of hyperkalaemia focuses on returning the serum potassium level to normal by treating the underlying cause and avoiding additional potassium intake. The choice of therapy for existing hyperkalaemia is based on the severity of the hyperkalaemia.

Diagnosis

The following laboratory and diagnostic tests may be ordered:

- *Serum electrolytes* show a serum potassium level greater than 5.0 mEq/L. Low calcium and sodium levels may increase the effects of hyperkalaemia; therefore, these electrolytes are usually measured as well.
- *ABG*s are measured to determine if acidosis is present.
- An *ECG* is obtained and *continuous ECG monitoring* is instituted to evaluate the effects of hyperkalaemia on cardiac conduction and rhythm.

Medications

Medications are administered to lower the serum potassium and to stabilise the conduction system of the heart. For moderate to severe hyperkalaemia, calcium gluconate is given intravenously to counter the effects of hyperkalaemia on the cardiac conduction system. While the effect of calcium gluconate lasts only for 1 hour, it allows time to initiate measures to lower serum potassium levels. To rapidly lower these levels, regular insulin and 50 g of glucose are administered. Insulin and glucose promote potassium uptake by the cells, shifting potassium out of ECF. In some cases, a $ß_2$-agonist such as salbutamol may be given by nebuliser to temporarily push potassium into the cells. Sodium bicarbonate may be given to treat acidosis. As the pH returns towards normal, hydrogen ions are released from the cells and potassium returns into the cells.

To remove potassium from the body, sodium polystyrene sulfonate, a resin that binds potassium in the GI tract, may be administered orally or rectally. If renal function is normal, diuretics such as frusemide are given to promote potassium excretion. Commonly prescribed drugs, their actions and nursing implications are listed in the 'Medication administration' box below.

Dialysis

When renal function is severely limited, either peritoneal dialysis or haemodialysis may be implemented to remove excess potassium. These measures are invasive and are typically used only when other measures are ineffective. See Chapter 27 for more information about dialysis.

Nursing care

Nursing care focuses related to hyperkalaemia include identifying the at-risk person, preventing hyperkalaemia and addressing problems resulting from the systemic effects of hyperkalaemia. A 'Nursing care plan: A person with hyperkalaemia' is found below.

Health promotion

Those at the greatest risk of developing hyperkalaemia include people taking potassium supplements (prescribed or over-the-counter), using potassium-sparing diuretics or salt substitutes, and experiencing renal failure. Athletes participating in competition sports such as body building and using anabolic steroids, muscle-building compounds or 'energy drinks' also may be at risk of hyperkalaemia.

Teach all people to read food and dietary supplement labels carefully. Discuss the importance of taking prescribed potassium supplements as ordered and not increasing the dose unless prescribed by the attending doctor. Advise the person taking a potassium supplement or potassium-sparing diuretic to avoid salt substitutes, which usually contain potassium. Discuss the importance of maintaining an adequate fluid intake (unless a fluid restriction has been prescribed) to maintain renal function and eliminate potassium from the body.

Assessment

Assessment data related to hyperkalaemia include the following:

- *Health history*: current manifestations, including numbness and tingling, nausea and vomiting, abdominal cramping, muscle weakness, palpitations; duration of symptoms and any precipitating factors such as use of salt substitutes, potassium supplements or reduced urine output; chronic diseases such as renal failure or endocrine disorders; current medications.
- *Physical assessment*: apical and peripheral pulses; bowel sounds; muscle strength in upper and lower extremities; ECG pattern.
- *Diagnostic tests*: serum electrolytes, potassium, sodium and calcium in particular; ABGs; digitalis levels; ECG.

MEDICATION ADMINISTRATION Hyperkalaemia

DIURETICS

Potassium-wasting diuretics, such as frusemide (Lasix), may be used to enhance renal excretion of potassium.

Nursing responsibilities

- Monitor serum electrolytes.
- Monitor and record weight at regular intervals under standard conditions (same time of day, balanced scale, same clothing).
- Monitor intake and output.

INSULIN, HYPERTONIC DEXTROSE AND SODIUM BICARBONATE

Insulin, hypertonic dextrose (10% to 50%) and sodium bicarbonate are used in the emergency treatment of moderate to severe hyperkalaemia. Insulin promotes the movement of potassium into the cell and glucose prevents hypoglycaemia. The onset of action of insulin and hypertonic dextrose occurs within 30 minutes and is effective for approximately 4 to 6 hours.

Sodium bicarbonate elevates the serum pH; potassium is moved into the cell in exchange for hydrogen ion. Sodium bicarbonate is particularly useful in the person with metabolic acidosis. Onset of effects occurs within 15 to 30 minutes and is effective for approximately 2 hours.

Nursing responsibilities

- Administer intravenous insulin and dextrose over prescribed interval of time using an infusion pump.
- Administer sodium bicarbonate as prescribed. It may be administered as an intravenous bolus or added to a dextrosein-water solution and given by infusion.
- In the person receiving sodium bicarbonate, monitor for sodium overload, particularly in the person with hypernatraemia, heart failure and renal failure.
- Monitor the ECG pattern closely.
- Monitor serum electrolytes (K^+, Na^+, Ca^{2+}, Mg^{2+}) frequently during treatment.

CALCIUM GLUCONATE AND CALCIUM CHLORIDE

Intravenous calcium gluconate or calcium chloride is used as a temporary emergency measure to counteract the toxic effects of potassium on myocardial conduction and function.

Nursing responsibilities

- Closely monitor the ECG of the person receiving intravenous calcium, particularly for bradycardia.
- Calcium should be used cautiously in the person receiving digitalis, because calcium increases the cardiotonic effects of digitalis and may precipitate digitalis toxicity, leading to arrhythmias.

SODIUM POLYSTYRENE SULFONATE AND SORBITOL

Sodium polystyrene sulfonate is used to treat moderate or severe hyperkalaemia. Categorised as a cation exchange resin, sodium polystyrene sulfonate exchanges sodium or calcium for potassium in the large intestine. Sorbitol is given with sodium polystyrene sulfonate to promote bowel elimination. Sodium polystyrene sulfonate and sorbitol may be administered orally, through a nasogastric tube or rectally as a retention enema. The usual dosage is 20 g three or four times a day with 20 mL of 70% sorbitol solution.

Nursing responsibilities

- Because sodium polystyrene sulfonate contains sodium, monitor the person with heart failure and oedema closely for water retention.
- Monitor serum electrolytes (K^+, Na^+, Ca^{2+}, Mg^{2+}) frequently during therapy.
- Restrict sodium intake in the person who is unable to tolerate increased sodium load (e.g. those with CHF or hypertension).
- Sodium polystyrene sulfonate should not be given to people at risk of intestinal necrosis, including postoperative patient, those who have a history of bowel obstruction, ischaemic bowel disease or those who have had a renal transplant.

Nursing diagnoses and interventions

The effects of excess potassium on the electrical conduction and contractility of the heart are the highest priority for nursing care, particularly when the serum potassium level is 6.5 mEq/L or higher.

Risk of decreased cardiac output

Hyperkalaemia affects depolarisation of the atria and ventricles of the heart. Severe hyperkalaemia can cause arrhythmias with ventricular fibrillation and cardiac arrest. The cardiac effects of hyperkalaemia are more pronounced when the serum potassium level rises rapidly. Low serum sodium and calcium levels, high serum magnesium levels and acidosis contribute to the adverse effects of hyperkalaemia on the heart muscle.

- Closely monitor the response to intravenous calcium gluconate, particularly in people taking digitalis. *Calcium increases the risk of digitalis toxicity.*

CONSIDERATION FOR PRACTICE

Monitor the ECG pattern for development of peaked, narrow T waves, prolongation of the PR interval, depression of the ST segment, widened QRS interval and loss of the P wave. Notify the physician of changes. Progressive ECG changes from a peaked T wave to loss of the P wave and widening of the QRS complex indicate an increasing risk of arrhythmias and cardiac arrest.

Risk of activity intolerance

Both hypokalaemia (low serum potassium levels) and hyperkalaemia (high serum potassium levels) affect neuromuscular activity and the function of cardiac, smooth and skeletal muscles. Hyperkalaemia can cause muscle weakness and even paralysis.

- Monitor skeletal muscle strength and tone. *Increasing weakness, muscle paralysis or progression of affected muscles to affect the upper extremities or trunk can indicate increasing serum potassium levels.*

NURSING CARE PLAN A person with hyperkalaemia

Monty Longacre, a 51-year-old male, has end-stage renal failure. He arrives at the emergency clinic complaining of shortness of breath on exertion and extreme weakness.

ASSESSMENT

Mr Longacre tells the nurse, Janet Allen, RN, that he normally receives dialysis 3 times a week. He missed his last treatment, however, to attend his father's funeral. During the past several days, he has eaten a number of fresh oranges he received as a gift. Physical assessment findings include T 37.3°C, P 100, R 28, BP 168/96, 2^+ pretibial oedema and a 3.6-kg weight gain since his last haemodialysis treatment 4 days ago. Laboratory and diagnostic tests show the following abnormal results:

- K^+ 6.5 mEq/L (normal 3.5 to 5 mEq/L)
- urea 118 mg/dL (normal 7 to 18 mg/dL)
- creatinine 14 mg/dL (normal 0.7 to 1.3 mg/dL)
- HCO_3^- 17 mEq/L (normal 22 to 26 mEq/L)
- peaked T wave noted on ECG.

Mr Longacre is placed on continuous ECG monitoring and the physician prescribes haemodialysis. As an interim measure to lower the serum potassium, the medical officer prescribes $D_{50}W$ (25 g of dextrose), one ampule, to be administered intravenously with 10 units of regular insulin over 30 minutes.

DIAGNOSES

- *Activity intolerance* related to skeletal muscle weakness.
- *Risk of decreased cardiac output* related to hyperkalaemia.
- *Risk of ineffective health maintenance* related to inadequate knowledge of recommended diet.
- *Excess fluid volume* related to renal failure.

PLANNING

- Advise the person that the nurses will be monitoring and documenting in the case notes all fluid intake and urine output.
- Explain the purpose of follow up treatments of venepuncture and electrocardiographs (ECGs).
- Educate the person about the causes of hyperkalaemia.
- Explain the importance of the medically prescribed requirement of haemodialysis.
- Discuss dietary sources and provide a list of foods to prevent hyperkalaemia.

Expected outcomes

- Gradually resume usual physical activities.
- Maintain serum potassium level within normal range.
- Verbalise causes of hyperkalaemia, the importance of haemodialysis treatments as scheduled and the role of diet in preventing hyperkalaemia.

IMPLEMENTATION

- To observe, monitor and document fluid intake and fluid output.
- To follow up on the collection of serum potassium and review serum potassium results and report abnormalities to the medical officer. Attend to ECGs as ordered by the medical officer or as required and report findings to the medical officer.
- Review Mr Longacre's knowledge pertaining to causes of hyperkalaemia and the relationship between haemodialysis and hyperkalaemia.
- Discuss and consult with Mr Longacre about the importance of avoiding foods high in potassium to prevent or control hyperkalaemia.

EVALUATION

Following emergency treatment and haemodialysis, Mr Longacre's ECG and serum potassium level have returned to normal. His muscle strength has returned to near normal and he verbalises an understanding of his prescribed haemodialysis regimen. Janet Allen provides verbal and written information about hyperkalaemia, and the importance of complying with the haemodialysis regimen and of limiting intake of dietary sources of potassium in renal failure. She also furnishes a list of foods high in potassium and cautions against using potassium-containing salt substitutes and non-prescription drugs.

CRITICAL THINKING IN THE NURSING PROCESS

1. What information given by Mr Longacre indicated that he might be experiencing hyperkalaemia?
2. Why was continuous ECG monitoring instituted as an emergency measure?
3. What additional emergency measures might have been instituted if Mr Longacre's serum potassium level had been 8.5 mEq/L and his ECG had shown changes in impulse conduction?
4. Develop a care plan for Mr Longacre for the nursing diagnosis of *Anxiety*.

REFLECTION ON THE NURSING PROCESS

1. Identify and outline what you have learned from this case study and how will you apply it to your future nursing practice.
2. What communication and education strategies could you use when caring for a person with a nursing diagnosis of *Anxiety*?

- Monitor respiratory rate and depth. Regularly assess lung sounds. *Muscle weakness due to hyperkalaemia can impair ventilation. In addition, medications such as sodium bicarbonate or sodium polystyrene sulfonate can cause fluid retention and pulmonary oedema in the person with pre-existing cardiovascular disease.*
- Assist with self-care activities as needed. *Increasing muscle weakness can lead to fatigue and affect the ability to meet self-care needs.*

Risk of imbalanced fluid volume

Renal failure is a major cause of hyperkalaemia. The person with renal failure also is at risk of fluid retention and other electrolyte imbalances.

- Closely monitor serum potassium, serum urea and creatinine. Notify the physician if serum potassium level is greater than 5 mEq/L or if serum urea and creatinine levels are increasing. *Serum urea and creatinine are the primary indicators of renal function. Levels of these substances rise*

rapidly in acute renal failure, more slowly in chronic renal failure (see Chapter 27).

- Maintain accurate intake and output records. Report an imbalance of 24-hour totals and/or urine output less than 30 mL/hour. *Oliguria (scant urine) or anuria (no urine output) may indicate renal failure and an increased risk of hyperkalaemia and fluid volume excess.*
- Monitor the person receiving sodium bicarbonate for fluid volume excess. *Increased sodium from injection of a hypertonic sodium bicarbonate solution can cause a shift of water into the extracellular space.*
- Monitor the person receiving cation exchange resins and sorbitol for fluid volume excess. *The resin exchanges potassium for sodium or calcium in the bowel. Excessive sodium and water retention may occur.*

Community-based care

Preventing future episodes of hyperkalaemia is the focus when preparing the person for home care. Include the family, a significant other or a caregiver when teaching the following topics:

- recommended diet and any restrictions, including salt substitutes and foods high in potassium
- medications to be avoided, including over-the-counter and fitness supplements
- follow-up appointments for lab work and evaluation.

CALCIUM IMBALANCE

Calcium is one of the most abundant ions in the body. The normal adult total serum calcium concentration is 8.5 to 10.0 mg/dL.

Overview of normal calcium balance

Calcium is obtained from dietary sources, although only about 20% of the calcium ingested is absorbed into the blood. The remainder is excreted in faeces. Extracellular calcium is excreted by the kidneys. Approximately 99% of the total calcium in the body is bound to phosphorus to form the minerals in bones and teeth. The remaining 1% is in extracellular fluid. About half of this extracellular calcium is ionised (free); it is this ionised calcium that is physiologically active. The remaining extracellular calcium is bound to protein or other ions. Ionised calcium is essential to a number of processes: stabilising cell membranes; regulating muscle contraction and relaxation; and maintaining cardiac function and blood clotting.

Serum calcium levels are regulated by the interaction of three hormones: parathyroid hormone (PTH), calcitonin and calcitriol (a metabolite of vitamin D). When serum calcium levels fall, the parathyroid glands secrete PTH, which mobilises skeletal calcium stores, increases calcium absorption in the intestines and promotes calcium reabsorption by the kidneys (see Figure 9.12).

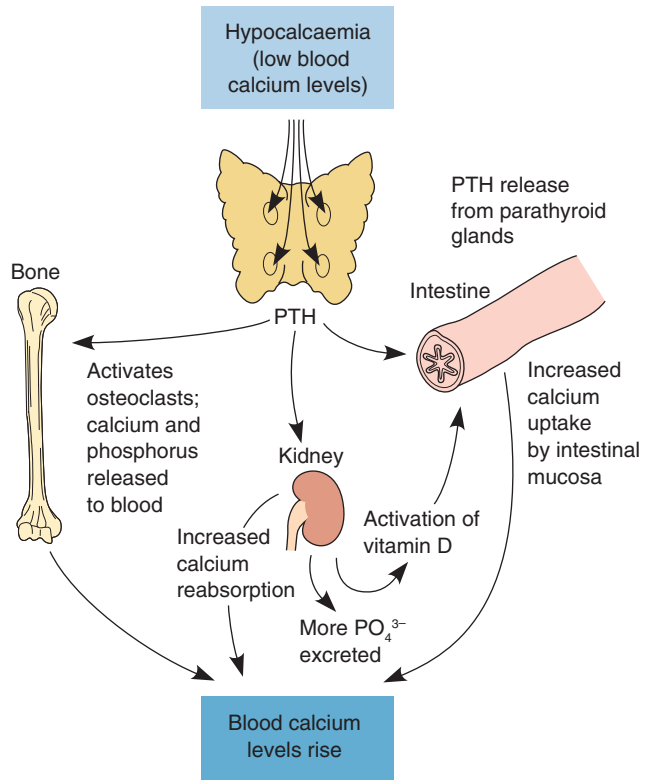


FIGURE 9.12 ■ Low calcium levels (hypocalcaemia) trigger the release of parathyroid hormone (PTH), increasing calcium ion levels through stimulation of bones, kidneys and intestines

Calcitriol facilitates this process by stimulating calcium release from the bones, absorption in the intestines and reabsorption by the kidneys. Calcitonin is secreted by the thyroid gland in response to high serum calcium levels. Its effect on serum calcium levels is the opposite of PTH: it inhibits the movement of calcium out of bone, reduces intestinal absorption of calcium, and promotes calcium excretion by the kidneys.

Serum calcium levels are also affected by acid–base balance. When hydrogen ion concentration falls and the pH rises (alkalosis), more calcium is bound to protein. While the total serum calcium remains unchanged, less calcium is available in the ionised, active form. Conversely, when hydrogen ion concentration increases and the pH falls (acidosis), calcium is released from protein, making more ionised calcium available.

Finally, the total amount of calcium in blood plasma fluctuates with plasma protein levels, particularly the albumin level. As the albumin level falls, the total amount of plasma calcium declines. Table 9.8 summarises the causes and manifestations of calcium imbalances.

The person with hypocalcaemia

Hypocalcaemia is a total serum calcium level of less than 8.5 mg/dL. Hypocalcaemia can result from decreased total body calcium stores or low levels of extracellular calcium with normal amounts of calcium stored in bone. The systemic effects of hypocalcaemia are caused by decreased levels of ionised calcium in extracellular fluid.

TABLE 9.8 Causes and manifestations of calcium imbalances

IMBALANCE	CAUSES	MANIFESTATIONS
Hypocalcaemia Serum calcium < 8.5 mg/dL or 4.3 mEq/L Critical value < 6.0 mg/dL	• Parathyroidectomy or neck surgery • Acute pancreatitis • Inadequate dietary intake • Lack of sun exposure • Lack of weight-bearing exercise • Drugs: loop diuretics, calcitonin • Hypomagnesaemia, alcohol abuse • Acute renal failure with hyperphosphataemia	Neuromuscular • Tetany • Paraesthesias • Muscle spasms • Positive Chvostek's sign • Positive Trousseau's sign • Laryngospasm • Seizures • Anxiety, confusion, psychoses Cardiovascular • Decreased cardiac output • Hypotension • Arrhythmias Gastrointestinal • Abdominal cramping • Diarrhoea
Hypercalcaemia Serum calcium >10 mg/dL or 5.3 mEq/L Critical value >13.0 mg/dL	• Hyperparathyroidism • Some cancers • Prolonged immobilisation • Paget's disease • Excess milk or antacid intake • Chronic renal failure with associated hyperparathyroidism	Neuromuscular • Muscle weakness, fatigue • Decreased deep tendon reflexes Behavioural • Personality changes • Altered mental status • Decreasing level of consciousness Gastrointestinal • Abdominal pain • Constipation • Anorexia, nausea, vomiting Cardiovascular • Arrhythmias • Hypertension Renal • Polyuria, thirst

Risk factors

Certain populations of people are at greater risk of hypocalcaemia: people who have had a parathyroidectomy (removal of the parathyroid glands), older adults (especially women), people with lactose intolerance and those with alcoholism. People who have undergone bariatric surgery for weight loss are at risk due to decreased food intake and malabsorption (Dewey & Heuberger, 2011). Older adults often consume less milk and milk products (good sources of calcium) and may have less exposure to the sun (a source of vitamin D). Older adults also may be less active, promoting calcium loss from bones. They are more likely to be taking drugs that interfere with calcium absorption or promote calcium excretion (e.g. frusemide). Older women are at particular risk after menopause because of reduced oestrogen levels. Intolerance to lactose (found in milk and milk products) causes diarrhoea and often limits the intake of milk and milk products, leading to possible calcium deficiency. Ethanol, or drinking alcohol, has a direct effect on calcium balance, reduces its intestinal absorption and interferes with other processes involved in regulating serum calcium levels.

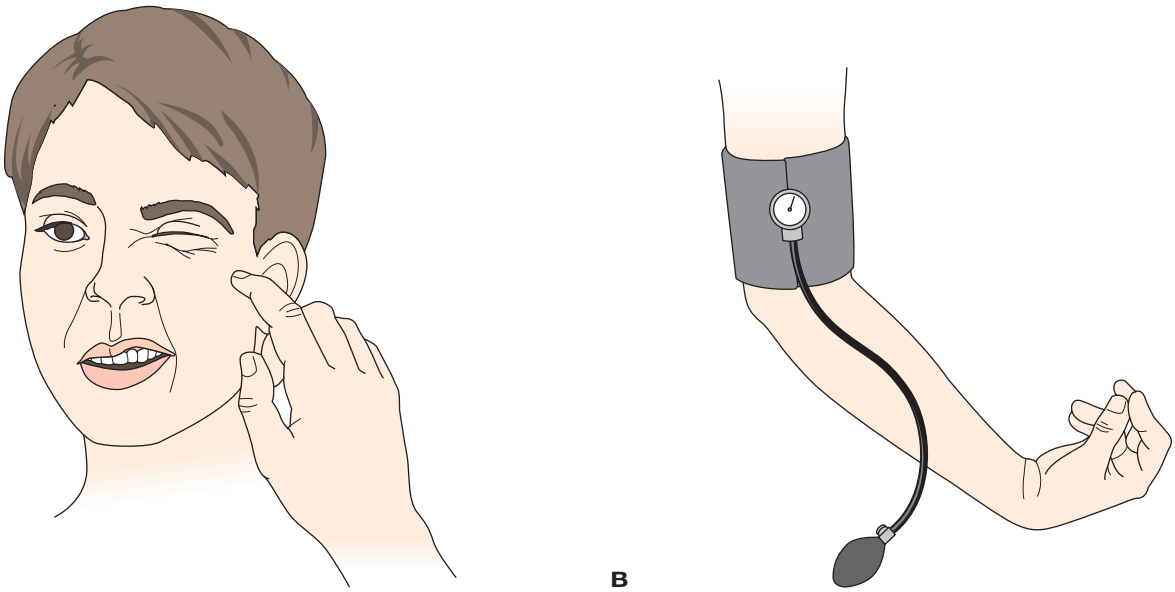
Pathophysiology

Common causes of hypocalcaemia are hypoparathyroidism (see Chapter 18) resulting from surgery (parathyroidectomy, thyroidectomy, radical neck dissection) and acute pancreatitis. In the person who has undergone surgery, symptoms of hypocalcaemia usually occur within the first 24 to 48 hours, but may be delayed.

CONSIDERATION FOR PRACTICE

Carefully monitor the person who has undergone neck surgery for manifestations of hypocalcaemia. Check serum calcium levels and report changes.

Additional causes of hypocalcaemia include other electrolyte imbalances (such as hypomagnesaemia or hyperphosphataemia), alkalosis, malabsorption disorders that interfere with calcium absorption in the bowel, and inadequate vitamin D (due to lack of sun exposure or malabsorption). Hyperphosphataemia often occurs in acute renal failure, with reciprocal hypocalcaemia. Massive transfusion of banked blood also can lead to hypocalcaemia. Citrate is added to blood to prevent clotting and as a preservative. When blood is administered faster than the liver can metabolise the citrate, it can bind with calcium, temporarily removing ionised calcium from circulation. Many drugs increase the risk of hypocalcaemia, including



A B

FIGURE 9.13 ■ *A*, Positive Chvostek's sign. *B*, Positive Trousseau's sign

loop diuretics (such as frusemide), anticonvulsants (such as phenytoin and phenobarbital), phosphates (including phosphate enemas) and drugs that lower serum magnesium levels (such as cisplatin and gentamicin) (Metheny, 2012).

Extracellular calcium acts to stabilise neuromuscular cell membranes. This effect is reduced in hypocalcaemia, increasing neuromuscular irritability. The threshold of excitation of sensory nerve fibres is lowered as well, leading to paraesthesias (altered sensation). The nervous system becomes more excitable and muscle spasms develop. In the heart, this change in cell membranes can lead to arrhythmias such as ventricular tachycardia and cardiac arrest. Hypocalcaemia decreases the contractility of cardiac muscle fibres, leading to decreased cardiac output.

Manifestations and complications

The most serious manifestations of hypocalcaemia are **tetany** (tonic muscular spasms) and convulsions. Numbness and tingling around the mouth (circumoral) and in the hands and feet develop. Muscle spasms of the face and extremities occur and deep tendon reflexes become hyperactive. Chvostek's sign, contraction of the facial muscles produced by tapping the facial nerve in front of the ear (see Figure 9.13A), and Trousseau's sign, carpal spasm induced by inflating a blood pressure cuff on the upper arm to above systolic blood pressure for 2 to 5 minutes (see Figure 9.13B), indicate increased neuromuscular excitability in the person without obvious symptoms.

Tetany can cause bronchial muscle spasms, simulating an asthma attack and visceral muscle spasms, producing acute abdominal pain. Cardiovascular manifestations include hypotension, possible bradycardia (slow heart rate) and ventricular arrhythmias.

Serious complications of hypocalcaemia include airway obstruction and possible respiratory arrest from laryngospasm, ventricular arrhythmias and cardiac arrest, heart failure and convulsions.

INTERPROFESSIONAL CARE

Management of hypocalcaemia is directed towards restoring normal calcium balance and correcting the underlying cause.

Diagnosis

The following laboratory and diagnostic tests may be ordered when hypocalcaemia is known or suspected.

- *Total serum calcium*, the amount of ionised (active) calcium available, is usually estimated. In the person who is critically ill, however, *ionised calcium* may be directly measured using ion selective electrodes. Direct measurement of ionised calcium requires special handling of the blood specimen, including placing the specimen on ice and analysing it immediately.
- *Serum albumin*, because the albumin level affects serum calcium results. When the albumin level is low (hypoalbuminaemia), the amount of ionised calcium may remain normal even though the total calcium level is low.
- *Serum magnesium*, because hypocalcaemia is often associated with hypomagnesaemia (serum magnesium < 1.6 mg/dL). In this case, normal magnesium levels must be restored to correct the hypocalcaemia.
- *Serum phosphate*; hyperphosphataemia (serum phosphate > 4.5 mg/dL) can lead to hypocalcaemia because of the inverse relationship between phosphorus and calcium. (As phosphate levels rise, calcium levels fall.)
- *Parathyroid hormone (PTH)*, to identify the possible diagnoses of hyperparathyroidism.
- An *ECG*, to evaluate the effects of hypocalcaemia on the heart, such as a prolonged ST segment.

Medications

Hypocalcaemia is treated with oral or intravenous calcium. The person with severe hypocalcaemia is treated with intravenous

BOX 9.7 Foods high in calcium

- Cottage cheese
- Canned sardines and salmon
- Cheese
- Rhubarb
- Milk
- Broccoli
- Cream
- Yoghurt
- Ice-cream
- Spinach
- Tofu

calcium to prevent life-threatening problems such as airway obstruction. The most common intravenous calcium preparations include calcium chloride and calcium gluconate. Although calcium chloride contains more elemental calcium than calcium gluconate, it also is more irritating to the veins and may cause venous sclerosis (hardening of the vein walls) if given into a peripheral vein. Intravenous calcium preparations can cause necrosis and sloughing of tissue if they extravasate into subcutaneous tissue. Rapid drug administration can lead to bradycardia and possible cardiac arrest due to overcorrection of hypocalcaemia with resulting hypercalcaemia. See the 'Medication administration' box below for further information on calcium administration.

Oral calcium preparations (calcium carbonate, calcium gluconate or calcium lactate) are used to treat chronic, asymptomatic hypocalcaemia. Calcium supplements may be combined with vitamin D, or vitamin D may be given alone to increase gastrointestinal absorption of calcium.

Nutrition

A diet high in calcium-rich foods may be recommended for the person with chronic hypocalcaemia or with low total body stores of calcium. Box 9.7 lists foods that are high in calcium.

Nursing care

Health promotion

Because of the large stores of calcium in bones, most healthy adults have a very low risk of developing hypocalcaemia. However, a deficit of total body calcium is often associated with ageing, increasing the risk of osteoporosis, fractures and disability. Women have a higher risk of developing osteoporosis than men, due to lower bone density and hormonal influences. Educate women of all ages about the importance of maintaining adequate calcium intake through diet and, as needed, calcium supplements. Stress the relationship between weight-bearing exercise and bone density, and encourage women to engage in a regular aerobic and weight-training exercise regimen. Discuss hormone replacement therapy and its potential benefits during and after menopause. See Chapter 39 for more information about osteoporosis.

Assessment

Assessment data related to hypocalcaemia include the following:

- *Health history*: current manifestations, including numbness and tingling around mouth and of hands and feet, abdominal pain, shortness of breath; acute or chronic

MEDICATION ADMINISTRATION Calcium salts

CALCIUM SALTS

Calcium carbonate
Calcium chloride
Calcium citrate
Calcium gluconate
Calcium lactate

Calcium salts are given to increase calcium levels when there is a deficit (a total body deficit or inadequate levels of extracellular calcium). Calcium is necessary to maintain bone structure and for multiple physiological processes, including neuromuscular and cardiac function as well as blood coagulation. In the presence of vitamin D, calcium is well absorbed from the gastrointestinal tract. Severe hypocalcaemia is treated with intravenous calcium preparations.

Nursing responsibilities

Oral calcium salts:

- Administer 1 to 1.5 hours after meals and at bedtime.
- Give calcium tablets with a full glass of water.

Intravenous calcium salts:

- Assess IV site for patency. Do not administer calcium if there is a risk of leakage into the tissues.
- May be given by slow IV push (dilute with sterile normal saline for injection prior to administering) or added to compatible parenteral fluids such as NS, lactated Ringer's solution or D_5W.
- Administer into the largest available vein; use a central line if available.
- Do not administer with bicarbonate or phosphate because a precipitate (insoluble salt) will form (Metheny, 2012).
- Continuously monitor ECG when administering IV calcium to the person taking digitalis, due to increased risk of digitalis toxicity.
- Frequently monitor serum calcium levels and response to therapy.

Health education for the person and their family

- Take calcium tablets with a full glass of water 1 to 2 hours after meals. Do not take with food or milk. If possible, do not take within 1 to 2 hours of other medications.
- Maintain adequate vitamin D intake through diet or exposure to the sun to promote calcium absorption.
- Calcium carbonate can cause constipation. Eat a high-fibre diet and maintain a generous fluid intake to prevent constipation.

diseases such as pancreatitis, liver or kidney disease; current medications.
- *Physical assessment*: muscle spasms; deep tendon reflexes; Chvostek's sign and Trousseau's sign; respiratory rate and depth; vital signs and apical pulse; heart rate and rhythm; presence of convulsions.
- *Diagnostic tests*: serum electrolytes (calcium, magnesium, phosphate and potassium, in particular), serum albumin, thyroid and parathyroid hormone levels; ECG.

Nursing diagnoses and interventions

The effect of hypocalcaemia on neuromuscular irritability, with the risk of muscle spasm and convulsions, is the highest priority for nursing care of the person.

Risk of injury

The person with hypocalcaemia is at risk of injury from possible laryngospasm, cardiac arrhythmias or convulsions. In addition, too rapid administration of intravenous calcium or extravasation of the medication into subcutaneous tissues can lead to injury.

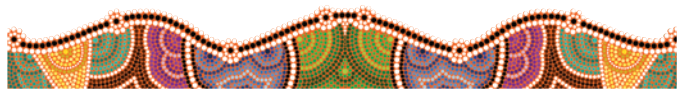
- Frequently monitor airway and respiratory status. Report changes such as respiratory **stridor** (a high-pitched, harsh inspiratory sound indicative of upper airway obstruction), or increased respiratory rate or effort, to the physician. *These changes may indicate laryngeal spasm due to tetany.*

CONSIDERATION FOR PRACTICE
Laryngeal spasm is a respiratory emergency, requiring immediate intervention to maintain ventilation and gas exchange.

- Monitor cardiovascular status, including heart rate and rhythm, blood pressure and peripheral pulses. *Hypocalcaemia decreases myocardial contractility, causing reduced cardiac output and hypotension. It also can cause bradycardia or ventricular arrhythmias. Cardiac arrest may occur in severe hypocalcaemia.*
- Continuously monitor ECG in the person receiving intravenous calcium preparations, especially if the person also is taking digitalis. *Rapid administration of calcium salts can lead to hypercalcaemia and cardiac arrhythmias. Calcium administration increases the risk of digitalis toxicity and resultant arrhythmias.*
- Provide a quiet environment. Institute seizure precautions such as raising the side rails and keeping an airway at the bedside. *A quiet environment reduces central nervous system stimuli and the risk of convulsions in the person with tetany.*

Community-based care

In preparing the person with hypocalcaemia for discharge and home care, consider the circumstances leading to low serum calcium levels. Discuss risk factors for hypocalcaemia specific to the person and provide information about managing these risk factors to avoid future episodes of hypocalcaemia. Educate about prescribed medications, including calcium supplements. Provide a list of foods high in calcium, as well as sources of vitamin D if recommended. Discuss symptoms to report to the attending doctor and stress the importance of follow-up care as scheduled.



The person with hypercalcaemia

Hypercalcaemia is a serum calcium value greater than 10.0 mg/dL. Excess ionised calcium in ECF can have serious widespread effects.

Pathophysiology

Hypercalcaemia usually results from increased resorption of calcium from the bones. The two most common causes of bone resorption are hyperparathyroidism and malignancies. In hyperparathyroidism, excess PTH is produced. This causes calcium to be released from bones, as well as increased calcium absorption in the intestines and retention of calcium by the kidneys. Hypercalcaemia is a common complication of malignancies. It may develop as a result of bone destruction by the tumour or due to hormone-like substances produced by the tumour itself. Prolonged immobility and lack of weight-bearing activity also causes increased resorption of bone with calcium release into extracellular fluids. Self-limiting hypercalcaemia also may follow successful kidney transplant. Levels of parathyroid hormone may be altered in chronic renal failure, leading to increased serum calcium levels.

Increased intestinal absorption of calcium also can lead to hypercalcaemia. This may result from excess vitamin D, overuse of calcium-containing antacids or excessive milk ingestion. Renal failure and some drugs such as thiazide diuretics and lithium can interfere with elimination of calcium by the kidneys, causing high serum calcium levels.

The effects of hypercalcaemia largely depend on the degree of serum calcium elevation and the length of time over which it develops. In general, higher serum calcium levels are associated with more serious effects. Calcium has a stabilising effect on the neuromuscular junction; hypercalcaemia decreases neuromuscular excitability, leading to muscle weakness and depressed deep tendon reflexes. Gastrointestinal motility is reduced as well. In the heart, calcium exerts an effect similar to digitalis (see Chapter 30), strengthening contractions and reducing the heart rate. Hypercalcaemia affects the conduction system of the heart, leading to bradycardia and heart blocks. The ability of the kidneys to concentrate urine is impaired by hypercalcaemia, causing excess sodium and water loss and increased thirst.

Extremely high serum calcium levels affect mental status. This is thought to be due to increased calcium in cerebrospinal fluid. Behavioural effects range from personality changes to confusion, impaired memory and acute psychoses.

Manifestations and complications

Manifestations of hypercalcaemia relate to its effects on neuromuscular activity, the central nervous system (CNS), the cardiovascular system and the kidneys. Decreased neuromuscular

excitability causes muscle weakness and fatigue, as well as gastrointestinal manifestations such as anorexia, nausea, vomiting and constipation. CNS effects may include confusion, lethargy, behaviour or personality changes, and coma. Cardiovascular effects include arrhythmias, ECG changes and possible hypertension. Hypercalcaemia causes polyuria and, as a result, increased thirst.

Complications of hypercalcaemia can affect several different organ systems. Peptic ulcer disease may develop due to increased gastric acid secretion. Pancreatitis can occur as a result of calcium deposits in pancreatic ducts. Excess calcium can precipitate out of urine to form kidney stones. Hypercalcaemic crisis, an acute increase in the serum calcium level, can lead to cardiac arrest.

INTERPROFESSIONAL CARE

The management of hypercalcaemia focuses on correcting the underlying cause and reducing the serum calcium level. Treatment is particularly important in the person who has one or more of the following: serum calcium levels greater than 12 mg/dL, overt symptoms of hypercalcaemia, compromised renal function and inability to maintain an adequate fluid intake.

Diagnosis

The laboratory and diagnostic tests that may be ordered and the resultant findings are as follows:

- *Serum electrolytes* show a total serum calcium greater than 10.0 mg/dL.
- *Serum PTH* levels are measured to identify or rule out hyperparathyroidism as the cause of hypercalcaemia.
- *ECG* changes in hypercalcaemia include a shortened QT interval, shortened and depressed ST segment and widened T wave. Bradycardia or heart block may be identified on the ECG.
- *Bone density* scans may be done to monitor bone resorption and the effects of treatment measures on mineralisation of bone.

Medications

Measures to promote calcium elimination by the kidneys and reduce calcium resorption from bone are used to treat hypercalcaemia. In acute hypercalcaemia, intravenous fluids are given (see the 'Fluid management' section that follows) with a loop diuretic such as frusemide to promote elimination of excess calcium. Calcitonin, which promotes the uptake of calcium into bones, also may be used to rapidly lower serum calcium levels.

A number of drugs that inhibit bone resorption are available. The bisphosphonates (pamidronate and etidronate) are commonly used to treat hypercalcaemia associated with malignancies. These drugs also are used to prevent and treat osteoporosis. Nursing implications for calcitonin and bisphosphonate drugs are presented in the 'Medication administration' boxes in Chapter 39. When a bisphosphonate drug is ineffective in correcting hypercalcaemia, mithramycin (a chemotherapeutic agent) may be used.

Rapid reversal of hypercalcaemia in emergency situations may be accomplished by intravenous administration of sodium phosphate or potassium phosphate. Calcium binds to phosphate, thus decreasing serum calcium levels. Paradoxically, complications of this therapy can include fatal hypocalcaemia resulting from binding of the ionised calcium and soft tissue calcifications.

Other drug therapies include the use of intravenous plicamycin to inhibit bone resorption. Glucocorticoids (cortisone), which compete with vitamin D, and a low-calcium diet may be prescribed to decrease gastrointestinal absorption of calcium, inhibit bone resorption and increase urinary calcium excretion. Also, calcitonin may be prescribed to decrease skeletal mobilisation of calcium and phosphorus, and to increase renal output of calcium and phosphorus. See Chapter 18 for more information about and nursing implications of glucocorticoid therapy.

Fluid management

Intravenous fluids, usually isotonic saline, are administered to the person with severe hypercalcaemia to restore vascular volume and promote renal excretion of calcium. Isotonic saline is used because sodium excretion is accompanied by calcium excretion. Careful assessment of cardiovascular and renal function is done prior to fluid therapy; the person is carefully monitored for evidence of fluid overload during treatment.

Nursing care

Health promotion

Identify and monitor the person at risk of hypercalcaemia. Promote mobility in the person when possible. Assist the hospitalised person to ambulate as soon as possible. In the home setting, discuss the benefits of regular weight-bearing activity with the person, families and caregivers. Encourage a generous fluid intake of up to 3 to 4 L per day. Encourage the person at risk to limit their intake of milk and milk products, as well as calcium-containing antacids and supplements. In addition, the person with prolonged immobility or hypercalcaemia is encouraged to consume fluids that increase the acidity of urine (which inhibits calcium stone formation), such as cranberry or prune juice.

Assessment

Assessment data related to hypercalcaemia include the following:

- *Health history*: current manifestations, including weakness or fatigue, abdominal discomfort, nausea or vomiting, increased urination and thirst; changes in memory or thinking; duration of symptoms and any risk factors such as excess intake of milk or calcium products, prolonged immobility, malignancy, renal failure or endocrine disorders; current medications.
- *Physical assessment*: mental status and level of consciousness; vital signs, including apical pulse; bowel sounds; muscle strength of upper and lower extremities; deep tendon reflexes.
- *Diagnostic tests*: serum electrolytes, urinary calcium, ECG and cardiac rhythm monitoring.

CONSIDERATION FOR PRACTICE

Remember, calcium has a stabilising or sedative effect on neuromuscular transmission. Therefore:

- Hypocalcaemia → increased neuromuscular excitability, muscle twitching, spasms and possible tetany
- Hypercalcaemia → decreased neuromuscular excitability, muscle weakness and fatigue.

Nursing diagnoses and interventions

Risk of injury

The person with hypercalcaemia is at risk of injury due to changes in mental status, the effects of hypercalcaemia on muscle strength and loss of calcium from bones.

- Institute safety precautions if confusion or other changes in mental status are noted. *Changes in mental status may impair judgment and the person's ability to maintain own safety.*

CONSIDERATION FOR PRACTICE

Monitor cardiac rate and rhythm, treating and/or reporting arrhythmias as indicated. Prepare for possible cardiac arrest; keep emergency resuscitation equipment readily available. Hypercalcaemia can cause bradycardia, various heart blocks and cardiac arrest. Immediate treatment may be necessary to preserve life.

- Observe for manifestations of digitalis toxicity, including vision changes, anorexia, and changes in heart rate and rhythm. Monitor serum digitalis levels. *Hypercalcaemia increases the risk of digitalis toxicity.*
- Promote fluid intake (oral and/or intravenous) to keep the person well hydrated and maintain dilute urine. Encourage fluids such as prune or cranberry juice to help maintain acidic urine. *Acidic, dilute urine reduces the risk of calcium salts precipitating out to form kidney stones.*
- If excess bone resorption has occurred, use caution when turning, positioning, transferring or ambulating. *Bones that have lost excess calcium may fracture with minimal stress or trauma (pathological fractures).*

Risk of excess fluid volume

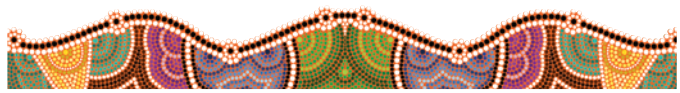
Large amounts of isotonic intravenous fluid often are administered to help correct acute hypercalcaemia, leading to a risk of hypervolaemia. The person with pre-existing cardiac or renal disease is at particular risk.

- Closely monitor intake and output. *A loop diuretic such as frusemide may be necessary if urinary output does not keep up with fluid administration.*
- Frequently assess vital signs, respiratory status and heart sounds. *Increasing pulse rate, dyspnoea, adventitious lung sounds and an S_3 on auscultation of the heart may indicate excess fluid volume and potential heart failure.*
- Place in semi-Fowler's to Fowler's position. *Elevating the head of the bed improves lung expansion and reduces the work of breathing.*
- Administer diuretics as ordered, monitoring response. *Loop diuretics may be ordered to help eliminate excess fluid and calcium.*

Community-based care

Discuss the following topics when preparing the person for discharge:

- Avoid excess intake of calcium-rich foods and antacids.
- Use prescribed drugs to prevent excess calcium resorption. Discuss their dose, use, and desired and possible adverse effects.
- Increase fluid intake to 3 to 4 L per day; increase the intake of acid ash foods (meats, fish, poultry, eggs, cranberries, plums, prunes); increase dietary fibre and fluid intake to prevent constipation.
- Maintain weight-bearing physical activity to prevent hypercalcaemia.
- Report early manifestations of hypercalcaemia.
- Follow recommended schedule for monitoring serum electrolyte levels.



MAGNESIUM IMBALANCE

Only about 1% of the magnesium in the body is in extracellular fluid; the rest is found within the cells and in bone. The normal serum concentration of magnesium ranges from 1.8 to 2.6 mg/dL (1.5 to 2.5 mEq/L).

Overview of normal magnesium balance

Magnesium is obtained through the diet (it is plentiful in green vegetables, grains, nuts, meats and seafood) and is excreted by the kidneys. Magnesium is vital to many intracellular processes, including enzyme reactions and synthesis of proteins and nucleic acids. Magnesium exerts a sedative effect on the neuromuscular junction, decreasing acetylcholine release. It is an essential ion for neuromuscular transmission and cardiovascular function. The physiological effects of magnesium are affected by both potassium and calcium levels. Approximately 65% of extracellular magnesium is ionised; the remainder is bound to protein. Table 9.9 summarises common causes and manifestations of magnesium imbalances.

The person with hypomagnesaemia

Hypomagnesaemia is a magnesium level of less than 1.6 mg/dL. It is a common problem, particularly in the critically ill person. Hypomagnesaemia may be caused by deficient magnesium intake, excessive losses, or a shift between the intracellular and extracellular compartments.

Risk factors

Loss of gastrointestinal fluids, particularly from diarrhoea, an ileostomy or intestinal fistula, is a major risk factor for hypomagnesaemia. Disruption of nutrient absorption in the small intestine also increases the risk. Chronic alcoholism is a common cause of deficient magnesium levels. Multiple factors associated with alcoholism contribute to hypomagnesaemia: deficient nutrient intake, increased gastrointestinal losses, impaired absorption and increased renal excretion. Other risk factors for hypomagnesaemia include:

- protein–kilojoule malnutrition or starvation
- endocrine disorders, including diabetic ketoacidosis

TABLE 9.9 Causes and manifestations of magnesium imbalances

IMBALANCE	CAUSES	MANIFESTATIONS
Hypomagnesaemia Serum magnesium < 1.6 mg/dL Critical value < 1 mg/dL	• Chronic alcoholism • GI losses: intestinal suction, diarrhoea, ileostomy • Impaired absorption • Inadequate replacement • Increased excretion: drugs, renal disease, osmotic diuresis	Neuromuscular • Muscle weakness, tremors • Tetany, seizures Gastrointestinal • Dysphagia • Anorexia, nausea, vomiting, diarrhoea Cardiovascular • Tachycardia • Arrhythmias • Hypertension CNS • Mood and personality changes • Paraesthesias
Hypermagnesaemia Serum magnesium > 2.6 mg/dL or 2.1 mEq/L Critical value > 4.7 mg/dL	• Renal insufficiency or failure • Excess intake of antacids, laxatives • Excess magnesium administration	Neuromuscular • Muscle weakness • Depressed deep tendon reflexes Gastrointestinal • Nausea and vomiting Cardiovascular • Hypotension • Bradycardia • Cardiac arrest CNS • Respiratory depression • Coma

- drugs such as loop or thiazide diuretics, aminoglycoside antibiotics, amphotericin B and cyclosporin
- rapid administration of citrated blood (banked blood)
- kidney disease.

Pathophysiology

Magnesium deficiency usually occurs along with low serum potassium and calcium levels. The effects of hypomagnesaemia relate not only to the magnesium deficiency but also to hypokalaemia and hypocalcaemia.

Hypomagnesaemia causes increased neuromuscular excitability, with muscle weakness and tremors. The accompanying hypocalcaemia contributes to this effect. In the CNS, this increased neural excitability can lead to seizures and changes in mental status. Deficient intracellular magnesium in the myocardium increases the risk of cardiac arrhythmias and sudden death. Hypokalaemia increases this risk. Hypomagnesaemia also increases the risk of digitalis toxicity. Chronic hypomagnesaemia may contribute to hypertension, probably due to increased vasoconstriction.

Manifestations and complications

Neuromuscular manifestations of hypomagnesaemia include tremors, hyper-reactive reflexes, positive Chvostek's and Trousseau's signs (see Figure 9.13), tetany, paraesthesias and seizures. CNS effects include confusion, mood changes (apathy, depression, agitation), hallucinations and possible psychoses. An increased heart rate and ventricular arrhythmias are common, especially when hypokalaemia is present or the person is taking digitalis. Cardiac arrest and sudden death may occur. Gastrointestinal manifestations include nausea, vomiting, anorexia, diarrhoea and abdominal distension.

INTERPROFESSIONAL CARE

Hypomagnesaemia is diagnosed by measuring serum electrolyte levels. The ECG shows a prolonged PR interval, widened QRS complex and depression of the ST segment with T-wave inversion. Treatment is directed towards prevention and identification of an existing deficiency. Magnesium is added to intravenous total parenteral nutrition solutions to prevent hypomagnesaemia.

In the person able to eat, a mild deficiency may be corrected by increasing the intake of foods rich in magnesium (see Box 9.8) or with oral magnesium supplements. Oral magnesium supplements may cause diarrhoea, however, limiting their use.

BOX 9.8 Foods high in magnesium

- Green, leafy vegetables
- Oranges
- Seafood
- Grapefruit
- Meat
- Chocolate
- Wheat bran
- Milk
- Coconut
- Legumes
- Refined sugar
- Bananas

The person with manifestations of hypomagnesaemia is treated with parenteral magnesium sulfate. Treatment is continued for several days to restore intracellular magnesium levels. Magnesium may be given intravenously or by deep intramuscular injection. Renal function is evaluated prior to administration, and serum magnesium levels are monitored during treatment. The intravenous route is used for severe magnesium deficiency or if neurological changes or cardiac arrhythmias are present. See the 'Medication administration' box below for the nursing implications of parenteral magnesium sulfate.

Nursing care

Health promotion

Discuss the importance of maintaining adequate magnesium intake through a well-balanced diet, particularly with those at risk (people with alcoholism, malabsorption or bowel surgery). Many hospitalised people are at risk of hypomagnesaemia due to protein–kilojoule malnutrition and other disorders. Monitor serum magnesium levels, reporting changes to the healthcare provider.

Assessment

In addition to asking questions related to risk factors for hypomagnesaemia, use the guidelines for assessing the person with hypokalaemia and hypocalcaemia for subjective and objective assessment data. Monitor diagnostic studies such as serum electrolytes, serum albumin levels and the ECG. Monitor GI function, including bowel sounds and abdominal distension.

Nursing diagnoses and interventions

Nursing care for the person with hypomagnesaemia focuses on careful monitoring of manifestations and responses to treatment, promoting safety, the person and family education, and administering prescribed medications.

Risk of injury

- Monitor serum electrolytes, including magnesium, potassium and calcium. *Magnesium deficiency often is accompanied by deficiencies of potassium and calcium.*
- Monitor gastrointestinal function, including bowel sounds and abdominal distension. *Hypomagnesaemia reduces gastrointestinal motility.*
- Initiate cardiac monitoring, reporting and treating (as indicated) ECG changes and arrhythmias. In the person receiving digitalis, monitor for digitalis toxicity. *Low magnesium levels can precipitate ventricular arrhythmias, including lethal arrhythmias such as ventricular fibrillation.*
- Assess deep tendon reflexes frequently during intravenous magnesium infusions and prior to each intramuscular dose. *Depressed tendon reflexes indicate a high serum magnesium level.*
- Maintain a quiet, darkened environment. Institute seizure precautions. *Increased neuromuscular and CNS irritability can lead to seizures. A quiet, dark environment reduces stimuli.*

Community-based care

Prior to discharge, instruct the person to increase dietary intake of foods high in magnesium and provide information about magnesium supplements. In addition, if alcohol abuse has precipitated a magnesium deficit, discuss alcohol treatment options, including inpatient treatment and support groups such as Alcoholics Anonymous, Al-Anon and/or Al Teen.

The person with hypermagnesaemia

Hypermagnesaemia is a serum magnesium level greater than 2.6 mg/dL. It is much less common than hypomagnesaemia. Hypermagnesaemia can develop in renal failure, particularly if magnesium is administered parenterally or orally (e.g. magnesium-containing antacids or laxatives). Older adults are at risk of hypermagnesaemia, as renal function declines with

MEDICATION ADMINISTRATION Magnesium sulfate

Magnesium sulfate is used to prevent or treat hypomagnesaemia. It is also used as an anticonvulsant in severe eclampsia or pre-eclampsia. It may be given intravenously or by intramuscular injection.

NURSING RESPONSIBILITIES

- Assess serum magnesium levels and renal function tests (serum urea and creatinine) prior to administering. Notify the attending doctor if magnesium levels are above normal limits or renal function is impaired.
- Frequently monitor neurological status and deep tendon reflexes during therapy. Withhold magnesium and notify the attending doctor if deep tendon reflexes are hypoactive or absent.
- Monitor intake and output.
- Administer IM doses deep into the ventral or dorsal gluteal sites.
- Intravenous magnesium sulfate may be given slowly by IV or by continuous infusion.

HEALTH EDUCATION FORTHE PERSON AND FAMILY

Explain purpose and duration of treatment. Discuss reason for frequent neurological and reflex assessments.

ageing and they are more likely to use over-the-counter laxatives and other preparations that contain magnesium.

Pathophysiology and manifestations

Elevated serum magnesium levels interfere with neuromuscular transmission and depress the central nervous system. Hypermagnesaemia also affects the cardiovascular system, potentially causing hypotension, flushing, sweating and brady-arrhythmias.

Predictable manifestations occur with increasing serum magnesium levels. With lower levels, nausea and vomiting, hypotension, facial flushing, sweating and a feeling of warmth occur. As levels increase, signs of CNS depression appear (weakness, lethargy, drowsiness, weak or absent deep tendon reflexes). Marked elevations cause respiratory depression, coma and compromised cardiac function (ECG changes, bradycardia, heart block and cardiac arrest).

INTERPROFESSIONAL CARE

The management of hypermagnesaemia focuses on identifying and treating the underlying cause. All medications or compounds containing magnesium (such as antacids, intravenous solutions or enemas) are withheld. In the person with renal failure, haemodialysis or peritoneal dialysis is instituted to remove the excess magnesium.

Calcium gluconate is administered intravenously to reverse the neuromuscular and cardiac effects of hypermagnesaemia. The person may require mechanical ventilation to support respiratory function and a pacemaker to maintain adequate cardiac output.

Nursing care

Nursing care includes instituting measures to prevent and identify hypermagnesaemia in the person at risk, monitoring for critical effects of hypermagnesaemia and providing measures to ensure the person's safety. Consider the following nursing diagnoses for the person with hypermagnesaemia:

- *Decreased cardiac output* related to altered myocardial conduction.
- *Risk of ineffective breathing pattern* related to respiratory depression.
- *Risk of injury* related to muscle weakness and altered level of consciousness.
- *Risk of ineffective health maintenance* related to lack of knowledge about use of magnesium-containing supplements, antacids, laxatives and enemas.

Community-based care

Discharge teaching and planning focus on instructions to avoid magnesium-containing medications, including antacids, mineral supplements, cathartics and enemas (see Box 9.9).

BOX 9.9 Medications containing magnesium

Antacids
- Gelusil
- Milk of magnesia
- Mylanta
- Gaviscon

Laxatives
- Milk of magnesia
- Magnesium oxide
- Magnesium citrate
- Epsom salts

PHOSPHATE IMBALANCE

Although most phosphate (85%) is found in bones, it is the primary intracellular anion. About 14% is in intracellular fluid and the remainder (1%) is in extracellular fluid. The normal serum phosphate (or phosphorus) level in adults is 2.5 to 4.5 mg/dL. Phosphorus levels vary with age, gender and diet.

Overview of normal phosphate balance

Phosphate is essential to intracellular processes such as the production of ATP, the fuel that supports muscle contraction, nerve cell transmission and electrolyte transport. Phosphate is vital for red blood cell function and oxygen delivery to tissues; nervous system and muscle function; and the metabolism of fats, carbohydrates and protein. It also assists in maintaining acid–base balance.

Phosphorus is ingested in the diet, absorbed in the jejunum and primarily excreted by the kidneys. When phosphate intake is low, the kidneys conserve phosphorus, excreting less. An inverse relationship exists between phosphate and calcium levels: when one increases, the other decreases. Regulatory mechanisms for calcium levels (parathyroid hormone, calcitonin and vitamin D) also influence phosphate levels. The causes and manifestations of phosphate imbalances are summarised in Table 9.10.

The person with hypophosphataemia

Hypophosphataemia is a serum phosphorus of less than 2.5 mg/dL. Low serum phosphate levels may indicate a total body deficit of phosphate or a shift of phosphate into the intracellular space, the most common cause of hypophosphataemia. Decreased gastrointestinal absorption of phosphate or increased renal excretion of phosphate also can cause low phosphate levels. Hypophosphataemia often is *iatrogenic*—that is, related to treatment. Selected causes of hypophosphataemia include the following:

- *Refeeding syndrome* can develop when the malnourished person is started on enteral or total parenteral nutrition. Glucose in the formula or solution stimulates insulin release, which promotes the entry of glucose and phosphate into the cells, depleting extracellular phosphate levels.
- Medications frequently contribute to hypophosphataemia, including intravenous glucose solutions, antacids (aluminium- or magnesium-based antacids bind with phosphate), anabolic steroids and diuretics.
- Alcoholism affects both the intake and absorption of phosphate.

TABLE 9.10 Causes and manifestations of phosphate imbalances

IMBALANCE	CAUSES	MANIFESTATIONS
Hypophosphataemia Serum phosphorus < 2.5 mg/dL Critical value < 1 mg/dL	• Shift of phosphorus into cells • IV glucose administration • Total parenteral nutrition without phosphorus • Aluminium- or magnesium-based antacids • Diuretic therapy • Alcoholism	• Paraesthesias • Muscle weakness • Muscle pain and tenderness • Confusion, decreasing level of consciousness • Seizures • Bone pain, osteomalacia • Anorexia, dysphagia • Decreased bowel sounds • Possible acute respiratory failure
Hyperphosphataemia Serum phosphate > 4.5 mg/dL Critical value > 90 mg/dL	• Renal failure • Chemotherapy • Muscle tissue trauma • Sepsis • Severe hypothermia • Heat stroke	• Circumoral and peripheral paraesthesias • Muscle spasms • Tetany • Soft tissue calcification

- Hyperventilation and respiratory alkalosis cause phosphate to shift out of extracellular fluids into the intracellular space.
- Other causes include diabetic ketoacidosis with excess phosphate loss in the urine, stress responses and extensive burns.

Pathophysiology and manifestations

Most effects of hypophosphataemia result from depletion of ATP and impaired oxygen delivery to the cells due to a deficiency of the red blood cell enzyme 2,3-DPG. Severe hypophosphataemia affects virtually every major organ system:

- *Central nervous system*: reduced oxygen and ATP synthesis in the brain causes neurological manifestations such as irritability, apprehension, weakness, paraesthesias, lack of coordination, confusion, seizures and coma.
- *Haematological*: oxygen delivery to the cells is reduced. Haemolytic anaemia (excessive red blood cell destruction) may develop due to lack of ATP in red blood cells.
- *Musculoskeletal*: decreased ATP causes muscle weakness and release of creatinine phosphokinase (CPK, a muscle enzyme); acute rhabdomyolysis (muscle cell breakdown) can develop. Muscle cell destruction, in turn, can lead to acute renal failure as myoglobin, a muscle cell protein, exerts a toxic effect on the kidney tubule.
- *Respiratory*: chest muscle weakness can interfere with effective ventilation, leading to respiratory failure.
- *Cardiovascular*: hypophosphataemia decreases myocardial contractility; decreased oxygenation of the heart muscle can cause chest pain and arrhythmias.
- *Gastrointestinal*: anorexia can occur, as well as dysphagia (difficulty swallowing), nausea and vomiting, decreased bowel sounds and possible ileus due to reduced gastrointestinal motility.

INTERPROFESSIONAL CARE

Treatment for hypophosphataemia is directed at prevention, treating the underlying cause of the disorder and replacing phosphate. An improved diet and oral phosphate supplement may restore normal phosphate levels in the person with a mild to moderate deficiency. Intravenous phosphate (sodium phosphate or potassium phosphate) is given when serum phosphate levels are less than 1 mg/dL. Oral phosphate supplements are then continued for up to 1 week to restore intracellular phosphate levels (Metheny, 2012).

Nursing care

Nurses can be instrumental in identifying the person at risk of phosphate deficiency and preventing it from developing. Nurses should closely monitor serum electrolyte values in the person at risk, including those who are malnourished, receiving intravenous glucose solutions or total parenteral nutrition, or being treated with diuretic therapy or antacids that bind with phosphate. Nursing diagnoses that may be appropriate for the person with hypophosphataemia include:

- *impaired physical mobility* related to muscle weakness and poor coordination
- *ineffective breathing pattern* related to weakened muscles of respiration
- *decreased cardiac output* related to reduced myocardial contractility
- *risk of injury* related to muscle weakness and altered mental status.

Community-based care

In preparing for discharge, educate the person and family about the causes and manifestations of hypophosphataemia. Discuss the importance of avoiding phosphorus-binding antacids, unless prescribed. Stress the need for a well-balanced diet to maintain an adequate intake of phosphate.

The person with hyperphosphataemia

Hyperphosphataemia is a serum phosphate level greater than 4.5 mg/dL. As with other electrolyte imbalances, it may be the result of impaired phosphate excretion, excess intake or a shift of phosphate from the intracellular space into extracellular fluids.

- Acute or chronic renal failure is the primary cause of impaired phosphate excretion.
- Rapid administration of phosphate-containing solutions can increase serum phosphate levels. This can include phosphate enemas. In addition, excess vitamin D increases phosphate absorption and can lead to hyperphosphataemia in the person with impaired renal function.
- A shift of phosphate from the intracellular to the extracellular space can occur during chemotherapy, due to sepsis or hypothermia, or because of extensive trauma or heat stroke.
- Because phosphate levels are affected by serum calcium concentrations, disruption of the mechanisms that regulate calcium levels (e.g. hypoparathyroidism, hyperthyroidism or vitamin D intoxication) can lead to hyperphosphataemia.

Pathophysiology and manifestations

Excessive serum phosphate levels cause few specific symptoms. The effects of high serum phosphate levels on nerves and muscles (muscle cramps and pain, paraesthesias, tingling around the mouth, muscle spasms, tetany) are more the result of hypocalcaemia that develops secondary to an elevated serum phosphorus level. The phosphate in the serum combines with ionised calcium and the ionised serum calcium level falls.

Calcification of soft tissues can occur with high phosphate levels. Phosphates bind with calcium to precipitate in soft tissues such as the kidneys and other organs. Soft tissue calcification can impair the function of affected organs.

INTERPROFESSIONAL CARE

Treatment of the underlying disorder often corrects hyperphosphataemia. When this is not feasible, phosphate-containing drugs are eliminated and intake of phosphate-rich foods such as organ meats and milk and milk products is restricted. Agents that bind with phosphate in the GI tract (such as calcium-containing antacids) may be prescribed. If renal function is adequate, intravenous normal saline may be given to promote renal excretion of phosphate. Dialysis may be necessary to reduce phosphate levels in the person with renal failure.

Nursing care

When providing nursing care for the person with hyperphosphataemia, monitor the person for laboratory data revealing an excess of phosphorus and a deficit of calcium, as well as the signs of hypocalcaemia.

Community-based care

Discuss the risk of hyperphosphataemia related to using phosphate preparations as laxatives or enemas, particularly with the person who has other risk factors for the disorder. When preparing the person for discharge, teach about the use of phosphate-binding preparations as ordered and dietary phosphate restrictions.

ACID–BASE DISORDERS

Homeostasis and optimal cellular function require maintenance of the hydrogen ion (H^+) concentration of body fluids within a relatively narrow range. Hydrogen ions determine the relative acidity of body fluids. **Acids** release hydrogen ions in solution; **bases** (or **alkalis**) accept hydrogen ions in solution. The hydrogen ion concentration of a solution is measured as its pH. The relationship between hydrogen ion concentration and pH is inverse; that is, as hydrogen ion concentration increases, the pH falls and the solution becomes more acidic. As hydrogen ion concentration falls, the pH rises and the solution becomes more alkaline or basic. The pH of body fluids is slightly basic, with the normal pH ranging from 7.35 to 7.45. (A pH of 7 is neutral.)

REGULATION OF ACID–BASE BALANCE

A number of mechanisms work together to maintain the pH of the body within this normal range. Metabolic processes in the body continuously produce acids, which fall into two categories: volatile acids and non-volatile acids. **Volatile acids** can be eliminated from the body as a gas. Carbonic acid (H_2CO_3) is the only volatile acid produced in the body. It dissociates (separates) into carbon dioxide (CO_2) and water (H_2O); the carbon dioxide is then eliminated from the body through the lungs. All other acids produced in the body are *non-volatile acids* that must be metabolised or excreted from the body in fluid. Lactic acid, hydrochloric acid, phosphoric acid and sulfuric acid are examples of non-volatile acids. Most acids and bases in the body are weak; that is, they neither release nor accept a significant amount of hydrogen ions.

Three systems work together in the body to maintain the pH despite continuous acid production: buffers, the respiratory system and the renal system.

Buffer systems

Buffers are substances that prevent major changes in pH by removing or releasing hydrogen ions. When excess acid is present in body fluid, buffers bind with hydrogen ions to minimise

the change in pH. If body fluids become too basic or alkaline, buffers release hydrogen ions, restoring the pH. Although buffers act within a fraction of a second, their capacity to maintain pH is limited. The major buffer systems of the body are the bicarbonate–carbonic acid buffer system, the phosphate buffer system and protein buffers.

The bicarbonate–carbonic acid buffer system can be illustrated by the following equation:

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$$

Bicarbonate (HCO_3^-) is a weak base; when an acid is added to the system, the hydrogen ion in the acid combines with bicarbonate and the pH changes only slightly. Carbonic acid (H_2CO_3) is a weak acid produced when carbon dioxide dissolves in water. If a base is added to the system, it combines with carbonic acid and the pH remains within the normal range. Although the amounts of bicarbonate and carbonic acid in the body vary to a certain extent, as long as a ratio of 20 parts bicarbonate (HCO_3^-) to 1 part carbonic acid (H_2CO_3) is maintained, the pH remains within the 7.35 to 7.45 range (see Figure 9.14).

The normal serum bicarbonate level is 24 mEq/L and that of carbonic acid is 1.2 mEq/L. Thus, the ratio of bicarbonate to carbonic acid is 20:1. It is this ratio that maintains the pH within the normal range. Adding a strong acid to extracellular fluid depletes bicarbonate, changing the 20:1 ratio and causing the pH to drop below 7.35. This is known as **acidosis**. Addition of a strong base depletes carbonic acid as it combines with the base. The 20:1 ratio again is disrupted and the pH rises above 7.45, a condition known as **alkalosis**.

Intracellular and plasma proteins also serve as buffers. Plasma proteins contribute to buffering of extracellular fluids. Proteins in intracellular fluid provide extensive buffering for organic acids produced by cellular metabolism. In red blood cells, haemoglobin acts as a buffer for hydrogen ions when carbonic acid dissociates. Inorganic phosphates also serve as extracellular buffers, although their roles are not as important as the bicarbonate–carbonic acid buffer system. Phosphates are, however, important intracellular buffers, helping to maintain a stable pH within cells.

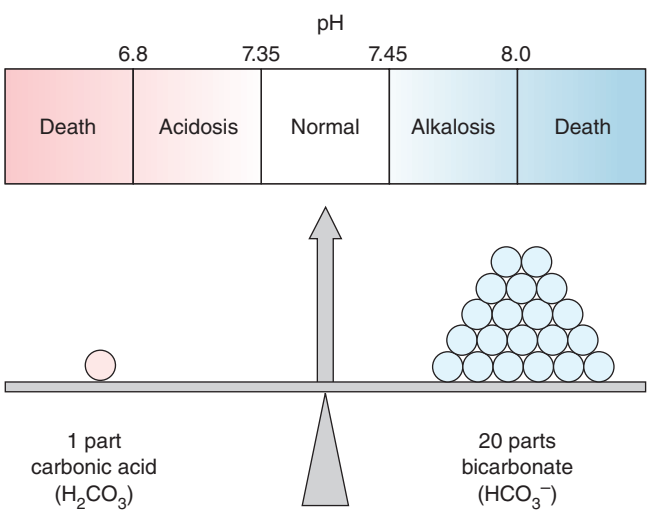


FIGURE 9.14 ■ The normal ratio of bicarbonate to carbonic acid is 20:1. As long as this ratio is maintained, the pH remains within the normal range of 7.35 to 7.45

Respiratory system

The respiratory system (and the respiratory centre of the brain) regulates carbonic acid in the body by eliminating or retaining carbon dioxide. Carbon dioxide is a potential acid; when combined with water, it forms carbonic acid (see previous equation), a volatile acid. Acute increases in either carbon dioxide or hydrogen ions in the blood stimulate the respiratory centre in the brain. As a result, both the rate and depth of respiration increase. The increased rate and depth of lung ventilation eliminate carbon dioxide from the body and carbonic acid levels fall, bringing the pH to a more normal range. Although this compensation for increased hydrogen ion concentration occurs within minutes, it becomes less effective over time. The person with chronic lung disease may have consistently high carbon dioxide levels in their blood.

Alkalosis, by contrast, depresses the respiratory centre. Both the rate and depth of respiration decrease and carbon dioxide is retained. The retained carbon dioxide then combines with water to restore carbonic acid levels and bring the pH back within the normal range.

Renal system

The renal system is responsible for the long-term regulation of acid–base balance in the body. Excess non-volatile acids produced during metabolism normally are eliminated by the kidneys. The kidneys also regulate bicarbonate levels in extracellular fluid by regenerating bicarbonate ions as well as reabsorbing them in the renal tubules. Although the kidneys respond more slowly to changes in pH (over hours to days), they can generate bicarbonate and selectively excrete or retain hydrogen ions as needed. In acidosis, when excess hydrogen ion is present and the pH falls, the kidneys excrete hydrogen ions and retain bicarbonate. In alkalosis, the kidneys retain hydrogen ions and excrete bicarbonate to restore acid–base balance.

Assessment of acid–base balance

Acid–base balance is evaluated primarily by measuring arterial blood gases (ABGs).

CONSIDERATION FOR PRACTICE

Arteries are high-pressure vessels, in contrast to veins. Obtaining an arterial blood sample requires specialised training. It may be done by a doctor, a Registered Nurse, a respiratory therapist or a laboratory technician who has been trained in drawing ABGs. Apply firm pressure to the puncture site for 2 to 5 minutes after the needle is withdrawn to prevent bleeding into the surrounding tissues.

Arterial blood is used because it reflects acid–base balance throughout the entire body better than venous blood. Arterial blood also provides information about the effectiveness of the lungs in oxygenating blood. The elements measured are pH, $PaCO_2$, PaO_2 and bicarbonate level.

The **$PaCO_2$** measures the pressure exerted by dissolved carbon dioxide in the blood, and reflects the respiratory component of acid–base regulation and balance. The $PaCO_2$ is regulated by the lungs. The normal value is 35 to 45 mmHg. A $PaCO_2$ of less than 35 mmHg is known as *hypocapnia*; a $PaCO_2$ greater than 45 mmHg is *hypercapnia*.

The **PaO_2** is a measure of the pressure exerted by oxygen that is dissolved in the plasma. Only about 3% of oxygen in the blood is transported in solution; most is combined with haemoglobin. However, it is the dissolved oxygen that is available to the cells for metabolism. As dissolved oxygen diffuses out of plasma into the tissues, more is released from haemoglobin. The normal value for PaO_2 is 80 to 100 mmHg. A PaO_2 of less than 80 mmHg is indicative of *hypoxaemia*. The PaO_2 is valuable for evaluating respiratory function, but is not used as a primary measurement in determining acid–base status.

CONSIDERATION FOR PRACTICE

You will see the abbreviations $PaCO_2$ and PaO_2 used interchangeably with PCO_2 and PO_2. The 'P' stands for partial pressure: the pressure exerted by the gas dissolved in the blood. The 'a' indicates that the sample is arterial blood. Because these measurements rarely are done on venous blood, the 'a' is often omitted from the abbreviation.

The **serum bicarbonate** (HCO_3^-) reflects the renal regulation of acid–base balance. It is often called the metabolic component of arterial blood gases. The normal HCO_3^- value is 22 to 26 mEq/L.

The **base excess (BE)** is a calculated value also known as *buffer base capacity*. The base excess measures substances that can accept or combine with hydrogen ions. It reflects the degree of acid–base imbalance by indicating the status of the body's total buffering capacity. It represents the amount of acid or base that must be added to a blood sample to achieve a pH of 7.4. This is essentially a measure of increased or decreased bicarbonate. The normal value for base excess for arterial blood is –3.0 to +3.0. Normal ABG values are summarised in Table 9.11.

TABLE 9.11 Normal arterial blood gas values

VALUE	NORMAL RANGE	SIGNIFICANCE
pH	7.35 to 7.45	Reflects hydrogen ion (H^+) concentration • < 7.35 = acidosis • > 7.45 = alkalosis
$PaCO_2$	35 to 45 mmHg	Partial pressure of carbon dioxide (CO_2) in arterial blood • < 35 mmHg = hypocapnia • > 45 mmHg = hypercapnia
PaO_2	80 to 100 mmHg	Partial pressure of oxygen (O_2) in arterial blood • < 80 mmHg = hypoxaemia
HCO_3^-	22 to 26 mEq/L	Bicarbonate concentration in plasma
BE	–3 to +3	Base excess; a measure of buffering capacity

ABGs are analysed to identify acid–base disorders and their probable cause, to determine the extent of the imbalance and to monitor treatment. When analysing ABG results, it is important to use a systematic approach. First evaluate each individual measurement, then look at the interrelationships to determine the person's acid–base status (see Box 9.10).

BOX 9.10 Interpreting arterial blood gases

1. Look at the pH.
   - pH < 7.35 = acidosis
   - pH > 7.45 = alkalosis
2. Look at the $PaCO_2$.
   - $PaCO_2$ < 35 mmHg = hypocapnia; more carbon dioxide is being exhaled than normal
   - $PaCO_2$ > 45 mmHg = hypercapnia; carbon dioxide is being retained
3. Evaluate the pH–$PaCO_2$ relationship for a possible respiratory problem.
   - If the pH is < 7.35 (acidosis) and the $PaCO_2$ is > 45 mmHg (hypercapnia), retained carbon dioxide is causing increased H^+ concentration and respiratory acidosis.
   - If the pH is > 7.45 (alkalosis) and the $PaCO_2$ is < 35 mmHg (hypocapnia), low carbon dioxide levels and decreased H^+ concentration are causing respiratory alkalosis.
4. Look at the bicarbonate.
   - If the HCO_3^- is < 22 mEq/L, bicarbonate levels are lower than normal.
   - If the HCO_3^- is > 26 mEq/L, bicarbonate levels are higher than normal.
5. Evaluate the pH, HCO_3^- and BE for a possible metabolic problem.
   - If the pH is < 7.35 (acidosis), the HCO_3^- is < 22 mEq/L and the BE is < –3 mEq/L, then low bicarbonate levels and high H^+ concentrations are causing metabolic acidosis.
   - If the pH is > 7.45 (alkalosis), the HCO_3^- is > 26 mEq/L and the BE is > +3 mEq/L, then high bicarbonate levels are causing metabolic alkalosis.
6. Look for compensation.
   Renal compensation:
   - In respiratory acidosis (pH < 7.35, $PaCO_2$ > 45 mmHg), the kidneys retain HCO_3^- to buffer the excess acid, so the HCO_3^- is > 26 mEq/L.
   - In respiratory alkalosis (pH > 7.45, $PaCO_2$ < 35 mmHg), the kidneys excrete HO_3^- to minimise the alkalosis, so the HCO_3^- is < 22 mEq/L.

   Respiratory compensation:
   - In metabolic acidosis (pH < 7.35, HCO_3^- < 22 mEq/L), the rate and depth of respirations increase, increasing carbon dioxide elimination, so the $PaCO_2$ is < 35 mmHg.
   - In metabolic alkalosis (pH > 7.45, HCO_3^- > 26 mEq/L), respirations slow, carbon dioxide is retained, so the $PaCO_2$ is > 45 mmHg.
7. Evaluate oxygenation.
   - PaO_2 < 80 mmHg = hypoxaemia; possible hypoventilation
   - PaO_2 > 100 mmHg = hyperventilation

ACID–BASE IMBALANCE

Acid–base imbalances fall into two major categories: acidosis and alkalosis. Acidosis occurs when the hydrogen ion concentration increases above normal (pH below 7.35). Alkalosis occurs when the hydrogen ion concentration falls below normal (pH above 7.45).

Acid–base imbalances are further classified as *metabolic* or *respiratory* disorders. In metabolic disorders, the primary change is in the concentration of bicarbonate. In metabolic acidosis, the amount of bicarbonate is decreased in relation to the amount of acid in the body (see Figure 9.15A). It can develop as a result of abnormal bicarbonate losses or because of excess non-volatile acids in the body. The pH falls below 7.35 and the bicarbonate concentration is less than 22 mEq/L. Metabolic alkalosis, by contrast, occurs when there is an excess of bicarbonate in relation to the amount of hydrogen ion (see Figure 9.15B). The pH is above 7.45 and the bicarbonate concentration is greater than 26 mEq/L.

In respiratory disorders, the primary change is in the concentration of carbonic acid. Respiratory acidosis occurs when carbon dioxide is retained, increasing the amount of carbonic acid in the body (see Figure 9.16A). As a result, the pH falls to less than 7.35 and the $PaCO_2$ is greater than 45 mmHg. When too much carbon dioxide is 'blown off', carbonic acid levels fall and respiratory alkalosis develops (see Figure 9.16B). The pH rises to above 7.45 and the $PaCO_2$ is less than 35 mmHg.

Acid–base disorders are further defined as primary (simple) and mixed. Primary disorders usually are due to one cause. For example, respiratory failure often causes respiratory acidosis due

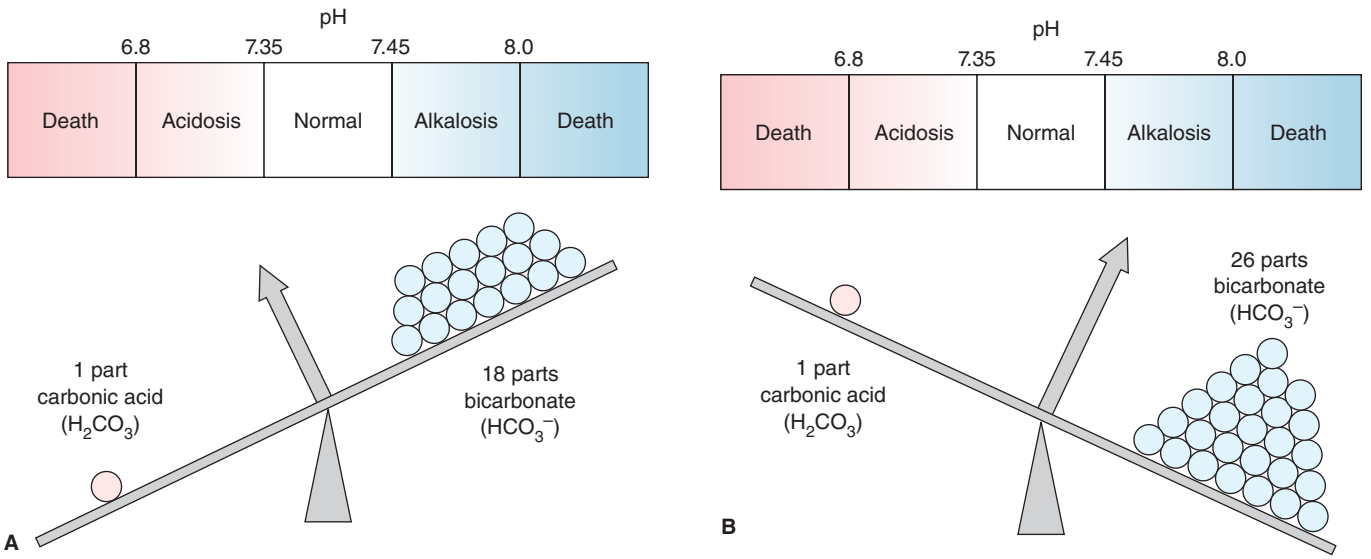


FIGURE 9.15 ■ Metabolic acid–base imbalances. *A*, Metabolic acidosis. *B*, Metabolic alkalosis

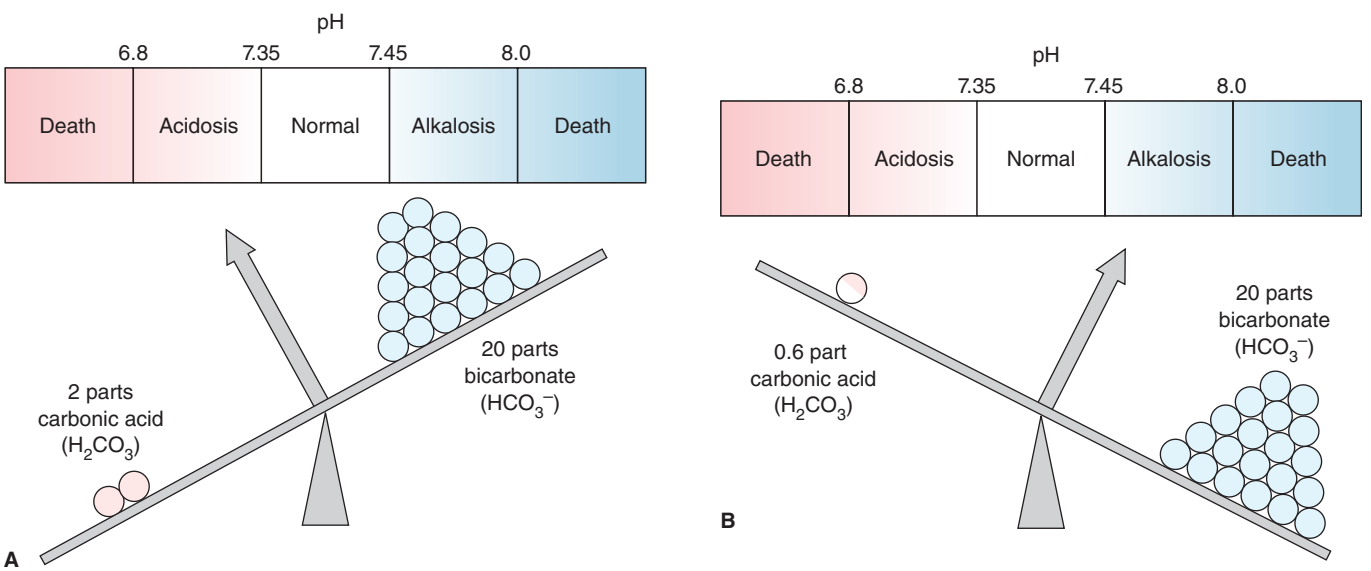


FIGURE 9.16 ■ Respiratory acid–base imbalances. *A*, Respiratory acidosis. *B*, Respiratory alkalosis

to retained carbon dioxide; renal failure usually causes metabolic acidosis due to retained hydrogen ion and impaired bicarbonate production. Table 9.12 summarises primary acid–base imbalances with common causes of each. Mixed disorders occur from combinations of respiratory and metabolic disturbances. For example, a person in cardiac arrest develops a mixed respiratory and metabolic acidosis due to lack of ventilation (and retained CO_2) and hypoxia of body tissues that leads to anaerobic metabolism and acid by-products (excess non-volatile acids).

FAST FACTS

- Simple acid–base imbalances are more commonly seen than mixed imbalances. Common causes of simple acid–base imbalances include:
  - diabetic ketoacidosis (metabolic acidosis)
  - chronic obstructive lung disease (respiratory acidosis)
  - anxiety-related (psychogenic) hyperventilation (respiratory alkalosis).
- The critically ill person is at higher risk of mixed acid–base imbalances.

Compensation

With primary acid–base disorders, compensatory changes in the other part of the regulatory system occur to restore a normal pH and homeostasis. In metabolic acid–base disorders, the change in pH affects the rate and depth of respirations. Therefore, this affects carbon dioxide elimination and the $PaCO_2$, helping restore the carbonic acid to bicarbonate ratio. The kidneys compensate for simple respiratory imbalances. The change in pH affects both bicarbonate conservation and hydrogen ion elimination (see Table 9.12).

Compensatory changes in respirations occur within minutes of a change in pH. These changes, however, become less effective over time. The renal response takes longer to restore the pH, but is a more effective long-term mechanism. If the pH is restored to within normal limits, the disorder is said to be *fully compensated*. When these changes are reflected in ABG values but the pH remains outside normal limits, the disorder is said to be *partially compensated*.

The person with metabolic acidosis

Metabolic acidosis (bicarbonate deficit) is characterised by a low pH (< 7.35) and a low bicarbonate (< 22 mEq/L). It may be

TABLE 9.12 Common causes of primary acid–base imbalances

IMBALANCE	COMMON CAUSES
Metabolic acidosis pH < 7.35 HCO_3^- < 22 mEq/L *Critical values* pH < 7.20 HCO_3^- < 10 mEq/L	↑ Acid production • Lactic acidosis • Ketoacidosis related to diabetes, starvation or alcoholism • Salicylate toxicity ↓ Acid excretion • Renal failure ↑ Bicarbonate loss • Diarrhoea, ileostomy drainage, intestinal fistula • Biliary or pancreatic fistulas ↑ Chloride • Sodium chloride IV solutions • Renal tubular acidosis • Carbonic anhydrase inhibitors
Metabolic alkalosis pH > 7.45 HCO_3^- > 26 mEq/L *Critical values* pH > 7.60 HCO_3^- > 40 mEq/L	↑ Acid loss or excretion • Vomiting, gastric suction • Hypokalaemia ↑ Bicarbonate • Alkali ingestion (bicarbonate of soda) • Excess bicarbonate administration
Respiratory acidosis pH < 7.35 $PaCO_2$ > 45 mmHg *Critical values* pH < 7.2 $PaCO_2$ > 77 mmHg	Acute respiratory acidosis • Acute respiratory conditions (pulmonary oedema, pneumonia, acute asthma) • Opiate overdose • Foreign body aspiration • Chest trauma Chronic respiratory acidosis • Chronic respiratory conditions (COPD, cystic fibrosis) • Multiple sclerosis, other neuromuscular diseases • Stroke
Respiratory alkalosis pH > 7.45 $PaCO_2$ > 35 mmHg *Critical values* pH > 7.60 $PaCO_2$ < 20 mmHg	• Anxiety-induced hyperventilation (e.g. anxiety) • Fever • Early salicylate intoxication • Hyperventilation with mechanical ventilator

caused by excess acid in the body or loss of bicarbonate from the body. When metabolic acidosis develops, the respiratory system attempts to return the pH to normal by increasing the rate and depth of respirations. Carbon dioxide elimination increases and the $PaCO_2$ falls (< 35 mmHg).

Risk factors

Metabolic acidosis rarely is a primary disorder; it usually develops during the course of another disease:

- *Acute lactic acidosis* usually results from tissue hypoxia due to shock or cardiac arrest.
- A person with type 1 diabetes mellitus is at risk of developing *diabetic ketoacidosis*. (See Chapter 19 for more information about diabetes and its complications.)
- *Acute* or *chronic renal failure* impairs the excretion of metabolic acids.
- Diarrhoea, intestinal suction or abdominal fistulas increase the *risk of excess bicarbonate loss*.

Other common causes of metabolic acidosis are listed in Table 9.12.

Pathophysiology

Three basic mechanisms that can cause metabolic acidosis are:

- accumulation of metabolic acids
- excess loss of bicarbonate
- an increase in chloride levels.

An accumulation of metabolic acids can result from excess acid production or impaired elimination of metabolic acids by the kidney. Lactic acidosis develops due to tissue hypoxia and a shift to anaerobic metabolism by the cells. Lactate and hydrogen ions are produced, forming lactic acid. Both oxygen and glucose are necessary for normal cell metabolism. When intracellular glucose is inadequate due to starvation or a lack of insulin to move it into cells, the body breaks down fatty tissue to meet its metabolic needs. In this process, fatty acids are released, which are converted to ketones; ketoacidosis develops. Aspirin (acetylsalicylic acid) breaks down into salicylic acid in the body. Substances such as aspirin, methanol (wood alcohol) and ethylene (contained in antifreeze and solvents) cause a toxic increase in body acids by either breaking down into acid products (salicylic acid) or stimulating metabolic acid production (Grossman & Porth, 2014). Renal failure impairs the body's ability to excrete excess hydrogen ions and form bicarbonate.

Excess metabolic acids increase the hydrogen ion concentration of body fluids. The excess acid is buffered by bicarbonate, leading to what is known as a high **anion gap** acidosis (see Box 9.11).

The pancreas secretes bicarbonate-rich fluid into the small intestine. Intestinal suction, severe diarrhoea, ileostomy drainage or fistulas can lead to excess loss of bicarbonate. Hyperchloraemic acidosis can develop when excess chloride solutions (such as NaCl or ammonium chloride) are infused, causing a rise in chloride concentrations. It may be related to renal disease or administration of carbonic anhydrate inhibitor diuretics. The anion gap remains normal in metabolic acidosis due to bicarbonate loss or excess chloride.

Acidosis depresses cell membrane excitability, affecting neuromuscular function. It also increases the amount of free calcium in ECF by interfering with protein binding. Severe acidosis (pH of 7.0 or less) depresses myocardial contractility, leading to a fall in cardiac output. If kidney function is normal, acid excretion and ammonia production increase to eliminate excess hydrogen ions.

Acid–base imbalances also affect electrolyte balance (see Table 9.13). In acidosis, potassium is retained as the kidney excretes excess hydrogen ions. Excess hydrogen ions also enter the cells, displacing potassium from the intracellular space to maintain the balance of cations and anions within the cells. The effect of both processes is to increase serum potassium levels. Also in acidosis, calcium is released from its bonds with plasma proteins, increasing the amount of ionised (free) calcium in the blood. Magnesium levels may fall in acidosis. See Box 9.11 for information on anion gap acidosis.

Manifestations

Metabolic acidosis affects the function of many body systems. Its general manifestations include weakness and fatigue, headache and general malaise. Gastrointestinal function is affected, causing anorexia, nausea, vomiting and abdominal pain. The level of consciousness declines, leading to stupor and coma. Cardiac arrhythmias develop and cardiac arrest may occur. The skin is often warm and flushed. Skeletal problems may develop in chronic acidosis, as calcium and phosphate are released

TABLE 9.13 Compensation for simple acid–base imbalances

PRIMARY DISORDER	CAUSE	COMPENSATION	EFFECT ON ABGs
Metabolic acidosis	Excess non-volatile acids; bicarbonate deficiency	Rate and depth of respirations increase, eliminating additional CO_2	↓ pH ↓ HCO_3^- ↓ $PaCO_2$
Metabolic alkalosis	Bicarbonate excess	Rate and depth of respirations decrease, retaining CO_2	↑ pH ↑ HCO_3^- ↑ $PaCO_2$
Respiratory acidosis	Retained CO_2 and excess carbonic acid	Kidneys conserve bicarbonate to restore carbonic acid : bicarbonate ratio of 1:20	↓ pH ↑ $PaCO_2$ ↑ HCO_3^-
Respiratory alkalosis	Loss of CO_2 and deficient carbonic acid	Kidneys excrete bicarbonate and conserve H^+ to restore carbonic acid : bicarbonate ratio	↑ pH ↓ $PaCO_2$ ↓ HCO_3^-

BOX 9.11 Unravelling the anion gap

Calculation of the anion gap can help identify the underlying mechanism in metabolic acidosis if it is unclear.

The number of cations (positively charged ions) and anions (negatively charged ions) in ECF normally is equal (see Figure 9.2). Not all of these ions, however, are measured in laboratory testing (e.g. organic acids and proteins). The anion gap is calculated by subtracting the sum of two measured anions, chloride and bicarbonate, from the concentration of the major cation, sodium (see figure). The normal anion gap is 8 to 12 mEq/L.

Excess acids in ECF are buffered by bicarbonate, reducing serum bicarbonate levels and the total measured concentration of anions. This increases the anion gap (B in figure). When bicarbonate is lost from the body or chloride levels increase, however, the anion gap remains within normal limits (C in figure). This occurs because an increase or decrease in one of these negatively charged ions causes a corresponding change in the other to maintain balance (e.g. $\downarrow HCO_3^- \leftrightarrow \uparrow Cl^-$) and there is no change in the amount of unmeasured anions.

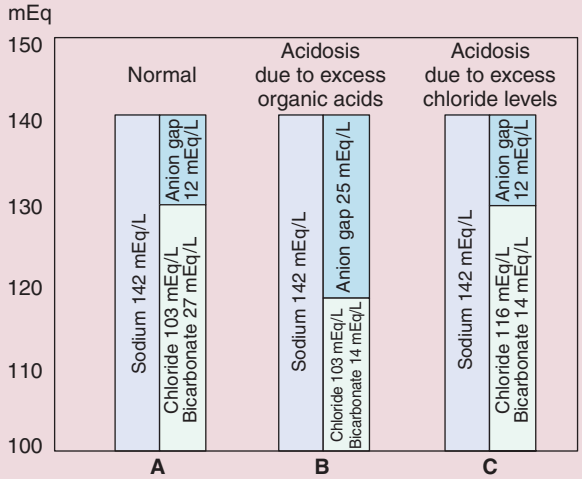


Illustration of the anion gap in metabolic acidosis. *A*, Normal anion gap. *B*, High anion gap caused by excess acids. *C*, Normal anion gap with hyperchloremia

from the bones. Manifestations of compensatory mechanisms are seen. The respirations are deep and rapid, known as Kussmaul's respirations. The person may complain of shortness of breath or dyspnoea. (See 'Manifestations' box below.)

MANIFESTATIONS Metabolic acidosis

- Anorexia
- Nausea and vomiting
- Abdominal pain
- Weakness
- Fatigue
- General malaise
- Decreasing levels of consciousness
- Arrhythmias
- Bradycardia
- Warm, flushed skin
- Hyperventilation (Kussmaul's respirations)

INTERPROFESSIONAL CARE

Management of metabolic acidosis focuses on treating the underlying cause of the disorder and correcting the acid–base imbalance.

Diagnosis

The following laboratory and diagnostic tests may be ordered:

- *ABGs* generally show a pH of less than 7.35 and a bicarbonate level of less than 22 mEq/L. A compensatory decrease in $PaCO_2$ to less than 35 mmHg is usually present.
- *Serum electrolytes* demonstrate elevated serum potassium levels and possible low magnesium levels. The total calcium may remain unchanged, although more physiologically active ionised calcium is available. Sodium, chloride and bicarbonate levels are used to calculate the anion gap.
- The *ECG* may show changes that reflect both the acidosis (particularly when severe) and the accompanying hyperkalaemia.
- Other diagnostic studies such as the blood glucose and renal function studies may be ordered to identify the underlying cause of metabolic acidosis.

Medications

An alkalinising solution such as bicarbonate may be given if the pH is less than 7.1 to reduce the effects of the acidosis on cardiac function. Sodium bicarbonate is the most commonly used alkalinising solution; others include lactate, citrate and acetate solutions (which are metabolised to bicarbonate). Alkalinising solutions are given intravenously for severe acute metabolic acidosis. In chronic metabolic acidosis, the oral route is used.

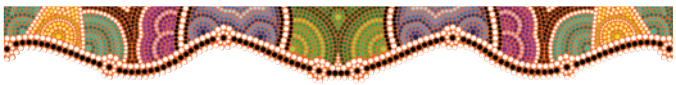
The person treated with bicarbonate must be carefully monitored. Rapid correction of the acidosis may lead to metabolic alkalosis and hypokalaemia. Hypernatraemia and hyperosmolality may develop as well, leading to water retention and fluid overload.

CONSIDERATION FOR PRACTICE

As metabolic acidosis is corrected, potassium shifts back into the intracellular space. This can lead to hypokalaemia and cardiac arrhythmias. Carefully monitor serum potassium levels during treatment.

Treatment for diabetic ketoacidosis includes intravenous insulin and fluid replacement. (See Chapter 19 for the treatment of diabetic ketoacidosis.) Alcoholic ketoacidosis is treated with saline solutions and glucose. Treatment for lactic acidosis

from decreased tissue perfusion (e.g. shock or cardiac arrest) focuses on correcting the underlying problem and improving tissue perfusion. The person with chronic renal failure and mild or moderate metabolic acidosis may or may not require treatment, depending on the pH and bicarbonate levels. When metabolic acidosis is due to diarrhoea, treatment includes correcting the underlying cause and providing fluid and electrolyte replacement.



Nursing care

Nurses frequently provide care for people diagnosed with metabolic acidosis, although the focus of care often is the disorder underlying the acidosis (e.g. diabetes mellitus, renal failure) rather than the acidosis itself. For this reason, it is vital for the nurse to be aware of the effects of the acidosis and its implications for nursing care.

Health promotion

To promote health in the person at risk of metabolic acidosis, it is important for the nurse to discuss the management of the underlying disease process (e.g. type 1 diabetes or renal failure) preventing complications such as diabetic ketoacidosis and metabolic acidosis. Because early manifestations of metabolic acidosis (e.g. fatigue, general malaise, anorexia, nausea, abdominal pain) resemble those of common viral disorders such as 'the flu', stress the importance of promptly seeking treatment if these symptoms develop.

Assessment

Assessment data related to metabolic acidosis include the following:

- *Health history*: current manifestations, including anorexia, nausea, vomiting, abdominal discomfort, fatigue, lethargy, other symptoms; duration of symptoms and any precipitating factors such as diarrhoea, ingestion of a toxin such as aspirin, methanol or ethylene; chronic diseases such as diabetes or renal failure, cirrhosis of the liver or endocrine disorders; current medications.
- *Physical assessment*: mental status and level of consciousness; vital signs including respiratory rate and depth; apical and peripheral pulses; skin colour and temperature; abdominal contour and distension; bowel sounds; urine output.
- *Diagnostic tests*: ABGs, serum electrolytes, tests for underlying disorders.

Nursing diagnoses and interventions

Nursing management of a person with metabolic acidosis often focuses on the primary disorder (e.g. diabetic ketoacidosis or renal failure); however, the acidosis itself has effects that must be attended to when providing care.

Decreased cardiac output

Metabolic acidosis affects cardiac output by decreasing myocardial contractility, slowing the heart rate and increasing the risk of arrhythmias. The accompanying hyperkalaemia increases the risk of decreased cardiac output as well (see earlier discussion about hyperkalaemia).

- Monitor vital signs, including peripheral pulses and capillary refill. *Hypotension, diminished pulse strength and slowed capillary refill may indicate decreased cardiac output and impaired tissue perfusion. Poor tissue perfusion can increase the risk of lactic acidosis.*
- Monitor the ECG pattern for arrhythmias and changes characteristic of hyperkalaemia. Notify the physician of changes. *Progressive ECG changes such as widening of the QRS complex indicate an increasing risk of arrhythmias and cardiac arrest. Arrhythmias further decrease cardiac output, possibly intensifying the degree of acidosis.*
- Monitor laboratory values, including ABGs, serum electrolytes and renal function studies (serum urea and creatinine). *Frequent monitoring of laboratory values allows evaluation of the effectiveness of treatment as well as early identification of potential problems.*

Risk of excess fluid volume

Administering bicarbonate to correct acidosis increases the risk of hypernatraemia, hyperosmolality and fluid volume excess.

- Monitor and maintain fluid replacement as ordered. Monitor serum sodium levels and osmolality. *Bicarbonate administration can cause hypernatraemia and hyperosmolality, leading to water retention.*
- Monitor heart and lung sounds, CVP and respiratory status. *Increasing dyspnoea, adventitious lung sounds, a third heart sound (S3) due to the volume of blood flow through the heart, and high CVP readings are indicative of hypervolaemia and should be reported to the attending doctor.*
- Assess for oedema, particularly in the back, sacral and periorbital areas. *Initially, oedema affects dependent tissues—the back and sacrum in the person who is bedridden. Periorbital oedema indicates more generalised oedema.*
- Assess urine output hourly. Maintain accurate intake and output records. Note urine output less than 30 mL/hour or a positive fluid balance on 24-hour total intake and output calculations. *Heart failure and inadequate renal perfusion may lead to decreased urine output.*
- Obtain daily weights using consistent conditions. *Daily weights are an accurate indicator of fluid balance.*
- Administer prescribed diuretics as ordered, monitoring the person's response to therapy. *Loop or high-ceiling diuretics such as frusemide can lead to further electrolyte imbalances, especially hypokalaemia. This is a significant risk like that seen during correction of metabolic acidosis.*

Risk of injury

Mental status and brain function are affected by acidosis, increasing the risk of injury.

- Monitor neurological function, including mental status, level of consciousness and muscle strength. *As the pH falls, mental functioning declines, leading to confusion, stupor and a decreasing level of consciousness.*

- Institute safety precautions as necessary: keep the bed in its lowest position, side rails raised. *These measures help protect the person from injury resulting from confusion or disorientation.*
- Keep clocks, calendars and familiar objects at the bedside. Orient to time, place and circumstances as needed. Allow significant others to remain with the person as much as possible. *An unfamiliar environment and altered thought processes can further increase the risk of injury. Significant others provide a sense of security and reduce anxiety.*

Nursing care also includes measures to treat the underlying disorder, such as diabetic ketoacidosis. Refer to the chapters on diabetes (Chapter 19) and renal failure (Chapter 27) for specific interventions.

Community-based care

Discharge planning and teaching focus on the underlying cause of the imbalance. The person who has developed ketoacidosis as a result of diabetes mellitus, starvation or alcoholism needs interventions and teaching to prevent future episodes of acidosis. Diet, medication management and alcohol dependency treatment are vital teaching areas. When metabolic acidosis is related to renal failure, the person should be referred for management of the renal failure itself. The person who has experienced diarrhoea or excess ileostomy drainage leading to bicarbonate loss requires information about appropriate diarrhoea treatment strategies and when to call their healthcare provider.

The person with metabolic alkalosis

Metabolic alkalosis (bicarbonate excess) is characterised by a high pH (> 7.45) and high bicarbonate (> 26 mEq/L). It may be caused by loss of acid or excess bicarbonate in the body. When metabolic alkalosis develops, the respiratory system attempts to return the pH to normal by slowing the respiratory rate. Carbon dioxide is retained and the $PaCO_2$ increases (> 45 mmHg).

Risk factors

As is the case with other acid–base imbalances, metabolic alkalosis rarely occurs as a primary disorder. Risk factors include hospitalisation, hypokalaemia and treatment with alkalinising solutions (e.g. bicarbonate).

Pathophysiology

Hydrogen ions may be lost via gastric secretions, through the kidneys or because of a shift of H^+ into the cells. Metabolic alkalosis due to loss of hydrogen ions usually occurs because of vomiting or gastric suction. Gastric secretions are highly acidic (pH 1 to 3). When these are lost through vomiting or gastric suction, the alkalinity of body fluids increases. This increased alkalinity results from both the loss of acid and selective retention of bicarbonate by the kidneys as chloride is depleted. (Chloride is the major anion in ECF; when it is lost, bicarbonate is retained as a replacement anion.)

Increased renal excretion of hydrogen ions can be prompted by hypokalaemia as the kidneys try to conserve potassium, excreting hydrogen ions instead. Hypokalaemia contributes to metabolic alkalosis in another way as well. When potassium shifts out of cells to maintain extracellular potassium levels, hydrogen ions shift into the cells to maintain the balance between cations and anions within the cell.

Excess bicarbonate usually occurs as a result of ingesting antacids that contain bicarbonate or overzealous administration of bicarbonate to treat metabolic acidosis. Common causes of metabolic alkalosis are summarised in Table 9.12.

In alkalosis, more calcium combines with serum proteins, reducing the amount of ionised (physiologically active) calcium in the blood. This accounts for many of the common manifestations of metabolic alkalosis. Alkalosis also affects potassium balance: hypokalaemia not only can cause metabolic alkalosis (see above), but it also can result from metabolic alkalosis. Hydrogen ions shift out of the intracellular space to help restore the pH, prompting more potassium to enter the cells and depleting ECF potassium. The high pH depresses the respiratory system as the body retains carbon dioxide to restore the carbonic acid to bicarbonate ratio.

Manifestations and complications

Manifestations of metabolic alkalosis (see the box below) occur as a result of decreased calcium ionisation and are similar to those of hypocalcaemia, including numbness and tingling around the mouth, fingers and toes; dizziness; Trousseau's sign; and muscle spasm. As the respiratory system compensates for metabolic alkalosis, respirations are depressed, and respiratory failure with hypoxaemia and respiratory acidosis may develop.

MANIFESTATIONS Metabolic alkalosis

- Confusion
- Decreasing level of consciousness
- Hyperreflexia
- Tetany
- Arrhythmias
- Hypotension
- Seizures
- Respiratory failure

INTERPROFESSIONAL CARE

Interprofessional management of metabolic alkalosis focuses on diagnosing and correcting the underlying cause.

Diagnosis

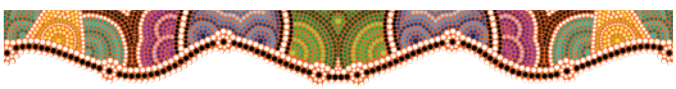
The following laboratory and diagnostic tests may be ordered:

- *ABGs* show a pH greater than 7.45 and bicarbonate level greater than 26 mEq/L. With compensatory hypoventilation, carbon dioxide is retained and the $PaCO_2$ is greater than 45 mmHg.
- *Serum electrolytes* often demonstrate decreased serum potassium (< 3.5 mEq/L) and decreased chloride (< 95 mEq/L)

levels. The serum bicarbonate level is high. Although the total serum calcium may be normal, the ionised fraction of calcium is low.
- *Urine pH* may be low (pH 1 to 3) if metabolic acidosis is caused by hypokalaemia. The kidneys selectively retain potassium and excrete hydrogen ions to restore ECF potassium levels. Urinary chloride levels may be normal or greater than 250 mEq/24 hours.
- The *ECG pattern* shows changes similar to those seen with hypokalaemia. These changes may be due to hypokalaemia or to the alkalosis.

Medications

Treatment of metabolic alkalosis includes restoring normal fluid volume and administering potassium chloride and sodium chloride solution. The potassium restores serum and intracellular potassium levels, allowing the kidneys to more effectively conserve hydrogen ions. Chloride promotes renal excretion of bicarbonate. Sodium chloride solutions restore fluid volume deficits that can contribute to metabolic alkalosis. In severe alkalosis, an acidifying solution such as dilute hydrochloric acid or ammonium chloride may be administered. In addition, drugs may be used to treat the underlying cause of the alkalosis.



Nursing care

Health promotion

Health promotion activities focus on teaching a person the risks of using sodium bicarbonate as an antacid to relieve heartburn or gastric distress. Stress the availability of other effective antacid preparations and the need to seek medical evaluation for persistent gastric symptoms. In the hospital setting, carefully monitor laboratory values for the person at risk of developing metabolic alkalosis, particularly people undergoing continuous gastric suction.

Assessment

Focused assessment data related to metabolic alkalosis include the following:
- *Health history*: current manifestations, such as numbness and tingling, muscle spasms, dizziness, other symptoms; duration of symptoms and any precipitating factors such as bicarbonate ingestion, vomiting, diuretic therapy or endocrine disorders; current medications.
- *Physical assessment*: vital signs, including apical pulse and rate and depth of respirations; muscle strength; deep tendon reflexes.
- *Diagnostic tests*: ABGs, serum electrolytes.

Nursing diagnoses and interventions

As with metabolic acidosis, nursing care of the person with metabolic alkalosis often focuses on intervening for responses to the primary problem, rather than the alkalosis itself. However, the risk of impaired gas exchange is a priority problem, especially with severe metabolic alkalosis.

Risk of impaired gas exchange

Respiratory compensation for metabolic alkalosis depresses the respiratory rate and reduces the depth of breathing to promote carbon dioxide retention. As a result, the person is at risk of impaired gas exchange, especially in the presence of underlying lung disease.
- Monitor respiratory rate, depth and effort. Monitor oxygen saturation continuously, reporting an oxygen saturation level of less than 95% (or as ordered). *The depressed respiratory drive associated with metabolic alkalosis can lead to hypoxaemia and impaired oxygenation of tissues. Oxygen saturation levels of less than 90% indicate significant oxygenation problems.*
- Assess skin colour; note and report cyanosis around the mouth. *Central cyanosis, seen around the mouth and oral mucous membranes, indicates significant hypoxia.*
- Monitor mental status and level of consciousness (LOC). Report decreasing LOC or behaviour changes such as restlessness, agitation or confusion. *Changes in mental status or behaviour may be early signs of hypoxia.*
- Place in semi-Fowler's or Fowler's position as tolerated. *Elevating the head of the bed facilitates alveolar ventilation and gas exchange.*
- Schedule nursing care activities to allow rest periods. *The person who is hypoxaemic has limited energy reserves, necessitating frequent rest and limited activities.*
- Administer oxygen as ordered or necessary to maintain oxygen saturation levels. *Supplemental oxygen can help maintain blood and tissue oxygenation despite depressed respirations.*

Deficient fluid volume

A person with metabolic alkalosis often has an accompanying fluid volume deficit.

CONSIDERATION FOR PRACTICE

Assess intake and output accurately, monitoring fluid balance. In acute situations, hourly intake and output may be indicated. Urine output of less than 30 mL/hour indicates inadequate tissue perfusion, inadequate renal perfusion and an increased risk of acute renal failure.

- Assess vital signs, CVP and peripheral pulse volume at least every 4 hours. *Hypotension, tachycardia, low CVP and weak, easily obliterated peripheral pulses indicate hypovolaemia.*
- Weigh daily under standard conditions (time of day, clothing and scale). *Rapid weight changes accurately reflect fluid balance.*
- Administer intravenous fluids as prescribed using an electronic infusion pump. Monitor for indicators of fluid overload if rapid fluid replacement is ordered: dyspnoea, tachypnoea, tachycardia, increased CVP, jugular vein distension and oedema. *Rapid fluid replacement may lead*

to hypervolaemia, resulting in pulmonary oedema and cardiac failure, particularly in a person with compromised cardiac and renal function.

- Monitor serum electrolytes, osmolality and ABG values. *Rehydration and administration of potassium chloride will affect both acid–base and fluid and electrolyte balance. Careful monitoring is important to identify changes.*

Community-based care

When preparing the person with metabolic alkalosis for discharge, consider the cause of the alkalosis and any underlying factors. For example, provide teaching about the following:

- using appropriate antacids for heartburn and gastric distress
- using potassium supplements as ordered or eating high-potassium foods to avoid hypokalaemia if taking a potassium-wasting diuretic or if aldosterone production is impaired
- contact the primary attending doctor if uncontrolled or extended vomiting develops.

The person with respiratory acidosis

Respiratory acidosis is caused by an excess of dissolved carbon dioxide or carbonic acid. It is characterised by a pH less than 7.35 and a $PaCO_2$ greater than 45 mmHg. Respiratory acidosis may be either acute or chronic. In chronic respiratory acidosis, the bicarbonate is higher than 26 mEq/L as the kidneys compensate by retaining bicarbonate.

Risk factors

Acute or chronic lung disease (e.g. pneumonia or chronic obstructive pulmonary disease (COPD)) is the primary risk factor for respiratory acidosis. Other conditions that depress or interfere with ventilation, such as excess narcotic analgesics, airway obstruction or neuromuscular disease, also are risk factors for respiratory acidosis. Selected causes of respiratory acidosis are listed in Table 9.12.

Pathophysiology

Both acute and chronic respiratory acidosis results from carbon dioxide retention caused by alveolar hypoventilation. Hypoxaemia (low oxygen in the arterial blood) frequently accompanies respiratory acidosis.

ACUTE RESPIRATORY ACIDOSIS Acute respiratory acidosis occurs due to a sudden failure of ventilation. Chest trauma, aspiration of a foreign body, acute pneumonia and overdoses of narcotic or sedative medications can lead to this condition. Because acute respiratory acidosis occurs with the sudden onset of hypoventilation—for example, with cardiac arrest—the $PaCO_2$ rises rapidly and the pH falls markedly. A pH of 7 or lower can occur within minutes (Metheny, 2012). The serum bicarbonate level initially is unchanged because the compensatory response of the kidneys occurs over hours to days.

Hypercapnia (increased carbon dioxide levels) affects neurological function and the cardiovascular system. Carbon dioxide rapidly crosses the blood–brain barrier. Cerebral blood vessels dilate and, if the condition continues, intracranial pressure increases and *papilloedema* (swelling and inflammation of the optic nerve where it enters the retina) develops. Peripheral vasodilation also occurs and the pulse rate increases to maintain cardiac output.

CHRONIC RESPIRATORY ACIDOSIS Chronic respiratory acidosis is associated with chronic respiratory or neuromuscular conditions such as COPD, asthma, cystic fibrosis or multiple sclerosis. These conditions affect alveolar ventilation because of airway obstruction, structural changes in the lung or limited chest wall expansion. Most people with chronic respiratory acidosis have COPD with chronic bronchitis and emphysema. (See Chapter 36 for more information about COPD.)

In chronic respiratory acidosis, the $PaCO_2$ increases over time and remains elevated. The kidneys retain bicarbonate, increasing bicarbonate levels, and the pH often remains close to the normal range.

The acute effects of hypercapnia may not develop because carbon dioxide levels rise gradually, allowing compensatory changes to occur. When carbon dioxide levels are chronically elevated, the respiratory centre becomes less sensitive to the gas as a stimulant of the respiratory drive. The PaO_2 provides the primary stimulus for respirations. A person with chronic respiratory acidosis is at risk of developing *carbon dioxide narcosis*, with manifestations of acute respiratory acidosis, if the respiratory centre is suppressed by administering excess supplemental oxygen.

CONSIDERATION FOR PRACTICE

Carefully monitor neurological and respiratory status in the person with chronic respiratory acidosis who is receiving oxygen therapy. Immediately report a decreasing LOC or depressed respirations.

Manifestations

The manifestations of acute and chronic respiratory acidosis differ. In acute respiratory acidosis, the rapid rise in $PaCO_2$ levels causes manifestations of hypercapnia. Cerebral vasodilation causes manifestations such as headache, blurred vision, irritability and mental cloudiness. If the condition continues, the level of consciousness progressively decreases. Rapid and dramatic changes in ABGs can lead to unconsciousness and ventricular fibrillation, a potentially lethal cardiac arrhythmia. The skin of the person with acute respiratory acidosis may be warm and flushed and the pulse rate is elevated.

The manifestations of chronic respiratory acidosis include weakness and a dull headache. Sleep disturbances, daytime sleepiness, impaired memory and personality changes also may be manifestations of chronic respiratory acidosis (see 'Manifestations' box).

MANIFESTATIONS Respiratory acidosis

ACUTE RESPIRATORY ACIDOSIS
- Headache
- Warm, flushed skin
- Blurred vision
- Irritability, altered mental status
- Decreasing level of consciousness
- Cardiac arrest

CHRONIC RESPIRATORY ACIDOSIS
- Weakness
- Dull headache
- Sleep disturbances with daytime sleepiness
- Impaired memory
- Personality changes

INTERPROFESSIONAL CARE

A person with acute respiratory failure usually requires treatment in the emergency department or intensive care unit. The focus is on restoring adequate ventilation and gas exchange. Hypoxaemia often accompanies acute respiratory acidosis, so oxygen is administered as well. Supplemental oxygen is administered with caution to the person with chronic respiratory acidosis.

Diagnosis

The following laboratory and diagnostic tests may be ordered:
- *ABGs* show a pH of less than 7.35 and a $PaCO_2$ of more than 45 mmHg. In acute respiratory acidosis, the bicarbonate level is initially within normal range but increases to greater than 26 mEq/L if the condition persists. In chronic respiratory acidosis, both the $PaCO_2$ and the HCO_3^- may be significantly elevated.
- *Serum electrolytes* may show hypochloraemia (chloride level < 98 mEq/L) in chronic respiratory acidosis.
- *Pulmonary function tests* may be done to determine if chronic lung disease is the cause of the respiratory acidosis. However, these studies would not be done during the acute period.

Additional diagnostic tests may be done to identify the underlying cause of the respiratory acidosis. *Chest x-ray* and *sputum studies* (cytology and culture) may be ordered to identify an acute or chronic lung disorder. If drug overdose is suspected, *serum levels* of the drug may be obtained.

Medications

Bronchodilator drugs may be administered to open the airways and antibiotics prescribed to treat respiratory infections. If excess narcotics or anaesthetic has caused acute respiratory acidosis, drugs to reverse their effects (such as naloxone) may be given.

Respiratory support

Treatment of respiratory acidosis, either acute or chronic, focuses on improving alveolar ventilation and gas exchange. The person with severe respiratory acidosis and hypoxaemia may require intubation and mechanical ventilation. (See Chapter 36 for more information about these procedures.) The $PaCO_2$ level is lowered slowly to avoid complications such as cardiac arrhythmias and decreased cerebral perfusion. In a person with chronic respiratory acidosis, oxygen is administered cautiously to avoid carbon dioxide narcosis.

Pulmonary hygiene measures, such as breathing treatments or percussion and drainage, may be instituted. Adequate hydration is important to promote removal of respiratory secretions.

Nursing care

See 'Nursing care plan: A person with acute respiratory acidosis' below.

Health promotion

Health promotion activities related to respiratory acidosis focus on identifying, monitoring and teaching the person at risk. Carefully monitor the person receiving anaesthesia, narcotic analgesics or sedatives for signs of respiratory depression. Monitor the response of a person with a history of chronic lung disease to oxygen therapy. Educate the person who has an identified risk of respiratory acidosis (such as people using narcotic analgesia for cancer pain and those with chronic lung disease) and their families about early manifestations of respiratory depression and acidosis, and instruct them to contact their attending doctor immediately if manifestations develop.

Assessment

Assessment data related to respiratory acidosis include the following:
- *Health history*: current manifestations, including headache, irritability or lethargy, difficulty thinking, blurred vision and other symptoms; duration of symptoms and any precipitating factors such as drug use or respiratory infection; chronic diseases such as cystic fibrosis or COPD; current medications.
- *Physical assessment*: mental status and level of consciousness; vital signs; skin colour and temperature; rate and depth of respirations, pulmonary excursion, lung sounds; examination of optic fundus for possible papilloedema.
- *Diagnostic tests*: ABGs, serum electrolytes; white blood cell count (indicator of infection), sputum culture results, serum drug and toxicology results.

Nursing diagnoses and interventions

Restoring effective alveolar ventilation and gas exchange is the priority of interprofessional and nursing care for the person with respiratory acidosis.

Impaired gas exchange

- Frequently assess respiratory status, including rate, depth, effort and oxygen saturation levels. *Decreasing respiratory rate and effort along with decreasing oxygen saturation levels may signal worsening respiratory failure and respiratory acidosis.*

NURSING CARE PLAN A person with acute respiratory acidosis

Marlene Hurd, age 76, is eating lunch with her friends when she suddenly begins to choke and is unable to breathe. After several minutes of trying, an attendant at the senior centre successfully dislodges some meat caught in Ms Hurd's throat using the Heimlich manoeuvre. Ms Hurd is taken by ambulance to the emergency department for follow-up because she was apnoeic for 3 to 4 minutes, her respirations are shallow and she is disoriented.

ASSESSMENT

Ms Hurd is placed in an observation room. Oxygen is started at 4 L/min per nasal cannula. David Love, the nurse admitting Ms Hurd, makes the following assessments: T 37.6°C, P 102, R 36 and shallow, BP 146/92. Skin is warm and dry. Alert but restless and not oriented to time or place; she responds slowly to questions. Stat ABGs are drawn, a chest x-ray is done and D_5 1/2 NS is started intravenously at 50 mL/h.

The chest x-ray shows no abnormality. ABG results are pH 7.38 (normal: 7.35 to 7.45), $PaCO_2$ 48 mmHg (normal: 35 to 45 mmHg), PaO_2 92 mmHg (normal: 80 to 100 mmHg) and HCO_3^- 24 mEq/L (normal: 22 to 26 mEq/L).

DIAGNOSES

- *Impaired gas exchange* related to temporary airway obstruction.
- *Anxiety* related to emergency hospital admission.
- *Risk of injury* related to confusion.

PLANNING

- Explain the need to monitor vital and the need for oxygen therapy to the person and significant others.
- Explain the purpose for follow-up treatments of ABGs and continuous monitoring.
- Maintain a calm, quiet environment.
- Provide reorientation and explain all activities.
- Explain the importance of keeping the side rails in place and call bell within reach.

Expected outcomes

- Regain normal gas exchange and ABG values.
- Be oriented to time, place and person.
- Regain baseline mental status.
- Remain free of injury.

IMPLEMENTATION

- Monitor ABGs as per medical officer orders and collect ABG every 2 hours and report findings to the medical officer.
- Review ABG collection site pre and post collection of bloods.
- Monitor vital signs and respiratory status (including oxygen saturation) every 15 minutes for the first hour then every hour.
- Assess colour of skin, nail beds and oral mucous membranes every hour.
- Provide mouth hygiene hourly while O_2 being administered.
- Assess mental status and orientation every hour.
- Monitor anxiety level as evidenced by restlessness and agitation.

EVALUATION

Ms Hurd remains in the emergency department for 6 hours. Her ABGs are still abnormal and David Love now notes the presence of respiratory crackles and wheezes. She is less anxious and responds appropriately when asked about her name, time and place. Because she has not regained normal gas exchange, Ms Hurd is admitted to the hospital for continued observation and treatment.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the pathophysiological process that leads to acute respiratory acidosis in Ms Hurd.
2. Describe the effect of acidosis on mental function.
3. What teaching would you provide to Ms Hurd to prevent future episodes of choking?

REFLECTION ON THE NURSING PROCESS

1. Identify and outline what you have learned from this case study and how you will apply it to your future nursing practice.
2. As the Registered Nurse, what communication and education strategies can you use to ensure Ms Hurd is able to prevent further episodes of choking?

CONSIDERATION FOR PRACTICE

Frequently assess level of consciousness. A decline in LOC may indicate increasing hypercapnia and the need for increasing ventilatory support (such as intubation and mechanical ventilation).

- Promptly evaluate and report ABG results to the physician and respiratory therapist. *Rapid changes in carbon dioxide or oxygen levels may necessitate modification of the treatment plan to prevent complications of overcorrection of respiratory acidosis.*
- Place in semi-Fowler's to Fowler's position as tolerated. *Elevating the head of the bed promotes lung expansion and gas exchange.*
- Administer oxygen as ordered. Carefully monitor response. Reduce the oxygen flow rate or percentage and immediately report increasing somnolence. *Supplemental oxygen can suppress the respiratory drive in the person with chronic respiratory acidosis.*

Ineffective airway clearance

- Frequently auscultate breath sounds (whether on or off a mechanical ventilator). *Increasing adventitious sounds or decreasing breath sounds (faint or absent) may indicate worsening airway clearance due to obstruction or fatigue.*
- Encourage the person with chronic respiratory acidosis to use pursed-lip breathing. *Pursed-lip breathing helps maintain open airways throughout exhalation, promoting carbon dioxide elimination.*

- Frequently reposition and encourage ambulation as tolerated. *Repositioning, sitting at the bedside and ambulation promote airway clearance and lung expansion.*
- Encourage fluid intake of up to 3000 mL per day as tolerated or allowed. *Fluids help liquefy secretions and hydrate respiratory mucous membranes, promoting airway clearance.*
- Administer medications such as inhaled bronchodilators as ordered. *Inhaled bronchodilators help to relieve bronchial spasm, dilating airways.*
- Provide percussion, vibration and postural drainage as ordered. *Pulmonary hygiene measures such as these help to loosen respiratory secretions so they can be coughed out of airways.*

Community-based care

Planning and educating for home care focuses on the problem that caused the person to develop respiratory acidosis. The person who developed acute respiratory acidosis as a result of acute pneumonia or chest trauma may only require education to prevent future problems. If acute respiratory acidosis occurred secondary to a narcotic overdose, determine if the drug was prescribed for pain or if it was an illicit street drug. Provide education to the person who requires narcotic medication on a continuing basis. Refer the person using illicit drugs to a substance abuse counsellor or treatment centre as appropriate.

For a person with chronic lung disease, discuss ways to avoid future episodes of acute respiratory failure. Encourage the person to be immunised against pneumococcal pneumonia and influenza. Discuss ways to avoid acute respiratory infections and measures to take when respiratory status is further compromised.

The person with respiratory alkalosis

Respiratory alkalosis is characterised by a pH greater than 7.45 and a $PaCO_2$ of less than 35 mmHg. It is always caused by hyperventilation leading to a carbon dioxide deficit.

Risk factors

Anxiety with hyperventilation is the most common cause of respiratory alkalosis; therefore, anxiety disorders increase the risk of this acid–base imbalance. In the person who is critically ill, mechanical ventilation is a risk factor for respiratory alkalosis.

Pathophysiology

In acute respiratory alkalosis, the pH rises rapidly as the $PaCO_2$ falls. Because the kidneys are unable to rapidly adapt to the change in pH, the bicarbonate level remains within normal limits. Anxiety-based hyperventilation is the most common cause of acute respiratory alkalosis. Other physiological causes of hyperventilation include high fever, hypoxia, gram-negative bacteraemia and thyrotoxicosis. Early salicylate intoxication (aspirin overdose), encephalitis and high progesterone levels in pregnancy directly stimulate the respiratory centre, potentially leading to hyperventilation and respiratory alkalosis. Hyperventilation also can occur during anaesthesia or mechanical ventilation if the rate and tidal volume (depth) of ventilations are excessive.

If hyperventilation continues, the kidneys compensate by eliminating bicarbonate to restore the bicarbonate to carbonic acid ratio. The bicarbonate level is lower than normal in chronic respiratory alkalosis and the pH may be close to the normal range.

Alkalosis increases binding of extracellular calcium to albumin, reducing ionised calcium levels. As a result, neuromuscular excitability increases and manifestations similar to hypocalcaemia develop. Low carbon dioxide levels in the blood cause vasoconstriction of cerebral vessels, increasing the neurological manifestations of the disorder.

Manifestations

The manifestations of respiratory alkalosis include lightheadedness, a feeling of panic and difficulty concentrating, circumoral and distal extremity paraesthesias, tremors and positive Chvostek's and Trousseau's signs. The person also may experience tinnitus, a sensation of chest tightness and palpitations (cardiac arrhythmias). Seizures and loss of consciousness may occur. (See 'Manifestations' box below.)

MANIFESTATIONS Respiratory alkalosis

- Dizziness
- Numbness and tingling around mouth, hands and feet
- Palpitations
- Dyspnoea
- Chest tightness
- Anxiety/panic
- Tremors
- Tetany
- Seizures, loss of consciousness

INTERPROFESSIONAL CARE

Management of respiratory alkalosis focuses on correcting the imbalance and treating the underlying cause.

Diagnosis

ABGs generally show a pH greater than 7.45 and a $PaCO_2$ of less than 35 mmHg. In chronic hyperventilation, there is a compensatory decrease in serum bicarbonate to less than 22 mEq/L and the pH may be near normal.

Medications

A sedative or anti-anxiety agent may be necessary to relieve anxiety and restore a normal breathing pattern. Additional drugs to correct underlying problems other than anxiety-induced hyperventilation may be ordered.

Respiratory therapy

The usual treatment for anxiety-related respiratory alkalosis involves instructing the person to breathe more slowly and having the person breathe into a paper bag or rebreather mask. This allows rebreathing of exhaled carbon dioxide, increasing $PaCO_2$ levels and reducing the pH. If excessive ventilation by a mechanical ventilator is the cause of respiratory alkalosis,

ventilator settings are adjusted to reduce the respiratory rate and tidal volume as indicated. When hypoxia is the underlying cause of hyperventilation, oxygen is administered.

Nursing care

Health promotion

Identify the person at risk in the hospital (e.g. a person on mechanical ventilation or who has a fever or infection) and monitor assessment data and ABGs to identify early manifestations of hyperventilation and respiratory alkalosis.

Assessment, diagnoses and interventions

Ineffective breathing pattern

The usual cause of hyperventilation and respiratory alkalosis is psychological, although physiological disorders also can lead to hyperventilation. It is important not only to address the hyperventilation but also to identify the underlying cause.

- Assess respiratory rate, depth and ease. Monitor vital signs (including temperature) and skin colour. *Assessment data can help identify the underlying cause, such as a fever or hypoxia.*
- Obtain subjective assessment data such as the circumstances leading up to the current situation, current health and recent illnesses, or medication use and current manifestations. *Subjective data provide cues to the cause and circumstances of the hyperventilation response.*
- Reassure the person that they are not experiencing a heart attack and that symptoms will resolve when breathing returns to normal. *Manifestations of hyperventilation and respiratory alkalosis such as dyspnoea, chest tightness, or pain and palpitations can mimic those of a heart attack.*
- Instruct the person to maintain eye contact and breathe with you to slow the respiratory rate. *These measures help to make the person aware of respirations and provide a sense of support and control* (Ackley & Ladwig, 2011).
- Have the person breathe into a paper bag. *This allows the person to rebreathe exhaled carbon dioxide, increasing the $PaCO_2$ and decreasing the pH.*
- Protect the person from injury. *If hyperventilation continues to the point at which the person loses consciousness, respirations will return to normal, as will acid–base balance.*
- If the person has experienced repeated episodes of hyperventilation or has a chronic anxiety disorder, refer for counselling. *Counselling can help the person develop alternative strategies for dealing with anxiety.*

Community-based care

Planning and teaching for home care are directed towards the underlying cause of hyperventilation. If anxiety precipitated the episode, discuss anxiety management strategies with the person. Refer the person and family to a counsellor if appropriate. Teach the person to identify a hyperventilation reaction and how to breathe into a paper bag to manage it at home.

CHAPTER HIGHLIGHTS

- The volume and composition of body fluid are normally maintained by a balance of fluid and electrolyte intake; elimination of water, electrolytes and acids by the kidneys; and hormonal influences. Change in any of these factors can lead to a fluid, electrolyte or acid–base imbalance that adversely impacts on health.
- Fluid, electrolyte and acid–base imbalances can affect all body systems, especially the cardiovascular system, the central nervous system and the transmission of nerve impulses. Conversely, primary disorders of the respiratory, renal, cardiovascular, endocrine or other body systems can lead to an imbalance of fluids, electrolytes or acid–base status.
- Fluid and sodium imbalances commonly are related; both affect serum osmolality.
- Potassium imbalances are commonly seen in the person with acute or chronic illnesses. Both hypokalaemia and hyperkalaemia affect cardiac conduction and function. Carefully monitor cardiac rhythm and status in the person with very low or very high potassium levels.
- Calcium imbalances primarily affect neuromuscular transmission: hypocalcaemia increases neuromuscular irritability; hypercalcaemia depresses neuromuscular transmission. Magnesium imbalances have a similar effect.
- Acid–base imbalances may be caused by either metabolic or respiratory problems. Simple acid–base imbalances (respiratory or metabolic acidosis or alkalosis) are more commonly seen than mixed imbalances.
- Buffers, lungs and kidneys work together to maintain acid–base balance in the body. Buffers respond to changes almost immediately; the lungs respond within minutes; the kidneys, however, require hours to days to restore normal acid–base balance.
- The lungs compensate for metabolic acid–base imbalances by excreting or retaining carbon dioxide. This is accomplished by increasing or decreasing the rate and depth of respirations.
- The kidneys compensate for respiratory acid–base imbalances by producing and retaining or excreting bicarbonate and by retaining or excreting hydrogen ions.
- Careful monitoring of respiratory and cardiovascular status, mental status, neuromuscular function and laboratory values is an important nursing responsibility for the person with fluid, electrolyte or acid–base imbalances.

CONCEPT CHECK

1 A person is admitted to the emergency department with hypovolaemia. Which intravenous solution would the nurse anticipate administering?
1 Ringer's solution
2 10% dextrose in water
3 3% sodium chloride
4 0.45% sodium chloride

2 When assessing a person with fluid volume deficit, the nurse would expect to find:
1 increased pulse rate and blood pressure
2 dyspnoea and respiratory crackles
3 headache and muscle cramps
4 orthostatic hypotension and flat neck veins

3 The nurse caring for a person with acute hypernatraemia includes which of the following in the plan of care? (Select all that apply.)
1 Conduct frequent neurological checks.
2 Restrict fluids to 1500 mL per day.
3 Orient to time, place and person frequently.
4 Maintain intravenous access.
5 Limit length of visits.

4 Laboratory results for a person show a serum potassium level of 2.2 mEq/L. Which of the following nursing actions is of highest priority for this person?
1 Keep the person on bed rest.
2 Initiate cardiac monitoring.
3 Start oxygen at 2 L/min.
4 Initiate seizure precautions.

5 The nurse evaluates teaching about calcium supplement therapy as effective when the person states that she will take her calcium tablets:
1 all at one time in the morning
2 with meals
3 as needed for tremulousness
4 with a full glass of water

6 A person who is known to be an alcoholic presents with confusion, hallucinations and a positive Chvostek's sign. Which medication(s) should the nurse anticipate administering?
1 magnesium sulfate
2 calcium chloride
3 insulin and glucose
4 sodium bicarbonate

7 Arterial blood gas results for a person show pH 7.21, PaO_2 98 mmHg, $PaCO_2$ 32 mmHg and HCO_3^- 17 mEq/L. The nurse correctly interprets these values as indicative of which of the following acid–base imbalances?
1 metabolic acidosis
2 metabolic alkalosis
3 respiratory acidosis
4 respiratory alkalosis

8 A person is admitted with a suspected heroin overdose and a respiratory rate of 5 to 6 breaths per minute. Which of the following assessment data would the nurse anticipate? (Select all that apply.)
1 pH 7.29
2 alert and oriented
3 $PaCO_2$ 54 mmHg
4 HCO_3^- 32 mEq/L
5 skin warm and flushed

9 The nurse caring for a person undergoing several days of gastric decompression recognises that the person is at risk of which of the following acid–base imbalances?
1 metabolic acidosis
2 metabolic alkalosis
3 respiratory acidosis
4 respiratory alkalosis

10 A person undergoing mechanical ventilation following a severe chest wall injury and flail chest complains of chest tightness, anxiety and feeling as though she cannot get enough air. She is afraid she is having a heart attack. The nurse should first:
1 administer prescribed analgesic
2 contact respiratory therapy to evaluate ventilator settings
3 obtain arterial blood gases
4 notify the physician

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CHAPTER 10

NURSING CARE OF PEOPLE EXPERIENCING TRAUMA AND SHOCK

LISA GATZONIS

KEY TERMS

LEARNING OUTCOMES

- Describe the components and types of trauma including causes, effects to diagnose and management of life-threatening injuries.
- Discuss diagnostic tests, medications, blood transfusion and intravenous fluids used in assessing people experiencing trauma and shock.
- Explain organ donation and forensic implications of traumatic injury or death.
- Discuss the risk factors, aetiologies and pathophysiologies of hypovolaemic shock, cardiogenic shock, obstructive shock and distributive shock.
- Describe the role of the nurse in trauma prevention education and evaluate a plan of care to restore the functional health status of people experiencing trauma.

CLINICAL COMPETENCIES

- Obtain initial data about the person experiencing trauma to include history taking, assessment, review of medical history and communication with pre-hospital and other healthcare providers and family members.
- Communicate and document significant data and changes in the condition of a person who has sustained trauma.
- Evaluate responses to medical and surgical interventions for people sustaining multiple trauma and shock.
- Formulate nursing diagnoses based on manifestations recognised during the nursing assessment.
- Develop a plan of care for the person experiencing trauma based on scientific knowledge and individual diversity.
- Describe the role of the nurse in trauma prevention education and evaluate a plan of care to restore the functional health status of people experiencing trauma.
- Advocate for people's rights as indicated by documents that address end-of-life issues.

THE PERSON EXPERIENCING TRAUMA

Trauma is defined as injury to human tissues and organs resulting from the transfer of energy from the environment. In the past the term 'trauma' has been associated with the word 'accident'. *Accident* means that the injury occurred without intent, a result of random chance. We now know that a considerable number of injuries are preventable and not of random chance. Intentional and non-intentional trauma encompass a variety of injuries resulting from motor vehicle crashes, pedestrian injuries, gunshot wounds, falls, violence towards others or self-inflicted violence. The injuries, disabilities and deaths resulting from these acts constitute a major healthcare challenge.

- Traumatic injury is the leading cause of death in people under 45 years of age, a leading cause of morbidity, mortality and permanent disability, and a major source of health costs in Australia.
- Approximately 27 people die as a result of injury every day—almost 10 000 lives are lost each year (Australian Institute of Health and Welfare (AIHW), 2010).

Trauma usually occurs suddenly, leaving the person and family with little time to prepare for its consequences. Nurses provide a vital link in both the physical and psychosocial care for the injured person and family. In caring for the person who has experienced trauma, nurses must consider not only the initial physical injury but also its long-term consequences, including rehabilitation. Trauma may alter the person's previous way of life, potentially affecting independence, mobility, cognitive thinking and appearance.

Components of trauma

Trauma results from an abnormal exchange of energy between a host and a mechanism in a predisposing environment. The *host* is the person or group at risk of injury. Multiple factors influence the host's potential for injury: age, sex, race, economic status, pre-existing illnesses and use of substances such as street drugs and alcohol.

The *mechanism* is the source of the energy transmitted to the host. The energy exchanged can be mechanical, gravitational, thermal, electrical, physical or chemical. Table 10.1 lists the most common mechanisms for each type of energy. Mechanical energy is the most common type of energy transferred to a host in trauma. The most common mechanical source of injury in all adult age groups is the motor vehicle.

When describing a traumatic injury, *intention* is included as a component. Most gunshot and stab wounds are examples of intentional injuries. It is important to remember, however, that some gunshot wounds are unintentional, such as those that occur when children play with guns. Other common unintentional injuries result from motor vehicle crashes, falls, drowning and fires.

The final component of trauma is the *environment*. For example, a road that has become slippery after a storm is a physical environment that may contribute to an injury. Occupation is an important environmental factor to consider. Those in certain occupations face a high risk of trauma; examples include police officers, firefighters, professional athletes, race car drivers and taxi cab drivers. One's social environment also influences risk of injury; see the 'Meeting individualised needs' box on the next page for one example—domestic violence.

Types of trauma

Minor trauma causes injury to a single part or system of the body and is usually treated in a general practitioner's clinic or in the hospital emergency department. A fracture of the clavicle, a small second-degree burn and a laceration requiring sutures are examples of minor trauma. **Major** or **multiple trauma** involves

TABLE 10.1 Common mechanisms of injury by energy source

ENERGY SOURCE	COMMON MECHANISMS OF INJURY
Mechanical	Motor vehicles Firearms Machines
Gravitational	Falls
Thermal	Heating appliances Fire Freezing temperatures
Electrical	Wires, sockets and other electrical objects Lightning
Physical	Fists, feet and other body parts (as in physical assault) Sharp objects, such as knives Ultraviolet radiation Ionising radiation Water (drowning) Other submersion agents (e.g. grain) Explosions
Chemical	Drugs Poisons Industrial chemicals

serious single-system injury (such as the traumatic amputation of a leg) or multiple-system injuries. Multiple trauma is most often the result of a motor vehicle crash.

Trauma is further classified as either blunt or penetrating. **Blunt trauma** occurs when there is no communication between the damaged tissues and the outside environment. It is caused by various forces including *deceleration* (a decrease in the speed of a moving object), *acceleration* (an increase in the speed of a moving object), *shearing* (forces occurring across a plane, with structures slipping across each other), *compression* and *crushing*. Blunt forces often cause multiple injuries that can affect the head, spinal cord, bones, thorax and abdomen. Blunt trauma is frequently caused by motor vehicle crashes, falls, assaults and sports activities.

Penetrating trauma occurs when a foreign object enters the body causing damage to body structures. Structures commonly affected include the brain, lungs, heart, liver, spleen, the intestines and the vascular system. Examples of penetrating trauma are gunshot or stab wounds and impalement.

Other types of trauma include inhalation injuries from gases, smoke or steam; burn or freezing injuries; and blast injuries from explosions. Blast injuries result from the temperature and velocity of air movement and the force of projectiles from the explosion. Blast injuries are more severe in water than in air because blast waves travel further and faster in water. Trauma from blast injuries includes pulmonary oedema and haemorrhage, damage to abdominal organs, burns, penetrating injuries and ruptured tympanic membranes.

Outcome studies show a correlation between survival rates of multiple trauma victims and rapid response times by pre-hospital providers, coupled with appropriate decision making with regard to transporting the victim to a facility capable of treating their injuries (American College of Emergency Physicians, 2013). As a result, a system was devised to assist pre-hospital providers to make the appropriate decisions. People experiencing trauma are classified as class 1, 2 or 3 based on factors including mechanism of injury, vehicle speed, height of falls and location of penetrating injuries. Class 3 trauma is the least severe. An example would be a same-level fall without loss of consciousness or significant injury. Class 1 trauma involves life-threatening injuries likely to require medical specialists or immediate surgical intervention. While any hospital emergency department should be capable of caring for class 3 trauma people, people meeting class 1 or 2 criteria should be transported to a designated trauma centre when possible. Facilities designated as trauma centres have medical specialists and surgical coverage available or on call 24 hours a day.

Effects of traumatic injury

Death is a common result of serious traumatic injury, and falls into one of three categories related to the time span between injury and death: immediate, early or late. Immediate death happens within minutes at the scene from such injuries as a torn thoracic aorta or decapitation. Early death occurs within several hours of the injury from shock or delay in recognising injuries—causes may be major abdominal or thoracic injuries, or progression of intracranial haemorrhage. Late death generally occurs one or more days after the injury and results from multiple organ failure, sepsis and coagulopathies.

Because of the serious consequences of trauma, it is important to rapidly identify the person's injuries and institute appropriate interventions quickly. Following are common results of trauma and interventions necessary for good outcomes.

MEETING INDIVIDUALISED NEEDS **Assessing older adult abuse and domestic violence**

Intimate partner violence (IPV)

Most incidents of domestic violence are not reported; thus it is believed that the available data greatly underestimate the true magnitude of the problem. From the findings of the Personal Safety Survey conducted in 2012, it is estimated that 49% of men and 41% of women have experienced some form of violence since the age of 15, and that 62% of women compared to 8% of men have experienced their most recent incident of physical assault by a male in their home (Phillips & Vandenbroek, 2014). Domestic violence is one of the most common causes of injury to women in Australia.

Domestic violence is a widespread problem that occurs regardless of age, sex, race, socioeconomic status or education.

Violence and the older adult

Older adult abuse is defined as anything that endangers the life of an older adult. This can range from physical or emotional assault to intimidation, neglect or financial exploitation. Wilful deprivation of food or medical care is also included.

Data indicate that between 2% and 5% of Australians over the age of 65 years have experienced abuse, up to 80% of perpetrators are family members of the victims (the large majority being their children), financial and psychological abuse are the most common forms of abuse, and women are twice as likely to be victims of abuse (Lacey, 2012).

Diagnosis of abuse

The general approach to diagnosis in abuse situations is challenging as the abuse is often hidden. With spousal, older adult or child abuse, the task of identification is complex. The following are clues to identify violence-related injuries:

- injuries that do not correlate with the history
- injuries that suggest a defensive posture
- injuries during pregnancy
- pattern injuries
- pattern burns
- sexual abuse/rape
- unusual or unexplained fractures
- signs of confinement
- unusual interaction between the person and the caregiver
- lack of medical attention; immunisations not up to date; poor dental health
- unexplained dehydration or malnutrition.

Airway obstruction

Maintenance of the airway and cervical spine are the highest priority in the trauma patient. Other distracting injuries may take the inexperienced practitioner away from the airway, but if the airway is not patent (open) and the person is unable to deliver oxygen to vital organs all other interventions are futile.

Assessment includes determining airway patency. If the person is unresponsive, manual opening of the airway using a jaw-thrust or chin-lift manoeuvre is necessary. The jaw thrust is the recommended manoeuvre for people having suspected cervical spine injury. Once the airway is opened, the practitioner must identify any potential obstruction from the tongue, loose teeth, foreign bodies, bleeding, secretions, vomitus or oedema. If the person is responsive and can vocalise, that is a good indication that the airway is clear.

Any time the nurse performs an intervention it is important to reassess the effectiveness of the intervention. For example, if the nurse suctions the airway to remove vomitus, they would reassess the airway after suctioning to determine if that intervention was successful or if the airway needs to be suctioned a second time.

All trauma victims should receive high-flow oxygen until stabilised. Assessment of breathing effectiveness is paramount. Assessment should include whether the person has spontaneous breathing, good rise and fall of the chest, determination of skin colour, general rate and depth of respirations, use of abdominal or accessory muscles, position of the trachea, observation of chest wall integrity and presence of jugular vein distension, bilateral breath sounds and any surface trauma. Consider pulse oximetry and cardiac monitoring as well.

In addition to suctioning, other available airway adjuncts include oral or nasal pharyngeal airways, oxygen delivery devices, laryngeal mask airways, Combitubes and endotracheal intubation (see Figure 10.1). Intubation is the preferred method of airway management if the person is unable to maintain oxygenation or an open airway.

People experiencing trauma may exhibit several aspects of airway management that are unique and require special preparation and precautions, as discussed next.

CLOSED HEAD INJURY Changes in haemodynamics, oxygenation and ventilation should be minimised in order to maintain adequate cerebral perfusion pressure. Laryngoscopy causes a marked increase in intracranial pressure (ICP).

The goal is to maintain a $PaCO_2$ of 30 to 35 mmHg. Lignocaine administered 3 to 5 minutes prior to intubation can blunt an increase in ICP that is secondary to laryngeal stimulation. In a normotensive person, beta-blockers are given 2 to 3 minutes prior to intubation to attenuate the sympathetic response. Effective induction agents such as etomidate or thiopental have not been shown to increase ICP (Turner, Wakim & Secrest, 2005).

MAXILLOFACIAL TRAUMA Significant distortion of normal anatomy occurs in facial trauma and respiratory compromise is not uncommon. Even in victims who present with mild respiratory compromise, rapid deterioration from oedema or haemorrhage can occur. A surgical airway may be the only alternative.

DIRECT AIRWAY TRAUMA Penetrating trauma to the neck is associated with a high degree of morbidity and mortality.

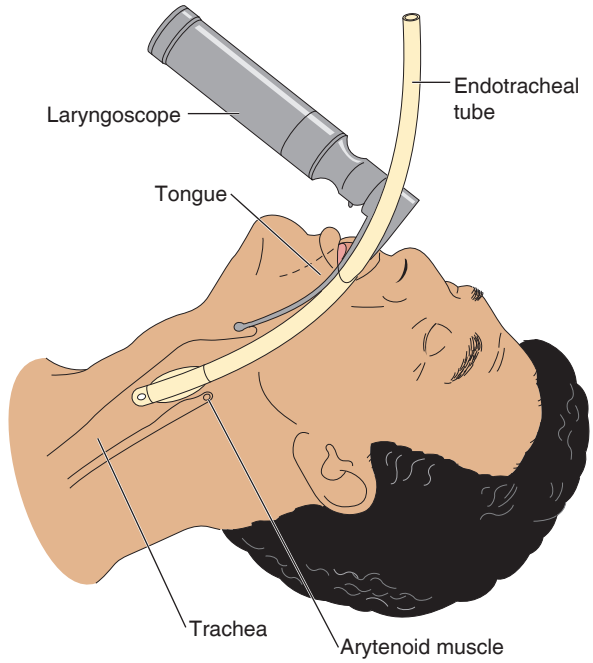


FIGURE 10.1 ■ Placement of an oral endotracheal tube (ETT) for intubation. When the ETT is in place, air or oxygen can be blown into the external opening of the tube and enter the trachea

Airway involvement includes dyspnoea, cyanosis, subcutaneous emphysema, hoarseness or air bubbling from the wound. Orotracheal intubation with rapid-sequence intubation is the technique of choice. The key is early identification of the need for intubation before the person has no airway at all. Tracheobronchial injury occurs in approximately 10% to 20% of people with penetrating neck injuries.

CERVICAL SPINE INJURY In the presence of a presumed cervical spine injury, precautions for securing an airway are consistently applied. Approximately 1.5% to 3% of major trauma victims have clinically significant cervical spine injuries (Desjardins, 2013). Oral intubation with manual in-line axial head and neck stabilisation is a safe method. The probability of cervical spine injury is decreased if the following criteria are met:

- absence of midline cervical spine tenderness
- normal alertness
- absence of intoxication
- absence of a painful distracting injury
- no focal neurological defects.

BURNS Burn victims with airway compromise require aggressive management. Upper airway oedema associated with inhalation or enclosed-space fires can progress during the post-burn phase. Securing an airway sooner rather than later is the goal. See Chapter 16 for nursing care of the person with burns.

Thoracic effects

TENSION PNEUMOTHORAX A **pneumothorax** results when air enters the potential space between the parietal and visceral pleura. The thorax is completely filled by the lungs, and

surface tension between the pleural surfaces holds the lungs to the chest wall. Air present in the pleural space will eventually collapse the lungs. A **tension pneumothorax** is a life-threatening condition and requires immediate intervention. On inspiration, air enters the pleural space which cannot escape on expiration; this then increases intrapleural pressure. This pressure collapses the lung and causes a shift in the mediastinal contents, resulting in compression of the heart, great vessels, trachea and, eventually, the unaffected lung. In turn, this causes the following signs and symptoms:

- severe respiratory distress
- hypotension
- jugular vein distension
- tracheal deviation towards the uninjured side
- cyanosis.

The immediate short-term lifesaving intervention is a needle thoracotomy, in which a large-bore needle is inserted into the second intercostal space at the midclavicular line (see Figure 10.2).

FLAIL CHEST Flail chest is the fracture of two or more ribs in two or more separate locations, leading to an unstable thoracic wall segment. Paradoxical movement of the chest wall is seen, with the area sinking into the chest cavity with inspiration and protruding with expiration. The area must be supported quickly to re-establish effective respiration and subsequent ventilation.

THORACIC CONTUSION AND RUPTURE Bruising of thoracic tissue is referred to as contusion. Pulmonary contusion is the most common traumatic chest injury, mostly from motor vehicle accidents. As a shock wave force travels through the parenchyma, diffuse haemorrhage and alveolar oedema develop, impairing gas exchange. Diaphragmatic rupture is a rare traumatic injury but can result in herniation of abdominal contents into the thoracic cavity, causing respiratory compromise.

Myocardial contusion results in extravasation of red blood cells into the myocardial fibres. As myocardial cells are injured, it is believed that cardiac output diminishes due to reduction in contractile strength. Myocardial rupture is an acute traumatic tear of any structures of the heart, and although rare it is fatal.

Cardiac tamponade occurs when blood or fluid collects in the pericardial sac. Resulting in myocardial compression, this condition is potentially life threatening and should be addressed immediately with pericardiocentesis (see Chapter 30).

Aortic rupture (transection) can result from acceleration–deceleration injury or blunt chest trauma. This injury is commonly fatal due to profuse bleeding.

Haemorrhage

When the person has suffered an injury that causes external haemorrhage, such as severing of an artery, the bleeding must be controlled immediately. This may be done by applying direct pressure over the wound and applying pressure over arterial pressure points (see Figure 10.3).

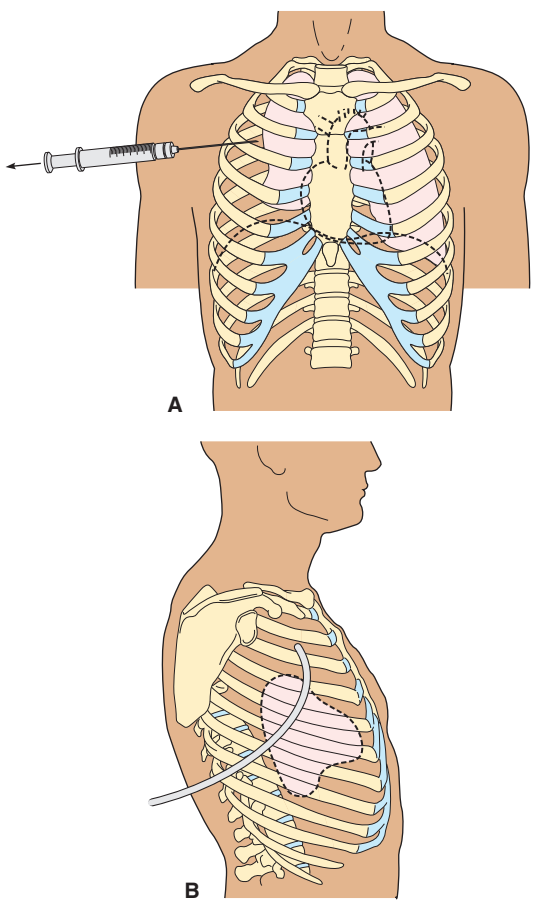


FIGURE 10.2 ■ A needle thoracostomy may be used in the emergency treatment of a tension pneumothorax. *A*, A large-gauge needle is introduced and air and fluid are aspirated. *B*, Alternatively, a chest tube may be inserted and connected to a chest drainage system

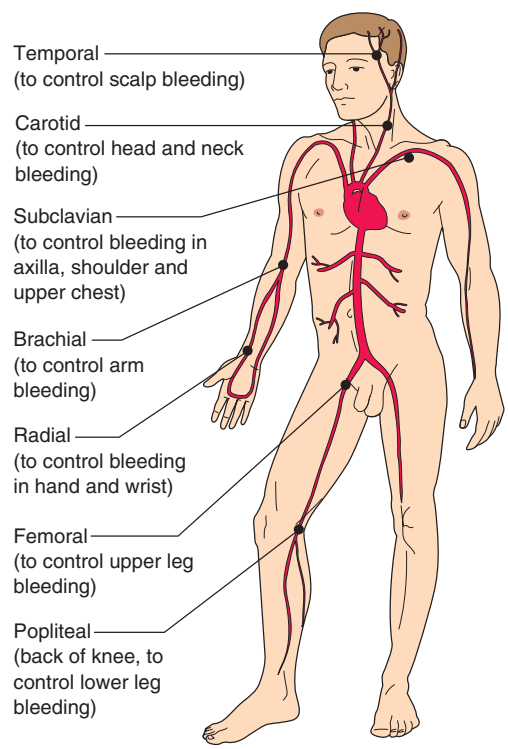


FIGURE 10.3 ■ The major pressure points used for the control of bleeding

Internal haemorrhage may result from either blunt or penetrating traumatic injury. Discovering the cause and location of the injury, as well as the extent of related blood loss, are the most important concerns. Several potential spaces in the body can accommodate large amounts of blood that may accumulate (called third spacing) following injury. For example, bleeding into the pleural space may occur with chest trauma (haemothorax) and bleeding into the abdominal cavity may occur with abdominal trauma. A pelvic fracture may cause massive haemorrhage into the retroperitoneal region. Once the source of internal haemorrhage has been recognised, interventions are initiated, including operative control of bleeding and continual assessment. Haemorrhage may result in hypovolaemic shock (discussed later in the chapter).

Integumentary effects

Injuries to the integument generally are not as serious as other injuries, with the exception of burns (see Chapter 16). The primary organ involved in integumentary trauma is the skin; however, underlying structures may also be injured. Injuries may result from either blunt or penetrating sources. It is important to evaluate all injuries to the integument because they may indicate a more serious injury such as an open fracture. Additionally, large wounds may contribute to significant blood loss.

Five specific injuries to the integument are contusions, abrasions, puncture wounds, lacerations and full-thickness avulsion injuries (see Figure 10.4). **Contusions**, or superficial tissue injuries, result from blunt trauma that causes the breakage of small blood vessels and bleeding into the surrounding tissue. **Abrasions** or partial-thickness denudations of an area of integument generally result from falls or scrapes. **Puncture wounds** occur when a sharp or blunt object penetrates the integument. **Lacerations** are open wounds that result from sharp cutting or tearing. Injuries to the integument are at risk of contamination from dirt, debris or foreign objects. Infection may cause further physical stress to the person with multiple injuries. **Full-thickness avulsion injuries** are injuries that result in loss of all of the layers of the skin, causing fat and muscle to be exposed. The size of the wound impacts on both the length of time necessary for healing to take place and the risk of infection. These types of injuries are treated by allowing new skin to grow from the edges, suturing the wound together, reattaching avulsed skin or by skin grafting.

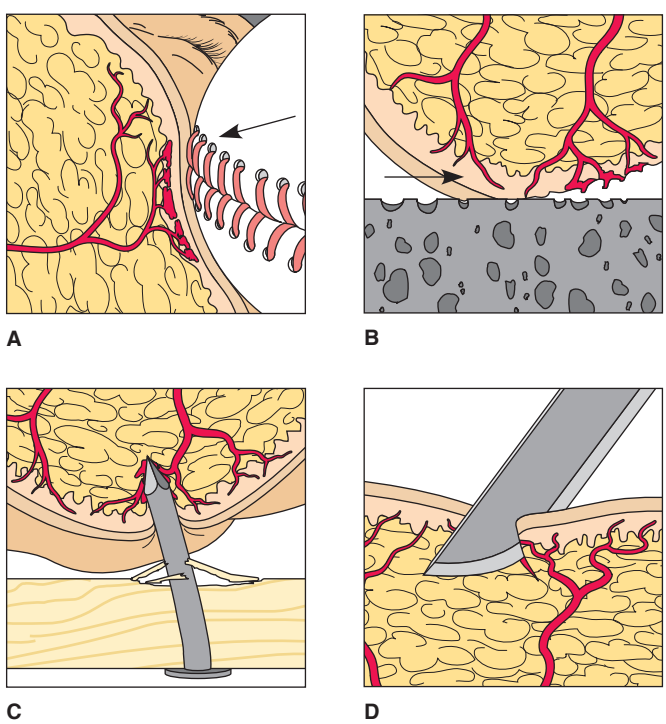


FIGURE 10.4 ■ Traumatic injuries to the skin include *A*, contusion; *B*, abrasion; *C*, puncture wound; *D*, laceration

Abdominal effects

The abdomen contains both solid organs (liver, spleen and pancreas) and hollow organs (stomach and intestines). Direct trauma to the abdomen can lacerate and compress the solid organs and cause burst injuries to the hollow organs. Blood vessels may be torn and organs may be displaced from their blood supply, producing life-threatening haemorrhage. Damage to the mesenteric vessels supplying the bowel can result in bowel ischaemia and infarction. Injury to the stomach, pancreas and small bowel may allow digestive enzymes to leak into the abdominal cavity. Rupture of the large bowel results in escape of faeces which causes peritonitis. Blunt or penetrating trauma to the abdomen may also cause rupture of the diaphragm with herniation of the abdominal organs into the thoracic cavity. The immediate threat following abdominal trauma is haemorrhage and the later threat is peritonitis.

Musculoskeletal effects

Musculoskeletal injuries may occur alone or with multiple injuries as the result of blunt or penetrating trauma. Musculoskeletal injuries usually are not considered a high priority in the care of the person with multiple injuries. Exceptions are the life- or limb-threatening musculoskeletal injury, such as a dislocated hip, pulseless extremity or significant blood loss such as from a femur or pelvic fracture. Musculoskeletal injuries may provide clues to the presence of other serious injuries; for example, a fractured clavicle may indicate an associated thoracic injury. Care of the person who has suffered a musculoskeletal injury is discussed in Chapter 39.

Neurological effects

Head injuries are a common type of injury sustained as the result of trauma. Injuries to the spinal cord resulting in loss of neurological function are devastating outcomes of trauma, but they are much less common than head injuries. Most head and spinal cord injuries result from blunt trauma and are sustained in motor vehicle crashes. Falls, sports injuries and assault are other sources of neurological injury. Care of the person with a neurological injury is discussed in Chapters 41 and 43.

Multiple organ dysfunction syndrome

Multiple organ dysfunction syndrome (MODS) is a common complication of severe injury and a frequent cause of death in intensive care units. MODS is a progressive impairment of two or more organ systems. This is the result of an uncontrolled inflammatory response to severe injury or illness.

People at risk of MODS are those with a disturbance in homeostasis resulting from one or a combination of the following conditions:

- infection
- injury
- inflammation
- ischaemia
- immune response
- intoxication of substances
- iatrogenic factors.

The primary organ systems involved in MODS are the respiratory, renal, hepatic, haematological, cardiovascular, gastrointestinal and neurological systems. Supportive therapy depends on the identification of correctable causes and this may be one or a combination of several therapies. Surgical intervention, antibiotic administration, corticosteroid administration and correction coagulopathies are some of the therapies used for this condition. The occurrence of MODS following traumatic injuries causes more than half of the late mortality following trauma.

Effects on the family

Trauma usually occurs suddenly and with little warning. It may result in death or cause injury serious enough to alter both the person's and the family's lives. The suddenness and seriousness of the event are precipitating factors in the development of a psychological crisis. During the past decade, some emergency departments have instituted care plans that allow families to be present during resuscitation. This policy is not without controversy, but it should be considered when appropriate in conjunction with institution work practices.

INTERPROFESSIONAL CARE

Interprofessional care of the trauma victim depends on a team approach. Providing trauma care with a team focus helps each team member know their role. Prompt delegation of tasks and responsibilities improves the person's chances of survival and decreases the morbidity that may result from traumatic injuries.

Pre-hospital care

The major functions of pre-hospital care include injury identification, critical interventions and rapid transport.

INJURY IDENTIFICATION Emergency care of the person experiencing trauma is based on rapid assessment to identify injuries and begin appropriate interventions. Injuries that indicate the need for trauma centre care include:

- penetrating injuries to the abdomen, pelvis, chest, neck or head
- spinal cord injuries with deficit
- crushing injuries to the abdomen, chest or head
- major burns
- injuries leading to airway compromise or obstruction.

Many methods help healthcare providers determine the seriousness of the person's injuries and the potential for survival. Scoring systems such as the Champion Revised Trauma Scoring System can be helpful (see Table 10.2). A primary trauma assessment must be rapid and comprehensive. As a prompt, using an alphabetical mnemonic can be helpful:

- **A** is airway assessment (with cervical spine immobilisation) to determine if the airway is patent, maintainable or non-maintainable.
- **B** is breathing evaluation for spontaneous respirations or ventilator impedance—such as by rib fractures or a collapsed lung.
- **C** is circulatory assessment by palpating peripheral and central pulses; assessing capillary refill, skin colour and temperature; and identifying any external sources of bleeding.
- **D** is disability and refers to neurological status. Assessment includes level of consciousness and pupillary function assessment, response to verbal or painful stimuli, and assessment of blood glucose level.
- **E** is exposure/environment where a whole-body assessment for any obvious injuries is completed while ensuring that hypothermia does not occur (i.e. use of heated blankets, warmed intravenous fluids).

Secondary assessment usually begins while the primary assessment is underway. This assessment extends the alphabetical mnemonic.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 9:
Recognising and Responding to Clinical Deterioration in Acute Health Care

'The intention of this standard is to ensure a patient's deterioration is recognised promptly and appropriate action is taken.' (ACSQHC, 2012, p. 61)

Implementing this standard is achieved by the establishment and maintenance of systems for recognising and responding to clinical deterioration.

These systems include processes that recognise clinical deterioration and escalating care to ensure appropriate action is taken in patients whose condition is deteriorating.

Effective communication should exist across all individuals involved in a person's care (including the person themselves and their significant others).

Caring for individuals experiencing trauma and shock requires the need to observe, recognise and monitor physiological changes that could signal a patient's deterioration.

Efficient and appropriate systems are imperative to ensure the safety of not only the person receiving appropriate care, but also any other individual involved in their care.

Source: © Australian Commission on Safety and Quality in Health Care.

TABLE 10.2 Champion Revised Trauma Scoring System

TEST	SCORE	CODED VALUE
Glasgow Coma Scale*	13 to 15	4
	9 to 12	3
	6 to 8	2
	4 to 5	1
	3	0
Systolic blood pressure (mmHg)	> 89	4
	76 to 89	3
	50 to 75	2
	1 to 49	1
	0	0
Respiratory rate (breaths/min)	10 to 29	4
	> 29	3
	6 to 9	2
	1 to 5	1
	0	0
	Total score:	_____

The highest possible total score is 12. The lowest possible score is 0. The higher the total score, the greater the chance of survival.

*See Chapter 40 for instructions for using the Glasgow Coma Scale.

Source: Based on Centers for Disease Control and Prevention (2009). Guidelines for field triage of injured patients. *Morbidity and Mortality Weekly Report,* 58(RR-1), 15. Retrieved from www.cdc.gov/mmwe/pdf/rr/rr5801.pdf.

- **F** is a full set of vital signs. It may also stand for having family members present during treatment.
- **G** is giving comfort measures, both physical and emotional, to the person and family.
- **H** is head-to-toe assessment and medical history that includes visual and manual assessment as well as auscultation.
- **I** is inspection of posterior surfaces for any injuries.

The Glasgow Coma Scale is another scoring system that is used to quantify the level of consciousness following traumatic brain injury (see Chapter 40).

CRITICAL INTERVENTIONS As life-threatening problems are identified during the primary assessment, appropriate on-the-scene interventions must be performed immediately. These include providing life support, immobilising the cervical spine, managing the airway and treating haemorrhage and shock.

Immobilisation of the person's cervical spine is a primary intervention. The person is placed on a spine board and a cervical collar and head immobiliser are applied (see Figure 10.5). The cervical spine may also be immobilised by log rolling the person onto a board, placing towel rolls or a head immobiliser along the sides of their head, and securing them to the board. If the person was wearing a helmet at the time of injury, the helmet should remain on until they arrive at the hospital, unless their airway is at risk. If necessary, healthcare personnel at the scene will remove the helmet by manipulating it over the person's nose and ears while holding their head and neck immobile; safe removal requires at least two people. Improper removal risks injury or additional injury to the spinal cord.

FIGURE 10.5 ■ Immobilisation of the cervical spine at the scene of the accident is essential to prevent further injury to the spinal cord. The combined use of a hard cervical collar, head blocks and tape best restricts flexion, extension, rotation and lateral bending of the neck

Source: © Michael Donne/SPL/Getty Images.

If the person's airway is patent, oxygen is administered. Ventilations may be assisted with a bag-valve-mask resuscitator until airway management is achieved. Active external bleeding is controlled by direct pressure. Measures to reverse shock (discussed later in the chapter) are initiated.

RAPID TRANSPORT People who have multiple injuries must be transported as soon as possible to a regional trauma centre. The most common modes of rapid transport are ground ambulance and air ambulance, which includes helicopters specially staffed and equipped to care for trauma victims. Figure 10.6 shows a flight nurse assessing a person. Stable people within access of a ground ambulance are best transported by ground. Unstable people and those injured in the wilderness or other areas in which ground access is difficult may best be transported by air. When these transport systems are unavailable, the person is transported by any possible means.

Emergency department care

ON ARRIVAL Multidisciplinary care and teamwork are required to ensure assessment and resuscitation strategies are implemented in a timely manner. Initial assessment and evaluation follows the process of primary survey, resuscitation, secondary survey, diagnosis, then definitive treatment or transfer to an appropriate healthcare facility that can provide this treatment.

DIAGNOSIS The diagnostic tests ordered once the person reaches the hospital depend on the type of injury they have sustained. Tests that may be ordered for victims of trauma include the following:

- *Blood type and crossmatch* involves typing the person's blood for ABO antigens and Rhesus (Rh) factor, screening the blood for antibodies and crossmatching the person's serum and donor red blood cells.

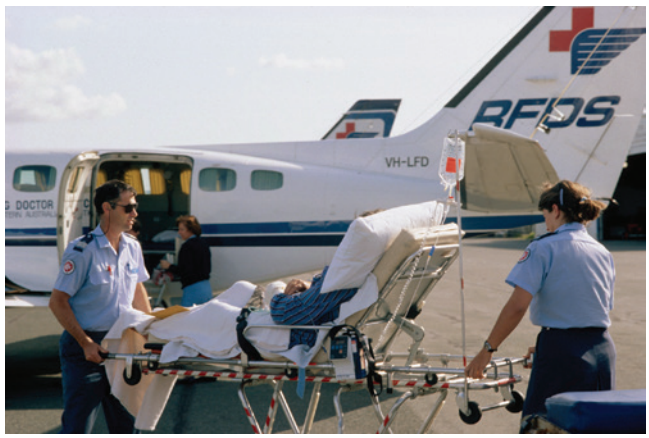


FIGURE 10.6 ■ Flight nurses provide initial assessment, stabilisation and support for people with trauma

Source: © Robert Garvey/Corbis.

- *Arterial blood gases* evaluates oxygenation, acid–base balance and the presence of metabolic or respiratory compensatory mechanisms.
- *Full blood examination, electrolytes and coagulation* may be ordered after stabilisation and resuscitation.
- *Blood glucose level* to identify correctable cause of decreased level of consciousness; also early hypoglycaemia has been linked to increase risk of infection and mortality post trauma.
- *Blood alcohol level* measures the amount of alcohol in a person's blood. Studies have found that between 20% and 50% of people who are injured may be intoxicated. Alcohol alters the person's level of consciousness and response to pain.
- *Urinalysis*—perform dipstick to exclude occult haematuria. *Urine drug screen* may also be ordered. Like alcohol, drugs such as cocaine alter the person's level of consciousness and overall response to the primary survey.
- *Radiography*—anterior posterior chest x-ray to aid in diagnosis and confirmation of endotracheal or invasive central line placement; pelvic film for identification of pelvic fractures.
- *Focused abdominal sonography in trauma (FAST)* exam is a portable ultrasound examination used to identify the presence of free fluid in body cavities where it is not supposed to be. Primary focus is on the peritoneum but can also look at the pleura and pericardium.
- *Diagnostic peritoneal lavage* determines the presence of blood in the peritoneal cavity, which may indicate abdominal injury. This test is generally done in the emergency department. A local anaesthetic (such as lignocaine) is injected subcutaneously and a small incision is made in the lower abdomen. A catheter is placed into the peritoneal cavity and any free blood is aspirated. If free blood is found, the person is taken to the operating room for exploratory surgery. If no free blood is aspirated, 1 L of a warm isotonic crystalloid solution (such as normal saline) is rapidly infused into the peritoneal cavity and then allowed to drain by gravity. If the solution returns pink and is found to have the presence of red blood cells, white blood cells, bile, food or faeces, the test is considered positive and the person is taken to the operating room for exploratory surgery. This procedure has been used less since the inception of the FAST exam.
- *Pregnancy test* for any woman of childbearing age rules out the potential for pregnancy and foetal injury.
- *Computed tomography (CT) scans* can discover injuries to the brain, skull, spine, spinal cord, chest and abdomen.
- *Magnetic resonance imaging (MRI) scans* can discover injuries to the brain and spinal cord.

MEDICATIONS

Medications used to treat the person who has experienced trauma depend on the type and severity of the injuries, as well as the degree of traumatic shock that is present. The following general categories of medications may be used. (Fluid administration and the drugs listed are covered later in the chapter in discussion of the collaborative care of the person in shock.)

- Blood components and crystalloids are administered intravenously in the initial treatment of traumatic shock to replace intravascular volume.
- Inotropic drugs (drugs that increase myocardial contractility) are given to increase cardiac output and improve tissue perfusion. These drugs, administered only after fluid volume restoration, include dopamine hydrochloride, dobutamine hydrochloride and isoprenaline hydrochloride (Isuprel).
- Vasopressors may be administered in conjunction with fluid replacement to treat neurogenic, septic or anaphylactic shock. Examples of vasopressors include dopamine, adrenaline and noradrenaline.
- Opioids administered by bolus or continuous infusion are used to treat pain as soon as possible. However, the effects of the pain medications may alter the person's responses and mask potential injuries. If pain medications are administered they must be carefully regulated and the person must be closely monitored.
- Immunisation: if the person has penetrating and open wounds, tetanus immunisation status must be determined. If they are unable to remember when the last tetanus immunisation was given or are unable to answer, tetanus prophylaxis is given.

BLOOD TRANSFUSIONS

Blood and blood components are initially produced in the body and then donated for use by another person through a **transfusion** (an infusion of blood or blood components). A person may be given whole blood, packed red blood cells (RBCs), platelets, plasma, albumin, clotting factors, prothrombin or cryoprecipitate (see Table 10.3). Blood and blood components increase the amount of haemoglobin available to carry oxygen to the cells, improve haemoglobin and haematocrit levels during active bleeding, increase intravascular volume and replace deficient substances such as platelets and clotting factors.

Each person has one of four blood types: A, B, AB or O. The blood group antigens A and B, present on RBC membranes, form the basis for the ABO blood categorisation. The presence or absence of these inherited antigens determines one's blood type. People with blood type A have A antigens, those with type B have B antigens, those with type AB have both antigens and those with neither antigen have blood type O (called a universal donor).

FAST FACTS

- Type AB blood is the 'universal recipient'.
- Type O blood is the 'universal donor'.

ABO antibodies develop in the serum of people whose RBCs lack the corresponding antigen; these antibodies are called anti-A and anti-B. The person with blood type B has A antibodies, the person with type A has B antibodies, the person with type O has both types of antibodies and the person with blood type AB has no antibodies (called a universal recipient).

A third antigen on the RBC membrane is D. People who are Rh positive (RH^+) have the D antigen, whereas people who are Rh negative (RH^-) do not. These antigens and antibodies may cause ABO and Rh incompatibilities.

TABLE 10.3 Volume resuscitation therapies

COMPONENT	INDICATIONS	ADVANTAGES	DISADVANTAGES
Compound sodium lactate (or Hartmann's solution)	Restoration of circulating volume Replacement of electrolyte deficits	Good availability Safe to use Low cost Aids in buffering acidosis	Rapid movement from the intravascular to the extravascular space, leading to three or more times requirement for replacement
Normal saline	Restoration of circulating volume Vehicle compatible with administration of blood	Good availability Low cost Safe to use	Hyperchloraemic acidosis associated with prolonged use of sodium solutions
Whole blood	Replaces blood volume and oxygen-carrying capacity in haemorrhage and shock	Contains RBCs, plasma proteins, clotting factors and plasma	Contains few platelets or granulocytes; deficient in clotting factors V and VII Greatest risks are for incompatibility or circulatory overload Risk of transmitting blood-borne pathogens
Packed RBCs	Restoration of intravascular volume Replacement of oxygen-carrying capacity	One unit of RBCs should increase the haemoglobin of a 70 kg adult by approximately 1 g/L in the absence of volume overload or continuing blood loss	Red cells require compatibility testing Risk of transmitting blood-borne pathogens Should be warmed to prevent hypothermia Contains little or no clotting factors
Platelets	Significant thrombocytopenia (platelet count less than 50×10^9/L) Continued haemorrhage	Compatibility testing is not required Typical platelet transfusion should raise the platelets of a 70 kg adult approximately $20–40 \times 10^9$/L	Post-exposure prophylaxis with anti-Rh immune globulin should be considered following Rh^+ platelet transfusion to an Rh^- woman Risk of transmitting blood-borne pathogens
Albumin	Expends blood volume in shock and trauma	Good availability	Is not a substitute for whole blood Hypersensitivity reactions can occur Risk of transmitting blood-borne pathogens
Fresh frozen plasma (FFP)	Documented coagulopathy Restoration of clotting factors Supplies plasma proteins	Crossmatching and Rh compatibility not required	Thawed by transfusion service provider—takes approx. 30 mins Should be ABO compatible Risk of transmitting blood-borne pathogens
Cryoprecipitate	Coagulopathy with low fibrinogen Restoration of fibrinogen	Rh type not important	Risk of transmitting blood-borne pathogens Contains haemagglutinins Should be ABO compatible, as intravascular haemolysis can occur if a large volume of ABO-incompatible cryoprecipitate is administered

TABLE 10.4 Blood group types and compatibilities

BLOOD GROUP	RBC AGGLUTINOGENS	SERUM AGGLUTINOGENS	COMPATIBLE DONOR BLOOD GROUPS	INCOMPATIBLE DONOR BLOOD GROUPS
A	A	Anti-B	A, O	B, AB
B	B	Anti-A	B, O	A, AB
AB	A, B	None	A, B, AB, O	None
O	None	Anti-A, anti-B	O	A, B, AB

Note: Group O is often called the universal donor, and group AB is called the universal recipient.

A transfusion of incompatible blood causes haemolysis (breakdown) of the RBCs and agglutination of erythrocytes. (*Agglutination* is the clumping of cells which results from their interaction with specific antibodies.) The ABO blood group names and compatibilities are listed in Table 10.4.

Before RBCs or whole blood can be administered, a series of procedures determine donor and recipient ABO types and Rh groups. These procedures—called a *type and crossmatch*—are performed by mixing the donor cells with the recipient's serum and watching for agglutination. If none occurs, the blood is considered compatible.

Despite meticulous procedures for matching blood types and antigens, blood transfusion reactions may still occur. The most common is a *febrile reaction*. Antibodies within the person receiving the blood are directed against the donor's white blood cells, causing fever and chills. Febrile reactions typically begin during the first 15 minutes of the transfusion. Using leucocyte-reduced blood avoids future febrile reactions.

Hypersensitivity reactions result when antibodies in the person's blood react against proteins, such as immunoglobulin A, in the donor blood. Hypersensitivity reactions may appear during or after the transfusion. The manifestations of hypersensitivity reaction include *urticaria* (the appearance of reddened wheals of various sizes on the skin) and itching.

Haemolytic reactions, the most dangerous transfusion reactions, usually result from an ABO incompatibility. Clumping RBCs block capillaries, decreasing blood flow to vital organs. In addition, macrophages engulf the clumped RBCs, releasing free haemoglobin into the circulating blood; the haemoglobin is then filtered by the kidneys and may block the renal tubules causing renal failure. Haemolytic reactions usually begin after infusion of 100 to 200 mL of the incompatible blood. Manifestations of a haemolytic reaction include flushing of the face, a burning sensation along the vein, headache, urticaria, chills, fever, lumbar pain, abdominal pain, chest pain, nausea and vomiting, tachycardia, hypotension and dyspnoea. If any of these manifestations appear, the blood transfusion must be immediately discontinued.

Other risks to people receiving blood include circulatory overload, electrolyte imbalances and infectious diseases such as hepatitis or cytomegalovirus.

People who have experienced trauma of any severity have had substantial blood loss and are usually in hypovolaemic shock. Blood replacement is the treatment of choice to restore oxygen-carrying capacity. People in severe shock with active bleeding are given universal type O red blood cells immediately. Those with less severe injuries or bleeding may be stabilised with other types of fluids until type-specific or crossmatched blood is available.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 7:
Blood and Blood Products

'The intention of this standard is to ensure that the patients who receive blood and blood products do so appropriately and safely.' (ACSQHC, 2012, p. 48)

Implementing this standard is achieved by the establishment of systems that ensure safe and appropriate prescription and administration of blood and blood products. These systems include processes facilitating accurate documentation, storage, transport, use and disposal. Effective communication regarding risks, benefits and use should exist across all individuals involved in a person's care (including the person themselves and their significant others).

Caring for individuals experiencing haematological conditions will often result in the need to administer blood or blood products in order to manage the person's condition. As with any biological material, various risks are involved in all facets of this treatment. Efficient and appropriate systems are imperative to ensure the safety of not only the person receiving the product, but also any other individual involved in their care.

Source: © Australian Commission on Safety and Quality in Health Care.

Some emergency departments and trauma centres use autotransfusion to provide blood for transfusions for the person with multiple injuries and/or severe shock. Autotransfusion is a method of blood administration in which special equipment collects and returns the person's own blood. The chest cavity is the typical source of blood to be autotransfused.

Nursing considerations for blood transfusion therapy are described in the following 'Medication administration' box.

MEDICATION ADMINISTRATION Blood transfusion

The risk of and seriousness of blood transfusion reactions require that extreme caution be taken when blood is administered. Most fatal transfusion reactions are the result of human error. Although general guidelines are provided here, each institution has specific policies and procedures that must be followed. Prior to beginning the transfusion, the nurse must determine that typed and crossmatched blood is available and collect the needed equipment: a blood administration set, a large-bore intravenous catheter (usually 18- or 19-gauge) and normal saline solution. Only normal saline is used with a blood transfusion as dextrose causes clumping of RBC and distilled water causes haemolysis.

NURSING RESPONSIBILITIES

- Obtain the person's consent.
- Assess for any previous reactions to blood.
- Explain the procedure to the person and answer any questions.
- Using aseptic non-touch technique, prepare the intravenous equipment. Prime the administration set with the saline.
- If venous access is not already in place, organise insertion of the intravenous needle (following body substance precautions) and begin administering the saline.
- Using institutional procedure, obtain the blood from the blood bank or laboratory. Administer the blood immediately; if this is not possible, return it to the blood bank or laboratory.
- Check and document that the donor and recipient blood have been tested and are compatible. This usually involves two nurses, each verifying that:
  a. An order for blood has been written.
  b. Type and crossmatch have been done.
  c. The name of the person and the name on the blood bag are identical.
  d. The number assigned to the unit of blood is identical to the one on the requisition for the blood.
  e. Blood type and Rh factor are compatible.
  f. The blood has not exceeded its expiration date.
  g. The unit of blood is intact and has no bubbles or discolouration.
- Identify the person by reading the armband identification and, if conscious, asking the person to tell you their name. Check the armband identification against the unit of blood labelling.
- Gently invert the blood bag several times to mix the plasma and RBCs.
- Take and record vital signs as a baseline.
- Attach the unit of blood to the administration set and begin the transfusion at a slow rate of about 2 mL per minute. (Some trauma victims may have blood infused at a rapid rate. If blood is infused rapidly, it may need to be warmed through an appropriate warming device during administration to prevent hypothermia.) Stay with the person for at least the first 15 minutes of the transfusion, monitoring for manifestations of a reaction and taking their vital signs.
- Continue to monitor the person during the transfusion, assessing for manifestations of hypersensitivity or haemolytic reactions, and taking and recording vital signs as directed by institutional policy.
- After the first 15 minutes the rate of infusion is increased. If there is no danger of fluid volume overload, most people can tolerate an infusion of a unit of blood (ranging from 250 to 500 mL depending on the blood component administered) in 2 hours. The unit of blood must be administered within 3 to 4 hours; after this time it has warmed and begins to deteriorate.
- Take the following actions if manifestations of a reaction occur:
  a. Stop the infusion of blood immediately and notify the physician. Continue to infuse the saline.
  b. Take vital signs and assess manifestations.
  c. Compare the blood slip with the unit of blood to ensure that an identification error was not made.
  d. Save the blood bag and any remaining blood for return to the laboratory for further tests to determine the cause of the reaction.
  e. Follow institutional policy for collecting urine and venous blood samples.
  f. Continue to monitor the person and provide prescribed interventions to treat hypersensitivity or haemolytic manifestations.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- In terms of viral safety, Australia has one of the safest blood supplies in the world. The possible risks of blood transfusions include transmission of infectious diseases and acquired immune deficiency syndrome (AIDS). However, with careful handling and storage of blood, bacterial contamination is rare. Although hepatitis may be transmitted by contaminated blood, new tests for hepatitis antibodies in donor blood are reducing this risk. Many people are afraid of contracting AIDS from blood even though donor screening and HIV-antibody testing of donor blood have virtually eliminated the transmission of HIV by blood transfusion.
- During the transfusion, immediately report any warm feelings, chills, itching, feelings of weakness or fainting, or difficulty breathing.
- Report any signs of a delayed transfusion reaction: chills, fever, cough, difficulty breathing, hives, itching or changes in circulation, and seek medical care immediately.
- Discuss any religious or cultural considerations related to blood transfusion.

Emergency surgery

Immediate surgical intervention is indicated when the person remains in shock despite resuscitation and there is no obvious external sign of blood loss. Abdominal and chest x-ray, ultrasound studies, diagnostic peritoneal lavage or CT scan may be performed to help identify the potential source of the blood loss. It is important for the emergency or trauma nurse to speak with the family as soon as possible and keep them informed about what is happening to their family member. Unfortunately, the need for emergency surgery may not allow time for family members or significant others to see their loved one before transfer to the operating room.

Organ donation

The *Australian Organ and Tissue Donation and Transplantation Authority Act 2008* was passed by the federal parliament in November 2008 and saw the establishment of the Australian Transplant Authority in January 2009. The focus of the Act was to implement the national reform package on organ and tissue donation by working with states and territories, clinicians, consumers and the community sector to build a world-leading organ and tissue donation and transplantation system for Australia. Under this Act, the Australian Organ Donor Registry (AODR) is the register of legal consent and is the only official national register for organ and tissue donation in Australia. The AODR ensures that consent (or objection) to donating organs and tissue for transplantation can be verified 24 hours a day, 7 days a week by authorised medical personnel, anywhere in Australia.

Consent for organ donation may be given not only by the donor but also by a spouse, adult children, parents, adult siblings, guardian or any adult authorised to do so. The Act also encourages people to carry donor cards. It is rare for a donation to proceed without the agreement of family or next-of-kin with their loved one's decision.

The increased success of organ transplant has made it a more common and valuable method of prolonging and improving life; however, many people are still waiting for organs and many people who may be suitable organ donors die each year from trauma. Organs and tissues that may be transplanted include bones, eyes, liver, lungs, skin, muscles and tendons, pancreas, intestines, kidneys, heart and heart valves.

The organ donation process begins with identification of the potential organ donor, which includes most people.

Certain factors are considered when determining if a person is an appropriate organ donor. They include:

- the circumstances of how, where and when a person dies
- any medical history
- age is considered, but it is more important to assess how the organs are working/functioning.

The family needs to be made aware of the person's prognosis and presented with the option of donating their organs. Both the family's and the person's feelings about organ donation must be explored. Even if the person carries an organ donation card, many institutions will not remove any organs without a signature from a family member or other authorised person. The nurse must always respect the family's concerns and feelings during this process. Some members of certain cultural groups may have religious constraints or issues of mistrust that may interfere with the donation process. Donation coordinators facilitate the process and are responsible for contacting family and discussing options.

Box 10.1 lists **brain death criteria**. Irreversible loss of all brain function is to be clinically confirmed. Along with the person being examined by two experienced medical practitioners, in some cases ancillary testing such as angiography or radionucleotide imaging will be used to provide additional evidence. Once brain death has been confirmed, the family must also understand the diagnosis and be allowed time to accept the person's death.

BOX 10.1 Brain death criteria

Clinical signs

- Irreversible condition
- Apnoea with a $PaCO_2$ greater than 60 mmHg
- No response to deep stimuli
- No spontaneous movement (some spinal cord reflexes may be present)
- No gag or corneal reflex
- No oculocephalic or oculovestibular reflex
- Absence of toxic or metabolic disorders

Confirmatory tests

- Cerebral blood flow study
- Electroencephalogram

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 5:
Patient Identification and Procedure Matching

'The intention of this standard is to correctly identify all patients whenever care is provided and correctly match patients to their intended treatment.' (ACSQHC, 2012, p. 40)

Implementing this standard is achieved by the establishment and maintenance of systems that use at least three approved patient identifiers to ensure that patients are correctly identified and crossmatched when providing care, therapy and/or services. Effective communication should exist across all individuals involved in a person's care (including the person themselves and their significant others). Clinicians and other members of the workforce use the patient identification and procedure matching systems.

The development of safety routines in patient identification and treatment matching ensures patient safety against any mistakes that may progress and cause harm, whether those components are diagnostic, therapeutic or supportive.

Source: © Australian Commission on Safety and Quality in Health Care.

When caring for an adult person who is an organ donor, the nurse ensures the following are maintained:

- systolic blood pressure of 90 mmHg to keep the person's organs perfused until removal
- urine output at >0.5 mL/kg/hr. This is usually accomplished by administering fluids and/or inotropic agents such as dopamine
- oxygen saturation at 90% or greater.

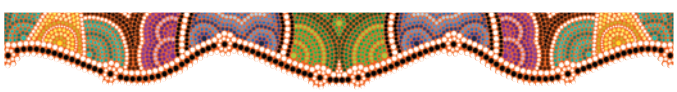
Forensic considerations

Injuries often happen under circumstances that require legal investigation. Many injuries, particularly penetrating trauma, may involve criminal activity. Therefore, the nurse must recognise the need to identify, store and properly transfer potential evidence for medico-legal investigations.

Each item of clothing removed from the person must be placed in a breathable container, such as a paper bag, and documented appropriately. Bullets or knives should be labelled, with their source specified and given to the proper authorities. Holes found in clothing should not be disturbed. When it is necessary to cut off clothing, these areas should be avoided and never cut through if at all possible.

The person's hands may yield important evidence, such as powder burns or residue on the skin, or tissue or hair samples beneath the fingernails. In the case of death, it is recommended that paper bags be placed over the person's hands if the presence of evidence is suspected; otherwise the evidence should be collected from nail clippings.

Identify all wounds and document these findings with pictures, diagrams or written descriptions. Once the evidence has been collected, identified and properly stored, ensure that it is given to the appropriate authorities. A chain of custody needs to be maintained throughout the entire process. All evidence must be identified and labelled, and documentation procedures must chronicle where and in whose possession the evidence has been. For the chain of custody to remain intact, the evidence must remain in the continuous possession of identified people and be marked and sealed in tamper-proof containers.



Nursing care

Nursing care of the person who has been injured begins with a primary assessment and the initiation of collaborative interventions for any life-threatening injuries. Nursing care is directed towards the person's specific responses to trauma.

Health promotion

Prevention efforts can reduce the incidence and severity of trauma. Areas of health promotion and trauma prevention interventions for individuals and communities include the following:

- *Motor vehicle safety*: seat belts, air bags, helmets, driving under the influence of alcohol or drugs, reckless driving, visual or cognitive deficits in the older adult, mobile phone use, driver fatigue.
- *Home safety*: electrical wiring, falls, burns, drowning, snow and ice removal.
- *Farm safety*: operating heavy equipment, safe storage of chemicals such as fertilisers.
- *Work safety*: operating work equipment, wearing safety equipment, removal of jewellery.
- *Relationships*: domestic violence, child abuse, older adult abuse or neglect.
- *Communities*: condition of streets, neighbourhood safety, gun control, gangs.

In providing information about trauma prevention to members of the community, the nurse serves as a healthcare educator, political activist and safety advocate.

Assessment

See 'Interprofessional care', above, for assessment of the person experiencing trauma.

Nursing diagnoses and interventions

The person experiencing trauma has many complex and interrelated actual or potential alterations in health. The nursing care in this section focuses on person and family problems with respirations, infection, immobility, spirituality and stress. Nursing interventions for decreased cardiac output and altered perfusion are discussed in the section of the chapter on nursing care of the person in shock.

Ineffective maintenance of airway

The person with multiple injuries is at great risk of developing airway obstruction and apnoea. Facial injuries, loose teeth, blood and vomitus increase the risk of aspiration and obstruction. Neurological injuries and cerebral oedema alter the person's respiratory drive and ability to keep the airway clear.

- Assess if airway is patent, maintainable or non-maintainable. Assess for manifestations of airway obstruction: stridor, tachypnoea, bradypnoea, cough, cyanosis, dyspnoea, decreased or absent breath sounds, changes in oxygen levels and changes in level of consciousness. *Assessing the airway and initiating interventions are the first steps in managing the person with multiple injuries.*
- Monitor oxygen saturation by applying a pulse oximeter. Adjust oxygen flow to maintain oxygen saturation above 95%. *Changes in oxygen saturation as measured by the pulse oximeter reflect the effectiveness of the person's ventilation. Pulse oximetry in people who have been exposed to carbon monoxide (i.e. house fires) is unreliable since it cannot differentiate carboxyhaemoglobin from oxyhaemoglobin.*
- Monitor level of consciousness. *An early sign of an ineffective airway is change in the person's behaviour. If the person becomes restless, anxious, combative or unresponsive, airway patency and ventilation effectiveness need to be immediately evaluated and appropriate interventions initiated.*

NURSING CARE PLAN A person with multiple injuries

Jane Souza is a 25-year-old married woman with two children who provides daycare for preschool children in her home. As she is driving on the highway at 100 km per hour, a car crosses the median strip and strikes her vehicle head on. Jane, who is not wearing a seat belt, is thrown forward against the steering wheel. The front of her car is pushed up against her by the car that struck her, entrapping her lower extremities.

After extensive efforts to extricate her from the car, Jane is transported to the local trauma centre. She is still conscious, is receiving high-flow oxygen by mask and has one intravenous line in place. Her vital signs are a palpable systolic blood pressure of 80, a pulse rate of 120 and a respiratory rate of 36. On arrival, she states that she is having difficulty breathing.

ASSESSMENT

- *Airway*: Maintainable with high-flow oxygen in place.
- *Breathing*: Respiratory rate of 36, decreased breath sounds on the right side, equal chest wall movement.
- *Circulation*: No palpable radial pulses; palpable brachial pulses. Cardiac monitor shows sinus tachycardia. No active external bleeding noted. Skin colour pale, cool to the touch and diaphoretic. One intravenous access in place and crystalloid fluids running.
- *Disability*: Moved her fingers when asked; complains of difficulty breathing; denies that she is hurt. Pupils 4 mm, equal and reacting to light. Extremity movement is limited due to broken limbs.
- *Exposure*: Has a broken right arm and an open fracture of the left ankle. Multiple bruising and abrasions on right side of chest.

Because of Jane's respiratory distress, she is intubated and ventilated with 100% oxygen. Another intravenous line is inserted and O-negative blood administered. It is determined that she has sustained a pneumothorax in the right side and an intercostal catheter is inserted.

DIAGNOSES

- *Ineffective breathing pattern* related to multiple bruises and abrasions on the right side of the chest and respiratory difficulty.
- *Deficient fluid volume* related to acute internal blood loss (presumed because no active bleeding can be found).
- *Risk of injury* related to trauma resuscitation.

PLANNING

- Continuously monitor airway and oxygenation.
- Continuously monitor circulatory status.
- Prepare for ongoing care.

Expected outcomes

- Maintain adequate oxygenation.
- Maintain adequate circulating blood volume.

IMPLEMENTATION

- Ensure patency of airway maintained—secure and monitor endotracheal tube.
- Assess and maintain ventilation—monitor ventilatory support, visualise chest expansion, assess air entry through lung auscultation, monitor oxygen saturations.
- Monitor the effects of fluid and blood administration, including any changes in blood pressure, heart rate and rhythm, skin colour and turgor.
- Insert urinary catheter and monitor urine output.
- Prepare for transfer to the operating room for emergency surgery.
- Explain all procedures.
- Keep family informed about her condition.

EVALUATION

- Jane is transferred to the operating room, where it is determined that she has a ruptured spleen and a serious pelvic fracture. Jane's treatment continues in the operating room.

CRITICAL THINKING IN THE NURSING PROCESS

1. Is the nursing diagnosis *Deficient fluid volume* appropriate for Jane Souza? Why or why not?
2. The assessment of a person who has experienced trauma is, in order: A = airway, B = breathing and C = circulation. What is the rationale for this sequence?
3. Following surgery, Jane is moved to the surgical intensive care unit. She is very anxious and restless. What assessments would you perform to identify the cause of her restlessness?
4. Infection is a common complication for a person experiencing trauma. Describe five risks for infection that are present from the time of injury to the time of hospital discharge.

REFLECTION ON THE NURSING PROCESS

1. What communication and education strategies would you use with Jane following surgery?

Risk of infection

Traumatic injuries are considered dirty wounds. Projectiles enter the body through dirty surfaces and clothing, carrying dirt and debris into the wound. Open fractures provide a portal for the entry of bacteria and dirt. Even with surgical intervention, the wounds often remain contaminated.

- Practise effective hand hygiene. *Handwashing remains the single most important factor in preventing the spread of infection.*
- Use standard precautions and aseptic technique when caring for wounds. *Standard precautions are essential to protect the person and the nurse from infection.*

In addition:

- Monitor wounds for odour, redness, heat, swelling and copious or purulent drainage.
- Monitor hidden wounds, such as those under casts, by asking the person whether pain has increased and by observing for increased drainage and heat over the area of the wound.
- Ensure that cross-contamination between wounds does not occur. Collect drainage in ostomy bags if it is copious. The skin is the first line of defence against infection; wounds provide a portal of entry for organisms. Risk factors for wound infection include contamination, inadequate wound care and the condition

of the wound at the time of closure. *Aseptic techniques used in applying and changing dressings reduce the entry of organisms.*

- Measure and record vital signs, including temperature every 2 to 4 hours. *Vital signs, particularly an elevated body temperature, may indicate the presence of an infection.*
- Provide adequate fluids and nutrition. *Adequate fluids, kilojoules and protein are essential to wound healing.*
- Assess for manifestations of gas gangrene: fever, pain and swelling in traumatised tissues; drainage with a foul odour. *Gas gangrene is usually caused by the organism* Clostridium perfringens. *This bacterium is found in the soil and can be introduced into the body during a traumatic injury. The organism grows in the tissues, causing necrosis; hydrogen and carbon dioxide are released with resultant swelling of tissues. If the infection continues, tissues are progressively destroyed and sepsis and death may result.*
- Assess status of tetanus immunisation and administer tetanus toxoid or human toxin–antitoxin as prescribed. *Tetanus is caused by an exotoxin produced by* Clostridium tetani*, usually introduced through an open wound. The organism is commonly found in the soil.*
- Use strict aseptic technique when inserting catheters, suctioning, administering parenteral medications or performing any other invasive procedure. *Using aseptic technique during invasive procedures reduces the risk of entry of organisms.*

Impaired physical mobility

The person with trauma injuries is often unable to change position independently and is at risk of complications of the integumentary, cardiovascular, gastrointestinal, respiratory, musculoskeletal and renal systems. At greatest risk are those who have had multiple injuries, spinal cord injuries, peripheral nerve injuries and traumatic amputations. Collaborate with the physiotherapist and occupational therapist (if available) to determine the most effective types and schedule of exercises and assistive devices.

The aim of therapy is to ensure the person will maintain joint range of motion and avoid development of contractures and pulmonary complications such as atelectasis.

- If active bleeding or oedema is not present, provide active or passive exercises to affected and unaffected extremities at least once every 8 hours. *Exercise improves muscle tone, maintains joint mobility, improves circulation and prevents contractures.*
- Assist the person to turn, cough and deep breathe, and use an incentive spirometer at least every 2 hours. *Changing positions, coughing, deep breathing and incentive spirometry reduce the risk of integumentary and respiratory complications.*
- If the person is unable to be moved and positioned, consider a specialty bed such as the kinetic continuous rotation bed (see Figure 10.7). *The kinetic continuous rotation bed allows continuous turning of the person; the motion decreases pulmonary complications, venous stasis, postural hypotension, urinary stasis, muscle wasting and bone demineralisation.*
- Monitor the lower extremities each day for manifestations of deep venous thrombosis: heat, swelling and pain. If anti-embolic stockings or intermittent compression stockings are used, remove them for 1 hour during each shift and assess the skin. *Venous stasis results when surrounding muscles are unable to contract and help move the blood through the veins. Thrombus (clot) formation in deep veins is a major risk of pulmonary embolism.*

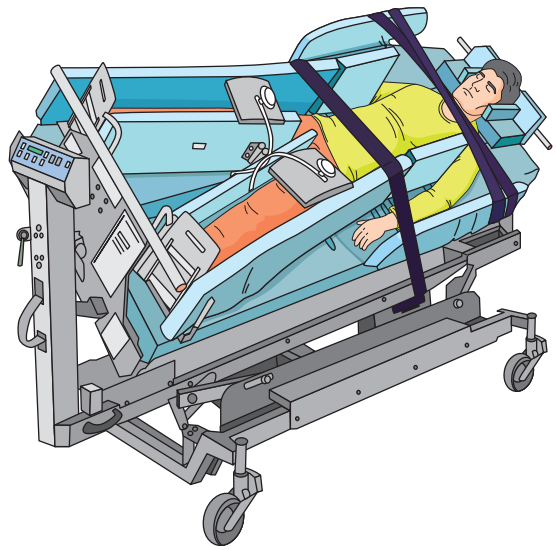


FIGURE 10.7 ■ A kinetic continuous rotation bed provides a means of turning the person with multiple injuries to decrease the hazards of immobility

Spiritual distress

Trauma generally strikes without warning and carries potentially devastating consequences, including severe alterations in the lives of the victim and family, and death. The traumatic death of a loved one may be the most difficult event a family ever experiences. The decision to cease life support systems or to donate organs challenges the family's belief systems and psychological stability. Nursing care of the family (or person) experiencing spiritual distress includes the following:

- Give the family information about the option to donate the person's organs. The decision to donate organs needs to be based on information about the person's condition, prognosis and the criteria by which brain death is determined. *It is important to convey to family members that organ donation is only an option and that they should not feel they are obligated to consent or are doing something wrong if they do not consent.*
- Encourage the family to ask questions and express their feelings about the traumatic event and/or organ donation. *Allowing families to express their feelings may help prevent long-term consequences such as guilt.*

- Refer the family for follow-up care. Long-term follow-up is important for the family facing the sudden death of a loved one. *Grieving is not an overnight process and providing the family with resources that may be used in the future may help prevent future crises and dysfunction.* (For more information see Chapter 4.)

Post-traumatic stress disorder

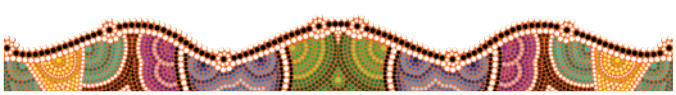
Post-traumatic stress disorder is an intense, sustained emotional response to a disastrous event. It is characterised by emotions that range from anger to fear and by flashbacks or psychic numbing. In the initial stage, the person may be calm or may express feelings of anger, disbelief, terror and shock. In the long-term phase, which begins anywhere from a few days to several months after the event, the person often experiences flashbacks and nightmares of the traumatic event. The person may call on ineffective coping mechanisms, such as alcohol or drugs, and withdraw from relationships.

- Assess emotional responses while providing physical care. Observe for excessive crying, sleep problems, suspiciousness and fear during the initial phase of treatment. If the person is unconscious, encourage family members and friends to express their feelings. *These assessments provide valuable information about the person's ability to cope with the trauma.*
- Be available if the person wishes to talk about the trauma and encourage expression of feelings. *The person may initially deny negative feelings; this denial is a coping mechanism in the initial phase of recovery.*
- Teach relaxation techniques such as deep breathing, progressive muscle relaxation or imagery (see Chapter 8). *These techniques are often useful in coping when thoughts of the trauma recur.*
- Refer the person and family members for counselling, psychotherapy or support groups as appropriate. *Continued therapy may be necessary in assisting the person and family to resolve the acute and long-term effects of trauma.*

Community-based care

Address the following topics to prepare the person and family for home care:

- the type of home environment to which the person will be returning, including any changes that will be required to let them function in that environment
- medications, dressings, wound care, equipment and supplies
- special diet, if needed
- rehabilitation plan and its effect on the person's family
- follow-up appointments with the general practitioner or at the trauma clinic
- emotional changes that the person may undergo as a result of the trauma
- helpful resources
  - home healthcare
  - community support groups
  - National Stroke Foundation.



THE PERSON EXPERIENCING SHOCK

Shock is a clinical syndrome characterised by a systemic imbalance between oxygen supply and demand. This imbalance results in a state of inadequate blood flow to body organs and tissues, causing life-threatening cellular dysfunction.

Overview of cellular homeostasis and haemodynamics

To maintain cellular metabolism, cells of all body organs and tissues require a regular and consistent supply of oxygen and the removal of metabolic wastes. This homeostatic regulation is maintained primarily by the cardiovascular system and depends on four physiological components:

1. A cardiac output sufficient to meet bodily requirements.
2. An uncompromised vascular system in which the vessels have a diameter sufficient to allow unimpeded blood flow and have good tone (the ability to constrict or dilate to maintain normal pressure).
3. A volume of blood sufficient to fill the circulatory system and a blood pressure adequate to maintain blood flow.
4. Tissues that are able to extract and use the oxygen delivered through the capillaries.

In a healthy person, these components function as a system to maintain tissue perfusion. During shock, however, one or more of these components are disrupted. An understanding of basic haemodynamics is necessary to understand the pathophysiology of shock:

- **Stroke volume (SV)** is the amount of blood pumped into the aorta with each contraction of the left ventricle.
- **Cardiac output (CO)** is the amount of blood pumped per minute into the aorta by the left ventricle. CO is determined by multiplying the stroke volume (SV) by the heart rate (HR): CO = SV × HR.

CONSIDERATION FOR PRACTICE

Cardiac output (CO) = stroke volume (SV) × heart rate (HR).

- **Mean arterial pressure (MAP)** is the product of cardiac output and systemic vascular resistance (SVR): MAP = CO × SVR. When CO, SVR or total blood volume rises, MAP and tissue perfusion increase. Conversely, when CO, SVR or total blood volume falls, MAP and tissue perfusion decrease. A MAP of 70 to 110 is normal. A MAP of 60 mmHg is required to maintain adequate perfusion to the brain, heart and kidneys.
- The sympathetic nervous system maintains the smooth muscle surrounding the arteries and arterioles in a state of partial contraction called *sympathetic tone*. Increased sympathetic stimulation increases vasoconstriction and SVR; decreased sympathetic stimulation allows vasodilation which decreases SVR.

Pathophysiology

When one or more cardiovascular components do not function properly, the body's haemodynamic properties are altered. Consequently, tissue perfusion may be inadequate to sustain normal

cellular metabolism. The result is the clinical syndrome known as shock. The manifestations of shock result from the body's attempts to maintain vital organs (heart and brain) and to preserve life following a drop in cellular perfusion. However, if the injury or condition triggering shock is severe enough or of long enough duration, cellular hypoxia and cellular death will occur.

Shock is triggered by a sustained drop in mean arterial pressure. This drop can occur after a decrease in cardiac output, a decrease in the circulating blood volume or an increase in the size of the vascular bed due to peripheral vasodilation. If intervention is timely and effective, the physiological events that characterise shock may be stopped; if not, shock may lead to death. See Table 10.5 for classification of haemorrhagic shock.

Stage I: Early, reversible and compensatory shock

The initial stage of shock begins when baroreceptors in the aortic arch and the carotid sinus detect a sustained drop in MAP of less than 10 mmHg from normal levels. The circulating blood volume may decrease (usually to less than 500 mL) but not enough to cause serious effects.

The body reacts to the decrease in arterial pressure. The cerebral integration centre initiates the body's response systems causing the sympathetic nervous system to increase the heart rate and the force of cardiac contraction, thus increasing cardiac output. Sympathetic stimulation also causes peripheral vasoconstriction, resulting in increased systemic vascular resistance and a rise in arterial pressure. The net result is that the perfusion of cells, tissues and organs is maintained.

Symptoms are almost imperceptible during the early stage of shock. The pulse rate may be slightly elevated. If the injury is minor or of short duration, arterial pressure is usually maintained and no further symptoms occur.

Compensatory shock begins after the MAP falls 10 to 15 mmHg below normal levels. The circulating blood volume is reduced by 25% to 35% (1000 mL or more), but compensatory mechanisms are able to maintain blood pressure and tissue perfusion to vital organs thereby preventing cell damage.

- Stimulation of the sympathetic nervous system results in the release of adrenaline from the adrenal medulla and the release of noradrenaline from the adrenal medulla and the sympathetic fibres. Both hormones rapidly stimulate the alpha- and beta-adrenergic fibres. Stimulated alpha-adrenergic fibres cause vasoconstriction in the blood vessels supplying the skin and most of the abdominal viscera. Perfusion of these areas decreases. Stimulated beta-adrenergic fibres cause vasodilation in vessels supplying the heart and skeletal muscles (beta-1 response) and increase the heart rate and force of cardiac contraction ($beta_2$ response). Further, blood vessels in the respiratory system dilate and the respiratory rate increases ($beta_2$ response). Thus, stimulation of the sympathetic nervous system results in increased cardiac output and oxygenation of these tissues.
- The renin–angiotensin response occurs as the blood flow to the kidneys decreases. Renin released from the kidneys converts a plasma protein to angiotensin II, which causes vasoconstriction and stimulates the adrenal cortex to release aldosterone. Aldosterone causes the kidneys to reabsorb water and sodium and to lose potassium. The absorption of water maintains circulating blood volume, while increased vasoconstriction increases SVR, maintaining central vascular volume and raising blood pressure.
- The hypothalamus releases adrenocorticotropic hormone causing the adrenal glands to secrete aldosterone. Aldosterone promotes the reabsorption of water and sodium by the kidneys, preserving blood volume and pressure.
- The posterior pituitary gland releases antidiuretic hormone, which increases renal reabsorption of water to increase intravascular volume. The combined effects of hormones released by the hypothalamus and posterior pituitary glands work to conserve central vascular volume.
- As MAP falls in the compensatory stage of shock, decreased capillary hydrostatic pressure causes a fluid shift from the interstitial space into the capillaries. The net gain of fluid raises the blood volume.

Working together, these compensatory mechanisms can maintain MAP for only a short period of time. During this period, the perfusion and oxygenation of the heart and brain are adequate. If effective treatment is provided, the process is arrested and no permanent damage occurs. However, unless the underlying cause of shock is reversed, these compensatory mechanisms soon become harmful and shock perpetuates shock.

TABLE 10.5 Classification of haemorrhagic shock and presentation of the person

	COMPENSATED/ CLASS I	MILD/CLASS II	MODERATE/ CLASS III	SEVERE/CLASS IV
Blood loss	Up to 750 mL	750–1500 mL	1500–2000 mL	>2000 mL
Percentage of blood volume loss	Up to 15%	15–30%	30–40%	>40%
Heart rate (bpm)	<100	>100	>120	>140
Blood pressure	Normal or increased	Normal	Decreased	Markedly decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Capillary refill	Normal	Mild increase	Usually delayed	Delayed
Respiratory rate	Normal	Mild increase	Moderate tachypnoea	Marked tachypnoea
Urine output	>0.5 mL/kg/hr	>0.3 mL/kg/hr	<0.3 mL/kg/hr	Anuria
Mental status	Normal–slightly anxious	Mildly anxious–agitated	Anxious–confused	Lethargic–obtunded

Stage II: Intermediate or progressive shock

The progressive stage of shock occurs after a sustained decrease in MAP of 20 mmHg or more below normal levels and a fluid loss of 35% to 50% (1800 to 2500 mL of fluid). Although the compensatory mechanisms in the previous state remain activated, they are no longer able to maintain MAP at a level sufficient to ensure perfusion of vital organs.

The vasoconstriction response that first helped sustain MAP eventually limits blood flow to the point that cells become oxygen deficient. To remain alive, the affected cells switch from aerobic to anaerobic metabolism. The lactic acid formed as a by-product of anaerobic metabolism contributes to an acidotic state at the cellular level. As a result, adenosine triphosphate, the source of cellular energy, is produced inefficiently. Lacking energy, the sodium–potassium pump fails. Potassium moves out of the cell while sodium and water move inward. As this process continues the cell swells, cell membrane integrity is lost and cell organelles are damaged. Lysosomes within the cell spill out their digestive enzymes which disintegrate any remaining organelles. Some enzymes spread to adjacent cells where they erode and rupture cell membranes.

The acid by-products of anaerobic metabolism dilate the precapillary arterioles and constrict the postcapillary venules. This causes increased hydrostatic pressure within the capillary and fluid shifts back into the interstitial space. The capillaries also become increasingly permeable, allowing serum proteins to shift from the vascular space into the interstitium. The build-up of plasma proteins increases the osmotic pressure in the interstitium, further accelerating the fluid shift out of the capillaries.

Throughout this period, the heart rate and vasoconstriction increase; however, perfusion of the skin, skeletal muscles, kidneys and gastrointestinal organs is greatly diminished. Cells in the heart and brain become hypoxic, while other body cells and tissues become ischaemic and anoxic. A generalised state of acidosis and hyperkalaemia ensues (see Chapter 9). Unless this stage of shock is treated rapidly, the person's chances of survival are poor.

Stage III: Refractory or irreversible shock

If shock progresses to the irreversible stage, tissue anoxia becomes so generalised and cellular death so widespread that no treatment can reverse the damage. Even if MAP is temporarily restored, too much cellular damage has occurred to maintain life. Death of cells is followed by death of tissues, which results in death of organs. Death of vital organs contributes to subsequent death of the body.

Effects of shock on body systems

Whatever its causes, shock produces predictable effects on the body's organ systems. (See 'Multisystem effects of shock' on the following page.)

CARDIOVASCULAR SYSTEM The perfusion and oxygenation of the heart are adequate in the early stages of shock. As shock progresses, myocardial cells become hypoxic and myocardial muscle function diminishes. Initially, the blood pressure may be normal or even slightly elevated (as a result of compensatory mechanisms) and the heart rate only slightly increased. Sympathetic stimulation increases the heart rate (a sinus tachycardia of 120 beats per minute is common) in an effort to increase cardiac output. As a result of vasoconstriction and decreased blood volume, the palpated pulse is rapid, weak and thready; as shock progresses, peripheral pulses are usually non-palpable.

Tachycardia reduces the time available for left ventricular filling and coronary artery perfusion, further reducing cardiac output. With progressive shock, altered acid–base balance, hypoxia and hyperkalaemia damage the heart's electrical systems and contractility. Consequently, cardiac arrhythmias may develop. Decreased blood volume with decreased venous return also decreases cardiac output, and blood pressure falls.

The blood pressure changes produced by shock are characterised by a progressive decrease in both systolic and diastolic pressures and a narrowing pulse pressure. Auscultation of blood pressure is often difficult or impossible and is an inaccurate reflection of blood pressure status. For this reason, haemodynamic monitoring is usually instituted to follow the person's cardiovascular status accurately.

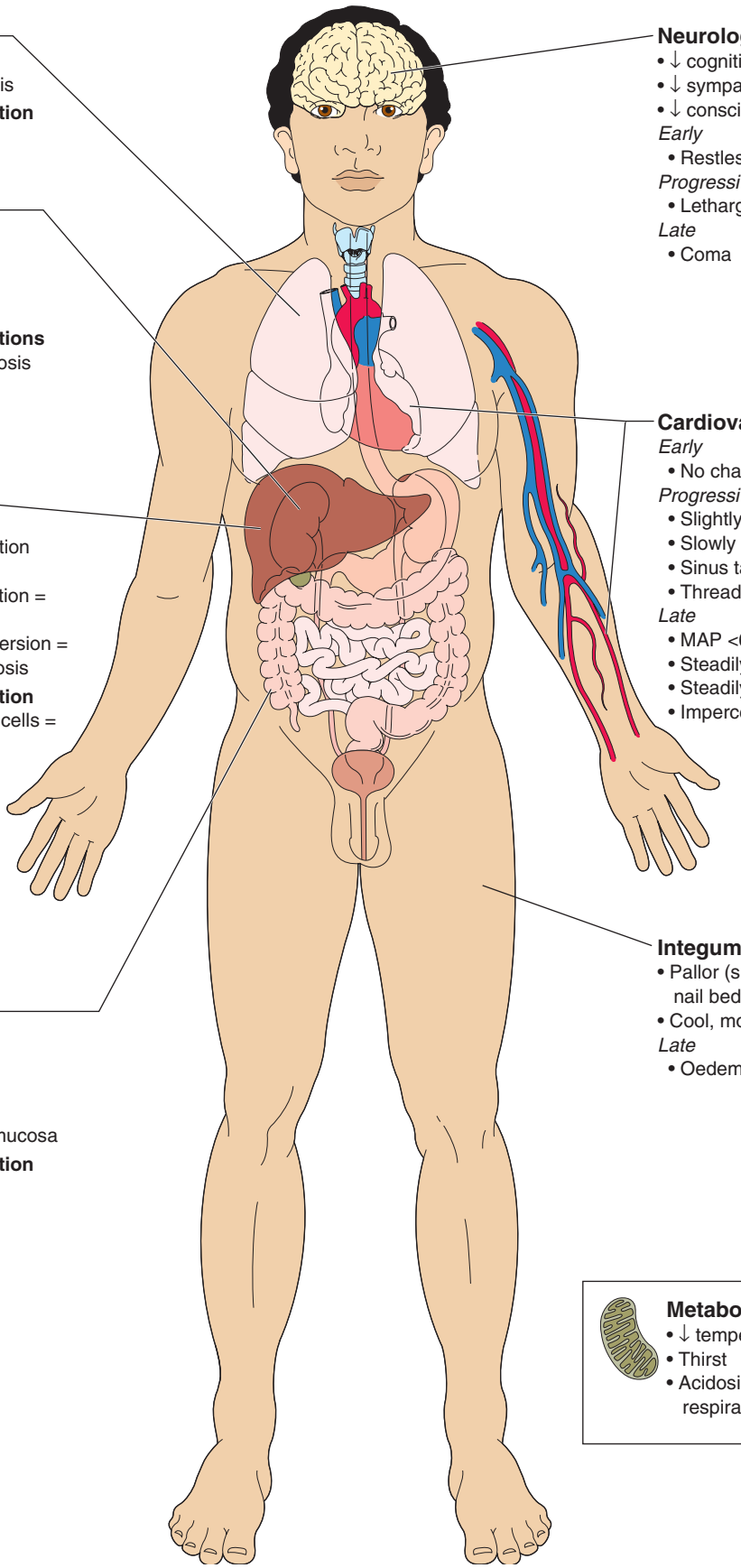
RESPIRATORY SYSTEM During shock, oxygen delivery to cells may be impaired by a drop in circulating blood volume or, in the case of blood loss, by an insufficient number of red blood cells that carry oxygen. Although the respiratory rate increases because of compensatory mechanisms that promote oxygenation, the number of alveoli that are perfused decreases and gas exchange is impaired. As a result, oxygen levels in the blood decrease and carbon dioxide levels increase. As perfusion of the lungs diminishes, carbon dioxide is retained and respiratory acidosis occurs.

A complication of decreased perfusion of the lungs is acute respiratory distress syndrome (ARDS), or 'shock lung'. The exact mechanism that produces ARDS is unknown, but some contributing factors have been identified. The pulmonary capillaries become increasingly permeable to proteins and water, resulting in non-cardiogenic pulmonary oedema. Production of surfactant (which controls surface tension within alveoli) is impaired and the alveoli collapse or fill with fluid. This potentially lethal form of respiratory failure may result from any condition that causes hypoperfusion of the lungs, but is more common in shock caused by haemorrhage, severe allergic responses, trauma and infection. (ARDS is discussed further in Chapter 36.)

GASTROINTESTINAL AND HEPATIC SYSTEMS The gastrointestinal organs normally receive 25% of the cardiac output through the splenic circulation. Shock constricts the splenic arterioles and redirects arterial blood flow to the heart and brain. Consequently, gastrointestinal organs become ischaemic and may be irreversibly damaged.

Gastric mucosa tends to ulcerate when it becomes ischaemic. Lesions of the gastric and duodenal mucosa (called *stress ulcers*) can develop within hours of severe trauma, sepsis or burns (Porth & Matfin, 2009). Gastrointestinal ulcers may haemorrhage within 2 to 10 days following the original cause of shock. In addition, the permeability of damaged mucosa increases, allowing enteric bacteria or their toxins to enter the abdominal cavity and then progress to the circulation, resulting in sepsis.

MULTISYSTEM EFFECTS OF SHOCK



Respiratory
- ↑ respiratory rate
- Respiratory acidosis

Potential complication
- ARDS

Urinary
- ↓ renal perfusion
- ↓ GFR

Late
- Oliguria

Potential complications
- Acute tubular necrosis
- Kidney failure

Hepatic

Early
- ↑ glucose production

Progressive
- ↓ glucose production = hypoglycaemia
- ↓ lactic acid conversion = metabolic acidosis

Potential complication
- Destroyed Kupffer cells = systemic bacterial infections

Gastrointestinal

Early
- ↓ GI motility

Late
- Paralytic ileus
- Ulceration of GI mucosa

Potential complication
- Bowel necrosis

Neurological
- ↓ cognition
- ↓ sympathetic activity
- ↓ consciousness

Early
- Restlessness, apathy

Progressive
- Lethargy

Late
- Coma

Cardiovascular

Early
- No change

Progressive
- Slightly ↑ BP
- Slowly ↑ HR
- Sinus tachycardia
- Thready pulse

Late
- MAP <60 mmHg
- Steadily ↓ BP
- Steadily ↓ CO
- Imperceptible pulses

Integumentary
- Pallor (skin, lips, oral mucosa, nail beds, conjunctiva)
- Cool, moist skin

Late
- Oedema

Metabolic processes
- ↓ temperature
- Thirst
- Acidosis (metabolic and respiratory)

Gastric and intestinal motility is impaired during shock and paralytic ileus may result. If the episode of shock is prolonged, necrosis of the bowel may occur. In many cases, alterations in the structure and function of the gastrointestinal tract impair absorption of nutrients such as protein and glucose.

Shock also alters the metabolic functions of the liver. Initially, *gluconeogenesis* (the process of forming glucose from non-carbohydrate sources) and *glycogenolysis* (the breakdown of glycogen into glucose) increase. This process allows blood glucose levels to increase as the body attempts to respond to the stressor; however, as shock progresses, liver functions are impaired and hypoglycaemia develops. Metabolism of fats and protein is impaired and the liver can no longer effectively remove lactic acid, contributing to the development of metabolic acidosis.

The destruction of the liver's reticuloendothelial Kupffer cells (phagocytes that destroy bacteria) causes a further problem. Bacteria may proliferate within the circulatory system, causing overwhelming bacterial infection and toxicity.

NEUROLOGICAL SYSTEM The primary effects of shock on the neurological system involve changes in mental status and orientation. Cerebral hypoxia produces altered levels of consciousness beginning with apathy and lethargy and progressing to coma. A common early symptom of cerebral hypoxia is restlessness. Continued ischaemia of brain cells eventually causes swelling, resulting in cerebral oedema, neurotransmitter failure and irreversible brain cell damage.

As cerebral ischaemia worsens, the sympathetic activity and vasomotor centres are depressed. This leads to a loss of sympathetic tone, causing systemic vasodilation and pooling of blood in the periphery. As a result, venous return and cardiac output further decrease.

CONSIDERATION FOR PRACTICE

An early sign of shock is a change in the level of consciousness. Late signs of shock include mental status changes, hypotension and marked tachycardia.

RENAL SYSTEM Blood that normally perfuses the kidneys is shunted to the heart and brain during the progressive stage of shock, resulting in renal hypoperfusion. The drop in renal perfusion is reflected in a corresponding decrease in the glomerular filtration rate. Urine output is reduced and the urine that is produced is highly concentrated. Oliguria of <0.5 mL/kg/hr indicates progressive shock.

Healthy kidneys can tolerate a drop in perfusion for only about 20 minutes; thereafter, acute tubular necrosis develops (Porth & Matfin, 2009). As tubular necrosis occurs, epithelial cells slough off and block the tubules, disrupting nephron function. The accumulating loss of functional nephrons eventually causes renal failure. Without normal renal function, metabolic waste products are retained in the plasma.

If treatment restores renal perfusion, the kidneys can regenerate the lost epithelial cells in the tubules and renal function usually returns to normal. However, in a person who is older or chronically ill or in sustained shock, loss of renal function may become permanent.

EFFECTS ON SKIN, TEMPERATURE AND THIRST In most types of shock, blood vessels supplying the skin are vasoconstricted and the sweat glands are activated. As a result, changes in skin colour occur. The skin of Caucasian people becomes pale. In people with darker skin (such as those of Indigenous Australian, African or Mediterranean descent), shock-related skin colour changes may be assessed as paleness of the lips, oral mucous membranes, nail beds and conjunctiva. The skin is usually cool and moist and, in the later stages of shock, often oedematous.

The body temperature decreases as shock progresses, the result of a decrease in overall body metabolism. Some people in shock become thirsty, probably a response to decreased blood volume and increased serum osmolality (Huether & McCance, 2013).

Types of shock

Shock is identified according to its underlying cause. All types of shock progress through the same stages and exert similar effects on body systems. Any differences are noted in the following discussion.

Hypovolaemic shock

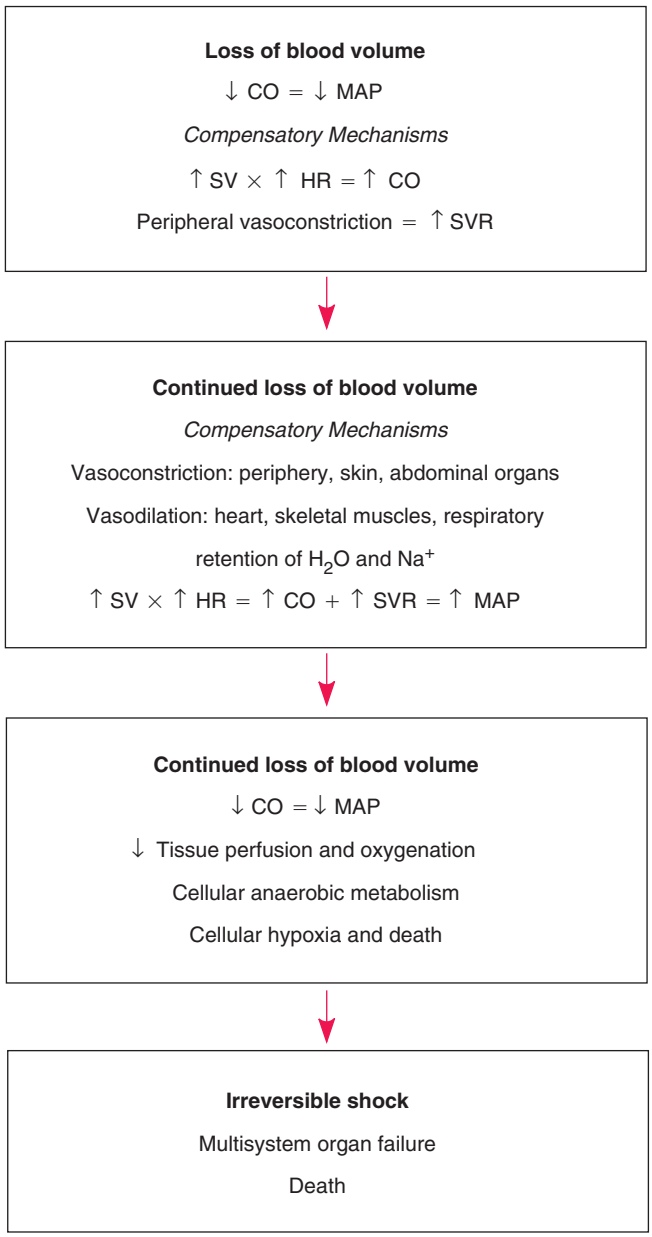
Hypovolaemic shock is caused by a decrease in intravascular volume of 15% or more (Huether & McCance, 2013). In hypovolaemic shock, the venous blood returning to the heart decreases and ventricular filling drops. As a result, stroke volume, cardiac output and blood pressure decrease. Hypovolaemic shock is the most common type of shock and often occurs simultaneously with other types.

The decrease in circulating blood volume that triggers hypovolaemic shock may result from:

- loss of blood volume from haemorrhage (from surgery, trauma, gastrointestinal bleeding, blood coagulation disorders, ruptured oesophageal varices)
- loss of intravascular fluid from the skin due to injuries such as burns (see Chapter 16)
- loss of intravascular volume from severe dehydration
- loss of body fluid from the gastrointestinal system due to persistent and severe vomiting or diarrhoea, or continuous nasogastric suctioning
- renal losses of fluid due to the use of diuretics or to endocrine disorders such as diabetes insipidus
- conditions causing fluid shifts from the intravascular compartment to the interstitial space
- third spacing due to such disorders as liver diseases with ascites, pleural effusion or intestinal obstruction.

Hypovolaemic shock affects all body systems. Its effects vary depending on the person's age, general state of health, extent of injury or severity of illness, length of time before treatment is provided and the rate of volume loss.

The manifestations of hypovolaemic shock result directly from the decrease in circulating blood volume and the initiation of compensatory mechanisms (see Figure 10.8). The loss of circulating blood volume reduces cardiac output by decreasing



Key CO: Cardiac output
HR: Heart rate
MAP: Mean arterial pressure
SV: Stroke volume
SVR: Systemic vascular resistance

FIGURE 10.8 ■ The stages of hypovolaemic shock

venous return to the heart. As a result, blood pressure drops. The carotid and cardiac baroreceptors sense the decrease in blood pressure and communicate it to the vasomotor centres in the brainstem. The vasomotor centres then induce the sympathetic compensatory responses. If the fluid loss is less than 500 mL, activation of the sympathetic response is generally adequate to restore cardiac output and blood pressure to near normal, although the heart rate may remain elevated.

With a sustained loss of blood volume (1000 mL or more), the shock stage progresses. Heart rate and vasoconstriction increase and blood flow to the skin, skeletal muscles, kidneys and abdominal organs decreases. Several renal mechanisms and a decline in capillary pressure help conserve blood volume. Eventually, the amount of blood flowing to cells is too low to oxygenate them and sustain production of cellular energy. Anaerobic metabolism begins, producing an acidotic environment for cells. As a result, cells lose their physical integrity. If untreated, shock causes multiple organ failure and death results. Manifestations of various stages of hypovolaemic shock are listed in the box below.

MANIFESTATIONS Hypovolaemic shock

INITIAL STAGE
- *Blood pressure*: normal to slightly decreased
- *Pulse*: slightly increased from baseline
- *Respirations*: normal (baseline)
- *Skin*: cool, pale (in periphery), moist
- *Mental status*: alert and oriented
- *Urine output*: slight decrease
- *Other*: thirst, decreased capillary refill time

COMPENSATORY AND PROGRESSIVE STAGES
- *Blood pressure*: hypotension
- *Pulse*: rapid, thready
- *Respirations*: increased
- *Skin*: cool, pale (includes trunk); poor turgor with fluid loss, oedematous with fluid shift
- *Mental status*: restless, anxious, confused or agitated
- *Urine output*: oliguria (less than 0.5 mL/kg/hr)
- *Other*: marked thirst, acidosis, hyperkalaemia, decreased capillary refill time, decreased or absent peripheral pulses

IRREVERSIBLE STAGE
- *Blood pressure*: severe hypotension (often systolic pressure is below 80 mmHg)
- *Pulse*: very rapid, weak
- *Respirations*: rapid, shallow; crackles and wheezes
- *Skin*: cool, pale, mottled with cyanosis
- *Mental status*: disoriented, lethargic, comatose
- *Urine output*: anuria
- *Other*: loss of reflexes, decreased or absent peripheral pulses

Cardiogenic shock

Cardiogenic shock occurs when the heart's pumping ability is compromised to the point that it cannot maintain cardiac output and adequate tissue perfusion. Cardiac disorders are discussed in Chapters 29 and 30; this section focuses only on the effects of shock caused by these disorders.

The loss of the pumping action of the heart may be caused by the following conditions:
- myocardial infarction
- cardiac tamponade
- restrictive pericarditis
- cardiac arrest
- arrhythmias, such as fibrillation or ventricular tachycardia
- pathological changes in the valves

- cardiomyopathies from hypertension, alcohol, bacterial or viral infections, or ischaemia
- complications of cardiac surgery
- electrolyte imbalances (especially changes in normal potassium and calcium levels)
- drugs affecting cardiac muscle contractility
- head injuries causing damage to the cardioregulatory centre.

Myocardial infarction is the most common cause of cardiogenic shock. People admitted to the hospital for treatment of myocardial infarction or cardiac surgery are at risk of cardiogenic shock. The severity and progression of shock are related to the amount of myocardial damage.

Whatever the cardiogenic cause, the decrease in cardiac output causes a decrease in MAP. Heart rate may increase in response to compensatory mechanisms. However, tachycardia increases myocardial oxygen consumption and decreases coronary perfusion. The myocardium becomes progressively depleted of oxygen, causing further myocardial ischaemia and necrosis. The typical sequence of shock is essentially unchanged in cardiogenic shock.

Cyanosis, however, is more common in cardiogenic shock because stagnating blood increases extraction of oxygen from the haemoglobin at the capillary beds. As a result, the skin, lips and nail beds may appear cyanotic. As cardiac failure and cardiogenic shock progress, left ventricular end-diastolic pressure increases. The increase is transmitted to the pulmonary capillary bed and pulmonary oedema may occur. Retention of blood in the right side of the heart increases right atrial pressure which leads to jugular venous distension as a result of backflow through the vena cava. Manifestations of cardiogenic shock are listed in the following box.

MANIFESTATIONS Cardiogenic shock

- *Blood pressure*: hypotension
- *Pulse*: rapid, thready; distension of veins of hands and neck
- *Respirations*: increased, laboured; crackles and wheezes; pulmonary oedema
- *Skin*: pale, cyanotic, cold, moist
- *Mental status*: restless, anxious, lethargic progressing to comatose
- *Urine output*: oliguria to anuria
- *Other*: dependent oedema; elevated central venous pressure (CVP); elevated pulmonary capillary wedge pressure; arrhythmias

Obstructive shock

Obstructive shock is caused by an obstruction in the heart or great vessels that either impedes venous return or prevents effective cardiac pumping action. The causes of obstructive shock are impaired diastolic filling (e.g. pericardial tamponade or pneumothorax), increased right ventricular afterload (e.g. pulmonary emboli) and increased left ventricular afterload (e.g. aortic stenosis, abdominal distension). The manifestations are the result of decreased cardiac output and blood pressure, with reduced tissue perfusion and cellular metabolism.

NURSING CARE OF THE OLDER ADULT

Experiencing hypovolaemia

With ageing comes a relative decrease in sympathetic activity in relation to the cardiovascular system. Cardiac compliance also decreases with age. Atherosclerosis affects many vital organs' sensitivity to even the slightest reduction in blood flow. Many older adults experience secondary volume depletion due to chronic diuretic use or malnutrition. Also, people prescribed beta-blockers may not present with tachycardia as an early indicator of shock. This important sign can be masked due to beta-adrenergic blockade. This group of people will require early invasive monitoring in order to avoid excessive or inadequate volume restoration. This should be considered early in the treatment phase.

Distributive shock

Distributive shock (also called **vasogenic shock**) includes several types of shock that result from widespread vasodilation and decreased peripheral resistance. Because the blood volume does not change, relative hypovolaemia results. Examples of distributive shock include septic, neurogenic and anaphylactic shock. Treatment is based on the underlying pathogenesis.

Septic shock

Septic shock, the leading cause of death for people in critical care units, is one part of a progressive syndrome called *systemic inflammatory response syndrome* (SIRS). This condition is most often the result of gram-negative bacterial infections (i.e. *Pseudomonas*, *E. coli*, *Klebsiella*) but may also follow gram-positive infections from *Staphylococcus* and *Streptococcus* bacteria. Gram-negative sepsis has greatly increased in the past 10 years with a 60% mortality rate despite treatment. The pathophysiology of septic shock is complex and not completely understood.

People at risk of developing infections leading to septic shock include those who are hospitalised, have debilitating chronic illnesses or have poor nutritional status. The risk is heightened after invasive procedures or surgery. Others at risk of septic shock include older adults and those who are immunocompromised. Portals of entry for infection that may lead to septic shock are as follows:

- *Urinary system*: catheterisations, suprapubic tubes, cystoscopy.
- *Respiratory system*: suctioning, aspiration, tracheostomy, endotracheal tubes, respiratory therapy, mechanical ventilators.
- *Gastrointestinal system*: peptic ulcers, ruptured appendix, peritonitis.
- *Integumentary system*: surgical wounds, intravenous catheters, intra-arterial catheters, invasive monitoring, decubitus ulcers, burns, trauma.
- *Female reproductive system*: elective surgical abortion, ascending infections from transmission of bacteria during the intrapartal and postpartal periods, tampon use, sexually transmitted infections.

Septic shock begins with *septicaemia* (the presence of pathogens and their toxins in the blood). As pathogens are destroyed, their ruptured cell membranes allow endotoxins to leak into the plasma. The endotoxins disrupt the vascular system, coagulation mechanism and immune system, and trigger an immune and inflammatory response. (See Chapter 11 for more information.) For this reason, the initial effects of septic shock differ from those of hypovolaemic and cardiogenic shock: cardiac output is high and systemic vascular resistance is low.

Endotoxins directly damage the endothelial lining of small blood vessels first; the small blood vessels of the kidneys and lungs are most susceptible. Cellular damage stimulates the release of vasoactive proteins and activates coagulation factor XII. The vasoactive proteins stimulate peripheral vasodilation and increase capillary permeability; the activation of coagulation factors results in the production of multiple intravascular blood clots.

As a result of the increased capillary permeability and vasodilation, fluid shifts from the intravascular space to the interstitial space. Hypovolaemia results as fluid volume is lost from the circulating blood. Hypovolaemia and intravascular coagulation alter oxygenation and cellular metabolism, leading to anaerobic metabolism, lactic acidosis and cellular death.

Septic shock has an early phase and a late phase. In early septic shock (sometimes called the *warm phase*), vasodilation results in weakness and warm, flushed skin, and the septicaemia often causes high fever and chills. In late septic shock (sometimes called the *cold phase*), hypovolaemia and activity of the compensatory mechanisms result in typical shock manifestations including cold, moist skin, oliguria and changes in mental status. Death may result from respiratory failure, cardiac failure or renal failure.

Toxic shock syndrome is an especially virulent form of septic shock occurring most frequently in menstruating women who use tampons. It is thought that bacterial toxins diffuse from the site of infection in the vagina into the circulation. The toxins then trigger a widespread inflammatory response and septic shock. The manifestations of toxic shock syndrome include extreme hypotension, hyperpyrexia, headache, myalgia, confusion, skin rash, vomiting and diarrhoea (Huether & McCance, 2013).

Disseminated intravascular coagulation (DIC), a generalised response to injury, is a potential risk in septic shock. This condition is characterised by simultaneous bleeding and clotting throughout the vasculature. Sepsis injures blood cells, causing platelet aggregation and decreased blood flow. As a result, blood clots form throughout the microcirculation. The clotting slows circulation further while stimulating excess fibrinolysis. As the body's stores of clotting factors are depleted, generalised bleeding begins. DIC is further discussed in Chapter 32.

MANIFESTATIONS Septic shock

EARLY (WARM) SEPTIC SHOCK
- *Blood pressure*: normal to hypotension
- *Pulse*: increased, thready
- *Respirations*: rapid and deep
- *Skin*: warm, flushed
- *Mental status*: alert, oriented, anxious
- *Urine output*: normal
- *Other*: increased body temperature; chills; weakness; nausea, vomiting, diarrhoea; decreased CVP

LATE (COLD) SEPTIC SHOCK
- *Blood pressure*: hypotension
- *Pulse*: tachycardia, arrhythmias
- *Respirations*: rapid, shallow, dyspnoeic
- *Skin*: cool, pale, oedematous
- *Mental status*: lethargic to comatose
- *Urine output*: oliguria to anuria
- *Other*: normal to decreased body temperature; decreased CVP

Neurogenic shock

Neurogenic shock is the result of an imbalance between parasympathetic and sympathetic stimulation of vascular smooth muscle. If parasympathetic overstimulation or sympathetic understimulation persists, sustained vasodilation occurs and blood pools in the venous and capillary beds.

Neurogenic shock causes dramatic reduction in systemic vascular resistance as the size of the vascular compartment increases. As SVR decreases, pressure in the blood vessels becomes too low to drive nutrients across capillary membranes and cellular metabolism is impaired.

The following conditions can cause neurogenic shock by increasing parasympathetic stimulation or inhibiting sympathetic stimulation of the smooth muscle of blood vessels:
- head injury
- trauma to the spinal cord (spinal shock, a form of neurogenic shock, is described in Chapter 42)
- insulin reactions (which cause hypoglycaemia, decreasing glucose to the medulla)
- central nervous system depressant drugs (such as sedatives, barbiturates or narcotics)
- anaesthesia (spinal and general)
- severe pain
- prolonged exposure to heat.

Bradycardia occurs early, but tachycardia begins as compensatory mechanisms are initiated. Central venous pressure drops as veins dilate, venous return to the heart decreases, stroke volume decreases and MAP falls. In early stages, the extremities are warm and pink (from the pooling of blood), but as shock progresses the skin becomes pale and cool. Manifestations of neurogenic shock are listed in the box on the next page.

Anaphylactic shock

Anaphylactic shock is the result of a widespread hypersensitivity reaction (called *anaphylaxis*). The pathophysiology in this type of shock includes vasodilation, pooling of blood in the periphery and hypovolaemia with altered cellular metabolism. These physiological alterations occur when a sensitised person has contact with an *allergen* (a foreign substance to which an individual is hypersensitive). Many different allergens can cause anaphylactic shock, including medications, blood administration, latex, foods, snake venom and insect stings.

Anaphylactic shock does not occur with the first exposure to an allergen. With the first exposure to a foreign substance

MANIFESTATIONS Neurogenic shock

- *Blood pressure*: hypotension
- *Pulse*: slow and bounding
- *Respirations*: vary
- *Skin*: warm, dry
- *Mental status*: anxious, restless, lethargic progressing to comatose
- *Urine output*: oliguria to anuria
- *Other*: lowered body temperature

(the *antigen*), the body produces specific immunoglobulin E (IgE) antibodies against this antigen. The person is thus sensitised to that specific antigen. With subsequent exposure, the antigen reacts with the already formed IgE antibodies, disrupting cellular integrity. In addition, large amounts of histamine and other vasoactive amines are released and distributed through the circulatory system. These substances cause increased capillary permeability and massive vasodilation resulting in profound hypotension and eventual vascular collapse.

Histamine also causes constriction of smooth muscles in the bladder, uterus, intestines and bronchioles. Respiratory distress, bronchospasm, laryngospasm and severe abdominal cramping result. Serotonin (a neurotransmitter with vasoconstrictive properties) is released, further affecting respiratory status by increasing capillary permeability in the lungs. As a result, plasma leaks into the alveoli, gas exchange is impaired and pulmonary oedema may occur.

Anaphylactic shock begins and progresses rapidly. Manifestations may begin within 20 minutes of contact with an antigen. Unless appropriate intervention is provided, death can occur within a matter of minutes. Because anaphylaxis is rapid and potentially lethal, people with known allergies should carry some form of warning (such as a MedicAlert® bracelet) informing others of their susceptibility. Some patients carry an Epipen (adrenaline) to use if required. Healthcare providers should be extremely careful to assess and document allergies or previous drug reactions. Manifestations of anaphylactic shock are listed in the box below.

Similar, but not related, are anaphylactoid reactions that are not humorally mediated and do not require prior exposure to a trigger. These can have similar symptoms and are treated in a similar manner.

MANIFESTATIONS Anaphylactic shock

- *Blood pressure*: hypotension
- *Pulse*: increased, arrhythmias
- *Respirations*: dyspnoea, stridor, wheezes, laryngospasm, bronchospasm, pulmonary oedema
- *Skin*: warm, oedematous (lips, eyelids, tongue, hands, feet, genitals)
- *Mental status*: restless, anxious, lethargic to comatose
- *Urine output*: oliguria to anuria
- *Other*: paraesthesias; pruritus; abdominal cramps, vomiting, diarrhoea

INTERPROFESSIONAL CARE

Medical care for the person in shock focuses on treating the underlying cause, increasing arterial oxygenation and improving tissue perfusion. Depending on the cause and type of shock, interventions include emergency care measures, oxygen therapy, fluid replacement and medications. Emergency care is often the first course of collaborative action taken to arrest shock, as discussed earlier in this chapter.

Diagnosis

The following diagnostic tests can help identify the type of shock and assess the person's physical status. Measurements include:

- *Blood haemoglobin* and *haematocrit* to detect the cell concentration that usually occurs in hypovolaemic shock which reflects the underlying aetiology. In hypovolaemic shock resulting from haemorrhage, the haemoglobin and haematocrit concentrations are lower than normal. In hypovolaemic shock resulting from intravascular fluid loss, by contrast, the haemoglobin and haematocrit concentrations are higher than normal.
- *Arterial blood gases (ABGs)* to determine oxygen and carbon dioxide levels and pH. The effects of shock and of the body's compensatory mechanisms cause a decrease in pH (indicating acidosis), a decrease in the partial pressure of oxygen (PaO_2) and in total oxygen saturation, and an increase in the partial pressure of carbon dioxide ($PaCO_2$).
- *Serum electrolytes* to monitor the severity and progression of shock. As shock progresses, glucose and sodium levels decrease, and potassium levels increase.
- *Blood urea nitrogen (BUN)*, *serum creatinine levels*, *urine specific gravity* and *osmolality* to check renal function. As perfusion of the kidneys is decreased and renal function is reduced, the BUN and creatinine levels increase as does urine specific gravity and osmolality.
- *Blood cultures* to identify the causative organism in septic shock.
- *White blood cell (WBC) count* and *differential* in the person with septic or anaphylactic shock. The total WBC count is increased in septic shock. Elevated neutrophils indicate acute infection, increased monocytes indicate a bacterial infection and increased eosinophils indicate an allergic response.
- *Serum cardiac enzymes*, which are elevated in cardiogenic shock: lactate dehydrogenase (LDH), creatine kinase (CK), creatinine kinase MB (CKMB) and troponin levels.

Other diagnostic tests may be ordered to determine the extent of injury or damage, or to locate the site of internal haemorrhage. These tests might include x-ray studies, computed tomography (CT) scans, magnetic resonance imaging (MRI), endoscopic examinations and echocardiograms. Newer diagnostic methods for hypoperfusion include gastric tonometry and sublingual $PaCO_2$. Gastric tonometry measures the partial pressure of carbon dioxide in the gastric lumen. The measurement of sublingual carbon dioxide correlates well with decreased MAP.

Medications

When fluid replacement alone is not sufficient to reverse shock, vasoactive drugs (drugs causing vasoconstriction or vasodilation) and inotropic drugs (drugs improving cardiac contractility) may be administered. When used to treat shock, these drugs increase venous return through vasoconstriction of peripheral vessels; they also improve the pumping ability of the heart by facilitating myocardial contractility and by dilating coronary arteries to increase perfusion of the myocardium.

Drugs used to treat shock are discussed in the following 'Medication administration' box. Other drugs that may be administered to the person in shock include:

- diuretics to increase urine output after fluid replacement has been initiated
- sodium bicarbonate to treat acidosis

MEDICATION ADMINISTRATION The person in shock

ADRENERGICS (SYMPATHOMIMETICS)

Vasoconstrictors
- **Adrenaline**
- **Noradrenaline**
- **Metaraminol (Aramine)**

Inotropes
- **Dopamine**
- **Dobutamine**
- **Isoprenaline**

Adrenergic drugs (also called sympathomimetics) mimic the fight-or-flight response of the sympathetic nervous system, selectively stimulating alpha-adrenergic and beta-adrenergic receptors. Many of these drugs have both vasopressor (vasoconstricting) effects and positive inotropic effects. Stimulation of alpha-adrenergic receptors results in vasoconstriction and increased systemic blood pressure. Stimulation of beta-adrenergic receptors increases the force and rate of myocardial contraction.

The physiological effect of these drugs includes improved perfusion and oxygenation of the heart, with increased stroke volume and heart rate and increased cardiac output. Increased cardiac output in turn increases tissue perfusion and oxygenation. The main disadvantage is that increases in stroke volume and heart rate also increase the oxygen requirements of the myocardium. These drugs may be used in the early stages of shock, especially in types of shock characterised by vasodilation.

Nursing responsibilities

- Carefully monitor responses in the older adult, who may be especially sensitive to sympathomimetics and require lower doses.
- Document lung sounds, vital signs and haemodynamic parameters before starting the medication and then according to institutional policy (usually every 5 to 15 minutes).
- When administering these drugs by the subcutaneous route, carefully aspirate the injection site to avoid injecting the drug directly into a blood vessel.
- Use the intravenous route only with continuous infusion pumps. Carefully adjust the dose to accommodate the person's cardiovascular status (as ordered by the physician or by written protocol).
- Record and monitor urine output. Report output of less than 0.5 mL/kg/hr.
- Be aware that the sympathomimetics are incompatible with sodium bicarbonate or alkaline solutions.
- When administering drugs that cause vasoconstriction, such as noradrenaline and metaraminol, monitor the intravenous insertion site for infiltration. If infiltration does occur, stop the infusion and notify the physician immediately as infiltration may cause ischaemia and necrosis of tissue.

Health education for the person and family

- Because these drugs mimic a physiological reaction to stress, they may cause feelings of anxiety.
- Close monitoring to adjust the dose will be carried out by qualified nurses using written protocols.
- Report heart palpitations or chest pain immediately.

VASODILATORS

Nitroglycerin (Glycerol trinitrate)
Nitroprusside (Nipride)

Drugs that cause vasodilation act directly on smooth muscle affecting both arterioles and veins. Peripheral resistance, cardiac output and pulmonary wedge pressure are all reduced as a result of the vasodilation. These effects decrease the oxygen need of the heart and decrease pulmonary congestion. Vasodilators are used primarily in the treatment of cardiogenic shock and may be combined with a sympathomimetic (e.g. dopamine).

Nursing responsibilities

- Protect these drugs from light by wrapping the intravenous bag in the package that is provided.
- Mix with 5% dextrose only.
- Infuse via an infusion pump and use within 4 hours of reconstitution.
- Do not add other medications to the solution.
- Assess mental status, blood pressure and pulse prior to initiating medication. Thereafter, assess blood pressure and pulse according to institutional policy (usually every 5 minutes initially, then every 15 minutes until stable and then hourly).
- Monitor for confusion, dizziness, tachycardia, arrhythmias, hypotension and adventitious breath sounds. Report these immediately if they occur and slow infusion to a keep-open rate.
- With nitroprusside infusions, monitor for signs of thiocyanate poisoning (nausea, disorientation, muscle spasms, decreased or absent reflexes) if infusion lasts longer than 72 hours.
- Keep the person in bed with side rails up.

Health education for the person and family

- It is important for the person to stay in bed and change positions slowly to avoid dizziness.
- The blood pressure and pulse are taken frequently to adjust the dose of medication.
- Headache is a common side effect.

- calcium to replace calcium lost as a result of blood transfusions
- antiarrhythmic agents to stabilise heart rhythm
- broad-spectrum antibiotics to suppress organisms responsible for septic shock
- adrenaline, antihistamines, corticosteroids and inhaled beta$_2$ agonists to treat anaphylactic shock
- morphine to dilate veins and decrease anxiety.

Oxygen therapy

Establishing and maintaining a patent airway and ensuring adequate oxygenation are critical interventions in reversing shock. All people in shock (even those with adequate respirations) should receive oxygen therapy (usually by mask or nasal cannula) to maintain the PaO_2 at greater than 80 mmHg during the first 4 to 6 hours of care. If the person's unassisted respiration cannot maintain PaO_2 at this level, ventilatory assistance may be necessary. Care of the person requiring ventilatory assistance is discussed in Chapter 36.

Fluid resuscitation

The most effective treatment for the person in hypovolaemic shock is the administration of intravenous fluids or blood. Fluids are also appropriate in the management of septic and neurogenic shock. However, depending on pulmonary artery pressure, the person with cardiogenic shock may require either fluid replacement or restriction.

Various fluids may be administered alone or in combination as part of fluid replacement therapy in treating shock. Fluid replacements are administered through two large-bore cannulae via peripheral intravenous access or through a central line. Current fluid resuscitation guidelines include rapid crystalloid infusion followed by blood transfusion. Whole blood or blood products increase the oxygen-carrying capacity of the blood and thus increase oxygenation of cells. Fluid replacements, such as crystalloid and colloid solutions, increase circulating blood volume and tissue perfusion.

CRYSTALLOID SOLUTIONS Crystalloid solutions contain dextrose or electrolytes dissolved in water; those used for management of shock are either isotonic or hypotonic. Isotonic solutions include normal saline (0.9%) and compound sodium lactate (Hartmann's solution). Hypotonic solutions include one-half normal saline (0.45%) and 5% dextrose in water (D_5W).

All crystalloid solutions increase fluid volume in both the intravascular and the interstitial space. Of the total amount infused, only about 25% remains in the intravascular system; the remaining 75% moves into the interstitial space. Consequently, fluid volume is only minimally expanded and the potential for peripheral oedema is increased when crystalloid solutions are used. However, compound sodium lactate (an electrolyte solution) and 0.9% saline are the fluids of choice in treating hypovolaemic shock, especially in the emergency phase of care while blood is being typed and crossmatched. Large amounts of these solutions may be infused rapidly, increasing blood volume and tissue perfusion.

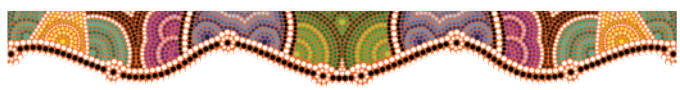
COLLOID SOLUTIONS Colloid solutions contain substances (colloids) that do not diffuse through capillary walls. Hence, colloids tend to remain in the vascular system and increase the osmotic pressure of the serum. This causes fluid to move into the vascular compartment from the interstitial space, resulting in the plasma volume expanding. Colloid solutions used to treat shock include 5% albumin, 25% albumin, Gelofusine, Haemaccel, plasma protein fraction and Dextran.

Colloid products reduce platelet adhesiveness and have been associated with reductions in blood coagulation. Consequently, the person's prothrombin time (PT), International Normalised Ratio (INR), platelet count and activated partial thromboplastin time (APTT) should be monitored when these solutions are administered. Normal values are as follows:

PT	10–15 seconds
INR	1–1.2 seconds
Platelets	150–400 × 10^9/L
APTT	<35 seconds

See the 'Medication administration' box on the next page for further information about colloid solutions and associated nursing responsibilities and teaching.

BLOOD AND BLOOD PRODUCTS If hypovolaemic shock is due to haemorrhage, the infusion of blood and blood products may be indicated. Available blood and blood products include fresh whole blood, stored whole blood, packed RBCs, platelet concentrate, fresh-frozen plasma and cryoprecipitate. Often, packed RBCs are given to provide haemoglobin concentration and are supplemented with crystalloids to maintain an adequate circulatory volume. (See discussion of blood administration earlier in the chapter.)



Nursing care

Nursing assessments and interventions to prevent shock are an essential part of the nursing care of every person. The primary nursing interventions to prevent shock are assessment and monitoring.

Health promotion and assessment

Nursing assessments are critical in preventing shock. Identifying people at risk and making focused assessments are essential. Although shock may occur at any age, physiological changes with ageing make the older adult a high-risk population. (See the 'Nursing care of the older adult' box on the next page).

- *Hypovolaemic shock*: people who have undergone surgery, have sustained multiple traumatic injuries or have been seriously burned are most likely to develop hypovolaemic shock. Monitoring fluid status is essential in preventing shock and includes daily assessments of weight, fluid intake by all routes, measurable fluid loss (e.g. urine, vomitus, wound drainage, gastric drainage and chest tube drainage) and fluid loss that must be estimated, such as fluid lost via

MEDICATION ADMINISTRATION Colloid solutions

COLLOID SOLUTIONS (PLASMA EXPANDERS)

Albumin 4% (Albumex 4)
Albumin 20% (Albumex 20)
Gelofusine (GelofuG)
Haemaccel
Stable plasma protein solution (SPPS)

These solutions are blood volume expanders and are used to treat hypovolaemic shock due to surgery, haemorrhage, burns or other trauma. Albumin and plasma protein solutions are prepared from healthy blood donors. Gelofusine and Haemaccel are synthetically prepared large molecules. The solutions promote circulatory volume and tissue perfusion by rapidly expanding plasma volume.

Nursing responsibilities

- Before infusion begins, establish baseline of vital signs, lung sounds, heart sounds and (if possible) CVP and pulmonary artery wedge pressure.
- Start administration of ordered intravenous fluids, using a large-gauge (18- or 19-gauge) infusion needle.
- Take and record vital signs as required by institutional policy (usually every 15 to 60 minutes) and assess status.
- Take and record intake and output every 1 to 2 hours.
- Monitor for manifestations of congestive heart failure or pulmonary oedema (dyspnoea, cyanosis, cough, crackles, wheezes). If these manifestations appear, stop the fluids and notify the physician immediately.
- Monitor for bleeding from new sites; an increase in blood pressure may cause bleeding in severed vessels that did not bleed with decreased blood pressure.
- Monitor for manifestations of dehydration (dry lips; scant, dark-coloured urine; loss of skin turgor). Increased intravenous fluids are usually ordered if the person becomes dehydrated.
- Monitor for manifestations of circulatory overload (jugular vein distension, increase in CVP, increase in pulmonary artery wedge pressure). If these manifestations occur, slow rate of infusion and notify physician.
- Monitor prothrombin time, partial thromboplastin time and platelet counts.
- If administering albumin or plasma protein solution, have adrenaline and antihistamines readily available for any manifestations of a hypersensitivity reaction (fever, chills, rash, headache, wheezing, flushing).
- Maintain the person on bed rest with side rails elevated.

Health education for the person and family

- The solutions are given to replace lost serum protein, which helps maintain the volume of blood.
- The vital signs are taken frequently to ensure the safety of the person.

profuse perspiration and wound drainage. Assessments for the critically ill person are ongoing and include fluid balance, haemodynamic values and vital signs.

- *Cardiogenic shock*: people with left anterior wall myocardial infarctions are at risk of developing cardiogenic shock. Nursing care to prevent the development of cardiogenic shock focuses on maintaining or improving myocardial oxygen supply by providing immediate pain relief, maintaining rest and administering supplemental oxygen.
- *Neurogenic shock*: the risk of neurogenic shock is increased in people who have spinal cord injuries and those who have received spinal anaesthesia. Preventive nursing care includes maintaining immobility of people with spinal cord trauma and elevating the head of the bed 15 to 20 degrees following spinal anaesthesia. Elevations of more than 20 degrees, however, can potentiate headaches following spinal anaesthesia and should be avoided.
- *Anaphylactic shock*: prevent anaphylactic shock by collecting information about allergies and drug reactions during the health history. Note these allergies clearly on all documents and place a special armband on the person. Careful and frequent assessments during blood administration may prevent serious reactions to blood or blood products.
- *Septic shock*: people who are hospitalised, are debilitated, are chronically ill or have undergone invasive procedures or tube insertions are at high risk of septic shock. Nursing care to prevent septic shock includes careful and consistent handwashing, the use of aseptic techniques for procedures (e.g. catheterisations, suctioning, changing dressings, starting and maintaining intravenous fluids or medications) and monitoring for local and systemic manifestations (e.g. WBC and differential counts) of infection.

NURSING CARE OF THE OLDER ADULT Variations in assessment findings—shock

- Cardiac changes may include a thickened left ventricular wall, decreased elasticity of the myocardium and more rigid valves. These changes result in a decreased stroke volume and cardiac output, thus decreasing responses to shock in general and increasing the risk of cardiogenic shock.
- Decreased arterial wall elasticity and vasomotor tone reduce the older adult's ability to respond to a decrease in oxygenation.
- Decreased elasticity and turgor of the skin make assessments of skin turgor more difficult.
- Previous medication and blood administration increase the risk of anaphylactic shock.
- Decreased immune system response increases the risk of septic shock.

Nursing diagnoses and interventions

Nursing care for the person in shock focuses on assessing and monitoring overall tissue perfusion and on meeting psychosocial needs of the person and the family. This section discusses nursing diagnoses that are appropriate for the person with hypovolaemic shock. See the accompanying 'Nursing care of the older adult'.

Decreased cardiac output

Decreased cardiac output is the primary problem for the person in shock. Although much of the care related to this diagnosis is collaborative, many independent nursing interventions are critical to the care of the person in shock.

- Assess and monitor cardiovascular function via the following:
  - blood pressure
  - heart rate and rhythm
  - pulse oximetry
  - peripheral pulses
  - haemodynamic monitoring of arterial pressures, pulmonary artery pressures and central venous pressures (CVPs). *A baseline assessment is necessary to establish the stage of shock. If palpable peripheral pulses and audible (to auscultation) blood pressure are lost, inserting central arterial, venous and pulmonary artery catheters is essential to establish progression of shock accurately and to evaluate the person's response to therapy.*
- Measure and record intake and output (total output and urinary output) hourly. *A decrease in circulating blood volume with hypotension and the effect of the compensatory mechanisms associated with shock can cause renal failure. Urinary output of <0.5 mL/kg/hr in an acutely ill adult indicates reduced renal blood flow.*
- Monitor bowel sounds, abdominal distension and abdominal pain. *Decreased splenic blood flow reduces bowel motility and peristalsis; paralytic ileus may result.*
- Monitor for sudden sharp chest pain, dyspnoea, cyanosis, anxiety and restlessness. *Haemoconcentration and increased platelet aggregation may result in pulmonary emboli.*
- Maintain bed rest and provide (to the extent possible) a calm, quiet environment. Place in a supine position with the legs elevated to about 20 degrees, trunk flat, and head and shoulders elevated higher than the chest (see Figure 10.9). *Limiting activity and ensuring rest decreases the workload of the heart. The supine position with legs elevated increases venous return; however, this position should not be used for people in cardiogenic shock. The Trendelenburg position is no longer recommended because it causes the abdominal organs to press against the diaphragm (limiting respirations), decreases filling of the coronary arteries and initiates aortic and carotid sinus reflexes.*

Ineffective tissue perfusion

As shock progresses, diminished tissue perfusion causes ischaemia and hypoxia of major organ systems. As shock worsens, blood flow and oxygenation of the lungs, heart and brain

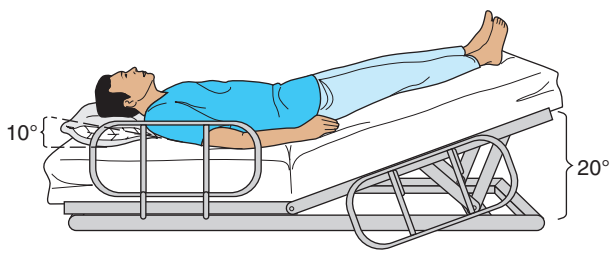
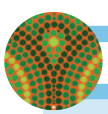


FIGURE 10.9 ■ The person in shock should be positioned with the lower extremities elevated approximately 20 degrees (knees straight), trunk horizontal and the head elevated about 10 degrees



TRANSLATION TO PRACTICE Evidence-based practice for care of ICU people sustaining multiple trauma

Ventilator-associated pneumonia (VAP) is an important person safety issue in critically injured people. The Association for Professionals in Infection Control and Epidemiology (APIC) published an evidence-based clinical guideline for the prevention and elimination of VAP in 2009 (APIC, 2009). The Association recommends the use of a facility-wide VAP infection prevention and control program and overall infection prevention and antimicrobial stewardship. The guideline describes the critical topics of problem identification, surveillance and how it is defined, risk assessment, and the development and use of a surveillance plan and prevention strategies. The guideline also provides examples of documents and reminders that can be used to provide systematic surveillance and management strategies.

IMPLICATIONS FOR NURSING

The APIC evidence-based guidelines recommend elements related to VAP prevention:

- prevention of complications in ventilated patients focusing on positioning specific to reduce VAP
- educational programs for healthcare personnel focusing on VAP prevention
- use of quality improvement projects with VAP prevention focus
- organisational issues related to leadership, staffing and informatics which can impact on patient outcomes
- setting a target of zero VAP cases
- key prevention strategies related to unit and institutional processes, supportive early innovators, consistent communication, connecting actions to outcomes and review of deviation from practice standards.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Considering the information from this guideline, how would you communicate the recommendations to the medical staff for the person with mechanical ventilation?
2. What is the rationale behind the preceding recommendations?

NURSING CARE PLAN A person with septic shock

Huang Mei Lan is a 43-year-old unmarried female who lives alone in a major city. Ms Huang came to Australia 15 years ago from China and now speaks English well. Her family still lives in China. She worked in a neighbourhood sewing shop until 3 years ago when she was diagnosed with breast cancer. Her treatment included mastectomy of the affected breast and follow-up chemotherapy.

Last month, Ms Huang experienced a recurrence of cancer in the lymph glands of the affected side. Surgery to remove the glands was performed and chemotherapy started. Ms Huang has a central line, a urinary catheter and a surgical incision. She is underweight, weak and depressed. Although she has multiple physical problems, she never complains or asks for any kind of medication.

ASSESSMENT

Ms Huang's nurse, Robert O'Brien, enters her room early in the morning to make an initial assessment. He finds Ms Huang huddled in the middle of the bed, shivering violently. Her vital signs are T 40°C, P 110, R 30 and BP 106/66. Her skin is hot, dry and flushed, with poor turgor. She is alert and oriented but is restless and appears anxious. Ms Huang states she is nauseated and suddenly begins vomiting and is incontinent of liquid stool. Laboratory data indicate leucocytosis, respiratory alkalosis and reduced platelet count. Blood cultures, as well as cultures of Ms Huang's sputum, urine and wound drainage, are conducted. She is diagnosed as having septic shock.

Gelofusine is ordered per intravenous line and intravenous broad-spectrum antibiotics are begun until the organism and its portal of entry can be determined. Despite treatment Ms Huang's condition worsens. Her blood pressure continues to drop, her skin becomes cool and cyanotic, and she begins to have periods of disorientation. She is transferred to the critical care unit. As she is being prepared for the transfer, she begins to cry and asks, 'Am I going to die?'.

DIAGNOSES

- *Ineffective breathing pattern* related to rapid respirations and progression of septic shock.
- *Ineffective tissue perfusion* related to progression of septic shock with decreased cardiac output, hypotension and massive vasodilation.
- *Deficient fluid volume* related to vomiting, diarrhoea, high fever and shift of intravascular volume to interstitial spaces.
- *Anxiety* related to feelings that illness is worsening and is potentially life threatening, and to the transfer to the critical care unit.

PLANNING

- Monitor respiratory status, including respiratory rate, rhythm and breath sounds.
- Monitor neurological status, including mental status and level of consciousness.
- Monitor cardiovascular status, including arterial blood pressure; rate, rhythm and quality of pulses; central venous pressure; pulmonary artery pressure and cardiac output.
- Monitor colour and character of skin.
- Monitor body temperature every 2 hours.
- Monitor results of ABGs, blood counts, clotting times and platelet counts.
- Monitor urinary output hourly, reporting any output of <0.5 mL/kg/hr.

Expected outcomes

- Maintain adequate circulating blood volume.
- Regain and maintain blood gas parameters within normal limits.
- Regain and maintain stable haemodynamic levels.
- Verbalise increased ability to cope with stressors.

IMPLEMENTATION

- Perform hourly neurological assessment, including mental status and level of consciousness.
- Implement continuous cardiac monitoring, including arterial blood pressure; rate, rhythm and quality of pulses; central venous pressure; pulmonary artery pressure and cardiac output.
- Regularly assess colour and character of skin.
- Take samples of blood for ABGs, blood counts, clotting times and platelet counts and report results.
- Assess respiratory status, including respiratory rate, rhythm and breath sounds.
- Assess body temperature every 2 hours.
- Measure urinary output hourly and report output of <0.5 mL/kg/hr.
- Explain procedures and provide comfort measures (oral care, skin care, turning, positioning).
- Report abnormal findings to medical team.

EVALUATION

Despite intensive nursing and medical care, Ms Huang's condition remains critical. The interventions are continued.

CRITICAL THINKING IN THE NURSING PROCESS

1. Vasoconstrictors may be used in the treatment of septic shock. Explain the rationale for their use.
2. While monitoring Ms Huang's ABGs, the nurse notes that her PaO_2 is < 60 mmHg and her $PaCO_2$ is > 50 mmHg. What do these findings indicate and why have they occurred?
3. Ms Huang has been given large amounts of colloids intravenously. Haemodynamic monitoring indicates a higher than normal CVP and pulmonary artery pressure. What do these findings indicate? What physical assessments would you make to confirm the changes?

REFLECTION ON THE NURSING PROCESS

1. Outline the component of Ms Huang's condition and physical status that posed potential risks for her developing septic shock.
2. What education strategies could have been used to assist Ms Huang to identify and communicate issues prior to her deterioration?

are also impaired. Hypoxia and ischaemia result from decreased tissue perfusion in the kidneys, brain, heart, lungs, gastrointestinal tract and the periphery.

- Monitor skin colour, temperature, turgor and moisture. *Decreased tissue perfusion is evidenced by the skin becoming pale, cool and moist; as haemoglobin concentrations decrease, cyanosis occurs.*
- Monitor cardiopulmonary function by assessing/monitoring the following:
  - blood pressure (by auscultation or by haemodynamic monitoring)
  - rate and depth of respirations
  - lung sounds
  - pulse oximetry
  - peripheral pulses (brachial, radial, dorsalis pedis and posterior tibial); include presence, equality, rate, rhythm and quality. (If unable to palpate pulses, use a device such as a Doppler ultrasound to assess peripheral arterial blood flow.)
  - jugular vein distension
  - central venous pressure measurements.
    Baseline vital signs are necessary to determine trends in subsequent findings. As shock progresses, the blood pressure decreases and the pulse becomes rapid, weak and thready. As perfusion of the lungs decreases, crackles, wheezes and dyspnoea are commonly assessed. Capillary refill is prolonged and peripheral pulses are weak or non-palpable. Neck veins that cannot be seen when the person is in the supine position indicate decreased intravascular volume. CVP is an accurate means of determining fluid status in the person in shock; the findings will be low (5 to 15 cm of water is normal) in hypovolaemic shock because of the decreased blood volume. (See Chapter 9 for a discussion of CVP.)
- Monitor body temperature. *An elevated body temperature increases metabolic demands, depleting reserves of bodily energy. It also increases myocardial oxygen demand and may place the person with previous cardiac problems at even greater risk of hypoperfusion.*
- Monitor urinary output per indwelling catheter hourly, using a urine drainage measure bag. *Urine output is a reliable indicator of renal perfusion.*
- Assess mental status and level of consciousness. *The appropriateness of the person's behaviour and responses reflects the adequacy of cerebral circulation. Restlessness and anxiety are common early in shock; in later stages, the person may become lethargic and progress to a comatose state. Altered levels of consciousness are the result of both cerebral hypoxia and the effects of acidosis on brain cells.*

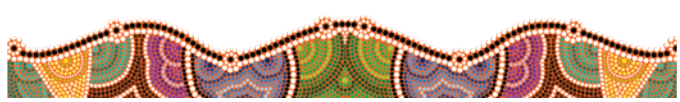
Anxiety

Many people in hypovolaemic shock have experienced some form of major trauma and may have life-threatening, multiple injuries. Following on-the-scene treatment, the person is usually admitted to the healthcare setting through the emergency department. Surgery may be required to treat injuries, followed by care in a critical care unit. Throughout this sequence of crisis events, treatment is invasive and contact with family is minimal. In situations of uncertainty, instability and change people and their families may respond with anxiety, fear and powerlessness. These responses are affected by age, developmental level, cultural and ethnic group, experience with illness and the healthcare system, and support systems.

- Assess the cause(s) of the anxiety and manipulate the environment to provide periods of rest. *Reducing stimuli that cause anxiety is calming and facilitates rest, which is necessary in the person at risk of bleeding.*
- Administer prescribed pain medications on a regular basis. *Pain precipitates and/or aggravates anxiety.*
- Provide interventions to increase comfort and reduce restlessness:
  - Maintain a clean environment.
  - Provide skin and oral care.
  - Monitor the effectiveness of ventilation or oxygen therapy.
  - Eliminate all non-essential activities.
  - Remain with the person during procedures.
  - Speak slowly and calmly, using short sentences.
  - Use touch to provide support.
    Unfamiliar sounds, sights and odours can increase anxiety. Damp skin or a dry mouth increases discomfort. Inadequate gas exchange with a decrease in oxygen or an increase in carbon dioxide in the blood may cause the person to experience a 'feeling of doom'. Activity increases the body's need for oxygen. Listening and touch provide support in an environment in which the person often feels alone and abandoned. Severe anxiety interferes with the ability to understand others and to respond appropriately.
- Provide support for the person and family:
  - Provide time, space and privacy for family members.
  - Allow family members access to the person when feasible.
  - Encourage the expression of feelings and concerns. Provide anticipatory guidance to prepare for recovery or death and to support realistic hope.
  - Acknowledge the beliefs, values and expectations of the person and family.
    Allowing the family access to the person reduces anxiety and gives both the person and the family some feeling of control. If the prognosis is poor, access and involvement allow the family to begin the grieving process. If recovery is expected, contact provides the person and family with a feeling of hope. Supporting the person and family facilitates concrete problem solving, promotes acceptance of the illness and its implications, and helps them begin to establish ways of managing the illness experience.
- Provide information about the current setting to both the person and family; give the family information about available resources (such as pastoral care, social services, temporary housing, meals). *Knowing what to expect and how to control the environment to meet basic needs reduces anxiety.*

Community-based care

Home care for the person who has experienced shock is highly individualised, depending on the cause and the illness or injury that caused shock. Therefore, topics for consideration are not included in this section.



CHAPTER HIGHLIGHTS

- Traumatic injuries affect human tissues and organs resulting from a transfer of energy from the environment. Energy sources can be mechanical, gravitational, thermal, electrical, physical or chemical.
- Trauma types include minor trauma, which causes minimal damage to underlying tissues, or major trauma, which can involve a serious single-system injury or multiple trauma. Trauma is further categorised into blunt and penetrating trauma. Blunt trauma is caused by various forces such as deceleration, acceleration, shearing, compression or crushing. Penetrating trauma occurs when a foreign object enters the body.
- Maintenance of the airway and cervical spine are the highest priority in the trauma victim, with airway assessment superseding all other interventions.
- The primary assessment conducted by the nurse identifies all life-threatening injuries and performance of appropriate interventions. The secondary assessment is when the nurse identifies all injuries in order to prioritise care.
- Shock is a clinical syndrome characterised by a systemic imbalance between oxygen supply and demand. This imbalance results in a state of inadequate blood flow to body organs and tissues, causing life-threatening cellular dysfunction.
- The symptoms of shock arise from the body's attempts to maintain vital organs (heart and brain) and to preserve life in response to a decrease in oxygen delivery to the cells.
- An important early sign of shock is a change in the level of consciousness, with restlessness being a common symptom of cerebral hypoxia.
- Shock is defined in three stages: compensatory (stage 1), an early and reversible stage; progressive (stage 2), occurring after a fluid loss of 35–50% (1800 to 2500 mL), where the affected cells switch from aerobic to anaerobic metabolism in order to remain alive; and the final stage is irreversible (stage 3), where tissue anoxia and cellular death become widespread.
- Hypovolaemic shock is the most common type of shock and is caused by a decrease in the circulating blood volume by 15% or greater.
- Cardiogenic shock is caused when the pumping ability of the heart is compromised to the point where adequate cardiac output cannot be maintained.
- Obstructive shock is caused by an obstruction in the heart or great vessels that either impedes venous return or prevents effective cardiac pumping action. Causes can include cardiac tamponade, pneumothorax, pulmonary embolism and aortic stenosis.
- Septic shock is a part of a progressive syndrome called systemic inflammatory response syndrome (SIRS), a condition most often caused by gram-negative infections.
- Anaphylactic shock is caused by a fulminating hypersensitivity reaction to a foreign substance.

CONCEPT CHECK

1 What is the most common mechanical source of injury in adults of all ages?
1. gunshot wounds
2. fire
3. drowning
4. motor vehicles

2 Severe facial injuries, such as those resulting from going through a windshield, increase the risk of all of the following. Which would you assess first?
1. airway obstruction
2. haemorrhage
3. contusions
4. fractures

3 Which on-the-scene intervention would be a priority?
1. Determine cause of injury.
2. Assess airway patency.
3. Assess peripheral capillary refill.
4. Palpate for internal haemorrhage.

4 You are monitoring blood administration to a trauma victim in shock. Which of the following assessments indicate a dangerous transfusion reaction?
1. red raised areas (wheals) on the skin that itch
2. an increase in body temperature by 3°C
3. decreasing blood pressure and dyspnoea
4. increasing blood pressure and pulse

5 What type of shock causes widespread vasodilation and decreased peripheral resistance?
1. cardiogenic shock
2. septic shock
3. hypovolaemic shock
4. obstructive shock

6 What is the best method to manage uncontrolled bleeding?
1. apply direct pressure
2. clamp a visible vessel
3. apply a tourniquet
4. elevate the injured part

7 Trauma is defined as:
1. injury to human tissues from the transfer of energy
2. result of random chance
3. accidental injury
4. an intentional injury

8 Shock is defined as:
1. a systemic imbalance between oxygen supply and demand
2. sufficient cardiac output
3. haemorrhage
4. abnormal blood pressure

9 Distributive shock is caused by:
1. blood loss
2. widespread vasodilation
3. ineffective cardiac pumping action
4. hypersensitivity reaction

10 What actions should you perform when receiving handover of care immediately postoperative in order to prevent the onset of hypovolaemic shock?
1. Elevate the head of the bed.
2. Provide immediate pain relief.
3. Monitor strict intake and output.
4. Practise careful and consistent hand hygiene.

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CHAPTER 11

NURSING CARE OF PEOPLE WITH INFECTIONS

LISA GATZONIS

LEARNING OUTCOMES

- Discuss the components and functions of the immune system and the immune response.
- Compare antibody-mediated and cell-mediated immune responses.
- Describe the pathophysiology of wound healing, inflammation and infection.
- Identify factors responsible for and the implications of healthcare-associated infections.
- Discuss the nursing implications and health education in the prevention and treatments of inflammation and infection.

CLINICAL COMPETENCIES

- Apply standard precautions and evidence-based practices to prevent the spread of infection within the person, to other people in the facility, to members of the interprofessional team and to visitors.
- Provide safe, effective and respectful patient-centred care for patients with inflammation and infection.
- Collaborate with the interprofessional care team to integrate care of people with infection.
- Promote therapeutic levels and completed dosage of anti-inflammatory and anti-infective medication through prompt administration and person and family teaching.
- Assess for hypersensitivities to anti-inflammatories and anti-infectives prior to and during administration.
- Participate in quality improvement processes to reduce the rates and risk of infection.

KEY TERMS

OVERVIEW OF THE IMMUNE SYSTEM

The human body is continually threatened by foreign substances, infectious agents and abnormal cells. The immune system is the body's major defence mechanism against these threats. Recent years have seen the emergence of resistant microorganisms such as methicillin-resistant *Staphylococcus aureus* (MRSA) and altered strains of familiar diseases, such as multiple-drug-resistant tuberculosis. Other diseases have also emerged, including severe acute respiratory syndrome (SARS), *Clostridium difficile* and human immunodeficiency virus (HIV). The critical need to prevent healthcare-associated infections and their resulting impact on the patient and healthcare costs is an emerging theme.

A thorough knowledge of the immune system increases understanding of inflammatory responses, resistance to infectious disease and the importance of immunisation. This foundation can help the nurse to promote health by preventing and identifying infections and teaching people and families about recommended treatment regimens.

The immune system is a complex and intricate network of specialised cells, tissues and organs. Cells of the immune system seek out and destroy damaged cells and foreign tissue, yet recognise and preserve host cells. The immune system defends and protects the body from invading pathogens, removes and destroys damaged or dead cells, and identifies and destroys malignant cells, thereby preventing their further development into tumours.

The immune system is activated by minor injuries, such as small lacerations or bruises, or by major injuries, such as burns, surgeries and systemic diseases (e.g. pneumonia). The immune response may be innate or adaptive. **Innate immunity** provides non-specific, generic responses to harmful events. These responses prevent or limit the entry of invaders into the body, thereby limiting the extent of tissue damage and reducing the workload of the adaptive immune system. Inflammation is a non-specific response activated by both minor and major injuries. When the inflammatory process is unable to destroy invading organisms or toxins, a more specific response called the **adaptive immune response** is activated. Adaptive immunity provides a response that is specific to unique organisms. It includes memory that hastens future responses to the organism.

Immune system components

The immune system consists of molecules, cells and organs that produce the immune response (see Table 11.1). These components may be involved in the non-specific inflammatory response, the specific immunological response, or both.

Leucocytes

Leucocytes, or white blood cells (WBCs), are the primary cells involved in both innate and adaptive immune system responses. Like all blood cells, leucocytes derive from stem cells, the haemocytoblasts, in the bone marrow (see Figure 11.1). Leucocytes are not confined to the circulation; they use it to transport themselves to the site of an inflammatory or immune response. As the mobile units of the immune system, leucocytes detect, attack and destroy anything that is recognised as 'foreign'. They are able to move through tissue spaces, locating damaged

TABLE 11.1 Cells and tissues of the immune system

COMPONENT	LOCATION	FUNCTION
Leucocytes		
Granulocytes		
Neutrophils	Circulation	Phagocytosis and chemotaxis
Eosinophils	Circulation, respiratory tract and gastrointestinal tract	Phagocytosis Protection against parasites Involved in allergic response
Basophils	Circulation	Release of chemotactic substances
Monocytes and macrophages	Circulation (monocytes) and body tissue, such as skin (histocytes), liver (Kupffer cells), alveoli, spleen, tonsils, lymph nodes, bone marrow, brain	Trapping and phagocytising of foreign substances and cellular debris Secretion of interleukin-1 to stimulate lymphocyte growth
Lymphocytes		
T cells (mature in thymus gland)	Circulation, lymph system, tissues	Activation of T and B cells Control of viral infections and destruction of cancer cells Involved in hypersensitivity reactions and graft tissue rejection
B cells (mature in bone marrow)	Circulation, spleen	Production of antibodies (immunoglobulins) to specific antigens
NK (natural killer) cells	Circulation	Cytotoxic; killing of tumour cells, fungi, viral-infected cells and foreign tissue
Lymphoid tissues		
Primary or central lymphoid structures	Bone marrow and thymus gland	Production of immune cells; sites for cell maturation
Secondary or peripheral lymphoid structures	Lymph nodes, spleen, tonsils, intestinal lymphoid tissue, lymphoid tissue in other organs	Sites for activation of immune cells by antigens

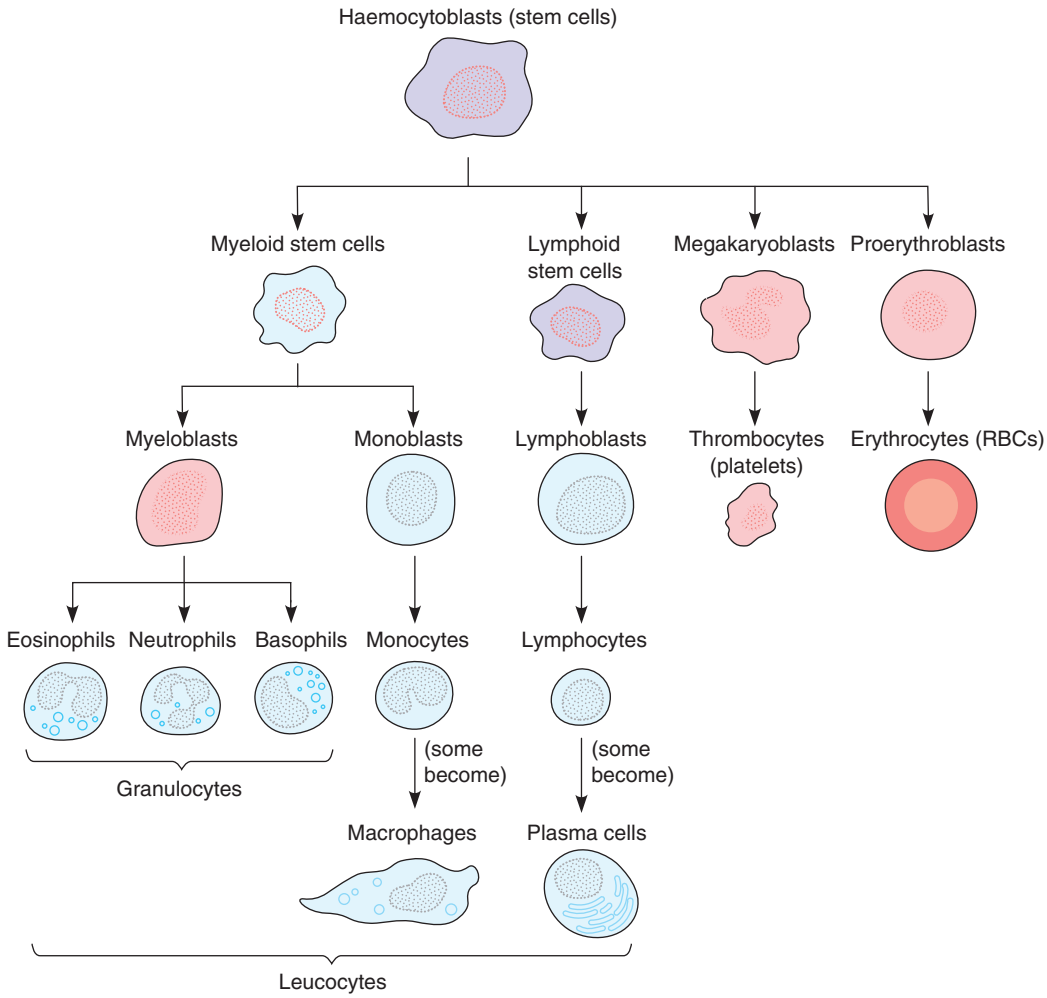


FIGURE 11.1 ■ The development and differentiation of leucocytes from haemocytoblasts

tissue and infection by responding to chemicals released by other leucocytes and damaged tissue.

The normal number of circulating leucocytes is 4500 to 10 000 cells per cubic millimetre (mm^3) of blood. Many more leucocytes are marginated. Margination refers to adhesion of leucocytes to vascular epithelial cells along the vessel walls, in other tissue spaces or in the lymph system. Marginated leucocytes migrate into injured areas or areas where pathogens infiltrate as part of the innate immune response. In the presence of an attack such as an infection, additional WBCs are released from the bone marrow, leading to **leucocytosis**, a WBC count of greater than 10 000/mm^3. As WBCs move out of the bone marrow into the blood, the bone marrow increases its production of additional leucocytes. A decrease in the number of circulating leucocytes, known as **leucopenia**, occurs when bone marrow activity is suppressed or leucocyte destruction increases.

Leucocytes are divided into three major groups: granulocytes, monocytes and lymphocytes. The granulocytes and monocytes derive from the myeloid stem cells of the bone marrow and are instrumental in the inflammatory response. Lymphocytes derive from the lymphoid stem cells of the bone marrow and are the primary cells involved in the specific immune response. In laboratory tests, the WBC count indicates the total number of circulating leucocytes. The White Cell Count (WCC) differential identifies the portion of the total represented by each type of leucocyte.

GRANULOCYTES Granulocytes constitute 60% to 80% of the total number of normal blood leucocytes. Their cytoplasm has a granular appearance and their nuclei are distinctively multilobular (see Figure 11.1). Granulocytes have a short life span, measured in hours to days, compared to the lifespan of monocytes, which is measured in months to years. Granulocytes play a key role in protecting the body from harmful microorganisms during acute inflammation and infection. There are three types of granulocytes: neutrophils, eosinophils and basophils.

Neutrophils, also called polymorphonuclear leucocytes (PMNs or polys), are the most plentiful of the granulocytes, constituting 55% to 70% of the total number of circulating leucocytes. Neutrophils are phagocytic cells, responsible for engulfing and destroying foreign agents, particularly bacteria and small particles. Neutrophils are the first phagocytic cells to arrive at the site of invasion, drawn by chemicals released by damaged tissue and invading organisms.

Neutrophils are produced in the bone marrow and released into the circulation when they mature. Segmented neutrophils (or segs) are mature forms and usually account for about 55% of total leucocytes. *Bands* are immature neutrophils and usually comprise 5% of leucocytes. As neutrophils mature, their nucleus changes from round to kidney-bean-shaped (banded) and then the nucleus separates into small, attached segments; thus the designations 'banded' versus 'segmented' neutrophils. It takes about 10 days for a neutrophil to mature and be released into the circulation. Once released, neutrophils have a circulating half-life of 6 to 10 hours. They cannot replicate and must be replaced constantly to maintain adequate numbers in the circulation. They do not return to the bone marrow.

Eosinophils account for 1% to 4% of the total number of circulating leucocytes. They mature in the bone marrow in 3 to 6 days before being released into the circulation. Eosinophils have a circulating half-life of 30 minutes and a tissue half-life of 12 days. They are phagocytic cells but are less efficient at this process than neutrophils. Eosinophils are found in large numbers in the respiratory and gastrointestinal tracts, where they are thought to be responsible for protecting the body from parasitic worms, including tapeworms, flukes, pinworms and hookworms. Eosinophils surround the parasite and release toxic enzymes from their cytoplasmic granules. The parasite, although too large to be phagocytised, is destroyed. Eosinophils are also involved in a hypersensitivity response, inactivating some of the inflammatory chemicals released during the inflammatory response.

Basophils constitute about 0.5% to 1% of the circulating leucocytes. These cells are not phagocytic. Granules within basophils contain proteins and chemicals such as heparin, histamine, bradykinin, serotonin and slow-reacting substances of anaphylaxis (leukotrienes). These substances are released into the bloodstream during an acute hypersensitivity reaction or stress response.

MONOCYTES, MACROPHAGES AND DENDRITIC CELLS Monocytes, macrophages and dendritic cells are the mediators of immunity. They recognise foreign matter (from molecules to cells) and initiate immune responses. *Monocytes* are the largest of the leucocytes and constitute 2% to 3% of circulating leucocytes. After their release from the bone marrow, monocytes circulate in the serum for 1 to 2 days. They then migrate throughout the body, attaching themselves to the tissues, where they remain for months or even years until they are activated. Monocytes mature into **macrophages** after settling into the tissues. Once they have migrated and matured, macrophages are differentiated by the tissues in which they reside. *Histiocytes* are tissue macrophages in loose connective tissue, *Kupffer cells* are found in the liver, *alveolar macrophage*s in the lungs and *microglia* in the brain. Tissue macrophages are also found in the spleen, tonsils, lymph nodes and bone marrow. Dendritic cells are star-shaped cells that serve as intermediaries between the innate and adaptive immune systems. Dendritic cells capture antigens, transporting them to lymphoid organs such as regional lymph nodes (Grossman & Porth, 2014). Monocytes, macrophages and dendritic cells are antigen-presenting cells (APCs) which activate immune responses in both B and T lymphocytes.

Monocytes, macrophages and dendritic cells are actively phagocytic with the capacity to phagocytise large foreign particles and cell debris. Like neutrophils, macrophages are drawn to an inflamed area by chemicals released from damaged tissue in a process known as chemotaxis. Once they are in the tissue, macrophages can multiply to encapsulate and trap foreign matter that cannot be phagocytised. Monocytes and macrophages activate the immune response against chronic infections such as tuberculosis, viral infections and certain intracellular parasitic infections. Dendritic cells have long processes that can capture antigens and migrate to lymphoid tissue. They serve as sentinels for antigens in most organs including the heart, lungs, liver, kidneys and gastrointestinal tract. Like neutrophils, macrophages are drawn to an inflamed area by chemicals released from damaged tissue, a process known as chemotaxis. Dendritic cells activate T cells against cancer, assist B lymphocytes to produce antibodies and down regulate the immune system.

LYMPHOCYTES **Lymphocytes** account for 20% to 40% of circulating leucocytes. Lymphocytes are the principal effector and regulator cells of specific immune responses that protect the body from microorganisms, foreign tissue, and cell mutations or alterations. Through a process known as immune surveillance, lymphocytes monitor the body for cancerous cells and eliminate or destroy them.

Like other leucocytes, lymphocytes derive from the stem cells in the bone marrow (see Figure 11.2). Lymphocytes have 'homing' patterns: they constantly circulate then return to concentrate in lymphoid tissues (the lymph nodes, spleen, thymus, tonsils, Peyer's patches in the submucosa of the distal ileum and the appendix).

The three types of lymphocytes are **T lymphocytes (T cells)**, **B lymphocytes (B cells)** and **natural killer cells (NK cells)**. None of these cells acts independently. Their functions are closely interrelated. T cells mature in the thymus gland, whereas B cells complete their maturation in the bone marrow. T cells and B cells are integral to the adaptive immune response. On contact with an antigen, B lymphocytes are activated and mature into either plasma cells, which secrete antibodies, or memory cells. On contact with APCs, T lymphocytes mature into active helper T cells, cytotoxic T cells or memory T cells. Memory cells stay inactive, sometimes for years, but activate immediately with subsequent exposure to the same antigen. They then proliferate rapidly, producing an intense immune response. Memory cells are responsible for providing acquired immunity.

NK cells are large, granular cells found in the spleen, lymph nodes, bone marrow and blood. They constitute 15% of circulating lymphocytes. NK cells provide immune surveillance and resistance to infection, and they play an important role in the destruction of early malignant cells. Like B cells and T cells, NK cells are cytotoxic; however, unlike T cells, they do not require a specific antigen to become activated and kill cancer cells, virus-infected cells and cells infected with microbes (Grossman & Porth, 2014). Fortunately, NK cells are inhibited when contact is made with normal host cells.

ANTIGENS Substances the immune system recognises as foreign or 'non-self' are called **antigens**. Antigens provoke a specific immune response when introduced into the body.

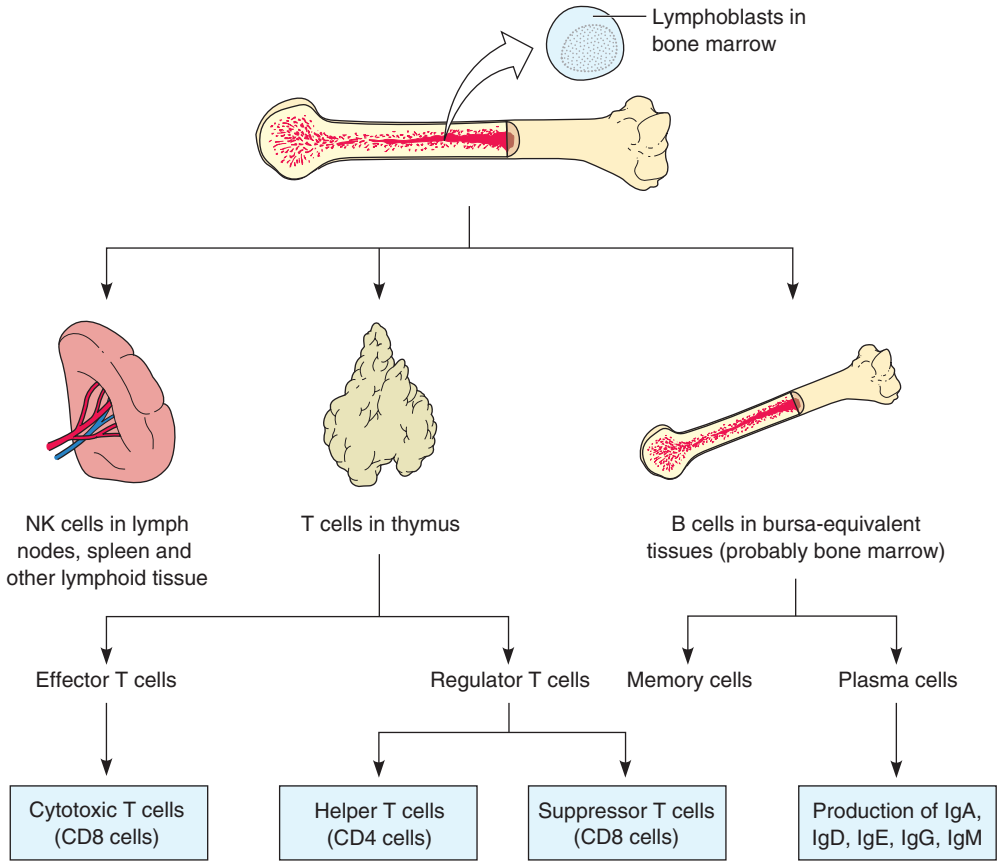


FIGURE 11.2 ■ The development and differentiation of lymphocytes from the lymphoid stem cell (lymphoblasts)

Typically, antigens are large protein molecules found on the cell membrane or cell wall of microorganisms or tissues such as transplanted tissue or organs. Other potentially antigenic substances include pollens, insect venom and the resin of poison ivy (Grossman & Porth, 2014).

Complete antigens, known as immunogens, have two characteristics: (1) *immunogenicity,* the ability to stimulate a specific immune response, and (2) *specific reactivity,* the stimulation of specific immune system components. In contrast, haptens are small molecules (e.g. chemical toxins or dust) that must link with proteins to evoke an antigenic response. When an antigen is encountered in the body, generation of an effective immune response involves two major groups of cells: lymphocytes and antigen-presenting cells (APCs). Macrophages and dendritic cells function as APCs as they capture, process and present antigens to the lymphocytes. Lymphocyte receptors recognise and respond to specific antigens, generating the immune response. Two separate but overlapping immune responses may occur, depending on the antigen itself and the type of immune cell activated by contact with the antigen. The B cell or humoral branch of the immune system mainly targets extracellular antigens such as bacteria, bacterial toxins and free viruses through the production of **antibodies**, molecules that bind with the antigen and inactivate it. The five classes of antibodies are IgG, IgA, IgM, IgD and IgE. These proteins make up the **antibody-mediated (humoral) immune response**. Intracellular pathogens, such as viral-infected cells, cancer cells and foreign tissue, activate T lymphocytes which are the primary agents of the **cell-mediated (cellular) immune response**. In this immune response, the lymphocytes themselves in the form of helper T cells, cytotoxic T cells and NK cells inactivate the antigen either directly or indirectly.

Lymphoid system

The *lymphoid system* consists of the lymph nodes, spleen, thymus, tonsils, lymphoid tissue scattered in connective tissues and mucosa, and the bone marrow. The thymus and bone marrow, in which T cells and B cells mature, are considered central lymphoid organs. The spleen, lymph nodes, tonsils and other peripheral lymphoid tissue are peripheral lymphoid organs (see Figure 11.3). The lymphoid system recovers proteins such as albumin for the vascular system and protects the bloodstream from invading organisms. Immune cells continuously circulate through lymphoid tissues and organs, identifying and destroying foreign antigens. Lymph nodes, the most numerous elements of the lymphoid system, are small, round or bean-shaped encapsulated bodies that vary in size from 1 mm to 2 cm. Lymph nodes generally occur in groups at the junction of the lymphatic vessels. They can be found in the neck, axillae, abdomen and groin.

Lymph nodes filter foreign products or antigens from the lymph and house and support proliferation of lymphocytes and macrophages. Lymph, a clear, protein-containing fluid transported within lymph vessels, enters the node through afferent lymphatic vessels. Inside the node, the lymph flows through sinuses in the

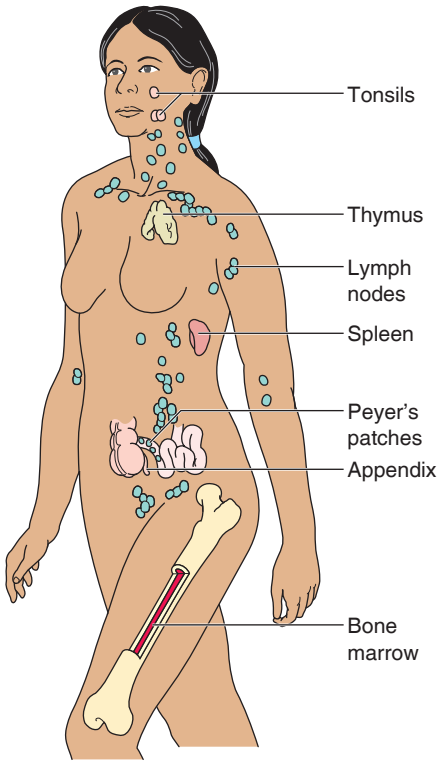


FIGURE 11.3 ■ The lymphoid system: the central organs of the thymus and bone marrow, and the peripheral organs, including the spleen, tonsils, lymph nodes and Peyer's patches

cortex of the lymph node where T and B lymphocytes and macrophages are abundant, then through sinuses of the medulla of the lymph node which contains macrophages and plasma cells. The presence of a foreign antigen stimulates lymphocytes and macrophages to proliferate in the lymph nodes. Macrophages destroy the antigen by phagocytosis. Immune cells and lymph then leave the lymph node through efferent vessels. An abundant blood supply to the node also facilitates lymphocyte movement.

The spleen is the largest lymphoid organ in the body and the only lymphoid organ that can filter blood. The spleen is located in the upper left quadrant of the abdomen. The spleen has two kinds of tissue: white pulp and red pulp. White pulp is lymphoid tissue that serves as a site for lymphocyte proliferation and immune surveillance. B cells predominate in the white pulp. Blood filtration occurs in the red pulp. In blood-filled venous sinuses, phagocytic cells dispose of damaged or aged RBCs and platelets. Other debris and foreign matter, such as bacteria, viruses and toxins, are also removed from the blood. The spleen also stores blood and the breakdown products of RBCs for future use. The spleen is not essential for life. If it is removed because of disease or trauma, the liver and the bone marrow assume its functions.

The *thymus gland* is located in the superior anterior mediastinal cavity beneath the sternum. It reaches its maximum size at puberty, then begins to atrophy slowly. By adulthood, it is difficult to differentiate from surrounding adipose tissue even though it remains active. In the older adult, the vast majority of thymus tissue has been replaced by adipose and fibrous connective tissue. During foetal life and childhood, the thymus serves as a site for the maturation and differentiation of thymic lymphoid cells, the T cells. Thymosin, an immunoregulatory hormone of the thymus, stimulates lymphopoiesis, the formation of lymphocytes or lymphoid tissue.

Bone marrow is soft organic tissue found in the hollow cavity of the long bones, particularly the femur and humerus, as well as the flat bones of the pelvis, ribs and sternum. Bone marrow produces and stores haematopoietic stem cells from which all cellular components of the blood are derived (see Figure 11.1).

Lymphoid tissues are also located at key sites of potential invasion by microorganisms: the submucosa of the genitourinary, respiratory and gastrointestinal tracts and the skin. Plasma cells in these lymphoid tissues defend the body against bacterial invasion at areas exposed to the external environment. In general, these tissues are known as *mucosa-associated lymphoid tissue* (MALT). Diffuse collections of lymphocytes, plasma cells and phagocytes are scattered throughout the respiratory tract, concentrating at bifurcations of the bronchi and bronchioles. Peyer's patches, or gut-associated lymphoid tissue (GALT), comprise the largest collection of immune cells in the body. Ingestion and absorption of solid foodstuffs and liquids continually expose the lining of the gut to resident microflora and infectious pathogens. Unlike peripheral lymph nodes, which respond to pathogens with acute inflammatory responses, GALT processes common intestinal antigens without producing acute inflammation. Collections of immune cells make up the GALT. Intraepithelial lymphocytes fill the spaces between mucosal epithelial cells. Beneath the basement membrane of gut epithelium lie abundant T cells and mature plasma cells, which are sources of IgA. Peyer's patches hold dense collections of lymphocytes in lymphoid nodules. As naive B and T cells migrate through Peyer's patches, they are sensitised to specific antigens. In mesenteric lymph nodes, these sensitised cells proliferate and circulate throughout the vascular tree where they produce secretory IgA. Secretory IgA coats mucosal cells and prevents attachment of intraluminal bacteria in the intestine, upper respiratory tract, bronchi, mammary ducts and salivary glands. Thus the GALT collection of immune cells effectively protects mucosa throughout the body that is exposed to resident and foreign pathogens.

Tonsils and adenoids protect the body from inhaled or ingested foreign agents. Skin-associated lymphoid tissue contains lymphocytes and dendritic cells such as Langerhans cells in the epidermis, which transport antigens to regional lymph nodes for destruction and development of specific immunity to the antigen.

Innate immune response

Innate or natural immunity is the first line of defence against infection. It is non-specific and includes skin and mucosal barriers, vascular and cellular responses and phagocytosis. Cells involved in innate immunity include phagocytic neutrophils and macrophages, and NK cells which target intracellular pathogens. Soluble molecules such as opsonins, cytokines, acute-phase proteins (such as C-reactive protein) and the complement system also are involved in innate immunity.

Barrier protection is the body's first line of defence against infection. Intact skin prevents invasion by external organisms. When the skin is damaged or lost (e.g. as a result of injury, surgery

or burns), infection is much more likely. A barrier of mucus, which traps microorganisms and other foreign substances, protects the membranes lining inner surfaces of the body. These can then be removed by other protective mechanisms, such as ciliary movement or the washing action of tears or urine. In addition, many body fluids contain bactericidal substances that provide barrier protection. These include acid in gastric fluid, zinc in prostatic fluid and lysosyme in tears, nasal secretions, saliva and sweat.

When these defences are breached, the resulting tissue damage or foreign material entering the body induces inflammation, another innate defence mechanism. **Inflammation** is a response to injury that brings fluid, dissolved substances and blood cells into the interstitial tissues where the invasion or damage has occurred. The response is *non-specific* as the same events occur regardless of the cause of the inflammatory process. Through the inflammatory reaction, the invader is neutralised and eliminated, destroyed tissue is removed and the process of healing and repair is initiated.

The inflammatory response has two stages: (1) a vascular response characterised by vasodilation and increased permeability of blood vessels, and (2) a cellular response. Phagocytosis sets the stage for healing (tissue repair).

Vascular response

After tissue cells are damaged, local blood vessels briefly constrict. Vasodilation of the capillary arterioles and venules follows almost immediately as inflammatory mediators such as histamine and kinins are released from damaged tissue (see Box 11.1). Increased blood flow causes vasocongestion at the injury site with resultant redness and heat. The congestion also increases local hydrostatic pressure. This, along with increased vessel permeability that results from chemical mediators, moves fluid out of the capillaries and into the interstitial spaces of the tissue. The escaping fluid, called fluid exudate, contains large amounts of protein. This protein increases osmotic pressure in the interstitial spaces, which draws fluid and causes local oedema. Fluid exudate provides protection to the injured tissue by transporting to the tissue certain nutrients needed for tissue healing, diluting bacterial toxins and transporting cells needed for phagocytosis. Exudate may range from *serous,* primarily plasma with some proteins, to *sanguineous* containing large amounts of blood cells. *Fibrinous* exudate forms a thick, sticky meshwork of fibrinogen, in effect 'walling off' inflamed tissues and preventing the spread of infection. In more severe or acute inflammation, the fluid contains fibrin, RBCs and dead and live bacteria. This type of exudate, called *purulent* exudate, has an odour and colour characteristic of the bacteria present.

The vascular response localises invading bacteria and keeps them from spreading. Increased capillary permeability enhances the release of clotting factors such as fibrinogen which converts to fibrin threads, entrapping the bacteria and walling them off from contact with the rest of the body.

Cellular response

The cellular stage of the inflammatory process begins within less than an hour after the injury. This stage is marked by changes in the lining of blood vessels and movement of phagocytic blood cells into the damaged tissue.

As serous fluid escapes the capillaries, the viscosity of blood in the area increases and its flow becomes more sluggish. Leucocytes move to the edges of the blood vessels where they accumulate, their movement slows and they begin to adhere to

BOX 11.1 Inflammatory mediators

Many of the manifestations of inflammation are produced by chemicals released as a result of immunological processes or tissue injury or damage. These inflammatory mediators are broadly classified as follows:

- Vasoactive substance (e.g. histamine, prostaglandins, leukotrienes and platelet-activating factor) produce smooth muscle constriction, vasodilation and increased capillary permeability.
- Chemotactic factors (e.g. complement fragments and chemokines) attract leucocytes to the damaged tissue.
- Plasma enzymes (proteases) activate the complement system, the clotting cascade and the vasoactive kinins system, contributing to the vascular phase of the inflammatory response.
- Miscellaneous cell products (e.g. oxygen metabolites and lysosomal enzymes) damage surrounding tissue.

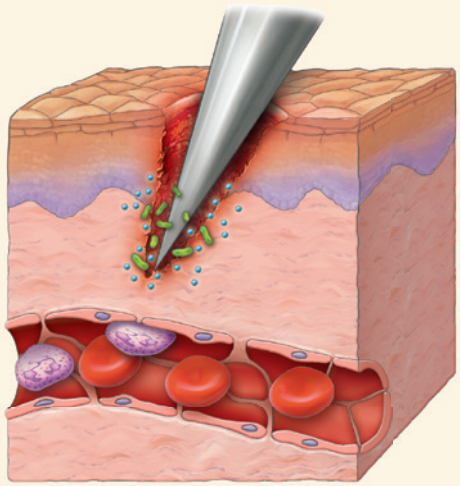
Many of the outward manifestations of inflammation result from vasoactive substances such as histamine, prostoglandins and leukotrienes. Stored in mast cells, basophils and platelets, *histamine* is released when an injury occurs or with stimulation by the immune system. An important component of the early inflammatory response, histamine causes vasodilation and vascular permeability in the affected area. Histamine is also a key factor in many hypersensitivity reactions. The *leukotrienes*, collectively known as slow-reacting substances of anaphylaxis (SRS-A), play a significant vasoactive role in the later stages of the inflammatory response.

Prostaglandins are chemotactic substances that draw leucocytes to the inflamed tissue. In addition, they play a vasoactive role and are pain and fever inducers. Aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs), as well as the glucocorticoids, inhibit prostaglandin synthesis, thereby reducing fever, pain and inflammation.

Plasma proteases activate the clotting cascade, kinin system and complement system. With activation of the clotting cascade, bacteria and other foreign substances are trapped in the area of tissue damage. Fibrin, which has vasoactive by-products, is also released. Activation of the complement system causes vasodilation, increases vessel permeability and facilitates the phagocytic process. Through the release of bradykinin, the kinin system has similar effects. Bradykinin also stimulates pain receptors.

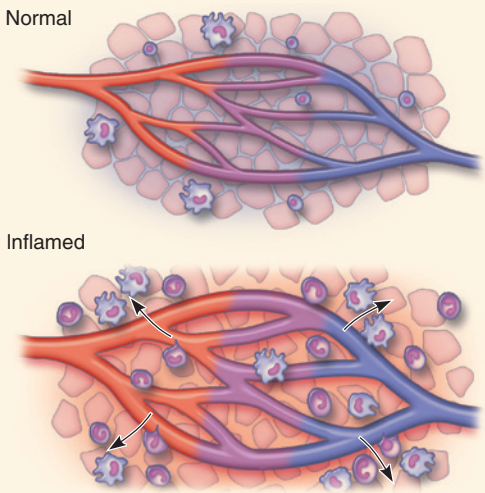
Major chemical mediators of inflammation are summarised in Table 11.2.

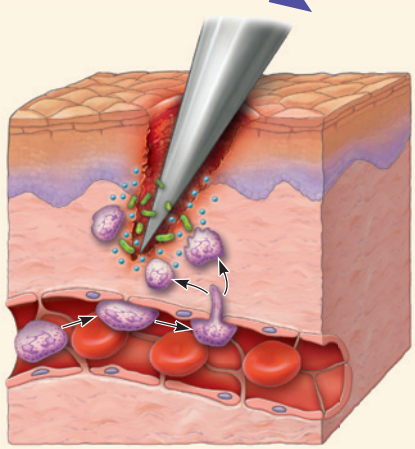
Acute inflammation



1. Inflammation is a key component of innate immunity, the body's immediate response to tissue damage or invasion of the body by foreign material. The inflammatory response serves to contain, control and eliminate damaged cells and tissue, microorganisms and antigens.

2. Vascular response. Tissue damage causes brief, initial vasoconstriction which is rapidly followed by vasodilation, with resulting redness and warmth. Inflammatory mediators (e.g. histamine, prostaglandins, bradykinins) released in the innate immune response and by damaged tissue dilate local blood vessels and increase the permeability of capillaries in the area. Protein-rich fluid (exudate) accumulates in interstitial spaces, causing swelling and pain. Resulting oedema slows blood flow and, together with activation of clotting in the area, helps localise and prevent microorganisms from spreading.





3. Cellular response. Within less than an hour after the injury, the cellular stage of the inflammatory process brings phagocytic blood cells into the damaged tissue. Loss of serous fluid from capillaries increases blood viscosity in the area and slows its flow. Leucocytes marginate, moving to the vessel periphery and adhering to the capillary endothelium. As a result, endothelial cells separate, allowing leucocytes to transmigrate through vessel walls into the tissue spaces. Chemotactic signals draw the leucocytes to the site of the injury or infection.

4. Phagocytosis. Once attracted to the inflammatory site, phagocytes engulf the foreign agent or target cell by projecting pseudopodia ('false feet') in all directions around it. This produces a phagosome containing the antigen, which is ingested into the cytoplasm. Once engulfed, lysosomes fuse with the phagosome, killing any live organism and releasing digestive enzymes, which destroy the antigen.

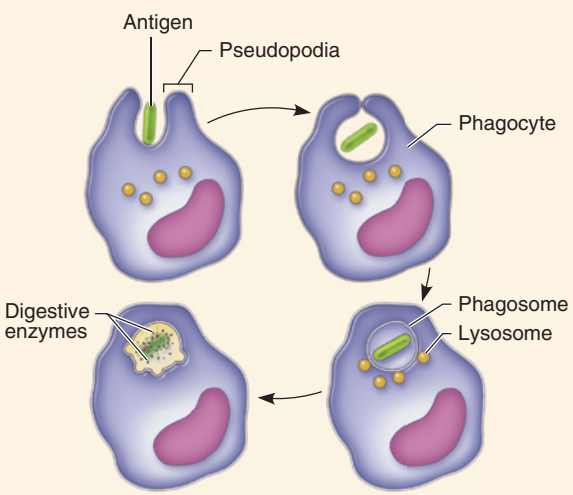


TABLE 11.2 Major chemical mediators of inflammation

FACTOR	SOURCE	EFFECT
Histamine	Mast cells, basophils and platelets	Vasodilation and increased capillary permeability producing tissue redness, warmth and oedema
Kinins (bradykinin and others)	Plasma proteins	Histamine-like effects; chemotaxis and pain inducers
Prostaglandins	Formed from arachidonic acid found in cell membranes	Histamine-like effects; chemotaxis, pain, and fever inducers
Leukotrienes	Formed from arachidonic acid	Smooth muscle constriction (especially bronchoconstriction), increased vascular permeability, chemotaxis

the capillary endothelium. This process is known as *margination*. Leucocyte adhesion causes separation of endothelial cells, allowing leucocytes to transmigrate through the blood vessel wall into the tissue spaces. Within hours, millions of leucocytes emigrate into the area of inflammation.

Once leucocytes have emigrated, they are drawn to the damaged or inflamed tissues by chemotactic signals. Infectious agents, damaged tissues and activated plasma substances such as complement fractions provide chemotaxic signals that attract an army of neutrophils, monocytes and macrophages to the injury site.

The number of neutrophils around the site increases to about 15 000 to 25 000/mm^3 and they begin their role in phagocytosis within a few hours. Monocytes become transient macrophages to augment the activity of the fixed macrophages and dendritic cells; together they engulf dead cells, damaged tissue, non-functioning neutrophils and invading bacteria.

Phagocytosis

Phagocytosis is a process by which a foreign agent or target cell is recognised, engulfed and destroyed. Neutrophils, monocytes and macrophages, known as *phagocytes*, are the primary cells involved in phagocytosis. Once attracted to the inflammatory site, phagocytes select and engulf foreign material.

The following factors or processes help phagocytes differentiate foreign tissue from normal cells:

- *Smooth surface*. Normal tissue has a smooth surface that is resistant to phagocytosis, whereas the rough surface of a foreign agent or target cell promotes phagocytosis.
- *Surface charge*. Healthy body cells present an electronegative surface charge that repels phagocytes. Cellular debris and foreign agents, by contrast, have an electropositive charge that attracts them.
- *Opsonisation*. This immune system process coats the surface of bacteria or target cells with soluble molecules (opsonins) such as complement, lectins and other proteins (see Box 11.2). Opsonisation enables the phagocyte to bind tightly with the foreign tissue, facilitating phagocytosis.

Phagocytes engulf the foreign agent or target cell by projecting pseudopodia in all directions around it. This produces a chamber called a *phagosome* containing the antigen, which is ingested into the cytoplasm. Once the phagosome has been engulfed, lysosomes fuse with the phagosome, releasing antimicrobial molecules and digestive enzymes which destroy the antigen. Phagocytes produce bactericidal agents that kill most pathogens. These agents include toxic oxygen and nitrogen radicals, such as nitric oxide, hydrogen peroxide and hydroxyl ions, as well as

BOX 11.2 The complement system

The *complement system* consists of approximately 20 complex plasma proteins that are activated by a tissue injury or antigen–antibody reaction. The complement system is involved in both innate and adaptive immune responses. Its activation results in the production of effector molecules that are involved in the processes of inflammation, phagocytosis, and cell lysis or destruction (Grossman & Porth, 2014). Specifically, complement activation leads to the following:

- *Mediation of the inflammatory response*. When the complement system is activated, chemical mediators such as histamine are released from mast cells and basophils, leading to smooth muscle contraction, increased vascular permeability and oedema, and the attraction of leucocytes.
- *Opsonisation* (or coating) of microbes and antigen–antibody complexes. Opsonisation facilitates recognition of and binding to the antigen by phagocytes, and activation of phagocytosis.
- *Alteration of the cell membrane or viral capsule*. Complement can alter cell membranes, forming pores that cause cell lysis and death. Bacteria and viruses are destroyed; certain normal cells such as RBCs that are damaged or old may also be destroyed through this process.

The complement system has three 'arms', or pathways, of protein and enzyme reactions. The *classic pathway* is activated by antibody-containing immunoglobulins and other substances such as DNA and C-reactive protein. The *alternative* and *lectin pathways* function in innate immunity; they do not require antibodies but are activated by tissue injury, properties of the microbial antigen and proteins produced in response to injury (e.g. C-reactive protein) (Grossman & Porth, 2014). Complement activation results in mediation of the inflammatory process, attraction of phagocytes, facilitation of phagocytosis and lysis of microbes.

digestive enzymes (e.g. lysozyme) that break down bacterial cell walls (Mayer, 2011). They also can produce antimicrobial molecules known as *defensins* (Grossman & Porth, 2014).

Some antigens, such as the tubercle bacillus, have coats or secrete substances that are resistant to lysosomal and bactericidal agents. To destroy such antigens, lysosomes release digestive enzymes into the phagosome. The lysosomes of neutrophils and macrophages contain an abundance of proteolytic (protein-destroying) enzymes that digest bacteria and other foreign protein components. The macrophage's lysosomes also contain lipases (fat-splitting enzymes) capable of digesting the thick lipid membranes of such bacteria as *Mycobacterium tuberculosis* and *Mycobacterium leprae*.

Once neutrophils have ingested toxic substances to their capacity, they in turn are destroyed. Neutrophils have the capacity to phagocytise 5 to 20 bacteria before they become inactive. Macrophages then digest the dead neutrophils. Monocytes or macrophages are capable of phagocytising up to 100 bacteria. Because of their size, they can ingest larger particles than neutrophils can ingest, such as whole RBCs, necrotic tissue, cell fragments, malarial parasites and dead neutrophils. Dendritic cells are also phagocytic and secrete IL-12, which is an important cytokine in the maturation of helper T cells. Macrophages have the ability to extrude (release) the toxic substances and lysosomal enzymes within their phagosomes. As a result, they can continue to function for months and even years.

Healing

During the inflammatory process, particulate matter, bacteria, damaged cells and inflammatory exudate are removed by phagocytosis. This process, called *debridement*, prepares the wound for healing. Adequate nutrition is essential for inflammation and healing to proceed. Protein, glucose and oxygen are needed by leucocytes for chemotaxis, phagocytosis and intercellular killings.

The second phase of the healing process, known as *reconstruction*, may overlap the inflammatory phase. The ideal result of the healing process is *resolution*, the restoration of the original structure and function of the damaged tissue. Simple resolution occurs when there is no destruction of the normal tissue and the body is able to neutralise and remove the offending agent through the inflammatory process.

Resolution may also occur when the damaged tissue is capable of regeneration. The ability to regenerate or replace lost *parenchyma* (functional tissue) with new functional cells varies by tissue and cell type.

- *Labile cells* continue to regenerate throughout life. These cells are found in tissues where there is a daily turnover of cells—namely, bone marrow and the epithelial cells of the skin, mucous membranes, cervix, gastrointestinal tract and genitourinary tract.
- *Stable cells* normally stop replicating when growth ceases but are capable of regeneration when stimulated by an injury. Osteocytes (which are found in bone) and parenchymal cells of the kidneys, liver and pancreas are stable cells.
- *Permanent* or *fixed cells* are unable to regenerate. When these cells are destroyed they are replaced by fibrous scar tissue. Nerve cells, skeletal muscle cells and cardiac muscle cells are fixed cells.

When regeneration and complete resolution are not possible, healing occurs by replacement of the destroyed tissue with collagen scar tissue. This process is known as *repair*. Although tissue that has undergone repair lacks the physiological function of the destroyed tissue, the scar fills the lesion and provides tensile tissue strength.

Adaptive immune response

The adaptive immune response is a more specific reaction than innate immunity. On the first exposure to an antigen, a change occurs in the host, resulting in a specific and rapid response following subsequent exposures.

The adaptive immune response has the following distinctive properties:

- The immune response typically is directed against materials recognised as foreign (i.e. from outside the body) and is not usually directed against the self (i.e. cells or structures produced by the body). This property is known as *self-recognition*.
- The immune response is *specific*. It is initiated by and directed against particular antigens (such as a specific virus, bacterium or transplanted tissue).
- Unlike a localised inflammatory response, the immune response is systemic. Immunity is not restricted to the initial site of infection or entry of foreign tissue.
- The immune response has memory. Repeated exposures to an antigen produce a more rapid response.

A person whose immune system is able to identify antigens and effectively destroy or remove them is said to be **immunocompetent**.

There are two types of adaptive immune responses: humoral or antibody-mediated immunity, and cellular or cell-mediated immunity.

Antibody-mediated immune response

The antibody-mediated (humoral) immune response is produced by B lymphocytes (B cells). B cells are constantly replaced through cell division and proliferation in the bone marrow. It is believed that B cells mature in the bone marrow and then migrate to the spleen to await activation. They normally constitute 10% to 15% of circulating lymphocytes.

B cells are activated by contact with an antigen and by T cells (discussed in the next section). Each B cell has receptor sites for a specific antigen or antigens. When the antigen is encountered, the activated B cell proliferates and differentiates into antibody-producing plasma cells and memory cells (see Figure 11.4). Plasma cells are short-lived, lasting only about 1 day. While alive, however, they can produce thousands of antibody molecules per second. Memory cells retain antibody-producing information, allowing a rapid response if the antigen is again encountered.

An antibody is an **immunoglobulin (Ig)** molecule with the ability to bind to and inactivate a specific antigen. Immunoglobulins fall into five classes: IgG, IgA, IgM, IgD and IgE. Each has a slightly different structure and function. Their roles are summarised in Table 11.3.

Antibodies are Y-shaped molecules with two light and two heavy polypeptide chains (see Figure 11.5). The top portion of the Y, called the *Fab* or *antigen-binding fragment*, is chemically variable and specific to the antigen. The lower portion, the

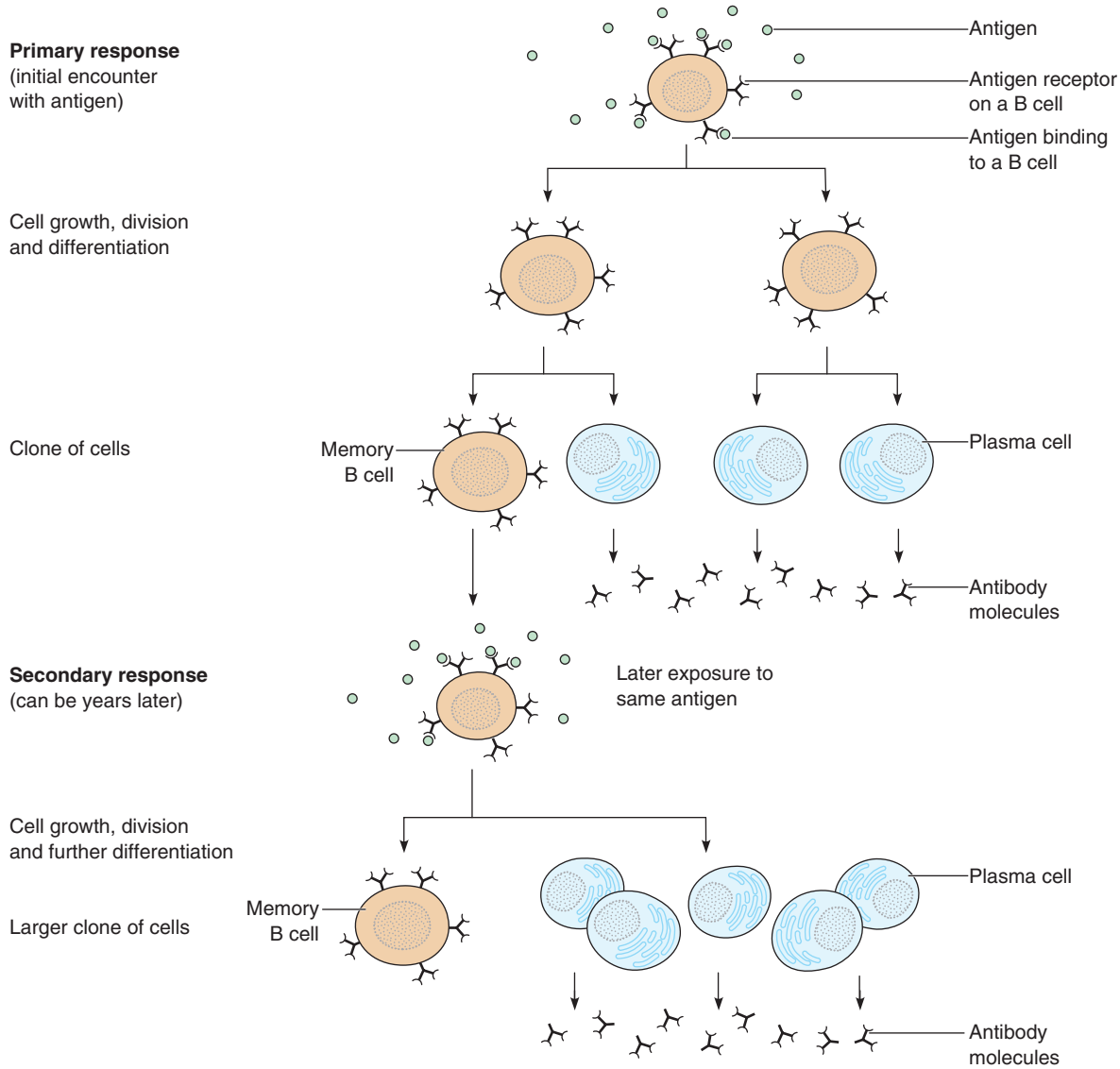


FIGURE 11.4 ■ Antibody-mediated (humoral) immunity. On initial exposure to the antigen, B cells with appropriate receptor sites are activated to become plasma cells and produce antibodies or memory cells. This is known as the primary response. With subsequent exposures, memory cells respond rapidly with antibody production. This is known as the secondary response.

Fc or *crystallised fragment*, is constant for its class of immunoglobulin and directs the biological activity of the immunoglobulin (the manner in which it functions). For example, the lower portion of immunoglobulin molecules produced against hepatitis A and hepatitis B are the same (IgG), but the upper portion is different and specific to the virus.

The antibodies produced by B cells (see Figure 11.5) link with the antigen (see Figure 11.6) and inactivate it through one or more of the following processes:

- covering the antigen with antibodies to attract phagocytes, including neutrophils, macrophages and eosinophils
- precipitation—combining with soluble antigens to form an insoluble complex or precipitate that can be captured and destroyed by phagocytes
- neutralisation—combining with a virus or toxin to neutralise its effects by preventing it from attaching to cells and tissues; the antigen–antibody complex is then destroyed by the process of phagocytosis
- complement activation and fixation to the antigenic cell surface, leading to cell lysis
- agglutination (clumping) of insoluble antigens (e.g. a cell or virus) to form a large complex
- opsonisation—coating of the antigen with antibodies and complement, making them more susceptible to phagocytosis.

The complete antibody-mediated response occurs in two phases. With initial exposure to an antigen, the primary response develops. B cells are activated to proliferate and begin producing antibodies. There is a latency period of 3 to 6 days before antibodies become detectable in the blood. Levels then continue to rise, peaking at 10 to 14 days after the initial exposure. With many illnesses (e.g. chickenpox), this peak correlates with recovery.

TABLE 11.3 Immunoglobulin characteristics and functions

CLASS	PERCENTAGE OF TOTAL	CHARACTERISTICS AND FUNCTION
IgG	75%	Most abundant Ig; also known as gamma globulin; found in blood, lymph and intestines Active against bacteria, bacterial toxins and viruses Activates complement and binds to macrophages The only Ig to cross the placenta, providing immune protection to neonates
IgA	10–15%	Found in saliva, tears and bronchial, gastrointestinal, prostatic and vaginal secretions, as well as blood and lymph Provides local protection on exposed mucous membrane surfaces and potent antiviral activity by preventing binding of the virus to epithelial cells Levels decrease during stress
IgM	5–10%	Found in blood and lymph First antibody produced with primary immune response High concentration early in infection decreases within about a week Mediates cytotoxic response and activates complement
IgD	< 1%	Found in blood, lymph and surfaces of B cells Exact function unknown; may be receptor-binding antigens to B-cell surface
IgE	< 0.1%	Found on mast cells and basophils Involved in release of chemical mediators responsible for immediate hypersensitivity (allergic and anaphylactic) response and parasitic infections

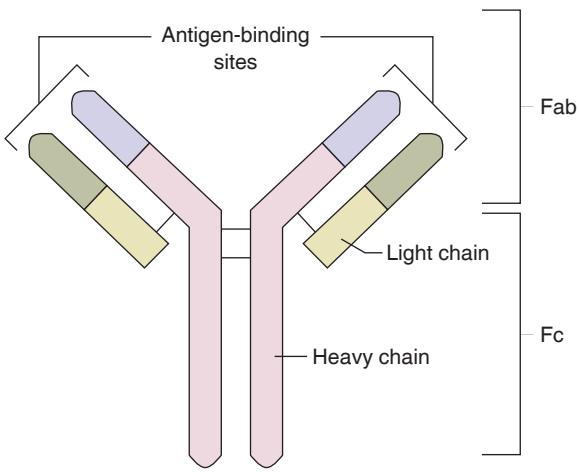


FIGURE 11.5 ■ An antibody molecule. The Fab section is unique, providing an antigen-specific binding site. The Fc section is common to each class of immunoglobulin (IgG, IgA, IgM, IgD, IgE)

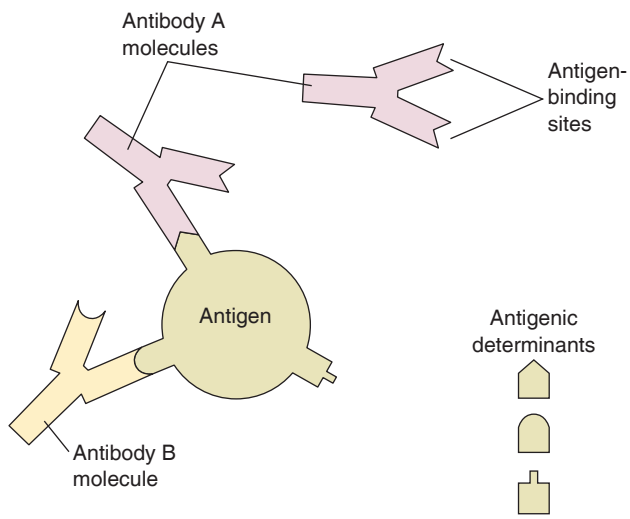


FIGURE 11.6 ■ Antigen–antibody binding. The unique Fab site on the antibody binds with specific receptor sites on the antigen. As shown, more than one kind of antibody may be produced for an antigen

Subsequent exposure to the same antigen elicits a secondary response. Memory cells (see Figure 11.4) formed during the primary response stimulate the production of plasma cells and an almost immediate rise in antibody levels occurs (see Figure 11.7). This rapid secondary response is the basis of acquired immunity and is instrumental in preventing disease. It is also the mechanism through which vaccines provide protection from disease.

Cell-mediated immune response

Many antigens cannot stimulate the antibody-mediated response or are hidden from it because they live inside the body's cells (e.g. viruses and mycobacteria). The cell-mediated immune response, also known as *cellular immunity,* provides protection against these antigens. T lymphocytes (T cells) initiate this type of immune response.

Approximately 70% to 80% of circulating lymphocytes are T cells. T cells migrate to the thymus during foetal and early life, establishing the lifetime pool of cells. T cells have a lifespan measured in years, maintaining their numbers through proliferation, primarily in the lymph nodes.

T cells are much more complex than B cells. There are two major classes of T cells: CD4 cells and CD8 cells, *differentiated by their cell surface proteins (or markers).*

T cells are antigen-specific; that is, each subset is activated by a particular antigen. The antigens that activate T cells must

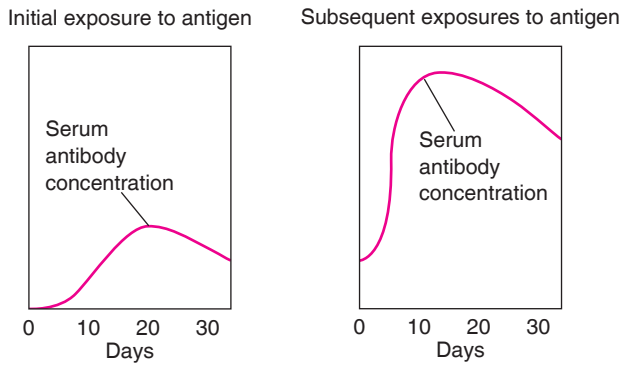


FIGURE 11.7 ■ Antibody production in the primary and secondary responses of the antibody-mediated immune response. Note the more rapid and effective production following subsequent exposure

be presented on another cell surface, such as pieces of virus presented on the surface of an infected cell or the histocompatibility locus antigen on a cell of transplanted tissue. When activated, T cells divide and proliferate, forming antigen-specific *clones* (see Figure 11.8). Activated T cells further differentiate to become *cytotoxic cells*, *helper cells* or *suppressor cells.* *Memory cells* are also formed; these remain in reserve for future encounters with the antigen.

The *cytotoxic T cell* (T_C cell), an effector cell with the CD8 markers, seeks out and destroys abnormal cells and cells harbouring anything foreign (e.g. viruses). Cytotoxic T cells bind with cell surface antigens on virus-infected or foreign cells. T_C cells destroy the identified cell by combining with it and then either destroying its cell membrane or releasing cytotoxic substances into the cell. They are vital in the control of viral and bacterial infections.

Helper T cells (T_H cells) develop from T cell populations with the CD4 marker. T_H cells coordinate immune responses to an antigen. They stimulate the proliferation of other T cells, amplify the cytotoxic activity of T_C cells and amplify the innate immune response. T_H cells interact directly with B cells to promote their multiplication and conversion into plasma cells capable of producing antibodies.

Suppressor T cells (T_S cells), a much smaller subgroup of T cells, are important regulators of immune responses. Suppressor T cells release inhibitory cytokines, which inhibit the activity of other T cells and B cells and limit the extent of the immune response to an antigenic stimulus.

On activation, both effector and regulator T cells synthesise and release soluble proteins known as **cytokines** (see Box 11.3).

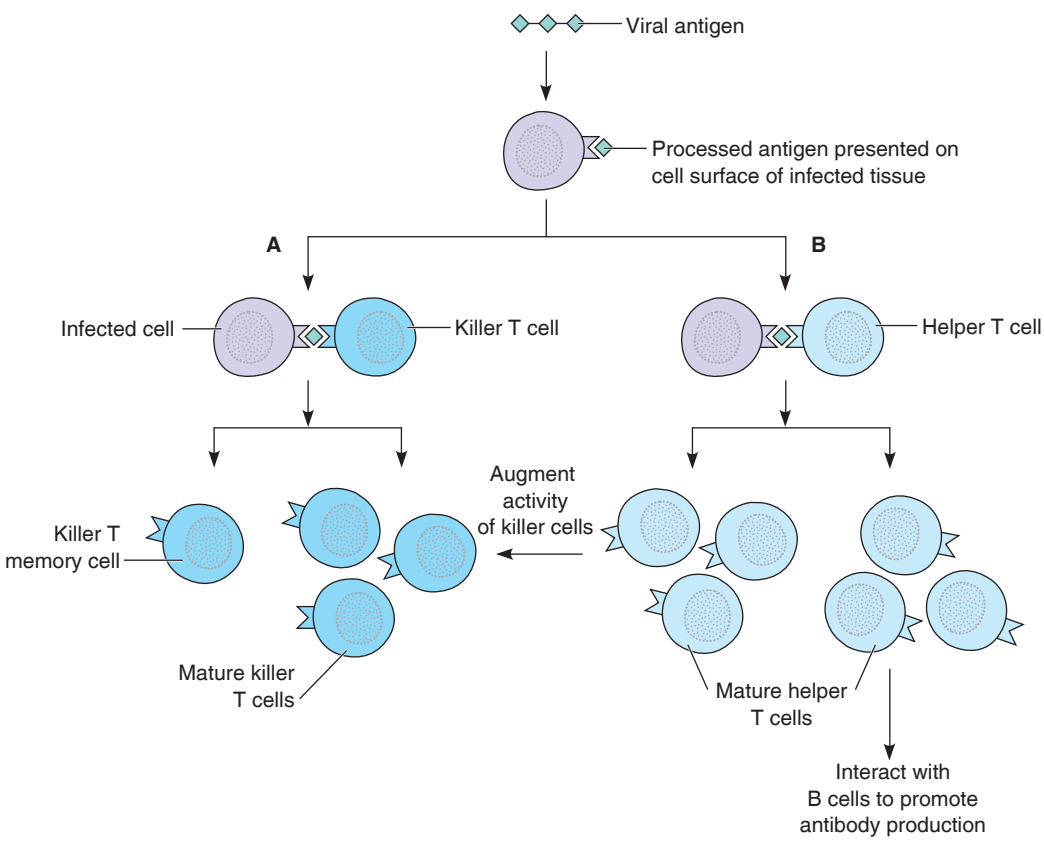


FIGURE 11.8 ■ Cellular immune response. *A*, An infected cell, abnormal cell or phagocyte presents antigen on its surface that binds with a receptor site on a killer T cell or a helper T cell. The killer T cell is activated to proliferate into memory cells or mature cytotoxic cells. *B*, The helper T cell is activated to augment the cytotoxic response and stimulate the antibody-mediated immune response

BOX 11.3 Cytokines

Cytokines, essential components of an adequate immune response, are hormone-like polypeptides produced primarily by cells of the immune system. Cytokines are produced in small quantities in many different tissues throughout the body. Cytokines act as messengers of the immune system, facilitating communication between the cells to adjust or vary the inflammatory reaction or to initiate immune cell proliferation and differentiation. The major cytokines and their functions are summarised in Table 11.4.

The inflammatory cytokines contribute to illness behaviours. People respond to increases in these chemicals with increased sleep, a need to seek warmth and reduced energy output. These are considered adaptive responses to illness. Interventions to reduce or eliminate the production of certain cytokines are common. Aspirin and NSAIDs to reduce pain and fever are commonly used. Because some cytokines cross the blood–brain barrier, their increase may explain depression and anxiety experienced during illness.

Interferons are a class of cytokine with broad antiviral and anticancer effects. A number of different forms of interferon exist, broadly grouped as alpha, beta and gamma interferons. Interferon is synthesised by cells infected with a virus and secreted into extracellular fluid. It then binds to specific receptors on uninfected neighbouring cells, protecting them from infection. The spread of the virus is thus inhibited and recovery from infection enhanced. It appears that interferons also moderate the activity of NK cells and may be involved in preventing the spread of abnormal malignant cells.

TABLE 11.4 Major cytokines and their functions

CYTOKINE	WHERE PRODUCED	PRIMARY FUNCTIONS
Interleukin-1 (IL-1)	Monocytes, macrophages and dendritic cells	Activates T and B cells Induces fever and tissue catabolism Enhances NK activity Attracts neutrophils, macrophages and lymphocytes Stimulates bone marrow and endothelial cell growth, collagen and collagenases
Interleukin-2 (IL-2)	Helper T cells	Stimulates T and B cell proliferation, aids in discriminating between self and non-self Activates killer T and NK cells
Interleukin-3 (IL-3) Interleukin-4 (IL-4)	T cells	Stimulates growth and differentiation of bone marrow stem cells Stimulates proliferation of T cells Increases IgE secretion by B cells
Interleukin-5 (IL-5)	T cells and activated mast cells	Promotes differentiation of B cells and eosinophils Stimulates production of IgA
Interleukin-6	T cells and macrophages	Is a pro-inflammatory and anti-inflammatory cytokine Induces fever
Interleukin-8	Macrophages	Mediates the innate immune response Induces fever Is angiogenic (stimulates vessel formation)
Gamma interferon	T and NK cells	Stimulates phagocytosis by neutrophils and macrocytes Activates NK cells Augments B cell proliferation, enhancing both cellular and humoral immune responses
Alpha and beta interferons	Virus-infected cells; macrophages	Activate macrophages and endothelial cells; beta interferon induces fever Augment NK cell activity Act at gene level to protect neighbouring cells from invasion by intracellular parasites, such as viruses, rickettsia, malaria
Macrophage inflammatory proteins (MP-1-4CC)	Macrophages, dendritic cells and lymphocytes	Are chemokines (CC), which are small cytokines Promote inflammatory response, chemotaxis and homeostasis (control migration of cells in maintenance and development)
Tumour necrosis factor (TNF)	Activated macrophages, T cells and NK cells	Major chemical mediator of inflammatory response Stimulates T-cell activation, antibody production and accumulation of leucocytes at inflammatory site Directly cytotoxic to some tumour cells Induces fever

Cytokines are important in amplifying innate immunity and the specific immune response. They stimulate:

- B cells to become plasma cells and produce antibodies
- attraction and activation of macrophages to become aggressive phagocytes
- proliferation of cytotoxic T cells and memory helper T cells
- cytotoxic T cells to destroy abnormal cells and pathogens.

Although T cells are activated by specific antigens, much of the resulting effect is non-specific—in other words, an enhanced inflammatory response. Like the antibody-mediated response, the cell-mediated response has memory. Subsequent exposures to an antigen result in a more rapid and effective inflammatory response and more effective phagocytosis by macrophages. This memory provides the basis for skin testing. For example. a person previously exposed to tuberculosis develops a more pronounced inflammatory response when minute amounts are injected under the skin.

The person with natural or acquired immunity

Immunity refers to the protection of the body against disease. Immunity to disease may be either natural or acquired, active or passive.

Immunity develops from the activation of the body's immune response. Depending on the antigen, antibody-mediated or cell-mediated responses are activated. The immune response typically involves components of both. In the immunocompetent (having an immune system capable of responding to pathogens and tissue damage) person, these responses inactivate and remove the antigen, allowing recovery to occur or preventing the development of disease. People with suppressed or impaired immune function are more susceptible to disease and require protection from exposure to environmental elements. Isolation techniques are employed to prevent the spread of disease and protect immunosuppressed people.

Pathophysiology

The processes of antibody-mediated and cell-mediated immunity result in the development of **active immunity**. Active immunity occurs when the body produces antibodies or develops immune lymphocytes against specific antigens. Memory cells, which can produce an immediate immune response on re-exposure to the antigen, provide long-term immunity. Active immunity can develop naturally, resulting from contact with the disease-producing antigen and subsequent development of the disease.

For many diseases, however, the potential consequences of a single disease episode for the individual and society make prevention desirable, especially for highly contagious diseases capable of causing epidemics. In these instances, immunisation, or vaccination, is used to provide artificially acquired immunity. The purpose of vaccination is to establish adequate levels of antibody and/or memory cells to provide effective immunity. Vaccination introduces the disease-producing antigen into the body in a manner that will stimulate the immune system to form antibodies and memory cells but will not produce disease. Vaccines may be made of killed organisms or of live organisms that have been attenuated or modified to reduce their disease-producing capability. Typhoid is an example of a killed organism vaccine; measles-mumps-rubella (MMR) vaccine, by contrast, is made from attenuated organisms. Many newer vaccines use subunits of the antigen; these are portions of the organism that have antigenic properties but are unable to produce disease.

Passive immunity provides temporary protection against disease-producing antigens. Antibodies produced by other people or animals are the source of passive immunity. These acquired antibodies are used up; they either combine with the antigen or they are naturally degraded by the body and their protection is gradually lost. The transfer of maternal antibodies via the placenta and breast milk to the infant provides naturally acquired passive immunity. Rabies human immune globulin and hepatitis B immune globulin (HBIG) are examples of immunisations used to provide artificially acquired passive immunity. The types of active and passive immunity are summarised in Table 11.5.

INTERPROFESSIONAL CARE

Interprofessional care is preventive, focusing primarily on assessing the person's immune status and ensuring acquired immunity to prevent disease.

Diagnosis

A number of diagnostic tests can be performed to assess the person's immune status.

- *Serum protein* measures the total protein in the blood, including albumin and globulins. Normal levels for the adult are 62 to 80 g/L; albumin is approximately 60% (32 to 45 g/L) of the total serum protein; and globulins are normally 23 to 34 g/dL. Total protein levels, albumin and globulin are decreased in malnutrition and liver disease. Decreased globulin levels are noted with immunological deficiencies.

TABLE 11.5 Types of acquired immunity

TYPE OF IMMUNITY		HOW DEVELOPED	EXAMPLES
Active immunity	Natural	Acquired by infection with an antigen, resulting in the production of antibodies	Chickenpox
	Artificial	Acquired by immunisation with an antigen, such as attenuated live virus vaccine	MMR, polio, DTP, hepatitis B vaccine
Passive immunity	Natural	Acquired by transfer of maternal antibodies to the foetus or neonate via the placenta or breast milk	Neonate initially protected against MMR if mother immune
	Artificial	Acquired by administration of antibodies or antitoxins in immune globulin	Gamma globulin injection following hepatitis A exposure

- *Protein electrophoresis* analyses protein content, especially for albumin and gamma globulin, and is used to assess immune function. Gamma globulins subjected to further electrophoresis separate into immunoglobulins: IgA, IgD, IgE, IgG and IgM (see Table 11.3). Analysis of specific levels of each provides clues about the immune status of the person. IgG levels are increased during acute infection. Decreased levels of IgG, IgA and IgM are found in malignancies.
- *Antibody testing* is ordered to determine if a person has developed antibodies in response to an infection or immunisation. Antibodies for hepatitis, HIV, rubella, varicella (chickenpox) and certain other diseases can be identified. An elevated titre level for varicella and rubella indicates immunity. Antibody testing may also be used to determine if the person has the disease.
- *Skin testing* can assess cell-mediated immunity. A known antigen such as streptokinase, tuberculin-purified protein derivative or *Candida* protein is injected intradermally. The site is then observed for induration and erythema, which typically peak at 24 to 48 hours. An induration of at least 10 mm in diameter is a positive reaction, indicating previous exposure and sensitisation to the antigen. No reaction to common antigens, or **anergy**, indicates depressed cell-mediated immunity.

Immunisations

Vaccines are suspensions of whole or fractionated bacteria or viruses that have been treated to make them non-pathogenic. Vaccines are given to induce an immune response and subsequent immunity. Although vaccine development has been a major factor in improving public health, no vaccine is completely effective or entirely safe. Table 11.6 outlines the vaccines recommended for the adult person to maintain optimal health and immune status (Centers for Disease Control and Prevention (CDC), 2014).

Adults born before 1966 are generally considered to be immune to measles, mumps and rubella by prior infection. Individuals born after 1966 should have documentation of one or more doses of MMR vaccine unless they have a medical contraindication to the vaccine or laboratory evidence of immunity to the three diseases (CDC, 2014).

Vaccination against diphtheria, tetanus and pertussis (DTP) is part of the National Immunisation Program (NIP) schedule. A combined vaccine is administered in a series of injections. The paediatric form of the vaccine is known as DTPa, while the adult

TABLE 11.6 Recommended immunisations for adults

VACCINE	TYPE	DOSE	INDICATIONS	PRECAUTIONS AND NURSING IMPLICATIONS
Measles-mumps-rubella (MMR)	Live virus	0.5 mL SC	All adults born after 1966, particularly those who are at risk of infection, such as healthcare workers, those working with children, and overseas travellers. MMR vaccination is particularly recommended for males without history of previous infection; rubella vaccination recommended for all seronegative females.	As a live virus vaccine, should not be administered to pregnant women or immunocompromised people. Do not administer to people with a history of anaphylactic reaction to egg protein or neomycin.
Diphtheria, tetanus and pertussis toxoids (dTpa)	Inactivated toxins	0.5 mL IM	Initial series of 3 injections (minimum intervals of 4 weeks) if never immunised then booster at 10 and 20 years; adults who reach 50 and have not had booster in previous 10 years; following a major or contaminated wound if more than 5 years since last booster.	Do not give in first trimester of pregnancy or to people with a history of anaphylactic reaction to previous dose or any vaccine component; administer deep IM in deltoid of dominant arm.
Hepatitis B (HB)	Inactive viral antigen	1.0 mL IM	Series of 3 doses: initial then at 1 to 2 months, then 2 to 5 months after second dose. Recommended for anyone at risk of exposure and for postexposure prophylaxis.	Use with caution in pregnant or lactating females, older people and people with active infection.
Influenza	Inactivated virus or viral components	0.5 mL IM	Yearly for all people > 6 months of age; strongly recommended for over age 65, all Aboriginal and Torres Strait Islander people > 15 years of age and those with predisposing conditions to severe influenza, including debilitated people and people with chronic disease.	Do not administer to people who are acutely ill or people with history of anaphylactic reaction to egg protein.
Pneumococcal	Bacterial polysaccharides	0.5 mL IM or SC	One dose for people over age 65, and Aboriginal and Torres Strait Islander people > 50 years of age; and those aged > 10 who are at risk of pneumococcal pneumonia, including people with chronic lung disease or other chronic diseases.	Do not administer to pregnant women.

form is dTpa. The vaccine stimulates active immunity by inducing the production of antibodies and antitoxins. After an initial series of a three-dose primary schedule as an infant, a booster dose is given around 4 years of age. A recent update of the *Immunisation Handbook* (Department of Health and Ageing and National Health and Medical Research Council (NHMRC), 2013) in March 2015 recommends a further booster at 18 months of age in addition to 4 years of age. A further booster dose is recommended for adolescents around 11 to 13 years. It is also recommended that pregnant women in the third trimester of each pregnancy receive a dose of dTpa. Adults who reach 50 years of age without having a booster dose in the prior 10 years should receive a booster dose.

Hepatitis B (HB) vaccine is given as a series of three immunisations to promote active immunity to hepatitis B. This vaccine is recommended for everyone at high risk of exposure through blood or other body fluids, including healthcare workers, and emergency and essential service workers. Other high-risk populations include intravenous drug users, sexual partners of infected individuals, people on haemodialysis, prison guards, tattooists, body piercers and embalmers. A universal hepatitis B vaccination program was recommended for infants and adolescents in 1996. The adolescent program commenced in 1997 and the universal infant program, with the first dose given at birth, began nationally in 2000.

Influenza vaccine is recommended annually for all adults. The antigenic strains included in the influenza vaccine vary each year according to the predicted predominant strains affecting the population; therefore, yearly re-immunisation is required.

Pneumococcal vaccine is recommended for all children and is included in the infant immunisation program.

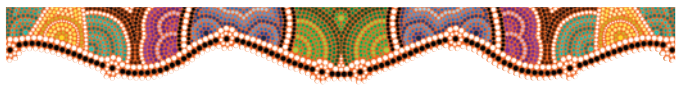
Indigenous Australians historically have had a very high rate of infectious diseases. In the early period of European colonisation, this was mainly due to a lack of previous exposure, followed by high-density living in newly established settlements. In recent decades, for some diseases such as diphtheria, polio, tetanus, hepatitis B, measles, mumps and rubella, vaccination has been very successful in eliminating or substantially reducing disease in all Australians, significantly contributing to reducing child mortality in Indigenous Australian populations. However, other diseases such as pneumococcal disease and influenza have a higher rate of illness in Indigenous compared to non-Indigenous people and therefore are specifically recommended for administration of vaccine to a broader age group.

In addition to routine immunisations, people travelling outside Australia should receive vaccines against diseases that are endemic in the regions of the world that they intend to visit.

Other immunological substances may be administered as indicated. Immunoglobulins provide passive immunity as protection against a known or potential exposure to an antigen. Normal human immunoglobulin is given to household contacts of people with hepatitis A and people travelling to areas in which it is endemic. HB immunoglobulin contains higher titres of antibody to hepatitis B virus and is used for people exposed by blood or sexual contact. Following confirmed or suspected contact with a pathogen, selected vaccines may be administered to stimulate an immediate immune response.

For most vaccines, a sensitivity test should be performed prior to administration to detect sensitivity to substances such as horse serum or eggs. The substance is injected intradermally; if after 20 minutes there is no evidence of a reaction, the selected vaccine can be administered.

Moderate to severe local reactions may occur following administration of an immunisation. Common reactions include redness, swelling, tenderness and muscle ache. Administering the vaccine in the dominant arm of the person helps minimise local reactions because use and movement of the arm facilitates absorption of the solution. Applying heat to the site is also beneficial. Occasionally local ulcerations occur; when they do, warm wet packs or sterile wet-to-dry dressings may be prescribed.



Nursing care

Maintaining a population that is fully immunised against common, potentially epidemic and devastating diseases is a major public health task for nursing. Nurses not only recommend and administer vaccines to individual people and their families, but also plan and implement preventive care for whole communities.

Although this process may appear to be straightforward, multiple issues affect society's ability to immunise the entire population. For some people, for example, religious beliefs may preclude the use of immunisations to prevent disease. Also, people who are not citizens and the medically indigent population have difficulty accessing immunisation services. Lack of immunisation not only puts the individual at increased risk of infectious disease, but also increases the cost of medical services and the possibility of exposing immunocompromised people to disease.

Health promotion

In the public health setting, the nurse looks at the immunisation needs and illness risk of an entire community. Communities include not only cities and localities, but also groups of people, such as university populations and employees in a workplace. Public education needs may be met through presentations to groups of people, feature articles in newspapers and other local publications, advertising, radio presentations, public service announcements, one-to-one discussion and teaching.

Assessment

Collect the following data through the health history and physical examination. Further focused assessments are described with nursing interventions in the next section.

- *Health history*: age, medication use (corticosteroids and antibiotics) and blood transfusion, nutrition, known allergies, pregnancy status, infection, immunisations, autoimmune disorders, chronic diseases such as asthma, diabetes mellitus, cancer.
- *Physical assessment*: skin lesions or rashes, breath sounds, respiratory rate.

Nursing diagnoses and interventions

Nursing care focuses on preventing injury from the immunisation and educating the person. See the accompanying 'Nursing care plan' box.

CONSIDERATION FOR PRACTICE

Observe the person for 20 to 30 minutes following vaccine administration to monitor for possible adverse reactions.

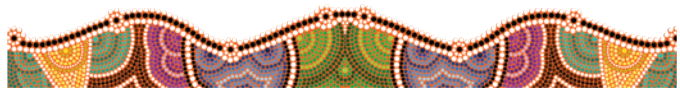
Readiness for enhanced immunisation status

For individual people and their families, nurses promote immunocompetence by assessing immune status, recommending appropriate immunisations and administering vaccines as ordered or indicated. Once a person reaches adulthood, routine immunisations often become a neglected part of healthcare.

- Determine knowledge level, understanding, attitudes and religious beliefs about immunisation. *This provides a basis for further education and determines if religious beliefs may contraindicate immunisation.*
- Discuss the value and reasons for recommended immunisations. *Understanding promotes adherence.*
- Reinforce positive health-seeking behaviours. *This will help promote future health maintenance activities.*
- Using recommended immunisation schedules, develop a plan to attain optimal immunisation status. *Adherence with recommended schedules for immunisation is important in preventing disease and disability.*
- Do not administer MMR or influenza vaccine if allergic to eggs, or tetanus antitoxin if sensitive to horse serum. *Vaccines prepared from chicken or duck embryos are contraindicated in people who are allergic to eggs. Tetanus antitoxin is prepared from horse serum. Both will cause a severe allergic reaction.*
- Withhold administration of active immunological products in the presence of an upper respiratory infection or other infection. *Active immunisations can cause a greater inflammatory reaction in the presence of infections.*
- Do not administer oral polio vaccine, MMR or any live virus vaccine to immunosuppressed people or to those who are in close household contact with an immunosuppressed person. *Live virus vaccines can cause disease in the immunosuppressed person. The virus may be transmitted from close household contacts during the initial post-vaccination period.*
- Do not administer vaccines such as MMR, pneumococcal or varicella to women who are pregnant. *Although the risk to the developing foetus is greatest during the first trimester, these vaccines are avoided throughout pregnancy.*
- Do not administer live attenuated virus vaccines and passive immunisations such as gamma globulin simultaneously. *Passive antibodies interfere with the response of the live attenuated virus.*
- Prior to administering the prescribed vaccine, check the expiration date and manufacturer's instructions. *Outdated vaccines cannot provide adequate immunisation protection. Certain injection sites have better absorption than others.*
- Keep adrenaline 1:1000 readily available for subcutaneous injection when administering immunisations. *Adrenaline causes vasoconstriction and reduces laryngospasm; in acute anaphylaxis, it can be lifesaving.*

Community-based care

Clinically significant medical events including high fever, injection-site hypersensitivity, unspecified rash and injection-site oedema that occur after vaccination should be reported as an adverse event following immunisation (AEFI). Reporting an AEFI is important as it provides a better understanding of the safety issues around vaccines. The ongoing reporting of adverse events following immunisation allows the Therapeutic Goods Administration (TGA) to monitor rates and trends across Australia and assist in identifying issues such as incorrect vaccine administration, manufacture, storage and delivery.



NORMAL IMMUNE RESPONSES

The person with tissue inflammation

Inflammation is a non-specific response to injury that serves to destroy, dilute or contain the injurious agent or damaged tissue. Acute inflammation is a short-term reaction of the body to all types of tissue damage. It is immediate and aimed at protecting the body and preventing further invasion or injury. Acute inflammation usually lasts less than 1 to 2 weeks. Once the injurious agent is removed, the inflammation subsides. Healing with tissue repair or scar formation occurs and the body functions in normal or near-normal capacity.

Chronic inflammation is slower in onset and may not have an acute phase. Its clinical manifestations occur over months or years. While the effects of some chronic inflammatory processes may be evident (such as the joint damage and destruction associated with rheumatoid arthritis), the role chronic inflammation plays in diseases such as asthma, obesity and heart disease has only recently been recognised.

Pathophysiology and manifestations

The tissue damage that evokes an inflammatory response may be caused by specific or non-specific agents. These agents may be *exogenous*, from outside the body, or *endogenous*, from within the body. Causes of inflammation include the following:

- mechanical injuries such as cuts or surgical incisions
- physical damage such as burns
- chemical injury from toxins or poisons
- microorganisms such as bacteria, viruses or fungi
- extremes of heat or cold
- immunological responses, such as hypersensitivity reactions
- ischaemic damage or trauma, such as a stroke or myocardial infarction.

Acute inflammation

Regardless of the cause, location or extent of the injury, the acute inflammatory response follows the previously outlined sequence of vascular response, cellular and phagocytic response, and healing.

Many of the manifestations of inflammation are produced by inflammatory mediators, such as histamine and prostaglandins, released when tissue is damaged (see Box 11.1).

The primary manifestations of inflammation include the following:

- erythema (redness)
- local heat caused by the increased blood flow to the injured area (hyperaemia)

NURSING CARE PLAN A person with acquired immunity

Terry Adams is a 48-year-old executive who is planning a trip to central Africa. In preparation, he contacts his local healthcare provider to obtain the necessary immunisations. Jane Wong, the Registered Nurse in the clinic, obtains a nursing history of Mr Adams.

ASSESSMENT

Mr Adams' history reveals that he has always been very healthy and active, apart from a mild case of asthma. As an adult he has had little problem with his asthma, 'except for those rare occasions when I am dumb enough to smoke more than one cigarette!'. He is divorced and is not currently in a continuing relationship. He has two grown daughters with whom he has a good relationship. Since contracting hepatitis A several years ago, he drinks alcohol only rarely and never more than one or two drinks at any one time. He confesses to doing little organised exercise, but plays golf two or three times a week. He says he is such a hyperactive workaholic that he rarely sits for any length of time. Mr Adams has not seen a physician since recovering from the hepatitis and is unsure when he last received any immunisations. He does not know if he had all the recommended childhood immunisations, but recalls getting both Salk and Sabin polio vaccines when they became available. His physical examination reveals an alert and healthy individual with no abnormalities noted. His vital signs are as follows: T 37.1°C, P 64, R 14 and BP 142/82.

The physician orders the following immunisations for Mr Adams:

- measles-mumps-rubella (MMR)
- combined tetanus, diphtheria and pertussis toxoids (dTpa)
- yellow fever vaccine
- typhoid vaccine
- meningococcal meningitis vaccine.

DIAGNOSES

- *Health-seeking behaviours: immunisation* related to impending international travel.
- *Ineffective health maintenance* related to apparent lapse in immunisation status.
- *Risk of injury* related to adverse response to immunisation.

PLANNING

- Obtain MMR, dTpa and meningococcal meningitis vaccines for administration.
- Schedule return visit in 1 week for typhoid vaccine.
- Refer Mr Adams to a registered vaccination centre for yellow fever vaccine.
- Ensure instructions are provided for comfort measures to relieve local and systemic adverse effects of vaccines and manifestations that should be reported to the physician.
- Complete documentation of immunisations on a permanent record at the clinic and for Mr Adams.
- Provide information for ongoing requirements to maintain immunisation status.

Expected outcomes

- Complete necessary immunisations.
- Verbalise a schedule for maintaining up-to-date immunisation status.
- Experience no significant adverse effects from immunisation.

IMPLEMENTATION

- Administer MMR, dTpa and meningococcal meningitis vaccines prior to discharge from clinic.
- Observe closely for 30 minutes following immunisation for potential adverse responses.
- Schedule return visit in 1 week for typhoid vaccine.
- Provide referral to a registered vaccination centre for yellow fever vaccine and documentation of vaccination.
- Provide instructions for comfort measures to relieve local and systemic adverse effects of vaccines.
- Provide written instructions on signs and symptoms that should be reported to the physician.
- Document immunisations on a permanent record at the clinic and for Mr Adams.
- Discuss ongoing requirements to maintain immunisation status.

EVALUATION

Terry Adams completes his prescribed immunisations without major adverse effects, although he does complain of fever, malaise and general achiness for several days following the typhoid vaccination. His trip to Africa is successful and he returns to Australia without contracting any infectious diseases.

CRITICAL THINKING IN THE NURSING PROCESS

1. Explain why it is important for adults to continue receiving immunisations throughout their lifespan.
2. If a person says to you, 'I don't believe in immunisations. I hear they are dangerous', how would you respond?
3. When should a person contact the primary caregiver after receiving an immunisation?

REFLECTION ON THE NURSING PROCESS

1. What sources of knowledge or evidence influenced the decision making in this case study?
2. Outline what you have learned from this case study that you could apply in your future practices.

- swelling due to accumulated fluid at the site
- pain from tissue swelling and chemical irritation of nerve endings
- loss of function caused by the swelling and pain.

The degree of functional loss depends on the location and extent of the injury. With increased tissue damage, more fluid exudate is formed, resulting in more swelling, pain and functional impairment. Pain may be immediate or delayed. Prostaglandins intensify and prolong the pain. Kinins cause irritation to the nerve endings and contribute to the pain sensation.

Dead neutrophils, necrotic tissue and, if the tissue is infected, digested bacteria accumulate as a result of inflammation and phagocytosis, forming *pus*. Pus usually forms and remains until after the infection subsides. Pus may push itself to the surface of the body or become internalised. In the latter case, pus is gradually autolysed (self-digested) by enzymes

over a period of days. The end product is then absorbed by the body. On occasion, pus may remain after the infection is resolved. An abscess, or localised collection of pus, may form, necessitating drainage by a surgical procedure.

Systemic responses to inflammation include lymph node swelling (*lymphadenopathy*) due to the proliferation of macrophages within the nodes in response to microorganisms in the lymph. Enlarged lymph nodes are usually noted in the groin, axillae and neck, Fever, often precipitated by inflammatory mediators or bacterial toxins, inhibits the growth of many microorganisms and increases tissue repair functions. Loss of appetite and fatigue may occur in the effort to conserve energy during the inflammatory process. Leucocytosis occurs with increased WBC production to support inflammation and phagocytosis.

Chronic inflammation

Whereas acute inflammation is a self-limiting process lasting less than 2 weeks, chronic inflammation tends to be self-perpetuating, lasting weeks to months or years. Chronic inflammation may develop when the acute inflammatory process has been ineffective in removing the offending agent. Persistent low-grade fever or irritation by chemicals, particulate matter or physical irritants such as talc, asbestos or silica may also result in chronic inflammation.

GRANULOMATOUS INFLAMMATION *Granulomatous inflammation* is characterised by dense infiltration of the site by lymphocytes and macrophages. The macrophages mass to surround the site; they in turn are surrounded by lymphocytes and other immune cells, forming a lesion called a *granuloma*. The granuloma isolates the offending agent from the rest of the body; however, the infectious agent or irritant may not be destroyed and can survive within the granuloma for a long period of time. Chronic inflammation and granuloma formation are common with *Mycobacterium tuberculosis*. The granuloma formed in tuberculosis is called a tubercle. *M. tuberculosis* can survive for many years within the tubercle, emerging when the person's immune system is no longer able to contain it.

NON-SPECIFIC CHRONIC INFLAMMATION *Non-specific chronic inflammation* is implicated in such disorders as pulmonary airway disease (asthma and chronic obstructive lung disease), cardiovascular disease and autoimmune disorders such as inflammatory bowel disease (Reilkoff et al., 2011). This type of chronic inflammation is characterised by diffuse accumulation of macrophages and proliferation of fibroblasts in response to ongoing chemotaxis. The fibroblasts and fibrocytes, unique cells formed from monocytes, lead to formation of excessive fibrous connective tissue, disrupting normal tissue functions (Reilkoff et al., 2011). Inflammatory markers such as C-reactive protein, complement fractions and inflammatory cytokines are present in non-specific chronic inflammation and associated disorders (Bartunek & Vanderheyden, 2012: Engström, 2011; Pellizzaro & Heuertz, 2010; Strowig et al., 2012).

Complications

Inflammation and wound healing are highly metabolic processes that may be affected by a number of factors. Without adequate nutrition, blood supply and oxygenation, tissues cannot effectively complete the process. Impaired inflammatory and immune processes can interfere with phagocytosis and preparation of the wound for healing. Infection prolongs the inflammatory process and delays healing.

Chronic diseases may also impair healing. High blood glucose levels and small-blood-vessel disease associated with diabetes mellitus impair chemotactic and phagocytic function. Collagen formation and tensile strength of the wound are also impaired. Arterial and venous disorders impair the delivery of oxygen and nutrients to healing tissues, as well as the removal of toxins, bacteria and other waste products from the area. Drug therapy, particularly corticosteroid medications, may suppress the immune and inflammatory responses, delaying healing (Porth & Matfin, 2009). Other external factors, such as exposure to ionising radiation and wound cleansing agents, can also affect healing. Table 11.7 summarises major factors that affect the inflammatory process and wound healing.

TABLE 11.7 Factors that may impair healing

FACTOR	EFFECT
Malnutrition	
Protein deficit	Prolongs inflammation and impairs healing process
Carbohydrate and kilocalorie deficit	Impairs metabolic processes and promotes catabolism; proteins are used for energy rather than for healing
Fat deficit	Impairs cell membrane synthesis in tissue repair
Vitamin deficiencies	
Vitamin A	Limits epithelialisation and capillary formation
B-complex	Inhibits enzymatic reactions that contribute to wound healing
Vitamin C	Impairs collagen synthesis
Tissue hypoxia	Associated with an increased risk of infection and impaired healing because oxygen is required to support cell function and collagen synthesis
Impaired blood supply	Results in inadequate delivery of oxygen and nutrients to healing tissues and removal of waste products
Impaired inflammatory and immune processes	Results in decreased phagocytosis and wound debridement; increased risk of infection; delayed healing

INTERPROFESSIONAL CARE

Management of the person with inflamed tissue focuses on promoting healing. Care is generally supportive, allowing the person's own physiological processes to remove foreign matter and damaged cells. Wound care may be minimal, involving only simple cleaning, or may require irrigations and debridement. The person is encouraged to rest, to increase fluid intake and to eat a well-balanced, nutritious diet. Antibiotics may be prescribed to help eliminate infectious causes of inflammation.

Diagnosis

The following diagnostic tests may be ordered to identify the source and extent of inflammation.

- *WCC with differential* provides information about the type and extent of inflammatory response. The differential count (the percentage of the total WBC made up by each type of leucocyte) provides further clues about inflammatory processes (see Table 11.8).
- *Erythrocyte sedimentation rate (ESR)* is a non-specific test to detect inflammation. The rate at which RBCs fall to the bottom of a vertical tube is an indicator of inflammation. An increased ESR may indicate acute or chronic inflammation.
- *C-reactive protein (CRP) test* is used to detect CRP. This abnormal glycoprotein is produced by the liver and is excreted into the bloodstream during the acute phase of an inflammatory process. The expected result of this test is negative for CRP. A positive result indicates an acute or chronic inflammatory process.

In addition to the above diagnostic tests, cultures of the blood and other body fluids may be ordered to determine if infection is the cause of inflammation.

Medications

Although inflammation is a beneficial process to prepare acutely injured tissue for healing, its manifestations can be distressing. Chronic inflammation can lead to tissue damage and scarring with resulting loss of function. Anti-inflammatory medications may be prescribed to manage these effects. Anti-inflammatory medications fall into three broad groups: salicylates such as aspirin, other NSAIDs and corticosteroids.

Aspirin (acetylsalicylic acid or ASA) is a NSAID with antipyretic, analgesic and antiplatelet effects. Its beneficial effects are largely dose related. Low doses (as little as 75 mg/day) inhibit platelet aggregation and normal blood clotting. A 600 mg dose of aspirin is an effective analgesic and antipyretic dosage. Higher doses (600 to 900 mg, 4 to 5 times per day) are required to produce its anti-inflammatory effects. To relieve pain, aspirin acts primarily on peripheral sensory nerves by inhibiting the synthesis of prostaglandins and kinins, which are chemical stimuli of sensory nerves. As an antipyretic, aspirin acts both centrally and peripherally. It inhibits the formation of pyrogenic substances that raise the hypothalamic thermostat. It also dilates peripheral blood vessels and promotes diaphoresis, increasing the dissipation of heat (Adams, Holland & Urban, 2014).

In therapeutic doses, aspirin mediates the inflammatory process by inhibiting the enzyme cyclooxygenase (COX) and preventing synthesis of prostaglandins. Inflammation is reduced, along with the swelling, redness and impaired function that accompanies it.

The other NSAIDs have activity similar to that of aspirin. They inhibit COX and prostaglandin synthesis, reducing the inflammatory and pain responses. Each NSAID has a slightly different mode of action; sometimes several different agents must be tried before the most effective is identified. Side effects also differ to a certain extent; however, all have a potential

TABLE 11.8 The white blood cell count and differential

CELL TYPE AND NORMAL VALUE	INCREASED	DECREASED
Total WBCs: 4.5–13.5 × 10^9/L	*Leucocytosis:* infection or inflammation, leukaemia, trauma or stress, tissue necrosis	*Leucopenia:* bone marrow depression, overwhelming infection, viral infections, immunosuppression, autoimmune disease, dietary deficiency
Neutrophils (segs, PMNs or polys): 2.0–7.5 × 10^9/L	*Neutrophilia:* acute infection or stress response, myelocytic leukaemia, inflammatory or metabolic disorders	*Neutropenia:* bone marrow depression, overwhelming bacterial infection, viral infection, Addison's disease
Eosinophils (eos): 0.04–0.4 × 10^9/L	*Eosinophilia:* parasitic infections, hypersensitivity reactions, autoimmune disorders	*Eosinopenia:* Cushing's syndrome, autoimmune disorders, stress, certain drugs
Basophils (basos): < 0.1 × 10^9/L	*Basophilia:* hypersensitivity responses, chronic myelogenous leukaemia, chickenpox or smallpox, splenectomy, hypothyroidism	*Basopenia:* acute stress or hypersensitivity reactions, hyperthyroidism
Monocytes (monos): 0.2–0.8 × 10^9/L	*Monocytosis:* chronic inflammatory disorders, tuberculosis, viral infections, leukaemia, Hodgkin's disease, multiple myeloma	*Monocytopenia:* bone marrow depression, corticosteroid therapy
Lymphocytes (lymphs): 1.5–4.0 × 10^9/L	*Lymphocytosis:* chronic bacterial infection, viral infections, lymphocytic leukaemia	*Lymphocytopenia:* bone marrow depression, immunodeficiency, leukaemia, Cushing's syndrome, Hodgkin's disease, renal failure

cross-sensitivity with aspirin, all irritate the gastrointestinal tract and all are associated with an increased risk of cardiovascular events. NSAIDs are more costly than aspirin, but they have a longer duration of action; therefore, fewer daily doses are required to achieve the desired effect.

For acute hypersensitivity reactions, or for inflammation that cannot be managed using NSAIDs, corticosteroid therapy may be prescribed. The glucocorticoids are hormones produced by the adrenal cortex that have widespread effects on body metabolism and the immune response. Glucocorticoids inhibit inflammation and may be lifesaving in acute fulminating or chronic progressive inflammation. When glucocorticoids are prescribed to manage inflammation, the smallest possible effective dose is used. Wherever possible, a local-acting preparation such as a topical agent, metered-dose inhaler or intra-articular injection is prescribed to minimise systemic effects of the drug.

The incidence of potentially harmful side effects increases with higher doses and prolonged therapy. Wound healing is impaired and the metabolism of fats, proteins and carbohydrates is altered. Blood glucose control is impaired. Fat distribution changes, producing a cushingoid appearance with a moon face and increased truncal fat. Fluid retention and hypertension are potential problems, as are osteoporosis, gastrointestinal bleeding and emotional disturbances.

Paracetamol may be administered to reduce the fever and pain associated with inflammation. Paracetamol has no anti-inflammatory effect, and will not reduce the inflammatory process, but can relieve associated symptoms such as fever and pain.

Antibiotics may be used either prophylactically to prevent infection from interfering with the healing process of damaged tissue, or therapeutically to treat the infection. If infection is present, the organism and its response or sensitivity to various antibiotics is used to guide therapy. Antibiotic therapy is presented in greater depth in the section of this chapter on infectious diseases.

Nutrition

Healing depends on cell replication, protein synthesis and the function of specific organs—the liver, heart and lungs, in particular. Malnutrition and protein depletion are risk factors for poor healing and wound complications. Even a few days of severely impaired nutritional intake can noticeably affect healing (Tucker & Dauffenbach, 2011).

The person with an inflammatory process or healing wound requires a well-balanced diet of sufficient kilojoules to meet the metabolic needs of the body (see Table 11.7). Inflammation often produces *catabolism*, a state in which body tissues are broken down. By contrast, healing is a process of *anabolism*, or building up. Without sufficient kilojoules and nutrients, catabolism may predominate, impairing healing.

Adequate protein is necessary for tissue healing and the production of antibodies and WBCs. Lack of adequate protein increases the risk of infection. Complete protein sources, those that provide the essential amino acids, are preferred. Carbohydrates are important to meet energy demands, as well as to support leucocyte function. Care is taken to avoid hyperglycaemia in persons with diabetes, as hyperglycaemia interferes with oxygen delivery to the tissues as well as with the chemotactic and phagocytic function of neutrophils, impairing healing. Dietary fats are used in the synthesis of cell membranes.

Vitamins A, B-complex, C and K are also important to the healing process. Vitamin A is necessary for capillary formation and epithelialisation. B-complex vitamins promote wound healing, and vitamin C is necessary for collagen synthesis. Vitamin K provides a vital component for the synthesis of clotting factors in the liver.

Although it has been established that minerals contribute to the inflammatory and healing processes, less is known about required amounts. Minerals serve important roles in maintaining normal cell function and as cofactors in enzyme reactions necessary for cell proliferations. Zinc, a micronutrient, is involved in cell growth and in T-cell development (Tucker & Dauffenbach, 2011).

Oxygen is another important element in healing. It is necessary for collagen synthesis. Phagocytes such as neutrophils and macrophages require oxygen to digest bacteria engulfed in the phagocytic process. Impaired oxygen delivery to the tissues slows healing and increases the risk of infections. Supplemental oxygen administered via nasal cannula or mask improves the oxygen saturation of haemoglobin and its availability to tissues. Hyperbaric oxygen delivery improves leucocyte and fibroblast function as well as the development of new blood vessels, and may be beneficial to promote healing of inflamed ischaemic tissue (Bennett & Mitchell, 2012).

Nursing care

Acute inflammation may be self-limiting, or extensive and require hospitalisation. Nursing care includes teaching people with acute and chronic inflammatory conditions self-management at home.

Health promotion

Health promotion activities to prevent inflammation focus on reducing the risk of accidents and exposure to harmful agents that can result in subsequent injury. It is important to educate the public about potential hazards in both the work and home environments. In addition, safety education guidelines, such as not drinking and driving, wearing a protective helmet when riding a bicycle and using a seat belt in the car, are important areas for discussion. Because most injuries occur at home, it is also important to discuss ways to make the home safer.

Assessment

The following data are collected through the health history and physical examination. Further focused assessments are described with nursing interventions in the next section.

- *Health history*: risk factors, nutrition, medication use (anti-inflammatory and corticosteroids), location, duration and type (redness, heat, pain, swelling and impaired function) of symptoms.
- *Physical assessment*: movement of injured area, circulation, wounds, lymph nodes.

Nursing diagnoses and interventions

The nursing care needs of the person with an inflammatory process are related to the manifestations of inflammation and resulting altered tissue integrity. Nursing care priorities focus on relieving pain, supporting tissue healing and preventing infection.

Acute pain

Along with redness, warmth, swelling and impaired function, pain is one of the primary manifestations of inflammation. Depending on the cause, affected area and degree of inflammation, pain may be acute and immobilising or chronic and demoralising. It is important to remember that pain is a subjective experience and that people's responses to pain vary.

- Assess pain using a scale of 0 to 10, with 0 being no pain and 10 being the worst pain; note the character and location of the pain. *Because pain is subjective, the person provides the most accurate information regarding their pain experience.*
- Use physical and non-verbal cues to further assess the level of pain. *This intervention is especially important if the person is non-verbal or tends to under-report pain.*
- Administer anti-inflammatory medications as prescribed. *These medications help reduce the pain resulting from acute inflammation. Most NSAIDs also have analgesic and antipyretic effects, further promoting comfort* (Adams et al., 2014).
- Administer analgesic medications as prescribed. *Moderate to severe pain may require treatment with an analgesic (e.g. opioid). Paracetamol and opioid analgesics act within the CNS to reduce pain. Opioids provide the most effective pain relief overall, activating pain inhibitory neurons and inhibiting pain transmission neurons* (Rathmell & Fields, 2012).

CONSIDERATION FOR PRACTICE

Because opioids can depress respirations, it is important to monitor oxygen saturation and encourage the person to take deep breaths to maintain adequate oxygen saturation.

- Provide comfort measures, such as back rubs, position changes or relaxation techniques. *These measures reduce muscle tension, relieve areas of pressure and provide distraction.*
- Encourage activities such as reading, watching television and taking part in social interactions. *Such activities provide distraction from the pain experience.*
- Encourage rest. *Strenuous activity or exercising an inflamed body part may increase discomfort and tissue damage.*
- Provide cold or heat as pain relief measures as ordered. *For an acute injury, cold reduces swelling and relieves pain; after the initial stage, heat increases blood flow to the affected tissue and relieves pain and swelling by promoting absorption of oedema. Do not apply either heat or cold for more than 20 minutes at a time and ensure there is a covering between the skin and the application.*
- Elevate the inflamed area if possible. *Elevation promotes venous return and reduces swelling.*
- Teach about the appropriate use and expected effects of anti-inflammatory medications. *If the person's pain continues after the initial doses of anti-inflammatory medication, they may become discouraged and stop taking the medication before it becomes fully effective.*

CONSIDERATION FOR PRACTICE

Use heat or cold application cautiously in older people who have fragile skin and are at risk of tissue injury.

Impaired tissue integrity

The inflammatory response can either precipitate or result from an impairment in the integrity of skin, support or other tissues.

- Assess general health and nutritional status. *Poor general health or chronic diseases such as diabetes mellitus or renal failure interfere with the healing processes and increase the risk of infection.*
- Assess circulation to the affected area. *Adequate tissue perfusion and oxygenation are necessary for healing* (Grossman & Porth, 2014).
- Monitor the skin and surrounding tissue for increased signs of inflammation. *Inflammation can spread to adjacent tissues, leading to conditions such as cellulitis.*
- Provide protection and support for inflamed tissue. *This reduces discomfort and decreases the risk of further tissue damage.*
- Clean inflamed tissue gently; if possible, use water, normal saline or non-toxic wound cleansers only. *Soap and harsh cleansers such as povidone-iodine (Betadine) and hydrogen peroxide can cause further drying and tissue damage. Granulation tissue in a healing wound is fragile and easily damaged.*
- Keep the inflamed area dry and expose it to air as much as possible. *This promotes healing and helps prevent infection.*
- Balance rest with activity. *Rest decreases metabolic demands and allows for cell regeneration, while mobility helps to promote oxygenation and perfusion of the tissues.*
- Provide supplemental oxygen as ordered. *Supplemental oxygen improves tissue oxygenation and reduces hypoxia.*
- Provide a well-balanced diet with adequate kilojoules to meet the body's metabolic and healing needs. If the person is allowed nothing by mouth (NBM), suggest parenteral or enteral nutrition. For the person who is unable to consume an adequate diet, consult with a dietitian for between-meal supplements and/or multivitamin supplements. *Careful attention to diet and nutrient intake is important to provide the nutrients necessary for immune function and healing and to prevent catabolism* (Tucker & Dauffenbach, 2011).

Risk of infection

The inflammatory response often indicates that body defence mechanisms have been set in motion to protect against invading microorganisms. The person with a healing wound is at particular risk of infection.

- Assess the wound for specific signs of infection, including purulent drainage, odour and delayed healing. *The normal inflammatory response can indicate infection and, on occasion, mask its presence.*
- Evaluate complete blood counts for adequate WBC response. *Leucocytosis may indicate infection or healthy*

response to injury and protection from infection. Immune-impaired people may not respond with increased WBCs, and manifestations of inflammation may be diminished in those individuals.

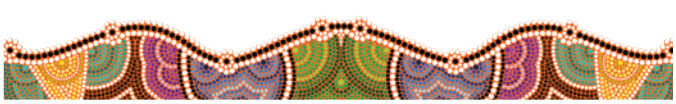
- Monitor vital signs at least every 4 hours. *In response to the inflammatory process the temperature rises, usually in the range 37.4ºC–38.2ºC. A temperature of 38.3ºC or above usually indicates infection. Fever is usually accompanied by increased heart and respiratory rates.*
- Culture purulent or odorous wound drainage. *Wound culture is used to determine the infectious organism and to direct antibiotic therapy.*
- Apply dry or moist heat to the affected area for no longer than 20 minutes several times a day. Monitor the temperature closely to prevent burns and further damage to the affected area. *Heat increases the circulation of blood to and from the inflamed tissue. Time is limited to prevent burns.*
- Provide and encourage fluid intake of 2500 mL/day as allowed. *Adequate hydration promotes blood flow and nutrient supply to the tissues and also dilutes and removes waste products from the body.*
- Ensure adequate nutrition. *Adequate nutrition enhances the function and production of T cells and B cells, which are important in the immune response.*
- Use good hand hygiene techniques consistently. *Hand hygiene removes transient microorganisms and is the best mechanism to prevent the spread of infection to a susceptible person.*
- Use aseptic technique when providing wound care. *Using sterile gloves and aseptic technique helps prevent further contamination of the wound and the spread of infection to other people.*

Community-based care

Education for the person and their family enhances understanding of the inflammatory process, its cause and its management. Teaching is also important to prevent further compromise that could result in infection.

Instructions, verbal and written, should include the following:

- Increase fluid intake to 2500 mL per day.
- Eat a well-balanced diet high in vitamins and minerals and with adequate protein and kilojoules for healing.
- Use good hand hygiene, particularly when caring for wounds or inflamed tissue and after using the bathroom.
- Elevate the inflamed area to reduce swelling and pain.
- Apply heat or cold for no longer than 20 minutes at a time to reduce the risk of tissue damage from burns or frostbite.
- Take all medications as prescribed, notifying the physician if adverse effects or hypersensitivity responses are noted.
- Rest acutely inflamed tissue; do not engage in strenuous activity until the inflammation has subsided.



The person with an infection

Microorganisms—including bacteria, viruses, fungi and parasites—often invade the human body and proliferate if undetected and not controlled or eliminated by inflammatory and immune responses. In most cases, contact between humans and microorganisms is incidental and may even be beneficial to both organisms. Resident bacteria of the skin, mucous membranes and gastrointestinal tract are an important part of the body's defence system. However, many microorganisms are virulent; that is, they have the ability to cause disease.

Pathogens are virulent organisms rarely found in the absence of disease. Some microorganisms, known as opportunistic pathogens, rarely cause harm to people with intact immune systems but are capable of producing infectious disease in the immunocompromised host (Porth & Matfin, 2009).

To a certain extent, modern medicine has contributed to the development of infectious diseases caused by antibiotic-resistant strains of microorganisms. Tuberculosis is on the rise in many countries, partially because organisms have become resistant to standard therapies. People receive immunosuppressive therapy following organ or tissue transplant, or in the treatment of neoplasms, making them more susceptible to infection. Metal and plastic prosthetic devices are implanted, providing potential sites for colonisation by disease-producing organisms. It has also become apparent that many diseases long considered unrelated to microorganisms may actually be infectious; for example, colonisation of the gastric mucosa with *Helicobacter pylori* is the predominant cause of peptic ulcer disease, and oncogenic viruses have the ability to transform normal cells into malignant cells.

Pathophysiology

Infection occurs when an organism is able to colonise and multiply within a host. The host can be any organism capable of supporting the nutritional and physical growth requirements of the microorganism—for example, humans. When the host experiences injury, pathological changes, inflammation or organ dysfunction in response to an infection or from intoxication by cellular poisons produced by a pathogen, the host is said to have an infectious disease.

For a microorganism to cause infection, it must have disease-causing potential (virulence), be transmitted from its reservoir and gain entry into a susceptible host. This is known as the chain of infection (see Figure 11.9).

PATHOGENS Pathogens capable of infecting and causing disease in a susceptible host include bacteria, viruses, *Mycoplasma*, *Rickettsia*, *Chlamydia*, fungi and parasites such as protozoa, helminths (worms) and arthropods (see Box 11.4). Each organism causes a different specific reaction in the host.

A number of different mechanisms have evolved in pathogens to facilitate their transmission and increase their ability to invade the host and cause disease. Factors influencing the transmission of an organism include its resistance to drying and to variations in environmental temperature. For example, spore-forming organisms are extremely resistant to drying.

Adhesion factors produced by or incorporated into the cell wall or membrane of the pathogen improve its ability to attach

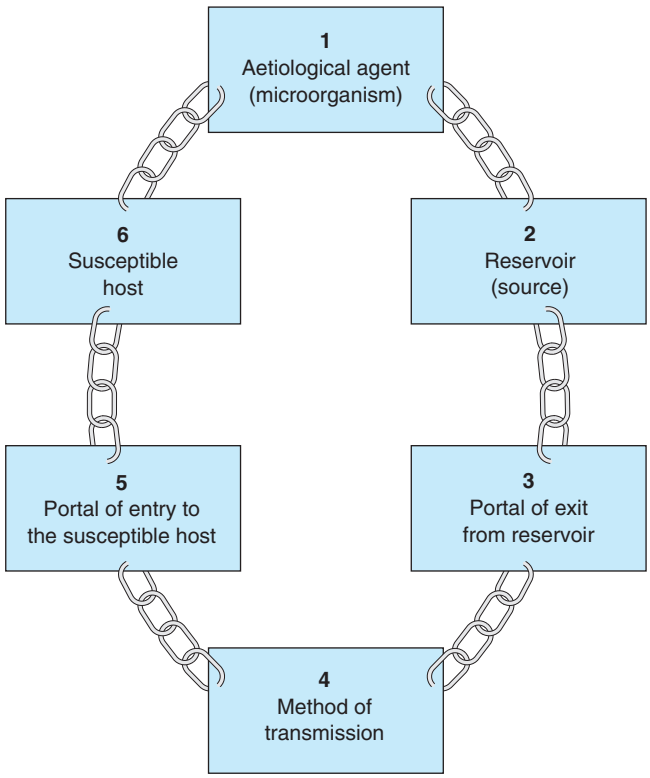


FIGURE 11.9 ■ The chain of infection

and colonise the host. Pathogens may also produce enzymes to enhance their spread to local tissues, chemicals to block specific immune processes or deplete neutrophils and macrophages, or extracellular capsules to discourage phagocytosis.

Pathogens are often capable of producing toxins that alter or destroy the normal function of host cells and promote colonisation, proliferation and invasion by the pathogen. Toxins often increase the disease-producing capability of the pathogen and, in some cases, are totally responsible for it; for example, cholera, tetanus and botulism result from bacterial toxins, not from the direct effects of the infection. **Exotoxins** are soluble proteins secreted into surrounding tissue by the microorganism. Exotoxins are highly poisonous, causing cell death or dysfunction. **Endotoxins** are found in the cell wall of Gram-negative bacteria and are released only when the cell is disrupted. Endotoxins have less-specific effects than exotoxins but they act as activators of many human regulatory systems, producing fever, inflammation and, potentially, clotting, bleeding or hypotension when released in large quantities.

RESERVOIR AND TRANSMISSION The reservoir or source, where the pathogen lives and multiplies, may be either endogenous or exogenous. Organisms that reside on skin or mucosal surfaces of the host are endogenous. Exogenous sources can include other humans, animals, soil, water, intravenous fluid or equipment. Infectious diseases are usually transmitted from human sources—that is, people who have clinical

BOX 11.4 Pathogenic organisms

Bacteria

Bacteria are single-celled organisms capable of autonomous reproduction. Relatively small and simple organisms, they contain a single chromosome. A flexible cell membrane and rigid cell wall surrounds their cytoplasm, giving them a distinctive shape; some also have an extracellular capsule for additional protection. Bacteria have different characteristics and growth requirements: the colonies formed by replicating bacteria differ from one another. *Aerobes* require oxygen for survival, whereas *anaerobes* cannot survive in the presence of oxygen; *Gram-positive* bacteria stain purple when subjected to crystal violet stain, whereas *Gram-negative* bacteria do not stain with crystal violet but turn red when subjected to safranin stain.

Prions

Prions are not independent organisms but small molecules that can modify host proteins. They primarily affect the neurological system, causing neurological degeneration in diseases such as mad cow disease (bovine spongiform encephalopathy) in animals and Creutzfeldt-Jakob disease in humans. These are slowly progressive, non-inflammatory conditions leading to dementia, lack of coordination and death. Prions' entry into the neurological cells makes them resistant to the host immune system and antibacterial and antiviral medications. They enter the host by injections, transplantation of contaminated tissue or medical devices, and possibly food. They are very resistant to disinfection, requiring special procedures for sterilising instruments, especially those used in CNS surgeries (Prusiner & Miller, 2012; Rothrock, 2011).

Viruses

Viruses are obligate intracellular parasites that are incapable of reproducing outside of a living cell. Viruses consist of a protein coat around a core of either DNA or RNA. Some viruses are shed continuously from infected cell surfaces; others, after inserting their genetic material into that of the infected cell, remain latent until they are stimulated to replicate. Viruses may or may not cause lysis and death of the host cell during replication. Oncogenic viruses are able to transform normal cells into malignant cells.

Mycoplasma

Although similar to bacteria, *mycoplasma* are smaller and have no cell wall, making them resistant to antibiotics that inhibit cell wall synthesis (e.g. penicillins).

Rickettsia and *Chlamydia*

As obligate intracellular parasites with a rigid cell wall, *Rickettsia* and *Chlamydia* have some features of both bacteria and

(continued)

BOX 11.4 Pathogenic organisms (continued)

viruses. Rather than depending on the host cell for reproduction, they use vitamins, nutrients or products of metabolism (e.g. ATP) from the host. *Chlamydia* are transmitted by direct contact, whereas many *Rickettsiae* infect the cells of arthropods (e.g. fleas, ticks and lice) and are transmitted from these vectors to humans.

Fungi

Fungi are prevalent throughout the world but few are capable of causing disease in humans. Most fungal infections are self-limited, affecting the skin and subcutaneous tissue. Some fungi, such as *Pneumocystis carinii*, can cause life-threatening opportunistic infections in the immunocompromised host.

Parasites

The term *parasite* is typically applied to members of the animal kingdom that infect and cause disease in other animals. Protozoa, helminths and arthropods are considered parasites. Protozoa are single-celled organisms (e.g. *Giardia lamblia* and *Trichomonas vaginalis*) transmitted via direct or indirect contact or an arthropod vector. Helminths are worm-like parasites: roundworms, tapeworms and flukes are examples. They gain entry into humans primarily through ingestion of fertilised eggs or penetration of larvae through the skin or mucous membranes. Arthropod parasites, such as scabies (mites), lice and fleas, typically infest external body surfaces, causing localised tissue damage and inflammation. Transmission is by direct contact with the arthropod or its eggs.

disease or are carriers with subclinical infection. Carriers harbour the pathogen without showing evidence of clinical disease. Pathogens exit human hosts via respiratory secretions, body fluids from the gastrointestinal and genitourinary tracts, skin or mucous membrane lesions, the placenta and blood.

Organisms may be transmitted from the source to the susceptible host by direct or indirect contact, droplet or airborne transmission, or a vector. Direct contact includes person-to-person spread or contact with infected body fluids, as well as transmission from contaminated food or water. Indirect contact occurs when the infectious agent is contracted by use of inanimate objects such as dirty eating utensils. Sneezing, talking and coughing allow transmission by droplet contact when the host is within 1 metre of the source. Smaller respiratory particles that stay suspended in air and are carried via air currents allow airborne transmission. Vectors are insects and animals such as flies, mosquitoes or rodents that act as intermediate hosts between the source and host. Microorganisms usually first colonise the portal of entry: non-intact skin, wounds, mucous membranes and the respiratory, gastrointestinal or genitourinary tracts.

HOST FACTORS The susceptible host is the final link in the chain of infection. Exposure to pathogens does not automatically cause infection or infectious disease. The outcome of contact with a pathogenic microorganism is determined by the balance of microbial virulence and host resistance. Factors that can enable the host to resist infection include the following:

- physical barriers such as the skin and mucous membranes
- the hostile environment created by acid stomach secretions, urine and vaginal secretions
- antimicrobial factors in saliva, tears and prostatic fluid
- respiratory defences, including humidification, filtration, the mucociliary escalator, cough reflex and alveolar macrophages
- innate and adaptive immune responses to pathogenic invasion.

Stages of the infectious process

When infectious disease develops in the host, it typically follows a predictable course with stages based on the progression and intensity of manifestations.

The initial stage is the *incubation period*, during which the pathogen begins active replication but does not yet cause manifestations. Depending on the organism and host factors, the incubation period may last from hours, as with *Salmonella* infection, to years, as with HIV infection.

The *prodromal stage* follows, during which symptoms first begin to appear. At this stage, manifestations are often nonspecific and include general malaise, fever, myalgias, headache and fatigue.

Maximal impact of the infectious process is felt during the *acute phase* as the pathogen proliferates and disseminates rapidly. Toxic by-products of microorganism metabolism and cell lysis, along with the immune response, produce tissue damage and inflammation during this stage (Grossman & Porth, 2014). Manifestations are more pronounced and specific to the infecting organism and site during the acute stage. Fever and chills may be significant during this phase. However, people with alcoholism and the very old may respond to severe infection by becoming hypothermic. The person is often tachycardic and tachypnoeic because of increased metabolic demands. Localised manifestations include redness, heat, swelling, pain and impaired function. When the infectious disease affects an internal organ, manifestations are related to inflammatory changes in that organ and surrounding tissue. The person may experience tenderness to palpation over the site or show signs of impaired function, such as the haematuria and proteinuria characteristic of renal infections.

If the infectious process is prolonged, manifestations of the continuing immune response may become apparent. Catabolic and anorexic effects of the infection can lead to loss of body fat and muscle wasting. Immune complexes may be deposited at sites other than the primary infection, resulting in an inflammatory process. Glomerulonephritis (e.g. following strep throat) and vasculitis are possible results. Another possible consequence of prolonged infection and immune response is the triggering of an autoimmune disease process such as rheumatic cardiomyopathy or coeliac disease.

As the infection is contained and the pathogen eliminated, the *convalescent stage* of the disease occurs. During this stage, affected tissues are repaired and manifestations resolve.

Resolution of the infection is total elimination of the pathogen from the body without residual manifestations. If a balance between organism and host factors occurs, with neither predominating, chronic disease may develop or the organism may be driven into a protected site such as an abscess. A carrier state develops when host defences eliminate the infectious disease but the organism continues to multiply on mucosal sites.

Complications

Multiple and varied complications are associated with infectious diseases. They are typically specific to the infecting organism and the body system affected.

Acute invasion of the blood by certain microorganisms or their toxins can result in septicaemia and septic shock. Whereas *bacteraemia*, the presence of bacteria in the blood, may not have serious effects, **septicaemia** refers to systemic disease associated with their presence or toxins. Septic shock indicates a state of hypotension and impaired organ perfusion resulting from sepsis. Unless treated aggressively, septic shock leads to diffuse cell and tissue injury and potentially to organ failure.

FAST FACTS

- Urinary tract infection is the most common type of **healthcare-associated infection** (HAI), usually associated with indwelling urinary catheters or urological procedures.
- Other common HAIs are bloodstream infections, pneumonia, surgical wound infections and *Clostridium difficile* colitis (CDC, 2011; Chin-Hong & Guglielmo, 2012).
- Pathogens associated with HAI are often different from those causing community-acquired infections and frequently are multidrug resistant, necessitating treatment with multiple, broad-spectrum and potentially toxic antibiotics (Chin-Hong & Guglielmo, 2012).

Healthcare-associated infections

Healthcare-associated infections (HAIs) are acquired in any healthcare setting, such as a hospital or nursing home. Also called *nosocomial* infections, HAIs account for an estimated 200 000 infections, 2 million bed days lost and, for the surgical post-discharge infection subgroup, $21 million in excess healthcare costs annually in Australia (ACSQHC, 2008). HAIs add hospital days, reduce admissions by occupying available beds and add to the cost of healthcare (CDC, 2010; Mirza, 2012).

Many HAIs result from the use of invasive devices such as intravascular catheters, urinary catheters and endotracheal tubes for ventilator support. People developing HAIs often are critically ill and among those least able to mount an effective immune defence against infection. HAIs also occur when antibiotic therapy has altered natural defences and impaired resistance to harmful microorganisms. Endogenous organisms outside their normal habitats (such as in *Escherichia coli* in the urinary tract) become a threat to the person. Other pharmacological and therapeutic procedures such as chemotherapy, the use of corticosteroids or radiation therapy also contribute to HAIs. Surgical site infections rank second in frequency of HAIs and add up to 7 to 10 extra days to postoperative hospitalisation (CDC, 2010; Weinstein, 2012).

BOX 11.5 Interventions to reduce healthcare-associated infections

- Central venous catheter infections have decreased by using chlorhexidine antiseptic for disinfection and maximal barrier precautions during insertion.
- Ventilator-associated pneumonia is decreased by weaning patients off ventilators as soon as possible, limiting sedation of the patient, positioning patients with the head of the bed elevated to prevent gastric reflux and for maximal ventilation, and using proper hand hygiene and sterile technique for all ventilator-associated care.
- Surgical site infections are reduced by administering a prophylactic antibiotic 1 hour before the incision and discontinuing it within 24 hours after surgery, limiting hair removal (no shaving), controlling perioperative glucose levels (especially in cardiac surgeries) and ensuring normothermia for the patient during the perioperative period (especially in colorectal surgeries).
- Insert urinary catheters only when clearly indicated, using aseptic technique during insertion; minimise manipulation or opening of drainage systems (Weinstein, 2012).

Superficial or deep wounds may be contaminated by endogenous or exogenous sources. Infections in body cavities or those associated with prosthetics are difficult to diagnose and may necessitate removal of the prosthetic device. Box 11.5 lists interventions that should be used to prevent HAIs.

Hospital-acquired pneumonia accounts for 15% of HAIs. It is usually associated with ICU stays and mechanical ventilation. Organisms causing the infection are often resistant to many drugs, not responding to antibiotics usually effective in treating infections acquired outside the hospital. More deaths are associated with hospital-acquired pneumonia than any other site of infections (Weinstein, 2012).

CONSIDERATION FOR PRACTICE

Existing guidelines and literature reviews agree that hand hygiene using alcohol-based hand rubs is more effective against the majority of common infectious agents on hands than hand hygiene with plain or antiseptic soap and water (NHMRC, 2010).

A soap-and-water wash is recommended for visibly soiled hands. Wearing gloves does not eliminate the need to perform hand hygiene.

Prevention is the most important control measure for HAIs. Cross-infection—the spread of pathogens from one person to another on the inadequately cleaned hands of healthcare workers—is one of the primary sources of HAIs (Weinstein, 2012). *Effective hand hygiene is the single most important measure in infection control* (WHO, 2009) (see Figure 11.10). Although infections may also be transmitted by the airborne

FIGURE 11.10 ■ 5 Moments for Hand Hygiene

Source: Reprinted from *Hand Hygiene When and How* leaflet, p. 2. © 2009. <www.who.int/gpsc/5may/background/5moments/en/index.html>. ©World Health Organization 2009. All rights reserved.

route, from contaminated equipment or from the environment, these are less significant causes. Invasive procedures and equipment should be used only when absolutely necessary; for example, it is not appropriate to insert an indwelling catheter when the only indication is incontinence. Peripheral intravenous equipment and sites must be kept clean and changed regularly: intravenous bags and bottles every 24 hours, tubing every 24 to 72 hours and sites every 2 to 3 days according to agency policy.

Antibiotic-resistant microorganisms

Antibiotic-resistant microorganisms are increasing at an alarming rate, primarily due to prolonged or inappropriate use of antibiotic therapy. Although antibiotic therapy is expected to eradicate all targeted microorganisms, sometimes a few bacteria survive, leading to bacteria that reproduce with antibiotic resistance already encoded into their genetic make-up (ACSQHC, 2010; Lehne, 2012). Other bacteria produce enzymes that inactivate drugs, change drug-binding sites or alter their cell membrane to prevent drug absorption.

Standard precautions, most importantly hand hygiene and the use of carefully selected antibiotics, are critical actions for stopping the spread of these diseases. Equipment such as stethoscopes, blood pressure cuffs and thermometers should be restricted to use by each person identified with one of these diseases. Personal protective equipment, used and disposed of appropriately, is an important safeguard.

NURSING CARE OF THE OLDER ADULT Infections

Because immune function declines with ageing, older adults are more susceptible to infections. Infections are among the top five causes for hospitalisation and among the leading causes of death among people over 65 years of age (Weir et al., 2011). Physiological changes that often occur with ageing place the older adult at greater risk than younger people of acquiring an infection.

- *Cardiovascular changes:* decreased tissue perfusion delays the inflammatory response and healing.
- *Respiratory system changes:* decreased mucociliary clearance, decreased elastic recoil and diminished cough and laryngeal reflexes decrease the clearance of respiratory secretions and increase the risk of pneumonia. The older adult with pneumonia may not present with cough or sputum production due to decreased immune function. The leading causes of pneumonia in older adults include *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Staphylococcus aureus*. Influenza A, a viral infection, is a significant risk factor for secondary bacterial pneumonia in older adults (Tabloski, 2014). Both pneumonia and influenza cause high mortality rates in the older person.
- *Genitourinary changes:* loss of muscle tone, reduced bladder contractility, altered bladder reflexes and prostatic hypertrophy in men increase the risk for incomplete bladder emptying, urinary incontinence and urinary tract infection (UTI). UTI is the most common infection and the leading cause of bacteraemia and sepsis in older adults, particularly those aged 85 and older.
- *Gastrointestinal system changes:* impaired swallow reflex, decreased gastric acidity and delayed gastric emptying increase the risk of aspiration with subsequent pneumonia.
- *Skin and subcutaneous tissue changes:* thinning of skin, decreased cushioning, decreased sensation and decreased vasculature increase the risk of injury, ulceration and infection.
- *Immune changes:* decreased phagocytosis, reduced inflammatory response, diminished antibody-mediated and cellular immune responses, and slowed or impaired healing processes increase the risk for infection. Immunoglobulin levels remain relatively stable, but primary and secondary antibody responses decline with ageing. The thymus gland atrophies and some T-cell populations decrease or decline in function. T-cell activation and the ability to proliferate following activation also decline with advancing age (Grossman & Porth, 2014). Resistance to antigens such as *Mycobacterium tuberculosis*, influenza and varicella-zoster viruses, malignant cells and tissue grafts is reduced.

Other factors, such as a lower activity level, poor nutrition and an increased risk for dehydration, a higher prevalence of chronic diseases such as diabetes, use of multiple medications and altered mentation contribute to the older adult's risk for infection.

HAIs are more common in older adults. The nurse must steadfastly adhere to principles of infection control. Nursing interventions to reduce the risk of HAIs include: (1) avoiding prolonged bed rest, (2) encouraging patients to take deep breaths, (3) providing adequate fluids, (4) providing regular toileting schedules with good hygiene, and (5) avoiding use of invasive devices such as indwelling catheters unless medically necessary.

The older adult may not exhibit the classic manifestations of inflammation and infection. The manifestations of inflammation—redness, heat and swelling—tend to be diminished or absent in older adults. The classic manifestations of infection—fever and chills—may be absent altogether because of age-related changes in the immune system, loss of central temperature control mechanisms, decreased muscle mass and loss of shivering ability. The older adult may have only subtle manifestations of infection or sepsis, including changes in mental status, disorientation, restlessness and tachypnoea.

Prompt identification and treatment of infection improves outcomes in the older adult. In addition to monitoring for changes in the patient's mental status or behaviour, the nurse should assess fluid intake and urinary output, activity levels, complaints of fatigue and respiratory status. Older adults are at increased risk for dehydration due to diminished thirst sensation and impaired water conservation by the kidneys. Carefully evaluate intake and output to determine if input is adequate.

INTERPROFESSIONAL CARE

The goals of care for the person with an infection are to identify the organ system affected by the infection and the causative agent, and to achieve a cure by the least toxic, least expensive and most effective means. Fortunately, most infectious diseases are self-limiting and will resolve with little or no medical care. However, medical treatment can be lifesaving in an overwhelming infection or immunocompromised host.

The site of the infection is often obvious from the person's history and presenting manifestations. Identifying the affected organ system allows the range of possible infecting organisms to be narrowed to those known to affect that system.

Once the infecting agent has been identified, either positively or by probability, therapy can be specifically tailored to the person's needs. Viral infections often resolve without treatment other than supportive care, such as providing rest and fluids. Skin infections may respond to a topical agent, avoiding the potential adverse effects of one administered systemically.

Diagnosis

To assess the person's response to infection, identify the infecting organism and monitor the progress of therapy, the following diagnostic tests may be ordered:

- *WBC count* provides clues about the infecting organism and the body's immune response to it.
- *WCC differential* is also ordered (see Table 11.8). Neutrophilia, increased numbers of circulating neutrophils (or PMNs),

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 3:
Preventing and Controlling Healthcare Associated Infections

'The intention of this standard is to prevent patients from acquiring preventable healthcare associated infections and effectively manage infections when they occur.' (ACSQHC, 2012, p. 26)

Implementing this standard is achieved by implementing systems to prevent and manage healthcare-associated infections and communicate these to the workforce to achieve appropriate outcomes. This includes governance and systems for infection prevention, control and surveillance; infection prevention and control strategies; infections or colonisations; antimicrobial stewardship (the safe and appropriate antimicrobial prescribing); and cleaning, disinfection and sterilisation.

Infection prevention and control is necessary to reduce the development of resistant pathogens and to minimise the risk of transmission by isolating the infectious organism or the patient, and by using standard and transmission-based precautions.

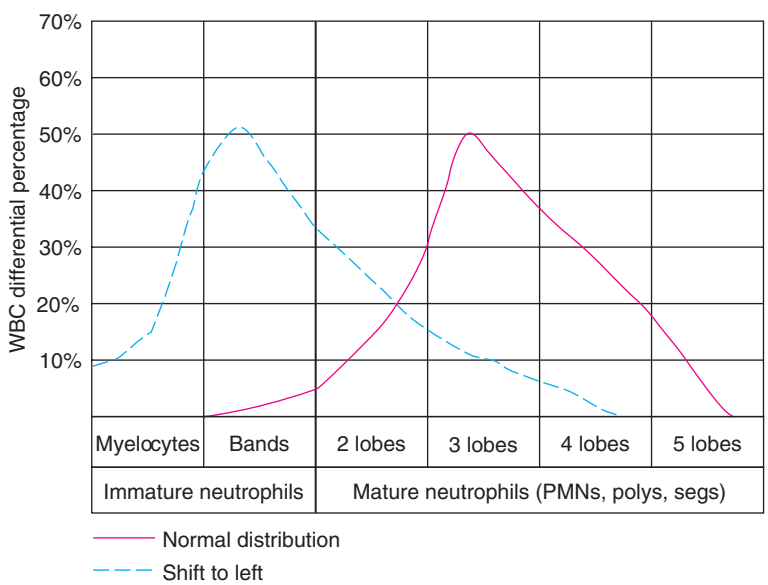
Efficient and appropriate systems are imperative to ensure the safety of not only the person receiving appropriate care, but also any other individual involved in their care.

Effective communication should exist across all individuals involved in a person's care (including the person themselves and their significant others).

Source: © Australian Commission on Safety and Quality in Health Care.

is a common response with infection as the bone marrow responds to an increased need for phagocytes. Along with neutrophilia, a shift to the left is common in acute infection. This means that there are more immature neutrophils in circulation than normal, indicating an appropriate bone marrow response (see Figure 11.11).

- *Procalcitonin (CTpr) and C-reactive protein (CRP)* are diagnostic markers of infection that can be measured in the blood. Blood levels of CTpr and CRP increase dramatically with serious bacterial infection and sepsis, making these markers useful early indicators of systemic infections.
- *Cultures of the wound, blood or other infected body fluids* are used to identify probable microorganisms by their characteristics, such as shape, growth patterns and Gram-staining qualities. After the organism is cultured, it is subjected to various antibiotics known to be effective against its particular strain to determine which antibiotic is likely to be most effective. Generally, 24 to 48 hours are required to grow the organism, potentially delaying the institution of therapy. Because antibiotics can alter the ability to culture an organism, specimens should be obtained before instituting therapy.
- *Serological testing* provides an indirect means of identifying infecting agents by detecting antibodies to the suspected organism. When the antibody titre against a specific organism rises during the acute phase of an infectious disease and



Type of WBC	Normal differential	Shift to left
Myelocytes	0%	Present
Band neutrophils (bands)	3–5%	Increased
Segmented neutrophils (segs, polys, PMNs)	50–65%	May be stable, increased, or decreased

FIGURE 11.11 ■ Neutrophils by stage of maturity and normal distribution in the blood

begins to fall during convalescence, the diagnosis is supported. Although it is not as accurate as culture, serology is particularly useful for organisms that cannot easily be cultured, such as hepatitis B or HIV.

- *Direct antigen detection methods* use monoclonal antibodies (purified antibody forms) to detect antigens in specimens from the diseased host. The tests offer rapid and accurate identification of the offending microorganism.
- *Antibiotic peak and trough levels* monitor therapeutic blood levels of the prescribed medication(s). The therapeutic range—that is, the minimum and maximum blood levels at which the drug is effective—is known for a given drug. By measuring blood levels at the predicted peak (1 to 2 hours after oral administration, 1 hour after intramuscular administration and 30 minutes after intravenous administration) and trough (lowest level, usually a few minutes before the next scheduled dose), healthcare personnel can determine that the person is maintaining a level within the therapeutic range at all times, ensuring maximal effect from the drug. It is also possible to determine whether the drug is reaching a toxic or harmful level during therapy, increasing the likelihood of adverse effects.
- *Radiological examination of the chest, abdomen or urinary system* may be ordered to detect organ abnormalities indicating an inflammatory response or tissue damage.
- *Lumbar puncture* is performed to obtain cerebrospinal fluid (CSF) for examination and culture if a central nervous system (CNS) infection, such as meningitis or encephalitis, is suspected.
- *Ultrasonic examination* is a non-invasive diagnostic test such as an echocardiogram or renal ultrasonography to identify an infectious site or evaluate the effects of an infection on organ function.

Medications

After the infecting organism and affected body system have been identified, specific therapy to cure the infectious disease can be instituted.

Antimicrobial preparations are broadly classified as bacteriostatic or bactericidal. **Bacteriostatic agents** inhibit the growth of the microorganism, leaving its destruction to the host's immune system. These agents are generally not indicated for the immunocompromised host. Tetracyclines, erythromycin and chloramphenicol are bacteriostatic preparations. **Bactericidal agents**, including pencillins, cephalosporins and aminoglycoside antibiotics, are capable of killing the organism without immune system intervention.

The activity of antimicrobial agents on bacteria, fungi and viruses falls under five basic mechanisms:

1. impairing cell wall synthesis, leading to lysis and cell destruction
2. inhibiting protein synthesis, causing impaired microbial function
3. altering cell membrane permeability, causing intracellular contents to leak
4. inhibiting the synthesis of nucleic acids
5. inhibiting cell metabolism and growth.

Many microorganisms have the ability to develop resistance to an anti-infective agent; that is, the pathogen continues to live and grow in the presence of the anti-infective. Resistance develops as a result of a chance mutation by the pathogen, allowing a subpopulation of cells to survive. The chance of an organism becoming resistant to an agent is partially related to the dose delivered. Resistance is less likely to occur when a lethal dose is administered; therefore, it is vital that people understand the need to take all doses of the prescribed drug as ordered.

ANTIBIOTICS

Medications used to treat bacterial infections are generally known as antibiotics. Most antibiotics are biological substances; that is, substances produced by other microorganisms. Antibiotics fall into classes of drugs with related chemical structure and activity. Some are effective against only Gram-positive bacteria and others are effective against only Gram-negative organisms. Broad-spectrum antibiotics have activity against a wide variety of bacteria including both Gram-positive and Gram-negative forms.

No antibiotic is totally safe. Hypersensitivity responses occur, so always check for allergies before administering the first dose. Some drugs are toxic to organ systems, exhibiting hepatotoxicity, nephrotoxicity, ototoxicity or bone marrow suppression. The antibiotics presented in the following 'Medication administration' box are organised according to their antibacterial action.

ANTIVIRALS

Most antibiotics have little effect on viruses because the virus has no cell wall and no cytoplasm, produces no enzymes and sequesters itself in a host cell to reproduce. Antiviral agents must be very selective in differentiating normal cellular activity from viral activity. In addition, the immune function of the host is a vital component in fighting viral infections; antiviral therapy may be relatively ineffective in the severely immunocompromised host. Timely diagnosis of viral infections can be an additional problem because viruses are less easily identified using laboratory techniques. Antiviral agents in common use are summarised in the 'Medication administration' box.

ANTIFUNGALS

Antifungal agents are available in both topical and systemic forms. They act by interfering with the cytoplasmic membrane of the fungus. Topical agents include preparations for cutaneous use to treat candidiasis, tineas and ringworm. Vaginal preparations to treat vulvovaginal candidiasis are also available, as are several non-prescription topical and vaginal antifungal agents.

Amphotericin B is a systemic antifungal agent for parenteral administration. It is used to treat severe, life-threatening fungal infections including histoplasmosis, blastomycosis and candidiasis. Another systemic antifungal in current use is flucytosine (Ancobon), which can be administered orally. It is used to treat severe candidiasis infections such as *Candida* septicaemia, endocarditis, pulmonary or urinary tract infections, and *Cryptococcus* meningitis.

Fluconazole (Diflucan) has the broadest use as an antifungal agent. It can be administered either orally or parenterally and is

used to treat candidiasis infections as well as *Cryptococcus* meningitis. It is generally better tolerated than other systemic antifungal medications.

ANTIPARASITICS Drugs used to treat parasitic infections are as varied as the organisms that cause them. Generally, agents classified as antiparasitic are both expensive and likely to be toxic. Quinine was one of the first antiparasitic drugs developed in the treatment of malaria. Quinine is highly toxic, but newer forms such as chloroquine, hydroxychloroquine (Plaquenil) and paludrine (Proguanil hydrochloride) are widely used as antimalarial drugs. Metronidazole (e.g. Flagyl) is used to treat infections of protozoan parasites (see the 'Medication administration' box).

MEDICATION ADMINISTRATION Antibiotic therapy

I. Cell wall synthesis inhibitors

PENICILLINS

Penicillin G
Penicillin V
Amoxicillin (Amoxil)
Amoxicillin and clavulanic acid (Augmentin)
Ampicillin (Austrapen)
Dicloxacillin (Diclocil, Distaph)
Flucloxacillin (Staphylex, Floxapen)
Piperacillin and tazobactam (Tazocin)
Ticarcillin and clavulanic acid (Timentin)

Penicillins are bactericidal and interfere with cell wall synthesis and the enzymes involved in cell division and synthesis. They are more effective on Gram-positive than Gram-negative organisms. Penicillins are considered to be safe, effective and of low toxicity. Resistance is now more common among *Streptococci* and *Staphylococci.* Penicillins and related antibiotics such as cephalosporins contain a molecular structure known as a beta-lactam ring. Some bacteria produce enzymes (beta-lactamases or penicillinases) that cleave this ring, making the antibiotics ineffective. To combat this *resistance, beta-lactamase or penicillinase inhibitors such as sulbactam and clavulanate are combined with some antibiotics to create an antibiotic effective against drug-resistant bacterial strains.*

Nursing responsibilities

- Monitor for hypersensitivity responses such as local erythema and itching at the site of injection, skin rashes, urticaria (hives), itching, fever, chills and anaphylaxis.
- Observe people receiving parenteral penicillin for at least 30 minutes.
- Discontinue the drug immediately if any hypersensitivity response occurs. Be prepared to administer antihistamines or corticosteroids for a mild reaction. Anaphylaxis is treated with adrenaline subcutaneously or intravenously and with airway support.
- Do not administer penicillin to anyone with a history of a severe allergic reaction to any form of the drug; a cross-reactivity may occur in people allergic to cephalosporin or carbapenem antibiotics.
- Assess for superinfection (vaginitis, stomatitis or diarrhoea) due to elimination of resident bacteria.

Health education for the person and family

- Notify the physician if you see white patches on the oral mucosa or if vaginitis develops. An antifungal drug may be prescribed and the antibiotic continued.
- Consuming yoghurt may prevent superinfection. Do not take these products within 1 hour of taking the drug.

CEPHALOSPORINS

1st generation
Cephalexin (Keflex, Ibilex)
Cephazolin (Kefzol)

2nd generation
Cefoxitin (Mefoxin)
Cefaclor (Aclor, Ceclor)

3rd generation
Cefotaxime (Claforan)
Ceftazidime (Fortrum)
Ceftriaxone (Rocephin)

4th generation
Cefepime (Maxipime)

Cephalosporins are structurally similar to the penicillins and also inhibit cell wall synthesis. They are divided into four groups or generations. First-generation cephalosporins act primarily against Gram-positive organisms. Second- and third-generation drugs are more effective against Gram-negative organisms than against Gram-positive ones. Fourth-generation cephalosporins act effectively against both Gram-positive and Gram-negative organisms.

Nursing responsibilities

- Monitor for previous hypersensitivity response to cephalosporins or penicillins.
- Assess intravenous site for phlebitis; intramuscular site may cause local pain.
- Monitor laboratory results for adverse response, such as leucopenia and thrombocytopenia, nephrotoxicity (elevated BUN and serum creatinine) or hepatotoxicity (elevated bilirubin, LDH, ALT, AST and alkaline phosphatase).
- Assess for signs of superinfection.

Health education for the person and family

- Take the medication on an empty stomach, 1 hour before or 2 hours after meals.
- Avoid alcohol as alcohol intolerance can develop with these antibiotics. These same drugs intensify bleeding tendencies.
- Space doses of the medication relatively evenly throughout the day and evening hours.
- Increased consumption of yoghurt may prevent intestinal superinfection.

CARBAPENEMS

This class of antibiotics includes only three drugs and all must be given parenterally. Imipenem has the broadest antimicrobial spectrum of any drug (Lehne, 2012). This makes it especially useful against mixed-organism infections. Imipenem, meropenem and ertapenem cross the meninges and achieve therapeutic doses in CSF; they are

MEDICATION ADMINISTRATION Antibiotic therapy (continued)

effective against methicillin-resistant *Staphylococcus aureus* (MRSA). These antibiotics cause bacterial cell wall lysis and subsequent death of the bacteria. Side effects include nausea and vomiting, diarrhoea, hypersensitivity reactions, occasional superinfections with bacteria or fungi, and, rarely, seizures.

Nursing responsibilities

Ertapenem should not be mixed with dextrose or other drugs containing dextrose. IV infusions should be slow and given over at least 30 minutes.

- Check for history of hypersensitivity to cephalosporins and penicillins and monitor for signs of reactions.
- Assess for signs of superinfection.
- Monitor laboratory indicators of renal function.

Health education for the person and family

- Instruct the person to report any signs or symptoms of allergy such as skin rash, itching or hives.

VANCOMYCIN

This antibiotic inhibits cell wall synthesis and is used for serious infections. It is only effective against Gram-positive bacteria, especially *S. aureus* and *Staphylococcus epidermidis*, including the strains resistant to methicillin. *C. difficile* is also susceptible to this antibiotic, but infection with *C. difficil*e is often treated first with metronidazole to delay emergence of resistance to vancomycin.

Nursing responsibilities

- Infuse slowly over 60 minutes or more to avoid 'red man' syndrome. The syndrome is characterised by erythematous rash, flushing, tachycardia and hypotension. People may become dizzy and agitated. The occurrence is usually associated with a first dose of vancomycin and is seen within 4 to 6 minutes of the start of a dose or after completion.
- Ototoxicity is a more serious adverse effect of vancomycin because hearing loss may be irreversible. Notify the physician immediately if a sensation of fullness in the ears is reported, as this indicates ototoxicity.

II. Bacterial protein synthesis inhibitors

TETRACYCLINES

Tetracycline HCl **Minocycline HCl (Minomycin, Akamin)**
Doxycycline (Vibramycin)

Tetracyclines are active against many Gram-positive and Gram-negative bacteria, such as *Mycoplasma*, *Rickettsia* and *Chlamydia*. They are bacteriostatic, interfering with microbial protein synthesis. Tetracycline binds readily with metal and solid elements in the bowel, limiting its absorption when administered with food; the other preparations are highly soluble in lipids and can be administered with food.

Nursing responsibilities

- Schedule doses 1 hour before or 2 hours after meals. Do not give with milk, milk products or antacids.
- Monitor for signs of superinfection.
- If the person is taking an anticoagulant, monitor prothrombin time and for signs of bleeding.

Health education for the person and family

- Avoid excessive sun exposure to reduce the risk of photosensitivity reactions.
- Tetracyclines can stain the enamel of developing teeth when taken during pregnancy; although deciduous (baby) teeth are affected, permanent teeth are not.

MACROLIDES

Erythromycin (Eryc) **Clarithromycin (Klacid)**
Azithromycin (Zithromax) **Roxithromycin (Rulide)**

Macrolides are bacteriostatic and act effectively against Gram-positive and Gram-negative organisms. Erythromycin is used to treat streptococcal pharyngitis in people who are allergic to penicillin. Azithromycin produces less nausea than erythromycin, increasing patient adherence.

Nursing responsibilities

- Administer erythromycin on an empty stomach or immediately before meals.
- Give the drug with a full glass of water. Do not administer with acidic fruit juice.
- Intravenous doses are very irritating to veins; give slowly (20 to 60 minutes per gram).

Health education for the person and family

- Gastric distress is a common side effect with erythromycin.

AMINOGLYCOSIDES

Amikacin (Amikin) **Gentamicin**
Neomycin (Neosulf) **Tobramycin**

Aminoglycosides are bactericidal, interfering with protein synthesis in the pathogen. They are especially effective against Gram-negative organisms. To provide a broader spectrum of activity, they are often combined with other antibiotics, especially penicillins. Aminoglycosides can be administered in multiple or single daily doses. They are ototoxic and nephrotoxic; the risk is highest for older adults, people with pre-existing renal disease and people receiving other ototoxic or nephrotoxic drugs.

Nursing responsibilities

- Assess renal function before and during aminoglycoside therapy. Monitor intake and output, daily weight, BUN and serum creatinine.
- Assess for adverse effects on hearing such as loss of perception of high tones, tinnitus and vertigo.
- Notify the physician if the person is receiving other nephrotoxic or ototoxic drugs such as frusemide (Lasix) and ethacrynic acid (Edecrin).
- Administer intravenous preparations separately from other drugs; flush tubing before and after administration.

Health education for the person and family

- Monitor for a sudden weight gain, which may indicate adverse effects on the kidney, and report it to the physician.

(continued)

MEDICATION ADMINISTRATION Antibiotic therapy (continued)

OXAZOLIDINONES

Linezolid is the first antibiotic in the class of oxazolidinones. This antibiotic inhibits protein synthesis and is effective against organisms that are resistant to both vancomycin and methicillin. Because of its usefulness against those organisms, it should be reserved for infections caused by vaoncomycin-resistant enterococci (VRE) and MRSA (Lehne, 2012).

Nursing responsibilities

- Monitor for side effects including nausea, diarrhoea, hypertension and headache.
- Monitor platelets if person is at risk for bleeding; this drug may cause thrombocytopaenia.

Health education for the person and family

- It can be taken with or without food.
- Avoid taking adrenaline, pseudoephedrine, methylphenidate or cocaine with this drug as hypertension may develop.

III. Bacterial nucleic acid inhibitors

FLUOROQUINOLONES

Ciprofloxacin (Ciproxin) **Norfloxacin (Noroxin)**
Moxifloxacin (Avelox)

Fluoroquinolones are bactericidal and especially active against Gram-negative and some Gram-positive organisms. They are used to manage infections of the respiratory, gastrointestinal and genitourinary tracts. Rarely, drugs in this class can cause tendon rupture, with the highest risk in people aged 60 and older and in those taking glucocorticoid medications (Adams et al., 2014).

Nursing responsibilities

- Increase fluid intake to 2000 to 3000 mL/day, unless contraindicated, to prevent crystalluria.
- Monitor laboratory results for hepatotoxicity (elevated ALT, AST).

Health education for the person and family

- If tendon inflammation or pain develops, stop taking the drug and immediately report to your healthcare provider.
- Drink 6 to 8 glasses of water per day.
- Avoid exposure to sunlight while taking these drugs.

SULFONAMIDES AND TRIMETHOPRIM

Sulfadiazine
Trimethoprim (Alprim)
Timethoprim and Sulfamethoxazole (Bactrim, Septrin)

Sulfonamides are bacteriostatic. Trimethoprim is an antibiotic effective against most Gram-positive and many Gram-negative organisms. It is often combined with sulfamethoxazole to manage urinary tract infections, *P. carinii* pneumonia and otitis media. Skin rashes and pruritus are the most common hypersensitivity reactions. Severe reactions include exfoliative dermatitis and Stevens–Johnson syndrome.

Nursing responsibilities

- Assess for history of hypersensitivity to sulfonamides and related medications, such as thiazide diuretics and hypoglycaemic preparations.
- Monitor intake and output. Unless contraindicated, maintain a fluid intake of at least 1500 mL/day.
- Assess for evidence of bleeding, easy bruising or systemic infection, and monitor blood count for possible bone marrow depression.

Health education for the person and family

- Take medication on an empty stomach with a full glass of water. Maintain a fluid intake of at least 2 L per day.
- Protect the skin from excessive sun exposure with clothing and sunscreens to reduce the risk of photosensitivity.

NITROIMIDAZOLES

Metronidazole (Flagyl)
Tinidazole (Fasigyn)

Nitroimidazoles are metabolised to active metabolites that are thought to interfere with DNA synthesis. Metronidazole is effective against anaerobic Gram-negative and Gram-positive bacteria and protozoan infections caused by amoebiasis, giardiasis and trichomoniasis. It is commonly used to prevent and treat infections following intestinal surgery and is the drug of first choice with *C. difficile*.

Nursing responsibilities

- Monitor for CNS effects of dizziness, headache, ataxia, confusion, depression and peripheral neuropathy.
- Administer with food to minimise gastric distress and metallic taste. Infuse intravenous metronidazole over 60 minutes.
- Discontinue the medication and notify the physician if neurological reactions occur.
- Increase fluid intake to 2500 mL/day to minimise the risk of nephrotoxicity.

Health education for the person and family

- This medication may turn urine reddish brown; caution the person that this is expected and not harmful.
- Stop taking the drug and notify the physician if hypersensitivity reaction or adverse effects occur, such as changes in mentation or coordination, painful or frequent urination, painful or difficult intercourse, impotence.
- Do not drink alcohol while taking this medication; an Antabuse-type reaction (flushing, sweating, headache, vomiting and abdominal cramps) may occur.
- Maintain a fluid intake of 2.5 to 3 L per day.
- When the drug is prescribed for *Trichomonas* infections, treatment of both partners is necessary. Use condoms to prevent cross-contamination during intercourse.

MEDICATION ADMINISTRATION Antiviral agents

NEURAMINIDASE INHIBITORS

Oseltamivir (Tamiflu) and zanamivir (Relenza) are used to prevent and treat influenza. They are both active against both influenza A and B. They are generally well tolerated.

ADAMANTANES

Amantadine is used to treat influenza A. When administered within 48 hours of symptom onset, common manifestations are reduced. Generally well tolerated, CNS side effects such as dizziness, anxiety, insomnia and difficulty concentrating may occur.

ACICLOVIR (ZOVIRAX) GANCICLOVIR (CYMEVENE)

Guanine analogues are used primarily in the treatment of herpes viruses. Aciclovir is prescribed mainly in the treatment of genital herpes simplex and varicella. Although it does not kill the virus, it is effective in reducing the severity, duration and frequency of recurrence of symptoms. Ganciclovir is indicated primarily in the treatment of cytomegalovirus infection. Although aciclovir is generally well tolerated with little toxicity, ganciclovir may profoundly suppress bone marrow function.

PROTEASE INHIBITORS

Protease inhibitors prevent viral maturation and replication of HIV. These drugs are used alone or in combination with some taken orally or administered parenterally. Many people are unable to tolerate recommended doses due to adverse effects, including nausea, anorexia, malaise, severe anaemia and granulocytopenia.

INTERFERONS

Interferons (IFNs) are naturally produced cytokines that have antiviral activity. Pegylated interferon is used to treat chronic hepatitis B and hepatitis C, often in combination therapy. Common adverse effects include fatigue, flu-like symptoms, muscle and joint pain, and possible depression and insomnia (Adams et al., 2014; Wells et al., 2012).

Isolation precautions

Controlling the spread of infectious diseases in the hospital or long-term care setting is particularly important to preventing healthcare-associated infections. Hand hygiene remains the single most important factor in preventing the transmission of infections. Most infectious diseases are transmitted by either direct or indirect contact and their spread is prevented through the use of standard precautions. However, diseases such as chickenpox (caricella) are highly contagious and are spread by the airborne route, requiring special precautions to protect other hospitalised patients.

In determining the need for transmission-based precautions, healthcare personnel consider the usual reservoir or source of the microorganism, the mode of transmission and the susceptibility of hospital staff and other persons.

Standard precautions

The *Australian guidelines for the prevention and control of infection in healthcare* (NHMRC, 2010) describe *standard precautions* as work practices that are applied to everyone, regardless of their perceived or confirmed infectious status. The aim of standard precautions is to prevent or reduce the likelihood of transmission of infectious agents from one person or place to another and to maintain objects and areas as free as possible from infectious agents.

Standard precautions include the following work practices:

- Personal hygiene practices, particularly hand hygiene.
- Use of personal protective equipment (PPE) including gloves, plastic aprons and gowns, masks, face shields and eye protection.
- Appropriate handling and disposal of sharps.
- Environmental controls including cleaning and spills management.
- Appropriate processing of reusable equipment and instruments.
- Practising respiratory hygiene and cough etiquette.
- Aseptic non-touch technique.
- Appropriate handling of waste and linen.

Barrier protection (i.e. use of PPE) is used to prevent exposing skin and mucous membrane surfaces to blood and body fluids. Barrier protection involves using gloves for touching and handling body fluids, and adding other protection such as gowns, masks and goggles if splashing or spraying is likely. Use of aseptic technique, sterile single-use disposable needles and syringes, single-use vials for preparing and administering parenteral medication is emphasised. Needles and other sharp objects are not recapped or bent, but disposed of in puncture-proof containers to prevent inadvertent percutaneous (needle-stick) exposure.

Transmission-based precautions

In addition to the use of hand hygiene and standard precautions, additional work practices are recommended in situations where standard precautions are not sufficient to prevent transmission. Not all infectious diseases spread readily; however, diseases such as chickenpox (varicella) and pulmonary tuberculosis are highly contagious and are spread by the airborne route, requiring special precautions to protect other hospitalised people.

The NHMRC (2010) identifies three types of transmission-based precautions: contact, droplet and airborne precautions. Transmission-based precautions may be combined for diseases that have multiple routes of transmission. Indications for the use of transmission-based isolation precautions and the specific work practices to be taken are outlined in Table 11.9.

TABLE 11.9 Transmission-based precautions

CATEGORY	INFECTIOUS DISEASES	PURPOSE	PRECAUTIONS
Contact precautions	Acute diarrhoea, multidrug-resistant organisms (MROs), intestinal tract pathogens, *C. difficile*, highly contagious skin infections	Reduce risk of transmission by direct or indirect contact. Direct contact transmission involves physical transfer of organisms from skin and blood. Indirect contact involves contact with a contaminated object.	Hand hygiene. Private room with handwashing and toilet facilities. Gown and glove on entry to care areas and remove before leaving to provide barrier protection. Single-use or dedicated devices (or decontamination prior to use on another person).
Droplet precautions	Influenza, pertussis respiratory syncytial virus (RSV), norovirus, meningococcus	Reduce risk of droplet transmission of infectious agents. Droplet transmission involves contact of mucosal surfaces with respiratory droplets generated during coughing, sneezing, talking or procedures such as suctioning.	Hand hygiene. Private room with handwashing and toilet facilities. Surgical mask, goggles or face shields to be worn by everyone entering room.
Airborne precautions	Pulmonary tuberculosis, chickenpox (with contact precautions), measles, SARS	Reduce risk of airborne transmission of infectious agents. Airborne transmission occurs by dissemination of either airborne droplet nuclei or particles containing the infectious agent.	Hand hygiene. Private room with negative pressure or special ventilation not allowing air to circulate to general facility ventilation, with handwashing and toilet facilities. P2 respirators for everyone entering room.

Nursing care

Nursing management related to infectious disease focuses on health promotion, prevention and prompt identification and treatment.

Health promotion

Preventing infection requires education of healthcare personnel and the general public. Education includes understanding the importance of immunisations, guidelines for using antibiotics to prevent the development of drug-resistant microorganisms and how to prevent the spread of infection.

Check immunisation records for all family members and encourage them to keep immunisations up to date. Increase public awareness regarding appropriate antibiotic use. Guidelines for preventing the spread of infection to others include the following:

- Avoid crowds and contact with susceptible people, especially those who are immunosuppressed (e.g. people who have HIV infection, who are undergoing therapy for cancer or who have had an organ transplant).
- Use disposable tissues to contain respiratory secretions when coughing or sneezing.
- Use appropriate food-handling precautions for diseases spread via the faecal–oral route, such as hepatitis A.
- Avoid contact with or sharing of body fluids. For example, do not share needles or razors; use a condom during sexual activity or abstain; have each person clean their own blood spills or wounds, if possible.

Assessment

The following data are collected through the health history and physical examination. Further focused assessments are described with nursing interventions in the next section.

- *Health history*: age, medication use (antipyretics and anti-infectives), nutrition, exposure to infectious people, immunisations, invasive procedures and therapies, chronic diseases such as diabetes mellitus, cancer.
- *Physical assessment*: vital signs, body system(s) where infection is suspected, lymph node enlargement and tenderness.

Nursing diagnoses interventions

People with an infection may be managed in the hospital or at home. During the acute phase, nursing care includes administering prescribed antibiotics, implementing and maintaining aseptic technique and infection control measures, and encouraging a balance of rest and activity, good nutritional intake, and other general health measures to support immunological function and healing.

Risk of infection

The spread of infection is a risk in any facility that houses many people. It is a particular risk in hospitals, where many people have at least some degree of immunosuppression and

many drug-resistant strains of pathogens are prevalent. It is vital that nurses use good hand hygiene techniques at all times, employ standard precautions in all situations and use transmission-based precautions as indicated to prevent infectious spread to other people, themselves and their families.

- Admit people with known or suspected infections to a private room. *This is important to minimise the risk of infection transmission to others.*
- Practise effective hand hygiene techniques following the '5 moments' (Hand Hygiene Australia, 2009). Utilise alcohol-based hand rubs or, when hands are visibly soiled, wash hands with soap or antibacterial scrub solution and water using a minimum 15-second hand rub. *The appropriate technique for cleansing of hands is as equally important as the selection of correct product.*
- Use standard precautions and personal protective equipment to reduce the risk of transmission. *Gloves, gowns and masks are to be worn whenever there is a risk of skin or mucous membrane contamination by direct contact with infectious material, airborne spread of organisms or droplet nuclei.*
- Explain the reasons for and importance of transmission-based precautions during hospitalisation. *Application of transmission-based precautions may make the person feel neglected, dirty or shunned. Explanation of reasons and procedures can enhance the person's and family's understanding and acceptance.*
- Place a mask on the person and/or cover all infectious lesions or wounds completely when transporting the person to other parts of the facility for diagnostic or treatment procedures. *These measures help minimise air contamination and the risk to visitors and personnel.*
- Collect a culture and sensitivity (C&S) specimen as ordered or indicated by purulent drainage, pyuria or other manifestations of infection. *C&S is performed to determine the presence and type of infectious organisms, as well as the antibiotics most likely to be effective in eradicating them.*
- Administer prescribed anti-infective agents. *Anti-infectives are used to destroy the invading microorganism.*
- Inform all healthcare workers and visitors in contact with the person requiring transmission-based precautions. *Utilise appropriate signage at the entrance to the person's room with appropriate personal protective equipment at hand so that appropriate precautions can be taken.*
- Use appropriate measures for disposing of contaminated tissues, dressings or other material, and for removing soiled linen and equipment from the person's room. *Check hospital policy or published guidelines for appropriate disposal and cleaning procedures.*

CONSIDERATION FOR PRACTICE

Collect the specimen for culture and sensitivity (C&S) before the first dose of antibiotics is administered to ensure adequate organisms for culture.

- Teach the importance of complying with prescribed treatment for the entire course of the regimen. *Because anti-infective agents kill only a portion of the pathogen population with each dose, completion of the entire course of therapy is necessary to reduce the risk of relapse and of creating drug-resistant organisms.*

Anxiety

The person with an infectious disease may experience anxiety related to their manifestations, treatment measures, the prognosis and expected outcome of the disease. The diagnosis of an infection can be traumatic, causing feelings of uneasiness, isolation, guilt (e.g. in regard to sexually transmitted infections), apprehension or depression.

- Assess level of anxiety. *The level of anxiety influences the person's response to and interpretation of the situation and the degree of threat it poses.*
- Discuss the infection, treatments, prognosis and outcomes. *Discussions help to allay fears and misconceptions.*
- Support and enhance the person's coping strategies. *A person uses intrapersonal and interpersonal mechanisms to reduce or relieve anxiety.*
- Include significant others in the plan of care. *Inclusion of family members or significant others promotes understanding and compliance supporting reassurance and confidence.*
- Explain transmission-based precautions and answer any concerns. *Separation may be necessary to prevent the spread of infection and could cause great anxiety for the person and family members.*
- Provide referrals as needed for continuing care—for example, to home health agencies or for dressing changes or periodic assessment. *Referrals are often necessary to provide ongoing interventions and to maintain continuity of care.*

Hyperthermia

Hyperthermia is an expected consequence of the infectious disease process. Fever may produce mild, short-term effects or, when prolonged, may cause serious life-threatening effects.

- Monitor temperature, especially during episodes of chills; note heart rate and rhythm. *Chills indicate a rising temperature. Hyperthermia can cause arrhythmias.*
- Administer prescribed antipyretic as indicated for elevated temperature. Although antipyretics lower the temperature and enhance comfort for the person, this benefit must be weighed against the possible beneficial effect of an elevated temperature in the immune response. *Fever increases the motility and activity of WBCs, stimulates the production of interferon and activates T cells. In addition, temperatures above the normal range inhibit the growth of many microorganisms* (Grossman & Porth, 2014).
- Promote body cooling through lowering the room temperature. *Rapid cooling stimulates the hypothalamus to increase the body's temperature; this increases both shivering and metabolic rate.*
- Monitor fluid loss; encourage increased fluid and electrolyte intake either orally or intravenously. *Hyperthermia causes fluid loss from evaporation and may result in dehydration and electrolyte imbalance.*

- If diaphoretic, bathe and provide dry clothing and bedding. *These measures increase comfort and decrease further water evaporation.*
- Promote rest periods. *Rest increases energy reserve that is depleted by an increased metabolic, heart and respiratory rate.*

CONSIDERATION FOR PRACTICE

Use ice packs, cool/tepid baths or fans with caution to prevent unnecessary shivering.

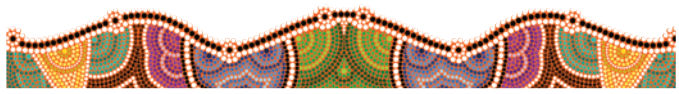
Acute pain

Pain often accompanies infections as part of the inflammatory process or secondary to delayed healing. Increasing pain in a wound may signal infection, especially if accompanied by erythema or purulence. *Keep the wound clean and dry and administer prescribed antibiotics to promote healing and decrease pain.*

Community-based care

Health education for the person and family is directed towards helping the person recover from the infection or disease, preventing its spread to others and preventing life-threatening complications. Instructions should include the following points:

- Promote the use of effective hand hygiene techniques, particularly after touching infected wounds or lesions, coughing, sneezing, blowing the nose or using the bathroom. Wash hands thoroughly before performing any procedures such as dressing changes. Wash hands with soap and water before and after preparing food or eating, using the toilet or handling nappies. Do not share eating utensils.
- Take all prescribed antibiotics as ordered, even after symptoms have subsided. Take the prescription at intervals around the clock as directed.
- Never allow anyone else to use your medications and never use anyone else's prescription even if they appear to be the same.
- Notify your healthcare provider in the following cases:
  - Symptoms do not improve within 24 to 48 hours after antibiotic therapy is instituted, or they worsen.
  - Signs of antibiotic allergy (itching, rash, difficulty breathing or swallowing, swelling of the face or tongue) occur. Discontinue medication and contact prescriber.
  - Adverse responses, such as gastrointestinal distress, that interfere with completion of the prescription.
  - Manifestations of infection that recur after completing prescribed antibiotic.
- Report redness, swelling or drainage around wounds, or persistent high fever.
- Increase fluid intake to at least 2500 mL per day.
- Report any signs of opportunistic infections: loose, watery and foul-smelling diarrhoea; vaginal discharge or itching; fuzzy growth or white plaques in mouth or on tongue; blood in urine; chills, fever or unusual cough.
- In addition, suggest the following resources:
  - state public health department
  - Australian Government Department of Health.



CHAPTER HIGHLIGHTS

- Innate immunity, a non-specific response to tissue injury, and the adaptive immune response, which directly targets invading microorganisms and invading cells, are critical components of the body's defences. Supporting these defences is a key nursing responsibility in promoting patient health.
- Both natural barriers and the immune system prevent the invasion and replication of pathogens.
- The adaptability and specificity of immune responses is possible because immune cells are genetically encoded to capture pathogens, move them to lymph nodes and develop specific immune reactions to destroy them.
- The inflammatory response serves to isolate invading antigens. When it occurs in response to acute injury, inflammation produces discomfort but serves a protective role. In contrast, chronic inflammation can damage affected tissue and may serve no protective function.
- Inflammation is a protective mechanism designed to prevent pathogens from entering the bloodstream and populating functional tissues such as heart, liver and kidney. Pain acts as a signal that tissue has been damaged and stimulates protective responses, such as cleansing wounds and limiting function, while healing progresses. Healing occurs as the inflammatory process isolates the injury and repairs damaged tissue.
- A fully immunised population is an important infection control strategy and a major factor in maintaining the health of individuals and the population as a whole.
- Nurses are instrumental in protecting vulnerable patients from infection, identifying early manifestations of infection, participating with the interprofessional team in treating infection and educating patients and their families about effective treatment of infection.
- Localised infections may damage tissue and create pain, but systemic infections are life threatening. Unfortunately, hospitals are hazardous environments populated with collections of pathogens. Healthcare-associated infections are often introduced into the body by medical procedures.
- Hygiene, protection from harm and nutrition support the immune defences. Antimicrobial medications limit the spread of pathogens but can lose their effectiveness when microbes mutate and develop resistance.

CONCEPT CHECK

1 When a person receives gamma globulin following exposure to hepatitis A, the nurse expects the person to develop:

1 natural active immunity
2 natural passive immunity
3 acquired active immunity
4 acquired passive immunity

2 The nurse is caring for the person with an infection. Which nursing action is a priority when providing the prescribed treatment?
1 Administer prescribed anti-infective.
2 Obtain specimen for culture and sensitivity.
3 Assess for history of hypersensitivities and allergies.
4 Monitor for reaction to prescribed anti-infective.

3 The nurse is providing medications to a patient with an inflammation. Which medication provided by the nurse will inhibit prostaglandin synthesis?
1 aspirin
2 penicillin
3 morphine sulfate
4 warfarin

4 While reviewing a patient's recent complete blood count, the nurse notes a large percentage of banded neutrophils. What does this finding indicate to the nurse?
1 renal failure
2 acute infection
3 hyperthyroidism
4 autoimmune disorder

5 A person is admitted with methicillin-resistant *Staphylococcus aureus* in a draining sacral wound. Which type of precaution should the nurse implement for this person?
1 droplet precautions
2 contact precautions
3 airborne precautions
4 protective precautions

6 A patient with a systemic inflammation is resting in bed, periodically sleeping, and wants additional blankets. Which part of the immune system is responsible for this patient's illness behaviour?
1 interferons
2 phagocytes
3 complement system
4 inflammatory cytokines

7 A patient is diagnosed with neutrophilia. What does this finding indicate to the nurse?
1 a decrease in total white blood cells
2 a decrease in circulating neutrophils
3 an increase in circulating neutrophils
4 an expected average number of white blood cells

8 The nurse is preparing discharge instructions for a person with an inflammation who is at risk for infection. What should the nurse include in the teaching?
1 Limit daily intake of calories.
2 Apply heat for 20 minutes at a time.
3 Resume normal activities of daily living.
4 Take prescribed antibiotics until fever drops.

9 The nurse is caring for an older patient recovering from an acute illness. Which intervention should the nurse implement to reduce the person's risk of developing a healthcare-associated infection?
1 Teach the patient to restrict fluids throughout the day.
2 Coach the patient to breathe deeply and cough frequently.
3 Recommend placement of an indwelling urinary catheter.
4 Wash hands with soap and water before entering the person's room.

10 The nurse is instructing unregistered health workers to use standard precautions when providing morning care to assigned patients. What should the nurse teach them specifically to do?
1 Perform hand hygiene, wear masks and recap needles.
2 Apply a mask and gown, and spray working surfaces with disinfectant.
3 Apply gloves, gowns and goggles if coming in contact with body fluids.
4 Wash hands with alcohol-based hand rub for visibly dirty or blood-contaminated hands.

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CHAPTER 12

NURSING CARE OF PEOPLE WITH ALTERED IMMUNITY

CATHERINE BETHELL

LEARNING OUTCOMES

- Review the normal anatomy and physiology of the immune system.
- Describe the four types of hypersensitivity reactions.
- Discuss the pathophysiology of autoimmune disorders and tissue transplant rejection.
- Discuss the characteristics of immunodeficiencies.
- Identify laboratory and diagnostic tests used to diagnose and monitor immune response.
- Describe pharmacological and other collaborative therapies used in treating people with altered immunity.
- Correlate the pathophysiological alterations with the manifestations of HIV/AIDS infection.

CLINICAL COMPETENCIES

- Assess functional health status of people with altered immunity and monitor, document and report abnormal manifestations.
- Use evidence-based practice to plan and implement nursing care for people with AIDS.
- Assess for hypersensitivities and anticipate treatment if signs and symptoms develop.
- Provide education about hypersensitivities, avoidance of sensitising agents and prophylactic treatment.
- Determine priority nursing diagnoses, based on assessment data, to select and implement individualised nursing interventions and education for people with altered immunity.
- Protect people who are immunosuppressed.
- Recognise manifestations of developing anaphylaxis.
- Recognise manifestations of infection and minimise healthcare-associated exposure.
- Utilise standard precautions to protect self and other people from HIV exposure.
- Recognise the burden and benefit of highly active antiretroviral therapy (HAART) for the person with HIV infection.
- Integrate interprofessional care into care of the person with altered immunity.
- Revise plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to people with altered immunity.

KEY TERMS

OVERVIEW OF THE IMMUNE SYSTEM

Recent years have seen the emergence of new diseases affecting the immune system. These diseases include human immunodeficiency virus (HIV) infection and altered strains of familiar diseases such as multidrug-resistant tuberculosis. At the same time, our understanding of the components of the immune system and specific immune responses is increasing. Therefore, it is vital that today's nurses understand the foundations of the immune system and the immune response.

The immune system functions to protect the body from invasion by foreign antigens, to identify and destroy potentially harmful cells and to remove cellular debris. These functions are accomplished by the lymphoid organs and specifically designed lymphocytes through the processes of antibody-mediated immune response and cell-mediated immune response.

The effectiveness of the immune system depends on its ability to differentiate normal host tissue from abnormal or foreign tissue. Body cells, tissues and fluids have unique antigenic properties recognised by the immune system as 'self'. External agents, such as microorganisms, cells and tissues from other humans or animals and some inorganic substances, have antigenic properties recognised by the immune system as 'non-self'.

Each body cell displays specific cell surface characteristics, or markers, that are unique to each person. These are known as human leucocyte antigens (HLAs). A person's HLA characteristics are coded within a large cluster of genes known as the major histocompatibility complex (MHC), located on chromosome 6. Recall that chromosomes are paired; each person inherits one member of the pair from each parent. A chromosome pair contains multiple genes, each carrying instructions for production of one polypeptide chain. The number of genes in the MHC results in a multitude of HLA combinations. As a result, the possibility of two people having the same HLA type is extremely remote. Identical twins may be the exception and some siblings have very similar HLA patterns. In tissue grafting and organ transplants, matching the HLA type as closely as possible tends to decrease rejection.

Immunocompetent people have an immune system that identifies antigens and effectively destroys or removes them. When the immune system functions improperly, the result may be an overreaction or deficiency, resulting in health problems. Overreaction of the immune system leads to hypersensitivity disorders, such as allergies. When the immune system loses the ability to recognise self, autoimmune disorders may ensue (see Table 12.1). Immunodeficiency diseases or malignancies can develop when the immune system is incompetent or unable to respond effectively, as is the case with acquired immunodeficiency disorder. These alterations in immunity are discussed later in this chapter.

As discussed previously in Chapter 11, the antibody-mediated immune response is accomplished by B lymphocytes (B cells) that are further divided into memory cells and plasma cells. They are activated by contact with an antigen and by T cells. B cells produce antibodies, also known as immunoglobulins (see Table 11.3), and serve to inactivate an invading antigen. One immunoglobulin in particular, IgM, forms natural antibodies, such as those for ABO blood group antigens, and is an important component of the immune system complexes seen in autoimmune disorders. Memory cells 'remember' an antigen and, when exposed to it a second time, immediately initiate the immune response. This action provides the foundation of acquired immunity.

In contrast, cell-mediated immunity acts at the cellular level by attacking antigens directly and by activating B cells. T lymphocytes comprise the cell-mediated immune response and are subdivided into effector cells and regulator cells. The cytotoxic cell or killer T cell is the primary effector cell. Regulator T cells are divided into two subsets known as helper T cells and suppressor T cells.

Proteins on the surface of the T cell help define its function and also provide a marker that can be used to identify the cell class. These proteins are known as the cluster of differentiation antigen or CD antigen. The two primary CD proteins are CD4 and CD8. Both cytotoxic and suppressor T cells carry the CD8 antigen. Helper T cells have the CD4 antigen and are often called CD4 cells. CD4 cells are the most numerous of the T lymphocytes, making up 70% of the circulating population.

Helper T cells initiate the immune response, whereas suppressor T cells limit it. Helper T cells accomplish their role by promoting growth of additional T cells, by stimulating proliferation of B cells and by activating killer T cells. It is believed that suppressor T cells are important in preventing autoimmune disorders. Proper immune system function depends on the correct balance between helper and suppressor T cells.

In addition to destroying viruses and bacteria, cytotoxic T lymphocytes also attack malignant cells. They also are responsible for the rejection of transplanted organs and grafted tissues.

CHANGES IN IMMUNE FUNCTION IN THE OLDER ADULT

Immune function declines with ageing, although many of the mechanisms leading to this decline are not clear. External factors, such as nutritional status and the effects of chemical exposure, ultraviolet radiation and environmental pollution, affect the older adult's immune status. Internal factors affect it as well, including genetics, the function of the neurological and endocrine systems, chronic and prior illnesses, and individual anatomical and physiological variations. These influences make it difficult to determine the effect of ageing on the immune system. In some older individuals, the immune system is as effective as that of a younger person.

Whereas the antibody response to foreign antigens is diminished, autoantibodies (antibodies that react to the person's own tissues) are more common in older people. The presence of autoantibodies suggests impaired regulation of the immune system, but it is not associated with an increased incidence of autoimmune disorders (Murasko & Gardner, 2003). The hypersensitivity response is also reduced or delayed.

TABLE 12.1 Selected autoimmune disorders

More organ specific	Hashimoto's thyroiditis	A chronic progressive inflammatory disease of the thyroid with lymphocyte infiltration and gradual destruction of the gland. See Chapter 18.
	Primary myxoedema	Thyroid deficiency resulting from destruction of the thyroid gland due to an autoimmune process, often Hashimoto's thyroiditis. See Chapter 18.
	Thyrotoxicosis	Hyperthyroidism resulting from thyroid-stimulating immunoglobulins that stimulate activity of the gland. See Chapter 18.
	Pernicious anaemia	Anaemia resulting from absence of intrinsic factor associated with loss of parietal cells; most people have antibodies to parietal cells. See Chapter 32.
	Addison's disease	Atrophy and hypofunction of the adrenal cortex, probably autoimmune in origin. See Chapter 18.
	Myasthenia gravis	A disease characterised by episodic muscle weakness caused by antibodies to the acetylcholine receptor of the neuromuscular junction. See Chapter 43.
	Insulin-dependent diabetes mellitus	Impaired insulin secretion, often the result of islet cell destruction by antibodies directed at the cell surface or cytoplasm. See Chapter 19.
	Goodpasture's syndrome	A type II hypersensitivity disorder with pulmonary haemorrhage and progressive glomerulonephritis characterised by circulating antiglomerular basement membrane antibodies. See Chapter 27.
	Multiple sclerosis	A probable autoimmune process resulting in disseminated patches of demyelination in the brain and spinal cord and varied neurological manifestations. See Chapter 43.
	Idiopathic thrombocytopenic purpura	A chronic disorder characterised by petechiae, purpura, mucosal bleeding and antibodies against platelets. See Chapter 32.
	Primary biliary cirrhosis	Inflammation and fibrosis of the bile ducts, probably of autoimmune origin. See Chapter 24.
	Active chronic hepatitis	A serious liver disease often resulting in hepatic failure and/or cirrhosis; may be autoimmune with infiltration by T cells and plasma cells. See Chapter 24.
Less organ specific	Ulcerative colitis	A chronic inflammatory disease of colon mucosa, possibly of autoimmune origin. See Chapter 23.
	Sjögren's syndrome	A systemic inflammatory disorder characterised by dryness of the mouth, eye and other mucous membranes with lymphocyte infiltration of affected tissues. See Chapter 39.
	Rheumatoid arthritis	A chronic syndrome with inflammation of peripheral joints and generalised manifestations, characterised by infiltration of synovium by lymphocytes and plasma cells. See Chapter 39.
	Scleroderma	Diffuse fibrosis, degenerative changes and vascular abnormalities of skin, joint structures and internal organs; probably of autoimmune origin. See Chapter 39.
Non-organ specific	Systemic lupus erythematosus	An inflammatory connective tissue disorder characterised by the presence of antinuclear antibodies. See Chapter 39.

ASSESSMENT OF ALTERED IMMUNE SYSTEM FUNCTION

Unlike body systems that are composed of a few closely related organs, the immune system is diverse and scattered. Optimal immune function depends on intact skin and mucous membrane barriers, adequate blood cell production and differentiation, a functional system of lymphatics and the spleen, and the ability to differentiate foreign tissue and pathogens from normal body tissue and flora. Because of this diversity of organs and function, assessment of the immune system is often integrated throughout the history and physical examination.

Health history

Prior to interviewing the person, review the biographical data, including age, sex, race and ethnic background. This information can provide valuable clues about possible immunological disorders. For example, many autoimmune disorders are more prevalent in women than in men. Family history is also important because there is a genetic component in the aetiology of many disorders affecting the immune system.

Many interview questions related to the immune system and disorders that affect it are of a sensitive nature. Be sure to provide for privacy prior to the interview. If family members are present, request that they leave as well. Establish a trusting relationship with the person prior to asking the most sensitive questions (e.g. those related to the use of illicit drugs or sexual activity). Epidemiological data show that social and racial groups have peculiar risks for HIV infection; cultural sensitivity is necessary for effective communication.

Physical assessment

The techniques of inspection and palpation are especially important in assessing a person's immune system.

- Assess the general appearance. Note whether the person's stated and apparent age coincide. Evident fatigue or weakness may indicate acute or chronic illness or immunodeficiency. Assess height, weight and body type for apparent weight loss or wasting. Observe ease of movement and note any evident stiffness or difficulty moving. Check vital signs. An elevated temperature may indicate an infection or inflammatory response.
- Inspect the mucous membranes of the nose and mouth for colour and condition. Pale, boggy (oedematous) nasal mucosa is often associated with chronic allergies. Note petechiae, white patches or lacy white plaques, in the oral mucosa; they may indicate haemolysis or immunodeficiency.
- Assess skin colour, temperature and moisture. Pale or jaundiced skin may indicate a haemolytic reaction. Pallor may also indicate bone marrow suppression with accompanying immunodeficiency. Inspect the skin for evidence of rashes or lesions, such as petechiae, numerous bruises, purple or blue patches, or lesions indicative of Kaposi's sarcoma, and wounds that are infected, inflamed or unhealed. Note the location and distribution of any rashes or lesions.
- Inspect and palpate the cervical lymph nodes for evidence of lymphadenopathy (swelling) or tenderness. Palpate the nodes of the axillae and groin as well.
- Assess the musculoskeletal system by inspecting and palpating the joints for redness, swelling, tenderness or deformity. Such changes may indicate an autoimmune disorder such as rheumatoid arthritis or systemic lupus erythematosus. Check joint range of motion as well, including that of the spine.

ALTERED IMMUNE RESPONSES

Considering the complexity of the immune system, it is not surprising that abnormal or harmful responses occur. Altered immune system responses include those characterised by hyperresponsiveness of the immune system and those characterised by an impaired immune response. Allergies, autoimmune disorders, and reactions to organ or tissue transplants are all examples of hyperresponsive immune function. AIDS and other immunodeficiency disorders result from impairment of the immune system.

THE PERSON WITH A HYPERSENSITIVITY REACTION

Hypersensitivity is an altered immune response to an antigen that results in harm to the person. When the antigen is environmental or exogenous, it is called an **allergy** and the antigen is referred to as an *allergen*. The tissue response to a hypersensitivity reaction may be simply irritating or bothersome, causing a runny nose or itchy eyes, or it may be life threatening, leading to blood cell haemolysis or laryngospasm.

Hypersensitivity reactions are primarily classified by the type of immune response that occurs on contact with the allergen. They may also be classified as immediate or delayed hypersensitivity responses. Anaphylaxis and transfusion reactions are examples of immediate hypersensitivity reactions; contact dermatitis is a typical delayed response. Allergies are sometimes referred to by the affected organ system (e.g. allergic rhinitis) or the allergen involved, as in hay fever. Classification by immunological response is the preferred means of studying allergies. Although more than one type of reaction may occur simultaneously, it is practical and insightful to study and treat allergy by classified types (King et al., 2005). The prevalence of allergy in Australia is one of the highest in the developed world, with 19.6% of the population having at least one allergy (Australasian Society of Clinical Immunology and Allergy (ASCIA), 2007).

Pathophysiology

In a hypersensitivity reaction, an antigen–antibody or antigen–lymphocyte interaction causes a response that is damaging to body tissues. Antigen–antibody responses characterise types I, II and III, also known as immediate hypersensitivity responses. Type IV hypersensitivity is an antigen–lymphocyte reaction, resulting in a delayed hypersensitivity response.

Type I: IgE-mediated hypersensitivity

Common hypersensitivity reactions, such as allergic asthma, allergic rhinitis (hay fever), allergic conjunctivitis, hives and anaphylactic shock, are typical of type I or IgE-mediated hypersensitivity. This type of hypersensitivity response is triggered when an allergen interacts with IgE bound to mast cells and basophils. The antigen–antibody complex prompts release of histamine and other chemical mediators, complement, acetylcholine, kinins and chemotactic factors (see Figure 12.1).

When a potent allergen such as bee or wasp venom or a drug is injected, resulting in widespread antibody–antigen reaction and response to these chemical mediators, a systemic response such as anaphylaxis, urticaria or angio-oedema results.

Anaphylaxis is an acute systemic type I response that occurs in highly sensitive people following injection of a specific antigen. Substances known to trigger anaphylaxis are summarised in Box 12.1. Anaphylaxis rarely follows oral ingestion, although this is possible. The reaction begins within minutes of exposure to the allergen and may be almost instantaneous. The release of histamine and other mediators causes vasodilation and increased capillary permeability, smooth muscle contraction and bronchial constriction. These chemical mediators cause the person to experience the typical manifestations of anaphylaxis. Initially, a sense of foreboding or uneasiness, light-headedness and itching palms and scalp may be noted. Hives may develop, along with angio-oedema (localised tissue swelling) of the eyelids, lips, tongue, hands, feet and

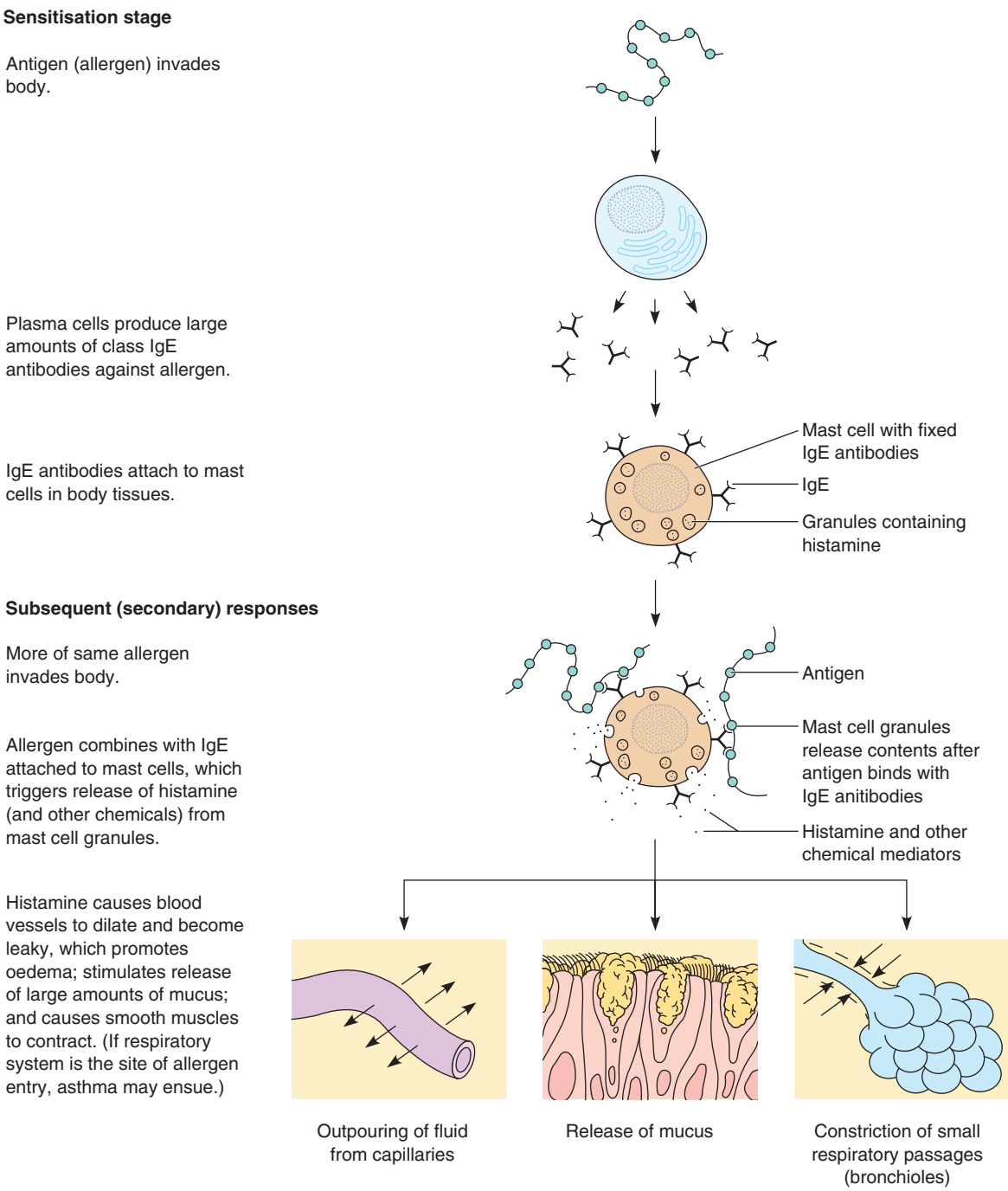


FIGURE 12.1 ■ Type I or IgE-mediated hypersensitivity response

genitals. Swelling can also affect the uvula and larynx, impairing breathing. This is further complicated by bronchial constriction. The person exhibits air hunger, stridor and wheezing, and a barking cough. These respiratory effects can be lethal if the reaction is severe and intervention is not immediately available. Vasodilation and fluid loss from the vascular system can lead to impaired tissue perfusion and hypotension, a condition known as *anaphylactic shock*.

Fortunately, localised responses are more common manifestations of type I hypersensitivity. These are typically atopic responses; that is, they have a strong genetic predisposition. Atopic reactions are the result of localised, rather than systemic, IgE-mediated responses to an allergen. They are prompted by contact of the allergen with cell-bound IgE in the bronchial tree, nasal mucosa and conjunctival tissues. Chemical mediators are released locally, producing symptoms such as asthma, allergic rhinitis (hay fever), conjunctivitis or atopic dermatitis. Allergens commonly associated with atopic reactions of this type include pollens, fungal spores, house dust mites, animal dander and feathers (Porth & Matfin, 2009). Food allergens can also cause localised responses such as diarrhoea or vomiting. If the gastrointestinal mucosa is altered by a

BOX 12.1 Substances known to trigger anaphylaxis in sensitised people

Hormones
- Insulin
- Vasopressin
- Parathormone

Enzymes
- Trypsin
- Chymotrypsin
- Penicillinase

Pollens
- Ragweed
- Grass
- Trees

Foods
- Eggs
- Seafood
- Nuts
- Grains
- Beans
- Chocolate
- Dairy products
- Royal jelly

Vitamins
- Thiamine
- Folic acid

Insect venom
- Australian native ants (Jack Jumper ant)
- Wasps
- Honey bee
- Tick

Occupational agents
- Rubber products
- Industrial chemicals (ethylenes)

Antibiotics
- Penicillins
- Cephalosporins
- Amphotericin B
- Nitrofurantoin

Local anaesthetics
- Procaine
- Lidocaine

Medical diagnostic agents
- Sodium dehydrocholate
- Sulfobromophthalein

Antiserum
- Antilymphocyte gamma globulin

local allergic response, then the allergen may be absorbed, leading to a systemic reaction. Urticaria (hives) is the most common systemic response to food allergies.

Type II: cytotoxic hypersensitivity

Cytotoxic hypersensitivity reactions are characterised by formation of IgG or IgM antibodies against normal or foreign cells or tissues (Grossman & Porth, 2014; Haynes et al., 2012). A haemolytic transfusion reaction to blood of an incompatible type is characteristic of a type II or cytotoxic hypersensitivity reaction. IgG or IgM type antibodies are formed to a cell-bound antigen such as the ABO or Rh antigen. When these antibodies bind with the antigen, the complement cascade is activated, resulting in destruction of the target cell (see Figure 12.2). Haemolytic disease of the newborn is caused by this type of reaction.

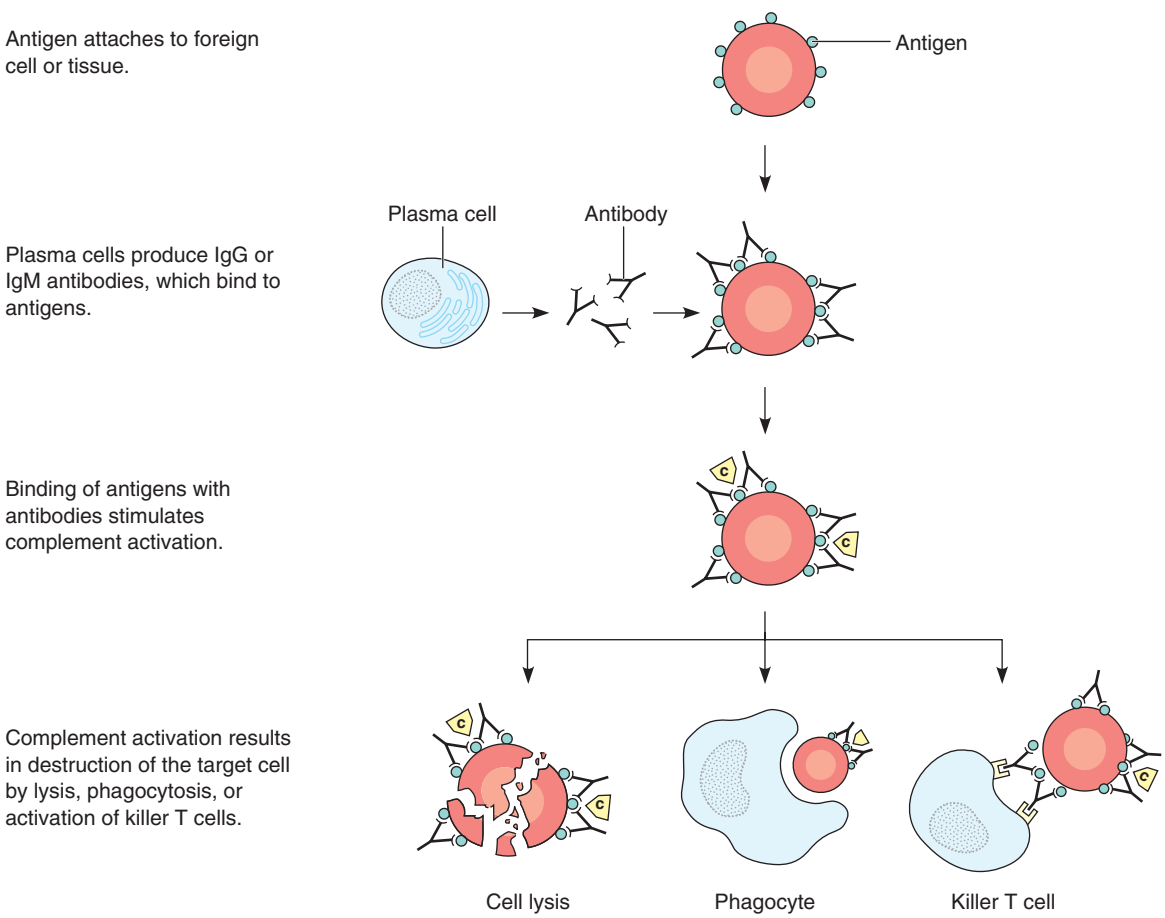


FIGURE 12.2 ■ Type II or cytotoxic hypersensitivity response

Type II reactions may be stimulated by an exogenous antigen, such as foreign tissue or cells, or a drug reaction in which the drug forms an antigenic complex on the surface of a blood cell, stimulating the production of antibodies. The affected cell is then destroyed in the resulting antigen–antibody reaction; for example, haemolytic anaemia is sometimes associated with the administration of drugs such as penicillins, cephalosporins and streptomycin. Withdrawal of the drug stops the reaction and cell destruction (Goldsby et al., 2003).

Endogenous antigens can also stimulate a type II reaction, resulting in an autoimmune disorder such as Goodpasture's syndrome, in which antigens are formed to specific tissues in the lungs and kidneys. Hashimoto's thyroiditis and autoimmune haemolytic anaemia are additional examples of autoimmune type II reactions.

Type III: immune-complex-mediated hypersensitivity

Type III hypersensitivity reactions result from the formation of IgG or IgM antibody–antigen immune complexes in the circulation. When these complexes are deposited in vessel walls and extravascular tissues, complement is activated and chemical mediators of inflammation such as histamine are released. Chemotactic factors attract neutrophils to the site of inflammation. When neutrophils attempt to phagocytise the immune complexes, lysosomal enzymes are released, increasing tissue damage (see Figure 12.3).

Either systemic or local responses may be seen with type III reactions. For example, serum sickness is a systemic response, so named because it was first identified after administration of foreign serum (e.g. horse antitetanus toxin). Although foreign serums are no longer administered, serum

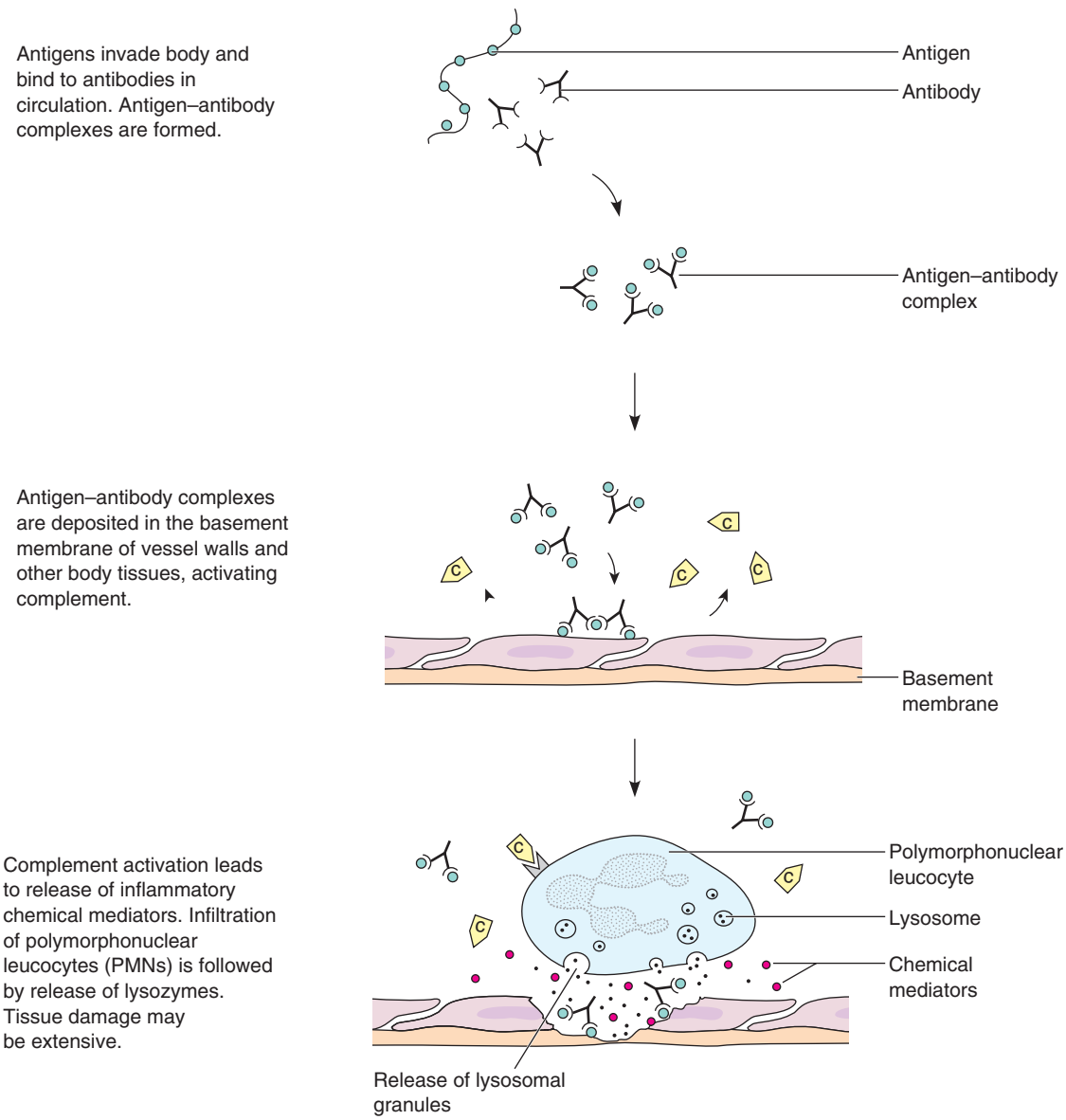


FIGURE 12.3 ■ Type III or immune-complex-mediated hypersensitivity response

sickness still occurs in response to some drugs, such as penicillin and sulfonamides. Immune complexes are deposited in walls of small blood vessels, the kidneys and joints. Manifestations of serum sickness include fever, urticaria or rash, arthralgias, myalgias and lymphadenopathy.

Localised responses may occur at a number of different sites. As immune complexes accumulate in the glomerular basement membrane of the kidneys—for example, following a streptococcal infection or with systemic lupus erythematosus—glomerulonephritis develops. When an antigen such as dust from mouldy hay is inhaled, an acute alveolar inflammatory response can occur. This condition can develop in agricultural workers.

Type IV: delayed hypersensitivity

Type IV reactions differ from other hypersensitivity responses in two ways. First, these reactions are cell mediated rather than antibody mediated, involving T cells of the immune system. Second, type IV reactions are delayed rather than immediate, developing 24 to 48 hours after exposure to the antigen. Type IV hypersensitivity responses result from an exaggerated interaction between an antigen and normal cell-mediated mechanisms. This exaggerated interaction results in the release of soluble inflammatory and immune mediators (from the lysozymes within the macrophages) and recruitment of killer T cells, causing local tissue destruction (see Figure 12.4).

Contact dermatitis is a classic example of a type IV reaction. Intense redness, itching and thickening affect the skin in the area exposed to the antigen. Fragile vesicles are often present as well. Many antigens can provoke this response; poison ivy is a prime perpetrator. In the healthcare setting, an allergic response to latex can also produce contact dermatitis. An estimated 8% to 13% of healthcare workers are allergic to latex (National Institute of Occupational Safety and Health (NIOSH), 2005), and recent Australian studies indicate the likely increase in the risk of latex allergy in Australian healthcare workers due to occupational use of latex gloves (ASCIA, 2010). Other examples of cell-mediated responses include a positive tuberculin test and graft rejection episodes.

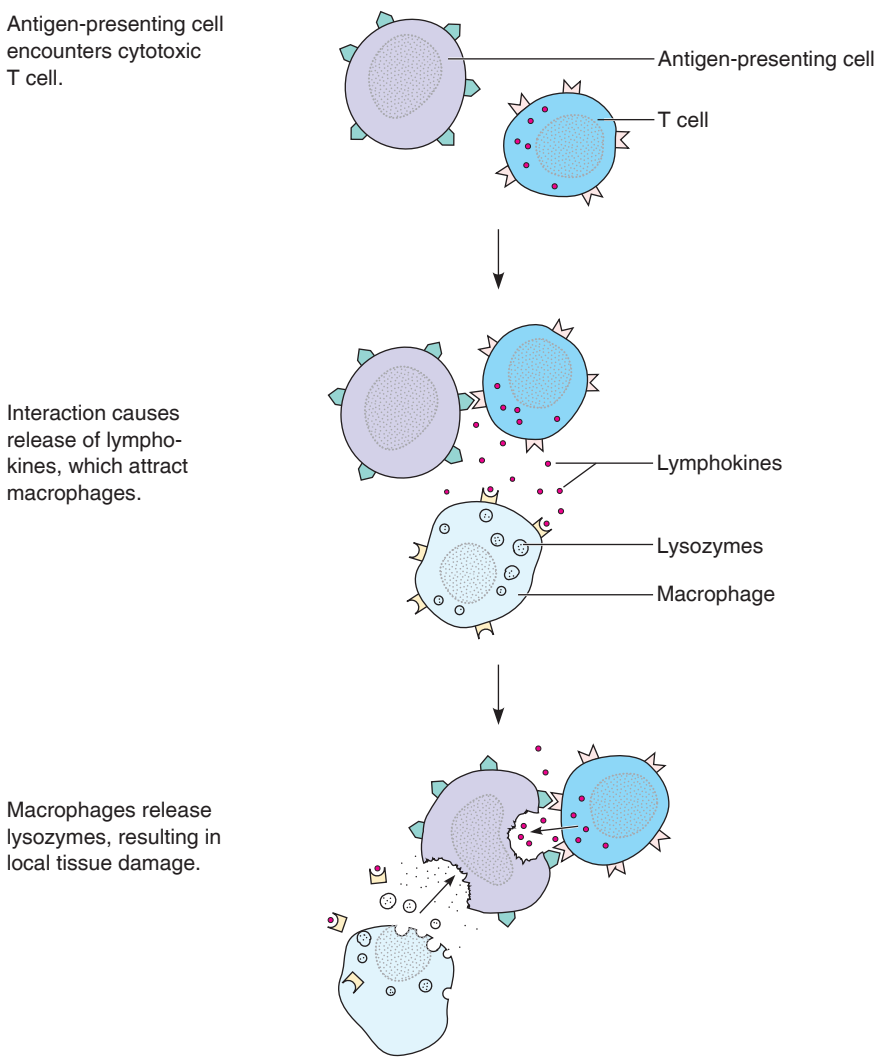


FIGURE 12.4 ■ Type IV or delayed hypersensitivity response

Latex allergy

Although protective against infection, the repetitive use of latex gloves creates a persistent exposure to latex for healthcare workers. Handwashing after using latex products limits exposure. When gloves are powdered with cornstarch to facilitate donning and removal, the cornstarch particles aerosolise when the gloves are removed. The cornstarch includes latex particles. This creates a respiratory exposure as well as dermal exposure to latex. In addition, chemicals used in the manufacture of latex products may be irritating. Products such as balloons, condoms and rubber bands are commonly made of latex.

Sensitivity to latex develops without the user being aware until a rash appears on the hands. Type IV hypersensitivity (contact dermatitis) can progress to type I systemic allergic reactions without previous symptoms signalling an escalation. It is important to protect the person and the healthcare worker who is allergic to latex. Prevention is aided by employers who select products free of latex (vinyl, nitrile, neoprene or polyurethane). Non-latex and synthetic gloves must be used. ASCIA (2010) provides strategies and guidelines for the management of latex-allergy individuals specific to various Australian healthcare settings.

INTERPROFESSIONAL CARE

The focus of care for people with allergic responses is on the following:

- Minimise exposure to the allergen.
- Prevent a hypersensitivity response.
- Provide prompt, effective interventions for allergic responses when they occur.

Identifying allergens for the individual to reduce the likelihood of exposure is a key aspect of management. A complete history of the person's allergies is obtained, including medications, foods, animals, plants and other materials. The type of hypersensitivity response is documented, as is its onset, manifestations and usual treatment.

When a documented or suspected hypersensitivity reaction occurs, the allergen (e.g. intravenous medication or transfusion) is withdrawn immediately. With a type I hypersensitivity response, managing the person's airway takes highest priority, followed by maintenance of cardiac output. Type II hypersensitivity responses may necessitate aggressive management of bleeding or renal failure. A type III (immune complex) reaction is treated by removing the offending antigen and interrupting the inflammatory response.

With a hypersensitivity response, supportive care is important to relieve discomfort. This often involves the administration of selected antihistamine or anti-inflammatory medications. Other therapies, such as plasmapheresis, may be prescribed in selected instances, a procedure that involves the continual withdrawal and reinfusion of blood from the patient, during which time the blood is processed to remove the allergic components of the plasma portion.

Diagnosis

To identify possible allergens or hypersensitivity reactions, the following laboratory tests may be ordered:

- *White blood cell (WBC) count with differential* can detect high levels of circulating eosinophils. Normally, eosinophils constitute a very small percentage (1–4%) of the total WBCs. Eosinophilia, however, is often present in people with type I hypersensitivities.
- *Radioallergosorbent test (RAST)* measures the amount of IgE directed towards specific allergens. Test results are compared with control values and used to identify hypersensitivities. RAST poses no risk of an anaphylactic reaction. It is particularly useful in detecting allergies to some occupational chemicals and toxic allergens (Goldsby et al., 2003).
- *Blood type and crossmatch* are ordered prior to any anticipated transfusions. The person's ABO blood group and Rh status are determined. Two major antigens, designated A and B, may be present on RBCs. People with the A antigen are designated as blood type A; those with blood type B have the B antigen. When neither antigen is found on the RBCs, the person is identified as type O. A third major RBC antigen is the Rh antigen. People with this antigen are called Rh positive; those without are Rh negative. Because a blood transfusion is actually a transplant of living tissue, antigen matching is vital to prevent significant hypersensitivity reactions. Once blood type is determined, a sample of the person's blood is mixed with a sample of matching donor blood and observed for antigen–antibody reactions in the crossmatch portion of this test. Although this procedure greatly reduces the risk of a haemolytic transfusion reaction (type II hypersensitivity), it does not totally eliminate it.
- *Indirect Coombs' test* detects the presence of circulating antibodies (other than ABO antibodies) against RBCs. The person's serum is mixed with the donor's RBCs. If the person's serum contains antibodies to an RBC antigen, agglutination (clumping together) will occur. This is called a positive response. The normal value is negative or no agglutination. This test is also part of the crossmatch of a blood 'type and crossmatch'.
- *Direct Coombs' test* detects antibodies on the person's RBCs that damage and destroy the cells. This is used following a suspected transfusion reaction to detect antibodies coating the transfused RBCs. It can also identify haemolytic anaemia when the cause is unknown. In the direct Coombs' test, the person's RBCs are mixed with Coombs' serum, which contains antibodies to IgG and several complement components. Agglutination will occur if the person's RBCs are coated with antibodies, resulting in a positive test. As with the indirect Coombs' test, the normal test result is negative.
- *Immune complex assays* may be performed to detect the presence of circulating immune complexes in suspected type III hypersensitivity responses. The assays are particularly useful in diagnosing suspected autoimmune disorders. Non-specific assays of IgG-, IgM- and IgA-containing immune complexes, which do not detect specific antibodies, as well as specific antibody assays, may be done. The normal result is a test negative for circulating immune

complexes. A negative test does not, however, rule out an immune complex hypersensitivity response. In some cases, a negative result may indicate that the disease process has reached a later stage, in which complexes are no longer circulating but have initiated extensive tissue damage, such as glomerulonephritis (Fauci et al., 2008).

- *Complement assay* is also useful in detecting immune complex disorders. In these disorders, complement is, in effect, used up by the development of antigen–antibody complexes. Decreased levels are seen on examination. Both total complement level and amounts of individual components of the complement cascade can be determined.

SKIN TEST FOR ALLERGIES Skin tests are also used to determine causes of hypersensitivity reactions. These tests are used to identify specific allergens to which a person may be sensitive. Allergens for testing are selected according to the person's history. Test solutions made from extracts of inhaled, ingested or injected materials, such as pollens, mites, venoms or some drugs, are used for the prick test and intradermal testing. Epicutaneous testing (prick testing) is generally done first to avoid a systemic reaction; it is followed by intradermal testing of allergens with a negative response to prick testing (Papadakis, McPhee & Rabow, 2013).

If the large-dose intradermal tests were done intially, individuals highly allergic to a substance would be at increased risk of an anaphylactic reaction. Substances that cause a reaction to the prick test should not be tested intradermally.

- *Skin prick (epicutaneous or puncture) test:* a drop of diluted allergenic extract is placed on the skin and the skin is then pricked or punctured through the drop. With a positive test, a localised pruritic wheal and erythema occur. The response is maximal at 15 to 20 minutes.
- *Intradermal:* a small amount (just enough to create a wheal) of allergen extract at a 1:500 or 1:1000 dilution is injected on the forearm or intrascapular area. If several allergens are being tested, injections are spaced 0.7 to 1.25 cm apart. As control measures, plain diluent (negative control) and histamine (positive control) are also injected. If there is no response to a particular allergen at 15 to 20 minutes, the test is negative. The appearance of a wheal and erythema, with a wheal diameter at least 5 mm greater than that produced by the control, indicates a positive response (see Figure 12.5). Intradermal is not recommended for routine use for aeroallergens and food allergens, as it carries a greater risk of anaphylaxis (ASCIA, 2010).
- *Patch:* a 2.5 cm patch impregnated with the allergen (e.g. perfume, cosmetics, detergents or clothing fibres) is applied to the skin for 48 hours. Absence of a response indicates a negative test result. Positive responses are graded from mild (erythema in the exposed area) to severe (erythema, papules, vesicles or ulceration).
- *Food allergy testing* is performed when a food allergy is suspected but the source or implicated food item has not been clearly identified. Food allergy symptoms are typically demonstrated within hours of eating. Initially, the person is asked to keep a diary of foods consumed and allergic responses for a week. An elimination diet is then prescribed. The diet excludes most common food allergens and all suspected foods for one week. Any foods that may contain allergens in combination, such as breads, are also eliminated. If symptoms do not improve, a different variation of the elimination diet is prescribed. If symptoms are relieved, foods are reintroduced to the diet one at a time until symptoms recur, indicating allergy to that food.

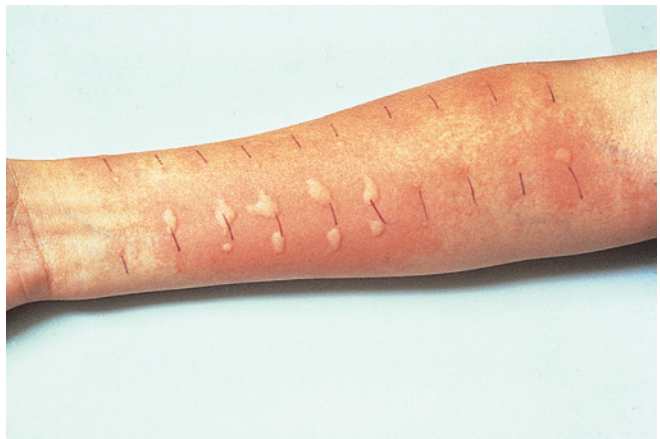


FIGURE 12.5 ■ Skin testing on the forearm showing induration and erythema typical of a positive response to an antigen

Source: © Southern Illinois University/Photo Researchers, Inc.

Medications

When it is impossible to avoid the offending allergen and allergic manifestations are severe or disrupt the person's activities of daily living (ADLs), pharmacological intervention is prescribed. *Immunotherapy*, also called hyposensitisation or desensitisation, consists of injecting an extract of the allergen(s) in gradually increasing doses. Immunotherapy is used primarily for allergic rhinitis or asthma related to inhaled allergens. It has also been shown to be effective in preventing anaphylactic responses to insect venom. With weekly or biweekly subcutaneous injections of the allergen, the person develops IgG antibodies to the allergen that appear to block effectively the allergic IgE-mediated response. Once a therapy plateau is reached, injections are continued indefinitely either monthly or bimonthly.

Antihistamines are the major class of drugs used in treating the symptoms of hypersensitivity responses, type I in particular. They are also useful to some extent in relieving manifestations (such as urticaria) of some type II and type III reactions.

Antihistamines block H1-histamine receptors, acting as a competitive antagonist to histamine, but they do not affect the production or release of histamine. The prototype antihistamine is diphenhydramine (Benadryl). It and other antihistamines alleviate the systemic effects of histamine such as urticaria and angio-oedema. They are also useful in relieving allergic rhinitis, although they are not effective in all people. Antihistamines are available in both prescription and nonprescription preparations. The preferred route of administration is oral, although diphenhydramine and others can be given parenterally, particularly when immediate action is needed, as

in anaphylaxis. They also dry respiratory secretions through an anticholinergic effect. Their use is limited by their side effects, especially drowsiness and dry mouth. Antihistamines are not effective in relieving asthmatic responses to allergens and may actually worsen symptoms by their drying effect on respiratory secretions.

Antihistamines are often combined with a sympathomimetic agent such as pseudoephedrine to improve their decongestant activity and counteract their sedative effect. Antihistamines and decongestants are discussed further in Chapter 34.

The immediate treatment for anaphylaxis is parenteral adrenaline, an adrenergic agonist (sympathomimetic) drug that has both vasoconstricting and bronchodilating effects. These qualities, combined with its rapid action, make adrenaline ideal for treating an anaphylactic reaction. For mild reactions with wheezing, pruritus, urticaria and angio-oedema, preferably an intramuscular injection of 0.3 to 0.5 mg (adults) of 1:1000 adrenaline is generally sufficient and can be repeated every 5 minutes if required as per the organisational protocol. Intravenous adrenaline using a 1:100 000 concentration may be used in the person with a more severe anaphylactic reaction. Refer to the organisational protocols, guidelines and doctor's orders.

People who have experienced an anaphylactic reaction to insect venom or other potentially unavoidable allergens should wear a Medic-Alert® pendant or bracelet identifying allergy triggers. They should also carry an adrenaline autoinjector kit for immediate treatment of future exposures. The adrenaline autoinjector kit contains a prefilled syringe of adrenaline (epinephrine), allowing prompt self-treatment or, in an emergency, administration by a person who has not been medically trained.

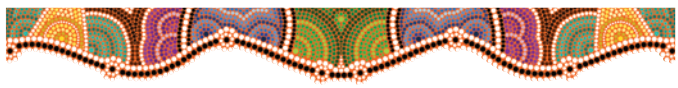
Cromolyn sodium is a drug used to treat allergic rhinitis and asthma. Cromolyn sodium acts by stabilising the mast cell membrane, thus preventing chemical mediator release (Lehne, 2012). It is most effective when applied directly to involved tissue by inhaler or nasal spray. It has few side effects and a wide margin of safety, making it a good choice for people in whom it is effective (Papadakis et al., 2013).

Glucocorticoids (corticosteroids) are used in both systemic and topical forms for many types of hypersensitivity responses. Their anti-inflammatory effects, rather than their immunosuppressive effects, are of most benefit. A short course of corticosteroid therapy is often used for severe asthma, allergic contact dermatitis and some immune-complex disorders: alternate day dosing is preferred in long-term use (Papadakis et al., 2013). Corticosteroids in topical forms or delivered by inhaler may be used for longer periods of time with few side effects; however, systemic absorption can occur.

Other therapies

Other treatments used for hypersensitivity responses are generally dictated by the severity of the response and the organ system affected. Airway management takes highest priority for the person with an acute anaphylactic reaction. Insertion of an endotracheal tube or emergency tracheostomy may be required to maintain airway patency with severe laryngospasm. Because anaphylaxis places the person at risk of vasomotor collapse and significant hypotension, it is necessary to insert an intravenous line and initiate fluid resuscitation with an isotonic solution such as compound sodium lactate (Hartmann's solution).

Plasmapheresis, removal of harmful components in the plasma, may be used to treat immune complex responses such as glomerulonephritis and Goodpasture's syndrome. Plasma and the glomerular-damaging antibody–antigen complexes are removed by passing the person's blood through a blood cell separator. The RBCs are then returned to the person along with an equal amount of albumin or human plasma. This procedure is usually done in a series rather than as a one-time treatment. It is not without risk and informed consent is required. Potential complications of plasmapheresis include those associated with intravenous catheters, shifts in fluid balance and alteration of blood clotting.



Nursing care

Nursing care related to hypersensitivity reactions is primarily directed towards prevention, early identification and providing prompt, effective treatment.

Health promotion

Health promotion activities include helping people to identify possible allergens that prompt a hypersensitivity response and discussing possible strategies to avoid these allergens. Anyone with severe food allergies may need assistance from a dietitian to discuss necessary dietary changes and ways to continue meeting nutrient needs. It is important that people with hypersensitivities inform healthcare personnel of all allergens. People who experience anaphylactic reactions should wear a MedicAlert® bracelet or pendant at all times to identify the substance(s) that provokes this response. Patients who have experienced an anaphylactic reaction to insect venom or other potentially unavoidable allergens may also be required to carry an adrenaline autoinjector (commonly known as an Epipen®) on their person at all times. The need to carry the adrenaline autoinjector is on a prescription basis and is only offered in conjunction with a full anaphylaxis management plan (ASCIA, 2010).

Assessment

Collect the following data through the health history and physical examination. Further focused assessments are described with nursing interventions in the next section.

- *Health history:* risk factors, hypersensitivities (medications, household dust, bee stings, etc.), reaction (rash, hives, difficulty breathing), type of treatment for hypersensitivity reactions; allergy skin testing; asthma, hay fever or dermatitis.
- *Physical assessment:* mucous membranes of nose and mouth, skin for lesions or rashes, eyes (tearing and redness), respiratory rate and adventitious breath sounds.

Nursing diagnoses and interventions

Priority nursing diagnoses will vary according to the type of hypersensitivity reaction experienced by the person. Because nurses are most likely to become involved with a person

experiencing a type I or type II response, this section focuses on diagnoses for these people. Airway, breathing and circulation (the ABCs) are of greatest importance for people with an anaphylactic reaction. When a haemolytic reaction to an incompatible blood transfusion occurs, the person is at risk of injury.

CONSIDERATION FOR PRACTICE

Anaphylaxis is a life-threading emergency. Maintain ABC (airway, breathing, circulation) and, if alone, call for help.

Ineffective airway clearance

In anaphylactic reactions, the airway may be obstructed due to facial angio-oedema, bronchospasm or laryngeal oedema. Establishing and maintaining a patent airway is of highest priority.

- Administer oxygen via nasal prongs at a rate of 2 to 4 L/min or facemask at a rate of > 6 L/min. Apply oxygen and obtain a doctor's order for oxygen administration as per the organisational protocol. *Oxygen administration increases the alveolar oxygen and its availability to cells of the body.*
- Assess respiratory rate and pattern, level of consciousness and anxiety, nasal flaring, use of accessory muscles of respiration, chest wall movement, audible stridor; palpate for respiratory excursion; auscultate lung sounds and any adventitious sounds, such as wheezes. *Extreme anxiety or agitation, nasal flaring, stridor and diminished lung sounds indicate air hunger and possible airway obstruction, necessitating immediate intervention.*
- Position in Fowler's to high Fowler's *to promote optimal lung expansion and ease of breathing*.
- Insert a nasopharyngeal or oropharyngeal airway if required and arrange for immediate intubation as indicated. *Ensuring an adequate airway is vital to preserve life.*
- Administer intramuscular adrenaline 1:1000, 0.3 to 0.5 mL, as prescribed. This may be repeated if necessary as per the prescribed protocol. Administer parenteral antihistamine (deep intramuscular or intravenous) as prescribed. *Adrenaline is a potent vasoconstrictor and bronchodilator, counteracting the effects of histamine. An antihistamine (e.g. promethazine hydrochloride) blocks histamine receptors and their effect. These medications can be effective in rapidly reversing manifestations of anaphylaxis*.
- Provide calm reassurance. Hypoxaemia and air hunger are terrifying for the person. *Anxiety can impair the person's ability to cooperate with treatment and can increase the respiratory rate, making breathing less effective.*

Decreased cardiac output

Peripheral vasodilation and increased capillary permeability from the release of histamine can significantly impair cardiac output. When it falls to the degree that tissue perfusion becomes impaired and hypoxia results, a state of anaphylactic shock exists.

- Monitor vital signs frequently, noting fall in blood pressure, decreasing pulse pressure, tachycardia and tachypnoea. *These vital sign changes may be early indicators of shock.*
- Assess skin colour, temperature, capillary refill, oedema and other indicators of peripheral perfusion. *As cardiac output falls, peripheral vessels constrict and tissue perfusion is impaired.*
- Monitor level of consciousness. *A change in level of consciousness (lethargy, apprehension or agitation) is often the first indicator of decreased cardiac output.*
- Insert one or more large-bore (18-gauge or larger) intravenous catheters. *It is important to insert intravenous catheters as soon as possible to provide sites for rapid fluid replacement.*
- Administer intravenous solutions of normal saline 0.9% or compound sodium lactate, as prescribed. These isotonic solutions help maintain intravascular volume. *Warmed solutions are used to prevent hypothermia from the rapid administration of large amounts of fluid at room temperature (about 21.1°C).*
- Insert an indwelling catheter and monitor urinary output frequently. As the cardiac output drops, the glomerular filtration rate (GFR) falls. *With an output of less than 30 mL/h, the person is at risk of acute renal failure from ischaemia.*

CONSIDERATION FOR PRACTICE

Aggressive fluid therapy may lead to hypervolaemia and acute cardiogenic pulmonary oedema; assess for shortness of breath and crackles in the lungs.

- Once breathing is established safely and maintained, place the person flat with the legs elevated. *This position enhances perfusion of the central organs, such as the brain, heart and kidneys.*

Risk of injury

As noted, the potential for hypersensitivity responses is high in people subjected to medical treatments. Because a blood transfusion is a transplant of living tissue, the risk of adverse immunological response and injury is particularly significant.

- Obtain and record a thorough history of previous blood transfusions and any reactions experienced, *no matter how mild*. Alert the doctor if previous transfusion reactions have occurred. *The person who has received prior blood transfusions is at increased risk of a hypersensitivity reaction, because antibody production may have been stimulated by prior exposure to antigens.*

CONSIDERATION FOR PRACTICE

Begin a blood transfusion within 30 minutes of its delivery from the blood bank to reduce bacterial contamination.

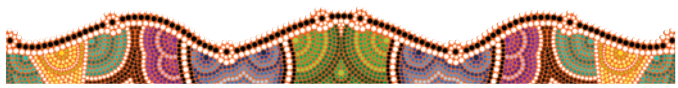
- Check for a signed informed consent as per organisational protocol to administer blood or blood products. *It is important to obtain informed consent for this invasive and risky procedure.*
- Using two registered healthcare professionals, double-check the person's identity, blood type, Rh factor,

crossmatch and expiration date for all blood and blood components received from the blood bank with the person's data. *Check bag for signs of leakage, clots and discolouration. This is an important safety measure to reduce the risk of a haemolytic transfusion reaction due to incompatible blood types.*
- Take and record vital signs within 15 minutes prior to initiating the blood infusion. *This provides a baseline for evaluating any changes related to the blood transfusion.*
- Infuse blood into a site separate from any other intravenous infusion. Use at least a 20-gauge cannula for the infusion to promote flow. This reduces the risk of damage to the blood cells due to incompatibility with other intravenous solutions or physical trauma. *When blood is administered with dextrose solutions (e.g. dextrose 5%, dextrose 5% and normal saline 0.9%), blood cell haemolysis and aggregation occur; administration with Hartmann's can cause agglutination of cells. Administer with normal saline to prime intravenous tubing.*
- Remain with the person for the first 15 minutes of the transfusion (from when the blood enters the vein) and observe for reactions. *Reactions generally occur within the first 15 minutes.*
- During transfusion, monitor observations as per organisational protocol and monitor for complaints of back or chest pain, an increase in the temperature of more than 1˚C, chills, tachycardia, tachypnoea, wheezing, hypotension, hives, rashes or cyanosis. *These signs may indicate an adverse reaction to the blood transfusion.*
- Stop the blood transfusion immediately if a reaction occurs, no matter how mild. Remove the blood bag and the tubing with blood in it. Flush new intravenous tubing with normal saline, keeping the intravenous line open. Notify the doctor and the transfusion service provider.
- If a reaction is suspected, send the blood and administration set to the transfusion service provider with a freshly drawn blood sample and urine specimen from the person. *These will be used to identify the cause of the reaction as well as its effect on the person.*
- If no adverse reaction occurs, administer the transfusion over 2 hours to a maximum of 4 hours as ordered. *This time frame is important to limit the risk of bacterial growth.*

Community-based care

The vast majority of hypersensitivity responses are appropriately treated by the person or family members with little or no medical intervention. Teaching, therefore, is a vital component of care. If the person is at risk of anaphylaxis, involving the family in teaching is essential because the response may occur with such rapidity that the person will be unable to provide self-care.

Include the following points in teaching the person and family about managing hypersensitivities:
- Importance of following the anaphylaxis action plan prepared by a general practitioner.
- When and how to use an anaphylaxis autoinjector pen containing adrenaline and antihistamines in injectable, inhaler and oral forms.
- When to seek medical attention.
- Use of and adverse reactions to prescription and non-prescription antihistamines and decongestants.
- Advantages of autologous blood transfusion if future surgery is scheduled.
- Preventing an immune complex reaction such as glomerulonephritis.
- Skin care to prevent contact dermatitis, including:
  - Expose affected areas to air and sun as much as possible.
  - Avoid direct contact with people who have an infection.
  - Wear cool, light, non-restrictive clothing made of natural fibres, such as cotton, to avoid irritating affected areas.
  - Avoid exposure to extremes of heat or cold.
  - Use bath oils or plain water instead of soaps and detergents.
  - Take baths in cool to lukewarm water rather than showers.
  - To decrease pruritus, maintain a cool environment and avoid exercising.
  - Trim fingernails to reduce the risk of skin damage.
- Helpful resources:
  - Australasian Society of Clinical Immunology and Allergy
  - Allergy and Anaphylaxis Australia
  - Asthma Foundation—Asthma Australia.



THE PERSON WITH AN AUTOIMMUNE DISORDER

Maintaining optimal health and preventing disease depends not only on the immune system's ability to recognise and destroy foreign tissues and other antigens, but also on the immune system's ability to recognise self. When this self-recognition is impaired and immune defences are directed against normal host tissue, the result is an **autoimmune disorder**.

Autoimmune disorders can affect any tissue in the body. Some are tissue or organ specific, affecting particular tissue or a particular organ. Hashimoto's thyroiditis is an example of an organ-specific autoimmune disorder. Circulating antibodies are formed to certain thyroid components, resulting ultimately in destruction of the gland. In other disorders, autoantibodies are formed that are not tissue specific, but tend to accumulate and cause an inflammatory response in certain tissue—for example, the renal glomerulus or the hepatic small bile ductules.

Additional factors are believed to contribute to the development of autoimmune disorders including age, gender and environmental elements—for example, infectious agents (Diamond & Lipsky, 2012). Autoimmune disorders may also be systemic, with neither antibodies nor the resulting inflammatory lesions confined to any one organ. Rheumatological disorders, such as rheumatoid arthritis and systemic lupus erythematosus (SLE), are characteristic of systemic autoimmune disorders (Goldsby et al., 2003). A list of selected autoimmune disorders is included in Table 12.1.

Pathophysiology

The mechanism that causes the immune system to recognise host tissue as a foreign antigen is not clear. The following factors are under study as possible contributors to the development of autoimmune disorders:

- the release of previously 'hidden' antigens into the circulation, such as DNA or other components of the cell nucleus, which elicit an immune response
- chemical, physical or biological changes in host tissue that cause self-antigens to stimulate the production of autoantibodies
- the introduction of an antigen, such as a bacteria or virus, whose antigenic properties closely resemble those of host tissue, resulting in the production of antibodies that target not only the foreign antigen but also normal tissue. Heart damage in rheumatic fever and encephalitis following rabies vaccination are examples of the development of antibodies against normal tissue (Porth & Matfin, 2009)
- a defect in normal cellular immune function that allows B cells to produce autoantibodies unchecked
- initiation of the autoimmune response by very-slow-growing mycobacteria.

Although the exact mechanism producing autoimmunity is unclear, several characteristics of autoimmune diseases are known. It is apparent that genetics plays a role because a higher incidence is seen in family members of people with autoimmune disorders. Autoimmune disorders are far more prevalent in females than in males. The disorders tend to overlap, so that the person with one autoimmune disorder may develop another or some manifestations of another. The onset of an autoimmune disorder is frequently associated with an abnormal stressor, either physical or psychological. Autoimmune disorders are frequently progressive relapsing–remissing disorders characterised by periods of exacerbation and remission.

Specific autoimmune disorders are discussed in the sections of this textbook related to the affected organ systems or functional disruption.

INTERPROFESSIONAL CARE

For the most part, the diagnosis of an autoimmune disorder is based on the person's clinical manifestations. Serum assays are useful to identify autoantibodies. Other diagnostic tests are generally specific to the suspected disorder and to identifying the degree of tissue damage and destruction. Although the manifestations of these disorders can often be managed, a cure typically is not possible unless the affected target tissue is removed (e.g. colectomy for the person with ulcerative colitis).

Diagnosis

Serological assays are used to identify and measure antibodies directed towards host tissue antigens or normal cellular components. Many detectable autoantibodies are not specific to a single autoimmune disorder and are used to establish the autoimmune process rather than the specific disorder. Although healthy people often have low levels of autoantibodies, levels are much higher in people affected by an autoimmune disorder. The following serological assays may be ordered:

- *Antinuclear antibody (ANA)* detects antibodies produced to DNA and other nuclear material. These antibodies can cause tissue damage characteristic of autoimmune disorders, such as SLE. The person's serum is combined with nuclear material and tagged antihuman antibody to detect ANA–antihuman antibody complexes. Titres are not necessarily comparable between laboratories; refer to the laboratory reference levels. This test is not specific for SLE, because high levels of ANA may be present in rheumatoid arthritis (RA) and cirrhosis of the liver; nevertheless, 95% of people with SLE have a positive ANA titre.
- *Lupus erythematosus (LE) cell test* is also used to detect SLE and monitor its treatment. Neutrophils that contain large masses of phagocytised DNA from the nuclei of PMNs are called LE cells. Like the ANA, the LE cell prep is non-specific for SLE. A positive result may also be seen in RA or with medications such as isoniazid, penicillin, phenytoin, procainamide, streptomycin, tetracycline, oral contraceptives or sulfonamide drugs.
- *Rheumatoid factor (RF)* is an immunoglobulin present in the serum of approximately 80% of people with rheumatoid arthritis. Low titre levels may be present in the elderly. An RF titre 1:80 or higher indicates RA. A titre between 1:20 and 1:80 could indicate SLE, scleroderma or liver cirrhosis (Pagana & Pagana, 2013). Reference factors are variable; refer to the laboratory reference range.
- *Complement assay* may also be useful in identifying autoimmune disorders. In these disorders, complement may be consumed in the development of antigen–antibody complexes. Decreased levels are seen on examination. Both total complement level and amounts of individual components of the complement cascade can be determined.
- *CCP (also known as anti-CCP antibody test)* is a blood test for RA. It measures cyclic citrullinated peptide antibody in the blood. This is a specific test to identify RA from other forms of arthritis.

Medications

Various approaches are used in the treatment of autoimmune disorders. Anti-inflammatory medications such as aspirin, non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids may be prescribed to reduce the inflammatory response and minimise tissue damage. (Refer to Chapter 8 for additional detail on these drugs.) When these agents are not effective or well tolerated by the person, slow-acting anti-inflammatory medications may be prescribed. Slow-acting or antirheumatic drugs include such medications as gold salts, hydroxychloroquine and penicillamine. Their use is further detailed in Chapter 39. Cytotoxic drugs may be used in combination with plasmapheresis in treating many autoimmune disorders. Cytotoxic drugs are discussed in further detail in the next

section of this chapter. Disease-modifying antirheumatic drugs (DMARDs) reduce signs and symptoms, reduce or prevent joint damage, and preserve the structure and function of the joints in people with RA. These drugs may reduce health costs for people with RA and allow them to remain active and productive. The most common DMARDs in current use are methotrexate, sulfasalazine, hydroxychloroquine, leflunomide and cyclosporin.

Another class of antirheumatic drugs, referred to as *biologicals* or *biological response modifiers*, consists of laboratory-produced proteins that decrease the inflammatory process. These antibodies bind tumour necrosis factor alpha (TNF-α) and interleukin-1, both inflammatory elements. These medications include infliximab or adalimumab, etanercept and anakinra.

Nursing care

Nursing interventions for the person with an autoimmune disorder are individualised and tailored to needs dictated by manifestations of the disorder. Nurses will often be involved with the person in an outpatient setting such as an office or home, evaluating the person's response to therapy and self-care management.

Consider the following nursing diagnoses in planning care for the person with an autoimmune disorder:

- *Risk of activity intolerance* related to inflammatory effects of autoimmune disorder.
- *Risk of ineffective coping* related to chronic disease process.
- *Risk of interrupted family processes* related to lack of understanding about autoimmune disorder and its effects.
- *Risk of ineffective protection* related to disordered immune function.
- *Risk of ineffective therapeutic regimen management* related to lack of understanding.

Community-based care

Because many autoimmune disorders are chronic, educating the person and family about the disorder and its management is a key nursing care component. The person may be taking drugs with multiple side effects or long-term effects, necessitating effective education. People with autoimmune disorders often do not appear to be ill, making it difficult for friends and families to understand their care needs. The chronicity of these disorders also puts the person at high risk of unproven remedies and quackery. Provide psychological support, listening and teaching. In addition, suggest resources such as local support groups.

THE PERSON WITH A TISSUE TRANSPLANT

Since the first kidney transplant was performed from one identical twin to the other in 1954, organ and tissue transplantation have become increasingly popular and viable treatment options. The transplantation of avascular tissues, such as skin, cornea, bone and heart valves, is considered routine, with little need for tissue matching and **immunosuppression**. Transplants of organs (e.g. the kidney, heart, heart and lung, liver and bone marrow) are increasingly common and are no longer considered experimental or extraordinary procedures. In 2014, 378 organ donors gave 1117 Australians a new chance at life, the highest number since the commencement of Australian record keeping (Australian Government Organ and Tissue Authority, 2015). Common organ transplants are outlined in Table 12.2.

Transplant success is closely tied to obtaining an organ with tissue antigens as close to those of the recipient as possible. As noted earlier in this chapter, every body cell has cell

TABLE 12.2 Organ transplants

ORGAN	GRAFT TYPE	INDICATIONS FOR TRANSPLANT	SUCCESS RATE
Kidney	Allograft; may be isograft	End-stage renal disease	81.1% at 5 years
Heart	Allograft	End-stage cardiac disease refractory to medical management	74.4% at 5 years
Lung	Allograft	Pulmonary hypertension, cystic fibrosis, pulmonary fibrosis, chronic obstructive pulmonary disease	52.6% at 5 years
Liver	Allograft	Severe liver dysfunction due to chronic active hepatitis, primary biliary cirrhosis, sclerosing cholangitis	73.6% 5-year survival
Bone marrow	Autograft or allograft	Leukaemia, aplastic anaemia, congenital immunological defects	30–70% cure
Skin	Autograft, allograft or xenograft	Severe burns, plastic surgery	> 95% at 5 years
Cornea	Allograft	Corneal ulceration and opacification	> 95% at 5 years
Pancreas	Allograft	Pancreatic insufficiency, diabetes	88.1%
Islet cells	Allograft (multiple donor)	Type 1 diabetes mellitus	100% > 2 years

Source: OPTN/Scientific registry of transplant recipients, *2010 Annual Report: Transplant Data Report*. Retrieved 8 January 2013 from www.srtr.org/local_stats.aspx.

surface antigens known as human leucocyte antigens that are unique to the individual. Although identical twins may have the same HLA type, the chance is reduced to 1 in 4 for siblings and less than 1 in several thousand for unrelated individuals (Papadakis et al., 2013). Matching the HLA type of the donor and recipient as closely as possible decreases the potential for rejection of the transplanted organ or tissue but does not eliminate it.

Pathophysiology

An **autograft**, a transplant of the person's own tissue, is the most successful type of tissue transplant. Skin grafts are the most common example of autografts. Increasingly, autologous bone marrow transplants and blood transfusions are being used to reduce immunological responses. When the donor and recipient are identical twins, the term **isograft** is used. Because of the high likelihood of an HLA match, the success of these grafts is good and rejection episodes are mild.

Few people, however, have an identical twin to provide tissue for donation and when the need is for an organ such as the heart, liver or lungs, a living-donor transplantation is not possible. Most often organ and tissue transplants are **allografts**, which are grafts between members of the same species but who have different genotypes and HLA. Allografts may come from living donors; examples are bone marrow, blood and a kidney. Most often, however, organs for transplantation are obtained from a cadaver. Donors are typically people who meet the criteria for brain death, are less than 65 years old, and are free of systemic disease, malignancy or infection, including HIV, hepatitis B or hepatitis C. The organ is removed immediately before or after cardiac arrest and preserved until it is transplanted into the waiting recipient. Finally, **xenograft** is a transplant from an animal species to a human. These transplants are the least successful but may be used in selected instances, such as the use of pigskin as a temporary covering for a massive burn.

Tissue typing is used to determine **histocompatibility**, the ability of cells and tissues to survive transplantation without immunological interference by the recipient. Tissue typing is performed in an attempt to match the donor and recipient as closely as possible for HLA type and blood type (ABO, Rh) and to identify preformed antibodies to the donor's HLA.

Both antibody-mediated and cell-mediated immune responses are involved in the complex process of transplant rejection. Host macrophages process donor antigen, presenting it to T and B lymphocytes. Activated lymphocytes (B and T cells) produce both antibody- and cell-mediated effects. Killer T cells bind with cells of the transplanted organ, resulting in cell lysis. Helper T cells stimulate the multiplication and differentiation of B cells, and antibodies are produced to graft endothelium. Complement activation or antibody-dependent cell-mediated cytotoxicity leads to transplant cell destruction. Rejection typically begins after the first 24 hours of the transplant, although it may present immediately. Rejection episodes are characterised as hyperacute, acute or chronic, as summarised in Table 12.3.

Hyperacute tissue rejection occurs immediately to 2 to 3 days after the transplant of new tissue. Hyperacute rejection is due to preformed antibodies and sensitised T cells to antigens in the donor organ. Hyperacute rejection is most likely to occur in people who have had a previous organ or tissue transplant, such as a blood transfusion. Hyperacute rejection may be evident even before the transplant procedure is completed. The grafted organ initially appears pink and healthy, but soon becomes soft and cyanotic, as blood flow is impaired. Organ function deteriorates rapidly and symptoms of organ failure develop.

Acute tissue rejection is the most common and treatable type of rejection episode. It occurs between 4 days and 3 months after the transplant. Acute rejection is mediated primarily by the cellular immune response, resulting in transplant cell destruction. The person experiencing acute rejection demonstrates manifestations of the inflammatory process, with fever, redness, swelling and tenderness over the graft site. Signs of impaired function of the transplanted organ may be noted (e.g. elevated blood urea nitrogen (BUN) and creatinine, liver enzyme and bilirubin elevations, or elevated cardiac enzymes and signs of cardiac failure).

TABLE 12.3 Transplant rejection episodes

TYPE	CAUSE	PRESENTATION	TREATMENT
Hyperacute	Pre-existing antibodies to donor ABO or HLA antigens	Occurs within minutes to hours or days of the transplant Rapid deterioration of organ function	The transplant usually cannot be saved; prevent with crossmatch and use antimetabolites or anti-inflammatory drugs before surgery
Acute	Primarily a cell-mediated immune response to HLA antigens; antibody-mediated response may also contribute	Occurs within days to months after the transplant Signs of inflammation and impaired organ function	Increase immunosuppression using steroids, cyclosporin, monoclonal antibodies or antilymphocyte globulins
Chronic	Probably antibody-mediated response; may also involve inflammatory damage to vessel endothelium	Occurs 4 months to years after the transplant Gradual deterioration of organ function	None; loss of graft will occur, requiring retransplant

Chronic tissue rejection occurs from 4 months to years after transplant of new tissue. Chronic rejection is most likely the result of antibody-mediated immune responses. Antibodies and complement are deposited in transplant vessel walls, causing narrowing and decreased function of the organ due to ischaemia. The gradual deterioration of transplanted organ function is seen with chronic tissue rejection.

Graft-versus-host disease (GVHD) is a frequent and potentially fatal complication of bone marrow transplant. When there is no close match between donor and recipient HLA, immunocompetent cells in the grafted tissue recognise host tissue as foreign and mount a cell-mediated immune response. If the host is immunocompromised, as is often the case when a bone marrow transplant is performed, host cells are unable to destroy the graft and instead become the targets of destruction. Of people with very closely matched bone marrow, 30% to 60% nevertheless develop GVHD. Acute GVHD occurs within the first 100 days following a transplant and primarily affects the skin, liver and gastrointestinal tract. The person develops a maculopapular pruritic rash beginning on the palms of the hands and soles of the feet. The rash may spread to involve the entire body and lead to desquamation. Gastrointestinal manifestations include abdominal pain, nausea and bloody diarrhoea. GVHD that lasts longer than 100 days is said to be chronic. If it is limited to the skin and liver, the prognosis is good. If multiple organs are involved, the prognosis is poor (Porth & Matfin, 2009).

INTERPROFESSIONAL CARE

Pre-transplant and post-transplant care are directed towards reducing the risk that transplanted tissue will be rejected or result in GVHD. Diagnostic studies are directed first at identifying a suitable donor, then at monitoring the immune response to the transplant. Immunosuppressive therapy with medications is a vital part of post-transplant care. Indeed, the development of effective immunosuppressive drugs is responsible for the success of organ transplants using allografts.

Diagnosis

The following diagnostic tests may be ordered prior to organ or tissue transplantation:

- *Blood type and Rh factor* of both the donor and recipient are determined. Although there is some question about the benefit of histocompatibility testing prior to transplant of a cadaver organ, there is no question about the need for ABO blood group compatibility.
- *Crossmatching* of the person's serum against the donor's lymphocytes is performed to identify any preformed antibodies against antigens on donor tissues. If present, these antibodies would likely result in an immediate or hyperacute graft rejection with probable loss of the transplant.
- *DNA sequencing* is made on blood cells to determine histocompatibility. Sequencing can be completed quickly. Quick response is important to minimise cold ischaemia in cadaverous organs. Diagnostic testing for recipients can be done less urgently.
- *HLA histocompatibility testing* identifies donors with an HLA type close to that of the recipient. It is used primarily to identify living donors for bone marrow and kidney transplant. Because of GVHD, histocompatibility tests to identify an identical or very close HLA match are particularly important in bone marrow transplant. HLA tests are performed using lymphocytes from a blood sample. The sample should not be obtained within 72 hours of a blood transfusion, because this will interfere with results.
- *Mixed lymphocyte culture (MLC) assay tests* also are used to determine histocompatibility between the donor and the recipient. This test identifies whether mononuclear cells of the recipient will react against the potential donor's leucocyte antigens. The disadvantage of this test is that results cannot be obtained until 7 to 10 days later (Chernecky & Berger, 2004). If the intended recipient is severely immunocompromised, the results may be falsely negative. People treated with chemotherapy within 2 weeks of specimen collection are potentially immunocompromised.
- *Ultrasonography* or magnetic resonance imaging (MRI) of the transplanted organ may be performed to evaluate its size, perfusion and function.
- *Tissue biopsies* of the transplanted organ are performed routinely to assess for evidence of tissue rejection.

Medications

Prior to transplantation, several antibiotic and antiviral drugs may be prescribed, including the following:

- trimethoprim-sulfamethoxazole, which decreases the incidence of gram-negative bacterial infections
- aciclovir, which prevents the development of the herpes simplex virus and pneumonia in bone marrow transplant recipients
- ganciclovir, which prevents the development of cytomegalovirus (CMV) pneumonia in bone marrow transplant recipients.

The mainstays of drug therapy for people following a tissue or organ transplant are immunosuppressive agents. Varying regimens of these drugs are used, depending on the transplanted tissue and the medical centre; however, a combination of corticosteroids and cyclosporin is common for maintenance therapy (see Figure 12.6). Antilymphocyte therapy and the use of monoclonal antibodies are increasingly common in the immediate post-transplant period and for treating steroid-resistant rejection episodes.

Corticosteroids, primarily prednisone and methylprednisolone, were among the first medications used to prevent transplant rejection and they remain important agents today. Although the exact anti-inflammatory and immunosuppressive activity of corticosteroids is unknown, they are known to suppress production of interleukin-1 and -2, decrease monocyte migration and suppress proliferative and cytotoxic T-cell activity. Although they are very effective, large doses of corticosteroids used post-transplant are associated with significant adverse effects. Wound healing is impaired and the metabolism of fats, proteins and carbohydrates is altered. Blood glucose increases with steroid use, impairing glucose control. Fat distribution changes, producing a cushingoid appearance with

moon face, increased truncal fat and 'buffalo hump'. Fluid retention and hypertension are potential problems, as are osteoporosis, gastrointestinal bleeding and emotional disturbances.

Azathioprine has been in use as an immunosuppressant for more than 25 years and continues to be a component of many regimens. Azathioprine inhibits both cell-mediated and antibody-mediated immunity, although its activity is more specific for T cells than B cells. Because it is rapidly metabolised by the liver, azathioprine can be given to people with impaired renal function but may not be effective in people with impaired hepatic function. Bone marrow suppression is the most common adverse effect of this drug, necessitating frequent evaluation of the full blood count (FBC). Hepatotoxicity, pancreatitis and increased risk of neoplasm are also associated with azathioprine administration. Nursing responsibilities related to azathioprine are listed in the following 'Medication administration' box. People who cannot tolerate azathioprine may receive a newer immunosuppressant, mycophenolate mofetil. Primarily, it is prescribed following renal and cardiac transplants.

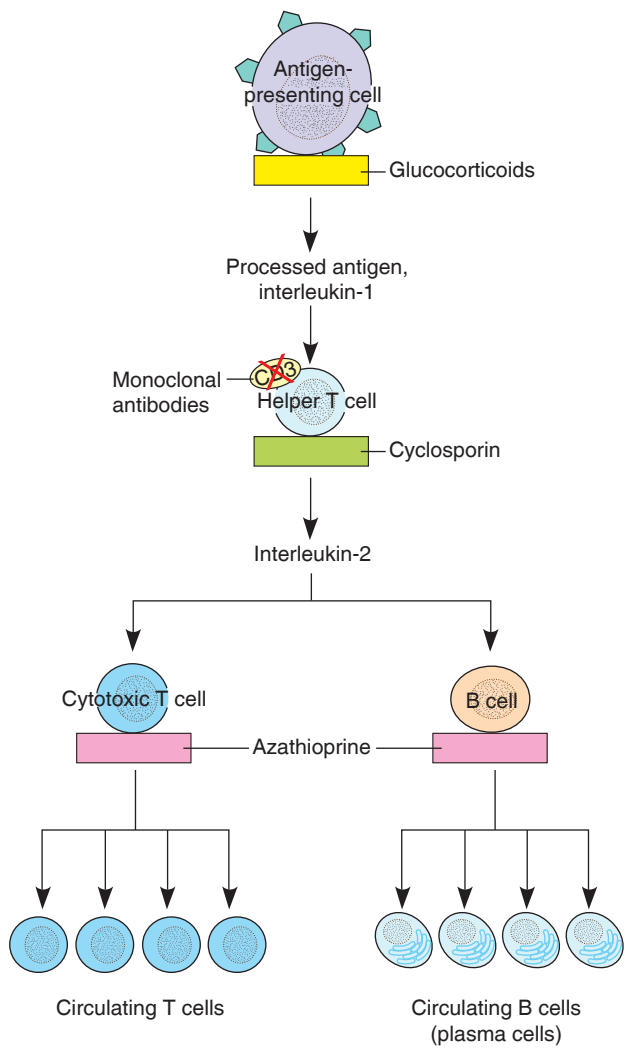


FIGURE 12.6 ■ Sites of action of immunosuppressive agents

Cyclosporin inhibits T-cell function and the normal cell-mediated immune response. The incidence of cyclosporin toxicity and side effects is related to blood levels, so blood levels are monitored closely. Cyclosporin is both nephrotoxic and hepatotoxic, especially at high doses. Observable toxic effects include hypertension and CNS symptoms such as flushing or tingling of the extremities, confusion, visual disturbances and seizures or coma.

Muromonab-CD3, also known as OKT3, is the first monoclonal antibody produced for therapeutic use in humans. As a monoclonal antibody, OKT3 is specific to T cells, blocking their generation and function. It binds with a surface antigen on T cells, inactivating and removing them from circulation. It also blocks killer T cells attached to the graft. Because of significant side effects, the use of OKT3 is limited primarily to treatment of steroid-resistant rejection. Two newer monoclonal antibodies, basiliximab (Simulect) and daclizumab (Zenapax), are a combination of mouse and human antibodies and cause fewer side effects.

Polyclonal antilymphocyte antibodies are also used as adjunctive immunosuppressant therapy. These are administered as antilymphocyte globulin (ALG) or antithymocyte globulin (ATG). These globulins contain antibodies against both T and B cells, as well as other mononuclear leucocytes. When administered, they deplete circulating lymphocytes, platelets and granulocytes.

Nursing care

The person who has an organ or tissue transplant has both immediate and long-term nursing care needs. Both the person and the family must be considered in providing nursing care.

Health promotion

Health promotion activities focus partly on preventing the need for a tissue transplant. It is important to increase public awareness regarding unhealthy lifestyle behaviours, such as excessive alcohol consumption and illegal drug use, and their relationship to organ failure. People with chronic diseases such as diabetes mellitus and hypertension must understand that inadequate management of these disorders could lead to end-stage renal disease. Other risk factors may simply relate to a person's heredity; it is important to understand how heredity could affect future health and might influence the person's lifestyle choices.

Assessment

Assessment data collected following a tissue transplant focus on identifying potential rejection episodes. Further focused assessments are described with nursing interventions in the next section.

Nursing diagnoses and interventions

Because of the continuing risk of transplant rejection and the need for immunosuppression, *Ineffective protection* and *Risk of impaired tissue integrity* are priority nursing care foci. The person's underlying disease process, the transplant and the

continuing need for immunosuppressive drug therapy also have emotional and psychological consequences. Many nursing diagnoses, such as *Powerlessness* or *Ineffective coping*, may be appropriate. The diagnosis *Anxiety* related to potential transplant rejection is considered in this section.

Ineffective protection

Ineffective protection is a problem for the transplant person at all stages. Before the transplant occurs, failure of the affected organ may put the person at risk of infection and other multisystemic problems. Incisions and invasive perioperative procedures impair skin and mucous membrane protection from infectious organisms and other antigens. Immunosuppressive drugs given postoperatively to prevent graft rejection disarm the immune response to a certain extent, increasing the risk of infections and neoplastic growths.

CONSIDERATION FOR PRACTICE

Use strict aseptic technique in changing dressings and caring for invasive catheters such as intravenous lines and indwelling urinary catheters to protect against external and resident host microorganisms.

- Wash hands on entering room and before providing direct care. *Handwashing removes transient organisms from the skin, reducing the risk of transmission to the person.*
- Assess frequently for signs and symptoms of infection. Monitor the temperature and vital signs every 4 hours. Assess for evidence of inflammation, abnormal wound drainage, changes in urine or other body secretions, complaints of pain or behaviour changes that may indicate infection. Culture abnormal wound drainage. The person on immunosuppressive therapy is more susceptible to infection, and the usual signs and symptoms may not be evident. Both the temperature and inflammatory response can be suppressed by therapy. *Prompt identification and intervention for infection is important in the immunosuppressed person.*
- Monitor laboratory values, including FBC and tests of organ function; report changes to the doctor. *An elevation in the WBC count with increased numbers of immature cells (bands) or a decline in function of the transplanted organ (e.g. a rising BUN and creatinine in the renal transplant person) may be early indications of infection or transplant failure.*
- Initiate reverse or protective isolation procedures as indicated by the person's immune status. *These procedures further protect the severely immunocompromised person from infection.*
- Instruct ill family members and visitors to avoid contact with the person. *A 'minor' upper respiratory infection can be a significant illness in the immunocompromised host.*
- Help ensure adequate nutrient intake, offering supplementary feedings as indicated or maintaining parenteral nutrition if necessary. *Adequate nutrition is important for healing and immune system function.*
- Change intravenous bags and tubing as per organisational protocol, generally every 24 hours, and change peripheral intravenous sites every 48 to 72 hours, unless contraindicated. Remove invasive catheters and lines as soon as they are no longer necessary. *Changing lines and sites is important to reduce bacterial contamination. Fewer invasive lines provide fewer sites for bacterial invasion of the body.*
- Emphasise the importance of washing hands thoroughly after using the bathroom and before eating. *This reduces the risk of infection with endogenous organisms.*
- Provide good mouth care. *Good mouth care reduces the population of oral microorganisms and helps maintain an intact mucous membrane lining.*
- Monitor for potential adverse effects of medications:
  - thrombocytopenia and possible bleeding
  - fluid retention with oedema and possible hypertension
  - loss of bone density, osteoporosis and possible pathological fractures
  - renal or hepatic toxicity
  - cardiac effects, particularly in the presence of fluid retention and hypervolaemia.

  Medications used to maintain immunosuppression and preserve the allograft have many potential adverse effects that can alter normal protective and homeostatic mechanisms.

Risk of impaired tissue integrity: allograft

As noted, the risk of transplant rejection is highest in the initial postoperative period, but it is never completely eliminated for the person who has had an allograft. The person who has had a bone marrow transplant has the additional risk of developing GVHD, which can affect the integrity of skin, mucous membranes and other organs.

- Administer immunosuppressive therapy as prescribed. *Suppression of the immune response is necessary to reduce the risk of graft destruction by normal immune responses and to preserve the graft's function.*
- Assess for evidence of graft rejection, including tenderness, erythema and swelling over the site; sudden weight gain, oedema and hypertension; chills and fever; malaise and an increased WBC count and sedimentation rate. Report any changes immediately. *Early identification of rejection allows adjustment of medication regimens and, possibly, preservation of the graft.*
- Monitor results of laboratory studies for function of the transplanted organ. *With a functional graft, results (e.g. renal or liver function studies) will improve; a functional decline may be an early indicator of rejection.*
- Assess for and report signs of GVHD immediately, including maculopapular rash, erythema of the skin and possible desquamation, hair loss, abdominal cramping and diarrhoea, or jaundice with elevated bilirubin and liver enzymes (AST, ALT). *GVHD is a potentially lethal complication in the immunosuppressed person and necessitates immediate intervention.*
- Stress the importance of maintaining immunosuppressive therapy and reporting signs of graft rejection promptly to the doctor. *Continued immunosuppression and prompt treatment of rejection are vital to preserving graft function.*

MEDICATION ADMINISTRATION Immunosuppressive agents

T-CELL SUPPRESSORS

Cyclosporin
Tacrolimus
Sirolimus

These drugs inhibit T-cell development and activation. They are given concurrently with a glucocorticoid and in combination with other immunosuppressants and inhibit immune system activity and organ rejection.

Nursing responsibilities

- Monitor BUN and creatinine for evidence of nephrotoxicity.
- Teach the signs and symptoms of infection unique to immunosuppressed individuals. A temperature of 38.1°C is significant evidence of infection. A sore throat may be a manifestation. Other signs and symptoms of inflammation and infection may be absent.
- Teach people good hygiene to avoid infection, with special emphasis on handwashing and avoiding infected individuals.
- Monitor blood pressure and availability and use of antihypertensive medications.
- Teach to avoid grapefruit juice, which can raise cyclosporin levels by 50% to 200% and increase the risk of toxicity. Sirolimus should not be taken with grapefruit juice. Sirolimus increases cholesterol and triglycerides. Lipid-lowering drugs may be necessary to prevent hyperlipidaemias.

CYTOTOXIC AGENTS

Azathioprine
Cyclophosphamide
Methotrexate
Mycophenolate

Certain drugs that are identified as cytotoxic or antineoplastic agents are effective as immunosuppressive agents. They act by decreasing the proliferation of cells within the immune system and are widely used to prevent rejection following a tissue or organ transplant. They are usually administered concurrently with corticosteroid therapy, allowing lower doses of both preparations and resulting in fewer side effects.

Nursing responsibilities

- Monitor blood count, with particular attention to the WBC and platelet counts. Notify the doctor of decreases in WBCs or platelet counts.
- Monitor renal and liver function studies, including creatinine, BUN, creatinine clearance and liver enzymes. Report abnormal levels to the doctor.
- Administer the drug as ordered. Administer oral preparations with food to minimise gastrointestinal effects. Antacids may be ordered.
- Have the person increase fluids to maintain good hydration and urinary output, void frequently and avoid taking the drug in the evening, which promotes dwelling of the drug in the bladder overnight.
- Monitor intake and output.
- Monitor for signs of abnormal bleeding, bleeding gums, bruising, petechiae, joint pain, haematuria and black or tarry stools.
- Use meticulous handwashing and other appropriate measures to protect the person from infection. Assess for signs of infection.
- Pulmonary fibrosis is a rare (< 1%) potential adverse effect of cyclophosphamides. Therefore, monitor respiratory function using pulmonary function studies, and monitor for clinical signs of dyspnoea or cough.

Health education for the person and family

- Avoid large crowds and situations where exposure to infection is probable.
- Report signs of infection—such as chills, fever, sore throat, fatigue or malaise—to the doctor.
- Use contraceptive measures to prevent pregnancy while on immunosuppressive therapy; these drugs are teratogenic.
- Avoid the use of aspirin or ibuprofen while taking these drugs. Report any signs of bleeding to the doctor. Many over-the-counter products contain aspirin; check labels for aspirin.
- With cyclophosphamide, amenorrhoea may occur. The menses will resume after the drug is discontinued.
- If taking cyclophosphamide, report any difficulty breathing or cough to the doctor.

MONOCLONAL ANTIBODY

Muromonab-CD3 or OKT3

This monoclonal antibody against T cells is formed by immunising a mouse with an antigen to produce a specific antibody. Lymphocytes producing the antibody, OKT3, are cloned and the antibody is harvested. When injected into humans, OKT3 binds with a surface antigen on T cells, removing them from circulation and inactivating those bound to allograft cells. Due to the high incidence of adverse effects, refer to the organisational protocol regarding the administration of initial and subsequent doses of OKT3 and to observation protocols following the administration of OKT3.

Nursing responsibilities

- Be sure a chest x-ray has been performed within 24 hours preceding initiation of OKT3 therapy and that no congestion is present. The risk of anaphylaxis is greater in the person with fluid overload.
- Premedicate as ordered with hydrocortisone, paracetamol and diphenhydramine to reduce potential adverse effects.
- Position the emergency trolley and/or emergency medications in the person's room or in proximity to it. Refer to the organisational protocol.
- Monitor vital signs following administration of OKT3 as per organisational protocols.
- Refer to the organisational protocols for administration of initial and subsequent doses of OKT3 and administer as per guidelines.

MEDICATION ADMINISTRATION Immunosuppressive agents (continued)

- Observe closely for potential adverse effects, including chills and fever; tachycardia; headache and tremor; hypertension or hypotension; nausea, vomiting and diarrhoea; chest pain, dyspnoea and wheezing.
- OKT3 can also cause anaphylaxis; observe for evidence of urticaria, angio-oedema, laryngeal oedema, wheezing or other signs of anaphylactic reaction.
- Monitor FBC for evidence of leucopenia or pancytopenia.
- Assess for infection. Remember that typical signs of infection, including symptoms such as fever and inflammation, may be masked or reduced by immunosuppressive therapy.

Health education for the person and family

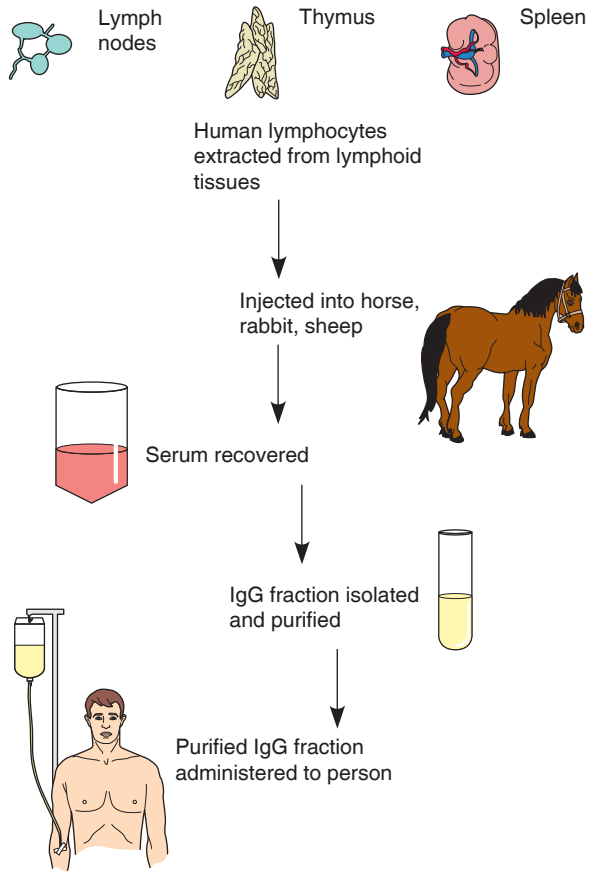
- Teach about the drug and its purpose.
- Discuss potential adverse and side effects and emphasise the need to report symptoms promptly.
- Inform the person that adverse effects are most likely to occur following the first two doses, necessitating close observation at that time. Reassure the person that this is standard protocol for this medication.

ANTILYMPHOCYTE GLOBULINS

Antithymocyte globulin or ATG (ATGAM)
Antilymphocyte globulin or ALG

These globulins containing antilymphocyte antibodies are produced by immunising horses (the main source), rabbits or sheep with human lymphocytes to stimulate production of antibodies (see figure). Serum from the animal is then recovered and the active IgG fraction is isolated, purified and administered parenterally to the person. It binds with peripheral lymphocytes and mononuclear cells, removing them from circulation.

ATG or ALG is used both to induce immunosuppression immediately following a transplant and to treat steroid-resistant rejection episodes. As with monoclonal antibody, multiple side effects are associated with ATG or ALG.



A horse is inoculated with washed human lymphocytes, stimulating the production of immunoglobulin with polyclonal antilymphocyte antibodies. These are then extracted from horse serum, purified, and administered intravenously to the person

Nursing responsibilities

- Ensure a skin test for sensitivity to horse serum has been carried out prior to initial dose. Report any positive reaction to the doctor and hold administration until desensitisation therapy has been completed.
- Premedicate as ordered with paracetamol and diphenhydramine prior to each dose. Steroids may also be administered before the initial dose. Have adrenaline and hydrocortisone injections available at the bedside in case of anaphylactic reaction.
- Administer by intravenous infusion into a central line over 4 to 6 hours as per organisational protocol.
- Monitor vital signs during infusion as per organisational protocol.
- Assess for adverse effects, including chills and fever, erythema and pruritus. Notify the doctor; these may be treated symptomatically.
- Monitor FBC daily; notify the doctor if WBC or platelet count decrease. The medication may be stopped or reduced.
- Assess renal function studies to monitor for serum sickness. Report complaints of joint pain.
- Monitor for signs of infection and report any signs promptly.

Health education for the person and family

- Explain the need for special precautions and close monitoring while this drug is being administered.
- Instruct the person to report any adverse effects, including malaise or joint pain, promptly.
- Ask the person to report any evidence of easy bruising, bleeding gums or black stools.
- Teach family members about the importance of not exposing the person to other people with infectious diseases.

Anxiety

The person who undergoes organ or tissue transplantation often faces the unwelcome choices of death from organ failure or receiving an organ that their body will likely attempt to reject. In most cases, the person understands that to receive this transplant, someone else must die and be willing to give up an organ. When the transplant (bone marrow or kidney) comes from a living donor, the person may not only worry about themselves but also about the condition of the donor. Fear of rejection and guilt may be even greater in this instance.

- Assess level of anxiety by noting such cues as expressions of apprehension, fear or inadequacy; facial expression, tension or shakiness; difficulty focusing; helplessness; poor eye contact and restlessness. *People may have difficulty identifying or verbalising feelings of fear and anxiety. Non-verbal cues are often useful in recognising states of anxiety.*
- Provide opportunities to express feelings. Use opening statements such as, 'Facing an organ transplant must be very stressful.' Listen attentively. *Encouragement and active listening allow the person to express feelings of anxiety or fear.*
- Arrange tasks to allow as much time with the person as possible. When leaving, tell the person when you will return. *Time spent with the person facilitates the development of trust.*
- Provide clear, concise directions. *Highly anxious people have difficulty focusing and retaining information.*
- Encourage involvement in care, but do not request unnecessary decisions. *The person needs to feel a sense of control but may become irritated if asked to make decisions unrelated to the situation.*
- Encourage family members to remain with the person as much as possible. *This can help reduce the person's anxiety.*
- Encourage the use of coping behaviours that have been effective for the person in the past. *Coping mechanisms and behaviours help lower anxiety to a more acceptable level.*
- Reduce or eliminate environmental stressors to the extent possible. *This gives the person a better sense of control.*
- Assist with stress reduction and relaxation techniques, such as guided imagery, meditation and muscle relaxation. *These techniques help the person gain control over physical responses to anxiety.*
- Refer to a counsellor or mental health specialist to work with the person. *Counselling can help the person identify and deal with their feelings.*
- Assess the person's preference and desire for spiritual counselling prior to transplant. *Because there is a risk of death if the transplant fails, the person may want to discuss their concerns with a spiritual counsellor.*

Community-based care

Teaching of the person and family regarding an organ or tissue transplant begins well before the transplant and continues throughout hospitalisation and follow-up treatment.

Initial teaching focuses on the options, risks and potential benefits of the transplant itself. Include the procedure by which the organ is selected and obtained, as well as the procedure by which it is transplanted into the person. If a living related donor is an option, discuss the risks and benefits for both the person and the donor. Outline the post-transplant treatment regimen, including any lifestyle changes that may be necessary.

Following the transplant, provide verbal and written instructions, including the following:

- manifestations of transplant rejection and the importance of notifying the doctor
- immunosuppressive drug regimen and side effects
- wound care
- avoiding exposure to infectious diseases, particularly respiratory infections and wearing a mask when going outside
- meticulous personal hygiene, handwashing technique and frequent mouth care
- wearing a MedicAlert® bracelet or pendant
- follow-up visits to the doctor or clinic
- helpful resources:
  - Transplant Australia
  - local and state support groups and organisations related to the specific organ transplant.

IMPAIRED IMMUNE RESPONSES

Disorders of impaired immune system responses may be either congenital or acquired. Often the function of either T or B cells is impaired, reducing the body's ability to defend against foreign antigens or abnormal host tissue.

No matter what the cause, people with immunodeficiency disorders demonstrate an unusual susceptibility to infection. When the antibody-mediated response is primarily affected, the person is at particular risk of severe and chronic bacterial infections. These people also do not develop long-lasting immunity to such diseases as chickenpox and are prone to recurrent cases. People with a defect of cell-mediated immunity tend to develop disseminated viral infections such as herpes simplex and CMV. Candidiasis and other fungal infections are also common. Because T cells are involved with activating antibody-mediated immune responses as well, overwhelming bacterial infections may occur. Immunodeficiency in its most

severe form occurs when both antibody-mediated and cell-mediated responses are impaired. People with combined immunodeficiency are susceptible to all varieties of infectious organisms, including those not normally considered to be pathogens.

Most immunodeficiency diseases are genetically determined and rare. They affect children more than adults. The noted exception is AIDS, an infectious disease caused by a virus.

THE PERSON WITH HIV INFECTION

In 1981, five cases of *Pneumocystis carinii* pneumonia (PCP) and 26 cases of a rare cancer, Kaposi's sarcoma, were diagnosed in young, previously healthy homosexual males in Los Angeles and New York City. The term **acquired immunodeficiency syndrome (AIDS)** was ascribed to this new phenomenon to describe the immune system deficits associated with these opportunistic disorders. Prior to this time, both PCP and Kaposi's sarcoma had been seen only in the older person, or in debilitated or severely immunodeficient people. Other groups at risk of AIDS were soon identified: injection drug users, people with haemophilia, recipients of blood transfusions and immigrants from Haiti.

Research to identify the cause of this apparently new disease progressed feverishly and, in 1983, a common antibody was identified in people with AIDS. The **human immunodeficiency virus (HIV)** was isolated in 1984. It then became apparent that AIDS was the final, fatal stage of HIV infection.

It began, like so many epidemics, with a few isolated cases and has become a worldwide plague (see the accompanying 'Focus on cultural diversity' box). AIDS invades our lives in ways we never imagined—testing our scientific knowledge, probing our private values and eluding a vaccine or a cure. Progression of HIV-positive disease to AIDS has slowed because of the effectiveness of highly active antiretroviral therapy (HAART) (Centers for Disease Control and Prevention (CDC), 2006). This change in progression to AIDS makes monitoring of AIDS less useful as an indicator of infected cases. For that reason, the CDC developed new surveillance methods based on infection rates in high-risk populations.

Although the incidence of HIV has levelled and mortality due to AIDS has declined, the epidemic is far from over.

Incidence and prevalence

In comparison with other countries, the overall incident rates of HIV/AIDS in Australia are low. In Australia, from the beginning of the HIV/AIDS epidemic to 31 December 2010 there have been 30 486 diagnosed cases of HIV (27 701 males and 2459 females), with an estimated 21 391 people living in Australia with HIV infection in 2010, and with 58% of newly diagnosed cases being Australian born (Kirby Institute, 2011a). A total of 10 446 people have been diagnosed with AIDS (9842 males and 568 females) and there have been a total of 6776 AIDS attributed deaths (6469 male and 307 female) (Kirby Institute, 2010). The relatively stable rates of AIDS diagnosis from 2001 to 2007 and the decrease in deaths from 149 in 2000 to 53 in 2007 are attributed to the use of effective antiviral therapies (Kirby Institute, 2009). Within Australia, funding is provided to professional and community organisations to assist in research and provision of education programs aimed at increasing knowledge and awareness within the community.

FOCUS ON CULTURAL DIVERSITY HIV/AIDS

In Australia, from 2001 to 2010, the rate of HIV among the Indigenous (Aboriginal and Torres Strait Islander) population remained similar to that of the non-Indigenous population. HIV transmission in Australia during 2006–2010 was primarily through male homosexual contact, in 70.8% of the non-Indigenous and 48.5% of the Indigenous cases. Exposure from heterosexual contact is reported as 16% of the non-Indigenous and 18.5% of the Indigenous cases. A higher proportion of incidence attributed to injection drug use was reported in Indigenous cases (19.4%) compared to non-Indigenous cases (2.5%). There was a higher proportion of HIV among Indigenous women (21.4%) compared to non-Indigenous women (8%). In 2010, the rate of HIV diagnosis in both the Indigenous and non-Indigenous Australian population was highest among residents in the major cities of Australia (Kirby Institute, 2011a; 2011b).

HIV/AIDS worldwide

There are an estimated 33 million people infected with AIDS worldwide, with virtually every country in the world reporting cases of AIDS (UNAIDS, 2007). The highest incidence is found in sub-Saharan Africa, South and South-East Asia, the United States, Western Europe, South America and Canada. Approximately 67% of all people infected with HIV or who have AIDS live in sub-Saharan Africa; another 15% live in South and South-East Asia, largely in Thailand and India. The most common mode of transmission is heterosexual intercourse. The cofactors that increase the risk of transmission include the presence of ulcerative or inflammatory sexually transmitted diseases, trauma, menses and lack of male circumcision (Papadakis et al., 2013; UNAIDS, 2007).

Critical thinking in person-centred care

1 Identify specific ways in which an HIV/AIDS prevention program focusing on injection drug use might be tailored to Aboriginal and Torres Strait Islander people.

2 What additional areas for research might further knowledge and guide strategies for HIV prevention in high-risk groups?

A continued decline in deaths is dependent on access to quality care and treatment and continued development of treatments for those already heavily treated (Papadakis et al., 2013).

In Australia, HIV risk criteria and indicators include sharing syringes, STI diagnosis or other clinical indicators of HIV, occupational or non-occupational exposure to HIV, unprotected intercourse with an HIV-positive person, unprotected intercourse between males and being born in or travelling to countries with a high HIV prevalence. Routine HIV testing is provided to antenatal women. Mandatory testing is carried out for specific visas; upon entry to the Australian Armed Forces; for all organ, tissue or blood donations; and in some legal or insurance requirements (National HIV Testing Policy, 2011).

The risk factors for HIV infection are behavioural. In adults in Australia, 79.7% of reported cases from 2006 to September 2008 involved male homosexual/bisexual contact; heterosexual contact was reported in 8.5% of cases. Unprotected anal intercourse is the major route of transmission in this group. Male homosexual/bisexual contact and injection drug use was reported in 5.1% of cases. Haemophiliacs, who require large amounts of intravenous clotting factors, and people infected through blood transfusion or tissue donation account for a small number of cases, approximately 2.8% (Kirby Institute, 2009). From 2004 to 2008, Australian-born people accounted for 58% of new diagnoses of HIV in Australia, and 9% of cases were reported to be in people who spoke English as their second language (Kirby Institute, 2009).

Declining immune system function in older adults significantly increases their risk of contracting HIV/AIDS, along with the belief that they cannot be affected by it. Just as younger people with HIV/AIDS contract the diseases primarily through sexual intercourse, so does the older population. Because older adults are beyond childbearing years, they often fail to use condoms when engaging in sexual activity. Manifestations may be overlooked by healthcare professionals, leading to a delayed diagnosis and increased severity of the disease.

HIV is a retrovirus transmitted by direct contact with infected blood and body fluids. Significant concentrations of the virus are present in blood, semen, vaginal and cervical secretions and cerebrospinal fluid (CSF) of infected individuals. It is also found in breast milk and saliva. Sexual contact is the primary mode of transmission. HIV is also transmitted through contact with infected blood via needle sharing during injection drug use or by transfusion. Approximately 13% to 40% of infants born to HIV-positive mothers are infected perinatally. Breastfeeding is a route of transmission and should be avoided (Papadakis et al., 2013).

Less than 0.04% of people voluntarily donating blood (a process that generally excludes people with high-risk behaviour) are found to be HIV positive. HIV is not transmitted by casual contact, nor is there any evidence of its transmission by vectors such as mosquitoes. Blood donation also poses no risk of contracting HIV to the donor, because only new, sterile equipment is used. A small but real occupational risk exists for healthcare workers. Percutaneous exposure to infected blood or body fluids through a needle-stick injury or non-intact skin is the primary route of transmission. Documented evidence indicates that parenteral exposure poses a 0.3% risk of becoming HIV positive (Carrico, 2001; Papadakis et al., 2013). Mucosal exposures, such as splashing in the eyes or mouth, pose a much smaller risk.

Pathophysiology and manifestations

As mentioned, HIV is a retrovirus, meaning it carries its genetic information in RNA. On entry into the body, the virus infects cells that have the CD4 antigen. Once inside the cell, the virus sheds its protein coat and uses an enzyme called *reverse transcriptase* to convert the RNA to DNA (see Figure 12.7). This viral DNA is then integrated into host cell DNA and duplicated during normal processes of cell division. Within the cell, the virus may remain latent or become activated to produce new RNA and to form *virions*. The virus then buds from the cell surface, disrupting its cell membrane and leading to destruction of the host cell.

Although the virus may remain inactive in infected cells for years, antibodies are produced to its proteins, a process known as **seroconversion**. These antibodies are usually detectable 6 weeks to 6 months after the initial infection. Helper T or CD4 cells are the primary cells infected by HIV. It also infects macrophages and certain cells of the CNS. Helper T cells play a vital role in normal immune system function, recognising foreign antigens and infected cells and activating antibody-producing B cells. They also direct cell-mediated immune activity and influence the phagocytic activity of monocytes and macrophages. The loss of these helper T cells leads to the immunodeficiencies seen with HIV infection (Porth & Matfin, 2009). Figure 12.8 illustrates the typical course of HIV infection.

The clinical manifestations of HIV infection range from no symptoms to severe immunodeficiency with multiple opportunistic infections and cancers (see the 'Manifestations' box below). It appears that the majority of people develop an acute mononucleosis-type illness within days to weeks after contracting the virus. Typical manifestations include fever, sore throat, arthralgias and myalgias, headache, rash and lymphadenopathy. Pathological changes are also noted in the CNS of many infected individuals, although the mechanism of neurological dysfunction is unclear. The person may also experience nausea, vomiting and abdominal cramping. The person often attributes this initial manifestation of HIV infection to a common viral illness such as influenza, upper respiratory infection or stomach virus.

Following this acute illness, people enter a long-lasting asymptomatic period. Although the virus is present and can be transmitted to others, the infected host has few or no symptoms. Clearly, the majority of HIV-infected people are in this stage of the disease. The length of the asymptomatic period varies widely, but its mean length is estimated to be 8 to 10 years.

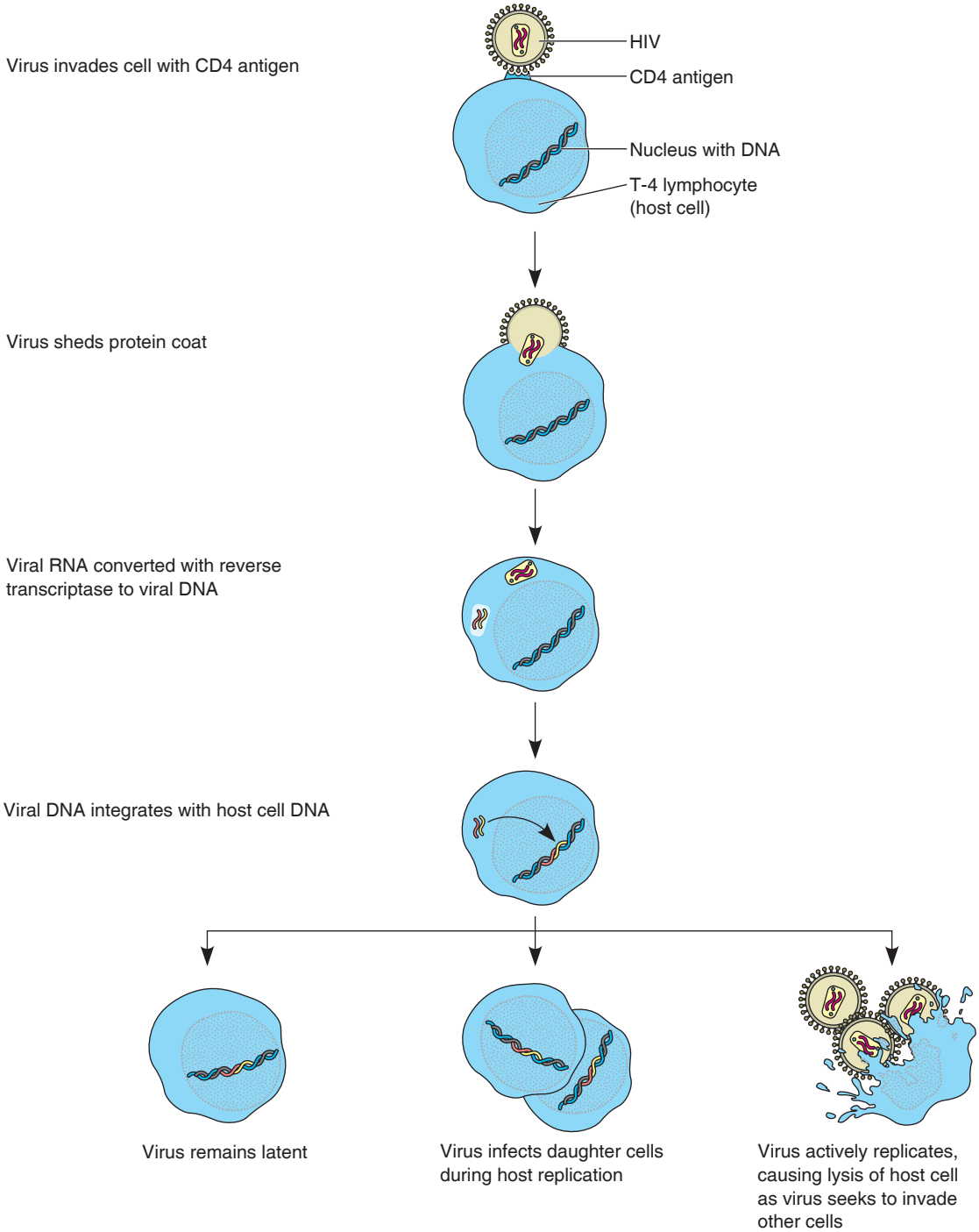


FIGURE 12.7 ■ How HIV infects and destroys CD4 cells

Some people with few other symptoms develop persistent generalised lymphadenopathy. This is defined as enlargement of two or more lymph nodes outside the inguinal chain, with no other illness or condition to account for the lymphadenopathy.

The move from asymptomatic disease or persistent lymphadenopathy to AIDS is often not clearly defined. The person may complain of general malaise, fever, fatigue, night sweats and involuntary weight loss. Persistent skin dryness and rash may be a problem. Diarrhoea is common, as are oral lesions such as hairy leucoplakia, candidiasis, gingival inflammation and ulceration. The development of advanced HIV typically occurs 10 to 11 years after initial infection; this varies according to the viral load, rate of disease progression and the development of resistance to antiretroviral therapy (Kenny, 2004).

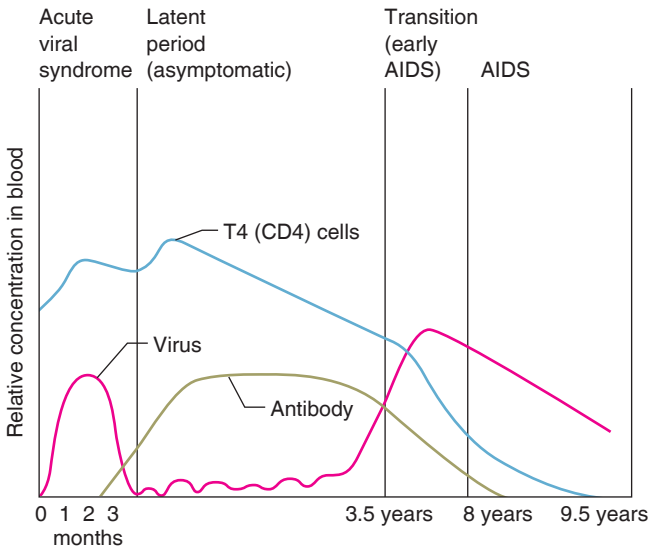


FIGURE 12.8 ■ The progression of HIV infection. Acute illness develops shortly after the virus is contracted, corresponding with a rapid rise in viral levels. Antibodies are formed and remain present throughout the course of infection. Late in the disease, viral activation results in a marked increase in virus while CD4 (T4) cells diminish as they are destroyed with viral replication. Antibody levels gradually decrease as immune function is impaired

With the development of significant constitutional disease, neurological manifestations or opportunistic infections or cancers, the person has manifestations that are characteristic of AIDS and a very poor prognosis. HIV infection and AIDS may be classified by using the CDC's matrix classification system. Under this system, HIV disease is determined by the presence of clinical symptoms (clinical categories A, B and C) and by T4 cell counts (categories 1, 2 and 3) (see Box 12.2). When a person's T4 cell count falls to < 200, they have late-stage AIDS; < 50 is end-stage AIDS (Coyne, Lyne & Watson, 2002).

When clinical manifestations develop, the outcome varies. With antiretroviral therapy (ART), many people are living longer after being diagnosed with AIDS. Today, PCP is most commonly diagnosed in those who are undiagnosed or have a late diagnosis of HIV infection, or who fail to take prophylactic antibiotics when their CD4 count is < 200. ART is credited with decreasing the incidence of opportunistic infections and improving survival (CDC, 2004). The time of survival has increased from about 13 months at the start of the AIDS epidemic; however, survival after diagnosis of HIV-related lymphomas still averages less than 8 months.

AIDS dementia complex and neurological effects

Neurological manifestations of HIV are common, affecting 40% to 60% of people with AIDS. Included among the manifestations are dementia, delirium and seizures. They result from both the direct effects of the virus on the nervous system and opportunistic infections.

AIDS dementia complex is the most common cause of mental status changes for people with HIV infection. This dementia results from a direct effect of the virus on the brain and affects cognitive, motor and behavioural functioning. Fluctuating memory loss, confusion, difficulty concentrating, lethargy and diminished motor speed are typical manifestations of AIDS dementia complex. People become apathetic, losing interest in work and social and recreational activities. As the complex progresses, the person develops severe dementia with motor disturbances such as ataxia, tremor, spasticity, incontinence and paraplegia (Fauci et al., 2008; Porth & Matfin, 2009).

Infections and lesions common with AIDS may also affect the CNS. Toxoplasmosis and non-Hodgkin's lymphoma are space-occupying lesions that may cause headache, altered mental status and neurological deficits. Cryptococcal meningitis and CMV infection also are common in people with AIDS. CNS complications have declined with the use of HAART therapy (Papadakis et al., 2013).

MANIFESTATIONS HIV infection and AIDS

I. Acute retroviral syndrome (ARS) or primary HIV infection
- Fever
- Sore throat
- Arthralgias and myalgias
- Headache
- Rash
- Nausea, vomiting and abdominal cramping

II. Asymptomatic infection
- None; converts to seropositive status

III. Persistent generalised lymphadenopathy
- Enlargement of two or more extrainguinal sites for more than 3 months

IV. Other acute disease symptoms
- General malaise, fatigue
- Low-grade fever
- Night sweats
- Involuntary weight loss
- Skin dryness or rashes

V. Other diseases and AIDS
A. *AIDS dementia complex*
B. *Secondary infectious diseases*
- *Pneumocystis carinii* pneumonia
- *Mycobacterium* tuberculosis
- *Mycobacterium avium* complex
- Candidiasis
- Cryptosporidiosis
- Cryptococcosis
- Toxoplasmosis
- Herpes simplex or herpes zoster
- Cytomegalovirus

C. *Secondary cancers*
- Kaposi's sarcoma
- Non-Hodgkin's lymphoma
- Cervical dysplasia and cervical cancer

D. *Other conditions*
- Pelvic inflammatory disease
- Human papillomavirus

BOX 12.2 Classification system for HIV infection and expanded AIDS surveillance case definition for adolescents and adults

DIAGNOSTIC CATEGORIES	CLINICAL CATEGORIES		
	A	B	C
CD4 + T-CELL CONDITIONS	ASYMPTOMATIC, ACUTE (PRIMARY) HIV OR PERSISTENT GENERALISED LYMPHADENOPATHY (PGL)	SYMPTOMATIC, NOT (A) OR (C) CONDITIONS	AIDS-INDICATOR CATEGORIES
(1) ≥500/mm^3	A1	B1	C1
(2) 200– 499/mm^3	A2	B2	C2
(3) <200/mm^3	A3	B3	C3

As of 1 January 1993, people with AIDS-indicator conditions (clinical category C) and those in categories A3 or B3 were considered to have AIDS.

Clinical category A

One or more of the following conditions in an adolescent or adult with documented HIV infection and without conditions in categories B and C:

- Asymptomatic HIV infection
- Persistent generalised lymphadenopathy
- Acute HIV infection with accompanying illness or history of acute HIV infection

Clinical category B

Examples of conditions, but not limited to:

- Candidiasis oral (thrush) or vulvovaginal (persistent, frequent or poorly responsive to therapy)
- Cervical dysplasia/cervical carcinoma in situ
- Constitutional symptoms, such as fever (38.5°C) or diarrhoea exceeding 1 month duration
- Hairy leucoplakia
- Herpes zoster involving at least two distinct episodes
- Pelvic inflammatory disease
- Peripheral neuropathy

Clinical category C

- Candidiasis of bronchi, trachea or lungs; oesophagus
- Coccidioidomycosis
- Cryptococcosis
- Cryptosporidiosis with persistent diarrhoea
- Cytomegalovirus infection (other than of liver, spleen or lymph nodes)
- CMV retinitis
- HIV encephalopathy
- Herpes simplex: chronic ulcers or bronchitis, pneumonitis or oesophagitis
- *Mycobacterium avium* complex or disseminated
- *Mycobacterium tuberculosis*
- *Pneumocystis carinii* pneumonia
- Progressive multifocal leucoencephalopathy
- *Salmonella* septicaemia
- Toxoplasmosis of the brain
- Kaposi's sarcoma
- Cervical cancer, invasive
- Lymphoma
- HIV wasting syndrome

Source: Adapted from CDC (1993). Revised classification system for HIV infection and expanded case definition for AIDS among adolescents and adults. *MMWR Recommendations and Reports, 41*(RR-17), 1–19.

Peripheral nervous system manifestations are also common in HIV-infected people. Sensory neuropathies with manifestations of numbness, tingling and pain in the lower extremities affect about 30% of people with AIDS. A Guillain-Barré type of inflammatory demyelinating polyneuropathy can also occur, resulting in progressive weakness and paralysis.

Opportunistic infections

Opportunistic infections are the most common manifestations of AIDS, often occurring simultaneously. The risk of opportunistic infections is predictable by the T4 or CD4 cell count. The normal CD4 cell count is 500 to 1350/mm^3. When the CD4 count falls to less than 500/mm^3, manifestations of immunodeficiency are seen. With a count of less than 250/mm^3, opportunistic infections and cancers are likely.

PNEUMOCYSTIS CARINII PNEUMONIA

Pneumocystis carinii pneumonia is the most common opportunistic infection affecting people with AIDS. Approximately 75% to 80% of people develop PCP at some point in their disease (Papadakis et al., 2013). It tends to be recurrent and is the cause of death in about 20% of people with AIDS. PCP is caused by a common environmental fungus that is not pathogenic in people with intact immune systems.

Unlike many pneumonias, the manifestations of PCP are non-specific and may progress insidiously. People often present with fever, cough, dyspnoea, tachypnoea and tacycardia. Complaints of mild chest pain and sputum may also be present. Breath sounds may initially be normal. With severe disease, the person may present with cyanosis and significant respiratory distress.

TUBERCULOSIS In some people, active tuberculosis results from reactivation of a prior infection. In other people, it is a new, primary disease facilitated by impaired immune function. Rapid progression, diffuse pulmonary infiltrates and disseminated disease occur more commonly in people with AIDS. Multidrug-resistant strains of tuberculosis present a significant problem (Papadakis et al., 2013).

People with pulmonary tuberculosis present with a productive cough of purulent sputum, fever, fatigue, weight loss and lymphadenopathy. Disseminated disease affects the bone marrow, bone, joints, liver, spleen, CSF, skin, kidneys, gastrointestinal tract, lymph nodes, brain and other sites.

CANDIDIASIS *Candida albicans* infection is a common opportunistic infection in people with AIDS. It is usually manifested as oral thrush or oesophagitis. In women, vaginal candidiasis is frequent and often recurrent. Oral thrush presents as white, friable plaques on the buccal mucosa or tongue and, in the HIV-infected person, is often the first indication of progression to AIDS. People with oesophagitis have difficulty swallowing and substernal pain or burning that increases with swallowing.

***MYCOBACTERIUM AVIUM* COMPLEX** *Mycobacterium avium* complex (MAC) affects up to 25% of people with AIDS, typically occurring late in the course of the disease when CD4 cell counts are less than 100/mm^3. MAC is more common in women than men. MAC is caused by organisms commonly found in food, water and soil. It is a major cause of 'wasting syndrome' in people with AIDS (see Figure 12.9). Manifestations of MAC include chills and fever, weakness, night sweats, abdominal pain and diarrhoea, and weight loss. Nearly every organ can be infected and most people with MAC develop disseminated disease.

OTHER INFECTIONS Herpesvirus infections are common in people with AIDS and may be severe. CMV can affect the retina, the gastrointestinal tract or lungs. Disseminated herpes simplex or herpes zoster may occur, although severe mucocutaneous manifestations are more common.

Parasitic infections with *Toxoplasma gondii* and *Cryptococcus neoformans* commonly affect the CNS. Toxoplasmosis occurs as encephalitis or an intracerebral mass lesion. Changes in mental status, focal neurological signs and seizures may result. *Cryptococcus* infection may present as either meningitis or disseminated disease primarily affecting the lungs. *Cryptosporidium*, a protozoon affecting the gastrointestinal tract, is an important cause of prolonged diarrhoea in people with AIDS. Bacterial *Salmonella* infections are also a relatively common cause of diarrhoea.

Women with AIDS have a high incidence of pelvic inflammatory disease (PID). Although the pathogens appear to be the same as those in PID affecting non-HIV-infected women, the disease is more severe. Inpatient treatment with intravenous antibiotics is often necessary.

Secondary cancers

As cell-mediated immune function declines, the risk of malignancy increases. The CDC classification of AIDS currently includes four cancers: Kaposi's sarcoma, non-Hodgkin's lymphoma, primary lymphoma of the brain and invasive cervical carcinoma.

KAPOSI'S SARCOMA **Kaposi's sarcoma (KS)** is often the presenting symptom of AIDS. It remains the most common cancer associated with the disease. Kaposi's sarcoma is caused by a virus called the Karposi-sarcoma-associated herpes virus, also known as human herpes virus 8. Men who have sex with men not only have a risk of HIV infection, but are also more likely to be infected with the virus responsible for KS. Women who have sex with these men also have a risk of HIV and KS. The virus associated with KS appears to be mainly transmitted through sexual contact, although cases have also been reported in injection drug users. People whose immune system is suppressed because they have received an organ transplant have a 1 in 200 risk of developing KS (American Cancer Society, 2005).

A tumour of the endothelial cells lining small blood vessels, KS presents as vascular macules, papules or violet lesions affecting the skin and viscera (see Figure 12.10). The face is a common site for skin lesions, especially the tip of the nose and

FIGURE 12.9 ■ Wasting syndrome in a person with AIDS

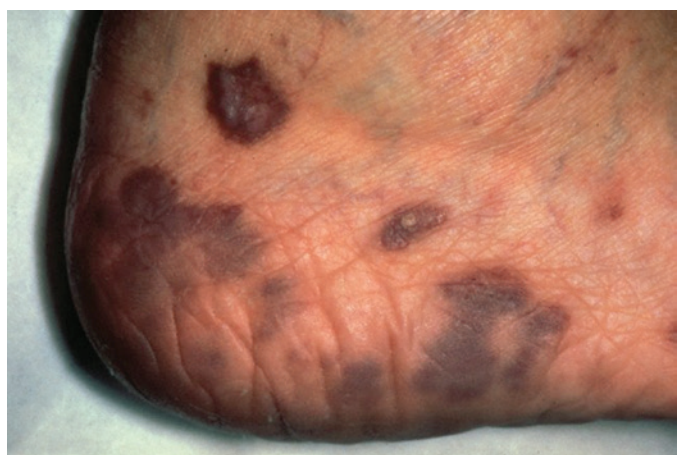


FIGURE 12.10 ■ Kaposi's sarcoma lesions

Source: © Carolina Biological/Visuals Unlimited/Corbis.

pinnae of the ears. Common sites for visceral disease include the gastrointestinal tract, lungs and lymphatic system.

The lesions of KS are usually painless initially, but may become painful as the disease progresses. Internally, the tumours may obstruct organ function or cause bleeding. When the lungs are involved, gas exchange may be severely impaired, resulting in pulmonary haemorrhage. KS is an indicator of late-stage HIV disease, with an average survival time of 18 months after diagnosis. The disease may progress slowly or rapidly. Rapidly progressing KS is treated with chemotherapy; milder forms may improve with the initiation of HAART therapy (Papadakis et al., 2013).

LYMPHOMAS Lymphomas are malignancies of the lymphoid tissue, including lymphocytes, lymph nodes and the lymphoid organs such as the spleen and bone marrow. In AIDS, two lymphomas are common: non-Hodgkin's lymphoma (including Burkitt's lymphoma) and primary lymphoma of the brain. Hodgkin's disease also occurs five times more frequently in people with HIV infection than in those without. The CNS is the usual site for these lymphomas, although they may be found in the bone marrow, gastrointestinal tract, liver, skin and mucous membranes. They are aggressive tumours, growing and spreading rapidly. Headache and changes in mental status are common early symptoms of lymphomas affecting the CNS.

CERVICAL CANCER Of women with HIV infection, 40% have cervical dysplasia. Cervical cancer develops frequently and tends to be aggressive. Women with concurrent HIV infection and cervical cancer usually die of the cervical cancer, not AIDS. Because of this, it is recommended that women with HIV infection have Papanicolaou (Pap) smears every 6 months and aggressive treatment of cervical dysplasia with colposcopic examination and cone biopsy.

INTERPROFESSIONAL CARE

Although multiple research studies to identify a cure for HIV infection and AIDS are under way, no cure is currently available. This fact, plus the apparent universally fatal nature of the disease, makes prevention a vital strategy in HIV care. New treatments are under investigation (see Box 12.3).

The goals of care for the person with HIV disease are as follows:

- early identification of the infection
- promoting health-maintenance activities to prolong the asymptomatic period as long as possible
- prevention of opportunistic infections
- treatment of disease complications, such as cancers
- providing emotional and psychosocial support.

Diagnosis

Diagnostic testing is used to screen and identify the infection, as well as to monitor the person's disease and immune status. The following diagnostic tests may be ordered. The likelihood that a positive screening test truly indicates the presence of HIV infection decreases as HIV prevalence in the tested population becomes lower. Therefore, false-positive HIV test results are more likely in settings where the tested population prevalence is lower than in settings where the tested population prevalence is higher. When a preliminary, positive rapid test is

BOX 12.3 Investigational immune-based treatment for HIV

HIV infection progressively alters the function of and destroys $CD4^+$ lymphocytes. $CD4^+$ cells are essential to the function of the immune system, including the body's ability to respond to infections. These cells initiate, direct and regulate immune responses and may also directly attack infected cells. They also are a source of cytokines, the chemical messengers of the immune system. Destruction of $CD4^+$ cells by HIV devastates the immune system, facilitating the development of fatal infections and neoplasms in the infected person. Immune-based treatments indirectly affect the HIV by improving the function of the immune system through actions that inhibit cytokines, replenish cytokines or restore immune function. These treatments, used alone or in combination with antiretroviral drugs, are being investigated for use in the treatment of HIV.

Inhibiting cytokines

Tumour necrosis factor alpha (TNF-α) is a cytokine secreted by activated monocytes and macrophages in response to infection, infestation or tumour growth. It causes a proliferation of B cells and T cells. However, high levels of this cytokine may actually facilitate the development of disease by blocking the normal inflammatory response. It is believed that blocking the effect of TNF-α can suppress HIV production, although caution must be used.

Replenishing cytokines

Some cytokines (interleukin-2, interleukin-12 and interferon alpha) may be helpful in treating HIV by stimulating the production of killer cells as well as increasing the function of lymphocytes. The interferons are part of the body's first line of defence against viruses. All of these agents have toxic side effects, requiring careful nursing assessment and care.

Restoring immune system function

HIV infection not only destroys $CD4^+$ cells, but also eventually destroys the lymphoid organs, such as bone marrow and the thymus gland. Lymphocytes, including the $CD4^+$ cells, are derived from stem cells in bone marrow and mature in the thymus. Two investigational treatments to restore the immune system are bone marrow transplant and thymus transplant. Bone marrow transplants have been used to correct other types of immune disorders (such as leukaemia or lymphoma) but have yet to be effective in people with HIV. A few thymus transplants have been done in HIV-infected people, but have provided only temporary benefits.

explained to people, phrases like 'a good chance of being infected' or 'very likely infected' can be used to indicate the likelihood of HIV infection and can be qualified based on the HIV prevalence in the setting and the person's individual risk.

Further testing is always required to confirm a reactive screening test result.

- *HIV rapid antibody tests* are available in many countries but not yet in Australia.
- *Enzyme-linked immunosorbent assay (ELISA)*, also called the HIV antigen/antibody test, is the most widely used screening test for HIV infection. The ELISA test was developed in 1985 to screen blood donors. ELISA tests for HIV antibodies; it does not detect the virus. Therefore, a person may have a negative ELISA test early in the course of infection, before detectable antibodies have developed. The test has a 99.5% or higher sensitivity when performed at least 13 weeks after infection. This means that more than 99.5% of tests performed on blood containing HIV antibodies will show a positive result. False positives can occur; therefore, an initial positive result is always tested repeatedly and confirmed using a different method of antibody detection, usually the Western blot.
- Immediate notification is critical because many people tested for HIV do not return to learn the results; many cannot be located to give the test results and educate about safe behaviours, whether they are positive or negative for HIV. Although confirmation of results is dependent on testing with a second source, ELISA or Western blot test, learning results immediately gives the person more information to make wise choices about their behaviours and self-care.
- *Western blot antibody testing* is more reliable but more time-consuming and more expensive than ELISA. When combined with ELISA, however, a specificity of greater than 99.9% is achieved. Specificity is a measure of the probability that a negative test result indicates that no antibodies are present. In this test, the person's serum is mixed with HIV proteins to detect reaction. If antibodies to HIV are present, a detectable antigen–antibody response will occur.
- *HIV viral load tests* measure the amount of actively replicating HIV. Levels correlate with disease progression and response to antiretroviral medications. Public health guidelines state treatment should be considered for asymptomatic people with HIV with viral loads greater than 5000 to 100 000 copies/mL. HIV viral load testing is used in conjunction with CD4 cell count to inform treatment decisions.
- *FBC* is performed to detect anaemia, leucopenia and thrombocytopenia, which are often present in HIV infection. Lymphopenia (or low levels of lymphocytes) is especially common in this disease.
- *CD4 cell count* is the most widely used test to monitor the progress of the disease and guide therapy. The CD4 cell count correlates very closely with the immunodeficiency disorders seen in AIDS. AIDS is now defined not only by the presence of opportunistic infections and other diseases indicative of immunodeficiency, but also by HIV-seropositive status; the CD4 count goes down as the disease progresses. The standard reference range for CD4 count is dependent on many factors. The doctor monitors the pattern for changes over several months to determine strategies for optimal treatment.

In addition to these widely used tests, several other diagnostic tests may be performed:

- *Blood culture for HIV* provides the most specific diagnosis but is an expensive and cumbersome test that is not widely available.
- *Immune-complex-dissociated p24 assay* is a test for p24 (HIV) antigen in the blood. This antigen indicates active reproduction of HIV and tends to be positive prior to seroconversion and with advanced disease. It is most useful in monitoring disease progression and the antiviral activity of experimental medications (Pagana & Pagana, 2013; Papadakis et al., 2013).

Other diagnostic tests are used primarily to detect secondary cancers and opportunistic infections in the person with HIV. Tests ordered are both general and specific to the person's manifestations and may include the following:

- *tuberculin skin testing* to detect possible tuberculosis infection
- *MRI* of the brain to identify lymphomas
- *specific cultures and serology examinations for opportunistic infections* such as PCP, toxoplasmosis and others
- *Pap smears* every 6 months for early detection of cervical cancer in women with cervical dysplasia (Papadakis et al., 2013).

Medications

Pharmacological management of the person with HIV disease has four primary foci:

1. to suppress the infection itself, decreasing symptoms and prolonging life
2. to provide prophylaxis of opportunistic infections
3. to stimulate haematopoietic response
4. to treat opportunistic infections and malignancies.

Effectiveness of treatment is monitored by viral load and CD4 cell counts; positive results are indicated by a reduction in viral load along with preserving the CD4 count above 350/mm^3. Treatment is recommended when the CD4 count falls below 200/mm^3. People with symptoms of severe disease are treated regardless of their CD4 level or viral load, so monitoring these individuals may reveal higher levels of CD4 or lower viral load. Initiating therapy in asymptomatic individuals with higher CD4 levels has not shown a protective effect and is thought to perhaps increase viral resistance. Today the drugs have been combined and dosing schedules simplified, which helps people adhere to medication administration schedules. Currently, researchers are using clinical trials of asymptomatic people receiving HAART to evaluate alternating drug regimens to prevent drug resistance by the viral organisms (Martinez-Picado et al., 2003).

Four classes of drugs used in antiretroviral treatment include nucleoside reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs) and entry inhibitors. HAART combines three or four antiretroviral drugs to reduce the incidence of drug

resistance. Combination therapies increase the likelihood of decreasing viral load and symptoms but also burden people with complicated and expensive medication schedules. People beginning the HAART protocol must understand the benefits, risks, costs and effects on daily life. HAART does not eradicate HIV infection. HAART medications are expensive, particularly the newer triple combinations such as Trizivir. These medications are scheduled for specific times throughout the day; therefore, leading a normal life becomes a challenge. In addition, as with most chronic diseases, all HAART medications cause major adverse reactions leading to less than perfect adherence; however, in this case, the outcome could be fatal.

Each person must be able to adhere to the treatment regimen. It may be preferable to delay initiating therapy until the person is able to agree to adherence so that irregular dosing does not lead to viral resistance. Some providers gauge a person's ability to follow the HAART regimen by their success with prophylaxis for an opportunistic infection.

Several methods to promote and ensure adherence are being used and studied. Wroe and Thomas (2003) found it helpful to distinguish between intentional and unintentional non-adherence and to treat them as separate entities. People's beliefs and internal logic were found to impact on intentional non-adherence; preparing people for the effects of HAART therapy by focusing on lessening people's reasons for not taking medication is believed to reduce intentional non-adherence. Enriquez and McKinsey (2004) emphasise the role of nursing in preventing drug resistance by assessing a person's readiness to adhere to the treatment regimen and intervening to overcome identified barriers to adherence prior to initiating therapy.

Another approach to adherence is the use of electronic monitoring devices EMDs (Bova et al., 2005). By placing a microprocessor in a medication cap, records are created of the time, date and frequency of bottle opening. Although this method does not guarantee that the medication will be taken even if the cap is removed, the record created is a source for follow-up and discussion between the provider and the person. Whether the person is asked to keep a diary of taking the medication, using an EMD to keep a record, or relying on pill count, adherence to medication regimen is critically important.

Ingersoll and Heckman (2005) found the most effective provider–person relationship for fostering adherence is a balance of appropriate challenge and support. Providers who were never confrontational seem to be perceived by people as giving permission to be less adherent. Although depression, substance use and financial considerations undoubtedly influence adherence to HAART therapy and need to be addressed, provider–person relationships seem to have the most influence on adherence behaviour.

NUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS

The NRTIs (also called nucleoside analogues) inhibit the action of viral reverse transcriptase, a retroviral enzyme that catalyses the substrates for conversion and copying of viral RNA to DNA sequences. This enzyme is necessary for viral integration into cellular DNA and replication. The nucleoside analogues act as a chemical decoy for building blocks of the formation of the DNA copy, preventing the RNA from being copied into DNA. Each drug substitutes for a particular nucleoside base at different points on the chain. See the 'Medication administration' guidelines below for this group of drugs.

Zidovudine (Retrovir, AZT) was the first antiretroviral agent approved for use with HIV infection. It remains in widespread use and has been shown to decrease symptoms and prolong the lives of people with AIDS. Zidovudine is often given to people with a CD4 cell count of less than 500/mm^3 because of evidence that it slows the progression to severe disease (Papadakis et al., 2013). Zidovudine may also be used prophylactically following a documented parenteral exposure to HIV. It is used in combination with ddI, ddC or 3TC (see below).

- Didanosine (ddI, Videx) also inhibits reverse transcriptase and viral replication. It is used in combination therapy with zidovudine.
- Stavudine (d4T, Zerit) is a retroviral inhibitor that has been shown to increase CD4 cell counts and decrease serum p24 antigen levels. Current use is for people who are intolerant of zidovudine.
- Lamivudine (3-TC, Combivir) is used for low CD4 cell counts or symptomatic disease as a first-line treatment in combination with zidovudine.
- Abacavir (Ziagen) is a potent inhibitor of reverse transcriptase; however, it may cause serious hypersensitivity reactions.
- Zidovudine plus lamivudine (Combivir) is a combination drug in use to decrease HIV zidovudine-resistant strains.

PROTEASE INHIBITORS

Protease is a viral enzyme necessary for the formation of specific viral protein needs for viral assembly and maturation. Protease inhibitors bond chemically with protease to block the function of the enzyme and result in the production of immature, non-infectious viral particles. When combined with other antiviral drugs, these chemicals increase the chance of eliminating the virus by interfering with different stages of its life cycle. However, viral resistance occurs rather quickly. PIs inhibit and induce metabolism of other drugs, so their use with other medications and the dose of those medications must be carefully planned. Some drugs will circulate longer because their metabolism is inhibited; others will be speedily metabolised and eliminated.

Protease inhibitors and nucleoside analogues are associated with serious metabolic derangements. These include elevated cholesterol and triglycerides, insulin resistance and diabetes mellitus, and changes in body fat composition, which are particularly distressing to the person. These body fat changes are primarily abdominal obesity and skeletal wasting. This set of symptoms is referred to as lipodystrophy (Papadakis et al., 2013). Elevated cholesterol should be treated with pravastatin or atorvastatin. Lovastatin and simvastatin react to PIs, so they need to be avoided. Dietary sources of cholesterol should be reduced.

- Saquinavir is used in combination with nucleoside analogues to treat progression of the disease.
- Ritonavir is used in combination with nucleoside analogues to treat progression of the disease.

MEDICATION ADMINISTRATION Antiretroviral nucleoside analogues

ZIDOVUDINE (AZT, AZIDOTHYMIDINE)

Zidovudine is the first antiretroviral agent developed to treat HIV infection. It interferes with reverse transcriptase, thus inhibiting replication of the virus. The usual dose is 250 mg twice daily. It is administered orally. Dose-limiting side effects are anaemia and neutropenia.

Nursing responsibilities

- Assess for possible contraindications to therapy, including allergic response or a CD4 count of greater than 350/mm^3.
- Administer by mouth, instructing the person to swallow capsules whole.
- Assess for adverse effects. Nausea and headache are common. They may be self-limiting, decreasing with time, or significant and continuing, necessitating a change of therapy. Nausea and neutropenia are treated with erythropoietin and G-CSF.
- Assess FBC with differential and creatinine phosphokinase. Notify the doctor of significant changes.

Health education for the person and family

- Antiretroviral medications will not cure HIV infection but slow its progress and reduce significant symptoms.
- Take the drug according to the directions for administration at least 0.5 hour before or 1 hour after meals, if tolerated.
- With this and all antiretroviral drugs, it is important to emphasise that the person is still infective and can pass the infection to others. Use safer sex practices and other measures to prevent transmission to partners. Do not donate blood or breastfeed.
- Notify the doctor if signs of an infection or adverse response to the medication develop: sore throat, swollen lymph glands, fever; unusual fatigue or weakness; easy bruising, bleeding gums or an injury that will not heal; persistent or intractable nausea; muscle pain or wasting.
- Continue all scheduled follow-up visits and laboratory studies to monitor for drug toxicity.
- Check with the doctor before taking any prescription or over-the-counter drug.

DIDANOSINE (DDI, VIDEX)

As with zidovudine, didanosine does not kill HIV but inhibits its replication within the cells. Its activity is similar to that of zidovudine. Didanosine has been shown to increase CD4 cell counts and lower p24 antigen levels (Papadakis et al., 2013). Didanosine is used alone for people who are intolerant or resistant to zidovudine. It is also being used with zidovudine in combination therapy regimens. Didanosine does not cause the anaemia associated with zidovudine, but it may cause neutropenia. Didanosine is also associated with an increased risk of pancreatitis, peripheral neuropathy and dry mouth.

Nursing responsibilities

- Assess for possible contraindications to didanosine therapy, including previous episodes of pancreatitis and impaired renal or liver function.
- Administer as directed and refer to the product information for administration instructions.
- Administer with caution to people taking vincristine, rifampin, pentamidine, ethambutol or metronidazole; the action of both drugs may be affected by concurrent administration. Intravenous pentamidine and trimethoprim-sulfamethoxazole taken concurrently may increase the risk of acute and fatal pancreatitis.
- Didanosine interferes with the absorption of ketoconazole and dapsone. Doses of these drugs should be scheduled at least 2 hours apart from didanosine doses.
- Evaluate for therapeutic response and possible adverse effects. Notify the doctor if manifestations of peripheral neuropathy, diarrhoea, depression or other adverse effects develop.
- Stop the drug and notify the doctor immediately if the person develops manifestations of pancreatitis or hepatic failure, including nausea and vomiting, severe abdominal pain, elevated bilirubin or elevated serum enzymes (e.g. amylase, AST, ALT).

Health education for the person and family

- Take the drug as directed. The prescribed dose must always be taken to get the required amount of antacid to prevent the drug from being destroyed by stomach acid.
- Take on an empty stomach, at least 1 hour before or 2 hours after meals.
- Do not drink alcohol while taking didanosine; alcohol may increase the risk of pancreatitis.
- Stop the drug and call the doctor immediately if nausea, vomiting, abdominal pain or diarrhoea develops. These may indicate pancreatitis.
- Call the doctor if extremity pain, weakness, numbness or tingling occurs. These side effects usually disappear when didanosine is discontinued.
- Other side effects to report to the doctor include unusual bleeding or bruising, fatigue, weakness, fever or persistent sore throat.

ABACAVIR

Abacavir is a nucleoside analogue with activity against some HIV strains resistant to other nucleoside drugs. It is prepared in combination with zidovudine and lamivudine (Trizivir) and one tablet is taken twice daily. This combination drug is composed exclusively of nucleoside analogues, lacking NNRTIs or PIs. As such, it is less effective at decreasing viral load and allowing immune system enhancement, but the ease of administration makes it a useful drug for people who cannot adhere to more complex regimens. The main toxicity is a hypersensitivity response in approximately 5% of people, manifested as flu-like symptoms. Avoid repeated use in those individuals.

Nursing responsibilities

- Assess for possible hypersensitivity reactions, anaemia and neutropenia.
- Evaluate for desired effect of increased CD4 counts and lower blood levels of p24 antigen.
- Notify the doctor if the person develops evidence of pancreatitis, impaired hepatic function or painful peripheral neuropathy.

Health education for the person and family

- Take without regard to food or water.
- Check with the doctor before taking any other prescription or over-the-counter medication.
- Report all signs and symptoms of hypersensitivity to the drug.
- Report to the doctor signs of infection, flu-like symptoms or changes in condition.

- Indinavir is used in combination with nucleoside analogues to treat progression of the disease.
- Nelfinavir is used in cases of failure of or intolerance to other protease inhibitors.
- Amprenavir is the newest protease inhibitor.
- Lopinavir/ritonavir is the first combination of protease inhibitors active against some HIV strains resistant to other protease inhibitors.

NON-NUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS Nevirapine, delavirdine and efavirenz are NNRTIs that may be used in combination with nucleoside analogues and protease inhibitors. However, one limitation to NNRTIs is the high incidence of cross-resistance to NRTIs. Some studies have shown that nevirapine and efavirenz may significantly reduce serum levels of the protease inhibitors. Only one NNRTI should be used at the same time. Nevirapine has a reported risk of liver toxicity and Stevens–Johnson syndrome (Bartlett & Weber, 2005).

ENTRY INHIBITORS: ENFUVIRTIDE (FUZEON) This new class of drugs became available in 2003. These entry or fusion inhibitors prevent HIV from entering target cells by binding to the protein envelope that surrounds the virus. When bound to the drug, the virus cannot morph in order to fit and adhere to cell membranes (Covington, 2005). Adding this new class of drug to the regimen of heavily pretreated individuals improves CD4 counts and lowers viral loads (Papadakis et al., 2013).

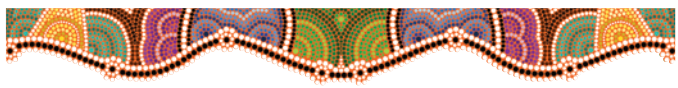
OTHER MEDICATIONS Other agents may also be administered in combination with antiretroviral therapy. Interferons, which are naturally occurring lymphokines, have been used alone and in combination. Interferon alpha may be used to treat KS and in combination with zidovudine to slow disease progression. Interferon gamma is also used. As more drugs become available, the burden to choose the best regimen increases for the healthcare provider. The most important limiting factor when choosing a regimen is a person's adherence. Second to that is selecting an effective combination of drugs without overlapping toxicities or toxicities so debilitating that adherence will be further impaired.

Some people undergoing HAART are developing body composition changes and metabolic abnormalities associated with the therapy, especially the PIs. Increased fat deposition in the midsection, breasts and neck, with atrophy in the face, buttocks and extremities, describes the body composition changes; metabolic abnormalities include increased low-density lipoprotein cholesterol and triglycerides and insulin resistance. The combination of changes is consistent with metabolic syndrome, which increases the risk of cardiovascular disease and diabetes. These conditions are commonly treated with medications. Robinson (2005) has observed these serious changes in HIV-positive people and hopes to prevent and treat the changes with diet and exercise without adding to the polypharmacy already experienced by those undergoing HAART.

A number of pharmacological agents are used to prevent and treat opportunistic infections and malignancies in the person with HIV. These agents are outlined in Table 12.4.

Many people at some point require a central venous access device, such as a Groshong catheter, to facilitate blood sampling, intravenous medication administration, transfusions and parenteral nutrition. See Chapter 13 for nursing care of the person with a central venous catheter.

It is recommended that all HIV-infected people receive pneumococcal, influenza, hepatitis B and *Haemophilus influenzae* b vaccines. People with a positive PPD and negative chest x-ray are given prophylactic isoniazid. When the person's CD4 cell count falls to less than 200/mm^3, prophylactic treatment for PCP is begun, usually with trimethoprim-sulfamethoxazole. People with a CD4 count of less than 100/mm^3 are started on prophylactic treatment for MAC.



Nursing care

The person with HIV and AIDS has many care needs, including both physical and psychosocial support (see the 'Translation to practice' box below). Because there is as yet no cure or effective treatment for HIV disease, many of these needs fall within the realm of nursing to promote knowledge and understanding, self-care, comfort and quality of life. As with many diseases that have an ultimately fatal outcome, the course of HIV infection may well be affected by the person's social support systems, control, perceived self-efficacy in management and coping mechanisms.

As the epidemic continues, nurses are providing care for increasing numbers of people with HIV infection at various stages of disease. These people are not only in special care settings but also in general units, maternal–child units, hospice and home settings. As people with HIV disease live longer, nurses will increasingly encounter people in whom HIV disease is a secondary diagnosis, with another primary diagnosis—for example, seizures, heart disease, diabetes mellitus or an operative procedure.

Prevention

To date, no safe immunisation to protect against HIV infection has been developed. Education, counselling and behaviour modification are the primary tools for AIDS prevention. The benefit of education and behaviour modification is evident in the homosexual male population. The incidence of new HIV infections in this population has declined dramatically in high-prevalence cities. Nurses play a vital role in providing education about this epidemic and infection prevention for individuals and communities.

All sexually active individuals need to know how HIV is spread. Following are the only *totally* safe sex practices:

- no sex
- long-term mutually monogamous sexual relations between two uninfected people
- mutual masturbation without direct contact.

TABLE 12.4 Pharmacological treatment of common opportunistic infections and malignancies in HIV disease

CONDITION	TREATMENT	POTENTIAL ADVERSE EFFECTS
Infections		
Pneumocystis carinii pneumonia	Trimethoprim/sulfamethoxazole Pentamidine	Rash, neutropenia, anaemia, thrombocytopenia, Stevens–Johnson syndrome Hypotension, altered blood glucose levels, hypocalcaemia, anaemia and leucopenia, liver and renal toxicity, pancreatitis
Tuberculosis	Combination drug therapy using isoniazid, rifampicin, ethambutol, pyrazinamide or streptomycin	Multiple; see Chapter 35
Candidiasis	Clotrimazole troches	Few toxic responses noted
Oral thrush	Nystatin suspension	
Oesophagitis or recurrent vaginitis	Ketoconazole Fluconazole Amphotericin B	Hepatitis, adrenal insufficiency Hepatitis Bone marrow toxicity, acute renal or hepatic failure; nausea, vomiting; chills, fever, headache
Mycobacterium avium complex	Combination therapy using • Clarithromycin, plus • Clofazimine • Ethambutol • Rifampin • Ciprofloxacin • Amikacin	 • Hepatitis, nausea, diarrhoea • Diarrhoea, nausea, vomiting; skin discolouration, pruritus, rash • Thrombocytopenia, hepatitis, optic neuritis • Bone marrow depression, renal failure, hepatitis • Nausea, rash • Bone marrow depression, renal failure, ototoxicity, hepatitis
Cytomegalovirus	Ganciclovir	Bone marrow depression, fever
	Foscarnet	Renal failure, electrolyte imbalances, seizures
Herpes simplex or herpes zoster	Aciclovir	Nausea, vomiting, diarrhoea; CNS effects; renal failure
Toxoplasmosis	Pyrimethamine, plus sulfadiazine or clindamycin and folinic acid	Bone marrow depression, rash; respiratory failure; nausea, vomiting, abdominal pain; haematuria
Malignancies		
Kaposi's sarcoma	Intralesional vinblastine	Inflammation and pain at injection site
Lymphoma	Combination chemotherapy	Nausea, vomiting; bone marrow toxicity; alopecia

People who do engage in sexual activity need to know and practise safer sex (see Box 12.4). Reducing the number of sexual partners—for example, by entering into and remaining in a long-term mutually monogamous relationship with an uninfected partner—reduces the risk. People should not engage in unprotected sex, especially if the HIV status of the partner is unknown. Latex condoms have been shown to reduce the risk of transmitting HIV. Their effectiveness is improved when nonoxynol-9, a spermicide, is used for lubrication; however, it may cause genital ulcers, which can facilitate HIV transmission. To be effective, condoms must be used with every sexual encounter involving vaginal, oral or anal intercourse. They also need to be applied and removed properly. A female condom is also available for use.

Healthcare workers exposed to HIV infection or adults who experience a high-risk exposure to HIV may choose postexposure prophylaxis. Risk of exposure for healthcare workers may be through needle sticks or cuts with a sharp object, contact with mucous membranes or non-intact skin, semen, vaginal secretions, fluids contaminated with visible blood and possibly CSF, synovial fluid and pleural, peritoneal, pericardial or amniotic fluids. CDC guidelines recommend treatment with HAART, which includes two NRTIs for lower-risk exposures and the addition of a third drug for higher-risk exposure. A 4-week course of treatment is recommended and should be started within 72 hours, preferably within 2 to 3 hours of exposure (Bartlett & Weber, 2005).

The most difficult group of high-risk people to reach and educate has been injection drug users. People in this group should never share needles, syringes or other drug paraphernalia. Australian cities have established needle-exchange programs, providing a sterile needle and syringe in exchange for a used one. It is important also to teach people in this population about safer sex practices, because most heterosexual HIV transmission occurs between injection drug users and their partners.

Australia's blood screening ensures one of the safest blood supplies in the world. Screening of voluntary blood donors and donated blood supplies commenced in 1985 and has reduced the risk of transmission of HIV/AIDS by transfusion, with no transmission via transfusion since 1985. Because current blood-screening methods use antibody testing, receiving donated blood

BOX 12.4 Guidelines for safer sex

- Practise mutual monogamy; if you are not in a mutually monogamous relationship, limit the number of sexual partners.
- Do not engage in unprotected sex, especially if the HIV status of your partner is unknown. (Remember that a person may be infected and infective for up to 6 months before converting to seropositive status.)
- When entering into a new monogamous relationship, both partners should undergo HIV testing initially. If both are negative, practise abstinence or safer sex for 6 months, followed by re-testing. If results still indicate that both partners are negative, sexual activity can probably be considered safe.
- Use latex condoms for oral, vaginal or anal intercourse; avoid natural or animal-skin condoms, which allow passage of HIV.
- For vaginal or anal sex, lubricate the condom with the spermicidal agent nonoxynol-9 for additional protection.
- Do not use an oil-based lubricant such as petroleum jelly, which can result in condom damage; water-based lubricants are acceptable.
- Women should carry and use a female condom.
- Remember that use of other means of birth control, such as oral contraceptives, provide no protection against HIV; barrier protection with a condom is necessary.
- Engage in safer sexual practices that are less damaging to sensitive tissues (e.g. mutual masturbation, avoiding anal or oral sex).
- Do not use drugs or alcohol.
- Do not share needles, razors, toothbrushes, sexual toys or other items that may be contaminated with blood or body fluids.
- If HIV positive:
  a. Do not engage in unprotected sexual activity.
  b. Inform all current and former sexual partners of HIV status.
  c. Inform all healthcare personnel—primary care providers, doctors and dentists, in particular—of HIV status.
  d. Do not donate blood, plasma, blood products, sperm organs or tissue.
  e. If female, do not become pregnant.

continues to carry a small risk. People in the *window period* between contraction of the virus and the development of detectable antibodies are able to transmit the virus to others, even though they do not yet test positive for HIV. This window period usually lasts from 6 weeks to 6 months; rarely, it lasts up to 1 year. When possible, encourage people to use autologous transfusion, donating their own blood prior to an anticipated surgery. Seeking donations from family members is not encouraged for several reasons. Family members may have engaged in high-risk behaviours but lie about their risk because of embarrassment or fear of discovery. Furthermore, the family member may have a different blood type or have other contraindications to donating.

Encourage HIV-positive people to abstain from donating blood organs or sperm. They should understand tactics to avoid exchange of body fluids by not sharing needles or other drug paraphernalia, not sharing razors and not obtaining a tattoo. Stress the importance of informing all medical personnel providing direct care (especially anyone performing a dental, surgical or obstetric procedure) about the diagnosis.

Healthcare workers can prevent most exposures to HIV by using standard precautions (see Figure 12.11). Testing to determine HIV status remains voluntary and relies on the use of antibody-screening methods. It is therefore impossible to identify every person who is HIV positive. With standard precautions, all people are treated alike, eliminating the need to know the person's HIV status. All high-risk body fluids are treated as if they are infectious and barrier precautions are used to prevent skin, mucous membrane or percutaneous exposure to them. Counselling and testing are provided to healthcare workers with a documented needle-stick exposure. Some clinicians and facilities recommend prophylactic AZT therapy after needle-stick or splash exposure; however, it must be initiated immediately and its effectiveness has yet to be established.

FIGURE 12.11 ■ This nurse is disposing of a needle and syringe in a special container, a necessary practice to avoid the transmission of HIV through needle sticks with contaminated needles

Assessment

Collect the following data through health history and physical examination. Further focused assessments are described with nursing interventions below.

- *Health history:* risk factors (transfusion, unprotected sex, needle exposure), infections (sexually transmitted infections, hepatitis, tuberculosis), medications, recreational drug use, foreign travel, pets.
- *Physical assessment:* height, weight, nutrition, skin and mucous membranes, vision, lymph nodes, breath sounds, abdominal tenderness, motor strength, coordination, cranial nerves, gait, deep tendon reflexes, genitourinary examination, mental status. Remember that symptoms must be interpreted and reported by the person. Like pain, the presence and severity of dyspnoea are determined and reported by the person. We must believe what the person tells us. Assessment is the basis for differential diagnosis; fitting appropriate treatment to the correct aetiology is critical. For example, delirium is an acute confusional state and, unlike dementia, is reversible. There are effective nursing interventions for these conditions (Coyne et al., 2002).

Nursing diagnoses and interventions

Nursing care needs for the person with HIV infection change over the course of the disease. Preventive healthcare measures, health maintenance activities, education and support of coping mechanisms are important in the early stages of the disease. Counselling the person with a new diagnosis of HIV infection is vital. HIV infection and AIDS continue to carry a social stigma that may interfere with the person's usual support systems and coping mechanisms. As the disease progresses and the person experiences more physical symptoms, direct care needs become more important while the need for psychosocial support continues. Acute exacerbation of opportunistic infections may necessitate hospitalisation, but typically the person is managed at home. See the following 'Nursing care plan'.

Ineffective coping

On receiving the test results indicating HIV seropositive status, the person with HIV infection is faced with multiple issues rarely affecting other people. First and foremost, HIV is a disease for which there is no known cure and which is, at this time, thought to be almost universally fatal. Social support systems, family relationships, and the ability to obtain and retain useful work and health insurance may be disrupted by the disease. The person may experience guilt about their lifestyle and how the disease was contracted. As the disease progresses, social isolation, fatigue, body image changes, medication side effects and multiple other issues affect the person's abilities to cope.

- Assess social support network and usual methods of coping. *This will help both the nurse and the person identify people and mechanisms that can help the person cope more effectively with the disease.*
- If possible, assign a primary nurse, whether the setting is community-based, hospice or acute care. *This helps promote the development of a therapeutic and trusting relationship and provides for continuity of care.*
- Plan for consistent, uninterrupted time with the person. *Time and a consistent presence encourage the person to express feelings and work through issues related to HIV infection.*
- Interact at every opportunity outside of providing specific nursing care treatments. *This purposeful interaction communicates caring and acceptance without fear of HIV disease.*
- Support the person's social network. Non-traditional families may offer more support than the traditional family. *This in turn may necessitate a liberal interpretation of the term 'family' if unit policy is 'immediate family' only.*
- Promote interaction between the person, significant others and family. *Hospitalisation and manifestations of HIV disease may bring about isolation from others and decrease the person's ability to cope.*
- Encourage involvement in making care decisions. *This gives the person a greater sense of self-worth and control over the situation, increasing coping abilities.*
- Set and maintain limits on manipulative and other destructive behaviours. *The person who is unable to limit inappropriate behaviours needs the external control established by setting limits.*
- Assist to accept responsibility for actions without blaming others. *Effective coping cannot occur without accepting responsibility for one's actions.*
- Support positive coping behaviours, decisions, actions and achievements. *As self-esteem is enhanced, coping improves* (Côté & Pepler, 2005).

Impaired skin integrity

Dryness, malnutrition, immobility from fatigue and skin lesions on pressure sites contribute to impaired integrity of the skin for the person with HIV disease. Maintaining skin integrity is important because of the progressive and debilitating nature of the disease. It is also a consideration both as the first line of defence against infection in an immunosuppressed person and as a site for secondary manifestations such as KS and herpes.

- Monitor and assess the skin frequently for lesions and areas of breakdown. *Early identification of impaired skin integrity allows prompt intervention.*
- Monitor lesions for signs of infection or impaired healing. *Infection or poor tissue perfusion not only impairs healing but may lead to further skin breakdown.*
- Turn at least every 2 hours, more frequently if necessary. *Turning decreases unrelieved pressure on bony prominences and improves circulation to the tissues.*
- Use pressure-relieving devices, such as alternating air mattresses and overlays, or sheep skin pads for elbows and heels. *These devices provide prophylactic relief of pressure.*
- Keep skin clean and dry using mild, non-drying soaps or oils for cleansing. Night sweats and diarrhoea, if present, can cause breakdown and damage to the skin. *Frequent cleansing with non-drying products discourages bacterial growth, thus reducing the risk of infection.*

TRANSLATION TO PRACTICE Evidence-based practice and nurses' willingness to care for people with AIDS

As reported by the Centers for Disease Control and Prevention in the United States (2006), the number of deaths from AIDS has declined. This is believed to be the result of both the slowing of the epidemic and of improved treatment, which has lengthened the lifespan of people with AIDS. However, as treatment continues to improve survival, a key challenge will be the increasing number of people living with HIV and AIDS and the additional resources needed for services, treatment and care.

Several studies have found that some professional nurses and students are resistant to caring for people with AIDS. One study by Sherman (1996) examined relationships between moral choices and nurses' willingness to care for people with AIDS. The willingness to provide care for people with this illness involves moral choices about one's own mortality (death anxiety), spirituality and social support. In a survey of 220 Registered Nurses employed in eight hospitals in the New York metropolitan area, Sherman found that willingness to care for people with AIDS was positively correlated with spirituality and perceived social support, and negatively correlated with death anxiety. It is suggested that nurses' willingness to care for people with AIDS may be related not only to nurses' personal values and beliefs (expressed in spirituality) but also to their professional identity and role expectations.

Addressing nursing reluctance to treat people with HIV infection, nursing educators Valois et al. (2001) researched the impact of persuasive messages on nursing students' beliefs and attitudes about caring for HIV-positive people. Nursing education certainly increases knowledge and awareness of the science of HIV infection, but it may not modify attitudes or behaviours. According to the underlying theory of the research, individuals who receive evidence-based persuasive messages may develop favourable beliefs that will alter their willingness to perform a given behaviour. Three main types of beliefs were considered in this study: behavioural belief (related to the expected consequences of adopting a behaviour); normative belief (related to perceived social pressures by significant others resulting from adopting a behaviour); and control belief (related to resources or barriers that seem to facilitate or hamper adoption of the behaviour).

In three sessions, the student nurses in the experimental group were given positive persuasive messages about caring for HIV-infected people. The persuasive messages were compelling and specific to caring for people with HIV; case studies provided opportunities for the students to discuss the elements of the case within the framework of the persuasive messages. Students in the control group studied the science of caring for people with HIV but did not receive the persuasive messages. When beliefs and attitudes about caring for HIV-positive people were compared, the researchers found significantly greater willingness to provide care in the experimental group. Nursing students proved to be well prepared and motivated to receive this information.

IMPLICATIONS FOR NURSING

To increase nurses' willingness to care for people with AIDS, students need to be better socialised into their roles and responsibilities. Incorporating education about HIV/AIDS into the undergraduate curriculum; analysing and defining effective nursing care in cases based on evidence-based framework; and utilising the standards of professional nursing all support the development of positive attitudes. Discussions within the classroom and clinical settings provide a safe means of bringing fears into the open and sharing experiences. Student groups can serve as support groups, improving communications, decreasing isolation and anxiety, and improving self-esteem and morale. Within the work setting, perceived support from colleagues and administrators as well as increased contact with people with AIDS are important factors in making caring a rewarding and positive experience.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. These studies were of student nurses and Registered Nurses. What differences do you think might have been found between the two groups today?
2. Carefully consider each of the following people with AIDS and write a brief paragraph about how you would feel if you were assigned to care for them:
   a. a heterosexual female, aged 25
   b. a homosexual male, aged 35
   c. a newborn baby girl
   d. a 40-year-old single mother of three teenagers
   e. a 30-year-old homeless drug user
   f. a 17-year-old male with haemophilia, infected by blood transfusions

- Gently massage around, but not over, affected pressure sites or bony prominence to increase circulation to the surrounding tissue. *Massaging over the affected area or bony prominences can cause skin breakdown.*
- If blisters are noted, leave intact and dress with a hydrocolloid dressing or as per organisational protocol. *Blisters provide natural sterile coverings for damaged tissue, improving healing and preventing bacterial invasion.*

CONSIDERATION FOR PRACTICE

Applying protective creams to reddened areas in the rectal area protects skin from the caustic effects of diarrhoea.

- Caution against scratching. If the person is confused, trim fingernails and use mitts or soft restraints to prevent scratching. Check for circulation of hands and fingers frequently if mitts or restraints are used. *Scratching and skin damage allow bacteria to be introduced into lesions, increasing the risk of infection. Tight or restrictive restraints or mitts may compromise circulation.*
- Avoid the use of heat or occlusive dressings. *Heat can further dry and damage the skin; occlusive dressings may impair circulation and lead to ulceration.*
- Prevent skin shearing by using a slide sheet and adequate personnel when repositioning. *Shearing causes tissue trauma that can lead to decubitus ulcers.*

NURSING CARE PLAN A person with HIV infection

Sara Lu is a 26-year-old primary school teacher who lives with her parents and two younger sisters. Ms Lu is very close to her parents and sisters; they share everything with each other. During a routine medical check-up, Ms Lu tells her doctor that lately she has felt fatigued. She also states that she has had a persistent sore throat, intermittent bouts of diarrhoea and mild shortness of breath for about a month. She takes no routine medications other than a daily multivitamin and an occasional paracetamol tablet for a headache. She is active in a drama club in her community and she jogs 5 km three to four times a week. She is engaged to be married; her wedding date is 6 months away. Her fiancé is the only person with whom she has had sexual relations. Her sexual activity has been unprotected. Ms Lu has a history of open-heart surgery 7 years ago to correct a congenital valve defect. She has been physically healthy since that time, until about a month or two ago. The doctor orders a mononucleosis test, ELISA, Western blot analysis, CD4T-cell count, a p24 antigen test and an erythrocyte sedimentation rate (ESR). Ms Lu has been asked to return in 1 week for follow-up.

ASSESSMENT

On Ms Lu's follow-up visit, Carole Kee, RN, obtains her nursing history. Ms Lu continues to have flu-like symptoms but has improved somewhat. She states that she has not been as active as usual and is worried about her health. Her appetite has decreased because of soreness in her mouth, and she has noted some whitish patches on her tongue and cheeks.

A chest x-ray film reveals no abnormality. The results of her laboratory tests are as follows:

- ELISA: positive for antibodies against HIV
- Western blot analysis: positive for antibodies against HIV
- p24 antigen test: positive for circulating HIV antigens
- ESR: increased to 55 mm/h (normal for women is 15 to 20 mm/h; normal for men is 10 to 15 mm/h)
- CD4T-cell count: 499/mm^3 (normal range is 500–1350/mm^3).

Ms Lu's physical examination reveals that she has enlarged lymph nodes in her neck and white patches on her oral mucosa. Her skin is warm to the touch. Her vital signs are as follows: T 37.7°C, P 84, R 20 and BP 120/78.

Ms Lu is told of the results of her laboratory tests and the medical diagnosis of HIV infection. Ms Lu is obviously distressed and wants to know how this happened, its meaning, whether she has infected her loved ones and whether she will get better.

DIAGNOSES

- *Imbalanced nutrition* related to soreness of mouth and throat as evidenced by nutritional intake being less than body requirements.
- *Risk of deficient fluid volume* related to decreased fluid intake and diarrhoea as evidenced by fluid intake being less than body requirements.
- *Risk of infection* related to altered immune protection as evidenced by elevated ESR.
- *Risk of anxiety* and fear related to diagnosis as evidenced by distress.
- *Knowledge deficit* related to the HIV disease process as evidenced by questions asked.

PLANNING

- Plan time to educate Ms Lu on the importance of nutritionally balanced diet and maintaining adequate fluid intake.
- Identify strategies for coping with anorexia and nausea.
- Referral for dietary consultation.
- Plan time for discussion of concerns and coping strategies.

Expected outcomes

- Maintain adequate nutrition for optimal body and cellular function.
- Consume at least 2500 mL of fluid per day.
- Remain free of infections and their complications.
- Verbalise anxiety and use appropriate coping mechanisms.
- Verbalise and demonstrate knowledge of HIV disease.
- Verbalise measures to prevent HIV transmission to others, including safer sex practices.

IMPLEMENTATION

- Monitor for signs of dehydration, such as poor skin turgor, oliguria and orthostatic hypotension.
- Monitor daily weight and intake and output.
- Monitor dietary habits and serum albumin levels.
- Assess bowel sounds and monitor elimination pattern.
- Administer antiemetic and antimotility medications as ordered.
- Increase fluid to 2500 mL daily.
- Use strict aseptic technique for all invasive procedures.
- Teach Ms Lu to avoid exposure to infection and people with known illnesses.
- Monitor response to prescribed medications.
- Encourage regular physical exercise.
- Provide opportunities for Ms Lu to verbalise her feelings.
- Avoid false reassurances.
- Provide appropriate and adequate information about HIV/AIDS.
- Teach safer sex practices and other measures to prevent HIV transmission.
- Teach anxiety-controlling techniques, such as deep breathing and meditation.

EVALUATION

Ms Lu is eager to learn about her illness and wants her family to come with her for further explanation. She states that she is sure her fiancé will be available as well. Ms Lu is taking home antifungal medication, diet plans and a schedule for increased exercise. She will return in 1 week for counselling and in 1 month for a follow-up medical.

CRITICAL THINKING IN THE NURSING PROCESS

1. How does age affect the body's response to fighting HIV? What other factors affect the risk of HIV infection and its progression?
2. Are the laboratory results for Ms Lu a true indication that she is HIV positive? What additional tests might be ordered?
3. What is the most likely source of Ms Lu's infection? What measures are used to reduce this risk and how did she contract HIV? What is another possible source of Ms Lu's HIV infection?
4. Ms Lu says that her fiancé would like to have a child. How will you counsel her regarding pregnancy and childbearing?

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. What communication and education strategies could you use when caring for a person newly diagnosed with HIV?

- Encourage ambulation if possible; if the person is confined to bed, encourage active or passive range-of-motion exercises. *Activity increases circulation, decreases pressure and skin breakdown, and helps maintain muscle tone.*
- Monitor nutritional intake and albumin levels. *Maintenance of optimal nutrition decreases the risk of tissue breakdown and improves resistance to infection.*

Imbalanced nutrition: less than body requirements

Many factors associated with HIV disease, including manifestations of the disease itself, put the person at risk of altered nutrition and weight loss. Nausea and anorexia may be manifestations of the disease or the result of antiretroviral therapy. Chronic diarrhoea is a common manifestation of constitutional HIV disease. Wasting syndrome is also common. It is manifested by involuntary weight loss of greater than 10% to 15% of baseline weight, severe diarrhoea, fever, and chronic fatigue and weakness. The exact cause of wasting syndrome is unclear, but the diarrhoea and fatigue contribute, as does the increased metabolic rate associated with fever. Oral and oesophageal candidiasis and KS of the gastrointestinal tract may cause painful swallowing, making eating difficult and thereby contributing to anorexia. Poor nutritional status in the person with HIV can ultimately result in altered comfort, a change in body image, muscle wasting, increased risk of infection, and higher mortality and morbidity.

- Assess nutritional status, including weight; body mass; kilojoule intake, and laboratory studies such as total protein and albumin levels, haemoglobin and haematocrit. *These factors provide a baseline to determine the effectiveness of interventions.*
- Identify possible causes of altered nutrition. *Identification of causes provides direction for planned interventions.*
- Administer prescribed medications for candidiasis and other manifestations as ordered. Eliminating this opportunistic infection improves comfort and facilitates food intake. *Topical viscous anaesthetic can help to reduce pain and improve oral intake.*
- Administer antidiarrhoea medications after stools and antiemetics prior to meals. Provide antipyretics as needed to control fever. Reducing diarrhoea will improve nutrient absorption; preprandial medication with an antiemetic reduces nausea and improves food intake. *Reduction of fever lowers the body's metabolic demands.*
- Provide a diet high in protein and kilojoules. *A high-protein, high-kilojoule diet provides the necessary nutrients to meet metabolic and tissue healing needs.*
- Offer soft foods and serve small portions. *Soft foods are easily digested. Small portions are more appealing to the anorectic or nauseated person.*

CONSIDERATION FOR PRACTICE

High-fibre foods can increase intestinal motility and the incidence of diarrhoea.

- Involve in meal planning and encourage significant others to bring favourite foods from home. *The person is more likely to consume adequate amounts of preferred foods. Allowing food choices enhances the person's sense of control.*
- Assist with eating as needed. *Fatigue and weakness can prevent the person from eating an adequate amount of food.*
- Provide supplementary vitamins and supplements, such as Ensure. *This improves nutritional status and kilojoule intake.*
- Provide or assist with frequent oral hygiene. *Oral hygiene improves comfort and appetite and reduces the risk of mucosal lesions.*
- Administer appetite stimulants, such as megestrol or dexamethasone, as ordered. *Both drugs may increase appetite and promote weight gain.*

Ineffective sexuality patterns

The diagnosis of HIV infection can significantly alter the person's expressions of sexuality. Guilt over the diagnosis may interfere with libido. The person may be angry with a significant other or partner if that person was the probable source of infection. The person may fear spreading the disease to others via sexual relations. As the disease progresses, its manifestations can affect body image and self-esteem, impairing sexuality. Other symptoms, such as nausea, fatigue and weakness, may also interfere with libido and sexual satisfaction.

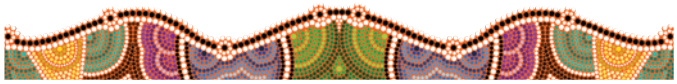
- Examine your feelings about sexuality, your role in dealing with a person's sexuality, the person's lifestyle and sexual preferences. *To deal effectively with the person's concerns, it is vital that the nurse is comfortable with their own feelings of sexuality and is able to accept the person's lifestyle. Referring the person to another nurse or counsellor may be necessary.*
- Establish a trusting, therapeutic relationship through the use of time, active listening, caring and self-disclosure. Maintain a non-threatening, non-judgmental attitude towards the person. *Sexuality is a private issue that will be uncomfortable or impossible for the nurse and person to discuss without a mutually trusting relationship.*
- Provide factual information about HIV infection and its effects. *This helps the person separate fears and myths from reality.*
- Discuss safer sex practices, including hugging, cuddling, non-sexual contact, the use of latex condoms and spermicidal lubricant, and mutual masturbation. *Alternative forms of sexual activity and expressing affection can allow the person and significant other to remain close throughout the course of the disease.*
- Encourage discussion of fears and concerns with significant other. *Open communication helps them to deal with issues related to sexuality.*
- For the person without a significant other, stress the need to continue to meet people and develop social relationships while practising safer sex. *The risk of isolation is high in the person with HIV infection, and relationships with others help the person to cope with the disease.*
- Refer the person and significant other to local support groups for people and partners of people with HIV. *Support groups provide a social and support network of people facing the same issues.*

Community-based care

Teaching needs for both the person and significant others are extensive. The primary need is information about the disease, its spread and its expected course. The person and family need current factual information to plan realistically and to combat myths, misperceptions and prejudices. At the same time, it is important to include information about current research and progress in treating the disease to maintain a sense of hopefulness.

The following topics should be discussed with the person and family to prepare for home care:

- guidelines for safer sex practices
- nutrition, rest and exercise, stress reduction, lifestyle changes and maintaining a positive outlook
- infection prevention and transmission, including handwashing and wearing gloves when handling people's secretions or excretions
- importance of regular medical follow-up and monitoring of immune status
- signs and symptoms of opportunistic infections and malignancies, as well as other symptoms that should be reported
- medications and adverse effects
- use and care of central venous access devices, total parenteral nutrition, intravenous pumps and continuous medication delivery systems, and intravenous or aerosolised medications
- cessation of smoking, alcohol and recreational or illicit drug use
- community health services
- hospice and respite care services
- community resources, such as support groups, social agencies and counsellors
- helpful resources:
  - National Association of People with AIDS
  - ACRON (www.acron.org.au)
  - Australian AIDS Information Services
  - Multicultural HIV/AIDS and Hepatitis C Service (MHAHS).



CHAPTER HIGHLIGHTS

- The immune system is a complex combination of cellular and humoral components that protect against disease. Immunity develops when the body recognises foreign proteins as 'non-self' and develops non-specific inflammatory responses and specific cellular responses to each foreign antigen.
- People suffer when the immune system is excessively or inadequately responsive or when recognition of self fails and reactions escalate against self. The latter occurs in autoimmune diseases.
- With ageing, there is a general decline in the sensitivity and regulation of the immune system, often resulting in autoimmune disease.
- Hypersensitivities are excessive responses to antigens that result in harm to the person. These range from benign to severely life-threatening. Damage to host tissue is caused by chemicals of the immune response, destruction of cells or creation of large antigen–antibody complexes that accumulate in the kidney glomerular capillaries.
- Allergic reactions are treated pharmacologically to prevent or moderate allergic responses. Another method of dampening allergic responses is by desensitisation, a weekly process of introducing increasing amounts of known allergens subdermally.
- People must be taught that the safest practice is to avoid contact with all known allergens.
- Latex allergy is a problem for healthcare professionals. Repeated exposure to latex-containing equipment and gloves results in delayed hypersensitivity.
- Any type of allergic reaction has the potential to escalate to anaphylaxis. Respiratory arrest and cardiac failure are risks with full-blown allergic reactions. Nurses must recognise early signs and symptoms and immediately signal for emergency care.
- Intentional immunosuppression is an essential step in preventing transplant rejection. The person receiving a transplanted organ will be treated with immune-suppressing drugs to prevent initial rejection, to maintain the transplant and to halt any rejection process that may develop. People will take the immune-suppressing, antirejection drugs for their lifetime. The drugs prevent cytokine production that up-regulates an immune reaction and targets the transplanted organ. Most immunosuppressing drugs are nephrotoxic; immunosuppression places people at greater risk of infection and cancers.
- HIV/AIDS continues to spread and many people are unaware they have the virus. AIDs is a profoundly immunosupressed condition that results from viral destruction of cellular components of host immunity.
- A major change in the AIDS epidemic is the disease profile, which has benefited from HAART. HAART is a combination of drugs that limits viral replication and host susceptibility to opportunistic infections and cancer. People are living much longer with the disease without progression to AIDS. 'Pill burden' refers to the number of pills the person must take daily to maintain immune function; side effects of the combination of drugs which make up HAART are appearing as people live longer. In addition to increased susceptibility to infections and cancers, people suffer from a dementia peculiar to AIDS.

CONCEPT CHECK

1 Which one of the following conditions is caused by a type I IgE-mediated hypersensitivity reaction?
   1 autoimmune haemolitic anaemia
   2 systemic lupus erythematosus
   3 graft rejection
   4 anaphylaxis

2 A person received a liver transplant 1 day ago. If the person were to develop an acute transplant rejection episode, when should the nurse expect to see the manifestations?
   1 approximately 4 days to 3 months later
   2 approximately 2 days later
   3 within the first 24 hours
   4 within the first 8 hours

3 The nurse notes a cough, shortness of breath and tachypnoea in a person with AIDS. Which opportunistic infection is probably causing these manifestations?
1 *Toxoplasma gondii*
2 Cytomegalovirus
3 *Pneumocystis carinii*
4 *Cryptococcus neoformans*

4 Which of the following explanations should the nurse give to a person who has tested positive for HIV?
1 'You have been diagnosed with AIDS.'
2 'At this point, AIDS is not active in your blood.'
3 'This means that you will not develop AIDS in the future.'
4 'Antibodies to the AIDS virus are present in the blood.'

5 People taking zidovudine should be monitored for which of the following adverse reactions?
1 cardiotoxicity
2 leucopenia
3 nephrotoxicity
4 polycythaemia

6 The order of administering antigens in allergy testing is based on prevention of anaphylaxis. Which method should be used first?
1 inhalation
2 prick test
3 intradermal injection
4 subcutaneous injection

7 If a hypersensitivity response is suspected when blood products are infusing, the nurse should:
1 discard the product immediately
2 replace all tubing and attach a new line with normal saline
3 backflush the line and run normal saline attached at the Y tubing
4 remove the intravenous catheter and establish access distal to the site

8 Protease inhibitors and nucleoside analogues share correlations to metabolic abnormalities, including:
1 lactose intolerance
2 diabetes mellitus
3 Hashimoto's thyroiditis
4 systemic lupus erythematosus

9 The priority when initiating or changing HIV drug therapy regimens is:
1 cost of therapy
2 access to dental care
3 toxicities associated with each drug
4 the person's willingness to adhere to the drug regimen

10 People receiving kidney transplants will receive immunosuppressant therapy. The agent used to induce immunosuppression immediately following a transplant is often:
1 azathioprine
2 corticosteroids
3 muromonab-CD3
4 antithymocyte globulin

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CHAPTER 13

NURSING CARE OF PEOPLE WITH CANCER

CATHERINE BETHELL

LEARNING OUTCOMES

- Define cancer and theories of carcinogenesis, and differentiate benign from malignant neoplasms.
- Explain known carcinogens and identify risk factors for cancer.
- Compare the mechanisms and characteristics of normal cells with those of malignant cells.
- Describe physical and psychological effects of cancer.
- Describe and compare laboratory and diagnostic tests for cancer.
- Discuss and compare the role of surgery, chemotherapy and classification of chemotherapeutic agents, radiation therapy and biotherapy in the treatment of cancer.
- Identify causes and discuss the nursing interventions for common oncological emergencies.
- Develop an appropriate care plan for people with cancer and their families regarding cancer diagnosis, treatment and coping strategies.

CLINICAL COMPETENCIES

- Assess functional health status of people with cancer and monitor, document and report abnormal manifestations.
- Incorporate evidence-based practice and research into the plan of nursing care for people with cancer.
- Prioritise nursing diagnosis based on assessment data and implement appropriate nursing interventions for people with cancer during cancer diagnosis, treatment and rehabilitation.
- Safely administer chemotherapeutic medications and other medications for pain, nausea and vomiting, mucositis or anaemia.
- Use the nursing process as a framework for planning and providing individualised care and integrating interprofessional care for people with cancer to meet their healthcare needs.
- Include cultural variation and diverse values in designing and implementing individualised plans of care for people with cancer.
- Design and provide individualised education to the person and family to restore, promote and maintain the person's functional status.
- Revise the plan of care as needed to provide effective interventions for people with cancer and their families.

KEY TERMS

CANCER AND THEORIES OF CARCINOGENSIS

Cancer is a group of complex diseases with various manifestations, depending on which body system is affected and the type of tumour cells involved. Cancer can affect people of any age, gender, ethnicity or geographical region. Although the incidence and mortality rates of cancer have continued to decline since 1990, it remains one of the most feared diseases. The fear engendered by even the suggestion of a cancer diagnosis often evokes feelings of hopelessness and helplessness (Galway et al., 2012).

This chapter focuses on the general pathogenesis, pathophysiology and aetiology of cancer; identifies current diagnostic and treatment modalities; and discusses nursing care appropriate for people with cancer. Discussions of cancers that affect specific body systems (e.g. leukaemia, lung cancer) can be found in corresponding body system chapters in the text.

Cancer results when normal cells mutate into abnormal, deviant cells that proliferate and spread within the body. Cancer can affect any body tissue. Nursing care of the person with cancer is holistic and comprehensive, focusing on cancer not as one disease but as a constellation of many diseases. The nurse recognises that cancer is a disruptive and life-threatening process that affects the person who has the diagnosis, their family and their significant others. Nursing interventions are based on the understanding that cancer is a chronic disease that has acute episodes, and that the person is usually treated with a combination of therapeutic modalities within the home or community setting. Equally important, the nurse recognises that caring for the person with cancer involves prevention, early detection, treatment, supportive care, education, rehabilitation, long-term follow-up and end-of-life care (Oncology Nursing Society, 2009).

Oncology is the study of cancer. The term is derived from the Greek word *oncoma* ('bulk'). Oncologists specialise in caring for people with cancer. They may be medical doctors, surgeons, radiologists, immunologists or researchers. The oncology nurse is an important and significant member of the multidisciplinary team who has received specialised training in cancer care and treatment. Oncology nurses have specialised skills and knowledge to assist the person and family with the psychosocial issues associated with cancer and terminal illness. Collaboration among healthcare professionals (e.g. surgeons, oncologists, nurses, social workers) ensures a multidisciplinary approach in implementing the most effective care and treatment for the person with cancer.

INCIDENCE AND MORTALITY

In Australia, 114 000 new cases of cancer were diagnosed in 2010 with an estimated 43 000 cancer-related deaths (Australian Institute of Health and Welfare (AIHW) and Australasian Association of Cancer Registries (AACR) 2010a). Cancer is the second leading cause of death in Australia with a mortality rate of 1 in 5 before 85 years of age, with a higher incidence rate in males. Mortality rates for different cancers vary. Lung cancer was the leading cause of all cancer deaths in both men and women in Australia, accounting for 19% of all cancer deaths in 2007, with the second most common cancer-related deaths being prostate cancer in males and breast cancer in females. During 2003 to 2007 it was identified that cancer mortality rates decrease as socioeconomic status increases; and for cervical cancer, lung cancer and cancer with an unknown primary site, the cancer mortality rates increase with the remoteness of the area in which the person resides (AIHW & AACR, 2010a; 2010b).

Due to advances in cancer prevention, early detection and treatment, more than 60% of people with cancer will survive more than 5 years following diagnosis, with increases in survival rates being identified for prostate cancer from 57% to 85%, breast cancer from 72% to 88% and bowel cancer from 49% to 62% (AIHW & AACR, 2010a). Indigenous Australians have a lower 5-year survival rate following a cancer diagnosis and a higher mortality rate (Shahid, Beckmann & Thompson, 2008). During 2003 to 2007 there were 1813 cancer-related deaths and 2291 Indigenous Australians newly diagnosed with cancer (AIHW & AACR, 2010a; 2010b). Cancer has a greater impact on Indigenous Australians, who are more likely to live in a remote area of Australia with limited access to health infrastructures, and are less likely to have an early diagnosis and receive adequate treatment, such as preventive, curative and palliative services. It has been identified that Indigenous Australians have a lower incidence of skin melanoma, lymphoma, bladder and kidney cancer; however, they have a higher incidence of cancers that have poorer prognosis but are largely preventable, such as lung, liver, cervix, lip and oropharynx cancers. The patterns of cancer are related to the higher prevalence of health risk behaviours such as smoking in conjunction with the barriers created by inadequate health systems (Cunningham et al., 2008).

For information about diversity and cancer risk and incidence, see the 'Focus on cultural diversity' box below.

FOCUS ON CULTURAL DIVERSITY Cancer in Indigenous Australians

- The incidence of cancer is lower, but mortality rates are significantly higher, for Indigenous people.
- Indigenous Australians have a higher incidence of cancers of the lung and cervix, and cancer with an unknown primary site.
- Lower incidence rates have been identified for bowel cancer, lymphoid cancer, melanoma of the skin and breast cancer in Indigenous females and prostate cancer in Indigenous males.
- Indigenous Australians have a lower 5-year post-cancer survival rate than non-Indigenous Australians.
- Mortality rate is higher in Indigenous Australians for breast cancer, lung cancer and cancer with an unknown primary site, but lower for melanoma of the skin.

Sources: AIHW and AACR (2010a). *Cancer in Australia: In brief, 2010.* Cancer Series no. 59 (Cat. no. CAN 55). Canberra: AIHW. AIHW and AACR (2010b). *Cancer in Australia: In brief, 2010.* Cancer Series no. 60 (Cat. no. CAN 56). Canberra: AIHW.

FAST FACTS

Cancer in Australia

- Breast cancer is the most commonly diagnosed cancer in women, with an incidence of 12 670 cases in 2010.
- Prostate cancer is the most commonly occurring cancer in men, with an incidence of 19 403 cases during 2010.
- The second most common cancer in males is bowel cancer (7804 cases) and the third most common cancer is melanoma of the skin (5980 cases); both of these as well as prostate cancer increased in incidence during 2010.
- The second leading cancer for females is bowel cancer (6430 cases) followed by melanoma of the skin (4362 cases); both breast cancer and melanoma of the skin increased in incidence during 2010.
- It is estimated that the number of cancers will continue to increase.
- During 2010 the five most common cancers attributed to 50% of all cancer-related deaths were lung cancer 7626 deaths, bowel cancer 4047 deaths, prostate cancer 2938 deaths, breast cancer 2706 deaths and lymphoid cancers 2552 deaths.

Sources: AIHW and AACR (2010a). *Cancer in Australia: In brief, 2010.* Cancer Series no. 59 (Cat. no. CAN 55). Canberra: AIHW. AIHW and AACR (2010b). *Cancer in Australia: In brief, 2010.* Cancer Series no. 60 (Cat. no. CAN 56). Canberra: AIHW.

Risk factors

Risk factors make an individual or a population vulnerable to a specific disease or other unhealthy outcome. Risk factors can be divided into those that are controllable and those that are not controllable. Knowledge and assessment of risk factors are especially important in counselling people and families about measures to prevent cancer.

Genetics and heredity

It is estimated that 5% to 10% of cancers may have a hereditary component. Even though the majority of people will not have an inherited form of cancer, it is important to determine which people have a genetic predisposition. The familial pattern of some breast and colon cancers has been well documented. Lung, ovarian and prostate cancers have also shown some familial relationships. Recurring patterns of cancer within a family are a risk factor for a hereditary component, but do not necessarily indicate that a specific gene or mutation is the cause. An increased rate of cancer between relatives can be due to genetics as well as shared environmental exposures, lifestyle and other non-genetic risk factors. The Human Genome Project has identified new cancer-linked genes (Futreal et al., 2001). Familial cancers generally occur during old age, whereas hereditary cancers usually happen at a younger age (Jorde, Carey & Barnshad, 2010). For most cancers, research has yet to distinguish true genetic transfer from environmental causes. Although further research is needed to identify cancers that are due to the inheritance of defective genes, familial predisposition to malignancies should be counted among risk factors so that people at risk can reduce behaviours that promote cancer. For example, a person with a family history of lung cancer should be counselled to avoid smoking, to avoid areas where smoking is allowed and to avoid working in an occupation that may expose them to inhaled carcinogens.

Age

Cancer is a disease associated with ageing; the risk of being diagnosed with cancer before the age of 75 years is 1:3 and before 85 years is 1:2 (AIHW & AACR, 2010a). A number of factors are associated with this increased risk in older adults. One possible factor is that at least five cycles of genetic mutations seem necessary to cause permanent damage to the afflicted cells. In addition, long-term exposure to high doses of promotional agents is usually necessary to allow the cancer to take hold. Also, the immune response alters with ageing, its actions becoming more generalised and less specific (Blaylock, 1998). Another problem is that free radicals (molecules resulting from the body's metabolic and oxidative processes) tend to accumulate in the cells over time, causing damage and mutation.

Hormonal changes that occur with ageing can be associated with cancer. Postmenopausal women receiving exogenous oestrogen have an increased risk of breast and uterine cancers. Older men are at risk of prostate cancer, possibly due to breakdown of testosterone into carcinogenic forms. See the 'Nursing care of the older adult' box for a discussion about older adults and cancer.

Severe and/or cumulative losses also are implicated in promoting cancer (Gregorio et al., 2012; Hasselbalch, 2013). These losses, which are common to older adults, include the death of a spouse or friends, loss of position and status in society and a decline in physical abilities. These repeated stressors are related to changes in the immune system that may lead to the development of cancer.

Gender

Gender is a risk factor for certain types of cancer. Breast cancer is the most frequently diagnosed cancer in women and prostate cancer in men. The incidence of laryngeal cancer is three times higher in men than in women (AIHW & AACR, 2010a; 2010b). Cancer of the peritoneum and the thyroid occurs more commonly among females (AIHW & AACR, 2010a; 2010b). See Chapters 47 and 48 for more information on gender-specific cancers.

Poverty

The poor are at a higher risk of cancer than the population in general. Inadequate access to healthcare, especially preventive screening and counselling, may be a major factor. Other factors that may be involved, such as diet and stress, usually come under the category of controllable risks; however, these risks are frequently uncontrollable in this population.

Stress

Continuous unmanaged stress that keeps hormones such as adrenaline (epinephrine) and cortisol at high levels can result in systematic 'fatigue' and impaired immunological surveillance. When the body attempts to adapt to physiological and psychological stressors, it goes through a series of stages called

NURSING CARE OF THE OLDER ADULT Older adults with cancer

Nurses need to be aware of how cancer and cancer treatments affect older adults. Cancer is the fourth leading cause of death in people over 75. The incidence of cancer increases with advancing age, probably as a result of the accumulated exposure to carcinogens and to age-related declines in the action of the immune system. The most commonly seen cancers in women are breast, colorectal and lung cancers, and melanoma of the skin. In men, prostate, lung and colorectal cancers, and melanoma of the skin, occur most frequently.

The importance of screening and early detection of cancer does not diminish with age. Unfortunately, older adults may be less likely to undergo cancer screening or seek treatment for cancer due to fear, depression, cognitive impairments, poor access to healthcare or financial constraints. Some older adults (and healthcare providers) mistake cancer symptoms for normal age-related changes. Believing that little can be done, they do not seek healthcare for their symptoms. Fear of the cancer diagnosis also keeps older adults from seeking appropriate healthcare. When they do seek treatment, chronic conditions frequently seen in older adults may make the diagnosis of cancer more difficult by masking or confounding the usual symptoms associated with cancer.

Older adults are at greater risk of side effects associated with cancer treatment because of age-related physiological changes and chronic conditions associated with ageing. This is particularly true for the side effects of chemotherapeutic agents. The incidence of toxic effects on the heart and central nervous system is increased. The side effects of chemotherapy can contribute to fatigue and cause problems related to immobility and functional decline. Alterations in the function of the immune system are also more frequent in older adults, which increase their risk of developing infection.

The problems associated with chemotherapy do not rule out its use, but the nurse must be aware of potential problems and monitor the person closely for the development of side effects. The nurse needs to consider the effect of ageing on responses to the disease and its treatment.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Discuss signs and symptoms of cancer.
- Stress the importance of seeking healthcare if any of the warning signs develop.
- Stress the importance of having an annual physical examination.
- For women, teach how to perform a monthly breast self-exam (BSE) and emphasise the importance of continuing BSE and regular mammography, available free from age 40 onwards.
- Teach men the early signs of prostate cancer and encourage them to have an annual digital rectal exam.

the general adaptation syndrome (Gregorio et al., 2012; Hasselbalch, 2013). First, the 'alarm reaction' occurs, in which adrenal hormones increase allowing the body to cope with the stressor. Eventually the body reaches the 'stage of resistance', in which the stress hormones are significantly reduced, indicating that adaptation has occurred. If the physiological adaptation is supported by appropriate coping strategies, the stressor is considered managed and the body systems return to pre-alarm functioning. However, if adaptation continues and the stress hormones remain elevated, the 'stage of exhaustion' sets in. This stage will maintain life but at great expense to body systems, resulting in general wear and tear and depression of the immune system (Antoni et al., 2012).

Diet

Some foods are considered genotoxic, such as the nitrosamines and nitrous indoles found in preserved meats and pickled, salted foods. Other foods, such as high-fat, low-fibre foods—the mainstay of many Australian diets—promote colon, breast and sex-hormone-dependent tumours. When fish and meat are excessively fried or grilled, potent carcinogenic compounds can form that may cause tumours in the mammary glands, colon, liver, pancreas and bladder. Also, repeatedly using fat to fry foods at high temperatures produces high levels of polycyclic hydrocarbons, which increase cancer risk considerably. Although many people profess to have changed their dietary habits, one only has to observe the large number of people who still lunch on hamburgers and chips (and teach their children to do the same) to realise that much more educational and motivational work is needed in this area. Other food-related substances believed to increase cancer risk include sodium saccharine, red food dyes and both regular and decaffeinated coffee.

Occupation

Occupational risk might be considered to be either controllable or uncontrollable. For many people, both education and ability limit their choice of occupation, particularly during times of high unemployment. Moreover, changing one's occupation because it poses risk factors may not be a viable option. Federal standards are designed to protect workers from hazardous substances, but many believe that these standards are not strict enough and that inspections are not frequent enough to prevent violations.

Specific risks vary according to the occupation. For example, outdoor workers such as farmers and construction workers are exposed to solar radiation; healthcare workers such as x-ray technicians and biomedical researchers are exposed to ionising radiation and carcinogenic substances; and exposure to asbestos is a problem for people who work in old buildings with asbestos insulation in the walls. Table 13.1 correlates known carcinogens and occupations.

Infection

As a number of viruses have been linked to some cancers, avoiding those specific infections will decrease risk. Although some infections may be unavoidable (e.g. Epstein-Barr) others, such as genital herpes and papillomavirus-induced genital warts, can

TABLE 13.1 Chemical carcinogens and relationship to occupation

CHEMICAL AGENT	ACTION	OCCUPATION AFFECTED
Polycyclic hydrocarbons (smoke, soot, tobacco, smoked foods)	Genotoxic	Miners, coal/gas workers, migrant workers
Benzopyrene		
Arsenic	Genotoxic	Pesticide manufacturers, mining
Vinyl chloride polymers	Promotional	Plastics workers
		Artists
Methylaminobenzine	Genotoxic	Fabric workers
		Rubber and glue workers
Asbestos	Promotional	Construction workers, workers in old, run-down buildings with asbestos insulation, insulation makers
Wood and leather dust	Promotional	Woodworkers, carpenters, leather toolers
Chemotherapy drugs	Genotoxic	Drug manufacturers, pharmacists, nurses

often be avoided by following safer sex practices (e.g. using condoms) or obtaining the human papillomavirus (HPV) vaccine. The HPV vaccine is currently given to 12–13-year-old females and males aged 14–15 years were able to receive it through a catch-up program as part of the Australian National Immunisation program (Department of Health and Ageing, 2012).

Tobacco use

Lung cancer is considered highly preventable because of its relationship to smoking. The genotoxic carcinogenic substances in tobacco are considered weak; therefore, stopping smoking can reverse the damage it causes. However, many other substances in tobacco are highly promotional, so that the larger the dose and longer the use, the higher the risk of developing cancer. Research has shown a significantly lower lung cancer death risk of former smokers compared to current smokers. Smokers who quit before middle age avoid more than 90% of the risk of lung cancer that can be attributed to tobacco.

Tobacco is also related to other forms of cancer. Smokers face an increased risk of oropharyngeal, oesophageal, laryngeal, gastric, pancreatic and bladder cancers. Pipe and cigar smokers are especially susceptible to oropharyngeal and laryngeal cancers. Oral and oesophageal cancers are more common among those who chew tobacco or use snuff. Smokers who have a genetic decrease in $alpha_1$-antitrypsin (an enzyme that protects lung tissue) that results in emphysema face an even higher cancer risk than smokers without this defect.

Additional research has documented the deleterious effects of second-hand tobacco smoke (Dietrich et al., 2002). Tobacco-specific nitrosamines were recovered in the urine of children living with smokers. It is now accepted that non-smokers exposed to tobacco smoke over long periods of time, whether in the workplace or the home, have an increased risk of lung or bladder cancers.

Alcohol use

Alcohol promotes cancer by enhancing the contact between carcinogens, such as those in tobacco, and the stem cells that line the oral cavity, larynx and oesophagus (Porth & Matfin, 2009). People who smoke and drink a considerable amount of alcohol daily have an increased risk of oral, oesophageal and laryngeal cancers.

Recreational drug use

Recreational drug use often promotes an unhealthy lifestyle that increases general cancer risk; for example, drug users often do not maintain adequate nutrition. Furthermore, recreational drugs are implicated as promoters because of their suppressive effect on the immune system. Although it has not been directly implicated in cancer development, marijuana has been demonstrated to cause chromosomal damage that may over time also result in cancer-causing DNA damage and genetic mutations. Marijuana smoke is also much more injurious to lung tissue than tobacco smoke.

Obesity

Excessive body fat has been linked to an increased risk of hormone-dependent cancers. Because sex hormones are synthesised from fat, obese people often have excessive amounts of the hormones that feed hormone-dependent malignancies of the breast, bowel, ovary, endometrium and prostate.

Sun exposure

As the protective ozone layer thins, more of the sun's damaging ultraviolet radiation reaches the earth. As a consequence, the rate of skin cancers has increased. Australia has one of the highest rates of skin cancer in the world, with the majority of skin cancers being caused by exposure to UV radiation in sunlight. Around 95% to 99% of skin cancers in Australia can be attributed to sun exposure (Cancer Council Australia (CCA), 2007). Sun-related skin cancers are considered to be a problem for all people, regardless of skin colour, but people with very fair skin, blue or green eyes, and light-coloured hair are most vulnerable. Older people with decreased pigment are also more at risk, even those with darker skin.

Figure 13.1 summarises the interaction of factors that promote cancer.

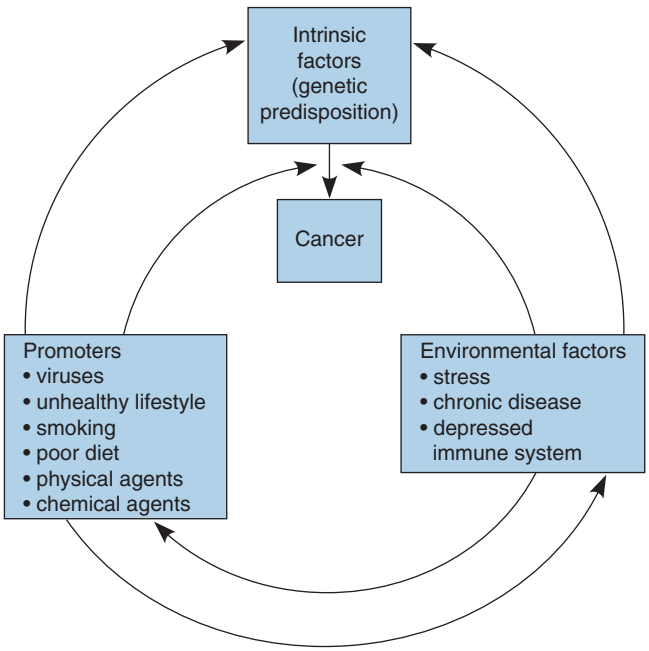


FIGURE 13.1 ■ Interaction of factors that promote cancer. Most people have immune systems that are competent enough to resist the establishment of cancer from an initiated cell. Cancer takes hold when a number of promotional factors occur together and over enough time to weaken immune resistance. Like factors are grouped together for ease of presentation but may occur in any combination

PATHOPHYSIOLOGY

Cancer is a complex disease with hundreds of agents that can contribute to its pathogenesis. Advances in research have greatly increased the understanding of how cancer develops. It is now known that the development of cancer is a process in which normal cells are changed and acquire malignant properties. Before moving on to discuss the various theories of the causes of cancer, it is useful to review how normal cells divide and adapt to changing conditions.

Normal cell growth

Mature normal cells are uniform in size and have nuclei that are characteristic of the tissue to which the cells belong. Within the nucleus of normal cells, chromosomes containing deoxyribonucleic acid (DNA) molecules carry the genetic information that controls the synthesis of polypeptides (proteins). Genes are subunits of chromosomes and consist of portions of DNA that specify the production of particular sets of proteins. Thus, genes control the development of specific traits. The genetic code in the DNA of every gene is translated into protein structures that determine the type, maturity and function of a cell. Any change or disruption in a gene can result in an inaccurate 'blueprint' that can produce an aberrant cell, which may then become cancerous. Box 13.1 lists some of the functions of DNA.

BOX 13.1 Functions of DNA

- Orders production of enzymes
- Instructs cells to produce specific chemicals
- Instructs cells to develop specific structures
- Determines individual traits and characteristics
- Controls other DNA by telling a cell to 'switch on' and use some portion of the genetic information stored in it

The cell cycle

Two coordinated events are responsible for cellular reproduction. Reproduction occurs as the result of replication of cellular DNA and mitosis, when the cell divides into two daughter cells with identical DNA.

The **cell cycle** consists of four phases. In the gap 1 or G_1 phase, the cell enlarges and synthesises proteins to prepare for DNA replication. During this phase the cell prepares to replicate and enter into the synthesis phase. During the synthesis (S) phase, DNA is replicated and the chromosomes in the cell are duplicated. During the next phase, gap 2 (G_2), the cell prepares itself for mitosis. Finally, with all preparation complete, the cell begins mitosis (M). This phase culminates in the division of the parent cell into two exact copies called daughter cells, each having identical genetic material. The cells then immediately enter G_1 where they begin the cell cycle again or divert into a resting phase called G_0. The cell cycle is controlled by cyclins, which combine with and activate enzymes called cyclin-dependent kinases. Some cyclins cause a 'braking' action and prevent the cycle from proceeding (Dunlop & Campbell, 2000). Checkpoints in the cell cycle ensure that it proceeds in the correct order.

A malfunction of any of these regulators of cell growth and division can result in the rapid proliferation of immature cells. In some cases, these cells are considered cancerous (malignant). Knowledge of cell cycle events is used in the development of chemotherapeutic drugs, which are designed to disrupt the cancer cells during different stages of their cell cycle. These drugs and their use are discussed later in the chapter.

Differentiation

Differentiation is a normal process occurring over many cell cycles that allows cells to specialise in certain tasks. For example, some epithelial cells lining the lungs develop into tall columnar cells with cilia. These columnar cells sweep potentially dangerous debris out of the lungs. When adverse conditions occur in body tissues during differentiation, protective adaptations can produce alterations in cells. Some of these alterations are helpful, but in other cases the cells mutate beyond usefulness and become liabilities (Porth & Matfin, 2009). Following are potentially unproductive cellular alterations that occur during cell differentiation:

- **Hyperplasia** is an increase in the number or density of normal cells. Hyperplasia occurs in response to stress, increased metabolic demands or elevated levels of

hormones. Examples include the hyperplasia of myocardial cells in response to a prolonged increase in the body's demand for oxygen and hyperplasia of uterine cells in response to rising levels of oestrogen during pregnancy. Hyperplastic cells are under normal DNA control.

- **Metaplasia** is a change in the normal pattern of differentiation such that dividing cells differentiate into cell types not normally found in that location in the body. The metaplastic cell is normal for its particular type, but it is not in its normal location. Some metaplastic cells are less functional than the cells they replace. Metaplasia is a protective response to adverse conditions. Metaplastic cells are under normal DNA control and are reversible when the stressor or other disruptive condition ceases.
- **Dysplasia** represents a loss of DNA control over differentiation occurring in response to adverse conditions. Dysplastic cells show abnormal variation in size, shape and appearance, as well as a disturbance in their usual arrangement. Examples of dysplasia include changes in the cervix in response to continued irritation, such as from the human papillomavirus or leucoplakia on oral mucous membranes in response to chronic irritation from smoking.
- **Anaplasia** is the regression of a cell to an immature or undifferentiated cell type. Anaplastic cell division is no longer under DNA control. Anaplasia usually occurs when a damaging or transforming event takes place inside the dividing, still undifferentiated cell, leading to loss of useful function. Anaplasia may occur in response to overwhelmingly destructive conditions inside the cell or in surrounding tissue (Porth & Matfin, 2009).

Although hyperplasia, metaplasia and dysplasia often reverse after the irritating factor is eliminated, they can lead to malignancy under certain conditions. This is especially true of dysplasia, which represents a loss of DNA control. Anaplasia is not reversible, but the degree of anaplasia determines the potential risk of cancer.

Theories of carcinogensis

Factors that cause cancer are both external (chemicals, radiation and viruses) and internal (hormones, immune conditions and inherited mutations). Causal factors may act together or in sequence to initiate or promote **carcinogenesis**. Ten or more years often pass between exposures or mutations and detectable cancer.

Central to these theories are two important concepts about the aetiology of cancer. First, damaged DNA, whether inherited or from external sources, sets up the necessary initial step for cancer to occur. Second, impairment of the human immune system, from whatever cause, lessens its ability to destroy abnormal cells.

Cellular mutation

The theory of cellular mutation suggests that carcinogens cause mutations in cellular DNA. It is believed that the carcinogenic process has three stages: initiation, promotion and progression. The initiation stage involves permanent damage in the cellular DNA as a result of exposure to a carcinogen (e.g. radiation, chemicals) that was not repaired or had a defective repair. Promotion may last for years and includes conditions, such as smoking or alcohol use, that act repeatedly on the already affected cells. In the progression stage, further inherited changes acquired during the cell replication develop into a cancer.

Oncogenes

Oncogenes are abnormal genes that promote cell proliferation and are capable of triggering cancerous characteristics. Oncogenes can be classified according to their overall function. Several oncogenes and their relationship to human cancers have been identified. For example, BRCA-1 and BRCA-2 are associated with breast cancer (Riley et al., 2012).

A decrease in the body's immune surveillance may allow the expression of oncogenes; this can occur during times of stress or in response to certain carcinogens. For example, people with AIDS, who have a decreased number of helper T lymphocytes, have a much higher than normal incidence of certain cancers, including non-Hodgkin's lymphoma and Kaposi's sarcoma (Petoumenos et al., 2013).

Tumour suppressor genes

Tumour suppressor genes normally suppress oncogenes. They can become inactive by deletion or mutation. Inherited cancers have been associated with tumour suppressor genes. An example is *p53*, a suppressor gene that has been associated with sarcoma and cancer of the breast and brain.

Known carcinogens

A number of agents are known to cause cancer, or at least are strongly linked to certain kinds of cancers. These known carcinogens include viruses, drugs, hormones and chemical and physical agents.

Carcinogens can be categorised into two groups: genotoxic carcinogens directly alter DNA and cause mutations, and promoter substances cause other adverse biological effects, such as cytotoxicity, hormonal imbalances, altered immunity or chronic tissue damage. Promoter substances do not cause cancer in the absence of previous cell damage (initiation) and often require high-level and long-term contact with the altered cells (see Table 13.1). Although everyone comes into contact with a vast number of substances that are considered carcinogenic, not everyone develops cancer. Other factors, such as genetic predisposition, impairment of the immune response and repeated exposure to the carcinogen, are necessary for a cancer to develop.

Viruses

Several viruses have been associated with the development of cancer. They damage cells and induce hyperplastic cell growth. Viral infection may play a role in cell mutation that can progress to malignant cells. Most people are able to suppress this progression (Spitalnick & diSant'Angnese, 2001). Box 13.2 identifies these viruses and the cancers with which they are associated.

In addition, viruses play a significant role in weakening immunological defences against neoplasms. For example,

BOX 13.2 Cancers associated with different viruses

Herpes simplex virus types I and II (HSV-1 and HSV-2)
- Carcinoma of the lip
- Cervical carcinoma
- Kaposi's sarcoma

Human cytomegalovirus (HCMV)
- Kaposi's sarcoma
- Prostate cancer

Epstein-Barr virus (EBV)
- Burkitt's lymphoma

Human herpesvirus-6 (HHV-6)
- Lymphoma

Hepatitis B virus (HBV)
- Primary hepatocellular cancer

Papillomavirus
- Malignant melanoma
- Cervical, penile and laryngeal cancers

Human T-lymphotropic viruses (HTLV)
- Adult T-cell leukaemia and lymphoma
- T-cell variant of hairy-cell leukaemia
- Kaposi's sarcoma

human immunodeficiency virus (HIV), which infects helper T lymphocytes and monocytes, impairs the person's protection against certain cancers such as lymphoma and Kaposi's sarcoma (Petoumenos et al., 2013).

Other viruses have also been associated with human malignancies. Hepatitis B virus integrates its DNA with liver cell DNA and is believed to cause primary hepatocellular carcinoma. Papillomaviruses cause plantar, common and flat warts, which are benign and usually regress spontaneously; however, they also cause genital warts and laryngeal papillomas, which are associated with malignant melanoma and cervical, penile and laryngeal cancers. Retroviruses have been found to cause cancer in animals. Adult T-cell leukaemia is the only human cancer known to be associated with a retrovirus (Hill, 2001).

Vaccines to prevent virus-induced cancers are being investigated. Preliminary results of the use of vaccines to treat malignancies, such as melanoma, have been encouraging (Berd, 2001).

Drugs and hormones

Certain drugs can be either genotoxic or promotional. For example, chemotherapeutic drugs used to disrupt the cell cycle of malignant cells can be genotoxic for normal cells. They can also be promotional: by drastically reducing the number of leucocytes, they impair immune function. Examples of these chemotherapeutic drugs include busulfan, chlorambucil and cyclophosphamide. Some recreational drugs also are implicated as carcinogens. These include the genotoxic betel nut chewed by many Pacific Islanders and the immunosuppressant promoters heroin and cocaine.

Hormones are also potential genotoxic carcinogens or promoters. Gonadotropic hormones often mediate cancers of the reproductive organs. Oestrogen, both natural and synthetic, and diethylstilbestrol (DES) have been linked to cervical, endometrial and breast cancers. Oestrogen-containing contraceptive pills have been implicated in breast cancer, but have also been shown to decrease the risk of ovarian cancer. Investigators have not reached a final conclusion about the cancer risk posed by contraceptives. Newer research suggests that alterations in the molecular structure of testosterone in older men may promote the development of prostate cancer. Also, glucocorticosteroids (cortisone) and anabolic steroids may act as promoters by altering the immune response or endocrine balance.

Chemical agents

Many chemicals have been demonstrated to be both genotoxic and promotional. As many of these substances are encountered in the workplace, they constitute occupational hazards. Examples of industrial and environmental carcinogens include polycyclic hydrocarbons, found in soot; benzopyrene, found in cigarette smoke; and arsenic, found in pesticides. These chemicals have some genotoxic action, with some altering DNA replication. Other industrial and environmental chemicals are considered promotional agents. These include wood and leather dust, polymer esters (used in plastics and paints), carbon tetrachloride, asbestos and phenol.

Natural substances in the body may also be carcinogenic or promotional. For example, end products of metabolism that are produced in excess amounts or are ineffectively eliminated, such as bile acids from a high-fat diet, may promote cancer.

Some foods contain carcinogens added during preparation or preservation. Examples include the sugar substitute sodium saccharine, and nitrosamines and nitrous indoles, which are found in pickled, salted foods. In some cases, food contaminants produce carcinogenic chemicals. The *Aspergillus* fungi produce aflatoxin, a highly potent carcinogen. These organisms grow on improperly stored vegetable products, such as grains and peanuts.

Polycyclic aromatic hydrocarbons, nitrosamines, phenols and other chemicals in tobacco act as either carcinogens or promoters of cancer (see Table 13.1).

Physical agents

It has been well documented that excessive exposure to radiation causes increased rates of cancer by damaging the DNA in cells, by activating other oncogenetic factors or by suppressing antitumour activity (protein inhibitors). Both solar radiation from ultraviolet rays and ionising radiation from industrial or medical sources are carcinogenic. This fact has implications for workers exposed to these agents and for the population in general. Radon, a naturally formed radioactive gas that can be found in the basements of some buildings, is also a known carcinogen. People who have lived in areas where nuclear weapons have been tested or whose groundwater has been polluted by nuclear wastes are at risk of developing cancers. The effects of high-dose radiation exposure and subsequent cancer

development have been demonstrated in the survivors of the atomic bombs at Nagasaki and Hiroshima and in workers exposed to radiation during the clean up of nuclear disaster sites, such as Chernobyl.

Types of neoplasms

A **neoplasm** is a mass of new tissue (a collection of cells) that grows independently of its surrounding structures and has no physiological purpose. The term *neoplasm* is often used interchangeably with *tumour*, from the Latin word meaning 'swelling'. Neoplasms are said to be autonomous because they grow at a rate uncoordinated with the needs of the body, they share some of the properties of the parent cells but with altered size and shape and they do not benefit the host and in some cases are actively harmful.

Neoplasms are not completely autonomous, as they require a blood supply with nutrients and oxygen to sustain their growth. Neoplasms are typically classified as benign or malignant on the basis of their potential to damage the body and on their growth characteristics.

Benign neoplasms

Benign neoplasms are localised growths. They form a solid mass, have well-defined borders and are frequently encapsulated. Benign neoplasms tend to respond to the body's homeostatic controls. Thus, they often stop growing when they reach the boundaries of another tissue (a process called *contact inhibition*). They grow slowly and often remain stable in size. Since they are usually encapsulated, benign neoplasms are often easily removed and tend not to recur.

Although typically harmless, benign neoplasms nevertheless can be destructive if they crowd surrounding tissue and obstruct the function of organs. For example, a benign meningioma (from the meninges of the brain and spinal cord) can cause severely increased intracranial pressure (ICP), which progressively impairs the person's cerebral function. Unless the meningioma can be successfully removed, the steadily rising ICP will eventually lead to coma and death.

Malignant neoplasms

In contrast to benign neoplasms, malignant neoplasms grow aggressively and do not respond to the body's homeostatic controls. Malignant neoplasms are not cohesive and present with an irregular shape. Instead of slowly crowding other tissues, malignant neoplasms cut through surrounding tissues, causing bleeding, inflammation and necrosis (tissue death) as they grow. This invasive quality of malignant neoplasms is reflected in the word origin of *cancer*, from the Greek *karkinos*, meaning 'crab'. Healthcare professionals are referring to a malignant neoplasm when they use the term *cancer*.

Malignant cells from the primary tumour may travel through the blood or lymph to invade other tissues and organs of the body and form a secondary tumour called a *metastasis*. This term also refers to the process by which such spreading of malignant neoplasms—perhaps their most destructive trait—occurs. Malignant neoplasms can recur after surgical removal of the primary and secondary tumours and after other treatments. Table 13.2 compares benign and malignant neoplasms.

TABLE 13.2 Comparison of benign and malignant neoplasms

BENIGN	MALIGNANT
Local	Invasive
Cohesive	Non-cohesive
Well-defined borders	Does not stop at tissue border
Pushes other tissues out of the way	Invades and destroys surrounding tissues
Slow growth	Rapid growth
Encapsulated	Metastasises to distant sites
Easily removed	Not always easy to remove
Does not recur	Can recur

Malignant neoplasms vary in their degree of differentiation from parent tissue. Highly differentiated cancer cells try to mimic the specialised function of the parent tissue, but undifferentiated cancers, consisting of immature cells, have almost no resemblance to the parent tissue and therefore no useful function. To make matters worse, undifferentiated cancers rob the body of its energy and nutrition as they grow. Undifferentiated anaplastic cells have little structural or functional relationship to the parent cells and are the basis of many malignant neoplasms. The degree of differentiation of anaplastic cells is a consideration in the classification and staging of neoplasms, discussed later in this chapter.

Characteristics of malignant cells

Malignant neoplasms may be identified by the following predictable cellular characteristics:

- *Loss of regulation of the rate of mitosis*. This results in rapid cell division and growth of the neoplasm.
- *Loss of specialisation and differentiation*. Malignant cells do not perform typical cellular functions. Many produce hormones and enzymes similar to those of the parent tissue but usually in excessive amounts, possibly revealing their presence.
- *Loss of contact inhibition*. Malignant cells do not respect other cellular boundaries. They easily invade and destroy other tissues.
- *Progressive acquisition of a cancerous phenotype*. Cellular mutation seems to be a sequential process involving successive generations of cells, with each generation becoming more deviant than the previous one. Additionally, malignant cells seem to be 'immortal' in that they do not stop growing and die, as do normal cells, which have a genetically determined lifespan.
- *Irreversibility*. The transformation into a malignant cell is irreversible. Rarely does a malignant neoplasm revert to a benign state.
- *Altered cell structure*. Cytological examination of malignant cells reveals distinct differences in the cell nucleus and cytoplasm, as well as an overall cell shape that differs from that of normal cells of the particular tissue type.
- *Simplified metabolic activities*. The work of malignant cells is simpler than that of normal cells. They show an increased

synthesis of substances needed for cell division and they have no need to create proteins for the specialised functions of the tissues they invade.

- *Transplantability*. Malignant cells often break away from the primary tissue site and travel to other locations in the body, where they establish new growths.
- *Ability to promote their own survival*. Malignant cells may create ectopic sites to produce the hormones they need for their growth. By their very presence and their ability to initiate vascular permeability, malignant cells promote the development of non-neoplastic stroma, a connective tissue framework consisting of collagen and other components, which then supports the neoplasm. They may also create their own blood supply. Through a process called angiogenesis, tumour cells secrete a polypeptide angiogenic growth factor that stimulates blood vessels from surrounding normal tissue to grow into the tumour. Finally, malignant cells divert nutrition from the host to meet their own needs, by diffusion when the tumour is less than 1 mm and thereafter by means of the newly formed blood vessels. If unchecked, malignant cells eventually destroy their host.

The characteristics of malignant cells are summarised in Box 13.3.

BOX 13.3 Characteristics of malignant cells

- Loss of regulation of mitotic rate
- Loss of cell specialisation
- Loss of contact inhibition
- Progressive acquisition of a cancerous phenotype and immortality
- Irreversibility of cancerous phenotype to greater aggressiveness
- Altered cell structure: differences in cell nucleus and cytoplasm
- Simplified metabolic activity
- Transplantability (metastasis)
- Ability to promote own survival

Tumour invasion and metastasis

The ability of cancer cells to invade adjacent tissues and travel to distant organs is considered their most ominous characteristic. This quality makes treatment a considerable challenge.

Invasion

Aggressive tumours possess several qualities that facilitate invasion (see Figure 13.2):

- *Ability to cause pressure atrophy*. The pressure of a growing tumour can cause atrophy and necrosis of adjacent tissues. The malignancy then moves into the vacated space.
- *Ability to disrupt the basement membrane of normal cells*. Many cancer cells can bind to elements of the basement membrane and secrete enzymes that degrade that physical barrier, thus facilitating their movement into normal tissues, lymph and blood circulation.
- *Motility*. Because malignant cells are less tightly bound to each other than normal cells (reduced adhesiveness), they easily separate from the neoplasm and move into surrounding body fluids and tissues.
- *Response to chemical signals from adjacent tissues*. Chemotaxis (the movement of cells in response to a chemical stimulus) calls the tumour cells into the normal

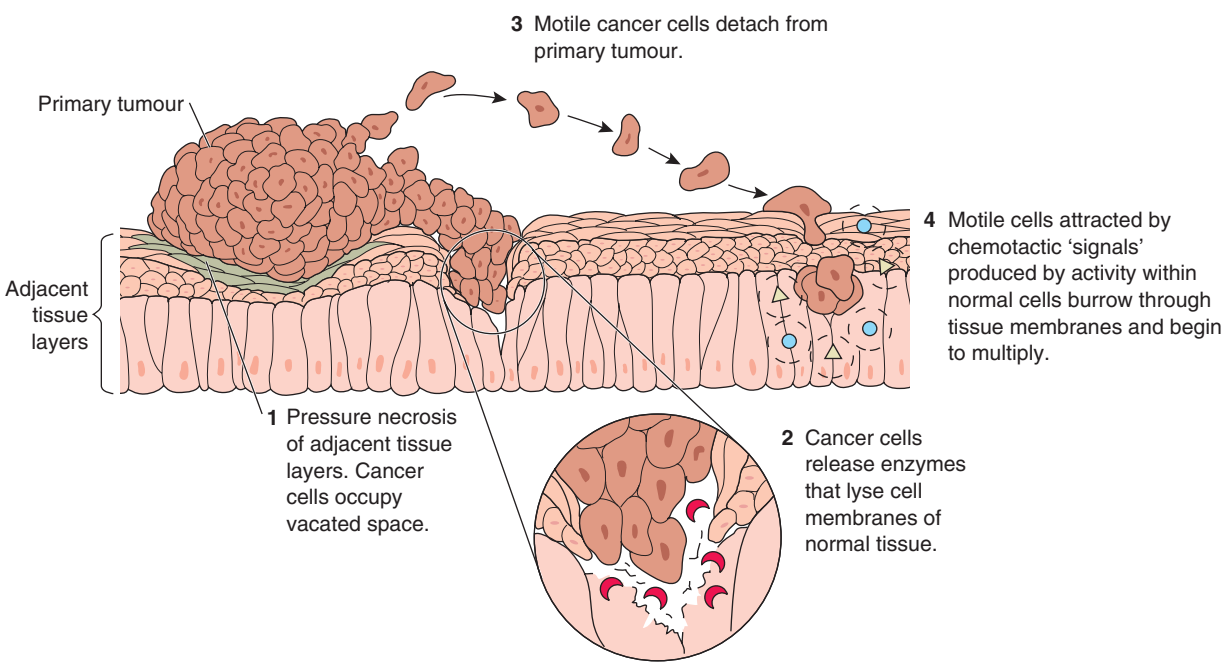


FIGURE 13.2 ■ How cancer cells invade normal tissue

tissues, possibly as a result of the degrading of the basement membranes of the normal cells. This breakdown of normal cellular membranes releases the chemical stimulus physiologically designed to draw normal phagocytic cells to clean up the debris. (See Chapter 11, on the inflammatory response, for more information on chemotaxis.) Malignant cells are also known to respond chemotactically to the end product of cellular metabolism. Some cancer cells even produce a substance called autocrine motility factor, which calls other malignant cells to a normal tissue. The first invading cells produce this substance, which then actively draws other malignant cells from the primary tumour into the invaded normal tissue.

Metastasis

The factors that favour invasion also contribute to the process of metastasis. **Metastasis** can occur by means of one or more mechanisms including embolism in the blood or lymph or spread by way of body cavities.

A blood- or lymph-borne metastasis allows a new tumour to be established in a distant organ. Figure 13.3 shows metastasis through the bloodstream. A tumour's ability to metastasise in this manner requires the following steps:

1. Intravasation of malignant cells through blood or lymphatic vessel walls and into the circulation.
2. Survival of the malignant cells in the blood. (To survive, the cells must escape the notice of the body's immune surveillance; only about 1 in 1000 cells does so.)
3. Extravasation from the circulation and implantation in a new tissue.

The tumour cells tend to clump together, forming an embolus, and continue growing until their size prevents further travel in the vessel or lymph channel. The growing neoplastic mass then uses its invasive abilities (secreting enzymes and motility factor) to move into the nearest organ.

About 60% of metastatic lesions tend to occur in a schema reflecting the pattern of blood or lymph circulation. However, it has been demonstrated that some malignant cells defy a blood-borne pattern and actually target specific organs to which they prefer to metastasise. For example, lung cancer frequently metastasises to the adrenal glands, and breast cancer frequently metastasises to bone. Malignant cells that gain access to the lymph channels may travel to a preferred organ and then move into it the same way they emigrate through blood vessels. Alternatively, the malignant cells may become trapped in the lymph node and continue to grow. Eventually, the malignant cells replace the node's tissues. At this point, emboli from the cancerous node disseminate to other nodes, creating a cascade reaction. The malignant cascade causes widespread transfer of the tumour to uncharacteristic sites.

A malignant tumour may break through the walls of the organ in which it is primarily housed, shedding cells into the nearby body cavity. The cells are then free to establish new tumours in a distant area of that cavity. For example, malignant cells from a colon cancer may be seeded into the peritoneal cavity, establishing a new tumour in the mesenteric epithelium.

Metastatic lesions are differentiated from primary neoplasms by cell morphology: metastatic cells do not resemble the tissue in which they reside. The most common sites of metastasis are the lymph nodes, liver, lungs, bones and brain. Table 13.3 lists different cancers and common sites of metastasis.

For metastasis to occur, the cancerous cells must avoid detection by the immune system. Thus, impairment of the immune system is a major factor in the establishment of metastatic lesions. Cells may escape detection in several different ways:

- Aggressive cancer cells may compile a large mass (greater than 1 cm) so rapidly that the immune system is unable to overcome the tumour before it takes hold in a new tissue.

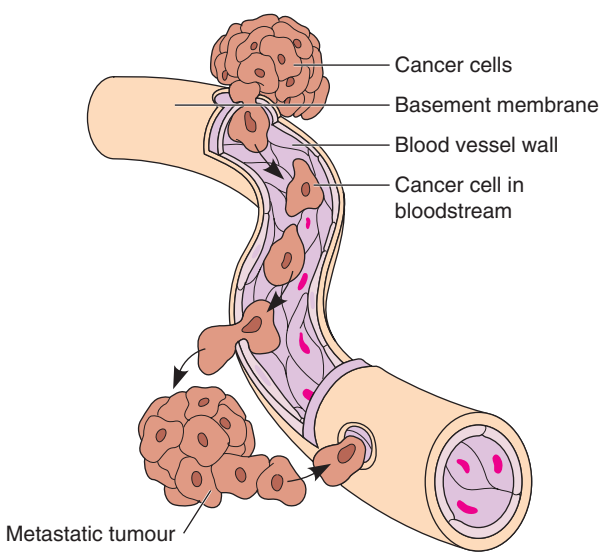


FIGURE 13.3 ■ Metastasis through the bloodstream. Cancer cells secrete enzymes and a motility factor that disrupt the basement membrane in the blood vessel. In this way, the cancer cells gain access to the circulation. Once in the blood, only about 1 cell in 1000 escapes immune detection, but that can be enough. Undetected cells move out of the blood, again secreting enzymes and cutting through the vessel wall into new tissue. The tissue selected for establishing a new tumour may be downstream from the original tumour or a chemical attraction may cause the malignant cells to target a specific site. Once in the new site, the malignant cells multiply and establish a metastatic tumour

TABLE 13.3 Various cancers and sites of metastases

PRIMARY TUMOUR	COMMON METASTATIC SITES
Bronchogenic (lung)	Spinal cord, brain, liver, bone
Breast	Regional lymph nodes, vertebrae, brain, liver, lung, bone
Colon	Liver, lung, brain, ovary, bone
Prostate	Bladder, bone (especially vertebrae), liver
Malignant melanoma	Lung, liver, spleen, regional lymph nodes, brain

BOX 13.4 Factors that may weaken or alter the immune response

- Accumulated stress
- Depression
- Increased age
- Pregnancy
- Chronic disease
- Chemotherapy treatment for the primary cancer

- For tumour cells to be recognised as foreign by the immune system, they must display on their surface a special antigen called tumour-associated antigen (TAA). TAA marks tumour cells for destruction by the lymphocytes. Some oncogenic viruses depress the expression of TAA on infected cells. Also, some tumours in advanced stages of growth no longer display TAA. Thus, such tumour cells escape detection as they travel through the blood or lymph.
- If the person's immune response is weakened or altered, then a metastatic tumour may take hold with little opposition. Factors that may weaken or alter the immune response are listed in Box 13.4.

An estimated 50% to 60% of all cancers have already metastasised by the time the primary tumour is identified. This may account for the current 50% death rate and certainly supports the need to educate people to facilitate early diagnosis. The time it takes for metastasis to occur is extremely variable and often difficult to predict. Some cancers, such as basal cell carcinomas, do not metastasise. The aggressiveness and location of the tumour and the state of the person's immune system determine how rapidly and whether metastasis will take place.

PHYSIOLOGICAL AND PSYCHOLOGICAL EFFECTS OF CANCER

Much of the nursing care for people with cancer is related to the generalised effects of cancer on the body and the side effects of the treatments used to remove or destroy the cancer. Although pathophysiological effects of the cancer vary with the type and location of the cancer, the following effects usually are observed.

Disruption of function

Physiological functioning can be upset by obstruction or pressure. For example, a large tumour in the bowel can stop intestinal motility, resulting in a bowel obstruction. Prostate tumours can obstruct the bladder neck or urethra, resulting in urine retention. Intracranial pressure can be dangerously increased by a glioma. Obstruction or pressure can cause anoxia and necrosis of surrounding tissues, which in turn cause a loss of function of the involved organ or tissue. For example, a kidney tumour may progress to renal failure. Pressure against the superior vena cava from an adjacent lung tumour or tumour-infiltrated lymph nodes can interrupt the blood flow to the heart.

In the liver, either a primary hepatocellular cancer or metastatic lesion can have several significant effects:

- In liver parenchymal tissue, it impairs the multiple life-sustaining functions of the liver, such as carbohydrate metabolism, synthesis of plasma proteins, detoxification and immunological functions. These functional impairments result in severe nutritional, hormonal, haematological and immunological problems. (See Chapter 20 for a more complete discussion of liver functions and effects of disruption.)
- Because more than 1 L of blood per minute passes through the liver via the portal vein, obstruction to this flow by a tumour can cause portal hypertension. This results in backup of fluid and increased pressure in the splanchnic circulation. The end result is ascites (third-spaced fluid in the peritoneal cavity) and varices (friable, over-distended blood vessels) of the oesophageal, gastric, mesenteric and haemorrhoidal vessels.

Haematological alterations

Haematological alterations can impair the normal function of blood cells. For example, in leukaemia, a malignant proliferative disease of the haematopoietic (blood-cell-producing) system, the immature leucocytes cannot perform the normal protective phagocytic functions and immunity is compromised. The excessive numbers of immature leucocytes in the bone marrow diminish erythrocyte and thrombocyte (platelet) production, resulting in secondary anaemia and clotting disorders.

Other examples of haematological alteration include the following:

- Gastrointestinal tumours disrupt the absorption of vitamin B12 and iron.
- Growing tumours need purines and folate and have a unique ability to accumulate and store these substances. Thus, the tumour deprives the bone marrow of these substances, which are needed for erythropoiesis (red blood cell production).
- Renal cell carcinoma produces its own erythropoietin hormone, which causes an excessively large number of red blood cells to be produced and dumped into the bloodstream. The resulting polycythaemia causes viscous blood, which impairs circulation, plugs small capillaries and promotes thrombus formation.

Infection

If the tumour invades and connects two incompatible organs, such as the bowel and bladder, and thus creates a fistula, infection becomes a serious problem. As they destroy viable tissue and thus their source of nutrition, tumours may become necrotic and septicaemia may result. Some tumours are less efficient in creating capillaries and, as a consequence, the centre of the tumour may become necrotic and infected. When a tumour grows near the surface of the body, it may erode through to the surface, thus breaking down the natural defences of intact skin and mucous membranes and providing a site for the entry of microorganisms. Any malignant involvement of the organs or tissues of immunity—such as the liver, bone

marrow, Peyer's patches in the small intestine, spleen or lymph nodes—can seriously impair the immune response, allowing infections to develop in vulnerable tissues.

Haemorrhage

Tumour erosion through blood vessels can cause extensive bleeding, giving rise to severe anaemia. Haemorrhage can be serious enough to cause life-threatening hypovolaemic shock.

Anorexia–cachexia syndrome

A characteristic feature of cancer is the wasted appearance of its victims, called **cachexia**. In many cases, unexplained rapid weight loss is the first symptom that brings the person to a healthcare provider. This can be due to a variety of problems associated with cancer, such as pain, infection, depression or the side effects of chemotherapy and radiation. However, usually the emaciation, malnutrition and loss of energy are attributed to the anorexia–cachexia syndrome.

This syndrome is specific to cancer because of the effect of cancer cells on the host's metabolism. The neoplastic cells divert nutrition to their own use while causing changes that reduce the person's appetite. Early in the disease, glucose metabolism is altered, causing an increase in serum glucose levels. Through the process of negative feedback, anorexia (loss of appetite) results. In addition, the tumour secretes substances that decrease appetite by altering taste and smell and producing early satiety. Pain, infection and depression also contribute to anorexia. Some types of cancers cause specific food aversions, such as to red meat, coffee or chocolate.

Avaricious cancer cells support their growth through widespread catabolism of the body's tissue and muscle proteins. This catabolism, coupled with inadequate nutrient intake, results in the typical cachexia. Normally, a starvation state reduces the body's basal metabolic rate. However, in many people with cancer, the metabolic rate is increased, probably because of the hyperactive metabolic and reproductive activities of the malignant cells. One theory suggests that cytokinins the body produces in response to the tumour are responsible for both early satiety and cachexia. One specific cytokine—called tumour necrosis factor alpha, or cachectin—is believed to enhance the increased metabolic consumption of nutrients. Cancers of the gastrointestinal system further promote anorexia–cachexia by decreasing absorption and use of nutrients; the side effects of some treatment modalities enhance this effect. Figure 13.4 shows the characteristic appearance of a cachectic person.

Paraneoplastic syndromes

Paraneoplastic syndromes are indirect effects of cancer. They may be early warning signs of cancer or indicate complications or return of a malignancy. The most frequently occurring paraneoplastic syndromes are endocrine, occurring when cancers set up ectopic sites of hormone production (Grossman & Porth, 2014). Table 13.4 lists laboratory indicators of ectopic functioning. These ectopic sites produce excessive amounts of the hormone, which harm the host. Consider the following examples:

FIGURE 13.4 ■ A cachectic person. Cancer robs its host of nutrients and increases body catabolism of fat and muscle to meet its metabolic needs

Source: Carole Gomez/Getty Images.

- Breast, ovarian and renal cancers may set up ectopic parathyroid hormone sites, causing severe hypercalcaemia.
- Oat cell and other lung cancers may produce ectopic secretions of insulin (causing hypoglycaemia), parathyroid hormone (PTH), antidiuretic hormone (ADH, which causes excessive fluid retention, hypertension and peripheral oedema) and adrenocorticotropic hormone (ACTH). See Chapter 18 for a description of the multiple problems caused by excessive secretions of cortisone.

Other paraneoplastic syndromes include haematological abnormalities such as anaemia, thrombocytopenia and coagulation abnormalities; nephrotic syndrome; cutaneous syndromes and neurological syndromes, such as distant tumours that produce increased ICP.

TABLE 13.4 Laboratory indicators of ectopic functioning

HORMONE	SPECIFIC LABORATORY TEST
Antidiuretic hormone (ADH)	Serum and urine osmolality
Adrenocorticotropic hormone (ACTH)	Plasma ACTH ACTH suppression test ACTH stimulation test Urine catecholamines
Calcitonin	Serum calcitonin
Insulin	Serum glucose Glucose tolerance test
Parathyroid hormone (PTH)	Serum PTH Serum calcium
Thyroxine	Serum thyroid-stimulating hormone (TSH), T_3, T_4

Pain

Pain is ranked as one of the most serious concerns of people with cancer, their families and oncology healthcare professionals. Despite extensive progress in the scientific understanding of pain, more than 60% of people with cancer experience moderate to severe pain at some time during their illness (Ogboli-Nwasor, Makama & Yusufu, 2013). Because pain management for people with cancer has a reputation for being ineffective, the anticipation of pain may engender fear in even the most stoic people. Most people fear pain and suffering even more than possible death, although pain management strategies have improved tremendously. The findings from a 10-year study of more than 2000 people in a palliative care program are encouraging. Following the World Health Organization guidelines for cancer pain relief, 88% of people reported good to satisfactory pain relief (Zech et al., 1995). Research on pain with its devastating statistics has led to great improvement in pain management strategies.

Types of cancer pain

Cancer pain can be divided into two main categories, acute and chronic, with subgroupings. These classifications serve to indicate appropriate therapeutic approaches. Acute pain has a well-defined pattern of onset, exhibits common signs and symptoms, and is often identified with hyperactivity of the autonomic system. Chronic pain, which lasts more than 6 months, frequently lacks the objective manifestations of acute pain, primarily because the autonomic nervous system adapts to this chronic stress. Unfortunately, chronic pain often results in personality changes, alterations in functional abilities and lifestyle disruptions that can seriously affect compliance with treatment and the quality of life.

Most people with cancer who cite acute pain as the primary symptom that led to the diagnosis tend to associate pain with the introduction to their disease. If these people experience pain during the illness or after therapy, they often perceive the pain as introducing another cancer or as a recurrence of the original cancer. Other people report experiencing pain as a component of cancer therapy. These people are often able to endure the pain in anticipation of a successful outcome of treatment (Rosedale & Fu, 2010).

Chronic pain may be related to treatment or may indicate progression of the disease. Identifying the pain as treatment-related rather than tumour-related is extremely important because it has a definite effect on the person's psychological outlook. For the person whose pain is due to the advancement of the disease, psychological factors play an even more important role. Hopelessness and fear of impending death intensify physiological pain and contribute to overall suffering, which goes well beyond just physical pain.

Three other categories used to classify people with cancer pain are worth mentioning: people with pre-existing pain, those with a history of drug abuse and people dying with cancer-related pain. The first two groups may have altered perceptions of pain and may not have the anticipated response to pain medication. For the dying person, pain is strongly associated with both the person and their family's confrontation of issues of hopelessness and death. Confronting these issues can intensify the perception of pain (see Chapter 4).

Causes of cancer pain

Direct tumour involvement is the primary cause of the pain experienced by people with cancer. This includes metastatic bone disease, nerve compression and involvement of visceral organs. The pain from tumour involvement is believed to be mechanical, resulting from stretching of tissues and compression. Chemicals from ischaemia or tumour metabolites and toxins that activate and sensitise nociceptors and mechanoreceptors are also responsible for tumour pain. See Chapter 8 for a more complete discussion of the mechanics of pain.

Side effects or toxic effects of cancer therapies (e.g. surgery, radiation and chemotherapy) may also cause cancer pain. These are usually the result of traumatised tissue; one example of this is the oropharyngeal ulcerations that occur with some types of chemotherapy. However, these therapies may also be used to manage pain, such as radiation to decrease pain associated with bone metastasis.

Physical stress

When the immune system discovers a neoplasm, it tries to destroy it using the resources of the body. The body mounts an all-out assault on the foreign invader, calling on many resources including chemical mediators, hormones and enzymes, blood cells, antibodies, proteins and inflammatory and immune responses.

These protective responses also mobilise fluid, electrolytes and nutritional systems. This massive effort requires tremendous energy. See Chapters 9 and 21 for specific information on these systems. If the neoplasm is small enough (i.e. microscopic), the immune system can destroy it and a tumour will never manifest. A neoplasm of 1 cm is large enough to overwhelm most immune systems; however, the body will continue to try to fight it until it reaches the stage of exhaustion and is no longer capable (Selye, 1984). Thus, many people with cancer present with fatigue, weight loss, anaemia, dehydration and altered blood chemistries (e.g. decreases in electrolytes).

Psychological stress

People confronted with the diagnosis of cancer exhibit a variety of psychological and emotional responses. Some people see cancer as a death sentence and experience overwhelming grief, often giving up. Others may feel guilt, considering the cancer a punishment for past behaviours, such as smoking or unhealthy eating habits, or for delaying diagnosis or treatment. The person may experience anger, especially if they believe that they had been practising a healthy lifestyle; beneath that anger may reside feelings of powerlessness. Fear is common: fear of the outcome of the illness, fear of the effects of treatment, fear of pain, fear of death. Some people feel isolated because of the stigma of cancer and old beliefs of contagion. Body image concerns and sexual dysfunction may be present but often unexpressed, especially if the cancer is of the breast or sexual organs or causes visible body changes. Box 13.5 summarises the physiological and psychosocial effects of cancer.

BOX 13.5 Physiological and psychosocial effects of cancer

- Disruption of function (due to obstruction or pressure)
- Haematological alterations
  a. Decreased leucocytes, erythrocytes and thrombocytes
  b. Altered erythropoiesis
- Infections
  a. Fistula between non-compatible organs
  b. Necrosis of tumour centre
  c. Malignant involvement of organs of immunity
- Haemorrhage (caused by erosion of neoplasm through blood vessels or surface of skin)
- Anorexia–cachexia syndrome
  a. Hyperglycaemia
  b. Catabolism of tissue and muscle proteins
  c. Altered taste and smell
- Creation of ectopic sites of hormones
  a. PTH
  b. Insulin
  c. ADH
  d. ACTH
- Paraneoplastic syndromes
  a. Deep venous thrombosis
  b. Peripheral nerve problems
  c. Increased intracranial pressure
  d. Anorexia–cachexia syndrome
  e. Nephrotic syndrome
- Pain
  a. Acute and chronic
  b. Caused by direct tumour involvement or side effects of therapy
- Physical stress
  a. Increased general adaptation syndrome activity
  b. Increased immunological activity
  c. Increased inflammatory response activity
  d. Nutritional, fluid and electrolyte alterations
- Psychological stress
  a. Grief
  b. Hopelessness
  c. Guilt
  d. Anger
  e. Fear
  f. Isolation
  g. Body image concerns
  h. Sexual dysfunction

Note: Manifestations depend on the type and location of the cancer.

INTERPROFESSIONAL CARE

Interprofessional care for the person with cancer begins with a variety of specialised laboratory and diagnostic tests.

Diagnosis

Several procedures are used to diagnose cancer. X-ray imaging, computed tomography (CT), ultrasonography and magnetic resonance imaging (MRI) can locate abnormal tissues or tumours. However, only microscopic histological examination of the tissue reveals the type of cell and its structural difference from the parent tissue. Tissue samples are acquired through biopsy, shedded cells (e.g. Papanicolaou smear) or collections of secretions (e.g. sputum). Lymph nodes are also biopsied to determine whether metastasis has begun. Simple screening procedures can be used to pick up substances secreted by the tumour, such as the prostatic-specific antigen (PSA) blood test which is being used to identify early prostatic cancers. Increases in enzymes or hormones released by normal tissues when they are damaged can also contribute to the diagnosis. Increased alkaline phosphatase noted in bone metastases and osteosarcoma is one example of an enzyme increase associated with cancer. Tumour markers are used for early diagnosis, for tracking responses to therapy and for devising immunological treatments.

Some investigators studying chemical mediators of the immune system have noted that there seems to be communication between the chemical mediators and the emotional centres of the brain. A person who states 'I feel I have cancer' should be listened to and the complaint investigated thoroughly.

CLASSIFICATION To help standardise diagnosis and treatment protocols, an elaborate identification system has been developed. This consists of naming the tumour (classification) and describing its aggressiveness (grading) and spread within or beyond the tissue of origin (staging).

Tumours are classified and named by the tissue or cell of origin. Tumour nomenclature often incorporates the Latin stem identifying the tissue from which the tumour arises. For example, a carcinoma arises from epithelial tissue; adjectives are added to further specify the location. A glandular malignancy arising from epithelial tissue is classified as an adenocarcinoma. A tumour arising from supportive tissues is called a sarcoma; the specific type of tissue is added as a prefix. For example, a cancer of fibrous connective tissue is called fibrosarcoma and a smooth muscle cancer is a leiomyosarcoma. A tumour from seminal or germ tissue is called a seminoma. Table 13.5 compares the nomenclature of benign and malignant neoplasms.

Other names for tumours incorporate the name of the discoverer of that particular cancer, such as Burkitt's lymphoma or Hodgkin's disease. Haematopoietic malignancies (also known as 'liquid tumours') are usually named by the type of immature blood cell that predominates. An example is myelocytic leukaemia, named for the immature form of the granulocyte that is predominant in this malignancy.

GRADING AND STAGING Grading evaluates the amount of differentiation (level of functional maturity) of the cell and estimates the rate of growth based on the mitotic rate. Cells that are the most differentiated—that is, most like the parent tissue

TABLE 13.5 Nomenclature for benign and malignant neoplasms

	TISSUE OF ORIGIN	BENIGN	MALIGNANT
Ectoderm/endoderm	Epithelium	Papilloma	Carcinoma
	Gland	Adenoma	Adenocarcinoma
	Liver cells	Hepatocellular adenoma	Hepatocellular carcinoma
	Neuroglia	Glioma	Glioma
	Melanocytes	Melanoma	Malignant melanoma
	Basal cells		Basal cell carcinoma
	Germ cells	Tetroma	Seminoma
Mesoderm	*Connective tissue*		
	Adipose tissue	Lipoma	Liposarcoma
	Fibrous tissue	Fibroma	Fibrosarcoma
	Bone tissue	Osteoma	Osteosarcoma
	Cartilage	Chondroma	Chondrosarcoma
	Muscle		
	Smooth muscle	Leiomyoma	Leiomyosarcoma
	Striated muscle	Rhabdomyoma	Rhabdomyosarcoma
	Neural tissue		
	Nerve cells	Ganglioneuroma	Neuroblastoma
	Endothelial tissues		
	Blood vessels	Haemangioma	Angiosarcoma
			Kaposi's sarcoma
	Meninges	Meningioma	Malignant meningioma
Haematopoietic tissues	Granulocytes	Granulocytosis	Leukaemia
	Plasma cells		Multiple myeloma
	Lymphocytes		Lymphoma

and therefore the least malignant—are classified as grade 1 and are associated with a better prognosis. Grade 4 is reserved for the least differentiated and most aggressively malignant cells. Because of the differences inherent in tumour appearance and biological behaviour, grading criteria may vary with different locations and types of tumours.

Staging is used to classify solid tumours and refers to the relative size of the tumour and extent of the disease. The TNM classification system is an internationally recognised staging system: T stands for the relative tumour size, depth of invasion and surface spread; N indicates the presence and extent of lymph node involvement; and M denotes the presence or absence of distant metastases. Table 13.6 shows the basic outline of the TNM system; however, other systems are also used to differentiate types and locations of tumours (e.g. melanomas, cervical cancer, Hodgkin's disease).

CYTOLOGICAL EXAMINATION

For malignant tissues to be identified by name, grade and stage, they must first be subjected to histological and cytological examination by light or electron microscope. Specimens are collected by three basic methods:

1. *Exfoliation from an epithelial surface*. Examples include scraping cells from the cervix (Pap smear) or bronchial washings.
2. *Aspiration of fluid from body cavities or blood*. Examples include white blood cells for evaluation of haematopoietic cancers, pleural fluid and cerebrospinal fluid.
3. *Needle aspiration of solid tumours*. This could include the breast, lung or prostate.

Cytological examination is also carried out on specimens from biopsied tissues or tumours and on collected body secretions, such as sputum or urine.

After collection, specimens are spread on a glass slide, fixed and stained if necessary. The morphological features of the cells are examined, with special attention to the nucleus and cytoplasm. Other special pathological procedures can be carried out on the specimen, but they must be ordered ahead of time if special preparations of the specimen are necessary. Several special diagnostic cytological procedures, such as cytogenetics, are proving useful in diagnosing and monitoring a person's response to treatment.

TUMOUR MARKERS

A **tumour marker** is a protein molecule detectable in serum or other body fluids. This marker is used as a biochemical indicator of the presence of a malignancy. Small amounts of tumour marker proteins are found in normal body tissues or benign tumours and are not specific for malignancy. However, high levels are suspicious and mandate follow-up diagnostic studies. Tumour marker tests are most useful for monitoring the person's response to therapy and for

TABLE 13.6 TNM staging classification system

	STAGE	MANIFESTATIONS
Tumour	T_0	No evidence of primary tumour
	T_{IS}	Tumour in situ
	T_1, T_2, T_3, T_4	Ascending degrees of tumour size and involvement
Nodes	N_0	No abnormal regional nodes
	N_{1a}, N_{2a}	Regional nodes—no metastasis
	N_{1b}, N_{2b}, N_{3b}	Regional lymph nodes—metastasis suspected
	N_x	Regional nodes cannot be assessed clinically
Metastasis	M_0	No evidence of distant metastasis
	M_1, M_2, M_3	Ascending degrees of metastatic involvement of the host including distant nodes

detecting residual disease. However, one marker, PSA, is a detector of prostate cancer. As a result, many healthcare practitioners recommend screening for it in men aged over 40, much as Pap smears and mammograms are recommended for women.

Tumour markers fall into two general categories: those derived from the tumour itself and those associated with host (immune) response to the tumour. Examples of tumour markers include the following:

- *Antigens.* These are present in foetal tissue but are normally suppressed after birth. Thus, their presence in large amounts may reflect an anaplastic process in tumour cells. Alpha-fetoprotein (AFP) and carcinoembryonic antigen (CEA) are oncofetal antigens.
- *Hormones.* Hormones are, of course, present in considerable amounts in human blood and tissues, but very high levels not related to other conditions may signify the presence of a hormone-secreting malignancy. Some common hormones seen as tumour markers include human chorionic gonadotropin (HCG), antidiuretic hormone (ADH), parathyroid hormone (PTH), calcitonin and catecholamines.
- *Proteins.* These narrow down the type of tissue that may be malignant, although they can also be increased in hyperplastic disorders. Examples of tissue-specific proeins include serum immunoglobin and beta-2 microglobulin.
- *Enzymes.* Rapid, excessive growth of a tissue may cause some of the enzymes and isoenzymes normally present in that particular tissue to spill into the bloodstream. Elevated levels can point to either hyperplasia of the tissue or cancer. Prostatic acid phosphatase (PAP) and neuron-specific enolase (NSE) are examples. Table 13.7 compares selected tumour-derived markers with their presence in neoplasms and other conditions.

ONCOLOGICAL IMAGING

Because physical assessment usually cannot detect cancer until the tumour has reached a size that poses a major risk of metastasis, radiological examination is extremely important in early diagnosis. This diagnostic process may involve routine x-ray imaging (usually for screening only), CT, MRI, ultrasonography, nuclear imaging, angiography and positron emission tomography.

TABLE 13.7 Tumour-derived markers associated with specific neoplasms

	TUMOUR MARKER	ASSOCIATED NEOPLASM
Oncofetal antigens	Carcinoembryonic antigen (CEA)	Adenocarcinomas of colon, lung, breast, ovary, stomach, pancreas
	Alpha-fetoprotein (AFP)	Hepatocellular carcinoma, gonadal germ-cell tumours (seminoma)
Hormones	Human chorionic gonadotropin (HCG)	Gonadal germ-cell tumours
	Calcitonin	Medullary cancer of thyroid
	Catecholamines/metabolites	Pheochromocytoma
Isoenzymes	Prostatic acid phosphatase (PAP)	Adenocarcinoma of prostate
	Neuron-specific enolase (NSE)	Small-cell lung carcinoma, neuroblastoma
Specific proteins	Prostate-specific antigen (PSA)	Adenocarcinoma of prostate
	Immunoglobin	Multiple myeloma
	CA 125	Epithelial ovarian cancer
	CA 19-9	Adenocarcinoma of pancreas, colon
	CA 15-3	Breast cancer

Source: Adapted from The pathologic evaluation of neoplastic disease by J. D. Pfeifer & M. R. Wick (1995). In A. I. Holleb, D. J. Fink & G. P. Murphy (eds), *American Cancer Society Textbook of Clinical Oncology*, pp. 75–95. Atlanta: American Cancer Society.

X-ray imaging Considered the least expensive and least invasive diagnostic procedure, film screen imaging (standard x-ray imaging) is the method of choice for screening such body areas as the breast (mammography), lung and bone to identify changes in tissue density that may indicate malignancies. X-ray studies are limited in that they do not easily distinguish between calcifications, benign cystic growths and true malignancies. However, as a screening tool, x-ray imaging can usually reassure the person if findings are negative or encourage follow-up studies if findings are suspicious. X-ray imaging is still the method of choice for lung cancer. Unfortunately, it does not usually reveal tumours until they have reached about 1 cm in size, which is late in their development.

Computed tomography CT has vastly advanced the effectiveness of traditional x-ray methods. CT allows the visualisation of cross-sections of the anatomy, and can reveal subtle differences in tissue densities; they provide much greater accuracy in tumour diagnosis. This procedure is useful in the screening for some cancers such as renal cell and most gastrointestinal tumours. CT scans are especially useful to evaluate possible lymph node involvement.

Magnetic resonance imaging MRI involves computerised mathematical technology. The person is placed within a strong magnetic field, pulsed radio waves are directed at the person and transmitted signals, based on tissue characteristics, are analysed by a computer. Related diagnostic imaging procedures—positron emission tomography (PET) and single photon emission computed tomography (SPECT)—create visible images by measuring electrical impulses from different body structures. MRI is the diagnostic tool of choice for both screening and follow-up of cranial and head and neck tumours.

Some people become claustrophobic during the MRI procedure because they must be placed inside the diagnostic imaging machine. The machines make loud thumping sounds that can be frightening if the person is not informed beforehand that this is normal.

Ultrasonography Ultrasonography measures sound waves as they bounce off various body structures, giving an image of normal anatomy as well as revealing abnormalities that indicate tumours. Ultrasonography has been adapted for diagnosing some specific tumours. For example, transrectal ultrasonography has provided excellent imaging of early prostate cancers and is used to guide needle biopsy. Ultrasound imaging is also more useful for detecting masses in the denser breast tissue of young women.

Nuclear imaging Nuclear imaging involves the use of a special scintillation scanner in conjunction with the ingestion or injection of specific radioactive isotopes. This is an invasive but usually safe diagnostic method for identifying tumours in various body tissues. This procedure is often used to check for possible bone or other organ metastases. This evaluation helps the healthcare provider determine appropriate treatment.

The procedure is usually minimally distressing for people. Drinking the isotope solution is not pleasant but is tolerable; some anxious people may have difficulty lying still during the scan. Anti-anxiety medication may help. Some people may experience nausea from drinking the isotope and require antiemetic drugs to complete the procedure. Preparation for the person may include allowing nothing by mouth or clear fluids only after midnight.

Angiography An expensive and invasive procedure, angiography is used infrequently for tumour diagnosis. Angiography is performed when the precise location of the tumour cannot be identified or there is a need to visualise the tumour's extent prior to surgery. The procedure involves injecting a radiopaque dye into a major blood vessel proximal to the organ or tissue to be examined. The movement of the dye through the vasculature of the organ or tissue is then traced by means of fluoroscopy or serial x-ray films. In some cases, small catheters are threaded through the vein under fluoroscopy to ensure the specific placement of the dye. Blockage to the flow of the dye indicates the tumour's location. Dye may also be used to identify blood vessels supplying a tumour, allowing the surgeon to know where to safely ligate vessels. Angiography requires preparation similar to that for minor surgery. This includes ensuring that the person takes in only fluids on the day of the examination, performing skin preparation at the insertion site and administering sedative drugs prior to the procedure. People should be informed that injection of the dye used to enhance imaging may cause a hot, flushing sensation or nausea and vomiting. Although angiography is usually done on an outpatient basis, the person will be kept in a short-stay unit for several hours and monitored for such complications as bleeding at the catheter insertion site.

DIRECT VISUALISATION Direct visualisation procedures are invasive but do not require the use of radiography. Examples include the following:

- sigmoidoscopy (viewing the sigmoid colon with a fibre-optic flexible sigmoidoscope)
- cystoscopy (viewing the urethra and bladder)
- endoscopy (viewing the upper gastrointestinal tract)
- bronchoscopy (inspecting the tracheobronchial tree).

These methods allow the visual identification of the organs within the limits of the scope and usually permit biopsy of suspicious lesions or masses. Flexible fibre-optic scopes may be more useful, because they allow deeper penetration than do traditional scopes. These procedures all require the person to complete some preparation, cause moderate to considerable discomfort and may require sedation or even anaesthesia, as in the case of bronchoscopy. Some procedures, such as sigmoidoscopy and cystoscopy, may be performed in the doctor's surgery and therefore cost less, making them more accessible screening procedures.

Preparation of the person includes a thorough bowel cleansing prior to sigmoidoscopy and cystoscopy; the person may ingest only liquids the morning of the procedure. Because anaesthesia may be required, people undergoing bronchoscopy and endoscopy may be instructed to have nothing by mouth from midnight until the procedure. These procedures are discussed in greater detail in later chapters of this textbook. When the tumour is exposed, a sample of tissue (biopsy) is sent to the pathology laboratory for a 'frozen-section' histological examination. This can be done rapidly while the person remains on the operating table under anaesthesia. If the initial report is

negative, the benign mass is usually removed to prevent further symptoms. If the report is positive for cancer, the tumour and, often, adjacent lymph nodes are resected, along with any other suspicious tissue. The tumour, nodes and any other specimens are sent to the pathology laboratory for more in-depth analysis. The person then receives the usual postoperative care.

LABORATORY TESTS Most laboratory tests of blood, urine and other body fluids are used to rule out nutritional disorders and other non-cancerous conditions that may be causing the person's symptoms. For example, a full blood count (FBC) helps screen for such problems as anaemia, infection and impaired immunity. Blood chemistries can point out nutritional disturbances and electrolyte imbalances. In conjunction with other diagnostic studies, some laboratory tests can be quite useful either in screening for other pathological conditions or for validating the cancer diagnosis (Kee, 2014). These tests include evaluating levels of enzymes such as alanine aminotransferase (alT), aspartate aminotransferase (AST) and lactic dehydrogenase (LDH) for liver metastases. Special protein tumour markers such as PSA for prostate cancer and CEA for colon cancer are also used. Table 13.8 identifies some useful laboratory tests, their normal values and their possible indications.

TABLE 13.8 Laboratory tests used for cancer diagnosis*

TEST	REFERENCE VALUE	ABNORMALITY INDICATED
Acid phosphatase (ACP)		No longer used for prostatic cancer diagnosis
Adrenocorticotropic hormone (ACTH)	8–80 pg/mL	Decreased in adrenal cancer Elevated in pituitary cancer or with tumour that secretes ACTH (bronchiogenic cancer)
Alanine aminotransferase (alT)	Female < 35 u/L Male < 40 u/L	Moderate elevation in liver cancer
Albumin	35–40 g/L	Decreased in malnutrition, metastatic liver cancer
Alkaline phosphatase (alP)	Adult 35–135 u/L	Elevated in cancer of liver, bone, breast and prostate, in leukaemia and in multiple myeloma
Alpha-fetoprotein (AFP)	Male and non-pregnant female: < 11 ng/mL	Elevated in germ-cell tumours (e.g. seminoma), testicular cancer
Aspartate aminotransferase (AST)	< 42 u/L	Elevated in liver cancer
Bilirubin	< 20 mg/dL	Elevated in liver and gallbladder cancer
Bleeding time	Ivy method: 2–9 minutes	Prolonged in leukaemia and metastatic liver cancer
Blood urea nitrogen (BUN)	3.0–8.0 mmol/L	Decreased in malnutrition; increased in renal cancer
Calcitonin	Male: < 40 pg/mL Female: < 20 pg/mL	Elevated to > 500 pg/mL in thyroid medullary cancer, breast cancer and lung cancer
Calcium (Ca)	2.15–2.65 mmol/L	Elevated in bone cancer and ectopic parathyroid hormone production (paraplastic syndrome)
Carcinoembryonic antigen (CEA)	2.5 ng/mL in non-smokers 5 ng/mL in smokers; > 12 ng/mL neoplasms	Elevated with GI cancers, lung, breast, bladder, kidney, cervical cancers and leukaemias. Used to evaluate effectiveness of cancer treatment
Chloride (Cl)	95–108 mmol/L	Decreased in vomiting, diarrhoea, syndrome of inappropriate antidiuretic hormone (SIADH)
C-reactive protein	< 10 mg/L	Elevated in metastatic cancer and Burkitt's lymphoma
Creatinine	Male: 50–110 µmol/L Female: 40–80 µmol/L	Decreased in malnutrition; elevated in most cancers
Dexamethasone suppression test	> 50% reduction in plasma cortisol	Non-suppression in adrenal cancer and ACTH-producing tumours, severe stress
Oestradiol	Female: 20–300 pg/mL Menopausal female: < 20 pg/mL Male: 15–50 pg/Ml	Elevated in oestrogen-producing tumours and testicular tumours
Fibrinogen	2.0–4.0 g/L	Decreased in leukaemia and as a side effect of chemotherapy
Gamma glutamyltransferase (GammaGT)	Male: < 60 u/L Female: < 40 u/L	Elevated in cancer of liver, pancreas, prostate, breast, kidney, lung and brain
Fasting blood sugar	3.5–5.5 mmol/L	Decreased in malnutrition, cancer of stomach, liver and lung

(continued)

TABLE 13.8 Laboratory tests used for cancer diagnosis* (continued)

TEST	REFERENCE VALUE	ABNORMALITY INDICATED
Haptoglobin	0.3–2.0 g/L	Elevated in Hodgkin's disease and cancer of lung, large intestine, stomach, breast and liver
Haemoglobin (Hgb)	Male: 132–170 g/L Female: 115–155 g/L	Decreased in anaemia, many cancers, Hodgkin's disease, leukaemia and malnutrition, and as a side effect of chemotherapy
Human chorionic gonadotropin (HCG)	Non-pregnant female: < 0.01 international unit/L	Elevated in choriocarcinoma
Insulin	5–25 microunit/mL	Elevated in insulinoma (islet cell tumour) and insulin-secreting cancers (e.g. lung cancer)
Lactic dehydrogenase (LDH)	200–400 u/L	Elevated in liver, brain, kidney, muscle cancers, acute leukaemia, anaemia
Occult blood	Negative	Positive in gastric and colon cancers
Serum osmolality	280–300 mOsm/kg H_2O	Decreased in SIADH
Urine osmolality	50–1200 mOsm/kg H_2O	Increased in SIADH
Parathyroid hormone (PTH)	1.5–6.5 pg/mL	Increased in PTH-secreting tumours
Platelet (thrombocyte) count	150–400 ($\times 10^9$/L)	Decreased in bone, gastric and brain cancer, in leukaemia and as a side effect of chemotherapy
Potassium (K)	3.4–5.5 mmol/L	Decreased in vomiting and diarrhoea, and in malnutrition
Prostatic-specific antigen (PSA)	Age-dependent 2.5–6.5 mg/L	Elevated from 10 to 120+ in prostate cancer
Total protein	60–80 g/L	Decreased in malnutrition, gastrointestinal cancer, Hodgkin's disease; elevated in vomiting, diarrhoea, multiple myeloma
Red cell count (RBCs)	Male: 4.5–5.5 million/mm^3 Female: 3.8–4.8 million/mm^3	Decreased in anaemia, leukaemia, infection, multiple myeloma
Sodium (Na)	134–146 mmol/L	Decreased in SIADH, vomiting; elevated in dehydration
Uric acid	Male: < 0.44 mmol/L Female: < 0.38 mmol/L	Increased in leukaemia, metastatic cancer, multiple myeloma, Burkitt's lymphoma and after vigorous chemotherapy
White blood cells (WBC)		
Total leucocytes	4500–10 000/mm^3	Elevated in acute infection, leukaemias, tissue necrosis; decreased as a side effect of chemotherapy
Neutrophils	50–70%	Elevated in bacterial infection and Hodgkin's disease; decreased in leukaemia and malnutrition, and as a side effect of chemotherapy
Eosinophils	1–3%	Elevated in cancer of bone, ovary, testes and brain
Basophils	0.4–1.0%	Elevated in leukaemia and healing stage of infection
Monocytes	4–6%	Elevated in infection, monocytic leukaemia and cancer; decreased in lymphocytic leukaemia and as a side effect of chemotherapy
Lymphocytes	25–35%	Elevated in lymphocytic leukaemia, Hodgkin's disease, multiple myeloma, viral infections and chronic infections; decreased in malnutrition, cancer and other leukaemias, and as a side effect of chemotherapy

*All values refer to serum values unless otherwise indicated. Values are approximate; check the reference standards specified by your own organisation's laboratory.

PSYCHOLOGICAL SUPPORT DURING DIAGNOSIS

Preparing for and awaiting the results of diagnostic tests can create extreme anxiety. Many people compare the experience to that of a prisoner awaiting trial and sentencing: after they know what the 'sentence' is, they can then prepare for the future. In addition to coping with the possibility of a life-threatening disease or at least a life-altering one, people often also face the prospect of uncomfortable, even painful, diagnostic procedures. They have important decisions to make that depend on the outcome of those tests. Many unspoken questions may exist, including the following:

- Do I have cancer?
- If so, what kind and how serious?
- Has it spread?
- Will I survive?
- What kind of treatment is needed?

- How will this affect my lifestyle?
- How will this affect my family members and friends?

Denial or intellectualisation serves some people well, but others display signs of anxiety and stress as they attempt to cope. The nurse can provide valuable support during this very difficult stage by helping people become actively involved in managing their life and disease. Talk with the person as soon as they enter the healthcare system, asking what they know already about what is going to happen and soliciting questions from them. Taking this approach and encouraging people to share what knowledge and experience they have allows them to maintain control. From there, the nurse can provide the information needed.

It is essential that people thoroughly understand the preparation required for their tests, especially if they will be preparing at home. They also need to be informed of any unusual effects that may occur as a result of the procedure, such as nausea from radioactive dye. If possible, a phone call the evening before to verify the person's understanding of the procedure and to answer questions can be helpful and supportive.

As the person begins to feel more comfortable with the nurse, they may express concerns, fears and other emotions. The nurse should actively listen and be supportive, but avoid giving advice and false reassurance, providing appropriate information when needed. For people who are not ready to discuss their concerns, or for those who appear angry, being non-judgmental and providing non-verbal support may facilitate more open communication. An atmosphere of calmness, warmth, caring and respect can ease the tension and often unspoken terror of this initial period.

Support of and communication with the person's significant others is extremely important. Often they try to be strong for the person but have many fears and emotional concerns that they do not feel comfortable expressing. The nurse needs to be available to the family while the person is undergoing diagnostic procedures. Allowing them to talk without the need to edit for the person's benefit can help them manage their own difficulties in coping with their loved one's potential cancer diagnosis.

Cancer treatment

The goals of cancer treatment are aimed at cure, control or palliation of symptoms. These goals may overlap. Cancer may be treated through surgery, chemotherapy, radiation therapy, biotherapy, photodynamic therapy, bone marrow and stem cell transplants, and complementary therapies. Once cancer is diagnosed, the initial focus is on surgical and medical treatment. The goals of treatment are:

- eliminating the tumour or malignant cells
- preventing metastasis
- reducing cellular growth and the tumour burden
- promoting functional abilities and providing pain relief to those whose disease has not responded to treatment.

SURGERY Surgery was once considered the only treatment for cancer before the mechanisms of cancer were understood. Today, surgery remains an important approach in cancer care. Surgical resection is used for diagnosis and staging of more than 90% of all cancers and for primary treatment of more than 60% of cancers. The goals of surgery have also expanded to include prophylaxis, diagnosis, treatment, reconstruction and palliation.

Prophylactic surgery aims to remove tissues or organs that are likely to develop cancer. Advances in identification of genetic markers make prophylactic surgery an option for individuals with a strong family history and genetic predisposition for the development of cancer. For example, a woman with a strong history of breast cancer, positive findings of BRCA-1 or BRCA-2 and abnormal finding on mammography may consider prophylactic mastectomy as one of the selective options. Other examples of prophylactic operations include colectomy and oophorectomy. With limited research on the long-term physiological and psychological effects on individuals undergoing prophylactic surgery for cancer, it is vital for nurses and other healthcare professionals to discuss the potential risks and postoperative outcomes of the prophylactic surgery thoroughly with the person and their family prior to the surgery. Nurses should respect the person's decision whether or not to pursue the prophylactic surgery. For those people who choose prophylactic surgery as a preventive measure for cancer, comprehensive preoperative teaching and counselling should be provided and long-term postoperative follow-up should be ensured to monitor the person's physiological and psychological adjustment to the surgery.

Diagnostic surgery aims to ensure histological diagnosis and staging of cancer through biopsy, endoscopy, laparoscopy and open surgical exploration. Table 13.9 provides information about common surgical diagnostic procedures.

As a primary treatment for cancer, the goal of surgery is to remove the entire tumour and involved surrounding tissue and lymph nodes as much as possible and feasible. This sometimes

TABLE 13.9 Surgical diagnostic procedures

PROCEDURE	EXPLANATION
Fine-needle biopsy	Use of a very thin needle to aspirate a small amount of tissue from the tumours
Needle core biopsy	Use of a slightly larger needle than that used for a fine-needle biopsy to extract a small amount of tissue from tumours that cannot be aspirated by fine-needle aspiration
Incisional biopsy	Removal of part of a larger tumour by cutting through the skin
Excisional biopsy	Removal of an entire tumour through operation
Endoscopy	Use of a small viewing lens or video camera through natural body openings to view tumours such as cancer of the oesophagus, stomach or colon
Laparoscopy	Use of a small viewing lens or video camera through a small incision in the abdominal wall

necessitates mutilation of the body and the creation of new structures to assume the function of the lost structures. For example, removal of the distal sigmoid colon and rectum requires a new means of bowel elimination, so the remaining healthy segment of the bowel is brought out through a created opening (stoma) in the abdominal wall, resulting in a permanent colostomy (see Chapter 23). In like manner, when the bladder is removed, the ureters are transplanted into a created pouch just under the abdominal wall. This serves as a continent ileostomy, a substitute reservoir for urine (see Chapter 26). Surgery can also destroy sensitive nerve plexuses, resulting in alteration or loss of normal functioning; for example, prostate surgery may result in incontinence and impotence. Surgical removal of involved regional lymph nodes can also lead to long-term lymphoedema (swelling in the affected area) that greatly inpacts on cancer survivors' quality of life; for instance, lymphoedema following surgery for breast cancer and melanoma (Norman et al., 2009).

Not all surgery results in such radical changes in functioning. The following surgeries can eliminate cancer successfully with less distressing results:

- removing a non-essential portion of the organ or tissue containing the tumour, such as in situ small-bowel tumours
- removing an organ whose function can be replaced chemically, such as the thyroid
- resecting one of a pair of organs when the unaffected organ can take over the function of the missing one, such as a lung.

Although the removal of any major body part has physiological and psychological consequences, the alternative—terminal disease—is usually less desirable.

If the tumour is in a non-resectable location or deeply invasive with metastases, surgery may be a palliative measure to allow the involved organs to function as long as possible, to relieve pain and provide comfort, or to bypass an obstruction. Surgery may also be done to reduce the bulk of the tumour in advanced disease, both at primary and metastatic sites. Decreasing the tumour size enhances the ability to control the remaining disease through other modalities. Surgery is often used in conjunction with other treatments to effect a cure. In cases when extensive removal of tissue is contraindicated (e.g. in surgical removal of a brain tumour), radiation may be used prior to surgery in an attempt to shrink the tumour before it is removed.

Surgical intervention may also be used for reconstruction and rehabilitation to achieve more desirable functional and cosmetic effects after curative or radical surgery. One example is the construction of transabdominal myocutaneous (TRAM) flaps in conjunction with or following modified radical mastectomy (see Chapter 48). For surgical interventions for cancers affecting specific body systems, refer to later chapters.

Surgical oncologists are working with researchers to identify premalignant disease earlier in high-risk populations and to conduct studies on ways to reverse oncogenic cell activity. Surgeons also work with molecular biologists using sophisticated techniques to develop monoclonal antibodies. Laser technology is being explored for use in different types of cancer surgery because it minimises blood loss, reduces deformity, increases the accuracy of tissue resection and enhances healing. Lasers are currently being used to treat radical prostatectomy in order to preserve urinary continence and sexual functioning. Another collaborative strategy under development is intraoperative radiation therapy, in which radiosensitive, non-diseased organs that may be damaged by radiation therapy are moved away from the radiation field and shielded. Radiation is then administered while the person is on the operating table. This technique allows more penetrating radiation to be directed to the malignant tumour with less trauma to normal, vulnerable tissues or organs.

Nursing responsibilities focus on preparing the person physically and psychologically for the specific surgery, as well as teaching routine postoperative care in which the person is expected to participate. For example, the nurse teaches the person about respiratory care and deep breathing to improve postoperative ventilation, about early ambulation to prevent circulatory problems and about how the person will receive fluids and nutrition (intravenously or orally, depending on the type of surgery). In addition, the nurse explains the specific surgical procedure and any anticipated alterations to the person's body, especially those that require major lifestyle adjustments, such as a colostomy. Before surgery, the nurse should give the person the opportunity to ask questions and to discuss concerns and fears. In some cases, the person may want to discuss alternative treatment options. In the latter case, the nurse should contact the oncologist and the surgeon and set up a conference for the person before surgery.

CHEMOTHERAPY

Chemotherapy involves the use of cytotoxic medications to cure some cancers, such as leukaemias, lymphomas and some solid tumours; to decrease tumour size, adjunctive to surgery or radiation therapy; or to prevent or treat suspected metastases. Chemotherapy may also be used in conjunction with biotherapy. All chemotherapy has side effects or toxic effects. The type and severity depend on the drugs used.

Chemotherapy disrupts the cell cycle in various phases by interrupting cell metabolism and replication. It also works by interfering with the ability of the malignant cell to synthesise vital enzymes and chemicals. Phase-specific drugs work during only some phases of the cell cycle; non-phase-specific drugs work through the entire cell cycle. Figure 13.5 lists some of the drugs useful in each phase of the cell cycle.

Most chemical treatment involves combinations of drugs in specific protocols given over varying periods of time. One protocol for adult acute lymphocytic leukaemia (alL) uses the acronym DVPA: daunorubicin given on days 1 to 3; vincristine given on days 1, 8, 15 and 22; prednisone given on days 1 to 28 and asparaginase given on days 17 to 28. The treatment regimen is given in cycles with rest periods allowed, especially if toxic effects such as liver dysfunction or severe neutropenia occur. The treatment is continued until the disease goes into remission. If the disease progresses, the particular protocol is abandoned and a new one may be tried.

The cell-kill hypothesis explains why several courses of chemotherapy are necessary. A 1-cm tumour contains about 10^9 (10 billion) total cells, most of which are viable. During

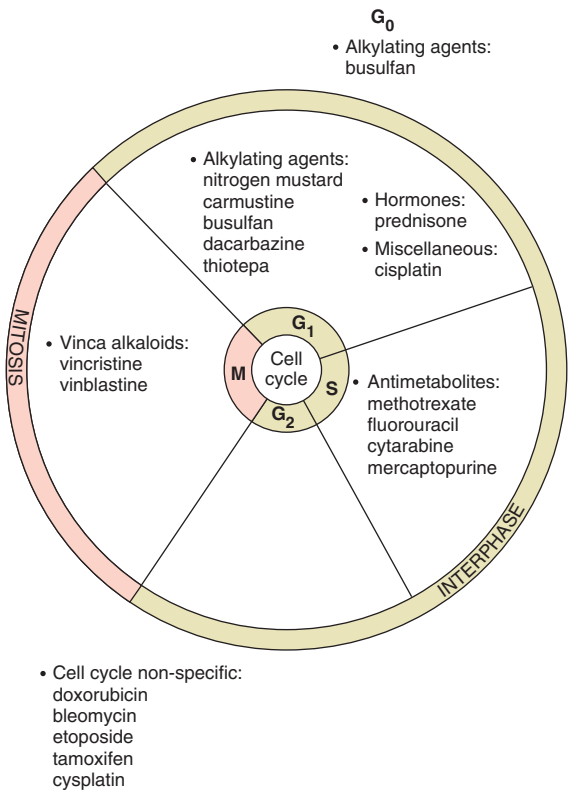


FIGURE 13.5 ■ Chemotherapeutic drugs useful in each phase of the cell cycle. Based on their chemical make-up and biological activity, different drugs used for cancer treatment act in specific phases and subphases of the cell cycle. Some drugs, called non-phase-specific drugs, are generalised and act throughout the cycle. Chemotherapy often involves combinations of drugs designed to attack the cancer cells at many different times in the cycle and thus to enhance effectiveness

each cell cycle, the chemotherapy kills a fixed percentage of cells, always leaving some behind. With each reduction, the tumour burden of cells decreases until the number of viable, clonogenic cells (i.e. those that are able to clone daughter cells) becomes small enough to allow the body's immune system to finish the job. Oncologists usually give the maximum amount of chemotherapy tolerated by the person. High-dose chemotherapy remains controversial.

Classes of chemotherapy drugs Chemotherapeutic agents can be classified either by the effects of the agent on the cell or by the pharmacological properties of the agent. According to the effects of the agent on the cell, chemotherapeutic agents can be divided into cell-cycle-specific and cell-cycle-non-specific agents. Cell-cycle-specific agents are effective at a specific phase (e.g. S and M phases) in the cell cycle to prevent cell replication by damaging cellular DNA and blocking production of protein necessary for DNA and RNA synthesis. Cell-cycle-non-specific agents are effective throughout all the phases of the cell cycle, including the resting phase. Both cell-cycle-specific and cell-cycle-non-specific agents are effective in rapidly dividing cells to prohibit the growth of fast-growing tumours.

The most common way of classifying chemotherapeutic agents is based on pharmacological properties of the agent. The classifications include alkylating agents, antimetabolites, antitumour antibiotics, mitotic inhibitors, hormones and hormone antagonists, and miscellaneous agents.

Alkylating agents Alkylating agents are not phase specific and basically act on preformed nucleic acids by creating defects in tumour DNA. They cause crosslinking of DNA strands, which can permanently interfere with replication and transcription.

Alkylating agents work with both proliferating and non-proliferating cells (those in the G_0 phase). Their toxicity relates to their ability to kill slowly cycling stem cells and manifests in delayed, prolonged or permanent bone marrow failure. Toxicity can also cause a mutagenic effect on bone marrow stem cells, culminating in a treatment-resistant form of acute myelogenous leukaemia. Because of the alkylating agents' effect on stem cells, they also cause irreversible infertility. Other common adverse effects include nephrotoxicity and haemorrhagic cystitis.

The several subclasses of alkylating agents include nitrogen mustard (mechlorethamine), nitrosoureas (carmustine), alkyl sulfonates (busulfan), triazines (dacarbazine), ethyleneimines (thiotepa) and cisplatin. Cisplatin is an alkylating agent containing platinum and chlorine atoms. It is most active in the G_1 subphase, but it is also not phase specific. Cisplatin binds to DNA and acts much like alkylating agents by forming intrastrand DNA crosslinks (gluing strands of DNA together so that they cannot separate). Its major toxic effect is reversible renal tubular necrosis. Cisplatin may be used alone or in combination with other chemotherapeutic drugs for testicular and ovarian cancers.

Antimetabolites The different types of antimetabolites include folic acid analogues (methotrexate), pyrimidine analogues (5-fluorouracil), cytosine arabinoside (ARA-C) and purine analogues (6-mercaptopurine). Antimetabolites are phase specific, working best in the S phase and having little effect in G_0. They interfere with nucleic acid synthesis by either displacing normal metabolites at the regulatory site of a key enzyme or by substituting for a metabolite that is incorporated into DNA or RNA molecules. Toxic effects usually do not occur until very high levels of the drug are administered. Toxicity is also more likely when the drugs accumulate in third-spaced fluid, such as pleural fluid (a characteristic that also makes them useful in treating malignant pleural effusions). Because the drug diffuses out slowly from the third-spaced fluid, exposure of the tissue to the drug is prolonged. Most toxic effects relate to rapidly proliferating cells, such as cells in the gastrointestinal tract, hair and skin, and white blood cells (WBCs). Signs and symptoms include nausea and vomiting, stomatitis, diarrhoea, alopecia and leukopenia. Some of the drugs can also cause liver and pulmonary toxicity.

Antitumour antibiotics Antitumour antibiotics derived from natural sources that are generally too toxic to be used as antibacterial agents. They are not phase specific and act in several ways: they disrupt DNA replication and RNA transcription; create free radicals, which generate breaks in DNA and other

forms of damage; and interfere with DNA repair. These drugs bind to cells and kill them, probably by damaging the cell membrane. Their main toxic effect is damage to the cardiac muscle. This limits the amount and duration of treatment. Examples of these antibiotics include actinomycin D, doxorubicin, bleomycin, mitomycin-C and mithramycin.

Mitotic inhibitors Mitotic inhibitors are drugs that act to prevent cell division during the M phase. Mitotic inhibitors include the plant alkaloids and taxoids. Plant alkaloids consist of medications extracted from plant sources: vinca alkaloids (e.g. vincristine and vinblastine) and etoposide (also called VP-16). The vinca alkaloids are phase specific, acting during mitosis. They bind to a specific protein in tumour cells that promotes chromosome migration during mitosis and serves as a conduit for neurotransmitter transport along axons. The toxicity of these drugs is characterised by depression of deep tendon reflexes, paraesthesias (pain and altered sensation), motor weakness, cranial nerve disruptions and paralytic ileus. Etoposide acts in all phases of the cell cycle, causing breaks in DNA and metaphase arrest. Although etoposide may cause bone marrow suppression and nausea and vomiting, the most common toxic effect is hypotension resulting from too rapid intravenous administration.

The taxoids act during the G_2 phase to inhibit cell division. Paclitaxel is used for the treatment of Kaposi's sarcoma and metastatic breast and ovarian cancer. Taxotere is used for breast cancer. Toxicities associated with these drugs include alopecia, bone marrow depression and severe hypersensitivity reactions (e.g. hypotension, dyspnoea and urticaria).

Hormones and hormone antagonists The main hormones used in cancer therapy are the corticosteroids (e.g. prednisone), which are phase specific (G_1). These act by binding to specific intracellular receptors, repressing transcription of mRNA and thereby altering cellular function and growth. Corticosteroids have multiple side effects such as impaired healing, hyperglycaemia, hypertension, osteoporosis and hirsutism.

Hormone antagonists work with hormone-binding tumours, usually those of the breast, prostate and endometrium. They block the hormone's receptor site on the tumour and prevent it from receiving normal hormonal growth stimulation. These drugs do not cure, but do cause regression of the tumour in about 40% of breast and endometrial tumours, and 80% of prostate tumours. Tamoxifen competes with oestradiol receptors in breast tumours. Raloxifene blocks oestrogen in the breast. Diethylstilbestrol competes with hormone receptors in endometrial and prostate tumours. Antiandrogen (Flutamide) and luteinising-hormone-releasing hormones block testosterone synthesis in prostate cancers. The main side effects of these drugs are alterations of the secondary sexual characteristics.

Miscellaneous agents Several miscellaneous agents act at different phases in the cell cycle. L-Asparaginase and hydroxyurea are examples of miscellaneous agents.

Effects of chemotherapeutic drugs As described, the side effects and toxic effects of chemotherapy vary with the drug used and the length of treatment. Because most of these drugs act on fast-growing cells, the side effects are manifestations of damage to normal rapidly dividing somatic cells. The side effects of hormones express the action of the hormone used or suppression of the normal hormone, such as the masculinising effects of male hormones administered for ovarian cancers.

Tissues usually affected by cytotoxic drugs include the following:

- Mucous membranes of the mouth, tongue, oesophagus, stomach, intestine and rectum. This may result in anorexia, loss of taste, aversion to food, erythema and painful ulcerations in any portion of the gastrointestinal tract, nausea, vomiting and diarrhoea.
- Hair cells, resulting in alopecia.
- Bone marrow depression affecting most blood cells (e.g. granulocytes, lymphocytes, thrombocytes and erythrocytes). This results in an impaired ability to respond to infection, a diminished ability to clot blood and severe anaemia.
- Organs, such as heart, lungs, bladder, kidneys. This kind of damage is related to specific agents, such as cardiac toxicity with doxorubicin or pneumonitis with bleomycin.
- Reproductive organs, resulting in impaired reproductive ability or altered foetal development.

Table 13.10 gives the classifications of chemotherapeutic drugs, common examples, target malignancies, adverse effects and side effects, and nursing implications. Consult current pharmacology textbooks for additional drugs and for new combination therapies as they are developed.

Preparation and administration Australia has legislative Acts and regulations (including, but not limited to, *Occupational Health and Safety Act (OH&S) 2000*, OH&S Regulations 2001, *Poisons & Therapeutic Goods Act 1966*, *Cytotoxic Drugs & Related Waste: Risk Management Guide 2008* and Australian Radiation Protection and Nuclear Safety Agency—Radiation Protection Series Codes and Guides) that govern the handling, labelling, transport, waste disposal, documentation and the designation, training and employee health monitoring of staff engaged in duties with the potential for exposure to radiation. Organisations are required to have procedures in place to minimise risk to staff (environmental and personal protective equipment) when preparing, administering and disposing of chemotherapeutic drugs, and individuals are required to demonstrate duty of care and to follow legislative and organisational guidelines. Refer to your organisation's policy/procedures. It is the nurse's responsibility to ensure they are working within their NMBA (formerly ANMC) competency standards. The nurse is required to teach people to dispose of their own body fluids safely. Oral medications pose a lesser risk of exposure, but a risk nonetheless, primarily through excretion in the urine.

Chemotherapeutic drugs, such as cyclophosphamide and chlorambucil, can be administered orally. Other drugs, such as hormones or hormone-blocking agents, may also be given intramuscularly. However, many drugs require intravenous infusion or direct injection into intraperitoneal or intrapleural body cavities. Intravenous preparations can be given through large peripheral veins, but the risk of extravasation or

TABLE 13.10 Classifications of chemotherapeutic drugs

DRUG CLASSIFICATION	COMMON DRUGS	TARGET MALIGNANCIES	ADVERSE EFFECTS OR SIDE EFFECTS	NURSING IMPLICATIONS
Alkylating agents	Mechlorethamine	Hodgkin's disease Lymphosarcoma Lung cancer Chronic leukaemia	Nausea and vomiting Leucopenia Thrombocytopenia Hyperuricaemia	Maintain good hydration. Alkalinise urine. Administer anti-emetics prior to chemotherapy. Monitor WBC, uric acid. Assess for infection.
	Busulfan	Chronic myelogenous leukaemia	Leucopenia Thrombocytopenia Renal failure Pulmonary fibrosis	Monitor WBCs, BUN. Maintain adequate fluid intake. Assess for infection. Assess lungs for fibrotic (coarse, loud) rales.
	Cyclophosphamide	Lymphomas Multiple myeloma Leukaemias Adenocarcinoma of lung and breast	Haemorrhagic cystitis Renal failure Alopecia Stomatitis Liver dysfunction Infertility	Encourage daily fluid intake of 2 to 3 L during treatment. Monitor WBCs, BUN, liver enzymes. Teach ways to manage hair loss.
Antimetabolites	Methotrexate	Acute lymphoblastic leukaemia Osteosarcoma Gestational trophoblastic carcinoma	Oral and gastrointestinal ulcerations Anorexia and nausea Leucopenia Thrombocytopenia Pancytopenia	Monitor CBC, WBC differential, BUN, uric acid, creatinine. Assess oral mucous membranes; treat ulcers prn. Assess for infection, bleeding.
	5-Fluorouracil (5-FU)	Colon carcinoma Rectal carcinoma Breast carcinoma Gastric carcinoma Pancreatic cancer	Stomatitis Alopecia Nausea and vomiting Gastritis Enteritis Diarrhoea Anaemia Leucopenia Thrombocytopenia Red sore peeling hands and feet	Monitor CBC with differential, BUN, uric acid. Administer anti-emetics prn. Assess for bleeding; check stool occult blood. Evaluate hydration and nutrition status. Teach oral care for stomatitis. Assess for infection. Teach care for hair loss.
Antitumour antibiotics	Doxorubicin	Acute lymphoblastic leukaemia (ALL) Acute myeloblastic leukaemia Neuroblastoma Wilms' tumour Breast, ovarian, thyroid, lung cancer	Stomatitis Alopecia Nausea and vomiting Gastritis Enteritis Diarrhoea Anaemia Leucopenia Thrombocytopenia Cardiac toxicity	Monitor ECG; assess for arrhythmias, gallops and congestive heart failure (CHF). Monitor CBC with differential, BUN, uric acid. Administer anti-emetics prn. Assess for bleeding; check stool for occult blood. Evaluate hydration and nutrition status. Teach oral care for stomatitis. Assess for infection. Teach care for hair loss.
Antitumour antibiotics	Bleomycin	Squamous cell carcinoma Lymphosarcoma Reticulum cell sarcoma Testicular carcinoma Hodgkin's disease	Mucocutaneous ulcerations Alopecia Nausea and vomiting Chills and fever Pneumonitis and pulmonary fibrosis	Check for fever 3 to 6 hours after administration. Have chest x-ray films taken every 2 to 3 weeks. Assess respiratory status and check for coarse rales. Evaluate hydration and nutrition status. Teach oral care for stomatitis. Assess for infection. Teach care for hair loss.

(continued)

TABLE 13.10 Classifications of chemotherapeutic drugs (continued)

DRUG CLASSIFICATION	COMMON DRUGS	TARGET MALIGNANCIES	ADVERSE EFFECTS OR SIDE EFFECTS	NURSING IMPLICATIONS
Plant alkaloids	Vincristine	Combination therapy for acute leukaemia, Hodgkin's and non-Hodgkin's lymphomas, rhabdomyosarcoma, neuroblastoma, Wilms' tumour	Areflexia Peripheral neuritis Constipation Paralytic ileus Mild bone marrow Depression	Assess neuromuscular function. Monitor FBC with differential. Evaluate gastrointestinal function. Manage constipation.
	Vinblastine	Combination therapy for Hodgkin's disease, lymphocytic and histocytic lymphoma Kaposi's sarcoma, advanced testicular carcinoma, unresponsive breast cancer	Areflexia Alopecia Nausea and vomiting Bone marrow depression Tingling and numbness of fingers and toes	Assess neuromuscular function. Monitor FBC with differential. Administer anti-emetics prn. Teach ways to manage hair loss.
	Etoposide, also called VP-16	Non-responsive testicular tumours Small-cell lung cancer	Alopecia Hypotension with rapid infusion Facial flushing Sweating Difficulty breathing/ wheezing	Hydrate adequately before administration. Administer for 60 minutes. Monitor vital signs every 15 minutes during administration and every 2 to 4 hours thereafter. Teach ways to manage hair loss.
	Prednisolone	Combination therapy for many tumours Leukaemia Lymphoma	Fluid retention Hypertension Steroid diabetes Emotional lability Silent bleeding ulcers Increased risk of infection Euphoria or insomnia	Monitor vital signs. Administer diuretics prn. Check blood glucose regularly. Evaluate mental status. Administer oral medications with food. Administer hydrogen ion antagonist drugs (antacids) as ordered. Monitor WBC with differential. Check for signs of systemic infection.
	Diethylstilbestrol (DES)	Advanced breast and prostrate cancers	Fluid retention Feminisation Uterine bleeding	Monitor vital signs. Administer diuretics prn as ordered. Explain reason for feminisation to men, bleeding to women. Monitor for excessive bleeding.
	Tamoxifen	Breast cancer	Hot flashes Nausea and vomiting	Explain reason for hot flashes. Teach ways to manage hot flashes. Administer anti-emetics as ordered.
Miscellaneous drugs	Cisplatin (CDDP) (Platinol)	Combination and single therapy for metastatic testicular and ovarian cancers, advanced bladder cancer, head and neck tumours, non-small-cell lung carcinoma, osteogenic sarcoma, neuroblastoma	Bone marrow depression: leucopenia and thrombocytopenia Renal tubular damage Deafness	Monitor WBC with differential and platelets, BUN, creatinine, uric acid. Watch for bleeding. Monitor for signs of infection. Evaluate hearing; check for tinnitus. Ensure that person is well hydrated before drug is administered. Encourage 2 to 3 L of fluid intake daily.

irritation to the vein may preclude this method for long-term therapy. Many people now receive central venous access devices (CVADs), especially if their treatment requires several cycles over weeks or months. CVADs are also useful for adjunctive parenteral nutrition in the person who needs continuous intravenous infusions to manage pain or frequent blood drawing to monitor blood counts. Different types of CVADs are available:

- Catheters inserted non-surgically by threading them through a large peripheral vein into the vena cava. Called *peripherally inserted central catheters (PICCs)*, they have multiple lumens that facilitate blood drawing. Placement is usually monitored by fluoroscopy.
- Catheters tunnelled under the skin on the chest into a major vein, such as the subclavian vein. Hickman or Groshong catheters may be used.
- Surgically implanted ports, placed under the skin with a connected catheter inserted into a major vein. These are accessed by means of a special needle with a 90-degree angle inserted through the skin directly into the rubber dome of the port, which has a hard plastic back to prevent tissue damage.

Figure 13.6 shows examples of different catheters and vascular access ports.

Risk of infection, catheter obstruction and extravasation are the main problems associated with CVADs. Nurses therefore must teach people and family members to observe for redness, swelling, pain or exudate at the insertion site, which may indicate infection; to observe for swelling of the neck or skin near the CVAD for extravasation and infiltration; and to flush catheters and provide site care (cleaning and dressing changes) on a regular basis. During each encounter with the person, the nurse always inspects the site; observes for infection, infiltration and catheter occlusion, and provides site care when necessary.

Management of people receiving chemotherapy In addition to providing the above nursing interventions, nurses help identify and manage toxic effects or side effects of the drugs and provide psychosocial support. Careful assessment and monitoring of the person's signs and symptoms, including appropriate laboratory tests, alert the nurse to the onset of toxicity. Nausea and vomiting, diarrhoea, inflammation and ulceration of oral mucous membranes, hair loss, skin changes, anorexia and fatigue require specific medical and nursing actions. These actions are discussed later in this chapter under the appropriate nursing diagnoses. Indicators of organ toxicities, such as nephrotoxicity, neurotoxicity or cardiac toxicity, must be reported immediately to the doctor. Another aspect of managing people undergoing chemotherapy is to teach them how to care for access sites and to dispose of used equipment and excretions safely. Nurses also teach people to increase fluid intake to flush out the drugs; to get extra rest, which can both assist therapy and help the person avoid other illnesses; to identify major complications of their particular drug protocol; to know when to call the doctor or emergency medical services; and, if their WBC count is low, to limit their exposure to other people, especially children or those with infections.

During chemotherapy, a number of psychological issues that can cause moderate to severe emotional distress may arise. The need to plan activities around chemotherapy treatments and their side effects can impair the person's ability to work, manage a household or care for family members, function sexually or participate in social and recreational activities. Weight loss and alopecia may prompt feelings of powerlessness and depression. The nurse can assist by carefully evaluating

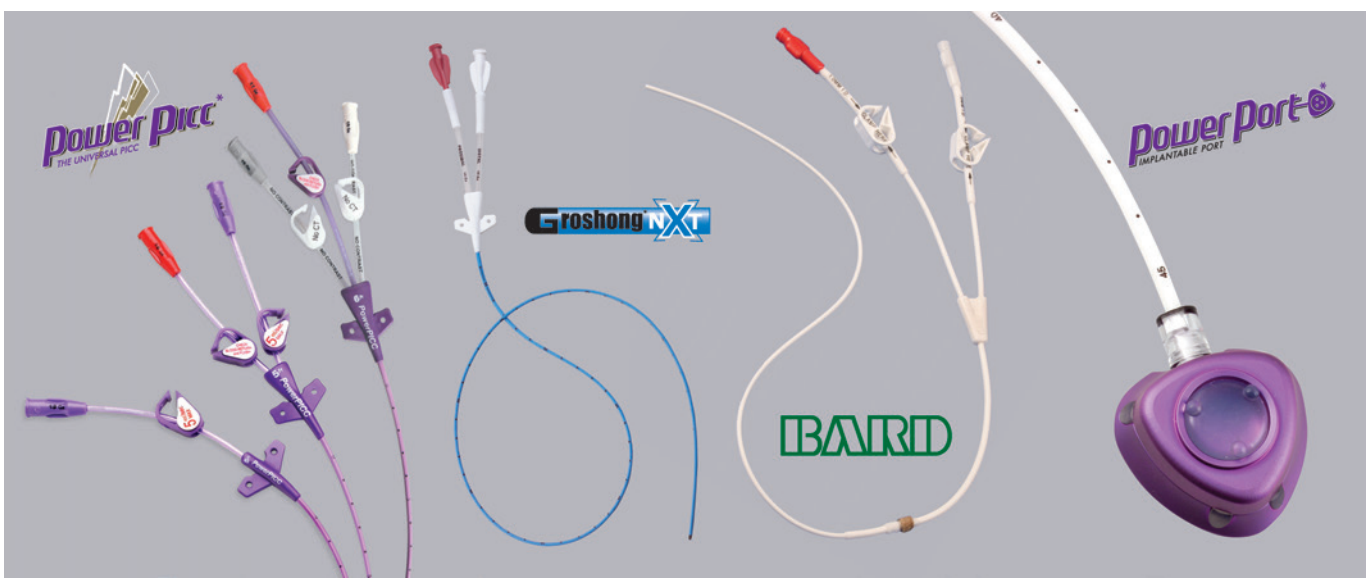


FIGURE 13.6 ■ Vascular access devices: peripherally inserted central catheters (single, dual and triple lumen PowerPICC and dual lumen Groshong), tunnelled central venous catheter (dual lumen Hickman) and implantable port (PowerPort)

Source: Photos courtesy of Bard Access Systems, Salt Lake City, UT.

symptoms, providing specific interventions as indicated and allowing people opportunities to express their fears, concerns and feelings. People should be encouraged to participate in their care and maintain control over their life as much as possible. Specific interventions are discussed later in the chapter under the appropriate nursing diagnoses. Table 13.10 includes nursing implications for specific adverse effects of common chemotherapy drugs.

RADIATION THERAPY Still the treatment of choice for some tumours or by some oncologists, radiation may be used to kill the tumour, reduce its size, decrease pain or relieve obstruction. Lymph nodes and adjacent tissues are irradiated when beginning metastasis is suspected. **Radiation therapy** consists of delivering ionising radiations of gamma and x-rays in one of two ways:

- *External radiation*, also known as teletherapy, involves delivery of radiation from a source at some distance from the person. A relatively uniform dosage is delivered to the tumour.
- *Brachytherapy*. In brachytherapy, the radioactive material is placed directly into or adjacent to the tumour, a technique that delivers a high dose to the tumour and a lower dose to the normal tissues. This allows delivery of high doses of radiation to the tumour while sparing adjacent tissue. Brachytherapy is also referred to as internal, interstitial or intracavitary radiation.

For many common neoplasms, a combination of these two therapies is used.

Lethal injury to DNA is believed to be the primary mechanism by which radiation kills cells, especially cells in faster-growing tumours and tissues. As a result, when given over time, radiation can destroy not only rapidly multiplying cancer cells but also rapidly dividing normal cells, such as those of the skin and mucous membranes. A malignant tumour is considered cured when there are no surviving tumour stem cells. The goal of radiation therapy is to achieve maximum tumour control with a minimum of damage to normal tissue.

Implanted or ingested radiation can be dangerous for those living with, taking care of or treating the person. Caregivers must use protection by shielding themselves from the source of radiation, limiting the time of exposure to the person, increasing the distance from the person and using specific safety procedures for handling secretions. Box 13.6 identifies safety principles to be followed by those caring for people undergoing internal radiation.

Tumours have differing sensitivities to radiation. Tumours that have the greatest number of rapidly proliferating cancer cells usually exhibit the best early response to radiation. The decision to use radiation rather than other modalities is based on balancing the probability of controlling the tumour against the probability of causing complications, such as tissue damage. The decision is usually made by risk–benefit analysis. Planning for radiation therapy includes assessing the disease site, tumour size and histological findings. Treatment schedules vary based on these factors.

BOX 13.6 General safety principles for radiation

These general procedures are taken from the *Code of Nursing Practice for Staff Exposed to Ionising Radiation* (Australian Radiation Protection and Nuclear Safety Agency, 1984):

- Maintain maximum distance from source of radiation (2.3) and work at a maximum practical distance when attending to people (4.1.5).
- Reduce time spent near a radioactive source (2.4).
- A single room should be used to minimise exposure to staff and other people (4.1.1).
- Nursing staff carrying out procedures with possibility of contamination should wear suitable gloves and gowns (4.6.5).
- It is preferable to assign staff who are unlikely to be pregnant (1.3).
- Staff should be subject to monitoring by personal monitors or an approved device. Records should be kept and available upon request (1.30).
- Nursing staff should never touch a radioactive source with the fingers (4.5.3). Special forceps or other remote control equipment should be used to handle radioactive sources (5.4).
- Specific precautions should be used to reduce risk of spill of body fluids that are radioactive (4.6.7).
- Handle bed linen and clothing with care and according to the organisation's protocol.
- Consult the organisation's radiation therapy department for any questions or problems in caring for people with radioactive implants.

Box 13.7 lists the degree of radiosensitivity for selected cancers.

The person receiving external radiation may experience skin changes such as blanching, erythema, desquamation, sloughing or haemorrhage. Ulcerations of mucous membranes may cause severe pain; in addition, oral secretions can decrease, making the person more vulnerable to infection and dental caries. Gastrointestinal effects include nausea and vomiting, diarrhoea or bleeding. Lungs may develop interstitial exudate, a condition called radiation pneumonia. Occasionally, external radiation therapy may cause fistulas or necrosis of adjacent tissues. Implanted radioactive materials can lead to similar problems; moreover, the excretory products of these people are usually considered dangerous and need special disposal. See the 'Nursing care' box below for nursing implications for people receiving radiation therapy.

BIOTHERAPY **Biotherapy** modifies the biological processes that result in malignant cells, primarily through enhancing the person's own immune responses. The development of this therapy was based on the immune surveillance hypothesis. Although it has been established that a competent immune system is the body's most important defence against any

BOX 13.7 Degree of radiosensitivity for selected cancers

Very radiosensitive
- Neuroblastoma
- Lymphomas
- Chronic leukaemia

Moderately radiosensitive
- Bronchogenic carcinoma
- Oesophageal carcinoma
- Squamous cell carcinoma
- Prostate carcinoma
- Cervical carcinoma
- Testicular carcinoma

Non-radiosensitive
- Many adenocarcinomas
- Fibrosarcoma
- Osteogenic carcinoma

disease, the role that various immune cells play in combatting different types of malignancies continues to be investigated. Currently, biotherapy is used for both haematological malignancies, such as lymphoma and hairy-cell leukaemia, and solid tumours, such as renal cancer and melanoma (Fox et al., 2013; Poust, Woolery & Green, 2013).

Tumour immunology has the following applications: detection screening in high-risk groups, differential diagnosis and classification of tumour cells, monitoring the course of the disease, with early detection of recurrence and active therapies to halt or limit the disease. The theory underlying tumour immunology is that most tumour cells have a structural appearance recognisable by the immune cells. Tumour-associated antigens (TAAs) exist on tumour cells but not on normal cells. TAAs elicit an immune response that, in a person with a competent immune system, destroys or inhibits tumour growth. Thus, TAAs can be isolated from serum and used for both diagnosis and various treatment modalities. The PSA is one such TAA currently in successful diagnostic use.

Tumour cells are often in a stage of arrested development (i.e. in the differentiation stage) for the cell type they represent; thus, they express antigens characteristic of that particular stage of development. The immaturity of the cells provides the doctor with information about the relative aggressiveness of the cancer.

Another aspect of immunotherapy is the development of monoclonal antibodies that enhance the immune system's ability to fight the cancer. Monoclonal antibodies are developed by inoculating an animal with the tumour antigen and recovering the specific antibodies produced. The antibodies are then given to the person with that cancer to assist in the destruction of the tumour. Monoclonal antibodies are also recreated or cloned in the genetic laboratory by recombining DNA to produce the specific antibody. Techniques involving recombinant DNA have been used to combine these antibodies with toxins and drugs that are then delivered selectively to the tumour sites.

A number of cytokines (normal growth-regulating molecules) with antitumour activity have been synthesised. Alpha interferon (IFN-α), bacillus Calmette-Guérin (BCG, which has been used for many years as an inoculation against tuberculosis) and interleukin-2 (IL-2) have shown some therapeutic benefit in eliciting increased immune responses. Combination strategies have also helped stimulate the function of macrophages.

A promising discovery has been the natural killer (NK) cells. These cells are like large granular lymphocytes, but have a cell surface phenotype different from that of T lymphocytes or macrophages. They have demonstrated a spontaneous cytotoxic effect on some types of cancer cells. They also provide a strong resistance to metastasis and secrete cytokines. When augmented by biological response modifiers such as IL-2, they show increased tumour destructive activity (Battiato & Wheeler, 2000).

The use of haematopoietic growth factors (HGFs) has been one of the most successful in biotherapy. HGFs, such as granulocyte colony-stimulating factor and erythropoietin, offset the suppression of granulocytes and erythrocytes that results from chemotherapy (Battiato & Wheeler, 2000).

Since the early 1990s, the combination of cytokines, particularly IFN-α and IL-2, with chemotherapy has been used in people with metastatic melanoma with promising results. Such a combination is referred to as either biochemotherapy or chemoimmunotherapy (Anderson et al., 1998; Cohen & Falkson, 1998; Legha et al., 1998). The rationale for biochemotherapy is based on the independent antitumour activity of both IFN-α and IL-2 against melanoma and their lack of cross-resistance with cytotoxic chemotherapy. Although the precise mechanism of the antitumour effect of biochemotherapy regimens is less well understood, two hypotheses have been proposed: (1) chemotherapy enhances the antitumour effect of biological agents; and (2) the biological agents enhance the antitumour cytotoxic effect of chemotherapy (Anderson et al., 1998; Legha et al., 1998).

As promising as these biotherapies or biochemotherapies are, they are accompanied by serious side effects and toxicities (Fu et al., 2002; Legha et al., 1998). IL-2 can cause acute alterations in renal, cardiac, liver, gastrointestinal and mental functioning. IFN-α causes mental slowing, confusion and lethargy and when used in combination with 5-fluorouracil or IL-2, severe flu-like symptoms—chills and fever of 39.4ºC to 41.1ºC, nausea, vomiting, diarrhoea, anorexia, severe fatigue and stomatitis—may result. The toxic effects are probably exaggerations of the normal systemic effects that these substances cause when fighting infection. For example, IL-2 is known to raise body temperature substantially in an attempt to create a hostile environment for foreign invaders.

The 'Nursing care' box below discusses nursing implications for people receiving immunotherapy. For nursing care of specific problems, refer to the appropriate nursing diagnoses later in this chapter.

NURSING CARE OF THE PERSON Receiving radiation therapy

NURSING RESPONSIBILITIES FOR EITHER EXTERNAL OR INTERNAL RADIATION THERAPY

- Assess and manage any complications, usually in collaboration with the radiation oncologist.
- Assist in documenting the results of the therapy; for example, people receiving radiation for metastases to the spine will show improved neurological functioning as tumour size diminishes.
- Provide emotional support, relief of physical and psychological discomfort, and opportunities to talk about fears and concerns. For some people, radiation therapy is a last chance for cure or even just for relief of physical discomfort.

EXTERNAL RADIATION

Prior to the start of treatments, the treatment area will be specifically located by the radiation oncologist and marked with coloured semi-permanent ink or tattoos. Treatment is usually given 5 days per week for 15 to 30 minutes per day over 2 to 7 weeks.

Nursing responsibilities

- Monitor for adverse effects: skin changes, such as blanching, erythema, desquamation, sloughing or haemorrhage; ulcerations of mucous membranes; nausea and vomiting, diarrhoea or gastrointestinal bleeding.
- Assess lungs for rales, which may indicate interstitial exudate. Observe for any dyspnoea or changes in respiratory pattern.
- Identify and record any medications that the person will be taking during the radiation treatment.
- Monitor white blood cell counts and platelet counts for significant decreases.

Health education for the person and family

- Wash the skin that is marked as the radiation site with plain water only, no soap; do not apply deodorant, lotions, medications, perfume or talcum powder to the site during the treatment period. Take care not to wash off the treatment marks.
- Do not rub, scratch or scrub treated skin areas. If necessary, use only an electric razor to shave the treated area.
- Apply neither heat nor cold (e.g. heating pad or ice pack) to the treatment site.
- Inspect the skin for damage or serious changes and report these to the radiologist or doctor.
- Wear loose, soft clothing over the treated area.
- Protect skin from sun exposure during treatment and for at least 1 year after radiation therapy is discontinued. Cover skin with protective clothing during treatment; once radiation is discontinued, use sun-blocking agents with a sun protection factor (SPF) of at least 15.
- External radiation poses no risk to other people for radiation exposure, even with intimate physical contact.
- Be sure to get plenty of rest and eat a balanced diet.

INTERNAL RADIATION

The radiation source, called an implant, is placed into the affected tissue or body cavity and is sealed in tubes, containers, wires, seeds, capsules or needles. An implant may be temporary or permanent. Internal radiation may also be ingested or injected as a solution into the bloodstream or a body cavity or be introduced into the tumour through a catheter. The radioactive substance may transmit rays outside the body or be excreted in body fluids.

Nursing responsibilities

- Place the person in a private room.
- Limit visits to 10 to 30 minutes and have visitors sit at least 2 metres from the person.
- Monitor for side effects such as burning sensations, excessive perspiration, chills and fever, nausea and vomiting, or diarrhoea.
- Assess for fistulas or necrosis of adjacent tissues.

Health education for the person and family

- While a temporary implant is in place, stay in bed and rest quietly to avoid dislodging the implant.
- For outpatient treatments, avoid close contact with others until treatment has been discontinued.
- If the radiologist indicates the need for such measures, dispose of excretory materials in special containers or in a toilet not used by others.
- Carry out daily activities as able; get extra rest if feeling fatigued.
- Eat a balanced diet; frequent, small meals often are better tolerated.
- Contact the nurse or doctor about any concerns or questions after discharge.

PHOTODYNAMIC THERAPY Photodynamic therapy is a method of treating certain kinds of superficial tumours. It is known by several different names: phototherapy, photoradiation and photochemotherapy. People suffering from tumours growing on the surface of the bladder, peritoneal cavity, chest wall, pleura, bronchus or head and neck are candidates for this treatment. The person is given an intravenous dose of a photosensitising compound, Photofrin, which is selectively retained in higher concentrations in malignant tissue. This drug is activated by a laser treatment that is started 3 days after the drug injection and administered for 3 days. The drug interacts with oxygen molecules in the tissue to produce a cytotoxic oxygen molecule called singlet oxygen.

At the time of the first intravenous injection, people are observed for adverse hypersensitivity reactions, such as nausea, chills and hives. Systemic or long-term toxicities are rare. The main side effects are local skin reactions and temporary photosensitivity, transiently elevated liver enzymes and inflammatory responses of the tissues being treated, such as peritoneal or pleural tissues. This treatment has been used successfully with early-stage lung cancer with response rates as high as 90% (Bruce, 2001).

The major nursing responsibilities associated with photodynamic therapy are to address the person and family's anxiety about undergoing a relatively new treatment procedure and

NURSING CARE OF THE PERSON Receiving immunotherapy

Immunotherapy can consist of various substances used alone, such as IL-2, or combination biotherapy, such as IFN-α with 5-fluorouracil. The nurse's role is to enhance the person's quality of life.

NURSING RESPONSIBILITIES

- Monitor for side effects: IFN-α may cause mental slowing, confusion and lethargy; combination therapy of 5-fluorouracil or IL-2 and IFN-α may cause severe flu-like symptoms, with chills and fever of 39.4°C to 41.1°C, nausea, vomiting, diarrhoea, anorexia, severe fatigue and stomatitis; erythropoietin may cause acute hypertension.
- Monitor enzymes and other appropriate biochemical indicators for acute alterations in renal, cardiac, liver or gastrointestinal functioning, which can be side effects of IL-2.
- Evaluate response to therapy by conducting a thorough evaluation of the person's symptoms.
- Assess the person's coping behaviours and teach new strategies as needed.
- Manage fatigue and depression.
- Encourage self-care and participation in decision making.
- Provide close supervision for people with altered mental functioning, either by caretakers or through frequent nursing visits to the person's home.
- If the person is unable to manage alone, teach medication administration and care of equipment to caregivers.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Minimise symptoms by managing fever and flu-like symptoms: increase fluid intake, take analgesic and antipyretic medications, and maintain bed rest until symptoms abate.
- Seek help for serious problems not managed by usual means, such as dehydration from diarrhoea.
- Use correct techniques for providing subcutaneous injections.
- Identify how to work and care for ambulatory pumps when medication is administered through an intercatheter or vascular access device.

to educate them in managing side effects. The drug remains in the subcutaneous tissues for 4 to 6 weeks after injection. Any direct or indirect exposure to the sun activates the drug, resulting in a chemical sunburn. People are taught to protect themselves from sunlight (even on cloudy days) by covering themselves from head to toe in opaque clothing, including a wide-brimmed hat, gloves, shoes and stockings, and sunglasses with 100% ultraviolet block. Long-term care of treated skin includes moisturising lotions and protection from trauma or irritation.

BONE MARROW AND PERIPHERAL BLOOD STEM CELL TRANSPLANTATIONS Bone marrow transplantation (BMT) is an accepted treatment to stimulate a non-functioning marrow or to replace marrow. BMT is given as an intravenous infusion of bone marrow cells from donor to person. Most commonly used in leukaemias, this therapy is being expanded to include treatment of other cancers including melanoma and testicular cancer. Chapter 32 provides an in-depth discussion of this procedure. Peripheral blood stem cell transplantation (PBSCT) is the process of removing circulating stem cells from the peripheral blood through apheresis and returning these cells to the person after dose-intensive chemotherapy. PBSCT has fewer side effects, shorter hospitalisation and decreased cost compared to BMT.

COMPLEMENTARY THERAPIES Although advances in cancer treatment have increased 5-year survival rates, the uncertainty of cure of cancer and cancer reoccurrence often compel some people to look for complementary therapies. It is estimated that approximately 30% to 50% of people with cancer may have had the experience of using some kind of complementary therapy. Complementary therapies are those that people choose as a complement to medical treatment. Common complementary therapies for cancer can be categorised into botanical agents, nutritional supplements, dietary regimens, mind–body modalities, energy healing, spiritual approaches and miscellaneous therapies. Box 13.8 provides detailed information about complementary therapies.

To provide sensitive nursing care, nurses should be knowledgeable about common complementary therapies. Nurses should use ethical principles of autonomy, beneficence, non-maleficence and justice to guide their professional practice and care for people who choose to use complementary therapies. It is also important for nurses to provide truthful, non-judgmental responses to the questions or inquiries about complementary therapies from people with cancer. Nurses should encourage people to report the use of any complementary therapies to their oncologist to prevent potential interactions of the complementary therapies with their medical treatment.

Pain management

Pain management is an important component of oncology care and is considered a crucial part of the collaborative treatment plan. It is estimated that more than 50% of people with early-stage cancer and up to 95% of people with advanced cancer experience pain that requires analgesia (Ogboli-Nwasor et al., 2013). There are three main categories of pain syndromes in people with cancer, and the category influences the type of treatment:

1. *Pain associated with direct tumour involvement*. The most common causes are metastases to bone, nerve compression or infiltration, and involvement of hollow visceral organs.
2. *Pain associated with treatment*. This may include postsurgical incisional or wound pain; peripheral neuropathy, ulceration of mucous membranes and pain from herpes zoster outbreaks secondary to chemotherapy; and pain in nerve plexuses, muscles and peripheral nerves from radiation therapy.
3. *Pain from a cause not related to either the cancer or therapy,* such as diabetic neuropathy.

BOX 13.8 Common complementary therapies for cancer

Type	Description
Botanical agents	Herbs are believed to be the most 'natural' and 'safe' plants ingested with the hope for a cure of cancer. Commonly used botanical agents include echinacea, essiac, ginseng, green tea, pau d'arco and hoxsey. The safety of many of these botanical agents has not been proven, especially as a complement to medical treatment.
Nutritional supplements	Chemical compounds include vitamins, minerals, enzymes, amino acids and essential fatty acids or proteins (such as shark cartilage). They are believed to have the ability to promote health and to help cure cancer. The safety of certain compounds such as vitamins has been established; however, in megadoses, many of the compounds can be toxic and have potential interactions with some therapeutic agents used for cancer, such as chemotherapy.
Dietary regimens	The ingestion of only natural substances is believed to have the effect of purifying the body and slowing down the growth of cancer. Popular regimens include the grape diet, the carrot juice diet and garlic, onions and liver intake. However, the effectiveness of these dietary regimens remains to be established.
Mind–body modalities	The harmony of mind and body is believed to facilitate physiological and psychological healing. Such modalities include relaxation, meditation or imagery. Recent research has shown that these modalities have helped individuals with cancer adjust to the experience of cancer.
Energy healing	The human body is believed to be an energy field and cancer might be the result of a disturbed energy field. Energy therapies, such as therapeutic touch and healing touch, can affect the energy field of the human body and promote physiological healing. Therapeutic touch uses the hands on or near the body with the intent to promote healing. Healing touch uses energy healing techniques to heal by restoring the harmony and balance of the body. Clinical practice and research on energy healing have shown positive findings of energy healing in a variety of people.
Spiritual approaches	Faith in God or a higher power of the universe is believed to help cancer healing. Spiritual approaches include faith healing, prayer to God, prayer groups and chain prayer. Research has shown that faith in God or a higher power also helped individuals with cancer to adjust to the experience of cancer.
Miscellaneous therapies	Aromatherapy has been used for people with cancer to relieve nausea, vomiting or retching, and to decrease anxiety. However, aromatherapy might not be appropriate for people who are highly sensitive to strong fragrance. Music, art and humour therapies have also been used to help people with cancer to reduce anxiety, to express feeling of loss and to promote optimism.

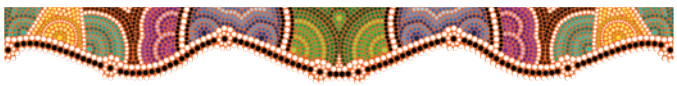
The goal of pain therapy is to provide relief that allows people to function as they wish and, in the case of terminally ill people, to die relatively free of pain. Drug therapy with opioid and non-opioid analgesics as well as adjuvant medications (those that enhance the effect of the analgesic) is the basis of most doctor-guided pain management. Other therapies include injection of anaesthetic drugs into spinal cord or specific nerve plexuses, surgical severing of nerves, radiation to reduce tumour size and pressure, and behavioural approaches. Pharmacological pain management follows these steps:

- Conduct careful initial and ongoing assessment of the pain.
- Evaluate the person's functional goals.
- Establish a plan with combinations of non-narcotic drugs (such as aspirin or ibuprofen) with adjuvants (such as corticosteroids or antidepressants).
- Evaluate the degree of pain relief.
- Progress to stronger drugs as needed, from mild narcotics such as oxycodone or propoxyphene to strong narcotics such as morphine or hydromorphone, and monitor side effects.
- Continue to try combinations and escalate dosages until maximal pain relief balanced with the person's need to function is achieved.

Medication is usually administered by the oral route as long as this route continues to be effective. Medication is given on a regular time schedule (e.g. every 4 hours), with additional medication prescribed to cover breakthrough pain. When the oral route alone becomes inadequate, the primary narcotic can be administered intramuscularly, subcutaneously or rectally on an intermittent schedule or continuously by transdermal patches, or intravenously by a continuous drip, usually controlled by an infusion pump. Some pumps are portable, deliver medication continuously and allow people to control their breakthrough pain with a limited number of boluses. When narcotic doses are increased gradually, there is no limit to the amount the person can receive, as long as adverse reactions can be managed. People have received up to 4800 mg daily (200 mg per hour) of morphine sulfate with up to six 200–400 mg breakthrough doses daily without major ill effects and with good pain control. The body develops tolerance to the sedative after a short period and most people are able to tolerate the level of medication needed to control the pain. Other side effects, such as constipation, nausea and vomiting, and itching, can be managed through the usual means and are discussed under the appropriate nursing diagnoses. If the person has persistent untoward

side effects that do not respond to treatment, or if the person does not get adequate relief from the narcotic, different narcotics and combinations are tried. Morphine sulfate and transdermal fentanyl are the most commonly used drugs for relief of cancer pain (Ferrell & McCaffery, 1997).

People receiving high-dose narcotics should not have the medication abruptly stopped, because withdrawal symptoms will occur. If the drug needs to be stopped, it must be tapered gradually. For more information on pain management, and on alternative therapies in particular, see Chapter 8.



Nursing care

Nurses face a major challenge in educating people about preventive measures and lifestyle changes to reduce the risk of cancer. At the same time, people with cancer must be reassured that they are not responsible for having acquired cancer.

Once a cancer diagnosis is established, nurses help people recover and support them during the rehabilitation phase. In cases of terminal cancer, nurses provide comfort and facilitate positive growth for the person and significant others.

Health promotion

Early detection and treatment are considered the most important factors influencing the prognosis of those who have cancer. However, many people do not seek early diagnosis and treatment because of denial, fear and anxiety, stigma or the absence of specific early signs such as pain or weight loss (which usually are late signs). For this reason, screening procedures such as mammograms, PSA, occult blood stool tests and sigmoidoscopy may be life saving.

Cancer Council Australia (CCA) advises the Australian government on policy and practices to help in the prevention, detection and treatment of cancer. CCA undertakes and funds cancer research as well as advocates for the rights of people with cancer to receive the best treatment, care and support. Recommendations on reducing preventable risk factors and early screening strategies are outlined in the *National Cancer Prevention Policy 2007–2009* by Cancer Council Australia (2007). The CCA highlights that one-third of cancer deaths in Australia can be attributed to known avoidable risk factors (CCA, 2007). Cancer Council Australia provides cancer-smart lifestyle fact sheets that provide information on common signs and symptoms that may indicate cancer. The signs and symptoms to look for (see Box 13.9) are helpful in promoting awareness but do not substitute for medical advice.

If a person is at special risk due to heredity, environment, occupation or lifestyle, special tests or more frequent examinations may be necessary. A routine cancer check-up should include counselling to improve health behaviours and physical examination with related tests of the breast, uterus, cervix, colon, rectum, testes, prostate, skin, thyroid and lymph nodes. Box 13.10 lists the recommendations for screening.

BOX 13.9 Cancer Council Australia: signs and symptoms to look for

- Lumps, sores or ulcers that do not heal.
- Coughs that persist or show blood, or a hoarseness that hangs around.
- Unexplained weight loss.
- Moles that have changed shape, size or colour or an inflamed skin sore that has not healed.
- Blood in a bowel motion.
- Persistent changes in toilet habits.

Men

- Unusual changes in your testicles—changes in shape, consistency or lumpiness.
- Urinary problems or changes.

Women

- Unusual changes in your breasts—lumps, thickening, unusual discharge, nipples that suddenly turn inwards, change in shape, colour or unusual pain.
- Blood loss between or following periods.
- Persistent abdominal pain or bloating.

These symptoms are often related to more common, less serious health problems. However, if you notice any unusual changes, or these symptoms persist, visit your doctor.

Source: Cancer Council Australia (2012). *Fact Sheets.* www.cancer.org.au.

Nurses have a special role in public education and should encourage all with whom they come into contact to schedule cancer check-ups. Nurses must be familiar with the cancer guidelines so that they can advise people, their families and significant others.

BOX 13.10 National Cancer Prevention Policy: cancer screening recommendations

Cervical screening

- Routine Pap tests from the age of 18, or within 1 to 2 years of becoming sexually active.

Breast cancer

- If you are over 40 you can have a free BreastScreen Australia mammogram (breast x-ray) every 2 years.
- Regular mammograms can reduce your risk of breast cancer death by 25%, particularly women in the 50–69 age group for whom the benefit is highest.

Bowel cancer

- Cancer Council Australia recommends all Australians aged over 50 have a faecal occult blood test (FOBT) every 2 years to screen for bowel cancer.

Source: Cancer Council Australia (2013a). *Early detection of breast cancer*. Retrieved from www.cancer.org.au/health-professionals/patient-factsheets/, 8 January 2013. For more information visit www.cancer.org.au.

Assessment

Focused interview

During this initial phase of the nursing process, collect the following significant data about the person:

- history of the person's disease, including the signs and symptoms that led the person to seek healthcare
- other concurrent diseases, such as diabetes
- current physical or psychological problems resulting from the cancer, such as pain or depression
- understanding of the treatment plan
- expectations of the treatment plan
- functional limitations due to illness or treatment (see Box 13.11)
- effect of the disease on current lifestyle
- reliable support systems or caretakers for the person
- coping strategies and how well they are working.

INTERVIEW QUESTIONS The following are appropriate questions to ask the person during the initial interview and at subsequent assessments:

- *'What brought you in to see the doctor?'* Asking this question allows people to tell their story in their own way, which may elicit more information than asking specific questions. The answer should elicit not only data about the signs and symptoms but also fears or concerns. If the cancer was discovered during a routine physical examination or check-up, the person may have some difficulty accepting the disease, especially if there were no symptoms. For people who offer insufficient information in response to this open-ended question, more specific questions may be necessary, such as 'Did you have pain or any specific physical problems that caused you to seek healthcare?'
- *'Do you have any other medical conditions or problems that are troubling you at this time?'* It may be necessary to ask about specific diseases to help the person focus. For example, 'Do you have high blood pressure?' or 'Are you having any problems with your lungs?' Information gained from these questions can help you anticipate problems and formulate potential nursing diagnoses related to other diseases that may interact with the cancer.
- *'What kinds of physical problems are you having at this time? Do you have pain? Are you nauseated? Have you lost a great deal of weight? Are you so tired you have difficulty carrying on your daily activities? Are you feeling depressed or discouraged because of your illness?'* For each positive response, ask follow-up questions to narrow down or define the exact nature of the problem. Again, these data help identify what nursing diagnoses should be included in the care plan.
- *'What options has your doctor suggested for treating your cancer?'* The answer will indicate the person's knowledge about their treatment and, possibly, their communication with the doctor. Often, under the stress of a cancer diagnosis, people do not hear or understand what the doctor is saying and are afraid to ask questions. Lack of knowledge indicates a need to collaborate with the doctor to explain the information to the person so that the person can absorb and understand it. If the person has a good understanding of the treatment plan, discussing how he or she feels about it can be useful in exposing fears, concerns and emotional responses.
- *'What do you expect to happen as a result of this treatment?'* The answer may reveal unrealistic expectations or lack of understanding of consequences of the treatment.
- *'What effect is the disease and/or treatment having on your ability to carry on with your usual daily activities?'* Additional questions may also be needed to pinpoint the types of limitations. The response to this question should provide information on the person's functional status, such as those shown in Box 13.11. This information can also be used to identify the need to collaborate with professionals from other disciplines. For example, if the person is the sole financial support of the family and is unable to work, a social worker may be able to help with resources; if the person is extremely weak, referral to a physiotherapist may help with energy conservation strategies and strengthening exercises.

BOX 13.11 Example of an instrument for assessing functional status for cancer patients (Australia-modified Karnofsky Performance Scale (AKPS))

100	Normal; no complaints; no evidence of disease.
90	Able to carry on normal activity; minor signs or symptoms of disease.
80	Normal activity with effort; some signs or symptoms of disease.
70	Cares for self; unable to carry on normal activity or to do active work.
60	Able to care for most needs but requires occasional assistance.
50	Considerable assistance and frequent medical care.
40	In bed more than 50% of the time.
30	Almost completely bedfast.
20	Totally bedfast and requiring extensive nursing care by professionals and/or family.
10	Comatose or barely rousable.
0	Dead.

Source: Abernethy et al. (2005). The Australia-modified Karnofsky Performance Status (AKPS) Scale: A revised scale for contemporary palliative care clinical practice [electronic version]. *BioMed Central Palliative Care*, *4*(7), 1–12, Table 1. http://creativecommons.org/licenses/by/2.0/.

- *'Who is available to help you at home and run errands for you? Who can provide transportation for you to get to your appointments or treatments? Who can you rely on to be a good listener when you're sad or just to be a comfortable companion? Is there someone you would like to make healthcare decisions for you if there is a time when you are unable to make them for yourself?'* It often seems that the person with cancer is the one who takes care of everyone else; asking for help may be difficult for this person. This information can identify how much support and help the person has access to. The last question introduces the concept of advance directives and enduring power of attorney regarding healthcare (see Chapter 4).
- *'How do you manage your stress or your feelings of discomfort? What helps you feel better? Do you think these measures work well for you?'* The responses to these questions provide information about the person's coping strategies and may identify maladaptive strategies such as alcohol or drug use. Lack of appropriate coping methods can interfere with the person's response to treatment and decrease overall quality of life.

Other assessment questions may be useful at different stages of the person's illness. For example, if the person is not expected to survive the cancer, it is important to ask whether the person has made decisions about last wishes (e.g. for a funeral and burial), whether these have been discussed with significant others and whether the person has made out a will.

Physical assessment

As soon as the person is admitted to the healthcare service or organisation, conduct a complete physical assessment to establish a baseline against which to evaluate later changes. It is especially important to document the nutritional status of the person using anthropomorphic measurements (i.e. frame size, height, weight, body fat and muscle mass) and to evaluate laboratory results and note any specific signs and symptoms. Table 13.11 compares the manifestations of good nutrition with those of malnutrition.

BOX 13.12 Factors to consider in assessing hydration status

- Intake and output
- Rapid weight changes
- Skin turgor and moisture
- Venous filling
- Vital sign changes
- Tongue furrows and moisture
- Eyeball softness
- Lung sounds
- Laboratory values

It is also important to assess the person's hydration status, especially if the person is not taking oral food and fluids well or is having bouts of vomiting. Box 13.12 lists specific assessments for hydration status. Other recommended assessments are discussed under the specific nursing diagnoses that follow. They can also be found in other chapters that address specific body systems affected by the cancer.

Nursing diagnoses and interventions

Nursing goals focus on supporting the whole person and managing specific problems such as pain, poor nutrition, dehydration, fatigue, adverse emotional responses, altered individual and family coping, and the side effects of medical treatment. Nursing also focuses on improving the quality of life by promoting rehabilitation for survivors of cancer and helping those who succumb to the disease maintain their dignity in the dying process. Because cancer affects the whole family, nursing care includes everyone involved with the person from the onset of diagnosis through the entire disease and treatment process and the ultimate outcome. Many diagnoses are pertinent to people with cancer; this section addresses only the most common diagnoses. See the accompanying 'Nursing care plan'. (Diagnoses specific to individual diseases can be found in their respective chapters.)

TABLE 13.11 Signs of nutritional status

SYSTEM	GOOD NUTRITION	POOR NUTRITION
General	Alert, energetic, good endurance, psychologically stable Weight within range for height, age, body size	Withdrawn, apathetic, easily fatigued, irritable Over- or underweight
Integumentary	Skin glowing, good turgor, smooth, free of lesions Hair shiny, lustrous, minimal loss	Skin dull, pasty, scaly-dry, bruises, multiple lesions Hair brittle, dull, falls out easily
Head, eyes, ears, nose and throat	Eyes bright, clear, no fatigue circles Oral mucous membranes pink-red and moist Gums pink, firm Tongue pink, moderately smooth, no swelling	Eyes dull, conjunctiva pale, discolouration under eyes Oral mucous membranes pale Gums red, spongy and bleed easily Tongue bright to dark red, swollen
Abdomen	Abdomen flat, firm	Abdomen flaccid or distended (ascites)
Musculoskeletal	Firm, well-developed muscles Good posture No skeletal changes	Flaccid muscles, wasted appearance Stooped posture Skeletal malformations
Neurological	Good attention span, good concentration, astute thought processes Good reflexes	Inattentive, easily distracted, impaired thought processes Paraesthesias, reflexes diminished or hyperactive

NURSING CARE PLAN A person with cancer

James Casey, aged 72, is of northern European heritage. He has been receiving medical care for chronic obstructive pulmonary disease, chronic bronchitis, post myocardial infarction and type I diabetes mellitus for over 15 years. He reports that he lost his wife to lung cancer 5 years ago and still 'misses her terribly'. He describes his bad habits as smoking two packets of cigarettes a day for 52 years (104 packs/year), one to two six-packs of beer a week, one 'bourbon and water' a night and 'a lot of sugar-free junk food, like chips'. He assures the nurse that he quit smoking 2 years ago, when he could no longer walk a block without considerable shortness of breath, and just quit drinking alcohol a few weeks ago at his doctor's insistence. About a year ago, he had a basal cell carcinoma removed from his right ear. Six months ago, cancerous tumours were discovered in his bladder and he underwent two 6-week chemotherapy courses of bladder instillations of BCG. His latest report indicates that the tumours have grown back and no further chemotherapy would be useful. The urologist had considered surgery but believed that Mr Casey's other medical problems would compromise his chances of survival. Mr Casey decides to let the disease run its course and to be managed at home through hospice care. Because he lives alone in a modest home, he asks his daughter, Mary, and her family to move in with him to provide care and support during his final months. His daughter accepts, saying she is glad to be able to spend this time with her father; she has been informed of the physical and emotional stress this will entail.

ASSESSMENT

Glynis Jackson, RN, the hospice nurse assigned as case manager for James Casey, completes a health history and physical examination during her first two visits in his home, 1 day apart. She gathers this information over 2 days to conserve his strength and allow more time for Mr Casey and his daughter to talk about their concerns.

During the physical assessment, Glynis notes that Mr Casey is pale with pink mucous membranes, thin with a wasted appearance and has a strained, worried facial expression. He complains of severe back pain no longer adequately relieved by Oxycodone and Panadeine Forte alternating every 2 to 4 hours. His blood pressure is 90/50, right arm in the reclining position with no significant orthostatic change; his apical pulse is 102, regular and strong; respiratory rate 24 and unlaboured; breath sounds are clear but diminished in the bases; oral temperature is 36°C.

A tunneled Groshong catheter as a CVAD is present in the right anterior chest. There is no drainage, redness or swelling at the site. The catheter was placed last week when the Mr Casey was being evaluated at the anaesthetist's office for pain management, but no medication is running via the CVAD. Mary reports that his urinary output is adequate. Approximately 200 mL of yellow, cloudy, non-malodorous urine is present in the urinal at the bedside from his last voiding.

Mr Casey states that he spends most of his time either in bed or sitting up in a chair in his room. He reports that he has no energy anymore and is unable to walk to the bathroom unassisted, dress himself or take care of his own personal hygiene. Glynis rates Mr Casey's functional level at ECOG level 4: capable of only limited self-care, confined to bed or chair 50% or more of waking hours (Karnofsky 10 to 20). Mr Casey tells the nurse that his daughter 'is working day and night to help me and is looking awfully tired'.

Mary reports that Mr Casey is eating very poorly: he usually eats a small bowl of oatmeal with milk for breakfast and vegetable soup and crackers for lunch, but he tells her that he is too tired for dinner and wants only fruit juice. Mr Casey tells the nurse that he has no appetite and eats just to please Mary. He does drink at least three to four glasses of water a day plus juice. His blood sugar levels remain within normal range.

His current weight is 54.6 kg at 170 cm tall, down from 81.8 kg 2 months ago.

Available laboratory values from his visit with the doctor show the following:

Total protein: 41 g/L (normal range: 62 to 8.0 g/L)
Albumin: 22 g/L (normal range: 35 to 50 g/L)
Haemoglobin: 102 g/L (normal range: 132 to 170 g/L)
Haematocrit: 30.5% (normal range: 40.0–54.0%)
BUN: 30 mml/L (normal range: 5 to 25 mm/L, slightly higher in older people)
Creatinine: 116 µmol/L (normal range: 50 to 110 µmol/L).

DIAGNOSES

- *Imbalanced nutrition related to dietary intake being less than body requirements* as evidenced by loss of appetite and weight loss.
- *Risk of caregiver role strain* related to severity of her father's illness and lack of help from other family members as manifested by physical exhaustion and emotional stress.
- *Chronic pain* related to progression of the disease process as evidenced by increased frequency of analgesia with limited effect.
- *Impaired physical mobility* related to pain, fatigue and beginning of neuromuscular impairment as evidenced by lack of energy and inability to ambulate without assistance.
- *Risk of impaired skin integrity* related to decreased physical mobility and malnourished state as manifested by skin breakdown.

PLANNING

- Plan to have a home health aide come to the home, give Mr Casey a shower or bed bath daily and assist his daughter with some of the household chores.
- Plan for a volunteer to spend up to 4 hours a day, twice a week with Mr Casey so that Mary can attend to outside activities and chores.
- Order a hospital bed with electronic controls to be delivered to the house.
- Order a special foam pad for bed and chair, and a bedside commode from the medical supply house.
- Request a physiotherapy consultation to evaluate current level of functioning and determine how to maintain current level.

NURSING CARE PLAN A person with cancer (continued)

Expected outcomes

- Increase oral intake and show improvement in serum protein values.
- Minimal pain for the rest of his life.
- Able to continue his current activity level.
- Maintain skin integrity.
- Daughter will be able to maintain supportive caretaking activities as long as Mr Casey needs them.

IMPLEMENTATION

- Talk with the doctor about prescribing a medication to help stimulate the appetite.
- Ask about favourite foods and ask Mary to offer a small portion of one of these foods each day.
- Encourage drinking up to four cans of liquid nutritional supplement with fibre a day, sipping them throughout the day.
- Discuss a pain control program with the specialist, using the CVAD and a CADD-PCA infusion pump with a continuous morphine infusion.
- Call a community nurse to set up the equipment and supplies (including the medication) for the morphine infusion.
- Teach Mary how to use the pump and about the side effects of the morphine infusion, including those that require a call to the nurse for assistance. Teach which untoward effects should be reported.
- Instruct Mary to allow ample rest periods for her father between activities.
- Instruct Mary and the community carer to inspect skin daily, give good skin care with emollient lotion after bathing and report any beginning lesions immediately to the nurse.
- Talk with Mary about having her adult son and daughter relieve her of the housework and stay with Mr Casey so that she can get out of the house occasionally. Offer to talk with them if she is uncomfortable doing so.

EVALUATION

James Casey did increase his oral intake a little, sometimes eating the special treats his daughter prepared and drinking one or two cans of liquid nutritional supplement a day. However, his weight did not increase; it stayed at about 54.6 kg until his death 2 weeks later. His daughter was very grateful for the extra help from the community carers and the volunteer, although she could not bring herself to ask her son and daughter for help and did not want the nurse to do so. She did become more rested and reported that 'Dad and I had some wonderful 3 am talks when he couldn't sleep'.

Mr Casey was started on 20 mg of morphine per hour with boluses of 10 mg 4 times a day, for breakthrough pain. This medication relieved his pain quite well; after 2 days he was alert enough most of the time to carry on a normal conversation and still walk to the bathroom with help up until 2 days before he died.

The hospital bed simplified Mr Casey's care and made it much easier for him to rest comfortably and change position. His skin remained intact and in good condition.

Mary reported that Mr Casey died peacefully in his sleep, about 2 weeks after care was started. She said that spending the last weeks of his life with him was a healing experience for both of them.

CRITICAL THINKING IN THE NURSING PROCESS

1. What other tests could be done to evaluate James Casey's nutritional status?
2. Mr Casey had severe back pain. What were the possible pathophysiological reasons for his pain?
3. One of the specified interventions was to consult the doctor regarding medication to increase Mr Casey's appetite. What medications might fulfil that function? What side effects might they have that would contraindicate these medications for him?
4. If Mr Casey had developed signs and symptoms of sepsis, what manifestations would you expect to see? As the nurse making the home visits, what would be your nursing actions and in what order of priority?

REFLECTION ON THE NURSING PROCESS

1. What support strategies could you recommend to assist Mr Casey and his family with anticipatory grieving?
2. Outline what you have learned from this case study that you can implement in your future practice.

Anxiety

Early in the disease process—for example, during diagnosis and treatment—threats to or changes in health status, physical comfort, role functioning or even socioeconomic status can cause anxiety. Later, anxiety may result from the anticipation of pain, disfigurement or the threat of death. In particular, people whose coping skills have been poor in the past (e.g. in managing anger) may find themselves at a loss to manage this current crisis. The person may manifest overt signs of anxiety: trembling, restlessness, irritability, hyperactivity, stimulation of the sympathetic nervous system (increased blood pressure, pulse, respiration, excessive perspiration, pallor), withdrawal, worried facial expressions and poor eye contact. The person may report insomnia and feelings of tension and apprehension, or express concerns regarding perceived changes brought about by the disease and fear of future events.

- Carefully assess the person's level of anxiety (moderate anxiety, severe anxiety or panic) and the reality of the threats represented in the person's current situation. The level of anxiety and the reality of the perceived threat influence the type of intervention that is appropriate for the person. *A person in panic may need medical intervention with appropriate medications, whereas those with moderate or severe anxiety are often managed by the nurse through counselling and teaching new coping skills.*
- Establish a therapeutic relationship by conveying warmth and empathy and using non-judgmental and effective listening. *A person who feels safe in the relationship with the nurse more readily expresses their thoughts and feeling. The person will be able to develop trust the nurse and perhaps be willing to try new behaviours as suggested.*

The amount of time this relationship may take to develop depends on the person's current emotional and mental state and the stage of the disease process.

- Encourage the person to acknowledge and express feelings, no matter how inappropriate they may seem to the person. *Just by expressing their feelings, people often can significantly diminish anxiety. Expressing feelings also allows the person to direct energy towards healing and thus has a positive therapeutic effect. Moreover, by acknowledging feelings, especially those the person considers unacceptable, the person can lay the groundwork for new coping behaviours.*
- Review the coping strategies the person has used in the past and build on past successful behaviours, introducing new strategies as appropriate. Explain why inappropriate strategies, such as repressing anger or turning to alcohol, are not helpful. *The person will be more willing to make changes that build on what has already worked in the past. The person will also be more willing to reject inappropriate strategies if they are given a persuasive reason why they have not had the desired effect in managing previous crises.*
- Identify resources in the community, such as crisis hotlines and support groups, that can help the person manage anxiety-producing situations. *The person may not have support systems available or the person's significant others may be having their own difficulties in dealing with the cancer diagnosis. Cancer Council Australia provides programs, support groups and counselling in all states and territories throughout Australia.*
- Provide specific information for the person about the disease, its treatment and what may be expected, especially for those people with obvious misinformation. *Knowing what is to come gives the person a sense of control and enables them to make decisions. Also, knowing that every effort will be made to keep the person as free of pain as possible can do a great deal to relieve anxiety.*
- Provide a safe, calm and quiet environment for the person in panic. Remain with the person and administer anti-anxiety medications as ordered. *Staying with the person and displaying calmness and confidence can protect the person from injury and prevent further panic. If the panic does not subside with the nurse's presence and support, referral to the doctor for medication management may be necessary.*
- Use crisis intervention theory to promote growth in the person and their significant others, regardless of the outcome of the disease. *During a major crisis, people can, with assistance, transform the experience from one that causes defeat and despair to one that enhances personal and spiritual growth.* If you are not skilled in this area, a referral to an appropriate mental health professional may be helpful to the person and their family.

Disturbed body image

Cancer and cancer treatments frequently result in major physiological and psychological body image changes. See the following box for manifestations of cancer. Loss of a body part (e.g. amputation, prostatectomy or mastectomy), skin changes and hair loss from chemotherapy or radiation therapy, disfigurement of a body part (e.g. lymphoedema in the affected upper and lower extremities) or the creation of unnatural openings on the body for elimination (e.g. colostomy or ileostomy) may have a major effect on the person's self-image. The gaunt, wasted appearance of the cachectic person, or draining, malodorous lesions that result when cancer breaks through the skin, are other significant aetiologies of body image disturbance. This may also give rise to fear of rejection, which plays a major role in sexual dysfunction. In addition to all of the other afflictions that cancer brings about, the person may undergo major changes in appearance and function. The person may exhibit a visible physical alteration of some portion of the body, verbalise negative feelings about the body and/or fear of rejection by others, refuse to look at the affected site and depersonalise the body change or lost part (e.g. by calling the colostomy 'that thing').

- Discuss the meaning of the loss or change with the person. *Doing so helps the nurse discover the best approach for this particular person and involves the person more actively in interventions. A small, seemingly trivial loss may have a big impact, especially when viewed in light of the other changes that are occurring in the person's life. Likewise, a major loss may not be as important as the nurse might imagine. To ensure more appropriate and individualised care, evaluate each situation in terms of the reactions of the specific person.*
- Observe and evaluate interaction with significant others. *People who are important to the person may unintentionally reinforce negative feelings about body image; on the other hand, the person may perceive rejection where none exists.*
- Allow denial, but do not participate in the denial; for example, if a person does not want to look at the wound, the nurse may say, 'I am going to change the dressing to your breast incision now'. *During the initial stage of shock at the loss of a body part, denial is a protective mechanism and should not be challenged, nor should it be promoted. A matter-of-fact approach and an empathetic attitude will go far to facilitate the eventual acceptance of the change.*
- Assist the person and significant others to cope with the changes in appearance:
  a. Provide a supportive environment.
  b. Encourage the person and significant others to express feelings about the situation.
  c. Give matter-of-fact responses to questions and concerns.
  d. Identify new coping strategies to resolve feelings.
  e. Enlist family and friends in reaffirming the person's worth.
- *A supportive, safe environment in which feelings are respected and new coping strategies can be tried promotes acceptance, as does reaffirming that the person's worth is not diminished by any physical changes.*
- Teach the person or significant others to participate in the care of the afflicted body area. Provide support and validation of their efforts. *Active involvement in providing*

care, such as changing a dressing or emptying a colostomy bag, empowers the person and/or significant others. This intimate involvement also desensitises feelings about disfigurement and promotes acceptance. Involving significant others reduces the risk of them rejecting the person and can promote closeness. Positive reinforcement from the nurse encourages them to continue these behaviours.

- Teach strategies for minimising physical changes, such as providing skin care during radiation therapy and dressing to enhance appearance and minimise change in the body part. *Early intervention can limit the negative side effects of treatment and actually promote recovery. Involving the person provides an additional way for the person to be in control of a difficult situation.*
- Teach ways to reduce the alopecia that results from chemotherapy and to enhance appearance until the hair grows back:
  a. Discuss the pattern and timing of hair loss. *This allows the person to cope with changes and incorporate them into daily activities.*
  b. Encourage wearing cheerful, brightly coloured head coverings; assist in colour coordinating them with usual clothing. *Attractive head coverings protect the bald head while allowing the person to feel stylish and well dressed.*
  c. Refer to a good wig shop before hair loss is experienced. *Hair colour and texture can be matched to minimise obvious changes in appearance.*
  d. Refer to support programs such as cancer support workshops that are provided by the Cancer Council in each state and territory. *A support group can diminish feelings of isolation and provide practical tips for managing problems. For a list of community resources available to people with cancer or for confidential information and support, contact the Cancer Council Helpline on 13 11 20. (The cost is that of a local phone call from anywhere in Australia.)*
  e. Reassure that hair will grow back after chemotherapy is discontinued, but also inform that the colour and texture of the new hair may be different. Hair loss has been identified as the most distressing symptom by many people (Ferrell, 2000). *Interventions to reduce that loss can have a significant impact on body image concerns. Moreover, knowing what to expect may decrease anxiety and distress.*

Anticipatory grieving

Anticipatory grieving is a response to loss that has not yet occurred. Overall, only 50% of people with cancer fully recover and certain types of cancer have a much higher death rate; thus, the person with cancer is often confronted with facing death and making preparations for it. This can be a healthy response that allows the person and family to work through the dying process and achieve growth in the final stage of life. Perceived changes in body image and lifestyle can also prompt anticipatory grieving. The person or significant others may show sorrow, anger, depression or withdrawal, expressing distress at the potential loss or verbalising concern about unfinished life business. (See Chapter 4 for more on nursing care of the person who is grieving or dying.)

MANIFESTATIONS Cancer

- Hair loss
- Depression
- Fever
- Bleeding gums
- Oropharyngeal ulcerations
- Stomatitis
- Anorexia
- Nausea and vomiting
- Diarrhoea
- Emaciation
- General weakness
- Flaccid muscles
- Stooped posture
- Pallor
- Excessive bruising
- Radiation burns
- Visible tumour (abdomen)
- Odour of decay
- Hypotension

- Use the therapeutic communication skills of active listening, silence and non-verbal support to provide an open environment for the person and significant others to discuss their feelings realistically and to express anger or other negative feelings appropriately. *This helps the person and family to get in touch with feelings and confront the possibility of the loss or death.*
- Answer questions about illness and prognosis honestly, but always encourage hope. *This allows for realistic appraisal of the situation and planning, and helps to combat feelings of hopelessness and depression.*
- Encourage the dying person to make funeral and burial plans ahead of time and to be sure the will is in order. Make sure the necessary phone numbers can be easily located. *This gives a sense of control and relieves family members of these concerns at a time when the person is most in need of their support and when they themselves are extremely stressed.*
- Encourage the person to continue taking part in activities he or she enjoys, including maintaining employment as long as possible. *This gives a sense of continuity of life even in the face of severe losses.*

Risk of infection

Malnutrition, impaired skin and mucous membrane integrity, tumour necrosis and suppression of the white blood cells (WBC) from chemotherapy or radiation may contribute to the risk of infection. Anorexia, as well as the disease itself, deprives the body of nutrients needed for healing, while impaired integrity of skin and mucous membranes (a result of chemotherapy

and/or radiation therapy) compromise the first lines of defence against microbial invasion. Cells in the centre of large or not very vascular tumours may die from malnutrition, eventually eroding through tissues to increase the risk of sepsis. Bone marrow depression resulting from the effects of certain types of cancers and from chemotherapy undermine the body's ability to respond to infection. The person may exhibit the classic signs of infection: lassitude, fever, anorexia, pain in the affected area and physical evidence of infection, such as a purulent, draining lesion or wound. If the bone marrow is compromised, the usual signs and symptoms of infection may be absent or reduced.

- Monitor vital signs. *Fever and sympathetic nervous system responses, such as increased pulse and respiration, are usual early signs of infection. However, severely immunosuppressed people may be unable to mount a fever; therefore, the absence of fever cannot rule out infection.*
- Monitor WBC counts frequently, especially if the person is receiving chemotherapy known to cause bone marrow suppression. *This allows the nurse to notify the doctor at the first sign of diminishing WBC counts so that corrective action can be taken.*
- Teach the person to avoid crowds, small children and people with infections when WBC count is at nadir (lowest point during chemotherapy) and to practise scrupulous personal hygiene. *During periods of leucopenia, the person may lose immunity to his or her own natural flora. Careful attention to hygiene reduces the risk of infection. Crowds, which promote contact with a greater variety of infectious agents, and friends with minor infections can be very dangerous for the immunosuppressed. Small children should be avoided because they often have microbes to which most people are usually immune but which the person with cancer may not be able to resist.*
- Protect skin and mucous membranes from injury. Teach appropriate skin care measures, such as good hygiene, use of a moisturising lotion to prevent dryness and cracking, frequent changes of position for the bed-bound and immediate attention to skin breaks or lesions. *Ensuring intact skin strengthens the first line of defence against infection.*
- Encourage the person to consume a diet high in protein, minerals and vitamins, especially vitamin C. *Improving nutrition decreases the risk of infection. Vitamin C has been shown to help prevent certain types of infection, such as colds.*

Risk of injury

In addition to infection, cancer can pose a risk of injury from, for example, obstruction by a large tumour or one located in a limited body space (e.g. in the brain, bowel or bronchial airways). If the cancer is one that creates ectopic sites of hormones, elevated levels of hormones that are not under the control of the pituitary gland can injure the person in a variety of ways. Signs of obstruction depend on the organ involved: bowel obstruction presents with pain, distension and cessation of bowel activities; obstruction in the brain gives signs of increased intracranial pressure or personality/behavioural change; bronchial obstruction manifests as respiratory distress, cyanosis and altered arterial blood gases. Ectopic production of parathyroid hormone manifests as high serum calcium levels as well as signs of hypercalcaemia; ectopic production of antidiuretic hormone causes fluid retention and manifests as hypertension and peripheral and pulmonary oedema.

- Assess frequently for signs and symptoms indicating problems with organ obstruction. *Early detection of major problems allows the nurse to seek medical help before the problem evolves into a physiological crisis.*
- Teach to differentiate minor problems from those of a serious nature. Encourage the person to consult with the nurse or doctor if in doubt, or to call 000 if they become very ill. Box 13.13 provides guidelines to help people identify serious problems. *Having guidelines for when to call the doctor provides an anxiety-reducing safety net for the person and family and promotes early detection of complications.*
- Monitor laboratory values that may indicate the presence of ectopic functioning and report abnormal findings to doctors immediately. (See Table 13.4 for laboratory indicators of ectopic functions.) *Early detection promotes early medical intervention and prevents serious consequences from the ectopic secretion.* Refer to Chapters 9, 18 and 19 for specific signs and symptoms of electrolyte imbalances and endocrine disorders.

BOX 13.13 When to call for help

Instruct the person or family member to call the nurse or doctor if any of the following signs or symptoms occurs:

- Oral temperature greater than 38.6°C.
- Severe headache; significant increase in pain at usual site, especially if the pain is not relieved by the medication regimen; or severe pain at a new site.
- Difficulty breathing.
- New bleeding from any site, such as rectal or vaginal bleeding.
- Confusion, irritability or restlessness.
- Withdrawal, greatly decreased activity level or frequent crying.
- Verbalisations of deep sadness or a desire to end life.
- Changes in body functioning, such as the inability to void or severe diarrhoea or constipation.
- Changes in eating patterns, such as refusal to eat, extreme hunger or a significant increase in nausea and vomiting.
- Appearance of oedema in the extremities or significant increase in oedema already present.

Instruct the person or family member to call 000 if the person:

- is having much difficulty breathing or if the face or lips have a bluish tinge
- becomes unconscious or has a convulsion
- exhibits unmanageable behaviour, such as being physically abusive, hurting self or engaging in uncontrollable activity.

Imbalanced nutrition: less than body requirements

The anorexia–cachexia syndrome (described earlier in this chapter) is a common cause of malnutrition in people with cancer. Metabolism increases in response to increased cancer cell production, while the cancer's parasitic activity reduces the nutrients available to the body. Loss of appetite, food aversion, nausea and vomiting, and painful oral lesions from chemotherapy or radiation may contribute to impaired nutrition. Tumours of the gastrointestinal tract that affect absorption also contribute to the problem. Manifestations include wasted appearance, considerable weight loss over a relatively short period of time, anthropometric measurements below 85% of standard for fat and muscle tissue, decreases in serum proteins and negative responses to antigen testing.

- Assess current eating patterns, including usual likes and dislikes, and identify factors that impair food intake. *This allows for a more individualised plan based on needs and preferences.*
- Evaluate degree of malnutrition:
  a. Check laboratory values for total serum protein, serum albumin and globins, total lymphocyte count, serum transferrin, haemoglobin and haematocrit. *These values represent the laboratory values that are most likely to decrease with malnutrition.*
  b. Calculate nitrogen balance and creatinine height index. Calculate skeletal muscle mass and compare findings to normal ranges. *Urinary creatinine is an index of lean body mass and decreases in malnutrition. Lean muscle mass is catabolised for energy in people with cancer.*
  c. Take anthropometric measurements and compare them to standards: height, weight, elbow breadth, arm circumference, triceps skinfold thickness and arm muscle mass. *This estimates the degree of wasting; findings below 85% of standard are considered malnutrition.*
- Teach the principles of maintaining good nutrition and adapting the diet to medical restrictions and current preferences. *This tailors the food plan to the person's needs and thereby promotes compliance.*
- Manage problems that interfere with eating:
  a. Encourage eating whatever is appealing and consider adding nutritional supplements such as Ensure Plus or Isocal to the diet. *It is better to eat something even if it is not nutritionally balanced.*
  b. Eat small, frequent meals. *These are more easily digested and absorbed and are usually better tolerated by the person with anorexia.*
  c. Encourage trying icy cold foods (such as ice-cream) or those that are more highly seasoned if food has no taste. *Chemotherapy and radiation therapy may harm taste buds and prevent distinguishing the taste of foods. Strong seasonings and coldness make food more enjoyable to the person with diminished taste. However, spicy foods are not recommended for people with stomatitis.*
  d. Encourage cold and bland semi-soft and liquid foods with painful oropharyngeal ulcers; use a non-alcohol anaesthetic mouthwash prior to eating. *These foods are less irritating to sensitive mucous membranes; deadening the pain can make chewing and swallowing easier.*
  e. Manage nausea and vomiting by administering antiemetic drugs. (Around-the-clock medication may be an effective preventive measure.) Encourage the person to eat small, frequent, low-fat meals with dry foods such as crackers and toast, to avoid liquids with meals and to sit upright for an hour after meals. Remove emesis basins and encourage oral hygiene before eating. *Dry, low-fat foods are more readily tolerated when nauseated. Removing vomiting cues, such as odour and supplies associated with vomiting, can reduce nausea.*
- Teach to supplement meals with nutritional supplements such as Ensure Plus or Isocal and to take multivitamin and mineral tablets with meals. Suggest increasing kilojoules by adding ice-cream or frozen yoghurt to the liquid supplement or commercial protein–carbohydrate powders to milk or fruit juice. *Because the food intake is usually less than that needed to maintain or gain weight, these supplements can add kilojoules in a manner often tolerated.*
- Teach to keep a food diary to document daily intake. If the person can see how little is being consumed, he or she may eat more. *A food diary also helps the nurse keep a kilojoule count and alert the doctor if more drastic nutritional measures, such as a feeding tube or parenteral nutrition, need to be instituted.*
- Teach to administer parenteral nutrition via a central line or other CVAD. Teach safety measures and care of the CVAD and explain how the pump delivering the solution works. Provide an emergency phone number for help with administration problems. (See Chapter 21 for safety guidelines for administering parenteral nutrition.) *The person with chronic or terminal cancer requiring parenteral nutrition is usually managed at home, so information on how to manage the entire process may be needed.*

Impaired tissue integrity

The most common impairment of tissue integrity occurs in the oral–pharyngeal–oesophageal mucous membranes. It is secondary to the effects of some chemotherapeutic drugs and radiation treatment to the head and neck. The oral–pharyngeal–oesophageal tissues are lined with cells with a high mitotic turnover rate and are therefore vulnerable to many chemotherapeutic drugs. Leukaemias, bone marrow transplants and herpes viral infections are other aetiological factors in the disruption of oral–pharyngeal–oesophageal tissue. Manifestations of this problem may include the following:

- Small ulcers occur on the tongue and mucous membranes in the mouth and throat.
- Herpes simplex type 1 lesions or vesicles evolve into ulcerations.
- Fungal infections, such as thrush (due to *Candida* infections), are manifested by a white, yellow or tan coating with dry, red, fissured tissue underneath.

- Red, swollen, friable gums bleeding with minimal or no trauma.
- **Xerostomia** is excessive dryness of the mucous membranes (due to chemotherapy or radiation).

Manage such problems with the following interventions:

- Carefully assess and evaluate the type of tissue impairment present. Identify possible sources, such as chemotherapy or radiation therapy to head and neck. *This allows the nurse to implement corrective measures appropriate to the type of problem.*
- Implement and teach measures for preventing oropharyngeal infection:
  a. Observe for systemic signs of infection. Be suspicious of any fever that has no apparent cause. *This facilitates early identification of an infection before it spreads.*
  b. Encourage cleaning teeth gently and using a non-alcohol mouthwash several times a day. This can be done after waking in the morning, after any oral intake and before bedtime. Soak dentures nightly in hydrogen peroxide and floss gently with waxed floss after meals and bedtime; this measure may be contraindicated for people with leukaemia or thrombocytopenia. *Disrupted mucous membranes allow the normal oral bacterial flora into the systemic circulation, which can result in sepsis in the immunocompromised person. Reducing the oral flora by frequent hygiene decreases the risk of infection.*
  c. Culture any oral lesions and report the problem to the doctor. *Herpes lesions may not follow a typical pattern in immunosuppressed people. Identifying the cause of the infection, whether viral, fungal or bacterial, allows the doctor to prescribe the appropriate treatment.*
- Implement and teach measures for reducing trauma to delicate tissues:
  a. Counteract dry mouth (xerostomia) with lubricating and moisturising agents, such as Gatorade, sugarless gum and Blistex. *This protects mucous membranes from infection and trauma.*
  b. Avoid putting sharp instruments in the mouth. Use smooth plastic spoons and forks for eating, especially with a bleeding disorder. Dental work should be done by dental oncologists.
  c. Brush teeth with a very soft toothbrush and obtain a new toothbrush monthly. If gums are friable and bleeding, clean teeth with a soft cloth or toothpaste over finger. Chlorhexidine mouthwash (Savacol) may be used. *This protects gums from trauma and decreases risk of haemorrhage.*
- Administer specific medications as ordered to control infection and/or pain:
  a. Aciclovir is often used to treat viral infections.
  b. Systemic antibiotics are used to treat bacterial infections.
  c. Nystatin or clotrimazole solution for 'swish and swallow' or lozenges that dissolve slowly in the mouth are used for fungal infections.

BOX 13.14 Combination mouthwashes for oropharyngeal pain control

Kaiser mouthwash
- Nystatin
- Hydrocortisone
- Tetracycline

Stanford mouthwash
- Nystatin
- Tetracycline
- Lignocaine
- Hydrocortisone

Xyloxylin suspension
- Benylin syrup
- Lignocaine
- Maalox suspension

Stomafate suspension
- Sucralfate
- Sterile water
- Benylin syrup
- Maalox suspension

  d. Use viscous xylocaine or various combination mouthwashes before meals and as needed. These agents reduce pain and inflammation. See Box 13.14 for the ingredients of combination mouthwashes. *Knowing the contents of each mouthwash can prevent hypersensitivity reactions (e.g. lignocaine) and assist when teaching.*

Nursing interventions for oncological emergencies

In caring for people with cancer, nurses may encounter a number of emergency situations in which their role may be pivotal to the person's survival. Most of these emergencies require astute observations, accurate judgments and rapid action once the problem has been identified. A brief description of the more common oncological emergencies with nursing interventions follows. In all cases, immediate notification of the doctor or emergency team is the first step.

Pericardial effusions and neoplastic cardiac tamponade

Malignant pericardial effusion is an accumulation of excess fluid in the pericardial sac that compresses the heart, restricts heart movement and results in a cardiac tamponade. The signs of cardiac tamponade are caused by compression of the heart, which leads to decreased cardiac output and impaired cardiac function. Signs include hypotension, tachycardia, tachypnoea, dyspnoea, cyanosis, increased central venous pressure, anxiety, restlessness and impaired consciousness.

Interventions include the following:

- Start oxygen and alert respiratory therapy for other respiratory support as needed.
- Insert an intravenous cannula if one is not already in place.
- Monitor vital signs and initiate haemodynamic monitoring.

- Prepare vasopressor drugs as ordered.
- Bring emergency cart to bedside.
- Set up for and assist doctor with a pericardial tap (pericardiocentesis).
- Reassure the person.

Superior vena cava syndrome

The superior vena cava can be compressed by mediastinal tumours or adjacent thoracic tumours. The most common cause is small-cell or squamous cell lung cancers. Occasionally the problem is caused by thrombus around a central venous catheter that then plugs up the vena cava, resulting in obstruction and backup of the blood flowing into the superior vena cava.

Obstruction of the venous system causes increased venous pressure, venous stasis and engorgement of veins that are drained by the superior vena cava. Signs and symptoms may develop slowly; facial, periorbital and arm oedema are early signs. As the problem progresses, respiratory distress, dyspnoea, cyanosis, tachypnoea, altered consciousness and neurological deficits may occur. Figure 13.7 illustrates the superior vena cava syndrome.

Emergency measures include the following:

- Provide respiratory support with oxygen and prepare for tracheostomy.
- Monitor vital signs.
- Administer corticosteroids (e.g. dexamethasone) to reduce oedema.
- If the disorder is due to a clot, administer antifibrinolytic or anticoagulant drugs.
- Provide a safe environment, including seizure precautions.

After the emergency is managed, the person often receives radiation or chemotherapy to reduce the tumour size.

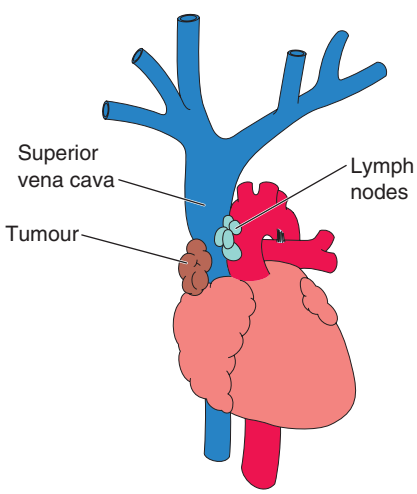


FIGURE 13.7 ■ The superior vena cava syndrome. The enlargement of a tumour adjacent to the superior vena cava (usually in the lung or mediastinum) compresses that major blood vessel, which leads into the right atrium of the heart. As a result, blood backs up into the venous system behind the obstruction, diminishing blood flow into the heart

Sepsis and septic shock

Tumour necrosis, immune deficiency, antineoplastic therapy, malnutrition and co-morbid conditions can lead to the development of sepsis. Bacteria gain entrance to the blood, grow rapidly and produce septicaemia. Because malignant tumours are more likely to use anaerobic metabolic pathways, the bacteria of tumour sepsis are usually Gram-negative and damage the body through a combination of bacterial endotoxins and an uncontrolled immune reaction. Gram-negative sepsis progresses to systemic shock and eventually results in multisystem failure. Signs and symptoms appear in two phases. The first phase is characterised by vasodilation with vascular dehydration, high fever, peripheral oedema, hypotension, tachycardia, tachypnoea, hot flushed skin with creeping mottling beginning in the lower extremities and anxiety or restlessness. Without treatment, the shock progresses to the second phase, which shows the more classic signs of shock: hypotension, rapid thready pulse, respiratory distress, cyanosis, subnormal temperature, cold clammy skin, decreased urinary output and altered mental state. Identifying the problem while the person is still in the hyperdynamic state is crucial to the person's survival. See Chapter 10 for further discussion of septic shock.

Spinal cord compression

Spinal cord compression is most commonly associated with pressure from expanding tumours of the breast, lung or prostate, and with lymphoma or metastatic disease. Spinal cord compression constitutes an emergency because of the potential for irreversible paraplegia. Back pain is the initial symptom in 95% of the cases of spinal cord compression. This may progress to leg pain, numbness, paraesthesias and coldness. Later, bowel and bladder dysfunction occur; finally, neurological dysfunction progresses from weakness to paralysis. Treatment often consists of radiation or surgical decompression, but early detection is essential. See Chapter 42 for further discussion of spinal cord compression.

Obstructive uropathy

People with intra-abdominal, retroperitoneal or pelvic malignancies, such as prostate, cervical or bladder cancers, may experience obstruction of the bladder neck or the ureters. Bladder neck obstruction usually manifests as urinary retention, flank pain, haematuria or persistent urinary tract infections, but ureteral obstruction is often not evident until the person is in renal failure. See Chapters 26 and 27 for further discussion of obstructive uropathy.

Hypercalcaemia

Hypercalcaemia in people with cancer results from the excessive ectopic production of parathyroid hormone and is most commonly associated with cancers of the breast, lung, oesophagus, thyroid, head and neck, and with multiple myeloma. Bone metastases may also cause hypercalcaemia. When the rate of calcium mobilisation from the bone exceeds the renal threshold for excretion, serum calcium levels can become dangerously elevated. People with hypercalcaemia often present with non-specific symptoms of fatigue, anorexia, nausea, polyuria and constipation. Neurological symptoms include muscle

weakness, lethargy, apathy and diminished reflexes. Without treatment, hypercalcaemia progresses to alterations in mental status, psychotic behaviour, cardiac arrhythmias, seizures, coma and death (see Chapter 9).

Hyperuricaemia

Hyperuricaemia usually is a complication of rapid necrosis of tumour cells after vigorous chemotherapy for lymphomas and leukaemias. Hyperuricaemia may be related to increased uric acid production or to the tumour lysis syndrome associated with Burkitt's lymphoma. Uric acid crystals are deposited in the urinary tract, causing renal failure and uraemia. People with hyperuricaemia manifest with nausea, vomiting, lethargy and oliguria.

Syndrome of inappropriate antidiuretic hormone secretion

Occurring in only about 2% of people with cancer, syndrome of inappropriate antidiuretic hormone secretion (SIADH) is related to an ectopic secretion of antidiuretic hormone that is usually associated with small-cell lung carcinoma, but also occasionally with prostate and adrenal cancers. The kidney secretes an excessive amount of sodium and conserves a disproportionate amount of free water, causing profound hyponatraemia. Signs and symptoms include anorexia, nausea, muscle aches and subtle neurological symptoms that can progress to lethargy, confusion, seizures and coma from cerebral oedema.

Tumour lysis syndrome

Tumour lysis syndrome (TLS) is a life-threatening emergency for people with cancer. TLS is characterised by a combination of two or more metabolic abnormalities, including hyperuricaemia, hyperphosphataemia, hyperkalaemia and/or hypocalcaemia (McBride & Westervelt, 2012). The syndrome develops because of massive and rapid destruction or death of cancer cells caused by cytotoxic treatment such as chemotherapy, radiation, biological therapy, hormonal therapy and surgery. It can also occur spontaneously with sudden death of tumour cell. A high incidence of TLS occurs in people with bulky, highly proliferating and chemosensitive tumours such as high-grade lymphomas (Burkitt's lymphoma) and acute lymphoblastic leukaemia (ALL). Although the incidence of TLS in solid tumours is rare, cases of TLS following chemotherapy have been reported in people with small-cell lung cancer, breast cancer, neuroblastoma, melanoma and ovarian cancer.

The major cause of TLS is chemotherapy to tumours with a high proliferative rate, a relatively large tumour burden and high sensitivity to cytotoxic agents, which leads to massive and rapid cell death. Usually, within a week of initiating chemotherapy, the body no longer can excrete the large amount of metabolic by-products from the cell death, resulting in the release of intracellular contents and metabolic by-products (such as potassium, phosphorus and nucleic acid) into the bloodstream (McBride & Westervelt, 2012). As a result, a combination of metabolic derangements occurs, including hyperkalaemia, hyperuricaemia and hyperphosphataemia with secondary hypocalcaemia. These metabolic abnormalities put people at risk of cardiac malfunction and renal failure.

Clinical manifestations of TLS include nausea, vomiting, lethargy, oedema, fluid overload, congestive heart failure, cardiac dysrhythmias, seizures, muscle cramps, tetany, syncope and possible sudden death. Diagnosis of TLS mainly depends on laboratory tests and clinical signs and symptoms. Prevention is crucial in management of TLS. People at risk of TLS include those with bulky chemosensitive cancer such as high-grade lymphomas and acute leukaemia, elevated serum uric acid, potassium, phosphorus and renal deficiency. Preventive and management measures include identifying people at risk, administration of allopurinol to inhibit the conversion of nucleic acid to uric acid, hydration and diuretic therapy to promote urinary excretion of uric acid and phosphate, urine alkalinisation to promote the urinary excretion of uric acid, administration of oral phosphate binder such as aluminium hydroxide to promote the excretion of phosphate through the bowel, administration of sodium polystyrene sulfonate (Kayexalate) to promote the excretion of potassium through the bowel and initiation of haemodialysis to people unresponsive to standard approaches to hyperkalaemia, hyperuricaemia or hyperphosphataemia.

Health education for the person and family

Prevention

Cancer Council Australia (2013b) promotes strategies to reduce cancer risk in addition to the screening measures discussed earlier in this chapter. Based on these strategies, nurses teach people and families to decrease risk factors by:

- quitting smoking
- eating healthy foods (see Box 13.15).
- staying in shape
  - maintain a healthy body weight range and body mass index
  - waist circumference below 94 cm for men and 80 cm for women
- being sun smart
  - slip on protective clothing
  - slop on SPF 30^+ sunscreen (20 minutes before going outside and re-apply every 2 hours)
  - slap on a broad-brimmed, legionnaire or bucket-style hat
  - seek shade
  - slide on some sunglasses
- limiting alcohol
  - limit intake—no more than two standard drinks per day
  - choose low-alcohol drinks
  - avoid binge drinking
  - have at least 2 alcohol-free days per week
  - eat some food when you drink
- moving your body
  - only 30 minutes of moderate intensity exercise each day is good for your health and 60 minutes can reduce your risk of developing cancer
  - increase walking—at lunchtime, park your car further away, take the stairs.

Source: Cancer Council Australia (2012; 2013b); www.cancer.org.au.

In addition, encourage people to report to the public health department any known leaking of chemicals or radioactive materials into the water or air and any noted increase in the incidence of cancer, especially of one specific type, in their communities.

BOX 13.15 Cancer Council Australia: strategies for eating healthy foods

Eat two serves of fruit and five serves of vegetables per day.
Eat cereals—preferably wholegrain.
Limit cured meats.
Red meat three to four times a week only and on other days eat fish, poultry, beans and lentils.
Choose foods low in salt, sugars and saturated fats.

Source: Cancer Council Australia (2013b). *Fact sheet: Maintain a healthy weight.* Retrieved from www.cancer.org.au/preventing-cancer/reduce-your-risk/maintain-a-healthy-weight.html, 8 January 2013. For more information visit www.cancer.org.au.

Rehabilitation and survival

Rehabilitation from cancer not only involves regaining strength, recovering from surgery or chemotherapy and learning to live with an altered body part or appliance, but also entails recovering from associated psychological and emotional turmoil.

Rehabilitation centres provide physiotherapy, occupational therapy, speech pathology, job retraining and an opportunity to recuperate before resuming full responsibilities. In addition, many people go home to convalesce and receive in-home support in the form of nursing supervision, direct care and teaching. Hygiene and home maintenance can be provided by home and community care organisations. Physiotherapists and occupational therapists provide muscle strengthening and mobility training (especially with prostheses) and home safety teaching.

Psychological rehabilitation of cancer survivors addresses quality-of-life issues. Three 'seasons of cancer survival' have been described (Mullan, 1985). The first starts with diagnosis but is dominated by treatment. The second stage is one of extended survival, which occurs when treatment ends and the watchful waiting period begins. This period is characterised by fear of recurrence. Permanent survival is said to begin when the survival period has gone on long enough that the risk of recurrence is small. In this period, the person has to deal with secondary problems related to health and social issues resulting from the cancer experience. Employment may be a problem, private health insurance may be cancelled and life insurance may be difficult to get. Relationships may have suffered from the strain of the illness on significant others and the essential self-focusing required for recovery. However, both the person and significant others may have undergone a personal and spiritual growth that ushers in a new and enriching period of their lives.

New self-help groups are emerging in many communities to support others through their 'seasons of survival'. Many cancer survivors speak to groups about assisting other cancer survivors. People and families need to be informed about the many resources available through community agencies as well as the survivor support groups.

Community-based care

Before the person is discharged, teach both the person and significant others or caregivers to manage the person at home. Discuss problems that may result from the type of cancer and the treatment received and provide information on how to manage these problems and when to call the doctor.

- Teach wound care to the person with an open wound or draining lesion and provide a referral to a community nurse to monitor progress.
- Explain special diets clearly or refer the person to a dietitian before discharge.
- Carefully review the doctor's instructions with the person and family, making sure they understand the medications to be taken, any other treatments and when to see the doctor for follow-up care.
- Provide or order equipment and supplies needed for home care, especially any specialised bed or equipment to aid mobility and ensure safety in the home.
- For the person who will need complex care, such as parenteral nutrition, provide a referral to the community nurse before discharge.

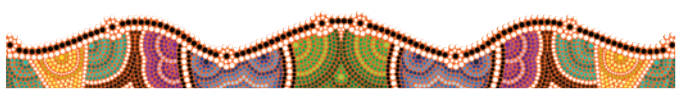
Because the hospital stay is often short, the person and family will benefit from follow-up phone calls at home for several days. People do not learn well under the stress of going home; give the person and family a number to call if they have concerns or questions.

Hospice care

More and more people with terminal disease are electing to die at home. This decision has been made easier by the increased availability of hospice programs. When a person and family or significant others elect **hospice** care, they are usually precluding additional hospitalisations other than those required to manage reversible problems. People in hospice care also refuse resuscitation measures (CPR and other extraordinary measures).

Many hospice services are connected with an inpatient respite care unit, where the person can receive 24-hour care for up to several weeks. This source provides the necessary care to the person if a family member becomes ill or needs to be relieved temporarily of the tremendous burden of caring for a dying loved one. Hospice care involves a multidisciplinary team and is designed to give the person comfort and to assist in a peaceful death with support to caretakers. The team usually consists of a nurse case manager, a doctor, an anaesthetist or pharmacist, an infusion therapist, a social worker, a physiotherapist, a home health aide and volunteers.

Studies of families that have participated in hospice services have found that family members were very positive about the experience (Teno et al., 2001). The aspects of hospice care they most appreciated were the 24-hour accessibility and availability of the healthcare team and the quality of communication from all team members. Family members emphasised that 'the nurses listened, answered questions honestly and prepared us for changes in the patient's condition'. Team members were rated as very professional, but more relaxed and friendly than hospital staff; they talked with the family and displayed accepting, non-judgmental attitudes. Team members were also seen as well informed, knowledgeable and competent with excellent problem-solving skills. Chapter 4 provides more information on hospice care.



CHAPTER HIGHLIGHTS

- Lung cancer was the leading cause of all cancer deaths in both men and women in Australia in 2005, accounting for 19% of all cancer deaths. Cancer can affect people of any age, gender, ethnicity or geographical region.
- The incidence of cancer increases with advancing age. The most commonly seen cancers in women are breast, colorectal and lung cancer, and melanoma of the skin. In men, prostate, lung and colorectal cancer, and melanoma of the skin occur most frequently.
- Oncogenes are genes that promote cell proliferation and are capable of triggering cancerous characteristics. Several oncogenes, such as BRCA-1 and BRCA-2, are associated with breast cancer.
- Tumour suppressor genes, which normally suppress oncogenes, can become inactive by deletion or mutation. Inherited cancers have been associated with tumour suppressor genes, such as *p53*, a suppressor gene that has been associated with sarcoma and cancer of the breast and brain.
- The diagnosis and treatment of cancer is a pivotal life-changing event that prompts individuals to make immediate and ongoing adjustment to this life-threatening illness.
- Effective physical and psychosocial adjustment to cancer diagnosis and treatment has been shown to lead to successful completion of treatment, enhancement of the person's ability to cope with disease, improvement of the person's quality of life and, ultimately, improvement of survival.
- The goals of cancer treatment are cure and control of cancer, as well as management of cancer-related and treatment-related symptoms.
- Chemotherapy uses cytotoxic medications to cure or control cancer by interrupting cell metabolism and replication and by interfering with the ability of the malignant cell to synthesise vital enzymes and chemicals.
- Pain management is an important component of care for people with cancer. It is estimated that 20% to 50% of people with early-stage cancer and up to 95% of people with advanced cancer experience pain.
- Complementary therapies are therapies that people choose as a complement to medical treatment. Common complementary therapies for cancer include botanical agents, nutritional supplements, dietary regimens, mind–body modalities, spiritual approaches and miscellaneous therapies.
- Tumour lysis syndrome (TLS), a combination of two or more metabolic abnormalities, is a life-threatening emergency for people with cancer. People at risk of TLS include those with bulky chemosensitive cancer such as high-grade lymphomas and acute leukaemia, elevated serum uric acid, potassium and phosphorus, and renal deficiency.

CONCEPT CHECK

1 Mr Lawrence has a history of colon cancer. He has been advised that the cells from the colon tumour have travelled to his liver. This process is called:
1. carcinogenesis
2. dysplasia
3. metastasis
4. mutation

2 A person diagnosed with lung cancer reports they are having difficulty sleeping and often feel tense. The most appropriate initial nursing intervention would be to:
1. encourage the person to express his feelings about the cancer diagnosis
2. document the person's report of difficulty sleeping and tenseness in the chart
3. obtain an order for medication for sleep from the doctor
4. offer an anti-anxiety drug such as Ativan (lorazepam)

3 Mr Palacci is receiving external radiation for treatment of lung cancer. Educating Mr Palacci to care for his skin in the marked area includes:
1. apply antibacterial ointment daily
2. avoid contact with others
3. avoid rubbing or scratching treated skin areas
4. cleanse the skin with plain water

4 Ms Hernandez complains of nausea and vomiting following her daily chemotherapy treatment. The most appropriate nursing intervention would be to:
1. keep Ms Hernandez on nil orally until her daily chemotherapy is completed
2. provide anti-emetic medication 30 to 40 minutes prior to each treatment
3. provide clear liquids until the chemotherapy is completed
4. schedule chemotherapy administration for bedtime

5 Mrs Smith experiences bone marrow depression as a result of chemotherapy. Which of the following would the nurse expect to find?
1. alopecia
2. nausea and vomiting
3. platelet count 50
4. temperature 38.9°C

6 Mr Wu, a 46-year-old businessman with a diagnosis of metastatic lung cancer, is going to have chemotherapy tomorrow. To help Mr Wu better understand chemotherapy, you have provided him with education about the role of the chemotherapeutic agents being used to treat his cancer. You determine that your teaching is effective when Mr Wu states:
1. 'Chemotherapy uses drugs that promote the normal growth of cells while killing the cancer cells.'
2. 'Chemotherapy only uses a single drug to treat cancer because drug resistance is rare.'
3. 'Chemotherapy includes drugs that not only attack cancer cells but also normal rapidly dividing cells.'
4. 'Chemotherapy is a preferred therapy because it has fewer adverse effects than radiation therapy.'

7 During training for new radiation nurses, you have learned that the delivery of high-energy radiation (e.g. electrons, x-rays, photons) to kill cancer cells by using a machine to focus a beam of radiation on the body is called:
1. external radiation therapy
2. internal-beam radiation therapy
3. brachytherapy
4. biochemotherapy

8 You are taking care of a person who just received the first cycle of chemotherapy for acute leukaemia 2 days ago. As an oncology nurse, you are closely monitoring the person's laboratory tests of uric acid, potassium, phosphorus and calcium as you are aware the person is at risk of:
1. spinal cord compression
2. tumour lysis syndrome
3. septic shock
4. superior vena cava syndrome

9 In which phase of the cell cycle does the DNA replicate to form two sets of chromosomes?
1 G_1
2 G_2
3 S
4 M

10 Oncogenes are genes that:
1 promote cell growth when activated
2 block cell growth
3 stimulate a complex signalling process
4 are strictly regulated

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UNIT 3 BUILDING CLINICAL COMPETENCE

Pathophysiology and patterns of health

CLINICAL SCENARIO

You have been assigned to work with the following four people for the 0700 shift on a medical–surgical unit. Significant data obtained during report are as follows:

- Allen Barber is a 55 year old with diabetes mellitus who is 4 days postoperative abdominal surgery with an inflammation of the incision site. Vital signs are T 38.3°C, P 94, R 24, BP 138/82. The abdominal incision appears red with warmth and oedema around the incision. Mr Barber states that his pain level is 8 on a pain scale of 1 to 10. Labs and wound cultures have been ordered.
- Tamra Sanders is a 22 year old with Down syndrome. She is admitted in sickle cell crisis with T 38.9°C, P 90, R 30 and shallow and BP 110/84. Tamra is complaining of severe chest pain with shortness of breath. She states that her pain scale level is 10 of 10. She has an order to begin morphine PCA.
- Mia Windham is a 26 year old who was admitted yesterday with a maculopapular rash on the hands and feet that is spreading to the arms and legs. This morning she is complaining of abdominal pain, nausea and bloody diarrhoea. She has a history of having a bone marrow transplant 3 months ago as treatment for leukaemia.
- Harry Anderson is a 40 year old in the late stages of AIDS. He is confused, incontinent and has severe spasticity. He is on seizure precautions. He needs to be turned every 2 hours to prevent pressure sores. He is currently yelling that he needs help.

Critical thinking questions

1 In what order would you visit these people after report?

1. _____
2. _____
3. _____
4. _____

2 What would you prioritise as the two most important nursing diagnoses for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Allen Barber		
Tamra Sanders		
Mia Windham		
Harry Anderson		

3 In which position does the nurse place the person with hypovolaemic shock?

1. semi-Fowler's position with legs straight
2. Trendelenburg position with legs elevated 10 degrees
3. left lateral position with legs bent towards chest
4. supine position with legs elevated 20 degrees

4 Tamra Sanders' family asks the nurse how sickle cell anaemia is transmitted from one family member to another. Which statement by the nurse is the correct response?

1. 'The mother carries the gene for sickle cell anaemia and passes it to the children.'
2. 'The father carries the gene for sickle cell anaemia and passes it to the children.'
3. 'Both parents carry the gene for sickle cell anaemia and children have a 25% chance of getting the disease process.'
4. 'One parent has the disease and one parent carries the affected gene and they have a 50% chance of passing it to the children.'

5 Mr Anderson, who has AIDS, has experienced weight loss. The dietitian teaches him meal planning in which type of diet?

1. high protein, high fibre
2. high protein, high kilojoule
3. low fibre, low protein
4. high carbohydrate, high vitamins

6 The nurse performs wound cleansing with which procedure?

1. Cleanse the wound with soap and water.
2. Use normal saline to cleanse the wound.
3. Cleanse the wound with povidone-iodine.
4. Hydrogen peroxide (half strength) is used to cleanse the wound.

7 Prior to administering cytotoxic agents, such as cyclophosphamide (Cytoxan), the nurse needs to notify the doctor of which lab results?

1. haemoglobin of 108 g/L, haematocrit of 35%
2. potassium of 3.4 mmol/L, sodium of 130 mmol/L
3. creatinine of 2 mg/dL, blood urea nitrogen of 3.0 mmol/L
4. white blood cell count of 3.9 ($\times 10^9$/L), platelets of 74 ($\times 10^9$/L)

8 Due to diarrhoea, Ms Windham's arterial blood gas results are pH, 7.30, $PaCO_2$, 35 mmHg; PaO_2, 90 mmHg, HCO_3^-, 19 mEq/L. The nurse interprets these results as indicating the person has:

1. metabolic acidosis
2. metabolic alkalosis
3. respiratory acidosis
4. respiratory alkalosis

9 Which laboratory studies would you expect to draw on a person who is 4 days postoperative with an inflammation of the incision site?

1. white blood cell count/differential, erythrocyte sedimentation rate, C-reactive protein
2. troponins, metabolic panel for electrolytes, cultures of wound site
3. blood cultures, haematocrit and haemoglobin, blood glucose level
4. full blood count, alkaline phosphatase, urine creatinine and blood urea nitrogen

10 The person is admitted to the emergency department for a severe anaphylactic reaction to aspirin. Which medication is ordered to be administered?

1. 0.5 mL of 1:1000 adrenaline subcutaneously
2. 0.3 mL of 1:10 000 adrenaline subcutaneously
3. intravenous infusion of 1:10 000 adrenaline
4. intravenous infusion of 1:100 000 adrenaline

11 When administering a blood transfusion, which manifestations indicate a haemolytic reaction to the blood being administered?

1. abdominal cramps and diarrhoea
2. bradycardia and hypertension
3. dyspnoea and hypotension
4. diaphoresis and tachycardia

12 The nurse educates people and families to decrease risk factors of cancer by following which cancer prevention recommendations? (Select all that apply.)

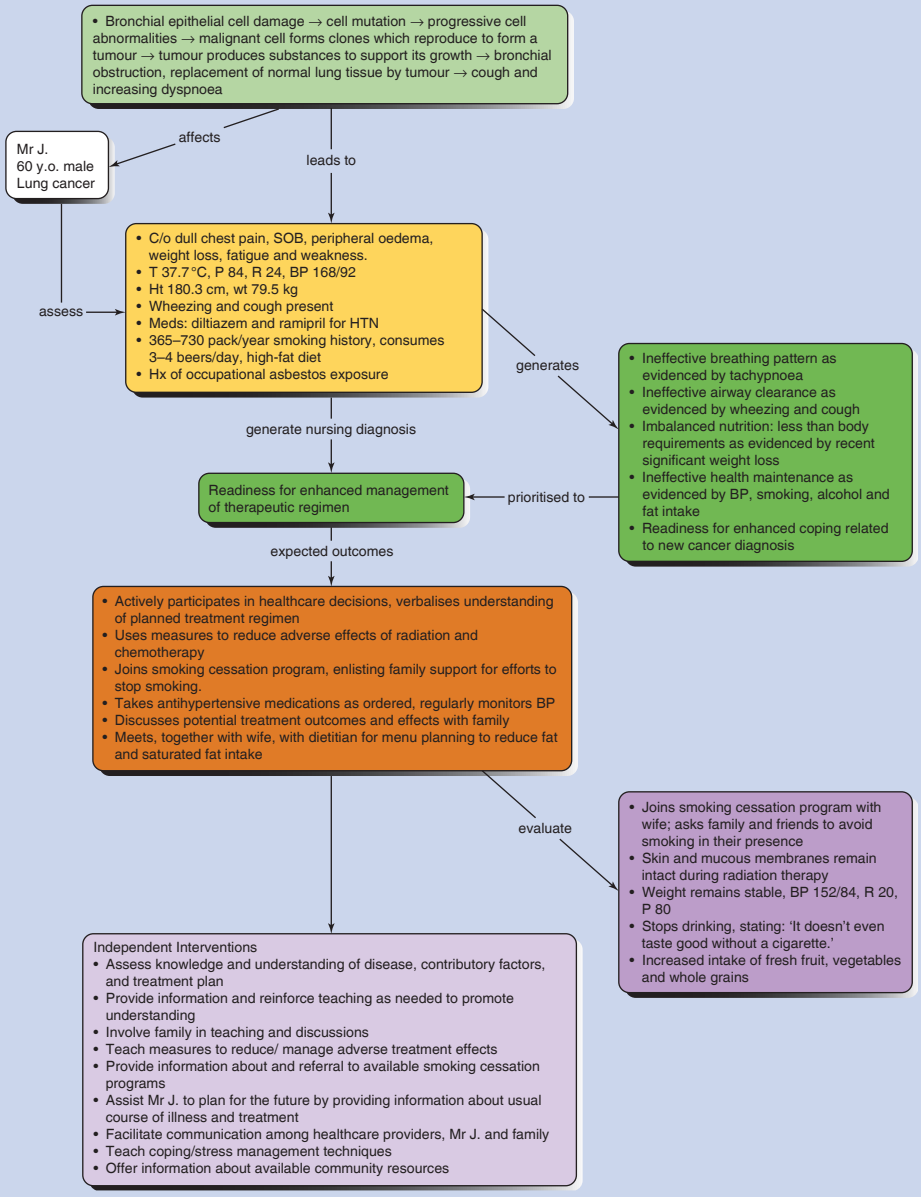
1. Avoid tobacco and excessive alcohol use.
2. Eat a diet low in fat and high in carbohydrates.
3. Increase intake of vitamins A, D, E and K.
4. Limit exposure in sun from 11 am to 4 pm.
5. Increase fruit and vegetables in the diet.
6. Eat meats grilled over a charcoal fire instead of fried.

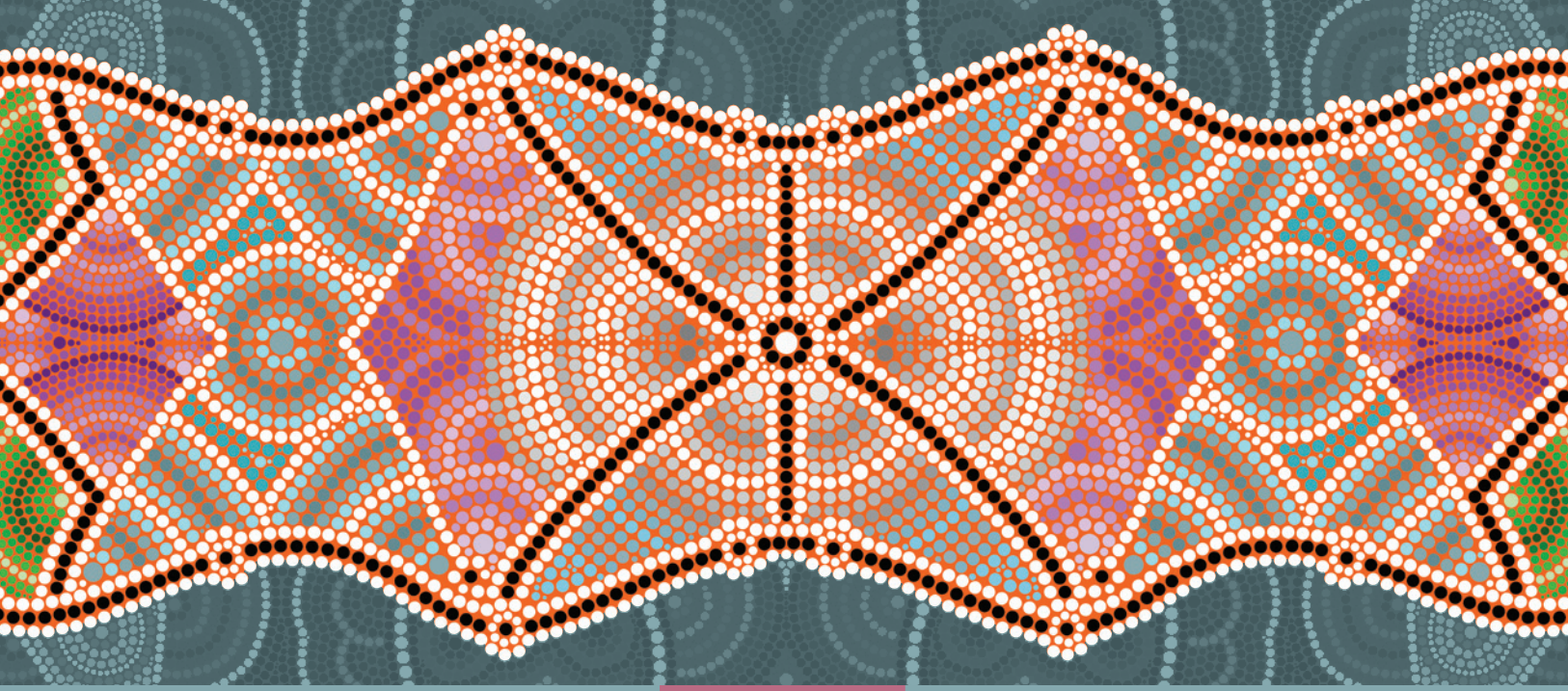
CASE STUDY

Mr Johnson is a 60-year-old construction worker who has presented at the doctors complaining of dull chest pain, shortness of breath, swelling of his hands and feet, weight loss, fatigue and weakness. On physical assessment, vital signs are temperature 37.7°C, pulse 84, respirations 24, blood pressure 168/92. His height is 180.3 cm and weight is 79.5 kg. Mr Johnson states that this is a loss of 15.9 kg during the past 3 months. Wheezing is heard when breath sounds are auscultated. Coughing is noted with deep breathing. The remainder of the physical assessment is unremarkable. He has a medical history of high blood pressure for which he takes diltiazem and ramipril. He has a history of smoking 1 to 2 packets of cigarettes a day since he was 15 years old. He states he has been exposed to asbestos in his employment. Mr Johnson's nutrition assessment indicates that his diet consists of fried meats (especially chicken), green vegetables cooked in pork fat, eggs and bacon for breakfast, and during breaks he eats biscuits. His fluid intake consists of coffee for breakfast and during breaks, soft drinks at lunch, and 3 to 4 beers at night.

Blood is drawn for a full blood count, electrolytes, blood glucose, calcitonin, CEA, haptoglobulin, GGT and creatinine. A sputum specimen is sent to the laboratory. A chest x-ray and CT scan are done. Based on the results of the chest x-ray, bronchoscopy and needle aspiration biopsies are performed to confirm a diagnosis of lung cancer. The oncologist recommends an initial treatment plan of radiation therapy followed by combination chemotherapy to reduce the tumour size prior to surgical resection of the tumour.

Based on Mr Johnson's medical diagnosis and treatment plan, *Readiness for enhanced therapeutic regimen management* is identified as the priority nursing diagnosis at this time.





UNIT 4

RESPONSES TO ALTERED INTEGUMENTARY STRUCTURE AND FUNCTION

CHAPTER 14

ASSESSING THE INTEGUMENTARY SYSTEM

KERYLN CARVILLE, KERRY REID-SEARL, KATE CROWLEY

KEY TERMS

alopecia 426
cyanosis 414
ecchymoses 421
erythema 414
hirsutism 426
jaundice 414
keratin 411
melanin 411
oedema 425
pallor 414
sebum 413
urticaria 421
vitiligo 421

LEARNING OUTCOMES

- Describe the anatomy, physiology and functions of the skin, hair and nails.
- Explain the integumentary assessment process, including the normal and abnormal findings which may arise.

CLINICAL COMPETENCIES

- Conduct and document a health history for people who have or are at risk of alterations in the skin, hair or nails.
- Conduct and document a physical assessment of the integumentary system.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Disposable gloves
- Ruler
- Torch or good light source

The skin and its accessory appendages, which include hair, nails and various glands, make up the integumentary system. The skin, the largest organ of the body, provides an external covering for the body, separating and protecting the body's organs and tissues from the external environment. Compared with all other body organs, it is the most exposed to infection and injury. In the average adult the skin receives one-third of the circulating blood volume. The pH of the skin is slightly acidic, ranging from 4.2 to 6, which ensures an 'acid mantle' for protective purposes and maintenance of normal skin flora. (Functions of the skin and its accessory structures are summarised in Table 14.1.)

Disorders of the integumentary structures may be caused by a variety of factors including environmental factors, allergies, infection, infestation, disease, malignancy, trauma and genetic influences.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE INTEGUMENTARY SYSTEM

The skin

The skin has an average total surface area of 7600 cm^2. It has been estimated that in 1 cm^2 there is almost 1 metre of blood vessels, 4 metres of nerves, 100 sweat glands, 15 sebaceous glands, 3000 sensory cells at the end of nerve fibres, 25 pressure apparatuses to record tactile stimuli, 200 nerve endings to record pain, 2 sensory apparatus for cold, 12 sensory apparatus for heat, 300 000 epidermal cells and 10 hairs (Klein, 1988). The appearance of the integumentary system is dependent upon age, ethnicity, general health and wellbeing, and cultural and occupational pursuits. Structurally, the skin consists of the epidermis, the dermis and the hypodermis (see Figure 14.1). Each layer will be discussed in the following sections of this chapter.

The epidermis

The epidermis, which comprises the surface or outermost part of the skin, consists of stratified squamous epithelium. It is avascular and is approximately 0.04 mm thick (Carville, 2012). The epidermis receives its nutrients from the dermal layer beneath it. The epidermis regenerates itself, and the normal epidermal turnover time of epidermal cells as they migrate from the stratum germinativum to the stratum corneum and are then shed is up to 28 days. The epidermis has either four or five layers, depending on its location; there are five layers over the palms of the hands and the soles of the feet, and four layers over the rest of the body.

The stratum germinativum, also known as the stratum basale, is the deepest layer of the epidermis. It is comprised of a single layer of basal cells that are mitotically active and constantly dividing. It contains melanocytes, cells that produce the pigment **melanin**, and keratinocytes, which produce **keratin**. Melanin forms a shield to protect the keratinocytes and the nerve endings in the dermis from the damaging effects of ultraviolet light. Melanocyte activity probably accounts for the difference in skin colour in humans. Keratin is a fibrous, water-repellent protein that gives the epidermis its tough, protective quality. As keratinocytes mature, they move upwards through the epidermal layers, eventually becoming dead cells at the surface of the skin. Millions of these cells are worn off by abrasion each day; simultaneously millions more are produced in the stratum germinativum.

TABLE 14.1 Functions of the skin and its appendages

STRUCTURE	FUNCTIONS
Epidermis	Protects tissues from physical, chemical and biological damage. Prevents water loss and serves as a water-repellent layer. Stores melanin, which protects tissues from harmful effects of ultraviolet radiation in sunlight. Converts cholesterol molecules to vitamin D when exposed to sunlight. Contains phagocytes, which prevent bacteria from penetrating the skin.
Dermis	Contains blood vessels, which supply nutrients to the stratum germinativum of the epidermis and the skin appendages. Regulates body temperature by dilating and constricting capillaries. Contains specialised sensory receptors and nerves, which transmit sensation signals to the central nervous system.
Hypodermis	Main support system for the skin. Protective layer for underlying organs and structures. Participates in temperature regulation. Storage depot for fat.
Sebaceous (oil) glands	Secrete sebum, which lubricates skin and hair. Play a role in maintaining the pH of the skin.
Eccrine sweat glands	Present at birth and regulate body heat by excretion of perspiration.
Apocrine sweat glands	Remnant of sexual scent gland, activated at pubescence.
Hair	Cushions the scalp. Eyelashes and cilia protect the body from foreign particles. Provides insulation in cold weather.
Nails	Protect the fingers and toes, aid in grasping and allow for various other activities, such as scratching the skin, picking up small items and so on.

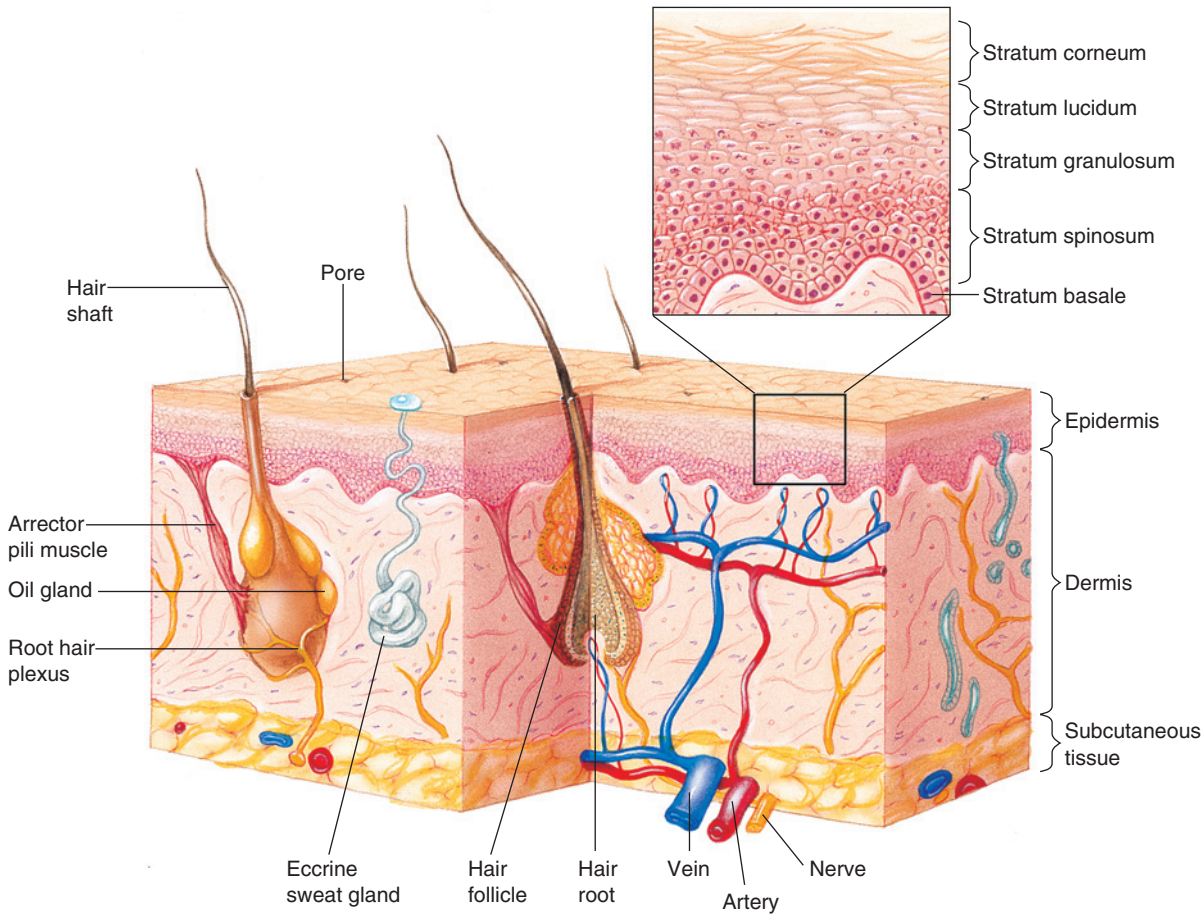


FIGURE 14.1 ■ Anatomy of the skin

The next layer of the epidermis is the stratum spinosum or prickle cell layer, which is composed of several stratified layers of polygonal cells which are attached by desmosomes, which appear as spiny processes. Mitosis occurs at this layer, although not as abundantly as in the stratum germinativum.

The stratum granulosum, the next layer, is two to three cells thick. The cells of the stratum granulosum contain a glycolipid that slows water loss across the epidermis. As cells migrate from stratum spinosum to stratum granulosum the nucleus is destroyed by enzymes, the cells flatten and cellular contents are converted into tough, insoluble keratin. Keratinisation, a thickening of the cells' plasma membranes, begins in the stratum granulosum.

The stratum lucidum is a transparent layer with no visible nuclei and is present only in areas of thick skin—for example, on the soles or palms. The outermost layer of the epidermis, the stratum corneum, is also the thickest, making up about 75% of the epidermis' total thickness. It consists of about 20 to 30 layers of dead cells filled with keratin fragments arranged in 'shingles' that flake off as dry skin.

THE BASEMENT MEMBRANE The basement membrane is an acellular, non-vascular and non-innervated membrane that separates the epidermis from the dermis. The basement membrane serves as an adherent and mechanical support layer between the epidermis and the dermis (Martini, Nath and Bartholomew, 2012). It is also thought to play a role in regulating the transfer of proteins, oxygen and nutrients across the dermal–epidermal junction (Carville, 2012).

The dermis

The dermis is attached to the basement membrane, but is often referred to as the second, deeper layer of skin. Made of a flexible connective tissue, this layer is richly supplied with blood cells, nerve fibres and lymphatic vessels. The hair follicles, sebaceous glands and sweat glands are located in the dermis, although they are derived from the epidermal layer. The dermis consists of a papillary (upper layer) and a reticular layer (deeper layer). The papillary layer contains reti ridges that indent the overlying epidermis. It also contains capillaries and receptors for pain and touch. The deeper, reticular layer contains blood vessels, sweat and sebaceous glands, deep pressure receptors and dense bundles of collagen fibres. The regions between these bundles form lines of cleavage in the skin, which are referred to as Langer's lines. Surgical incisions parallel to these lines of cleavage heal more easily and with less scarring than incisions or traumatic wounds across cleavage lines.

The hypodermis

The hypodermis (also called the subcutaneous layer) contains large blood vessels that supply the skin. Additionally, it serves as a storage depot for fat and thus contains adipose tissue as well as connective tissue. The function of the hypodermis is to insulate, afford protection to underlying structures and regulate temperature. The hypodermis plays a significant role as it acts as a support framework for the skin. This is because the fibres that extend from the dermis anchor the skin to the hypodermis, which then attaches to the underlying fascia (the connective tissue around muscles and bones) (Jenkins, Kemnitz & Tortora, 2010).

Skin appendages

Appendages of the skin include the glands (sebaceous, sudoriferous and ceruminous), the hair and the nails. The following section explains each appendage.

Glands of the skin

The skin contains sebaceous (oil) glands, sudoriferous (sweat) glands and ceruminous (cerumen or earwax) glands. Each of these glands has a different function.

Sebaceous glands enter halfway up the hair follicle; thus they are found all over the body except on the palms, soles, lips and nipples where there are no hair follicles. These glands secrete an oily substance called **sebum**, which is usually ducted into a hair follicle. Sebum softens and lubricates the skin and hair and also decreases water loss from the skin in low humidity. Sebum and sweat influence the pH of the skin, and the 'acid mantle' or slightly acidic pH protects the body against opportunistic infection. The secretion of sebum is stimulated by hormones, especially androgens. If a sebaceous gland becomes blocked, a pimple or whitehead appears on the surface of the skin; as the material oxidises and dries, it forms a comedone or blackhead. Acne vulgaris is an inflammation of the sebaceous glands.

There are two types of sweat glands: eccrine and apocrine. Eccrine sweat glands are present at birth and are more numerous on the forehead, palms and soles. The gland itself is located in the dermis; the duct to the skin rises through the epidermis to open in a pore at the surface. The secretion of the eccrine glands is composed mostly of water, but it also contains sodium, antibodies, small amounts of metabolic wastes, lactic acid and vitamin C. The production of sweat is regulated by the sympathetic nervous system and serves to maintain normal body temperature. Sweating also occurs in response to emotions.

Apocrine sweat glands are activated at pubescence. Most apocrine sweat glands are located in the axillary, anal and genital areas. The secretions from apocrine glands are similar to those of eccrine sweat glands, but they also contain fatty acids and proteins. Apocrine glands are a remnant of sexual scent glands.

Ceruminous glands are modified apocrine sweat glands. Located in the skin of the external ear canal, they secrete yellow-brown waxy cerumen. This substance provides a sticky trap for foreign materials.

The hair

Hair is distributed all over the body, except the lips, nipples, parts of the external genitals, the palms of the hands and the soles of the feet. Hair is composed of keratin. It is produced by a hair bulb and its root is enclosed in a hair follicle (see Figure 14.2). The exposed part, called the shaft, consists mainly of dead cells. Hair follicles extend into the dermis and in some places, such as the scalp, below the dermis. Many factors, including nutrition and hormones, influence hair growth. Humans have three types of hair. They are:

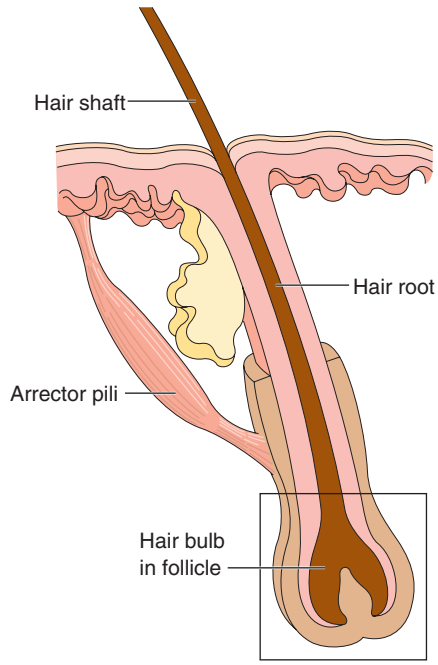


FIGURE 14.2 ■ Anatomy of a hair follicle

1. lanugo, the fine hair found on the body of premature neonates
2. vellus, the fine lightly coloured body hair that grows to 1 cm
3. terminal hair, which is found on the scalp, face of males, pubic region and armpits, and can grow to 50 cm.

Hair in various parts of the body has protective functions: the eyebrows and eyelashes protect the eyes; hair in the nose helps keep foreign materials out of the upper respiratory tract; and hair on the head protects the scalp from heat loss and sunlight. On average, an adult loses 50–100 head hairs a day. Scalp hair can last 2–4 years and eyelashes tend to last 3–5 months.

The nails

A nail is composed of keratin. Like hair, nails consist mainly of dead cells. They arise from the stratum germinativum of the epidermis. The body of the nail rests on the nail bed (see Figure 14.3). The nail matrix is the active, growing part of the nail. The proximal visible end of the nail has a white crescent, called a lunula. The sides of the nail are overlapped by skin, called nail folds. The proximal nail fold is thickened and is called the eponychium or cuticle. Nails form a protective coating over the dorsum of each digit on the fingers and toes.

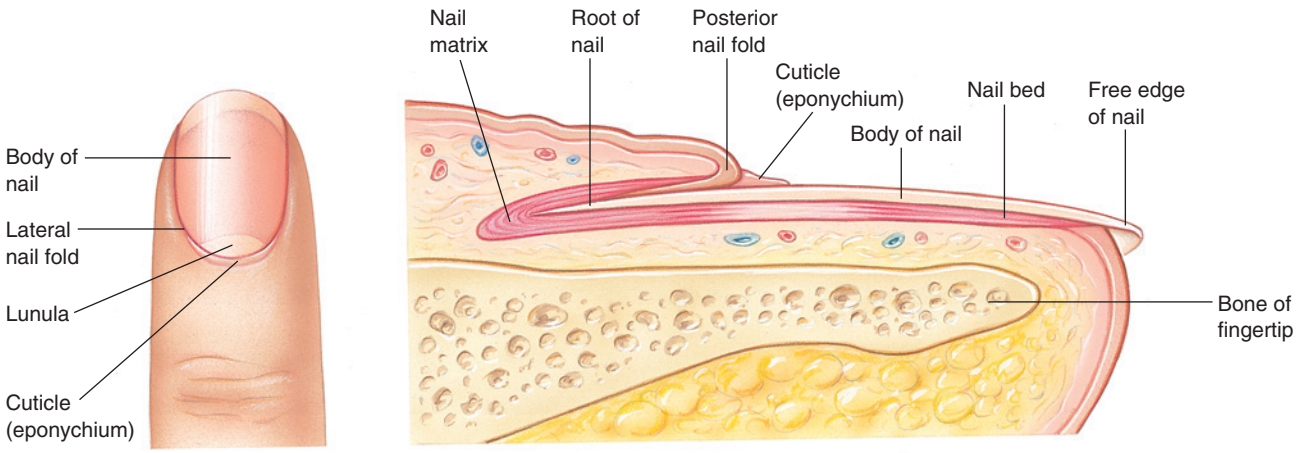


FIGURE 14.3 ■ Anatomy of a nail (frontal and side views)

Skin colour

Skin colour varies among individuals and among people of different races, ranging from a pinkish white to various shades of brown and black. Areas of the skin that are normally exposed to the sun and environment, such as the face and hands, may have a slightly different colour from areas that are usually covered with clothing. Special care must be taken when assessing changes in skin colour in people with darker skin, such as Indigenous Australians, Pacific Islanders, Asians, people of Mediterranean descent and Caucasians who are deeply suntanned.

Skin colour is the result of varying levels of pigmentation. Melanocytes produce melanin, a yellow-to-brown pigment, which is predominately responsible for skin tone and protects against damage from ultraviolet rays. All races have the same number of melanocytes, but melanin is produced in greater amounts in people with dark skin colour. Exposure to the sun causes a build-up of melanin and a darkening or tanning of the skin in people with light skin. Carotene, a yellow-to-orange pigment, is found mostly in areas of the body where the stratum corneum is thickest, such as the palms of the hands. Carotene is more abundant in the skins of people of Asian ancestry and, together with melanin, accounts for their golden skin tone. The epidermis in Caucasian skin has very little melanin and is almost transparent. Thus, the colour of the haemoglobin found in red blood cells (RBCs) circulating through the dermis shows through, lending Caucasians a pinkish skin tone.

Skin colour is influenced by emotions and illnesses. **Erythema**, a reddening of the skin, may occur with embarrassment (blushing), fever, hypertension or inflammation. It may also result from a drug reaction, sunburn, acne rosacea or other factors. A bluish discolouration of the skin and mucous membranes, called **cyanosis**, results from poor oxygenation of haemoglobin. **Pallor**, or paleness of skin, may occur with shock, fear or anger, or be a sign of anaemia and hypoxia. **Jaundice**, a yellow-to-orange colour visible in the skin and mucous membranes, is most often the result of a hepatic disorder or physiological jaundice in the newborn. Table 14.2 further defines these terms and compares and contrasts skin colour changes in people with light and dark skin.

ASSESSING THE INTEGUMENTARY SYSTEM

The functions of the integumentary system (skin, glands, hair and nails) are assessed by a health assessment interview to collect subjective data, a physical assessment to collect objective data and findings from diagnostic tests. See the box below for sample documentation of an assessment of the integument.

Health assessment interview

A health assessment interview to determine problems with the integumentary system may be conducted as part of a health screening or total health assessment, or it may focus on a chief complaint (such as itching or a rash). If the person has a skin problem, analyse its onset, characteristics and course, severity,

SAMPLE DOCUMENTATION

Assessment of the thyroid gland

21/01/2013
NURS 0900

A 27-year-old male with no history of skin lesions, hair loss or disorders of the nails. The person reports that he took an antibiotic for a respiratory infection approximately 10 days ago which resulted in the appearance of a fine, raised, red, itchy rash on his trunk and arms. The person further reports that he presented to the doctor, who prescribed an antihistamine and the rash cleared in 3 days. Upon current assessment the person's skin is light brown, warm, dry and supple. Patches of vitiligo are present over the dorsum of his hands. No lesions or oedema are noted. The person has a healed scar on his lower left abdomen (an appendectomy as a young adult). He has clean, dark brown hair which is greying at the temples. His nails are smooth, hard and immobile.———————— K Simpson
(KATE SIMPSON, RN)

TABLE 14.2 Skin colour assessment variations in people with light and dark skin

Pallor: *a decrease or absence in skin colour as the result of a decrease in tissue perfusion; a decrease in shape, size or amount of red blood cells (RBCs); or absence of melanin (local or generalised).*

DISORDER AND CAUSE	CHANGE IN LIGHT SKIN	CHANGE IN DARK SKIN
Anaemia (decreased or abnormal size and shape of RBCs)	Generalised paleness	Brown skin is dull and has a yellow cast; black skin is dull and has an ashen grey cast
Haemorrhage (decreased amount of circulating RBCs)	Generalised paleness	Brown skin is dull and has a yellow cast; black skin is dull and has an ashen grey cast
Shock (decreased amount of circulating RBCs or decreased perfusion)	Generalised paleness	Brown skin is dull and has a yellow cast; black skin is dull and has an ashen grey cast
Arterial insufficiency (trauma, acute arterial occlusion or arteriosclerosis)	Local paleness	Dull, ashen grey
Vitiligo (patchy loss of melanocytes)	Patches of white spots, most often found over skin of the face, hands or groin	Patches of white spots, most often found over skin of the face, hands or groin
Albinism (total absence of melanin)	White/pink	Light tan, cream or white

Cyanosis: *a bluish discolouration of the skin and mucous membranes resulting from a local or generalised excess of deoxygenated haemoglobin or a structural defect in the haemoglobin molecule.*

DISORDER AND CAUSE	CHANGE IN LIGHT SKIN	CHANGE IN DARK SKIN
Acute and chronic disorders of the structure and function of the heart and lungs (arterial insufficiency or respiratory distress). Peripheral cyanotic changes may also be due to exposure to cold or hypothermia)	Dusky blue (may be generalised or local, depending on cause)	Skin may appear darker, but will be dull; cyanosis is more readily assessed in the nail beds, oral mucous membranes and conjunctivae

Erythema: *redness of the skin or mucous membranes that is the result of dilatation and congestion of superficial capillaries.*

DISORDER AND CAUSE	CHANGE IN LIGHT SKIN	CHANGE IN DARK SKIN
Hyperaemia (inflammation, increased body temperature, hot environmental temperature, embarrassment, alcohol ingestion)	Red or bright pink	Difficult to assess; skin may have dark red cast
Carbon monoxide poisoning (carbon monoxide displaces oxygen on the haemoglobin molecule, causing hypoxia, carboxyhaemoglobinaemia)	Cherry red in face and upper torso	Cherry red lips, oral mucous membranes and nail beds
Pressure Reactive hyperaemia (a compensatory erythema in response to tissue pressure that fades when tissues are compensated). Non-reactive hyperaemia does not blanch after 30 minutes pressure relief and indicates a stage 1 pressure injury	Dusky red	Difficult to assess; may be warm to touch or bluish tint

Jaundice: *yellowish discolouration of the skin, mucous membranes and sclerae of the eyes, caused by increased amounts of bilirubin or other pigments such as elevated carotene in the blood.*

DISORDER AND CAUSE	CHANGE IN LIGHT SKIN	CHANGE IN DARK SKIN
Increased serum bilirubin to > 2–3 mg/100 mL or other pigments such as elevated carotene in haemolysis, such as following blood transfusion, severe burns or infections)	Yellowing of skin follows yellowing of sclerae and mucous membranes; may also be assessed in the fingernails and palms of the hands	Yellowing is best assessed at the junction of the hard palate and the soft palate or on the palms of the hands. Sclerae may be yellow near the limbus (do not confuse with normal yellow eye pigmentation)
Uraemia (retained urochrome pigments in the blood)	Orange-green or grey cast to skin. Yellowing of skin follows yellowing of sclerae and mucous membranes; may also be assessed in the fingernails and palms of the hands	Difficult to assess; may appear as yellowish green colour in the sclera
Physiological jaundice occurs in newborns around the third to fourth day and is due to haemolysing excess red cells	Obvious yellowing of the skin and sclerae	Yellowing is best assessed at the junction of the hard palate and the soft palate or on the palms of the hands. Sclerae may be yellow

precipitating and relieving factors, and note the timing and circumstances of any associated symptoms. For example, ask the person:

- When did the itching begin and how severe was it?
- When did you first notice a change in this mole?
- Did you change to any different kinds of shampoo or other hair products just before you started to lose your hair?

Ask about any change in health, rashes, itching, colour changes, dryness or oiliness, growth of or changes in warts or moles, and the presence of lesions. Precipitating causes, such as medications, the use of new soaps, skin care agents, cosmetics, pets, travel, stress or dietary changes, must also be explored. In assessing hair problems, ask about any thinning or baldness, excessive hair loss, change in distribution of hair, use of hair care products, diet and dieting. When assessing nail problems, ask about nail splitting or breakage, discolouration, change in shape, infection, diet and exposure to chemicals.

The person's medical history is important. Questions focus on identifying previous problems, allergies and the presence of lesions. Skin problems may be manifestations of other disorders, such as cardiovascular disease, endocrine disorders, hepatic disease and haematological disorders. The occupational and social history may provide cues to skin problems. It is important to ask the person about travel, exposure to toxic substances at work or socially, their use of alcohol and responses to stress. Family history may provide insight into hereditary-linked disorders such as atopic eczema or dermatitis (these terms are used interchangeably). Assess the presence of risk factors for skin cancer carefully. These include male gender; aged over 50; family history of skin cancer; extended exposure to sunlight; tendency to sunburn; history of sunburn or other skin trauma; light-coloured hair or eyes; residence in high altitudes or near the equator; and exposure to radiation, x-rays, coal, tar or petroleum products.

It is also important to explore the risk factors for malignant melanoma. These include the presence of a large number of moles, the presence of atypical moles, a family history of melanoma, prior melanoma, repeated severe sunburns, ease of freckling and sunburning, or inability to tan.

See 'Functional health pattern interview: integumentary system' below. Responses should be documented in the person's medical record.

Physical assessment

Physical assessment of the skin, hair and nails may either be performed as part of a total assessment, or it may be a focused assessment of the integument for people with known or suspected problems. Physical assessment of the skin, hair and nails is conducted using inspection and palpation techniques. Assess the skin for colour, presence and characteristics of lesions (observable changes from normal skin structure), temperature alterations, texture, moisture, turgor and presence of oedema (see Table 14.3).

TABLE 14.3 Age-related skin changes

AGE-RELATED CHANGE	SIGNIFICANCE
Epidermis: reduced thickness and miotic activity	• Skin is more fragile and at greater risk of tears or injury • Delayed wound healing • Hyperkeratoses and skin cancers in sun-exposed areas are more evident
Epidermis: increased permeability, reduced Langerhans cells	• Increased risk of reactions to irritants • Decreased inflammatory response
Epidermis: reduced number of active melanocytes	• Increased susceptibility to sun exposure
Epidermis: hyperplasia of melanocytes, especially in sun-exposed areas	• Small areas of hyperpigmentation (solar lentigines or 'liver spots')
Epidermis: impaired vitamin D production	• Increased risk of osteomalacia, osteoporosis
Epidermis: dermal–epidermal junction flattens	• Increased risk of skin tears, purpura and pressure injures
Dermis: reduced perfusion	• More susceptible to dry skin • Decreased sensation (pain, touch, temperature and peripheral vibration) • Increased risk of injury
Dermis: reduced vasomotor response	• Greater risk of hyperthermia and hypothermia
Dermis: elastic fibres degenerate	• Decreased tone and elasticity, with wrinkle formation
Dermis: proliferation of capillaries	• Cherry haemangiomas are common
Subcutaneous skin layer: thins	• Greater risk of hypothermia • Increased risk of pressure injuries
Subcutaneous skin layer: adipose tissue is redistributed	• Cellulite forms • Bags over and under the eyes • Double chin forms • Abdominal fat increases • Breasts sag • Skin returns to normal more slowly when pinched (tenting)
Glands: reduced eccrine and apocrine activity	• Dry skin is common • Reduced or absent perspiration

FUNCTIONAL HEALTH PATTERN INTERVIEW Integumentary system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Describe your current problem. How long has it lasted? What have you done to treat it? ■ Describe any past skin problems or injuries you have had. How were these treated? ■ Did you undergo any surgical procedures and, if so, why and when? ■ List prescribed or over-the-counter medications, herbs and vitamins you currently take. ■ Do you have allergies to plants, chemicals or pets? ■ Describe what you do each day to care for your skin, hair and nails.
Nutritional–Metabolic	■ Describe the type and amount of food and drink you consume in a 24-hour period. ■ Do you have a history of food allergies? If so, describe what you are allergic to and how you respond. ■ Have you recently eaten any new foods? ■ How well do your cuts and scratches heal?
Integumentary condition	■ Is your skin and scalp dry, oily or itchy? ■ Have you noticed swelling around your eyes or ankles? ■ Have you noticed any changes in your hair or nails? ■ Do you perspire a lot? ■ Do you bruise easily?
Activity–Exercise	■ Describe your physical activities in a typical day. ■ Do you use a sunscreen when you are outside? If so, what SPF? ■ Do you visit tanning salons?
Sleep–Rest	■ How many hours do you sleep each night? ■ Do you have trouble sleeping because of itching or sweating?
Pain and discomfort	■ Do you have any of the following: pain, discomfort, itching, tingling, burning, tenderness or numbness? If so, where?
Self-perception–Self-concept	■ How does this condition make you feel about yourself?
Role–Relationships	■ How does this condition affect your relationships with others? ■ Is there anything in your work environment that may have caused this condition?
Sexuality–Reproductive	■ Has this condition interfered with your usual sexual activities? ■ If you use a birth control method, could it have caused this condition?
Coping–Stress tolerance	■ Have you experienced any type of stress that may have worsened this condition? ■ Has this condition created stress for you? ■ Describe what you do when you feel stressed.
Value–Belief	■ Tell me how specific relationships or activities help you cope with this condition. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this condition. ■ Are there any specific treatments that you would not use to treat this condition?

Characteristics of lesions are described in Table 14.4.

Common skin lesions found on older adults are outlined in Box 14.1. Examine the hair for hygiene status, infestations, colour, texture, quality and distribution, and the presence of scalp lesions. Determine the shape, colour, contour and condition of the nails. Terminology of skin lesions with examples is outlined in Table 14.5.

The examination should be conducted in a warm, private room. The person should remove all clothing and put on a gown. The areas to be examined should be fully exposed, but protect the person's modesty by keeping other areas covered.

TABLE 14.4 Lesion characteristics

Configuration and shape	Single, grouped cluster, linear, annular (ring-shaped) or round, artiform (bite)
Appearance	Dimensions, raised, indented, flush, colour, mobile, non-mobile
Edges	Raised, rolled, undermined, coloured
Fluid-filled lesion or draining exudate	Colour, type, amount, consistency, odour

BOX 14.1 Common skin lesions of older adults

- Skin tags: soft brown or flesh-coloured benign papules
- Keratoses: horny growth of keratinocytes; may be seborrhoeic (benign) or actinic (premalignant)
- Lentigines ('liver' or 'age' spots); brown or black benign macules with a defined border
- Angiomas (haemangioma): benign vascular tumours with dilated blood vessels, found in the middle to upper dermis
- Telangiectases: single dilated blood vessels, capillaries or terminal arteries
- Venous lakes (phlebectases): small, dark blue, slightly raised benign papules that usually occur on sun-exposed areas
- Photoageing: wrinkling, mottling, pigmented areas, loss of elasticity, benign or malignant lesions

TABLE 14.5 Terminology of skin lesions with associated disorders

LESION	EXAMPLES OF DISORDERS
Pigmented	Freckle, seborrhoeic keratosis, naevus, melanoma
Scaly	Psoriasis, dermatitis, xerosis, tinea, actinic keratoses
Pustular	Acne vulgaris, folliculitis, candidiasis
Vesicular	Herpes simplex, herpes zoster, scabies
Nodular	Warts, basal cell carcinoma, acne
Weepy, crusted	Acute contact allergic dermatitis, impetigo
Figurate (shaped) erythema	Urticaria, cellulitis
Bullous	Pemphigus, toxic epidermal necrolysis
Pruritic	Xerosis, scabies, pediculosis
Ulcerated	Pressure injury, skin cancer, herpes simplex

The person may be standing, sitting or lying down at various times of the examination. The assessor should don disposable gloves when palpating open lesions, skin surfaces suspicious of infections or infestations, or discharge from lesions of the skin and mucous membranes. Standard precautions should be adhered to when conducting a skin assessment. A disposable ruler is used to measure the size of lesions. A torch is used to better visualise lesions.

Diagnostic tests

The results of diagnostic tests of the structure and function of the integumentary system are used to support the diagnosis of a specific injury or disease, to provide information to identify or modify the appropriate medication or treatments used to treat the disease and to help nurses monitor the person's responses to nursing care interventions. Diagnostic tests to assess the integumentary system are described in the following box and summarised in the 'Integumentary assessments' section. More information is given in Chapters 15 and 16.

The use of laboratory tissue analysis, exudate cultures or skin scrapings will not be routine for all wounds but is restricted to situations when clinical assessment indicates infection to be the cause of delayed healing. Conditions that indicate the need for investigation include:

- acute or chronic wounds that demonstrate signs of infection (erythema, swelling, heat, pain and increased exudate or purulence) and that are not responding to standard management
- a clinical diagnosis of infection has been made and drug sensitivities are required to ensure correct coverage for the organisms in the wound
- infected chronic wounds are deteriorating, despite appropriate debridement and antimicrobial treatment
- local surveillance is required to identify drug-resistant organisms (Expert Working Group, World Union of Wound Healing Societies, 2008; Young, 2012).

There are several recognised methods for collecting a tissue or fluid sample for diagnosing infection. A wound swab can be collected using a sterile swab for microscopy culture and this will identify the species and semi-quantitatively the number of organisms (normally reported as scant, low, moderate or heavy growth). This method does not accurately identify organisms in deeper tissues, and surface contamination results in over-reporting of organisms.

Although there are several methods reported for collecting a wound swab, there is no single, universally accepted method. Methods include:

- *Random swab sampling:* risks contamination with surrounding skin and topical isolates.
- *Levine method:* a swab is rotated over a 1 cm^2 area (necrotic tissue is debrided prior to this) with sufficient pressure to express fluid from within the wound tissue (Levine et al., 1976).
- *Zig-zag stroke method:* rotation of the collection swab between the fingers as the swab is manipulated from close to wound margin to margin in a 10-point zig-zag (Z) fashion (Angel et al., 2011; Cuzzell, 1993).

The Levine technique is considered to be superior to the Z technique because of possible sampling of a greater concentration of microorganisms from both the surface and slightly below the surface of the wound (Angel et al., 2011).

The wound should be cleaned with sterile water or normal saline prior to taking any wound swab in order to minimise the risk of contamination. Topical antiseptics should be avoided prior to taking the wound swab as they may alter the result. Avoid the use of topical anaesthetics such as Emla which can reduce the number of bacteria and alter the result (Batai et al., 2004).

DIAGNOSTIC TESTS The integumentary system: tissue biopsy

There are several methods to obtain a tissue biopsy.

NAME OF TEST: Punch biopsy

PURPOSE AND DESCRIPTION Used to obtain a tissue sample for histological study. Helpful in diagnosing pathology which lies in the epidermis, dermis and subcutaneous tissue. Best biopsy technique for undiagnosed rashes and suspected neoplasms.

Punch biopsy instrument is used to remove a small section of epidermis, dermis and subcutaneous tissue. Depending on the size of the biopsy removed, the incision may need to be sutured.

RELATED NURSING CARE Explain procedure to the person and ensure a consent form is signed (if required). Assist with procedure. Apply appropriate dressing and provide information about self-care. If sutures are used, advise the person when to return for suture removal. Document procedure and send labelled specimen to the laboratory.

NAME OF TEST: Incisional skin biopsy

PURPOSE AND DESCRIPTION Used to excise a section of tissue from the wound bed and wound edge.

Incision is made using a scalpel extracting a block of tissue, quadrangular at the skin surface. Biopsy rarely needs to be more than 5 mm deep. The incision is closed with sutures.

RELATED NURSING CARE As for a punch biopsy.

NAME OF TEST: Excisional skin biopsy

PURPOSE AND DESCRIPTION Incision is made using a scalpel and the *entire* skin lesion or tumour is removed for analysis. Excision is closed with sutures.

RELATED NURSING CARE As for a punch biopsy.

NAME OF TEST: Shave biopsy

PURPOSE AND DESCRIPTION Helpful in diagnosing diseases where the pathology is in or near the epidermis. Lumps and bumps as opposed to rashes are best biopsied with the shave technique.

Using a scalpel or curette, a slice of the top layer of the raised area is taken. Stitches are not usually required.

RELATED NURSING CARE Individual should be advised to clean wound with tap water and apply dressing to keep the wound moist. Change as required generally twice a week initially, then weekly until healed.

NAME OF TEST: Skin scrapings

PURPOSE AND DESCRIPTION Useful where fungal infections and some parasite infestations are suspected.

Scalpel is used to collect the sample and the sample is transported dry.

RELATED NURSING CARE As for a punch biopsy.

NAME OF TEST: Immunofluorescent slides

PURPOSE AND DESCRIPTION Used to identify IgG antibodies (present in pemphigus vulgaris) and to identify varicella in skin cells (for herpes zoster). Skin or blood samples collected in a sterile specimen container. Procedure should be documented and specimen clearly labelled and sent to the laboratory where it will be placed on a slide and examined microscopically for analysis.

RELATED NURSING CARE As for a punch biopsy.

(continued)

DIAGNOSTIC TESTS The integumentary system: tissue biopsy (continued)

NAME OF TEST: Wood's lamp **PURPOSE AND DESCRIPTION** Used in the clinical setting and uses an ultraviolet light that causes certain organisms to fluoresce (such as *Pseudomonas* organisms and fungi). Skin is examined under a special lamp.	**RELATED NURSING CARE** Explain the procedure to the person. Document the procedure.
NAME OF TEST: KOH (potassium hydroxide) preparation **PURPOSE AND DESCRIPTION** Used to examine for a fungal infection. Specimen of hair or nails is obtained, placed in a sterile specimen-labelled container and sent with the supporting documentation to the laboratory for analysis. In the laboratory the specimen is placed on a slide, potassium hydroxide solution is added and the specimen is examined microscopically.	**RELATED NURSING CARE** Explain the procedure to the person. Assist with or obtain the specimen, document the procedure and send the labelled specimen to the laboratory for analysis.
NAME OF TEST: Tzanck test **PURPOSE AND DESCRIPTION** Used to diagnose herpes infections, but it does not differentiate herpes simplex from herpes zoster. Fluid and cells from the vesicles are obtained, sent to the laboratory where they are put on a slide, stained and examined microscopically.	**RELATED NURSING CARE** Explain the procedure to the person. Use sterile procedure to assist with or obtain the specimen. Document the procedure and send the labelled specimen to the laboratory for analysis.
NAME OF TEST: Patch test, scratch tests **PURPOSE AND DESCRIPTION** Used to determine a specific allergen. Small amount of the suspected material is placed on the skin under an occlusive bandage. Patch tests generally detect delayed allergic reactions. In a scratch test, a needle is used to prick a small amount of a suspected allergen into the skin of the forearm. The type of allergen is selected from the patient's history. A positive reaction will usually occur within 20–30 minutes after exposure in the form of itchy, raised red wheals.	**RELATED NURSING CARE** Explain the procedure to the person, including the need to return, usually in 48 hours, to have the patched area or scratched areas evaluated. Document the procedure.

A tissue biopsy can be collected using a punch or scalpel, and the numbers of organisms will be able to be quantified more accurately. This method requires technical expertise and is usually restricted to hospitals or doctors' rooms. Although considered the 'gold standard' approach by many, quantitative biopsy provides information from only a small area of the wound bed and can therefore have poor sensitivity and reliability in large wounds (Angel et al., 2011; Gjødsbøl et al., 2012; Rondas et al., 2013; Sibbald et al., 2003).

A needle aspiration of adjacent tissues for tissue fluid can be collected to identify tissue organisms. This method also requires technical expertise and, like biopsy, is usually restricted to hospitals and doctors' rooms. Increased pain can be associated with this technique and it can underestimate the number of organisms when compared to biopsy (Bowler, Duerden & Armstrong, 2001; Kingsley, 2003).

Skin scrapings may be collected for diagnosing fungal infection. Other tests used to identify infections include immunofluorescent studies, Wood's lamp, potassium hydroxide and the Tzanck test.

Allergies may be determined through analysis of patch tests or scratch tests.

Some studies are conducted to identify bacterial carriers. For example, if people have repeated bacterial skin infections or if a healthcare unit or agency experiences numerous bacterial infections of people, nasal cultures may be performed to determine if the people or the healthcare workers are carriers of the bacteria. Regardless of the type of diagnostic test, the nurse

is responsible for explaining the procedure and any special preparation needed; for assessing for medication use that may affect the outcome of the tests; for supporting the person during the examination as necessary; for documenting the procedures as appropriate; and for monitoring the results of the tests.

Genetic considerations

When conducting a health assessment interview and physical assessment, it is important for the nurse to consider genetic and ageing influences on the health of the adult.

Ascertain age and, during the health assessment interview, ask about integumentary disorders or abnormalities among immediate family members. During the physical assessment, assess for any manifestations that indicate a possible genetic disorder. If data are found that indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

INTEGUMENTARY ASSESSMENTS

Technique/normal findings	Abnormal findings
Inspect skin colour and note any odours coming from the skin. *Skin colour should be even, appropriate to the age and race of the person, without foul odours.*	■ A strong odour of perspiration may indicate poor hygiene and a need for health education. A foul odour may indicate a disorder of the sweat glands. ■ Pallor and/or cyanosis are seen with exposure to cold and with decreased perfusion and oxygenation. In cyanotic dark-skinned people, skin appears dull. Cyanosis may be more visible in the mucous membranes and nail beds of these people. ■ In dark-skinned people, jaundice may be most apparent in the sclerae of the eyes. ■ Redness, swelling and pain are seen with various rashes, inflammations, infections and burns. Superficial burns cause areas of painful erythema and swelling. Red, painful blisters appear in superficial partial-thickness burns, whereas white or blackened areas are common in deep partial-thickness or full-thickness burns. ■ **Vitiligo**, an abnormal loss of melanin in patches, typically occurs over the face, hands or groin. Vitiligo can occur at any age and is thought to be an autoimmune disorder.
Inspect the skin for lesions and alterations, including calluses, scars, tattoos and piercings. Include inspection of skin creases and folds. *Skin should be intact without abnormal lesions.*	Primary, secondary and vascular lesions are described and shown in Tables 14.6 to 14.8. ■ Pearly edged nodules with a central ulcer are seen in basal cell carcinoma. ■ Scaly, red, fast-growing papules are seen in squamous cell carcinoma. ■ Dark, asymmetric, multicoloured patches (sometimes moles) with irregular edges appear in malignant melanoma. ■ Circular lesions are usually present in ringworm and in tinea versicolor. ■ Grouped vesicles may be seen in contact dermatitis. ■ Linear lesions appear in poison ivy and herpes zoster. ■ **Urticaria** (hives) appears as patches of pale, itchy wheals in an erythematous area. ■ In psoriasis, scaly red patches appear on the scalp, knees, back and genitals. ■ In herpes zoster, vesicles appear along sensory nerve paths, turn into pustules and then crust over. ■ Bruises (**ecchymoses**) are raised bluish or yellowish vascular lesions. Multiple bruises in various stages of healing suggest trauma or abuse.
Palpate skin temperature. *Skin should be warm.*	■ Skin is warm and red in inflammation and is generally warm with elevated body temperature. ■ Decreased blood flow decreases the skin temperature; this may be generalised, as in shock, or localised, as in arteriosclerosis.

TABLE 14.6 Primary skin lesions

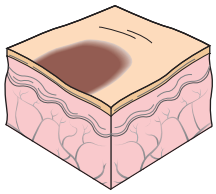
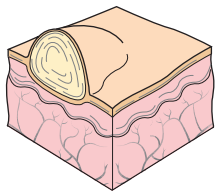
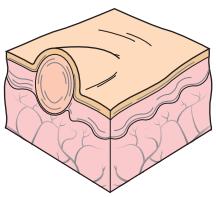
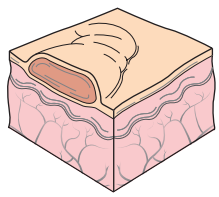
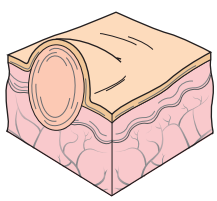
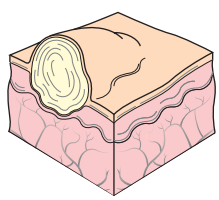
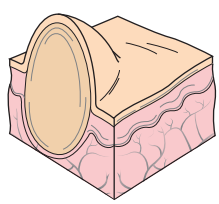
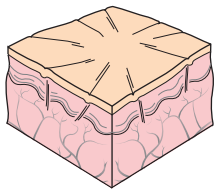
Macule, patch 	Flat, non-palpable change in skin colour. Macules are smaller than 1 cm, with a circumscribed border, and patches are larger than 1 cm and may have an irregular border. **Examples** Macules: freckles, measles and petechiae. Patches: Mongolian spots, port-wine stains, vitiligo and chloasma.	Vesicle, bulla 	Elevated, fluid-filled, round or oval shaped, palpable mass with thin, translucent walls and circumscribed borders. Vesicles are smaller than 0.5 cm; bullae are larger than 0.5 cm. **Examples** Vesicles: herpes simplex/zoster, early chickenpox and small burn blisters. Bullae: contact dermatitis, friction blisters and large burn blisters.
Papule, plaque 	Elevated, solid, palpable mass with circumscribed border. Papules are smaller than 0.5 cm; plaques are groups of papules that form lesions larger than 0.5 cm. **Examples** Papules: elevated moles, warts and lichen planus. Plaques: psoriasis, actinic keratosis and also lichen planus.	Wheal 	Elevated, often reddish area with irregular border caused by diffuse fluid in tissues rather than free fluid in a cavity, as in vesicles. Size varies. **Examples** Insect bites and hives (extensive wheals).
Nodule, tumour 	Elevated, solid, hard or soft palpable mass extending deeper into the dermis than a papule. Nodules have circumscribed borders and are 0.5 to 2 cm; tumours may have irregular borders and are larger than 2 cm. **Examples** Nodules: small lipoma, squamous cell carcinoma, fibroma and intradermal nevi. Tumours: large lipoma, carcinoma and haemangioma.	Pustule 	Elevated, pus-filled vesicle or bulla with circumscribed border. Size varies. **Examples** Acne, impetigo and carbuncles (large boils).
		Cyst 	Elevated, encapsulated, fluid-filled or semisolid mass originating in the subcutaneous tissue or dermis, usually 1 cm or larger. **Examples** Varieties include sebaceous cysts and epidermoid cysts.

TABLE 14.7 Secondary skin lesions

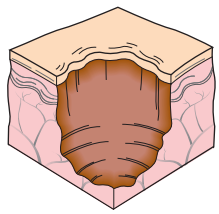
Atrophy



A translucent, dry, paper-like, sometimes wrinkled skin surface resulting from thinning or wasting of the skin due to loss of collagen and elastin.

Examples Striae, aged skin.

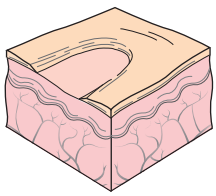
Ulcer



Deep, irregularly shaped area of skin loss extending into the dermis or sub cutaneous tissue. May bleed. May leave scar.

Examples Pressure injuries, venous stasis ulcers, chancres.

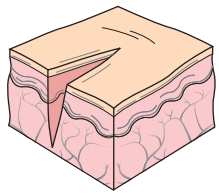
Erosion



Wearing away of the superficial epidermis causing a moist, shallow depression. Because erosions do not extend into the dermis, they heal without scarring.

Examples Scratch marks, ruptured vesicles.

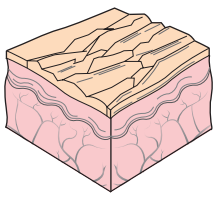
Fissure



Linear crack with sharp edges, extending into the dermis.

Examples Cracks at the corners of the mouth or in the hands, athlete's foot.

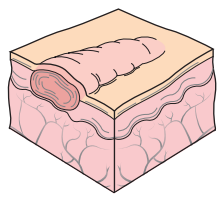
Lichenification



Rough, thickened, hardened area of epidermis resulting from chronic irritation such as scratching or rubbing.

Example Chronic dermatitis.

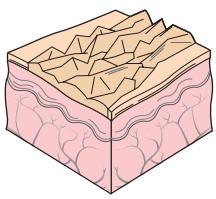
Scar



Flat, irregular area of connective tissue left after a lesion or wound has healed. New scars may be red or purple; older scars may be silvery or white.

Examples Healed surgical wound or injury, healed acne.

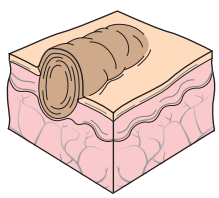
Scales



Shedding flakes of greasy, keratinised skin tissue. Colour may be white, grey or silver. Texture may vary from fine to thick.

Examples Dry skin, dandruff, psoriasis and eczema.

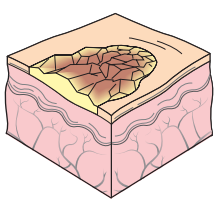
Keloid



Elevated, irregular, darkened area of excess scar tissue caused by excessive collagen formation during healing. Extends beyond the site of the original injury. Higher incidence in people of African descent.

Examples Keloid from ear piercing or surgery.

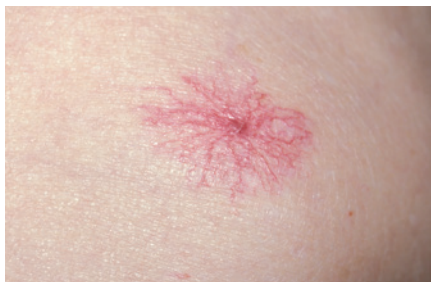
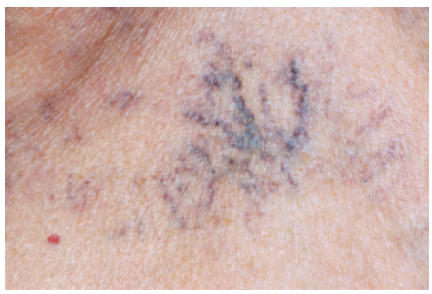
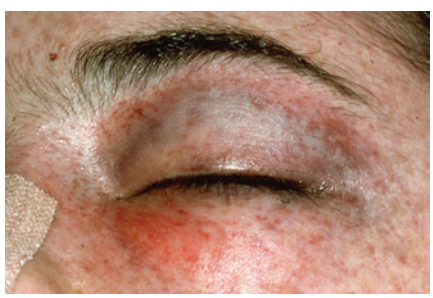
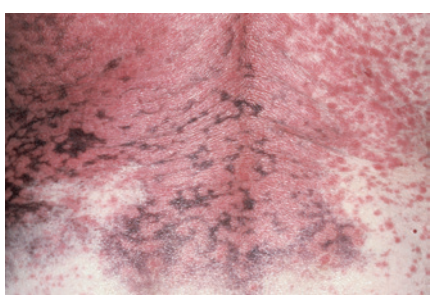
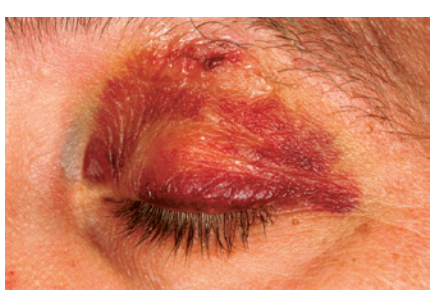
Crust



Dry blood, serum or pus left on the skin surface when vesicles or pustules burst. Can be red-brown, orange or yellow. Large crusts that adhere to the skin surface are called scabs.

Examples Eczema, impetigo, herpes or scabs following abrasion.

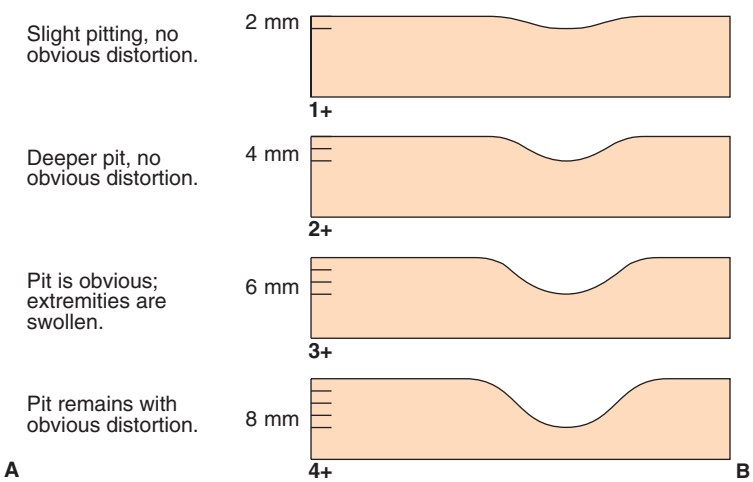
TABLE 14.8 Vascular skin lesions

Spider angioma	 *Source:* SPL/Custom Medical Stock Photo, Inc.	A flat, bright red dot with tiny radiating blood vessels ranging in size from a pinpoint to 2 cm. It blanches with pressure. **Cause** A type of telangiectasis (vascular dilatation) caused by elevated oestrogen levels, pregnancy, oestrogen therapy, vitamin B deficiency or liver disease, or may not be pathological. **Localisation/distribution** Most commonly appear on the upper half of the body.
Venous star	 *Source:* Photo Researchers, Inc./Science Source.	A flat blue lesion with radiating, cascading or linear veins extending from the centre. It ranges in size from 3 to 25 cm. **Cause** A type of telangiectasis (vascular dilatation) caused by increased intravenous pressure in superficial veins. **Localisation/distribution** Most commonly appear on the anterior chest and the lower legs near varicose veins.
Petechiae	 *Source:* Custom Medical Stock Photo, Inc.	Flat red or purple rounded freckles approximately 1 to 3 mm in diameter. Difficult to detect in dark skin. Do not blanch. **Cause** Minute haemorrhages resulting from fragile capillaries, petechiae are caused by septicaemias, liver disease or vitamin C or K deficiency. They may also be caused by anticoagulant therapy. **Localisation/distribution** Most commonly appear on the dependent surfaces of the body (e.g. back, buttocks). In the person with dark skin, look for them in the oral mucosa and conjunctivae.
Purpura	 *Source:* Custom Medical Stock Photo, Inc.	Flat, reddish blue, irregularly shaped extensive patches of varying size. **Cause** Bleeding disorders, scurvy and capillary fragility in the older adult (senile purpura). **Localisation/distribution** May appear anywhere on the body, but are most noticeable on the legs, arms and backs of hands.
Ecchymosis	 *Source:* Photo Researchers, Inc./Science Source.	A bruise. A flat, irregularly shaped lesion of varying size with no pulsation. Does not blanch with pressure. In light skin, it begins as a bluish purple mark that changes to greenish yellow. In brown skin, it varies from blue to deep purple. In dark skin, it appears as a darkened area. **Cause** Release of blood from superficial vessels into surrounding tissue due to trauma, haemophilia, liver disease or deficiency of vitamin C or K. **Localisation/distribution** Occurs anywhere on the body at the site of trauma or pressure.

Technique/normal findings	Abnormal findings
Palpate skin texture. *Skin should be smooth.*	■ Changes in the texture of the skin may indicate irritation or trauma. ■ The skin is soft and smooth in hyperthyroidism and coarse in hypothyroidism.
Palpate skin moisture. *Skin should be dry.*	■ Excessively dry skin often is present in older adults and people with hypothyroidism. ■ Oily skin is common in adolescents and young adults. Oily skin may be a normal finding or it may accompany a skin disorder such as acne vulgaris. ■ Excessive perspiration may be associated with shock, fever, increased activity or anxiety.
Palpate skin turgor. *Skin fold should return rapidly to normal position.*	■ Pinch the person's skin gently over the back of the hand or collarbone. Tenting, in which the skin remains pinched for a few moments before resuming its normal position, is common in older people who are thin (see Figure 14.4). ■ Skin turgor is decreased in dehydration. It is increased in oedema and scleroderma.

FIGURE 14.4 ■ Tenting in an older person

Assess for oedema. *No oedema should be present.*	■ Assess **oedema** (accumulation of fluid in the body's tissues) by depressing the person's skin (see Figure 14.5). Record findings as follows: 1+ Slight pitting, no obvious distortion 2+ Deeper pit, no obvious distortion 3+ Pitting is obvious; extremities are swollen 4+ Pitting remains with obvious distortion. ■ Oedema is common in cardiovascular disorders, renal failure and cirrhosis of the liver. It also may be a side effect of certain drugs.



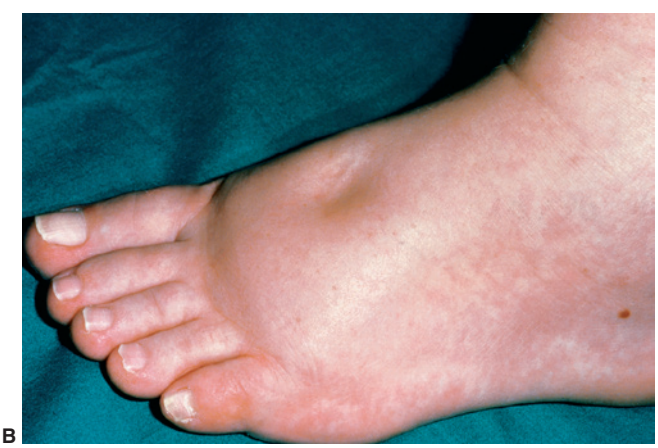


FIGURE 14.5 ■ *A*, Degrees of pitting in oedema. *B*, 4+ pitting

Source: B, © Dr P. Marazzi/SPL/Getty Images.

INTEGUMENTARY ASSESSMENTS (continued)

Technique/normal findings	Abnormal findings
Inspect distribution and quality of hair. *Hair should be evenly distributed for person's gender.*	■ A deviation in the normal hair distribution in the male or female genital area may indicate an endocrine disorder. **Hirsutism** (increased growth of coarse hair, usually on the face and trunk) is seen in Cushing's syndrome, acromegaly and ovarian dysfunction. **Alopecia** (hair loss) may be related to changes in hormones, chemical or drug treatment, or radiation. In adult males whose hair loss follows the normal male pattern, the cause is usually genetic.
Palpate hair texture. *Hair should be of even texture.*	■ Some systemic diseases change the texture of the hair. For instance, hypothyroidism causes the hair to coarsen, whereas hyperthyroidism causes the hair to become fine.
Inspect the scalp for lesions. *There should be no lesions on the scalp.*	■ Mild dandruff is normal, but excessive, greasy flakes indicate seborrhoea requiring treatment. ■ Hair loss, pustules and scales appear on the scalp in tinea capitis (scalp ringworm). ■ Red, swollen pustules appear around infected hair follicles and are called folliculitis. ■ Head lice may be seen as oval nits (eggs) adhering to the base of the hair shaft. Head lice are usually accompanied by itching.
Inspect nail curvature. *Nails should not be excessively curved.*	■ Clubbing (see Figure 14.6), in which the angle of the nail base is greater than 160 degrees, is seen in respiratory disorders, cardiovascular disorders, cirrhosis of the liver, colitis and thyroid disease. The nail becomes thick, hard, shiny and curved at the free end.

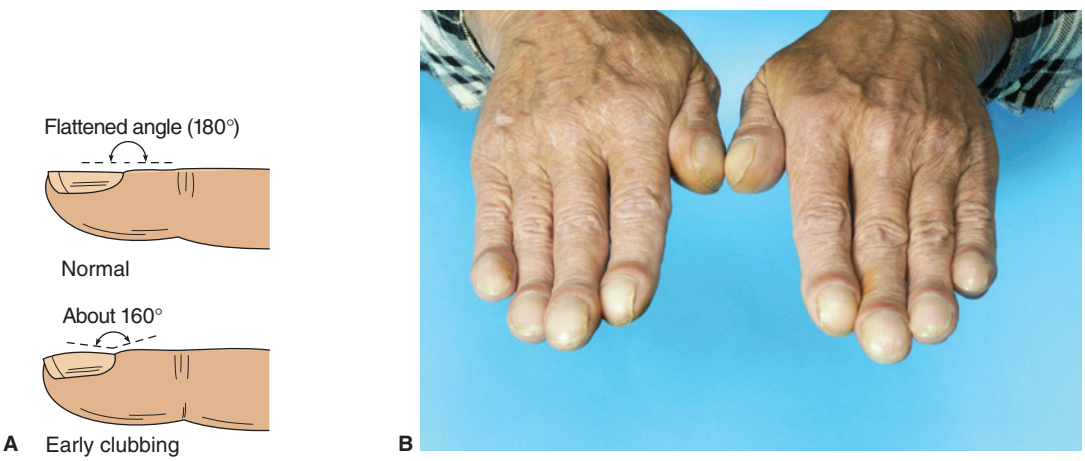


FIGURE 14.6 ■ *A*, Assessing clubbing of the nails. *B*, Hand with nail clubbing

Source: B, SPL/Science Source.

Inspect the surface of the nails. *Nail surfaces should be smooth and nail folds firm, without redness.*	■ The nail folds become inflamed and swollen and the nail may loosen in paronychia, an infection of the nail fold. ■ Inflammation and transverse rippling of the nail are associated with chronic paronychia and/or eczema. ■ The nail plate may separate from the nail bed in trauma, psoriasis and *Pseudomonas* and *Candida* infections. This separation is called oncolysis. ■ Nail grooves may be caused by inflammation, by lichen planus or by nail biting. ■ Nail pitting may be seen with psoriasis. ■ A transverse groove (Beau's line) may be seen in trachoma and/or acute diseases. ■ Thin spoon-shaped nails (see Figure 14.7) may be seen in anaemia.

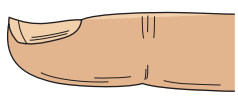


FIGURE 14.7 ■ Spoon-shaped nails

Technique/normal findings	Abnormal findings
Inspect nail colour. *Nail colour should be even.*	■ The sudden appearance of a pigmented band may indicate melanoma in Caucasians. ■ Yellowish nails are seen in psoriasis and fungal infections. ■ Dark nails occur with trauma, *Candida* infections and hyperbilirubinemia. ■ Blackish-green nails are apparent in injury and in *Pseudomonas* infection. ■ Red splinter longitudinal haemorrhages may be seen in injury and/or psoriasis.
Inspect nail thickness. *Nails should not be excessively thick.*	■ Trauma to the nails usually causes thickening. Other causes of thick nails include psoriasis, fungal infections and decreased peripheral vascular blood supply. ■ Thinning of the nails is seen in nutritional deficiencies.

CONCEPT CHECK

1 Which layer of the skin contains most of the hair follicles, sebaceous glands and sweat glands?
   1 epidermis
   2 dermis
   3 hypodermis
   4 stratum lucidium

2 What pigment is responsible for skin tanning?
   1 carotene
   2 red blood cells
   3 melanin
   4 sebum

3 Which of the four assessment techniques are used during assessment of the integumentary system? (Select all that apply.)
   1 inspection
   2 palpation
   3 percussion
   4 auscultation

4 Superficial skin damage that involves the epidermis only is referred to as:
   1 ulceration
   2 erosion
   3 atrophy
   4 lichenification

5 You are assessing a person who is complaining of severe itching. What would be an appropriate interview question?
   1 'Tell me how this itch feels.'
   2 'Why do you keep scratching it?'
   3 'Have you used a new soap?'
   4 'Describe your daily fluid intake.'

6 You are assessing the skin of an older person for dehydration. What finding would indicate this condition?
   1 decreased turgor
   2 increased moisture
   3 presence of lesions
   4 pallor or cyanosis

7 What part of the body would you commonly palpate to assess oedema in the older person?
   1 scalp
   2 fingers
   3 clavicle
   4 ankle/foot

8 You are assessing a person with chronic dermatitis and note that they have rough, thickened areas of skin. You document these areas as:
   1 ulcers
   2 papules
   3 atrophy
   4 lichenification

9 On assessment you observe clubbing of the fingernails. What disorders could this be related to?
   1 cirrhosis of the liver
   2 cardiovascular disorders
   3 emphysema
   4 excessive vitamin C intake

10 While assessing the hair, you note small white eggs on the hair shaft. What type of infestation are you assessing?
   1 bacterial
   2 viral
   3 head lice
   4 head lichens

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CHAPTER 15

NURSING CARE OF PEOPLE WITH INTEGUMENTARY DISORDERS

KERYLN CARVILLE, KERRY REID-SEARL, KATE CROWLEY

LEARNING OUTCOMES

- Describe the manifestations and nursing care, including the effects of treatment and medications, of common skin problems and lesions.
- Compare and contrast the aetiology, pathophysiology, interprofessional care and nursing care of people with infections and infestations of the skin.
- Compare and contrast the aetiology, pathophysiology, interprofessional care and nursing care of people with inflammation disorders of the skin.
- Differentiate between the various malignant skin conditions, including the nursing implications and treatment options for people with these conditions.
- Explain the risk factors for, pathophysiology of and nursing interventions to prevent and care for skin trauma.

CLINICAL COMPETENCIES

- Assess functional health status of people with integumentary disorders and monitor, document and report abnormal manifestations.
- Use evidence-based research to plan and implement nursing care for people with pressure injuries and skin tears.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with integumentary disorders.
- Administer topical, oral and injectable medications used to treat integumentary disorders knowledgeably and safely.
- Integrate interprofessional care into care of people with integumentary disorders.
- Provide teaching appropriate for prevention and self-care of disorders of the integumentary system.
- Revise plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to people with disorders of the integument.

KEY TERMS

The integumentary system is comprised of the skin and its accessory structures: hair, nails and glands (sebaceous, sudoriferous and ceruminous). The skin is the largest body organ, and provides protection by serving as a barrier between the internal and external environments. As described in Chapter 14, the functions of the skin are many, as the skin contains receptors for touch and sensation, helps regulate body temperature and maintains fluid and electrolyte balance. The skin also provides cues to racial and ethnic background and plays a major role in determining self-concept and cosmesis.

There are many disorders of the integument. Many of them are treated in an outpatient or community setting or by self-care. This chapter discusses disorders of the skin, hair and nails; Chapter 16 discusses the person with burns. Primary and secondary skin lesions are described and illustrated in Tables 14.6 and 14.7. The terms from these tables are used throughout this and the next chapter.

COMMON SKIN PROBLEMS AND LESIONS

The disorders discussed in this section are those experienced by a large number of people. Although they are considered minor health problems in terms of healthcare, they may cause major problems, such as impaired learning ability and earning potential for the person experiencing a high level of discomfort and loss of quality of life (Metz et al., 2013).

THE PERSON WITH PRURITUS

Pruritus is a subjective itching sensation that produces an urge to scratch. Pruritus may occur in a small, circumscribed area or it may involve a widespread area; it may or may not be associated with a rash. Pruritus is believed to result from either stimulation of itch receptors in the skin or as a response to the stimulation of skin receptors for pain and touch. The central nervous system (CNS) interprets these stimuli as an itch through central summation (Martini, Nath & Bartholomew, 2012).

Intrinsic and extrinsic factors can predispose a person to pruritus. Ingestion of foods or medications, contact with insects, animals, plants, fabrics or metals, allergic responses and even emotional distress are among the most common causes. Pruritus also may occur as a secondary manifestation of systemic disorders, such as certain types of cancer, diabetes mellitus, hepatic disease and renal failure. Although the exact physiology is unknown, it is known that heat and prostaglandins trigger pruritus and that histamine and morphine exacerbate the itch response.

The pathophysiological response of pruritus to stimulation or irritation follows a similar pathway, regardless of cause. The irritating agent stimulates receptors in the junction between the epidermis and dermis and may also trigger the release of histamine and other chemical mediators that either further stimulate or mediate the itch response. The response of the person experiencing the itch is to scratch or rub the affected area. This may irritate the skin and cause further inflammation, which in turn sets off a cycle of increasingly intense itching and scratching, called the *itch–scratch–itch cycle*.

Secondary effects of pruritus include skin excoriation, erythema, wheals, changes in pigmentation and infections. Persistent pruritus may interrupt sleep patterns, because the itching sensation is often more intense at night. Long-term pruritus may be debilitating and increases the risk of infection as excoriation occurs. In children, chronic pruritus has been shown to increase the risk of depression, anxiety and suicidal thoughts (Metz et al., 2013). Though not the most prevalent symptom in palliative care patients, pruritus remains the most puzzling. It can cause discomfort and has a major impact on the person's quality of life.

Treatment for this cohort remains challenging and requires an individualistic approach (Xander et al., 2013). Management of pruritus focuses on identifying and eliminating the cause and the topical application or the use of systemic medications to relieve the itch. Control of pruritus is crucial in breaking the itch/scratch cycle and preventing damage to the skin (excoriation and lichenification). A cool environment may cause vasoconstriction and decreasing itching. Antihistamines may relieve pruritus in some people. Tranquillisers may be prescribed to provide sedation, which may in turn relieve the emotional stress associated with pruritus. Systemic antibiotics are used to treat any infection resulting from the scratching and excoriation. Topical medications that contain corticosteroids are often used to relieve the pruritus and inflammation. Other topical preparations that contain menthol, camphor or phenol can be used to numb the itch receptors. Those with generalised itch may benefit from phototherapy (DermNet NZ, 2014). This is particularly useful for individuals with chronic renal failure on dialysis and eosinophilic infiltrations (DermNet NZ, 2014). Therapeutic baths or soaks with antipruritic agents such as cornflour, oatmeal, baking soda or coal tar concentrates may prove effective. Therapeutic baths are discussed in the following 'Medication administration' box. Table 15.1 lists examples of topical agents used to treat skin disorders. Creams containing a topical anaesthetic or antibiotic may also be prescribed.

THE PERSON WITH DRY SKIN (XEROSIS)

Dry skin, also called **xerosis**, is most often a problem in the older adult, as sebaceous and sweat gland activity reduces with ageing and this reduces the skin's lubrication and moisture retention. However, dry skin may occur at any age from overexposure to low humidity, sunlight, wind, excessive bathing and a decreased intake of liquids.

TABLE 15.1 Topical agents used to treat skin disorders

TYPE	USE	EXAMPLES
Creams and lotions	Moisturise the skin Cool the skin	Sorbolene Alpha Keri™ Ego QV™ skin lotion Calamine lotion (contains phenol which cools the skin, but limit use to a few days)
Ointments	Lubricate the skin Retard water loss	Urea Vaseline
Topical anaesthetics	Relieve itching	Xylocaine
Topical antibiotics (use prudently and only as prescribed)	Treat infection	Mupirocin (eye and nose mucous membrane) Silver sulfadiazine
Corticosteroids (use prudently and only as prescribed)	Suppress inflammation Relieve itching	Dexamethasone Hydrocortisone Betamethsone

MEDICATION ADMINISTRATION Therapeutic baths

AGENTS USED INTHERAPEUTIC BATHS

Saline or tap water
Antibacterial agents: potassium permanganate, Pinetarsol™ bath oil, hexachlorophene
Food substances: colloidal oatmeal, cornflour, sodium bicarbonate
Coal tar derivatives: Polytar™ and Alpha Keri™ tar bath preparations
Emollients: Alpha Keri™ and Ego QV™ bath oils

Coal tar preparations are not recommended for use under occlusive products, except under the direction and supervision of a physician; and coal tar preparations can cause skin irritation, rashes and skin photosensitivity (MIMS Online, 2013).

Therapeutic baths have a variety of uses in treating skin disorders. Depending on the agent used, therapeutic baths soothe the skin, lower the skin bacteria count, clean and hydrate the skin, loosen scales and relieve itching.

Nursing responsibilities

- Ensure that the bath water is at a comfortable tepid temperature that is neither too hot nor too cool, usually 45°C to 46°C.
- Fill the bath one-third to one-half full.
- Mix the agent well with the water.
- Assist the person into and out of the bath to prevent falls.
- Dry the person by blotting with the towel and avoid rubbing the skin.

Health education for the person and family

- Use a bath mat in the bath because the medications may cause the bath to become slippery.
- Keep the bathroom warm but adequately ventilated.
- If using a prescribed medication, follow directions carefully for the amount of medication to use in the bath.
- Fill the bath one-third to one-half full of water that is at a comfortable temperature.
- Stay in the bath for 20 to 30 minutes and immerse the areas to be treated.
- Do not get the medicated bathwater in your eyes.
- Dry by blotting (not rubbing) with the towel.
- If the medications cause staining, use old towels or linen.
- If the itching is not relieved or the skin becomes excessively dry, call your healthcare provider.

Two types of severe dry skin are xeroderma and ichthyosis. Xeroderma is a chronic skin condition characterised by dry, rough skin. Ichthyosis is an inherited dermatological condition in which the skin is dry, fissured and hyperkeratotic; the surface of the skin has the appearance of fish scales.

The primary manifestation of dry skin is pruritus. Other manifestations include visible flaking of surface skin and an observable pattern of fine lines over the area. If the skin has been excessively dry and pruritic for a long period, the person may have secondary skin lesions and lichenification (roughened thick epidermis with accentuated skin markings).

Nursing care focuses on teaching the person and family how to reduce the dry skin and relieve the pruritus, as outlined in Box 15.1.

THE PERSON WITH BENIGN SKIN LESIONS

The skin is subject to many different types and kinds of benign skin lesions, including cysts, hypertrophic scars, keloids, naevi, angiomas, skin tags and keratoses. Although these benign lesions are often considered more of a nuisance than an illness,

BOX 15.1 Teaching to reduce dry skin and relieve pruritus

- Wash clothing in a mild detergent and rinse twice; do not use fabric softeners.
- Avoid using perfumes and lotions containing alcohol.
- Apply skin moisturisers after a bath to help retain moisture.
- Soaps and hot water are drying. Clean the skin with tepid water and either a pH-neutral cleanser or mild soap. If soap is used, rinse it off thoroughly.
- It is not necessary to take a bath every day.
- If bath oils are used, add them to the bathwater at the end of the bath (the moist skin is more likely to retain the oil). Be careful not to slip in the bath.
- Pat the skin lightly and immediately apply a moisturising lotion or cream after bathing.
- Use a humidifier to humidify the air if the environment is very dry.
- Apply creams and lotions when the skin is slightly damp after bathing.
- Increase fluid intake.
- Keep nails trimmed short, wear loose clothing and keep the environment cool.
- A brief application of pressure or cold may relieve pruritus.
- Cotton gloves may be worn at night if scratching during sleep causes skin excoriation.
- Distraction or relaxation techniques may prove helpful.

they do require monitoring for malignant changes or an increase in size that interferes with the skin's appearance or function.

Most benign skin lesions do not require treatment, although excision or laser surgery may sometimes be desired or necessary for cosmetic or functional purposes. Cysts may enlarge, skin tags may become irritated and bleed, naevi may change in appearance or any of the lesions may cause discomfort with appearance.

Cysts

Cysts of the skin are benign closed sacs in or under the skin surface that are lined with epithelium and contain fluid or a semisolid material. Epidermal inclusion cysts and pilar cysts are the most common types.

Epidermal inclusion cysts may occur anywhere on the body, but are most often found on the head and trunk. Although they are painless, they may grow so large that they become irritated by contact with clothing (e.g. if located on the back of the neck) or cause obstruction (e.g. if located on the nose). The cysts contain a semisolid material composed mainly of keratin. Pilar cysts are painless and are found on the scalp and originate from sebaceous glands. Both types of cysts rarely require treatment unless they become large and bothersome.

Hypertrophic scar

Hypertrophic scars are characterised by an excess of collagen in the healed wound that results in excessive elevated scar; however, one which is contained within the perimeter of the original wound (Bullock & Hales, 2013). Hypertrophic scars can form following intentional or accidental trauma such as burns. Hypertrophic scarring is aberrant or abnormal wound healing (Carville, 2012). The mechanism and reasons for hypertrophic scarring are not fully understood, but such scarring commonly occurs following burn trauma. The use of custom-made pressure garments for the prevention and treatment of hypertrophic scarring is considered standard care. Although it is not definitively understood how pressure garments impact on scar formation, studies demonstrate that they do improve scar appearance and cause a scar to be softer and thinner (Monstrey et al., 2014). Pressure garments are often used in conjunction with silicone sheets which appear to aid in keeping the scar hydrated as they reduce moisture vapour transmission (Stavrou et al., 2010).

Keloids

Keloids are elevated, irregularly shaped, progressively enlarging scars that extend outside the perimeters of an original wound. Like hypertrophic scars, keloid formation is aberrant wound healing. They arise from excessive amounts of collagen in the stratum corneum during scar formation in connective tissue repair. These lesions are more common in young adults and appear within 1 year of the initial trauma.

This abnormal response most often occurs in people of African and Asian descent who sustain burns of the skin, but even seemingly minor trauma such as infection or an injection site can result in keloid formation (Bullock & Hales, 2013). There is a familial tendency to develop keloids. Other risk factors for keloid formation include excessive tension on a wound and poor alignment of skin edges following accidental or intentional skin trauma. Certain skin surfaces are also more likely to develop keloids: the chin, ears, shoulders, back and lower legs.

The excessive scar formation is associated with increased metabolic activity of fibroblasts and increased type III collagen. The principal cells of the keloids are myofibroblasts, which have characteristics of both fibroblasts and smooth muscle cells. The swollen appearance of the keloids is the result of an excess of extracellular material.

The keloids first appear as red, firm, rubbery plaques that persist for several months after the initial trauma (see Figure 15.1). Uncontrolled overgrowth over time causes the keloids to extend beyond the original scar. Eventually, the keloid becomes smooth and hyperpigmented. Intralesional steroid injections and excision may be tried to control keloid formation; however, they can recur following these treatments.

Naevi

Naevi, more commonly called *moles*, are flat or raised macules or papules with rounded, well-defined borders (see Figure 15.2). Naevi arise from melanocytes during early childhood, with the

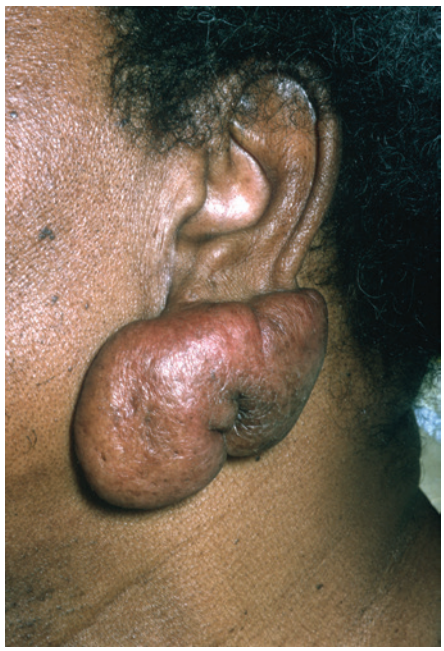


FIGURE 15.1 ■ Keloids form as a result of deposits of excessive amounts of collagen during scar formation

Source: © photographer/Alamy.

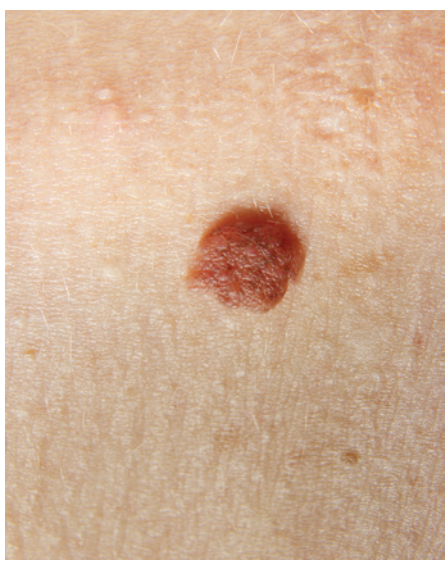


FIGURE 15.2 ■ Naevi (moles) arise from melanocytes and are common in all adults

Source: Nathalie Speliers Ufermann/Shutterstock.

cells initially accumulating at the junction of the dermis and epidermis. Over time, the cluster of cells moves into the dermis and the lesion becomes visible. Almost all adults have naevi.

Naevi range from flesh coloured to black and occasionally contain hair. They can occur on any skin surface of the body and may arise as single lesions or in groups. Some pigmented naevi can transform into malignant lesions. Australian malignant melanoma rates are on the rise. The whole body naevi count, when increased, is an independent risk factor for melanoma (Mar, Wolfe & Kelly, 2011). It is important to monitor naevi for changes in size, thickness, colour, bleeding or itching. If any of these changes occur, the person should seek immediate professional assessment.

Angiomas

Angiomas, also called *haemangiomas*, are benign vascular tumours. They appear in the adult in different forms:

- Naevus flammeus (port-wine stain) is a congenital vascular lesion that involves the capillaries. The lesions tend to occur on the upper body or face as macular patches that range from light red to dark purple. These lesions are present at birth and grow proportionately with the child into adulthood.
- Cherry angiomas are small, rounded papules that may occur at any age, but they most commonly arise in the forties and gradually increase in number. The lesions range in colour from bright red to purple. These lesions are often found on the trunk. They are also called senile angiomas or de Morgan spots, named after Campbell de Morgan, a 19th century British surgeon who first described them (Watkins, 2012).
- Spider angiomas are dilated superficial arteries. They are common in pregnant women and in people with hepatic disease. Spider angiomas occur most often on the face, neck and upper chest. The lesions are usually small, bright red papules with radiating lines, and they blanch with pressure.
- Telangiectases are single dilated capillaries or terminal arteries that appear often on the cheeks and nose. These lesions are most common in older adults and result from photoaged skin. The lesions look like broken veins.

Skin tags

Skin tags are soft papules on a pedicle. They can be as small as a pinhead or as large as a pea and are most often found on the front or side of the neck and in the axillae, as well as in areas where clothing (such as underwear) rubs the skin. These lesions have normal skin colour and texture.

Keratoses

A **keratosis** is any skin condition in which there is a benign overgrowth and thickening of the cornified epithelium. These lesions most often appear in adults after age 50. *Seborrhoeic keratoses* appear as superficial flat, smooth or warty-surfaced growths, 5 to 20 mm in diameter, most often on the face and trunk. The lesions may be tan, waxy yellow, dark brown or flesh coloured and they often appear greasy. They are most often seen in the older adult and do not appear to be related to damage from sun exposure.

THE PERSON WITH PSORIASIS

Psoriasis is a chronic immune skin disorder characterised by raised, reddened, round circumscribed plaques covered by silvery white scales (see Figure 15.3). The size of these lesions varies. The lesions may appear anywhere on the body, but they are most commonly found on the scalp, extensor surfaces of

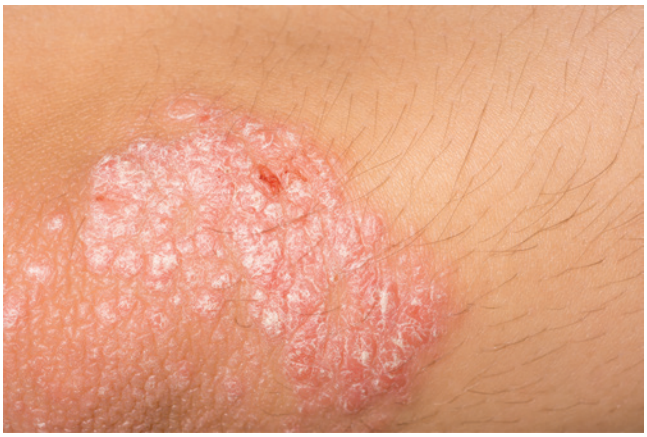


FIGURE 15.3 ■ The characteristic lesions of psoriasis are raised, red, round plaques covered with thick, silvery white scales

Source: © olavs/Shutterstock.com.

the arms and legs, elbows, knees, sacrum and around the nails. As with any chronic illness, the skin manifestations may occur and disappear throughout life, with no discernible pattern to the recurrence.

The incidence of psoriasis is lower in warm, sunny climates. Onset usually occurs in the person's twenties, but it may occur at any age. Psoriasis occurs more often in Caucasians; men and women are affected equally. Sunlight, stress, seasonal changes, hormone fluctuations, steroid withdrawal and certain drugs (such as alcohol, corticosteroids, lithium and chloroquine) appear to exacerbate the disorder. For more than 40 years the evidence has been identifying that psoriasis is linked to genetic susceptibility (Witte & Sabat, 2014). About one-third of people have a family history of psoriasis. Trauma to the skin from such events as surgery, sunburn or excoriation is also a common precipitating factor; lesions that result from trauma are called Köbner's reaction (Bullock & Hales, 2013).

Pathophysiology

Normally, the keratinocyte (an epidermal cell making up 95% of the epidermis) migrates from the basal cell to the stratum corneum (the outer skin layer) in about 14 days and is sloughed off 14 days later. Psoriatic skin cells, by contrast, have a shorter cycle of growth, completing the journey to the stratum corneum in only 4 to 7 days, a condition called *hyperkeratosis*. These immature cells produce an abnormal keratin that forms thick, flaky scales at the surface of the skin. The more rapid cell metabolism stimulates increased vascularity, which contributes to the erythema of the lesions. As part of the abnormal process, certain immune cells become overactive and release proteins called cytokines. One of the cytokines is tumour necrosis factor (TNF); in psoriasis, TNF causes inflammation, further contributing to plaque formation.

Psoriasis vulgaris is the most common form of psoriasis. The lesions can be found anywhere on the skin but most commonly involve the skin over the elbows, knees and scalp. Initially, the lesions are papules that form into well-defined erythematous plaques with thick, silvery white scales. The plaques in people with darker skin may appear purple.

Permanent remission of psoriasis is rare. The prognosis depends on the type, extent and severity of the initial attack. The age of onset is also a factor; early-onset disease is usually more severe.

Manifestations

The characteristic lesions in psoriasis are well-demarcated regions of erythematous plaques that shed thick, silvery white flakes. Pruritus is common over the psoriatic lesions. If the lesions are located in an intertriginous zone, such as between the toes, under the breasts or in the perianal region, the psoriatic scales may soften, allowing painful fissures to form. When psoriasis affects the nails, pitting and a yellow or brown discolouration result. The nail may separate from the nail bed, thicken and crumble. The involved nails, which are more often fingernails than toenails, are at high risk of infection. The person may also exhibit manifestations of psoriatic arthritis, seen most often in the distal interphalangeal joints, especially if the fingernails are involved.

INTERPROFESSIONAL CARE

Treatment is based on the type of psoriasis, the extent and location of the lesions, the age of the person and the degree of disfigurement or disability.

Diagnosis

Skin biopsy may be done if the person presents with atypical manifestations or to differentiate psoriasis from other inflammatory or infectious skin disorders. In addition, an ultrasound may reveal typical psoriatic changes in the stratum corneum and inflammation of the dermis.

Medications

A variety of medications and treatments may be prescribed, including topical medications and photochemotherapy. Although there is no cure, treatment decreases the severity and pain of the lesions.

Topical medications are administered to decrease inflammation, prolong the maturity time of keratinocytes and increase remission time. Corticosteroids, tar preparations, anthralin and the retinoids are typically used. Box 15.2 outlines general guidelines for teaching the person to apply topical medications.

Topical corticosteroids decrease inflammation, suppress mitotic activity of psoriatic cells and delay the movement of keratinocytes to the surface of the skin. The most effective topical corticosteroids are potent preparations that are well absorbed through the skin and are used under an occlusive dressing. Corticosteroids may also be taken systemically or injected directly into the lesions. However, corticosteroids rarely cause a lasting remission and may cause the psoriasis to become unstable (Horn et al., 2010). They are therefore used for repeated short periods of treatment and combined with other measures, such as tar preparations, occlusions or a topical retinoid.

BOX 15.2 General guidelines for applying topical medications

Each time a medication is applied, the skin surface must be clean and dry. Remove any medication from the previous application. Remove creams by washing the skin with tap water and a pH-neutral skin cleanser or mild soap.

- *To apply gels, creams and pastes:* squeeze about 1 to 2.5 cm of the gel or cream into the gloved palm of the hand. Rub the hands together until they are covered. Apply gels and creams to the affected areas with long gentle strokes until the skin is thinly covered. Differences from these general guidelines follow:
  a. Corticosteroids are usually applied two to three times a day in small amounts and rubbed directly on to the lesions. Apply the medication after a bath; it can be covered with an occlusive dressing if prescribed.
  b. Apply medications containing tar in the direction of hair growth. Do not apply these medications to the face, to the genitals or in skin folds. If the tar is water-based or oil-based, it will stain clothing.
  c. Wear gloves when applying dithranol stains.
- *To apply lotions:* shake the bottle of lotion well. Pour a small amount into the palm of the gloved hand and pat the medication on to the skin. If the lotion is thin, apply it with a gauze pad.
- *To apply sprays:* hold the container about 15 cm from the skin and apply the medication in a short spray.
- *To apply medicated shampoo:* apply the shampoo, massage into the hair and over the scalp carefully and allow it to remain for the prescribed time. Rinse.
- *To apply pastes:* use enough paste on an applicator (such as a wooden tongue depressor) to cover the lesion thinly.

Tar preparations (such as Psor-Asist™) suppress mitotic activity and are also anti-inflammatory. Tar preparations are made from distillation of coal and wood and they can stain clothing and have a pungent odour. The exact mechanism of action of tar preparations is unknown, but they are effective in removing scales and increasing remission time; however, they can make a person more sun sensitive.

Topical dithranol, which is extracted from tree bark, inhibits the mitotic activity of epidermal cells and is effective in some cases of chronic, localised psoriasis that do not respond to other topical agents. The medication is applied to the plaque patches at bedtime and left in place for 8 to 12 hours. The person should be tested for sensitivity to the drug before use and it should not be applied to inflamed or open areas of skin.

Calcipotrial (Daivovex™, a vitamin D analogue) has been effective and safe in both the short-term and long-term treatment of psoriasis. It inhibits cell proliferation in the epidermis and facilitates cell differentiation. Although a derivative of topical vitamin D, calcipotrial does not seem to affect bone or calcium metabolism; however, periodic monitoring is prudent if used in children or large doses (Harkin, 2014).

Ultraviolet light therapy

Psoriasis that is widespread (i.e. involves more than 30% of the body surface) is difficult to treat with topical medications. Treatments for generalised psoriasis include ultraviolet light therapy and photochemotherapy. Natural sunlight contains ultraviolet-B (UVB) which penetrates the skin and slows the growth of affected skin cells (National Psoriasis Foundation, 2015). In Australia, narrowband UVB, broadband UVB and a combination of oral psoralens (natural substances derived from plants such as celery) and ultraviolet-A light (PUVA) are used to treat psoriasis. The light is delivered by specially designed fluorescent tubes either within a cabinet (full body application) or in panels (for application to individual body parts) (Better Health Channel, 2015).

The light therapy is administered in gradually increasing exposure times, until the person experiences a mild erythema, like a mild sunburn. Treatments are given three times a week as an outpatient and are measured in seconds of exposure. The eyes are shielded during the treatment. The erythema response occurs in about 8 hours. Careful assessment is necessary to prevent more severe burning, which could exacerbate the psoriasis. In the person with extensive psoriasis, UVB treatments may be combined with tar preparations, which increase the photosensitivity of the skin.

PHOTOCHEMOTHERAPY In photochemotherapy, a light-activated form of the drug methoxsalen is used. This drug is an antimetabolite that inhibits DNA synthesis and thereby prevents cell mitosis, decreasing hyperkeratosis. Exposure to ultraviolet-A (UVA) rays activates methoxsalen; it is administered orally and the person is exposed to UVA 2 hours later. The eyes are covered by dark glasses during the treatment. Treatments are administered two to three times a week; usually, 10 to 20 total treatments are given over 1 to 2 months. Treatment causes tanning and direct sunlight must be avoided for 8 to 12 hours thereafter. If the person exhibits erythema, the treatments are stopped until the redness and swelling resolve.

Photochemotherapy has had a high success rate in achieving remission of psoriasis, but it can accelerate ageing of exposed skin, induce cataract development, alter immune function and increase the risk of melanoma.

Nursing care

The person with psoriasis requires nursing care to meet physical and psychological responses to the illness. The nurse provides teaching for self-care and emotional support through non-judgmental acceptance.

Nursing diagnoses and planning

The nursing care discussed in this section focuses on the diagnoses of:

- *Impaired skin integrity* related to psoriasis as evidenced by open lesions on lower legs and lower arms.

- *Disturbed body image* related to psoriasis evidenced by reluctance to wear clothing that may expose lesions.

Impaired skin integrity related to psoriasis

Psoriatic lesions range from several scales to large, open areas. Typical psoriatic skin lesions increase the risk of infection, which can further compromise healing. In addition, certain treatments (e.g. the use of UVA or retinoids) may cause erythema or peeling of the skin, further altering skin integrity.

- Teach methods to reduce injury to the skin when taking therapeutic baths or treatments:
  - Use warm, not hot, water.
  - Gently rub lesions with a soft washcloth, using a circular motion.
  - Dry the skin with a soft towel, using a blotting or patting motion.
  - Keep the skin lubricated at all times.
- Explain application of topical medications:
  - Apply the medication as prescribed in a thin layer, using gloved hands, wooden tongue depressors or a gauze pad.
  - Avoid getting medications in the eyes, on mucous membranes or in skin folds.
  - Apply a covering such as an occlusive dressing over the medicated areas if prescribed. Topical corticosteroids are often covered with occlusive dressings or wraps to increase absorption and thus facilitate treatment. However, constant occlusion may increase the effects of the medications to undesired levels and also increases the risk of infections.
- Teach manifestations of infection and how to contact the healthcare provider if these occur: elevated temperature, increased swelling, redness, pain, increase in drainage and any change in the colour of the drainage.
- Teach manifestations of the complications of treatment: excoriation, increased erythema, increased peeling and blister formation. The topical medications or treatments may damage cells through chemical burns or excessive exposure to ultraviolet light.

Disturbed body image related to psoriasis

The chronic skin lesions of psoriasis often cause people to isolate themselves from social contacts, withdraw from normal roles and responsibilities, and feel helpless or powerless.

- Establish a trusting relationship by expressing acceptance of the person, both verbally and non-verbally. For example, touch the person during social communications, demonstrating that the lesions are not contagious or offensive.
- Encourage the person to verbalise feelings about self-perception in view of the chronic nature of psoriasis and to ask questions about the disease and treatment.
- Promote social interaction through family involvement in care and by referral to support groups of people with psoriasis or other chronic skin conditions.

Community-based care

Person and family teaching focuses on treatments and skin care needs. The following topics should be addressed:

- The chronic nature of the disease, factors that may precipitate an exacerbation and methods to reduce stress.
- Interventions for pruritus and dry skin and specific care for psoriasis:
  - Expose the skin to sunlight, but avoid sunburn.
  - Avoid trauma to the skin (e.g. do not scrub off scales and use only an electric razor).
  - Avoid exposure to contagious illnesses such as influenza and colds.
  - Discuss current medications with the healthcare provider. Certain drugs (such as indomethacin (Indocin™), lithium and beta-adrenergic blocking agents) are known to precipitate exacerbations of psoriasis.
  - Suggest contact with resources such as Psoriasis Australia www.psoriasisaustralia.org.au.

INFECTIONS AND INFESTATIONS OF THE SKIN

The skin's resistance to infections and infestations is provided by protective mechanisms, including skin flora, sebum and the immune response. Although the skin is normally resistant to infections and infestations, these disorders may occur as a result of a break in the skin surface, a virulent agent, and/or decreased resistance due to a compromised immune system. This section discusses skin disorders resulting from bacterial infections, fungal infections, parasitic infestations and viral infections.

THE PERSON WITH A BACTERIAL INFECTION OF THE SKIN

A number of bacteria normally inhabit the skin and do not cause an infection. However, when a break in the skin allows invasion by pathogenic bacteria, an infection may occur. The most common bacterial infections are caused by gram-positive organisms commonly found on the skin, such as *Staphylococcus aureus*.

Bacterial infections of the skin may be primary or secondary. Primary infections are caused by a single pathogen and arise from normal skin; secondary infections develop in injured or diseased skin.

Most bacterial infections are treated by a primary care provider within the home environment. If the infection becomes more serious, however, inpatient care may be required. In addition, nosocomial infections of wounds or open lesions in hospitalised people are often the result of bacterial infections, especially by methicillin-resistant *Staphylococcus aureus* (MRSA).

Pathophysiology

Bacterial infections of the intact skin arise from the hair follicle, where bacteria can accumulate and grow and cause a localised infection. However, the bacteria also can invade deeper tissues and cause a systemic infection, a potentially life-threatening disorder. Various types of bacterial infections involve the skin, including folliculitis, furuncles, carbuncles, cellulitis and erysipelas.

Folliculitis

Folliculitis is a bacterial infection of the hair follicle, most commonly caused by *Staphylococcus aureus*. The infection begins at the follicle opening and extends down into the follicle. The bacteria release enzymes and chemical agents that cause an inflammation. The lesions appear as pustules surrounded by an area of erythema on the surface of the skin (see Figure 15.4). The lesions are accompanied by discomfort ranging from slight burning to intense itching. A major complication is abscess formation. Folliculitis is found most often on the scalp and extremities. It is also often seen on the face of bearded men (called sycosis barbae), on the legs of women who shave and on the eyelids (called a stye).

Although folliculitis may appear without any apparent cause, contributing factors include poor hygiene, poor nutrition, prolonged skin moisture, tight heavy fabrics on the upper legs, shaving and trauma to the skin.

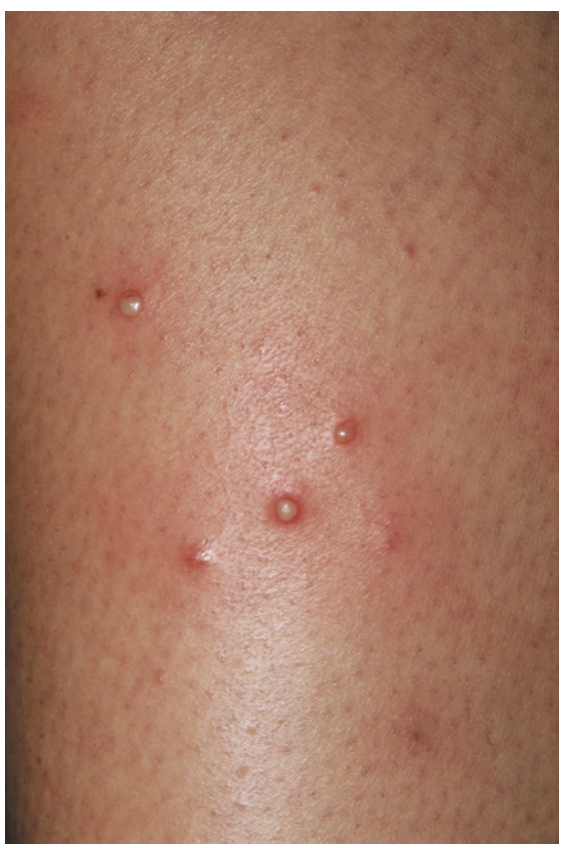


FIGURE 15.4 ■ The lesions of folliculitis are pustules surrounded by areas of erythema

Source: © Dr P Marazzi/Science Source.

CONSIDERATION FOR PRACTICE

A specific type of folliculitis, called 'hot tub folliculitis', is caused by *Pseudomonas aeruginosa* and is characterised by follicular or pustular lesions that occur 1 to 4 days after being in a hot tub or public swimming pool.

Furuncles

Furuncles, often called *boils*, are also inflammations of the hair follicle. They often begin as folliculitis, but the infection spreads down the hair shaft, through the wall of the follicle and into the dermis. The causative organism is commonly *Staphylococcus aureus*. A furuncle initially presents as a deep, firm, red, painful nodule from 1 to 5 cm in diameter (see Figure 15.5), but can result in a painful cystic nodule. The cysts may drain substantial amounts of purulent drainage.

Contributing factors include poor hygiene, heat and humidity, trauma to the skin, areas of excessive moisture (including perspiration) and systemic diseases such as diabetes mellitus and haematological malignancies.

Carbuncles

A **carbuncle** is a group of infected hair follicles. The lesion begins as a firm mass located in the subcutaneous tissue and the lower dermis. This mass becomes swollen and painful and has multiple openings to the skin surface. Carbuncles are most frequently found on the back of the neck, the upper back and the lateral thighs. In addition to the local manifestations, the person may experience chills, fever and malaise. The contributing factors for carbuncles are the same as for furuncles. Both infections are more common in hot, humid climates.

Cellulitis

Cellulitis is a localised infection of the dermis and subcutaneous tissue. Cellulitis can occur in intact skin or be associated with a wound or furuncles or carbuncles. The infection spreads as a result of a substance produced by the causative organism, called spreading factor (hyaluronidase). This factor breaks down the

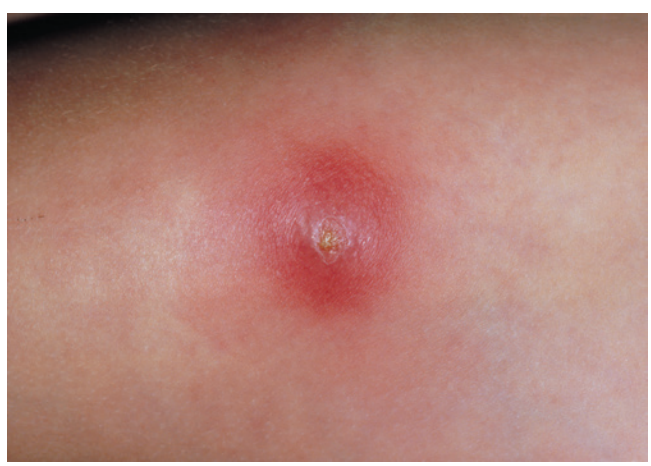


FIGURE 15.5 ■ A furuncle (boil) is a deep, firm, red, painful nodule

Source: © Dr P Marazzi/Science Source.

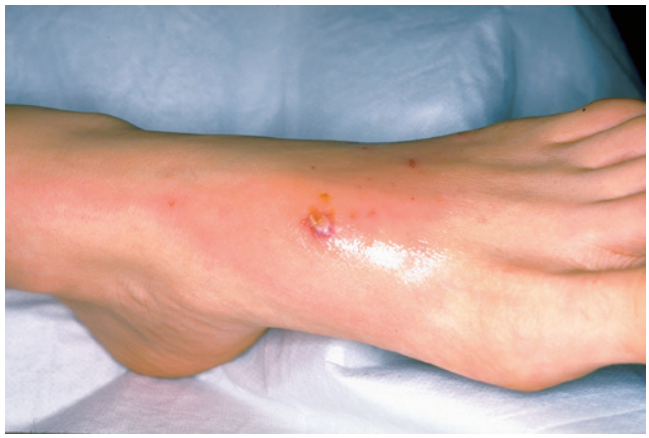


FIGURE 15.6 ■ Cellulitis is a bacterial infection localised in the dermis and subcutaneous tissue. The involved area is red, swollen and painful

Source: © Charles Stewart MD FACEP, FAAEM.

fibrin network and other barriers that normally localise the infection. The area of cellulitis is red, swollen and painful (see Figure 15.6). In some cases, vesicles may form over the area of cellulitis. The person may also experience systemic signs and symptoms such as fever, chills, malaise, headache and swollen lymph glands.

Erysipelas

Erysipelas is an infection of the skin most often caused by group A streptococci. Chills, fever and malaise are prodromal symptoms, occurring from 4 hours to 20 days before the skin lesion appears. The initial infection appears as firm red spots that enlarge and join to form a circumscribed, bright red, raised, hot lesion. Vesicles may form over the surface of the erysipelas lesion. The area usually is painful, itches and burns. Erysipelas most commonly appears on the face, ears and lower legs.

INTERPROFESSIONAL CARE

The diagnosis of a bacterial infection of the skin is made by assessing the appearance of the lesion and by identifying the causative organism. Antibiotics specific to the organism are used in treatment.

Diagnosis

Wound aspirate or swab is collected and sent to pathology for microbiological culture and sensitivity to identify the causative organism and determine the most effective antibiotic. People who experience repeated bacterial skin infections or who provide care for others who exhibit infections may have a culture taken from the external nares to determine whether they are carriers of bacteria (e.g. MRSA) and are reinfecting themselves or others.

Medications

The primary treatment for bacterial infections of the skin is an antibiotic specific to the organism. The antibiotic is usually taken orally, but for extensive or severe infections antibiotics may be administered intravenously. Multiple furuncles and carbuncles may be treated with cloxacillin (a penicillinase-resistant penicillin); the cephalosporins also are often effective. Topical antibiotics are not commonly prescribed as overuse can increase the risk of antibiotic resistance. However, prudent use of antiseptic-impregnated dressings can restore bacterial balance in the tissue without undue tissue toxicity (see Box 15.12).

Nursing care

Nursing care focuses on preventing the spread of infection and restoring normal skin integrity. Most people provide self-care at home, but the incidence of secondary bacterial infections in the inpatient population is great enough to warrant their inclusion in planning and implementing care.

Nursing diagnoses and interventions

Potential risk of infection related to ineffective infection control practices by healthcare personnel

- Practise good hand hygiene and teach its importance to all people.
- Teach the person and their family how to identify the signs and symptoms of localised and spreading infection. Local signs and symptoms include pain, swelling, erythema, heat and purulent or increased exudate. Signs of spreading infection may be manifested systemically by fever, tachycardia, chills and malaise.
- Prudent preventive use of modern antiseptic dressings such as cadexomer iodine (Iodosorb™), wound honey preparations, silver or polyhexamethylene-biguanide-impregnated dressings may be warranted in high-risk people or wounds.

Community-based care

Person and family teaching focuses on facilitating tissue healing and eliminating the infection. Address the following topics:

- the importance of maintaining good nutrition
- the importance of maintaining cleanliness through careful hand hygiene, and proper handling and disposal of dressings or contaminated materials
- preventing the spread of infection in the home by not sharing bed linen and towels, and washing clothing and linen in hot water
- the importance of not squeezing or trying to open a bacterial lesion
- avoiding plucking of nasal hair or picking nose
- the importance of taking the full course of prescribed antibiotics on a regular schedule until the prescribed supply is finished
- showering daily and using an antibacterial soap or lotion if warranted when bacterial skin infections exist.

THE PERSON WITH A FUNGAL INFECTION

Fungi are free-living plant-like organisms that live in the soil, on animals and on humans. The fungi that cause superficial skin infections are called dermatophytes. In humans, the dermatophytes live on keratin in the stratum corneum, hair and nails. Fungal disorders are also called *mycoses*.

Pathophysiology

Fungal infections include dermatophytoses (tinea or ringworm) and candidiasis (yeast) infections.

Dermatophytoses (ringworm or tinea)

Superficial fungal infections of the skin are called **dermatophytoses** or, more commonly, *ringworm or tinea*. Fungal infections occur when a susceptible host comes in contact with the organism. The organism may be transmitted by direct contact with animals or other infected people or by inanimate objects such as combs, pillowcases, towels and hats. The most important factor in the development of the infection is moisture; the onset and spread of the fungal infection is greatest in areas where moisture content is high, such as within skin folds, between the toes and in the mouth. Other factors that increase the risk of a fungal infection include the use of broad-spectrum antibiotics that kill off normal skin or mucosal flora and allow the fungi to grow, and conditions such as diabetes mellitus, immunodeficiencies, nutritional deficiencies, pregnancy, increasing age and iron deficiency. Fungal infections of the skin are more common in warm, humid climates. The dermatophyte infections are named by the body part affected, as follows:

- *Tinea pedis* is a fungal infection of the soles of the feet, the space between the toes, and/or the toenails (see Figure 15.7). More often called *athlete's foot*, this is the most common tinea infection. The lesions vary from mild scaliness to painful fissures with drainage and they are usually accompanied by pruritus and a foul odour. The infection is often chronic, absent in winter but reappearing in hot weather when perspiring feet are encased in shoes.
- *Tinea capitis* is a fungal infection of the scalp. The primary lesions are grey, round, bald spots, often accompanied by erythema and crusting. The hair loss is usually temporary. Tinea capitis is seen more often in children than in adults.
- *Tinea corporis* is a fungal infection of the body. It can be caused by several different fungi and the lesions vary according to the causative organism. The most common lesions, often called *ringworm,* are large circular patches with raised red borders of vesicles, papules or pustules. Pruritus and erythema are also present.
- *Tinea versicolour* is a fungal infection of the upper chest, back and sometimes the arms. The lesions are yellow, pink or brown sheets of scaling skin. The patches do not have pigment and do not tan when exposed to ultraviolet light.
- *Tinea cruris* is a fungal infection of the groin that may extend to the inner thighs and buttocks. Often called 'jock itch', it is often associated with tinea pedis and is more common in people who are physically active, are obese, and/or wear tight underclothing.

Candidiasis

Candidiasis infections are caused by *Candida albicans*, a yeast-like fungus. This fungus is normally found on mucous membranes, on the skin, in the vagina and in the gastrointestinal tract. The fungus becomes a pathogen when the following factors encourage its growth:

- a local environment of moisture, warmth or altered skin integrity
- the administration of systemic antibiotics

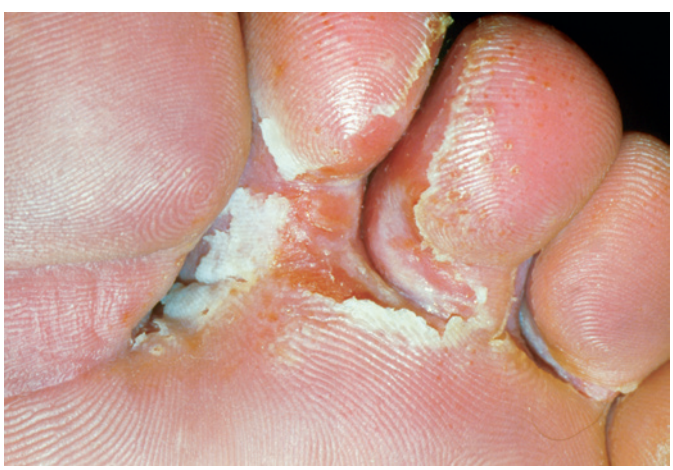


FIGURE 15.7 ■ *Tinea pedis* (athlete's foot) is a fungal infection that often occurs between the toes

Source: © SPL/Science Source.

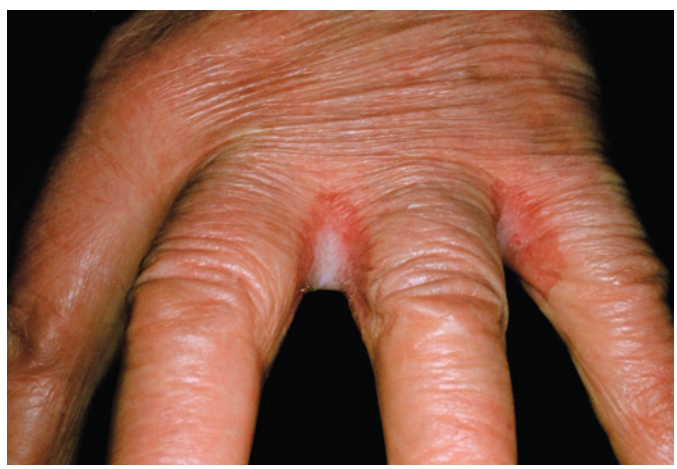


FIGURE 15.8 ■ *Candida albicans*, a fungus, causes a skin infection characterised by erythema, pustules and a typical white substance covering the area

Source: © CNRI/Science Photo Library.

TABLE 15.2 Characteristics of candidiasis infections by location

LOCATION	CHARACTERISTICS
Skin folds (under breasts, in groin, axillae, anus, umbilicus, abdominal pannus and between toes or fingers)	Erythematous lesions can be either dry or moist. The lesions have clear borders and satellite lesions are present.
Nails	Nail bed is red, swollen and painful.
Mouth (thrush)	Mucous membranes are red and may be swollen; surface is covered with white, creamy material. Eroded areas may be present over the tongue and the oral cavity.
Penis (balanitis) (glans and shaft)	The penis is covered with small, red, clearly demarcated lesions that are painful and itch. The lesions may be covered with a white plaque.
Vagina	Red mucous membranes contain brighter red, demarcated, oozing lesions. The cervix may be covered with white plaque. A white, cheesy, foul-smelling vaginal discharge is present, accompanied by itching and burning. The vaginal and labial membranes may be swollen; the infection may extend to the anus and groin.

- pregnancy
- the use of birth control pills
- poor nutrition
- the presence of diabetes mellitus, Cushing's disease or other chronic debilitating illnesses
- immunosuppression
- some malignancies of the blood.

Candidiasis affects the outer layers of the skin and mucous membranes of the mouth, vagina, uncircumcised penis, nails and deep skin folds (under large breasts or an abdominal pannus). The first sign of infection is a pustule that extends under the stratum corneum. The pustule has an inflamed base and often burns and itches. As the infection spreads, the accumulation of inflammatory cells and shedding of surface cells produce a white to yellow curd-like substance that covers the infected area (see Figure 15.8). Satellite lesions (maculopapular areas found outside the clearly demarcated border of the original infection) are characteristic of candidiasis. The appearance of the infection differs by location, as summarised in Table 15.2.

INTERPROFESSIONAL CARE

Fungal infections are primarily diagnosed in outpatient settings and treated at home, but may also occur in hospitalised people. The treatment is the same, regardless of the setting.

Diagnosis

Diagnostic tests are conducted to determine the causative fungi and may include cultures of skin scrapings and examination of the skin with ultraviolet light (Wood's lamp). See Chapter 14 for further information.

Medications

Fungal infections of the skin are treated by topical or systemic antifungal medications. Nursing implications for the antifungal medications are described in the 'Medication administration' box below.

- Candidiasis infections are treated, depending on the location, with oral medication or with powder or vaginal suppositories.

MEDICATION ADMINISTRATION Antifungal agents

Examples:

Nystatin (Mycostatin™, Nilstat™)
Itraconazole (Sporanox™)
Miconazole (Daktarin™)
Ketoconazole (Nizoral™)
Fluconazole (Diflucan™)
Amphotericin B (Fungilin™)
Griseofulvin (Fulcin™)
Terbinafine hydrocholoride (Lamisil™)

Antifungal medications are prepared in a variety of forms, depending on the specific drug: powders, creams, shampoos, suspensions, troches, vaginal suppositories and oral tablets. Some drugs interfere with the permeability of the fungal cell membrane; others interfere with DNA synthesis. Most of these medications are fungistatic, but in large doses they may be fungicidal.

Nursing responsibilities

- When taking the health history, ask about known hypersensitivity reactions to these agents; document carefully.
- Assess for side effects: skin rash, local irritation, gastrointestinal symptoms (if given orally) and cognitive status.
- Administer ketoconazole with food to minimise gastrointestinal irritation.
- Shake suspensions well before administration and ask the person to swish them around the mouth before swallowing.
- If advised by the manufacturer, advise the person to allow oral tablets to dissolve in the mouth.

Health education for the person and family

- Therapy usually continues over a long period of time, but regular use of medications for the recommended period

MEDICATION ADMINISTRATION Antifungal agents (continued)

is necessary. Do not miss doses and do complete the full treatment.
- *For griseofulvin:* take with meals or foods high in fat (such as ice-cream) to avoid stomach upset and help with absorption. Avoid alcohol (which may cause rapid pulse and flushing) and exposure to sunlight (this drug causes increased sensitivity).
- *For nystatin:* dissolve lozenges completely in the mouth. Hold suspensions in the mouth and swish throughout the mouth as long as possible before swallowing. Insert intravaginal medication high in the vagina. Continue with intravaginal applications throughout the menses.
- *For antifungal shampoo:* use two times a week for 4 weeks, allowing at least 3 days between each shampoo. Wet hair, apply shampoo to produce lather, leave in place for 1 minute, then rinse. Apply shampoo a second time, lather, leave in place for 3 minutes, then rinse thoroughly.
- *For topical application:* rub well into the affected areas, but do not get the medication in your eyes.
- *For vaginal candidiasis infections:* treatment with antifungal creams and pessaries can weaken condoms, so apply after sexual intercourse. Also, use plenty of lubricant during sexual intercourse as thrush can cause discomfort.
- A sexual partner will need to be treated at the same time so that you do not pass the infection back and forth to each other.

Nursing care

Many people treat themselves with over-the-counter antifungal medications. It is recommended, however, that the person be professionally diagnosed, especially if the infection is ongoing or repetitive. The interventions discussed for nursing care of the person with a bacterial infection are also appropriate for the person with a fungal infection. Teaching topics specific to fungal infections are as follows:
- Fungal diseases are contagious. Do not share linen or personal items with others.
- Use a clean towel and washcloth each day.
- Carefully dry all skin folds, including those under the breasts, arms, abdominal pannus and between the toes.
- Wear clean cotton underclothing each day.
- Fungi grow in moist environments, such as on sweaty feet. To prevent further infections:
  - Do not wear the same pair of shoes every day.
  - Wear socks that permit moisture to wick away from the skin surface.
  - Do not wear rubber- or plastic-soled shoes.
- For vaginal *Candida albicans* infection:
  - Avoid tight clothing, such as jeans and pantyhose.
  - Wear cotton or cotton-crotch underwear.
  - Shower more frequently and dry the genital area well.
  - Treat the sexual partner at the same time to avoid passing the infection back and forth to each other.

CONSIDERATION FOR PRACTICE

Recommend the person with repeated skin infections have a blood glucose test, because this may indicate diabetes mellitus.

THE PERSON WITH A VIRAL INFECTION

Viruses are pathogens that consist of an RNA or DNA core surrounded by a protein coat. They depend on live cells for reproduction and so are classified as intracellular pathogens. The viruses that cause skin lesions invade the keratinocyte, reproduce, and either increase cellular growth or cause cellular death.

An increase in the incidence of viral skin disorders has been attributed to a variety of causes. Some commonly used drugs, such as birth control medications and corticosteroids, are known to have immunosuppressive properties that allow the viruses to multiply. Other drugs, such as antibiotics, kill off normal skin bacteria that would otherwise serve as defence against viral infections.

Pathophysiology

Viral infections cause many different kinds of skin disorders, including warts, and herpes simplex and herpes zoster infections.

Warts

Warts or *verrucae*, are lesions of the skin caused by the human papillomavirus (HPV). More than 60 types of HPVs have been found on the human skin and mucous membranes (Bullock & Hales, 2013). Warts may be found on non-genital skin or genital skin and mucous membranes. Non-genital warts are benign lesions; genital warts may be precancerous. Warts are transmitted through skin contact. Wart lesions may be flat, fusiform (tapered at both ends) or round, but most are round and raised and have a rough, grey surface. There are many different types of warts; the location and appearance of the warts depend on the causative virus. Those most common are as follows:
- A common wart (*verruca vulgaris*) may appear anywhere on the skin and mucous membranes of the body, but it most commonly appears on the fingers. Common warts grow above the skin surface and may be dome-shaped with ragged borders (see Figure 15.9).

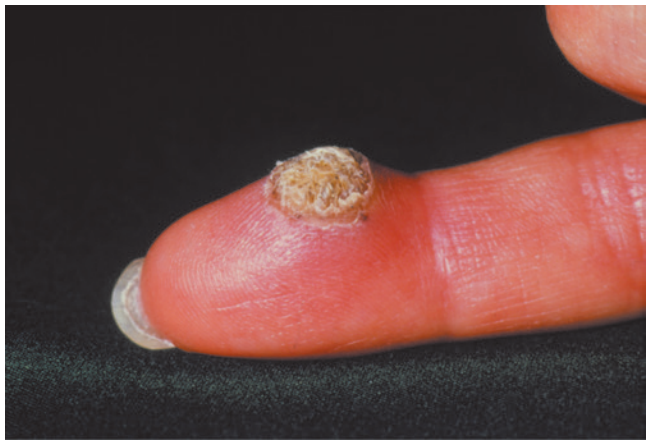


FIGURE 15.9 ■ The common wart, caused by a virus, appears as a raised, dome-shaped lesion

Source: Dr P Marazzi/Science Source.

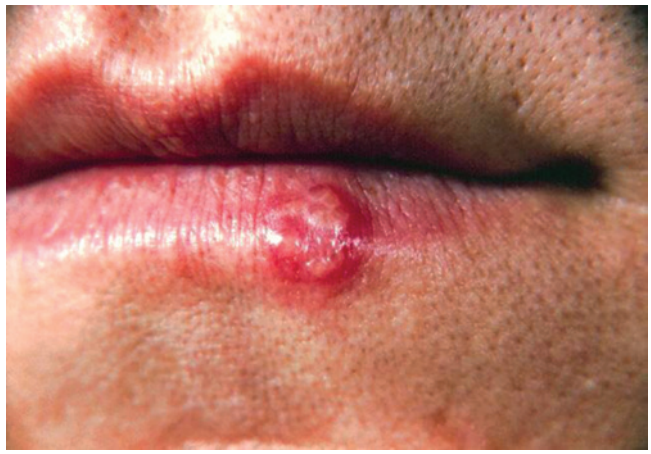


FIGURE 15.10 ■ Herpes simplex is a viral infection of the skin and mucous membranes

Source: CDC/Dr Hermann.

- Plantar warts occur at pressure points on the soles of the feet. The pressure of shoes and walking prevents these warts from growing outwards, so they tend to extend deeper beneath the skin surface than do common warts. Plantar warts are often painful.
- A flat wart (*verruca plana*) is a small flat lesion, usually seen on the forehead or the dorsum of the hand.
- *Condylomata acuminata*, also called HPV or venereal warts, occur in moist areas, along the glans of the penis, in the anal region and on the vulva and cervix. They are usually cauliflower-like in appearance and have a pink or purple colour.

Warts resolve spontaneously when immunity to the virus develops. This response may take up to 5 years.

Herpes simplex

Herpes simplex (also called a *cold sore*) virus infections of the skin and mucous membranes are caused by two types of herpes virus: HSV-1 and HSV-2. Most infections above the waist are caused by HSV-1, with herpes simplex lesions most often found on the lips, face and mouth. (Genital herpes infections, which result from either HSV-1 or HSV-2, are classified as sexually transmitted infections and are discussed in Chapter 49.) The virus may be transmitted by physical contact, oral sex or kissing.

The infection begins with a burning or tingling sensation, followed by the development of erythema, vesicle formation and pain (see Figure 15.10). The vesicles progress through pustules, ulcers and crusting until healing occurs in 10 to 14 days.

The initial infection is often severe and accompanied by systemic manifestations, such as fever and sore throat; recurrences are more localised and less severe. The virus lives in nerve ganglia and may cause recurrent lesions in response to sunlight, menstruation, injury or stress. Oral aciclovir (Zovirax™) may be used prophylactically to prevent reoccurrences and to treat recurrent outbreaks.

Herpes zoster

Herpes zoster, also called *shingles*, is a viral infection of a dermatome section of the skin caused by varicella zoster (the herpesvirus that also causes chickenpox). The infection is believed to result from reactivation of a varicella virus remaining in the sensory dorsal ganglia after a childhood infection of chickenpox. When reactivated, the virus travels from the ganglia to the corresponding skin dermatome area.

Herpes zoster most often affects adults over the age of 60 (Porth & Matfin, 2009). People with Hodgkin's disease, certain types of leukaemia and lymphomas are more susceptible to an outbreak of the disease. Herpes zoster is more prevalent in immunocompromised people, such as those with human immunodeficiency virus (HIV) infections, those receiving radiation therapy or chemotherapy, and those who have had major organ transplants. The appearance of the lesions in people with HIV infections may be one of the first manifestations of immunocompromise. The herpes eruption lasts for about 2 to 3 weeks and usually does not recur.

Herpes zoster lesions are vesicles with an erythematous base. The vesicles appear on the skin area supplied by the neurons of a single or associated group of dorsal root ganglia (although they may occur beyond this area in immunosuppressed people). The lesions usually appear unilaterally on the face, trunk and thorax (see Figure 15.11). New lesions continue to erupt for 3 to 5 days, then crust and dry. Recovery occurs in 2 to 3 weeks. The person often experiences severe pain for up to 48 hours before and during eruption of the lesions. The pain may continue for weeks to months after the lesions have disappeared. The older adult is especially sensitive to the pain and often experiences more severe outbreaks of herpes zoster lesions.

Eruption of vesicles over a single dermatome usually only occurs one time. Generalised herpes zoster may indicate an associated immunocompromised disease, such as Hodgkin's disease or HIV infection. People infected with HIV are 20 times more likely to develop herpes zoster (McPhee, Papadakis & Tierney, 2008).

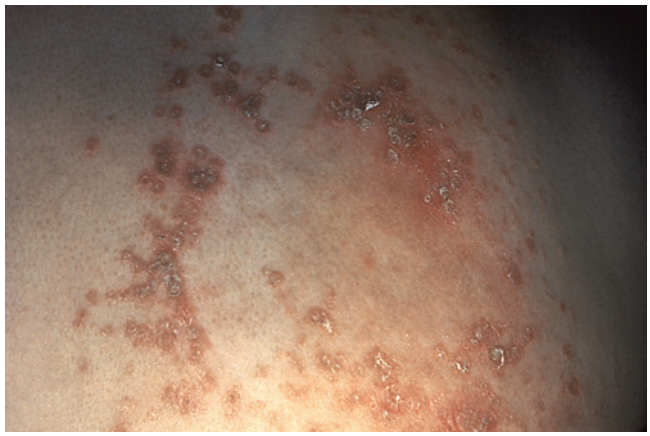


FIGURE 15.11 ■ Herpes zoster is a viral infection of a dermatome section of the skin. The typical lesions are painful vesicles lying along the path of the nerve

Source: CDC/Dr Dancewiez.

Complications of herpes zoster include post-herpetic neuralgia (a sharp, spasmodic pain along the course of one or more nerves) and visual loss. The neuralgia, described as burning or stabbing, results from inflammation of the root ganglia. This complication is more common in people over the age of 55 (McPhee et al., 2008). Permanent loss of vision may follow occurrence of lesions that arise from the ophthalmic division of the trigeminal nerve. The disease may disseminate in immunocompromised people, causing lesions beyond the dermatome, visceral lesions and encephalitis. This serious complication may cause death.

INTERPROFESSIONAL CARE

The treatment for viral skin infections focuses on stopping viral replication and treating the person's responses, such as itching and pain.

Diagnosis

Although diagnosis is usually based on manifestations and appearance of the lesions, laboratory tests may be necessary to differentiate herpes zoster from impetigo, contact dermatitis and herpes simplex. The laboratory tests include a Tzanck smear that identifies the herpesvirus but does not distinguish herpes zoster from herpes simplex. Cultures of fluid from the vesicles and antibody tests are used to make the differential diagnosis of herpesvirus types. HIV testing should be considered if the person has a history of HIV risk factors.

Medications

Most viral skin disorders are treated with antiviral medications, and other types of medications are used to relieve pruritus and pain in the person with herpes zoster.

- *Warts*. Depending on their size, location and any associated discomfort, warts may be treated with medications, cryotherapy or electrodesiccation and curettage. A common method of wart removal is acid therapy, using a colloidal solution of 16% salicylic acid and 16% lactic acid. The solution is applied to the wart every 12 to 24 hours; the wart disappears in 2 to 3 weeks. Other methods of eradicating warts are cryosurgery, freezing with liquid nitrogen and electrodesiccation of the wart with an electric current followed by excision of the dead tissue. Venereal warts are further described in Chapter 49.
- *Herpes simplex*. Herpes simplex lesions are treated with topical aciclovir (Zovirax™), an antiviral agent. Aciclovir shortens the time of symptoms and speeds healing.
- *Herpes zoster*. Antiviral drugs are used to treat herpes zoster infections. Aciclovir interferes with viral synthesis and replication. Although it does not cure herpes infections, it does decrease the severity of the illness and also decreases pain. It may be administered topically, orally or parenterally. It is more effective if administration begins within the first 1 to 2 days after the first vesicles appear. Nerve blocks may be needed to treat initial pain. Narcotic and non-narcotic analgesics are prescribed for pain management and antihistamines may be administered for relief of pruritus. People with eye involvement are treated with topical steroid ophthalmic ointments and mydriatics.

Nursing care

People with viral skin disorders require nursing care for infection, pruritus and pain. They also require teaching about preventing the spread of the virus to others. Of the viral disorders, herpes zoster is the most painful and debilitating. See below for nursing interventions that aim to relieve pain for the person with herpes zoster.

Nursing diagnoses and interventions

This section discusses the nursing care of the person with herpes zoster, focusing on the nursing diagnoses of *Acute pain* related to viral infection of herpes zoster.

Acute pain

The person with herpes zoster often experiences severe pain over the entire dermatome supplied by the affected nerve root. The pain is described as burning, tearing or stabbing. The person may avoid movement and does not want clothing or bed linen to touch the affected area.

- Monitor the location, duration and intensity of the pain.
- Explain the rationale for taking prescribed medications on a regular schedule.
- Teach measures to relieve pruritus:
  - Take prescribed antipruritic medications.
  - Topical application of amorphous hydrogels, especially those that contain tea tree oil, may soothe. Similarly, calamine lotion or wet compresses may be prescribed.
  - Keep the room temperature cool.
  - Use a bed cradle to keep sheets off affected areas of the body.

- Encourage the use of distraction (such as music) or a specific relaxation technique (such as progressive muscle relaxation or deep breathing).

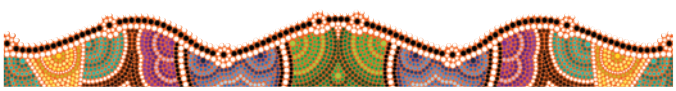
CONSIDERATION FOR PRACTICE

Pregnant women must avoid exposure to people with herpes zoster because the herpes virus can cross the placental barrier.

Community-based care

Most people with viral infections are self-caring in their home environment. Provide the following information and instructions:

- Herpes zoster infections are usually self-limiting and heal completely. Second occurrences of herpes zoster are rare.
- Do not have social contact with children or pregnant women until crusts have formed over the blistered areas with herpes zoster, because the disease is contagious to people who have not had chickenpox.
- Use pain medications regularly.
- Follow suggestions to help reduce itching, scratching and pain: use medications as prescribed, wear lightweight cotton clothing, keep room temperatures cool, wear cotton gloves at night if scratching is a problem and practise relaxation and distraction activities.
- Report to your healthcare provider any increase in pain, fever, chills, drainage that smells bad and has pus or a spread in the blisters.



THE PERSON WITH A PARASITIC INFESTATION

Infestations of the skin by parasites are more common in developing countries but may occur in any geographical area of the world. They affect people of all social classes but are associated with crowded or unsanitary living conditions.

Pathophysiology

In Australia, two of the more common parasitic infestations of the skin are caused by lice and scabies. These parasites do not normally live on the skin, but infest the skin through contact with an infested person or contact with clothing, linen or objects infested with the parasites.

Pediculosis

Pediculosis is an infestation with lice, parasites that live on the blood of an animal or human host. The louse is a 2 to 4 mm oval organism with a stylet that pierces the skin; an anticoagulant in its saliva prevents host blood from clotting while it eats. The female louse lays its eggs (small pearl-grey or brown eggs, called nits) on hair shafts. The louse within the egg hatches, reaches the adult reproductive stage and dies in 30 to 50 days (Bullock & Hales, 2013).

There are three types of human pediculosis:

1. Pediculosis corporis is an infestation with body lice. This type of infestation is more common in people who do not have access to facilities for bathing or washing clothes, such as the homeless. The lice live in clothing fibres and are transmitted primarily by contact with infested clothing and bed linen. The skin lesions occur at the site of a louse bite; macules appear initially, followed by wheals and papules. Pruritus is common and scratching often results in linear excoriations. Secondary infections cause hyperpigmentation and scarring. The lesions are most often seen on the shoulders, trunk and buttocks.
2. Pediculosis pubis is an infestation with pubic lice (often called crabs). This infestation is spread through sexual activity with someone already infested or by contact with infested clothing or linen. The lice are found in the pubic region and occasionally spread to the axillae or men's beards. The lice cause skin irritation and intense itching.
3. Pediculosis capitis is an infestation with head lice. The lice are most often found behind the ears and at the nape of the neck but may also spread to other hairy areas of the body: the eyebrows, pubic area or beard. The lice are transmitted by contact with an infected person or sharing combs, hairbrushes or hats. Manifestations of head lice include pruritus, scratching and erythema of the scalp. If untreated, the hair appears matted and crusted with a foul-smelling substance.

Scabies

Scabies is a parasitic infestation caused by a mite (*Sarcoptes scabiei*). The pregnant female mite burrows into the skin and lays two to three eggs each day for about a month. The eggs hatch in 3 to 5 days and the larvae migrate to the surface but burrow into the skin for food or protection. The larvae develop and the cycle repeats. Scabies infestation affects people of all socioeconomic classes. The infestation is found in webs between the fingers, the inner surfaces of the wrist and elbow, the axillae, the female nipple, the penis, the belt line and the gluteal crease. The lesions are a small red-brown burrow, about 2 mm in length, sometimes covered with vesicles, which appears as a rash. Pruritus in response to the mite or its faeces is common, especially at night, and excoriations may develop. The excoriations predispose the person to secondary bacterial infections (Bullock & Hales, 2013).

INTERPROFESSIONAL CARE

Parasitic infestations are diagnosed by identifying the organism and are treated with medications that kill the lice or scabies.

Diagnosis

When a person has manifestations of pediculosis, the hair shaft and the clothing are examined to identify the lice or the nits. Microscopic examination of the parasite provides a positive

diagnosis. Scabies is diagnosed by skin scrapings and microscopic examination for the mites or their faeces.

Medications

Lice are eradicated with agents that kill the parasite. Infestations of the body and pubic area are treated with topical medications that have a neurotoxic mode of action (permethrin, malathion, carbaril, spinosad) or a physical mode of action (demeicones, isopropyl myristate or herbal remedies). Ivermectin is the only oral treatment currently used as a pediculicide (Feldmeier, 2014).

Manufacturers' instructions are to be followed for use. Repeat applications may be required in some instances to kill newly hatched lice. A fine-toothed comb can be used to comb the dead nits off the hair shaft.

Scabies are treated with topical permethrin or lindane creams and the diligent treatment of other people who have been in close contact, as well as the laundry of contact linen and clothing to prevent reinfestation. Secondary skin infections are common with untreated scabies infestations or immunocompromised individuals.

The associated itching is treated with systemic or topical medications, including corticosteroids. Secondary bacterial infections are treated with the appropriate antibiotic.

Nursing care

Nursing care for the person with a parasite infestation most often focuses on teaching to prevent infestation or to eradicate an existing infestation.

Education for the person and family is necessary to facilitate treatment at home, to prevent the spread of the infestation and to dispel the myth that lice infest only people with poor hygiene or in dirty living conditions. Specific information includes the following:

- Wash clothing and linen in soap and hot water or have them dry-cleaned.
- Ironing the clothes kills any lice eggs.
- Personal care items, such as combs or brushes, may be boiled to kill the parasites.
- All family members and sexual partners must also be treated.
- Avoid using the combs, brushes or hats of others.
- Lice and mites may infest anyone.

INFLAMMATORY DISORDERS OF THE SKIN

The inflammatory skin disorders discussed in this section are dermatitis and acne.

THE PERSON WITH DERMATITIS

Dermatitis is an inflammation of the skin characterised by erythema and pain or pruritus. Dermatitis may be acute or chronic.

Pathophysiology

In dermatitis, various exogenous and endogenous agents cause an inflammatory response of the skin. Different types of skin eruptions occur, often specific to the causative allergen, infection or disease. The initial skin responses to these agents or illnesses include erythema, formation of vesicles and scales, and pruritus (see Figure 15.12). Subsequently, irritation from scratching promotes oedema, a serous discharge and crusting. Long-term irritation in chronic dermatitis causes the skin to become thickened and leathery and darker in colour.

Contact dermatitis

Contact dermatitis is a type of dermatitis caused by a hypersensitivity response or chemical irritation. The major sources known to cause contact dermatitis are dyes, perfumes, certain plants (ivy, oak, sumac), chemicals, latex and metals (see Box 15.3).

Allergic contact dermatitis is a cell-mediated or delayed hypersensitivity to a wide variety of allergens. Sensitising antigens include microorganisms, plants, chemicals, drugs, metals or foreign proteins. On initial contact with the skin, the allergen binds to a carrier protein, forming a sensitising antigen. The antigen is processed and carried to the T cells, which in turn become sensitised to the antigen. The first exposure is the sensitising contact and the person does not experience manifestations, which then occur with subsequent exposures. The manifestations include erythema, swelling and pruritic vesicles in the area of allergen contact. For example, a person hypersensitive to metal may have lesions under a ring or watch.

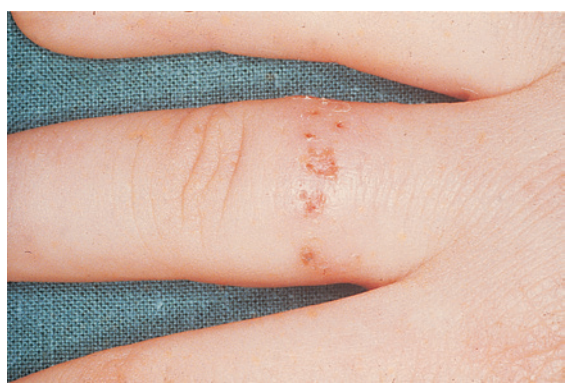


FIGURE 15.12 ■ Dermatitis may be a response to allergens, infections or chemicals. This person has contact dermatitis resulting from the metal salts in a ring

Source: © Biophoto Associates/Science Source.

BOX 15.3 Common causes of contact dermatitis

- Acids
- Alkalis: soaps, detergents, household ammonia, lye, cleaners
- Bromide
- Chlorine
- Cosmetics: perfumes, dyes, oils
- Dusts of lime, arsenic, wood
- Hydrocarbons: crude petroleum, lubricating oil, mineral oil, paraffin, asphalt, tar
- Iodine
- Insecticides
- Fabrics: wool, polyester, dyes, sizing
- Latex: gloves, catheters
- Metal salts: calcium chloride, zinc chloride, copper, mercury, nickel (common in costume jewellery and support bras), silver
- Plants: chrysanthemums, primula, tomato plants, grevillea, English ivy and rhus trees
- Colouring agents
- Rubber and leather products
- Soot

FAST FACTS

Latex allergy

- The increased use of latex gloves among healthcare providers has resulted in increased reporting of latex allergies. It is estimated that 10% to 17% of healthcare providers are allergic to latex (Gawchik, 2011).
- The most common type of allergic response to latex gloves is type IV, T-cell-mediated contact dermatitis.
- Type I, IgE-mediated hypersensitivity, manifested by urticaria, rhinoconjunctivitis, asthma or anaphylaxis, is far more serious than the T-cell-mediated type.
- The person with a latex allergy should be treated in a latex-free environment.
- Healthcare providers with severe allergic responses to latex may have to seek a different type of employment.

Irritant contact dermatitis is an inflammation of the skin from irritants; it is not a hypersensitivity response. Common sources of irritant contact dermatitis include chemicals (such as acids), soaps and detergents. The skin lesions are similar to those seen in allergic contact dermatitis.

Atopic dermatitis

Atopic dermatitis is an inflammatory skin disorder that is also called *eczema*. The exact cause is unknown, but related factors include depressed cell-mediated immunity, elevated IgE levels and increased histamine sensitivity. The disorder is seen more often in children, but chronic forms persist throughout life.

People with atopic dermatitis have a family history of hypersensitivity reactions, such as dry skin, eczema, asthma and allergic rhinitis. Although up to one-third of people with atopic dermatitis also have food allergies, a positive correlation has not been found.

The dermatitis results when mast cells, T lymphocytes, monocytes and other inflammatory cells are activated and release histamine, lymphokines and other inflammatory mediators. The immune response interacts with the allergen to create a chronic inflammatory condition. In the adult form of atopic dermatitis, characteristic lesions include chronic lichenification, erythema and scaling, the result of pruritus and scratching. The lesions are usually found on the hands, feet or flexor surfaces of the arms and legs (see Figure 15.13). Scratching and excoriation increase the risk of secondary infections, as well as invasion of the skin by viruses such as herpes simplex. Serum studies may find elevated eosinophil and IgE levels.

Seborrhoeic dermatitis

Seborrhoeic dermatitis is a chronic inflammatory disorder of the skin that involves the scalp, eyebrows, eyelids, ear canals, nasolabial folds, axillae and trunk. The cause is unknown. This disorder is seen in all ages, from the very young (called 'cradle cap') to the very old. People taking methyldopa (Aldomet™) for hypertension occasionally develop this disorder and it is a component of Parkinson's disease. Seborrhoeic dermatitis is also frequently seen in people with AIDS.

The lesions are yellow or white plaques with scales and crusts. The scales are often yellow or orange and have a greasy appearance. Mild pruritus is also present. Diffuse dandruff with erythema of the scalp often accompanies the skin lesions.

Exfoliative dermatitis

Exfoliative dermatitis is an inflammatory skin disorder characterised by excessive peeling or shedding of skin. The cause is unknown in about half of all cases, but a pre-existing skin disorder (such as psoriasis, atopic dermatitis, contact dermatitis or seborrhoeic dermatitis) is found in a majority of the cases (McPhee et al., 2008). Reactions to medications, such as sulfonamides, account for 20% to 40% of cases. Certain cancers (such as lymphoma) may also cause exfoliative dermatitis.

FIGURE 15.13 ■ Atopic dermatitis or eczema causes pruritus, resulting in lichenification, erythema and scaling

Source: Christine Langer-Pueschel/Shutterstock.

Both systemic and localised manifestations may appear. Systemic manifestations include weakness, malaise, fever, chills and weight loss. Scaling, erythema and pruritus may be localised or involve the entire body. In addition to peeling of skin, the person may lose their hair and nails. Generalised exfoliative dermatitis may cause debility and dehydration. The impairment of skin integrity increases the risk of local and systemic infections.

INTERPROFESSIONAL CARE

The person with dermatitis is treated primarily with topical medications and therapeutic baths. If the dermatitis is due to hypersensitivity to an allergen, the person avoids exposure to environmental irritants and suspected foods. The person also discontinues as many medications as possible to determine whether the dermatitis is the result of a drug allergy.

Diagnosis

The diagnosis is often based on the manifestations of the disorder and on a history of exposure to a known allergen. Patch tests and intradermal tests are used to identify a specific allergen.

Medications

The medications used depend on the cause of the dermatitis and the severity of the manifestations. Minor cases are treated with antipruritic medications, whereas more severe cases are treated with oral antihistamines, oral and/or topical corticosteroids and wet dressings. Topical anti-infectives may be prescribed.

Nursing care

Nursing care of the person with dermatitis focuses primarily on providing information for self-care at home. The person is responsible for managing skin problems and requires education and support. The following topics should be addressed in the person's management plan:

- Medications and treatments do not cure the disease; they only relieve the symptoms.
- Dry skin increases pruritus, which stimulates scratching. Scratching may in turn cause excoriation, and excoriation increases the risk of infection.
- It may be necessary to change the diet or environment to avoid contact with allergens.
- When using steroid preparations, apply only a thin layer to slightly damp skin (e.g. after taking a bath). See Box 15.4 for information on dosage for steroid preparations.
- Occlusive dressings or wraps may enhance the effect of topically applied steroids.
- When using oral corticosteroids, never abruptly stop taking the medication. Rather, follow instructions to taper the dosage gradually.

Box 15.4 Fingertip unit

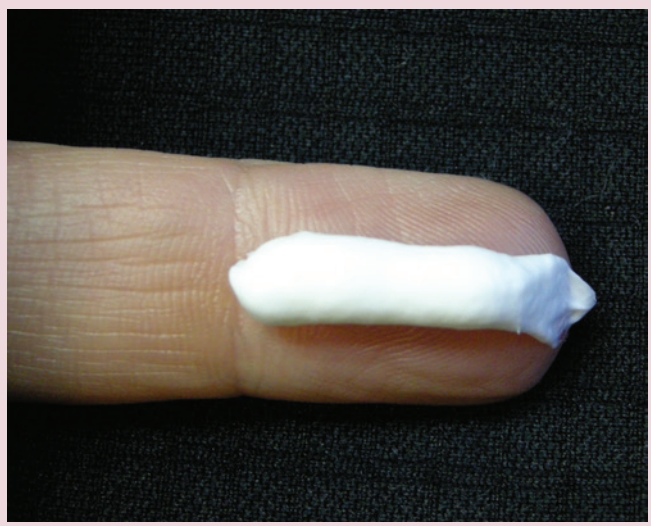
It can be hard to know how much cream or ointment to apply to an area. If you apply too little, it may not work; too much, and you risk side effects (Australian Medicines Handbook (AMH), 2012).

Dose of cream in a fingertip unit varies with age:

- adult male: one fingertip unit provides 0.5 g
- adult female: one fingertip unit provides 0.4 g
- children of 4 years: approximately one-third of adult amount
- infants 6 months to 1 year: approximately one-quarter of adult amount

Amount of cream used varies with body part:

- one hand: apply one fingertip unit
- one arm: apply three fingertip units
- one foot: apply two fingertip units
- one leg: apply six fingertip units
- face and neck: apply 2.5 fingertip units
- trunk, front and back: 14 fingertip units
- entire body: about 40 fingertip units (DermNet NZ, 2014).



A fingertip unit describes the amount of cream squeezed out of its tube onto the end of the finger as shown

Source: © DermNetNZ.

- Antihistamines cause drowsiness. When using these medications, avoid alcohol and use caution when driving or working around machinery.

THE PERSON WITH ACNE

Acne is a disorder of the pilosebaceous (hair and sebaceous gland) structure, which opens to the skin surface through a pore. The sebaceous glands, which empty directly into the hair

follicle, produce sebum, a lipid substance. Sebaceous glands are present over the entire skin surface except the soles of the feet and the palms of the hands, but the largest glands are on the face, scalp and scrotum. Sebum production is a response to direct hormonal stimulation by testicular androgens in men and adrenal and ovarian androgens in women.

Pathophysiology

Acne may be non-inflammatory or inflammatory. Non-inflammatory acne lesions are primarily **comedones**, more commonly called pimples, whiteheads and blackheads. Whiteheads are pale, slightly elevated papules categorised as closed comedones. Blackheads are plugs of material that accumulate in the sebaceous glands. They are categorised as open comedones. The colour is the result of the movement of melanin into the plug from surrounding epidermal cells. Inflammatory acne lesions include comedones, erythematous pustules and cysts (see Figure 15.14). Inflammation close to the skin surface results in pustules; deeper inflammation results in cysts. The inflammation is believed to result from irritation from fatty acid constituents of the sebum and from substances produced by *Propionibacterium acnes* bacteria, both of which escape into the dermis when the follicular walls of closed comedones rupture.

Several forms of acne occur at different periods of the lifespan. The most common are acne vulgaris, acne rosacea and acne conglobata.

Acne vulgaris

Acne vulgaris is the form of acne common in adolescents and young to middle adults. The actual cause of acne vulgaris is unknown. Possible causes include an androgenic influence on the sebaceous glands, increased sebum production and proliferation of the organism *P. acnes*. Many factors once thought to cause acne vulgaris, including high-fat diets, chocolate, infections and cosmetics, have been disproved (Bullock & Hales, 2013).

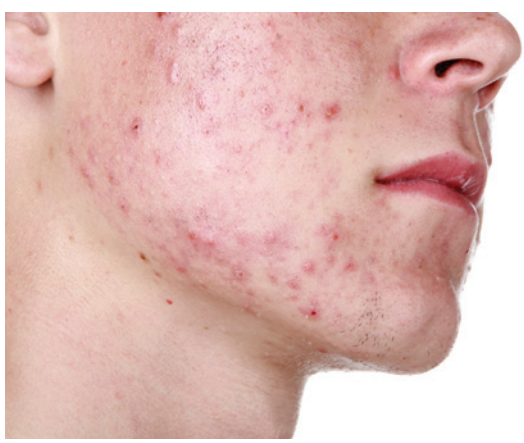


FIGURE 15.14 ■ Acne vulgaris lesions include comedones, erythematous pustules and cysts

Source: Suzanne Tucker/Shutterstock.

FAST FACTS

Acne vulgaris

- Acne vulgaris is the most common of all skin conditions.
- 12% of women and 3% of men over the age of 25 have acne vulgaris and the rate does not begin to decrease until after age 44 (McPhee et al., 2008).
- Scarring may be a sequela of the disease or may result from the person picking and manipulating the comedones.

Mild cases may involve only a few scattered comedones, but severe cases are manifested by multiple lesions of all types. Most acne vulgaris lesions form on the face and neck, but they also occur on the back, chest and shoulders. Women in their thirties and forties, often with no prior acne, may develop papular lesions on the chin and around the mouth. The lesions are usually mildly painful and may itch. The complications of acne vulgaris, especially in severe cases, are formation of cysts, pigment changes in people with dark skin, severe scarring and lowered self-concept from the skin eruptions.

Acne rosacea

Acne rosacea is a chronic type of facial acne that occurs more often in middle and older adults. The cause is unknown. The lesions of acne rosacea begin with erythema over the cheeks and nose. Other skin lesions may appear. Over the years, the skin colour changes to dark red and the pores over the area become enlarged. The soft tissue of the nose may exhibit *rhinophyma*, an irregular bullous thickening.

Acne conglobata

Acne conglobata is a chronic type of acne of unknown cause that begins in middle adulthood. This type causes serious skin lesions. Comedones, papules, pustules, nodules, cysts and scars occur primarily on the back, buttocks and chest, but may occur on other body surfaces. The comedones have multiple openings and a discharge that ranges from serous to purulent with a foul odour.

INTERPROFESSIONAL CARE

The management of acne is similar, regardless of type. Because acne vulgaris is most common, the discussions of interprofessional and nursing care focus on that type. Treatment is based on the type and severity of the lesions.

Diagnosis

The disease is diagnosed by the typical location and appearance of lesions. If the person has pustules, a culture of the drainage is performed to differentiate viral or bacterial dermatitis from acne.

Medications

The treatment of acne is tailored to the individual and is based on the severity of the lesions. For acne with comedones, tretinoin or benzoyl peroxide preparations are prescribed. Azelaic acid may

also be used. The administration of these vitamin A analogues is discussed in the 'Medication administration' box below. Benzoyl peroxide preparations are found in over-the-counter medications, and these products are keratolytic and loosen the comedones.

Mild forms of papular inflammatory acne are treated with topical clindamycin, a bacteriostatic agent that decreases the amount of fatty acids on the skin surface. This medication may be combined with tretinoin therapy.

Moderate forms of papular inflammatory acne are treated with oral or topical antibiotics, such as tetracycline, erythromycin and minocycline. These anti-acne antibiotics are administered for 3 to 4 months; if the person's skin is clear, the dose is lowered gradually to a maintenance dose that will maintain clear skin.

Severe forms of papular inflammatory acne are treated with isotretinoin. This drug is effective, but has serious side effects. Isotretinoin, with nursing responsibilities, is discussed in the 'Medication administration' box.

Treatments

Acne scars may alter the individual's self-concept. They may be removed by dermabrasion and laser treatment. Dermabrasion of inactive acne lesions can improve the person's appearance, especially if the scars are flat. Laser excision of deep scars may also be performed.

Nursing care

Nursing care is individualised to the person's developmental needs and is conducted primarily through teaching in clinics or the home setting. Regardless of the person's age or gender, it is important to remember that almost everyone with acne is embarrassed by and self-conscious about their

MEDICATION ADMINISTRATION Anti-acne retinoids

ANTI-ACNE RETINOIDS

Tretinoin

Isotretinoin

Tretinoin is a vitamin A derivative classified as an acne agent. This topical agent acts as an irritant to decrease the cohesiveness of follicular epithelial cells, thereby decreasing comedone formation while increasing the extrusion of comedones from the skin surface.

Isotretinoin is a vitamin A analogue classified as an acne product. It reduces the size of sebaceous glands, inhibits sebaceous gland differentiation to decrease sebum production and alters sebum lipid composition.

Nursing responsibilities

- Administer tretinoin with caution to pregnant women, because the effects of absorption on the developing foetus are not clearly defined.
- Isotretinoin is absolutely contraindicated for pregnant women or for women who want to become pregnant. The medication poses a high risk of major deformities in the infant if pregnancy occurs during use, even use that continues only for short periods.
- Do not administer to people with eczema or to those who are hypersensitive to the sun.

Health education for the person and family

Tretinoin

- Use the prescribed cream in a test area twice at night to test for sensitivity; if no reaction occurs, increase applications gradually to the prescribed frequency.
- A pea-sized amount of the cream is enough to cover the entire face.
- Apply the cream to clean, dry skin.
- Do not apply the cream to the eyes, mouth, angles of the nose or mucous membranes.
- Wash the face no more than two to three times a day, using a pH neutral cleanser or mild soap. Do not use skin preparations (such as aftershave lotion or perfumes) that contain alcohol, menthol, spice or lime; they may irritate your skin.
- The medication may cause a temporary stinging or warm sensation but should not cause pain.
- The skin where you apply the cream will be mildly red and may peel; if you experience a more severe reaction, consult your healthcare provider.
- The medication may cause increased sensitivity to sunlight; use sunscreens and wear protective clothing when outdoors.
- Acne may become worse during the first 2 weeks of treatment; this is an expected response.

Isotretinoin

- Take the prescribed medication with food.
- Acne may become worse during the initial period of treatment; this is an expected response.
- The medication causes dryness of the eyes, so the person will have trouble wearing contact lenses during and after treatment.
- Do not take vitamin A supplements; they will increase the effects of the medication.
- Avoid prolonged exposure to sunlight; use sunscreen and protective clothing when in the sun.
- Notify the physician at once if abdominal pain, severe diarrhoea, rectal bleeding, headache, nausea or vomiting, or visual disturbances occur.
- Do not drink alcohol while taking this medication. (It causes an increase in triglycerides.)
- Night vision may become worse; use caution when driving at night.
- Do not donate blood while, or for 1 month after, taking this medication.
- (For *women*) Use two reliable forms of contraception simultaneously for at least 1 month before, during and at least 1 month after therapy with this medication. The medication may cause deformities in a baby conceived at this time.

appearance. Prior to teaching, establish rapport with the person and clarify beliefs; for example, the person may believe some myths such as the lesions result from poor hygiene, masturbation, use of cosmetics, eating the wrong types of foods or lack of sexual activity. It is critical to teach the person about the causes of and factors involved in acne prior to teaching self-care.

The teaching plan for the person with acne includes general guidelines for skin care and health, as well as specific guidelines for care of the acne lesions. The following topics should be addressed in the person's management plan:

- Wash the skin with a pH-neutral cleanser or mild soap and water at least twice a day to remove accumulated oils.
- Shampoo the hair often enough to prevent oiliness.
- Eat a regular, well-balanced diet. Foods do not cause or increase acne.
- Expose the skin to sunlight, but avoid sunburn.
- Get regular exercise and sleep.
- Try to avoid putting your hands on your face.
- Do not squeeze a pimple. Squeezing forces the material of the pimple deeper into the skin and may cause the pimple to become larger and infected.
- The treatment for acne lasts months, and in some cases for the rest of one's life. It is very important to take the medications each day for the prescribed length of time.

MALIGNANT SKIN DISORDERS

THE PERSON WITH ACTINIC KERATOSIS

Actinic keratosis, also called senile or solar keratosis, is an epidermal skin lesion directly related to chronic sun exposure and photo damage. The prevalence is highest in people with light-coloured skin; these lesions are rare in people with dark skin. Actinic keratosis may progress to squamous cell carcinoma. Fewer than 1% of early lesions become malignant, but many of those that persist progress to malignancy (Porth & Matfin, 2009). Because of this tendency, the lesions are classified as premalignant.

The lesions are erythematous rough macules a few millimetres in diameter. They are often shiny but may be scaly; if the scales are removed, the underlying skin bleeds. They occur in multiple patches, primarily on the face, dorsa of the hands, the forearms and sometimes on the upper trunk (see Figure 15.15). Enlargement or ulceration of the lesions suggests transformation to malignancy.

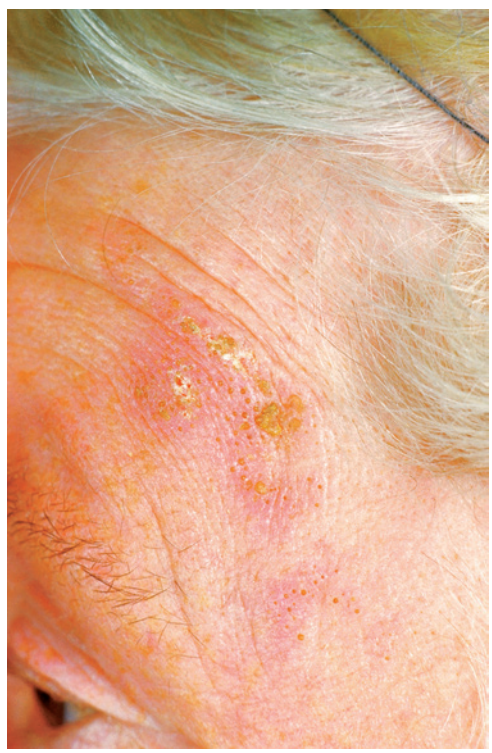


FIGURE 15.15 ■ The effects of long-term sun exposure are illustrated in this epidermal skin lesion, called actinic keratosis

Source: © Medical-On-Line/Alamy.

THE PERSON WITH NON-MELANOMA SKIN CANCER

The skin, despite its ability to protect the internal body from external damage, is a fragile organ and is subject to damage from ultraviolet radiation and chemicals. Over time, this damage results in alterations in cellular structure and function, and malignancies of the skin occur. The common skin cancers found among Australians are non-melanoma skin cancers (NMSCs) such as basal cell carcinoma and squamous cell carcinoma, as well as malignant melanoma.

The health burden of non-melanoma skin cancer

The leading cause of death in Australia is cancer, and non-melanoma skin cancers are the most commonly diagnosed cancers (Cancer Council Australia, 2015). However, although other invasive cancers are reportable by law to cancer registries, there is no such requirement for NMSCs and the scope of the health burden is determined from review of hospital and general medical practice records. The Australian Institute of Health and Welfare (AIHW) and Australasian Association of Cancer Registries (AACR) (2012) estimated that 815 Australians were diagnosed with NMSCs in 2012. The mortality rate is

higher among fair-skinned Australian-born people (83%) and those born in the United Kingdom and Ireland (9%) (AIHW & Cancer Council Australia, 2013). The risk of dying also increases with age, and males are more affected than females (AIHW & AACR, 2012).

Risk factors

Multiple aetiological factors are involved in the development of non-melanoma skin cancer, including environmental factors and host factors.

Environmental factors

The environmental factors implicated in the non-melanoma skin cancers are ultraviolet radiation, pollutants, chemicals, ionising radiation, viruses and physical trauma.

FAST FACTS

Risk factors for non-melanoma skin cancer

- Fair skin, freckles, blue or green eyes, and blonde or red hair
- Family history of skin cancer
- Unprotected and/or excessive exposure to UV radiation (natural or artificial)
- Radiation treatment
- Occupational exposures to coal, tar, pitch, creosote, arsenic compounds or radium
- Severe sunburn episodes as a child

Ultraviolet radiation (UVR) from the sun is believed to be the cause of most non-melanoma skin cancers. Sunlight contains both short-length rays (UVB) and long-length rays (UVA). UVB rays are absorbed by the top layer of skin and cause sunburn. UVA rays penetrate deeper into the skin layers, causing tissue damage. Both types of rays are believed to cause DNA alterations and also suppress T-cell and B-cell immunity. The amount of UVR reaching the earth is increasing, most likely from depletion of the ozone layer surrounding the planet (Diaz & Nesbitt, 2013).

Geographical, environmental and lifestyle factors affect the amount of exposure to the sun and the risk of NMSC. People who live in latitudes close to the equator and those who live at higher altitudes receive greater ultraviolet radiation exposure. The amount of clothing worn, the time of day and the amount of time in the sun also determine the amount of exposure. Exposure to ultraviolet radiation in tanning booths has also been implicated in the development of NMSC.

Certain chemicals have long been associated with NMSC. Polycyclic aromatic hydrocarbons, found in mixtures of coal, tar, asphalt, soot and mineral oils, have been linked with skin cancers. Psoralens, used in conjunction with UVA for treatment of psoriasis and cutaneous T-cell lymphoma, increase the risk of squamous cell carcinoma.

Other factors associated with NMSC are the use of ionising radiation, viruses and physical trauma. X-ray therapy for tinea capitis and the use of radium to treat other malignancies are risk factors. Human papillomavirus is implicated in the development of squamous cell carcinoma. Squamous cell changes can also occur in scar or chronic wounds and are referred to as Marjolin's ulcers (Pekarek, Buck & Osher, 2011).

Host factors

Certain host factors increase the risk of non-melanoma skin cancer. These include skin pigmentation, as well as the presence of premalignant lesions.

Skin pigmentation is an important factor in the development of NMSC. The amount of melanin pigment produced by the melanocytes determines a person's skin colour. The more melanin, the more the skin is protected from the damage produced by ultraviolet rays. Indigenous Australians, African Americans, Asians and people of Mediterranean descent have a much lower incidence of NMSC than do people who have fair complexions and tend to freckle or sunburn easily, such as people of northern European ancestry.

A major risk factor in the development of NMSC is a change in an existing lesion or the presence of a premalignant lesion, such as actinic keratosis. Organ transplant recipients who undergo immunosuppression to prevent rejection are also at risk of the development of squamous cell carcinoma.

Pathophysiology

Basal cell carcinoma and squamous cell carcinoma arise from epithelial tissue but have different pathophysiology, classifications and manifestations.

Basal cell carcinoma

Basal cell carcinoma (BCC) is an epithelial tumour believed to originate either from the basal layer of the epidermis or from cells in the surrounding dermal structures. These tumours are characterised by an impaired ability of the basal cells of the epidermis to mature into keratinocytes, with mitotic division beyond the basal layer. This results in a bulky neoplasm that grows by direct extension and destroys surrounding tissue, including healthy skin, nerves, blood vessels, lymphatic tissue, cartilage and bone. Basal cell carcinoma is the most common but least aggressive type of skin cancer, rarely metastasising.

Basal cell carcinomas tend to recur. Tumours greater than 2 cm in diameter have a high recurrence rate. Predisposing factors for metastasis are the size of the tumour and the person's resistance to treatment with surgery or chemotherapy. Even though they rarely metastasise, untreated BCCs invade surrounding tissue and may destroy body parts, such as the nose or eyelid.

Basal cell carcinoma is classified into different types: nodular, superficial, pigmented, morpheaform and keratotic. These types are described below and are summarised in Table 15.3.

Nodular basal cell carcinoma is the most common type of BCC and most often appears on the face, neck and head. The tumour is made up of masses of cells that resemble epidermal basal cells and grow in a bulky, nodular form from lack of keratinisation. In the early stages, the tumour is a papule that looks like a smooth pimple. It is often pruritic and continues to grow at a steady rate, doubling in size every 6 to 12 months. As the tumour grows, the epidermis thins, but it remains intact. The skin over the tumour is shiny and either pearly white, pink or flesh coloured. Telangiectasis may be visible

TABLE 15.3 Types and characteristics of basal cell carcinomas

TYPE	COMMON LOCATION	MANIFESTATION
Nodular	Face, neck, head	Small, firm papule; pearly, white, pink or flesh coloured; telangiectasis; enlarges; may ulcerate.
Superficial	Trunk, extremities	Papules or plaque that is flat, erythematous or scaling; pink colour; well-defined borders; may have shallow erosions and surface crusting.
Pigmented	Head, neck, face	Dark brown, blue or black colour; border is shiny and well defined.
Morpheaform	Head, neck	Looks like a flat scar; ivory or flesh coloured.
Keratotic	Ear	Small, firm papule; pearly, white, pink or flesh coloured; may ulcerate.

over the area of the tumour. As the tumour continues to increase in size, the centre or periphery may ulcerate and the tumour develops well-circumscribed borders. It bleeds easily from mild injury.

Superficial basal cell carcinoma, found most often on the trunk and extremities, is the second most common type of BCC. This tumour is a proliferating tissue that attaches to the undersurface of the epithelium. The tumour is a flat papule or plaque, often erythematous, with well-defined borders. The tumour may ulcerate and be covered with crusts or shallow erosions (see Figure 15.16).

Pigmented basal cell carcinoma, found on the head, neck and face, is less common. This tumour concentrates melanin pigment in the centre of the basal cancer cells, giving it a dark brown, blue or black appearance. The border of the tumour is shiny and well defined.

Morpheaform basal cell carcinoma, the rarest form of BCC, usually develops on the head and neck. The tumour forms finger-like projections that extend in any direction along dermal tissue planes. The tumour resembles a flat ivory or flesh-coloured scar. This form is more likely to extend into and destroy adjacent tissue, especially muscle, nerve and bone. It is often more difficult to diagnose because of its appearance.

Keratotic basal cell carcinoma (basosquamous) is found on the preauricular and postauricular groove. It contains both basal cells and squamoid-appearing cells that keratinise. Its appearance is much like that of nodular basal cell carcinoma. This type of BCC tends to recur locally and also is the type most likely to metastasise.

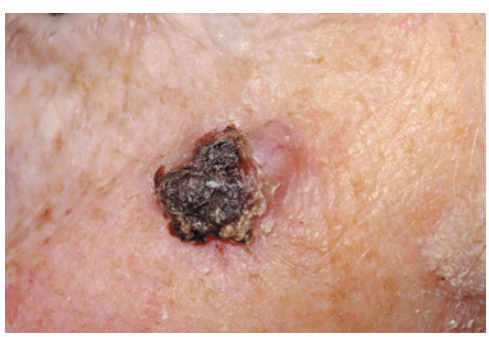


FIGURE 15.16 ■ A superficial basal cell carcinoma is characterised by erythema, ulcerations and well-defined borders

Source: Dr P. Marazzi/Science Source.

Squamous cell carcinoma

Squamous cell carcinoma (SCC) is a malignant tumour of the squamous epithelium of the skin or mucous membranes. It occurs most often on areas of skin exposed to ultraviolet rays and weather, such as the forehead, helix of the ear, top of the nose, lower lip and back of the hands. Squamous cell carcinoma may also arise on skin that has been burned or has chronic inflammation. This is a much more aggressive cancer than basal cell carcinoma, with a faster growth rate and a much greater potential for metastasis if untreated.

The tumours arise when the keratinising cells of the squamous epithelium proliferate, producing a growth that eventually fills the epidermis and invades the dermal tissue planes. Keratinisation of some cells is present and the formation of keratin 'pearls' is common. The keratin formation diminishes as the tumour grows. As the tumour grows, the tumour cells increase in number and rate of mitosis, forming odd shapes.

Squamous cell carcinoma begins as a small, firm red nodule. The tumour may be crusted with keratin products. As it grows, it may ulcerate, bleed and become painful. As the tumour extends into the surrounding tissue and becomes a nodule, the area around the nodule becomes indurated (hardened) (see Figure 15.17).

Recurrent squamous cell carcinoma can be invasive, increasing the risk of metastasis. Invasive squamous cell carcinoma

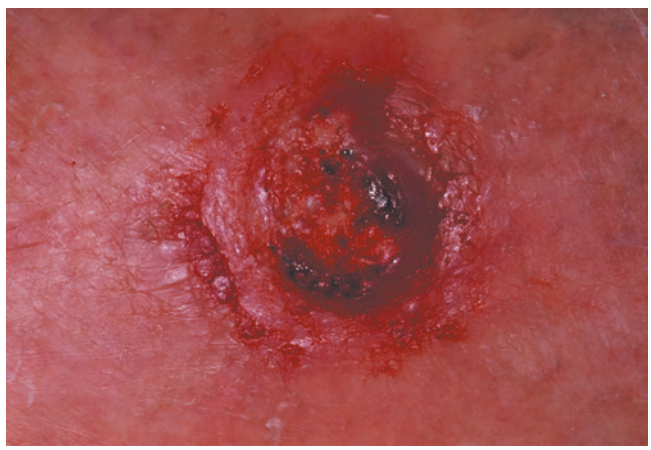


FIGURE 15.17 ■ As a squamous cell carcinoma grows, it tends to invade surrounding tissue. It also ulcerates, may bleed and is painful

Source: Dr P. Marazzi/Science Source.

may arise from pre-existing skin lesions, such as scars and actinic keratosis, and extend into the dermis (called intraepidermal squamous cell carcinoma). This form appears as a slightly raised erythematous plaque with well-defined borders. Metastasis occurs most often via the lymphatics. The degree of risk of metastasis depends on the size and depth of penetration of the tumour.

INTERPROFESSIONAL CARE

Treatment of non-melanoma skin cancer focuses on removal of all malignant tissue using such methods as surgery, curettage and electrodessication, cryotherapy or radiotherapy. These modalities offer a greater than 90% cure rate. After the malignant tissue is removed, the person should have regular examinations for recurrence.

Diagnosis

Non-melanoma skin cancer is diagnosed by microscopic examination of tissue biopsied from the tumour. The biopsy is usually done in a medical practice or clinic under local anaesthesia. The types of biopsy used are shave, punch, incisional and excisional. See Chapter 14 for further information.

Treatments

Depending on the type, size and location of a non-melanoma skin cancer, it may be treated with surgical excision, Mohs surgery, curettage and electrodessication, or radiation.

SURGICAL EXCISION Both basal cell carcinomas and squamous cell carcinomas are excised surgically. The surgery may be minor or major, depending on the size and location of the tumour. Surgery for small tumours is most often performed in the outpatient surgery department or in the surgeon's office. Surgical excision allows rapid healing and yields good cosmetic results, but, as with any surgery, carries the risk of infection.

The goal of surgical excision is to remove the tumour completely, so some surrounding tissue is excised along with the tumour. If the tumour is on the face, the incision is made along normal wrinkle or anatomical lines so that the scars will be less obvious. The incision is closed in layers to leave the smallest possible scar. A pressure dressing is usually applied over the incision to provide support.

If a large tumour is removed, a skin graft or skin flap may be performed in hospital to cover the excised area.

MOHS SURGERY In Mohs surgery (also known as chemosurgery), thin layers of the tumour are horizontally shaved off. A frozen section of the tissue is stained at each level to determine tumour margins. This method is the most accurate in assessing the extent of non-melanoma skin cancer and the method that conserves the most normal tissue. It is often used in areas such as the nose, the nasolabial fold, the medial canthus and the ear.

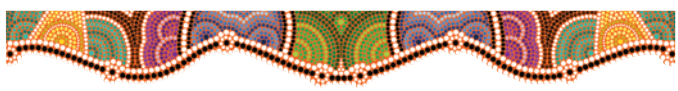
CURETTAGE AND ELECTRODESICCATION Curettage and electrodesiccation are used to treat BCCs that are less than 2 cm in diameter, are superficial or recur because of poor margin control. They may also be used for primary SCCs that are less than 1 cm in diameter and have distinct borders. This type of treatment is most successful for tumours on anatomical sites over a fixed underlying surface, such as the ear, chest and temple.

Abnormal tissue is scraped away (curettage) within 1 to 2 mm of the margin and then a low-voltage electrode is used to abrade the tumour base (electrodesiccation). Tumour tissue is much softer and more friable than normal tissue. Therefore, curettage and electrodesiccation is not used for lesions where the dermis is thin (such as the eyelid) or where the tumour extends into the subcutaneous tissue.

Curettage and electrodesiccation provide good cosmetic results and preserve normal tissue. However, healing time is longer and it is difficult to ensure that all tumour margins have been removed.

Instead of a low-voltage electrode, some physicians use a carbon dioxide laser to vaporise the tumour. When used in conjunction with curettage, this treatment is effective on superficial basal cell carcinomas. Carbon dioxide vaporisation results in minimal thermal injury to adjacent cells, less pain and quicker healing.

RADIATION THERAPY Radiation is most often used for lesions that are inoperable because of their location (such as tumours on the corner of the nose, the eyelid, the canthus and the lip) or size (between 1 and 8 cm). Radiotherapy is also used for people who are older or of poor surgical risk. Radiation is painless and can be used to treat areas surrounding the tumour if necessary. However, the treatment—given over 3 to 4 weeks in a clinical facility—does not allow control of tumour margins and may itself cause skin cancer.



Nursing care

The increasing number of people with skin cancer means that nurses must be involved in prevention and early detection. Nurses have the opportunity to teach preventive behaviours in all settings, including the hospital, home, community, school and clinic.

Nursing care for the person with non-melanoma skin cancer depends on the treatment used. Surgical excision is the most common form of treatment; nursing care depends on the extent of the procedure. However, regardless of the type of treatment, the person will have impaired skin integrity, an increased risk of infection and anxiety about the future following a diagnosis of cancer. Interventions with rationales for the person with any type of skin cancer are discussed in the following section on malignant melanoma.

Health promotion

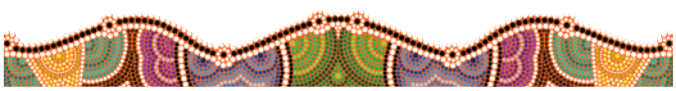
It is well known that cumulative sun exposure positively correlates with non-melanoma skin cancers. Many skin cancers can be prevented by limiting exposure to risk factors. Primary prevention behaviours are outlined in Box 15.5. Information about sunscreens is listed in Box 15.6.

BOX 15.5 Preventing skin cancer

- Minimise sun exposure between the hours of 10 am and 3 pm, when ultraviolet rays are the strongest.
- Cover up with a wide-brimmed hat, sunglasses, long-sleeved shirt and long pants made of tightly woven materials when in the sun.
- Apply a waterproof or water-resistant sunscreen with an SPF of 15 or higher at least 30 minutes before every exposure to the sun. If swimming or sweating heavily, reapply every hour.
- Apply sunscreen not only on sunny days but also on cloudy days (when ultraviolet rays can penetrate 70% to 80% of the cloud cover).
- Use sunscreen and protective clothing when you are on or near sand, snow, concrete or water (which can reflect more than 50% of the ultraviolet rays onto your skin).
- Avoid solariums as they emit ultraviolet radiation. Cancer Council Australia (2015) does not recommend solarium use for cosmetic tanning.

Nurses also provide person and family education for early detection of non-melanoma skin cancer. Numerous brochures describing the types of skin cancers, photographs of lesions and prevention behaviours are available from the Cancer Council Australia, health education and support agencies, and pharmaceutical companies that manufacture sunscreen. Most of this literature is free.

The person or family at risk of or diagnosed with a skin cancer must be taught how to conduct a regular self-examination of the skin, described in Box 15.7. The use of a mirror or assistance from family members can help with areas that are hard to examine, such as the ears, scalp and back.



THE PERSON WITH MALIGNANT MELANOMA

Malignant melanoma arises from melanocytes. Although melanoma is less common than NMSC, it is the most serious form of skin cancer and Australia and New Zealand have the highest incidence and mortality rates in the world (AIHW & AACR, 2012).

Incidence

In 2010, reported melanoma-related deaths were 11 545 in Australia. In the 15- to 24-years age group it is the most common cancer. There is a 1:24 for males, and 1:33 for females, risk of developing melanoma before the age of 75 years (Department of Health and Ageing, 2012). Melanoma is the third most common cancer after breast and bowel in women and the third most common in males after prostate and bowel (AIHW & AACR, 2010).

This disease is more than 10 times more common in fair-skinned people than in dark-skinned people. As with the non-melanoma skin cancers, an increase in the incidence of malignant melanoma is believed to be related to the thinning ozone layer and increased exposure to ultraviolet rays. The incidence is highest in Caucasian upper-middle-class professionals who work indoors. This group of people often had

BOX 15.6 Sunscreen information

Types of sunscreen

Chemical

Chemical sunscreens absorb ultraviolet light and act as a radiation filter. Examples follow:

- *p*-Aminobenzoic acid (PABA)
- Anthranilates
- Benzophenones
- Salicylates

Physical

Physical sunscreens reflect and scatter ultraviolet light. Examples follow:

- Zinc oxide
- Ferric chloride
- Titanium dioxide
- Kaolin
- Magnesium silicate
- Ichthyol

Adverse reactions associated with sunscreens

Adverse reactions associated with sunscreens include contact and photocontact dermatitis. People with previous hypersensitivity reactions to benzocaine, procaine, sulfonamides or paraphenylenediamine may develop hypersensitivity responses to PABA. People who are also taking systemic thiazide diuretics or sulfonamides may develop eczematous dermatitis.

Sunscreen ratings

Sunscreens need to be used in conjunction with other methods of sun protection, such as protective clothing and eliminating sun exposure times. Sun protection factor (SPF) ratings are awarded to sunscreen products subject to the amount of ultraviolet light they filter. In Australia, the ingredients in sunscreens are regulated by the Therapeutic Goods Administration (TGA, 2012). As well as preservatives, moisturisers, water, oils and emulsifiers, sunscreens contain agents that are described as either:

- chemical absorbers, which bind with the cells in the skin and absorb UV radiation and then release the energy as heat
- physical blockers, which reflect or scatter UV radiation (e.g. titanium dioxide and zinc oxide) (Cancer Council Western Australia, 2009).

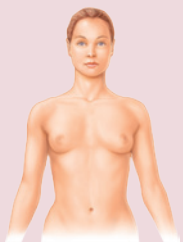
The SPF value is the ratio of the time required to produce minimal skin redness through a sunscreen product with the time required to produce the same degree of redness without the sunscreen. A person who can tolerate half an hour of sun without a sunscreen should be able to tolerate 3 hours of sun when a sunscreen of SPF 6 is applied to the skin. SPF values of sunscreens range from 2 to 50.

BOX 15.7 Skin self-examination

1. Choose the same day each month (such as the first day) to conduct a thorough skin examination.
2. The best time to do the examination is after you take a bath or shower.
3. Examine yourself in a well-lit room in front of a full-length mirror. Have a hand mirror, a chair and a hair dryer available. If you have difficulty seeing your back and scalp (or any other parts of your body), ask someone to help you.
4. Follow the same pattern with each examination:

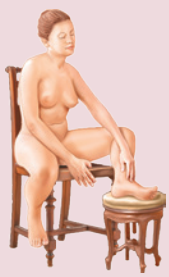
Examine head and face, using one or both mirrors. Use blow dryer to inspect scalp.

Check hands, including nails. In full-length mirror, examine elbows, arms, underarms.



Focus on neck, chest, torso. Women: Check under breasts.

With back to the mirror, use hand mirror to inspect back of neck and back, including buttocks.



Sitting down, check legs and feet, including soles, heels and nails. Use hand mirror to examine genitals.

severe sunburn with blistering during childhood and tend to holiday in areas of intense sun exposure. Malignant melanoma is also more common in people who live in sunny climates, burn easily and patronise tanning solariums. However, malignant melanoma may arise from already present lesions or from skin normally covered with clothing.

Risk factors

Although the exact cause of melanoma is unknown, it is known that certain risk factors are associated with the disease. The risk factors for melanoma are listed in Box 15.8.

Pathophysiology

Malignant melanomas arise from melanocytes, cells located at or near the basal layer (the deepest epidermal layer). These cells produce melanin, the dark skin pigment. Melanin is made in granules and transferred to keratinocytes, where it accumulates on the superficial side of each keratinocyte and forms a shield of pigment over the nucleus as protection against ultraviolet rays. Malignant melanomas can develop wherever there is pigment, but about one-third of them originate in existing naevi (moles).

Almost all malignant melanomas are more than 6 mm in diameter, are asymmetric and initially develop within the epidermis over a long period. While they are still confined to the epidermis, the lesions (called malignant melanoma in situ) are flat and relatively benign. However, when they penetrate the dermis, they mingle with blood and lymph vessels and are capable of metastasising. At this latter stage, the tumours develop a raised or nodular appearance and often have smaller nodules, called satellite lesions, around the periphery.

The prognosis for survival for people diagnosed with malignant melanoma is determined by several variables, including tumour thickness, ulceration, metastasis, site, age and gender. Younger people and women have a somewhat better chance of survival. Tumours on the hands, feet and scalp have a poorer prognosis; tumours of the feet and scalp are less visible and may not be diagnosed until they grow into the dermis.

Precursor lesions

The three specific precursor lesions for the development of malignant melanoma are congenital naevi, dysplastic naevi and lentigo maligna. A precursor lesion is also called a premalignant lesion, a name that indicates that the lesion's risk of becoming malignant is greater than normal.

BOX 15.8 Risk factors for melanoma skin cancer

- A high number of moles or large moles
- Fair skin, freckling, blonde hair or blue eyes
- Close relative with the disease
- Men with gene changes from a family history of breast or ovarian cancer
- Treatment with medications that suppress the immune system
- Too much exposure to UV radiation from sunlight, tanning lamps or solariums
- Over age 50
- Xeroderma pigmentosus, a rare inherited disease in which people are less able to repair damage caused by sunlight
- Past history of melanoma

CONGENITAL NAEVI Congenital naevi are present at birth. Some lesions are small; others are large enough to cover an entire body area. Their colour can range from brown to black. They are often slightly raised, with an irregular surface and a fairly regular border.

DYSPLASTIC NAEVI Dysplastic naevi are also called atypical moles. Although dysplastic naevi are not present at birth, they appear as normal naevi during childhood and become dysplastic (having abnormal development) after puberty. A person with classic dysplastic naevi has more than 100 naevi, at least one of which is larger than 8 mm in diameter, and at least one of which has the characteristics of malignant melanoma (asymmetry, irregular border, colour variegation and a diameter greater than 6 mm). A familial tendency to dysplastic naevi increases the risk of the development of malignant melanoma. However, it is not known whether people with dysplastic naevi and no family history of melanoma face a higher risk of melanoma.

Dysplastic naevi most often appear on the face, trunk and arms, but also are seen on the scalp, female breast, groin and buttocks. The pigmentation of the naevi is irregular, with mixtures of tan, brown, black, red and pink. An area of lighter pigmentation is surrounded by a papular area of deeper pigmentation (described as a 'fried egg appearance'). The borders of the naevi are irregular.

LENTIGO MALIGNA Lentigo maligna, also called Hutchinson's freckle, is a tan or black patch on the skin that looks like a freckle. It grows slowly, becoming mottled, dark, thick and nodular. It is usually seen on one side of the face of an older adult who has had a large amount of sun exposure.

Classification

Malignant melanomas are classified into different types. The major types are superficial spreading melanoma, lentigo maligna melanoma, nodular melanoma and acral lentiginous melanoma. Each of these tumours is characterised by a radial and/or vertical growth phase. During the initial radial phase, which may last from 1 to 25 years (depending on the type), the melanoma grows parallel to the skin surface. During this phase, the tumour rarely metastasises and is often curable by surgical excision. However, during the vertical growth phase, atypical melanocytes rapidly penetrate into the dermis and subcutaneous tissue, greatly increasing the risk of metastasis and death.

SUPERFICIAL SPREADING MELANOMA Superficial spreading melanoma is the most common type, comprising about 70% to 80% of all melanomas (Bullock & Hales, 2013). The lesions are usually flat and scaly or crusty and are about 2 cm in diameter. They often arise from a pre-existing naevus. This type of melanoma is found on the trunk and back of men and on the legs of women. Superficial spreading melanomas occur more often in women than in men. The median age of occurrence is the fifties.

The radial growth phase lasts from 1 to 5 or more years. When the lesion enters the vertical growth phase, it grows rapidly and its colour changes from a mixture of tan, brown and black to a characteristic red, white and blue. The lesion also develops irregular borders and often has raised nodules and ulcerations (see Figure 15.18).

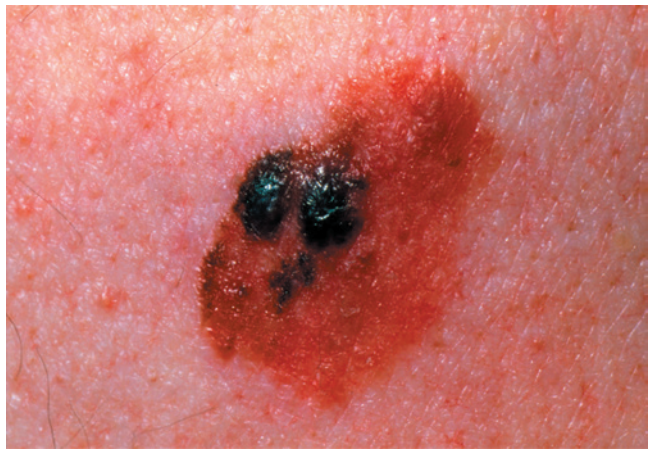


FIGURE 15.18 ■ Malignant melanoma is a serious skin cancer that arises from melanocytes

Source: Dr P. Marazzi/Science Source.

LENTIGO MALIGNA MELANOMA Lentigo maligna melanoma often arises from the precursor lesion, lentigo maligna. The lesions are large and tan with different shades of brown. This type of melanoma makes up 5% to 10% of malignant melanomas and is the least serious form (Bullock & Hales, 2013). It occurs on skin that has had long-term sun exposure, such as the face, neck and sometimes the dorsal surface of the hands and lower extremities. Lentigo maligna melanoma affects women more than men. It is typically diagnosed in people in their sixties and seventies.

Lentigo maligna melanoma is characterised by a proliferation of atypical melanocytes parallel to the basal layer of the epidermis. The radial growth phase may last from 10 to 25 years, with the lesion growing to as large as 10 cm. The lesion becomes malignant as soon as the melanocytes invade the dermis. In the vertical growth phase, raised nodules may appear on the surface of the lesion. The lesion tends to acquire a freckled or mottled appearance.

NODULAR MELANOMA Nodular melanoma lesions are raised, dome-shaped, blue-black or red nodules on areas of the head, neck and trunk that may or may not have been exposed to the sun. The lesions may look like a blood blister or they may ulcerate and bleed. The lesions arise from unaffected skin rather than from a pre-existing lesion. This type makes up 10% to 15% of malignant melanomas and is often diagnosed in people in their fifties (Bullock & Hales, 2013).

Nodular melanoma has only a vertical growth phase, but it grows aggressively during that phase. However, the absence of a radial growth phase makes this type more difficult to diagnose before it metastasises.

ACRAL LENTIGINOUS MELANOMA Acral lentiginous melanoma, also called mucocutaneous melanoma, is less common in people with fair skin and more common in people with dark skin.

The lesions progress from tan, brown or black flat lesions to elevated nodules and are about 3 cm in diameter. The radial phase lasts from 2 to 5 years. They are found on the palms of the hands, soles of the feet, the mucous membranes and the nail beds. Acral lentiginous melanoma affects both men and women equally and is most often diagnosed in people in their fifties and sixties.

INTERPROFESSIONAL CARE

The management of the person with malignant melanoma begins with identification, diagnosis and tumour staging. If treatable, the tumour is removed through surgical excision. Malignant melanoma is also treated with chemotherapy, immunotherapy and radiation therapy. Other therapies used with success include biological therapies with interleukin-2 and interferon, and therapeutic vaccines containing melanoma antigens.

Identification

Malignant melanoma can be found anywhere on the body and is most often found on the trunk of men and on the lower extremities of women. Nevertheless, it is important for the person to have a complete physical examination and total skin assessment. In addition to a visual examination of all skin surfaces, palpation of regional lymph nodes, the liver and the spleen is essential to assess for metastasis when a melanoma is suspected or found.

A change in the colour or size of a naevus is reported in 70% of people diagnosed with a malignant melanoma. The ABCD rule is used to assess suspicious lesions.

FAST FACTS

The ABCD rule

Using the ABCD rule to assess for melanoma:

- A = asymmetry (one half of the naevus does not match the other half)
- B = border irregularity (edges are ragged, blurred or notched)
- C = colour variation or dark black colour
- D = diameter greater than 6 mm (size of a pencil eraser)

Diagnosis

In addition to biopsy of any suspicious lesion, diagnostic tests are conducted to determine whether the tumour has metastasised. Because malignant melanoma may metastasise to any organ or tissue of the body, a variety of tests may be conducted, including microscopic examination, biopsy and tests for metastasis (liver function tests and computed tomography (CT) scan of the liver, a complete blood count, serum blood chemistry profile, chest x-ray, bone scan and CT scan, or magnetic resonance imaging of the brain).

Microstaging

The term *microstaging* describes the assessment of the level of invasion of a malignant melanoma and the maximum tumour thickness. In the Clark system of microstaging, the vertical growth of the lesion is measured from the epidermis to the subcutaneous tissue to determine the level of invasion (see Figure 15.19).

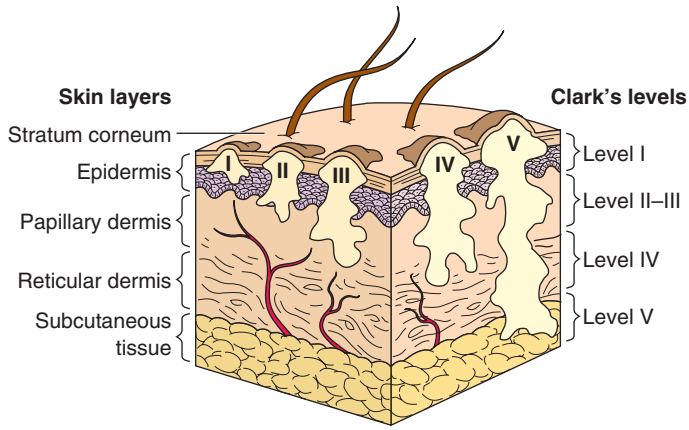


FIGURE 15.19 ■ Clark's levels for staging measure the invasion of a melanoma from the epidermis to the subcutaneous tissue

However, variations in individual skin thicknesses and different anatomical sites can affect the accuracy of the measurement. In the Breslow system, an adaptation of the Clark system of assessment, the vertical thickness is measured from the granular level of the epidermis to the deepest level of tumour invasion. This determination is important because as the thickness of the melanoma increases, survival rate decreases.

After the thickness and depth of the tumour are determined, a clinical stage is assigned. The traditional three-stage system is still used, although it does not include tumour thickness. The American Joint Committee on Cancer has adopted a four-stage system that includes tumour thickness, level of invasion, lymph node involvement and evidence of metastasis. This system is used in Australia.

Treatments

Surgical excision is the preferred treatment for malignant melanoma. Other methods of treatment are chemotherapy, immunotherapy and radiation therapy.

SURGERY If a biopsy identifies the lesion as a melanoma, a wide excision is performed that includes the full thickness of the skin and subcutaneous tissue. Because the risk of local recurrence for thin melanomas (those less than 0.76 mm) is quite low, margins of 0.5 to 1.0 cm of normal skin are excised around the tumour. Thick tumours require a 1 to 3 cm margin excision because they are at risk of local recurrence or satellite lesions.

Sentinel node biopsy is performed when a higher risk of primary melanoma has been diagnosed after an initial biopsy (melanoma 1.0 mm in depth, <40 years, Clark level ≥IV) (Keidan & Meyers, 2014). This will determine whether the melanoma has spread to the surrounding lymph nodes. This process starts with lymphatic mapping which will identify the first 'downstream' node—the sentinel node. This node is removed and sent to the pathologist to identify if malignant cells are present. Malignant cells are an indicator of the risk that melanoma may have spread to other parts of the body. Melanoma that has spread to sentinel nodes in the regional node groups in the armpits, neck or groin may linger before spreading to organs. In these cases melanoma

can sometimes be cured with surgery called regional lymph node clearance (Melanoma Institute of Australia, 2015). Regional lymph nodes are the most common sites for metastasis of malignant melanoma. Standard surgical treatment for clinically suspicious lymph node involvement includes excision of the primary lesions as well as surgical dissection of the involved lymph nodes. Elective lymph node dissection (ELND) in the treatment of localised malignant melanoma remains controversial. Advocates of ELND believe that the procedure benefits people with intermediate-thickness tumours because approximately 20% of people whose lymph nodes were clinically negative at diagnosis show some metastasis on removal of the nodes. Those opposed to ELND believe the risks associated with the procedure are too high for the 80% of people who have no evidence of metastasis after removal of the nodes.

Surgery may be indicated for palliative management of isolated metastasis. Removal of metastatic tumours in the brain, liver, lung, gastrointestinal tract or subcutaneous tissue may relieve symptoms and prolong life.

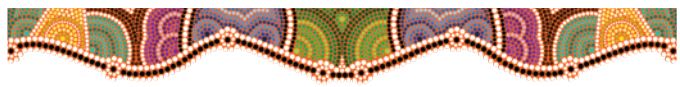
IMMUNOTHERAPY Immunotherapy is a relatively new treatment modality for malignant melanoma. The role of the immunological response initially was recognised because of the numerous spontaneous remissions seen in people with melanoma—a higher occurrence than with any other adult tumour. In addition, researchers have recently identified tumour-specific antigen antibodies in people with melanoma. This also has stimulated an interest in immunotherapeutic interventions for the treatment of malignant melanoma.

Agents such as interferons, interleukins, monoclonal antibodies, bacille Calmette-Guérin, levamisole, transfer factors and tumour vaccines have been used to treat melanoma, with varying response rates. The effectiveness of these agents, used either alone, in combination with chemotherapy or in combination with each other, is under investigation.

RADIATION THERAPY Melanoma responds to higher-dose radiation, especially if the tumour is small. Response rates to radiation therapy depend on the site of the tumour, its thickness, the type of melanoma and the person's general health, but may range from 0% to 71%. Radiation frequently is used for palliation of symptoms resulting from metastasis to the brain, bone, lymph nodes, gastrointestinal tract, skin or subcutaneous tissue. Liver and lung metastases are not treated with radiation therapy because a loss of organ function may result.

NEW METHODS OF TREATMENT Melanoma skin cancer research is ongoing and directed towards more specific methods of diagnosis and treatment. Examples are as follows:

- *Gene therapy:* clinical trials are in progress to test the effectiveness of adding certain genes to the malignant cells.
- *Melanoma DNA research:* knowledge of how ultraviolet light harms DNA is increasing, providing support for referral for genetic counselling for people with a strong family history of melanoma.
- *Targeted therapy:* this involves the development of drugs that attack the gene changes in melanoma cells. These drugs work differently from standard chemotherapy drugs. Sometimes targeted drugs work when chemotherapy does not and they can have less severe side effects. These drugs are new and we are still learning about the best way to use these drugs to treat melanoma (American Cancer Society 2015).
- *Immune therapy:* vaccines are being developed to make an individual immune to their own melanoma cells or to train the person's immune cells to fight the cancer.
- *Staging:* very sensitive new tests can better detect the spread of melanoma to lymph nodes and can possibly better identify people who could be helped by a treatment such as immunotherapy after surgery.



Nursing care

Nurses have the opportunity to assess the skin of people requiring care for many different health problems and may be the first healthcare provider to identify suspicious lesions. Wide excision and the high risk of metastasis from malignant melanoma usually require inpatient surgical treatment, with the nurse providing care and teaching.

Health promotion

The most important aspect of preventing malignant melanoma is identifying those at risk and performing regular skin assessments. Skin inspections should be performed monthly by those with actinic keratoses, at-risk individuals and those over 40 years of age. Mole clinics and summer beach skin inspection campaigns have become more popular and offer an alternative option for identification of sinister lesions. When self-assessing for melanoma, the person looks for a change in:

- colour, especially any lesion that becomes darker or variegated in shades of tan, brown, black, red, white or blue
- size, especially any lesion that becomes larger or spreads out
- shape, especially any lesion that protrudes more from the skin or begins to have an irregular outline
- appearance of a lesion, especially bleeding, drainage, oozing, ulceration, crusting, scaliness or development of a mushrooming outward growth
- consistency, especially any lesion that becomes softer or is more easily irritated
- skin around a lesion, such as redness, swelling or leaking of colour from a lesion into the surrounding skin
- sensation, such as itching or pain.

Assessment

Skin assessment is discussed in Chapter 14. Specific health history questions and assessments for skin cancer are outlined in Box 15.9.

Nursing diagnoses and interventions

Although many different nursing diagnoses may be appropriate for the person with a malignant melanoma, one potential diagnosis is as follows:

- *Impaired skin integrity* related to malignant melanoma.

BOX 15.9 Nursing assessment for skin cancer

Interview questions

- Have any members of your family ever been treated for skin cancer?
- Have you had a skin cancer removed from any part of your body?
- Have you noticed any change in the size, shape or colour of a mole, wart, birthmark or scar?
- Do you have any moles, warts, birthmarks or scars that itch, are painful, have crusting or bleed?
- In what parts of the country or world have you lived?
- Have you ever been badly sunburned?
- Do you visit tanning solariums?
- Are you exposed to any hazardous chemicals in your job?
- Have you been taught how to examine your skin? If so, how do you do this examination? How often?

Physical assessment

1. Ensure privacy and provide a warm environment. Ask the person to remove all clothing and put on an examination gown. Ensure good light; natural, bright light is best for inspection of lesions. The person may sit, stand or lie down.
2. Inspect and palpate the skin. Stretching the skin tightly during assessment facilitates assessment of nodular and scaly lesions and lesions in the dermis. Assess for:
   a. obvious lesions
   b. visible swellings
   c. alterations in normal contour and borders of naevi
   d. enlarged lymph glands
   e. skin or mucosal discolourations
   f. areas of ulceration, scaling, crusting or erosion.
3. The order of assessment follows:
   a. head and neck: entire scalp, eyelids, external ear, auditory canals, external surface of the nose, internal surface of the nose, the oral cavity, facial skin, the facial glands (parotid, submaxillary, sublingual)
   b. thyroid and neck, including lymph glands
   c. chest and abdomen, with special attention under pendulous breasts, in skin folds and in areas covered with hair
   d. back and buttocks, with special attention to the area between the buttocks
   e. extremities, with special attention to the axillae, nail beds, webs between the fingers and toes and soles of the feet
   f. external genitals, with special attention to skin folds, mucous membranes and areas covered with hair.
4. Measure and record a description of all skin lesions on an anatomical chart. Take photographs (if possible) of any suspicious lesion and include them in the person's record for future reference.

Impaired skin integrity related to malignant melanoma

Malignant melanomas not only destroy skin layers but also invade body structures. Certain types of melanomas may ulcerate prior to diagnosis, and treatment typically involves some type of surgical biopsy and excision. Any open lesion or incision increases the risk of secondary infection.

- Monitor for manifestations of infection: fever, tachycardia, malaise, erythema, swelling, pain or drainage that increases or becomes purulent. *Intact skin is the first line of defence against infection; impaired skin integrity increases the risk of infection.*
- Keep the incision line clean and dry by changing dressings as necessary.
- Follow principles of medical and surgical asepsis when caring for person's incision. Teach family members and visitors the importance of careful handwashing. Maintain standard precautions if drainage is present.
- Encourage and maintain adequate kilojoule and protein intake in the diet. Suggest a consultation with the dietitian if the person's appetite is poor. *Adequate kilojoules and protein are necessary for proper healing.*

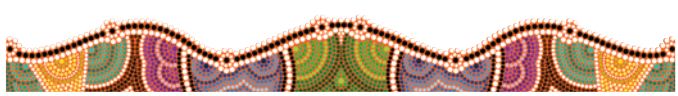
CONSIDERATION FOR PRACTICE

Nurses must use standard precautions with blood and body fluids to protect themselves from exposure to blood-borne viruses.

Community-based care

Health education for the person and family experiencing the diagnosis and treatment of non-melanoma and malignant melanoma involves self-care and ongoing self-monitoring. Education for the person and family is specific to the type of treatment. In addition to wound care for people who have had a surgical removal or lymph node dissection, they will need instructions on how to protect the extremity from bleeding, trauma and infection. People who have undergone lymph node dissection may develop lymphoedema and can benefit from manual lymph drainage therapy and compression therapy in the form of bandages and garments. Address the following topics:

- Schedule regular medical check ups every 3 months for the first 2 years, every 6 months for the next 5 years and yearly thereafter.
- Proper self-care combined with regular medical care can help the person lead a fairly normal life.
- If assistance for home care is necessary, provide referrals to a community health or home care organisation. In addition, refer the person to a local cancer support group if desired. Other resources are:
  - Cancer Council Australia: www.cancer.org.au
  - Melanoma Foundation Australia: www.sydney.edu.au/medicine/melanoma-foundation/
  - Melanoma Institute Australia: www.melanoma.org.au
  - Australasian Lymphology Association: www. lymphoedema.org.au.



SKIN TRAUMA

Trauma to the skin can be unintentional or intentional (as in the case of surgery). Pressure, friction and shear are common causes of skin trauma and can result in pressure injuries, while shear and friction can result in skin tears. Pressure injuries and skin tears are the most common wounds found in the elderly, and both types of wounds are largely preventable (Mulligan, Prentice & Scott, 2011). Thermal, chemical, electrical or radiation-induced burns are discussed in Chapter 16.

THE PERSON WITH A PRESSURE INJURY

Pressure injuries are synonymous with bed sores, decubitus ulcers and pressure ulcers, and are significant and preventable wounds. The term 'pressure injury' is the preferred term used in Australia (Australian Wound Management Association, 2012; Australian Commission on Safety and Quality in Health Care (ACSQHC), 2012). A pressure injury is 'a localised injury to the skin and/or underlying tissue. The injury is usually over a bony prominence and is caused as a result of pressure, or pressure in combination with shear and/or friction' (National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EUPAP) and Pan Pacific Pressure Injury Alliance (PPPIA), 2014; Australian Wound Management Association, 2012).

Pressure injuries are ischaemic lesions of the skin and underlying tissue caused by external pressure that impairs the flow of blood and lymph (Martini et al., 2012). The ischaemia causes tissue necrosis and eventual ulceration. Pressure injuries tend to develop over a bony prominence (such as the heels, greater trochanter, sacrum and ischia), but may appear on the skin of any part of the body subjected to external pressure, friction or shearing forces.

Incidence

The incidence of pressure injuries in hospitals, long-term care facilities and home settings is high enough to warrant the concern of healthcare providers. Pressure injury prevalence in the United Kingdom has been reported to range from 5.3% to 32% and in the United States from 1.4% to 36.4%. In Australia,

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 8:
Preventing and Managing Pressure Injuries

'The intention of this standard is to prevent patients from developing pressure injuries and effectively managing pressure injuries when they do occur.' (ACSQHC, 2012, p. 54).

Implementing this standard is achieved by the establishment of systems ensuring utilisation of best practice guidelines and risk assessment frameworks. This system includes implementation of processes which facilitate accurate identification and reporting, access to equipment and devices for prevention and treatment and best practice for treatment and monitoring. Meaningful communication regarding risk, prevention strategies and management should exist across all individuals involved in a person's care.

On admission, all patients, including children, should be screened for their risk of pressure injuries. Risk assessment, using a validated risk assessment scale, is conducted when screening indicates a patient is at risk of developing a pressure injury. Ongoing assessment and documentation should form part of the comprehensive nursing plan.

Source: © Australian Commission on Safety and Quality in Health Care.

TRANSLATION TO PRACTICE Pressure injury risk assessment and prevention

Nurses need to have an understanding of pressure injury risk assessment and preventive strategies. It is nurses who assess and manage patients' skin on a daily basis. Unfortunately knowledge deficits have been identified among Australian nurses (Lawrence, Fulbrook & Miles, 2015), as they have in other countries (Chianca et al., 2010; Gunningberg et al., 2015). To address this deficit, the *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline (2014),* which was produced by the European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Ulcer Advisory Panel (NPUAP) and the Pan Pacific Pressure Injury Alliance (PPPIA—Australia, Hong Kong, New Zealand and Singapore), includes dedicated chapters with recommendations for implementation of best practice guidelines; advancing health professional, consumer and caregiver education; and detailing quality indicators for monitoring guideline implementation and translation in the healthcare setting. This document can be accessed from www.internationalguideline.com.

pressure injury prevalence in public hospital surveys conducted in Victoria, Queensland, Western Australian, Tasmania and the Australian Capital Territory between 2003 and 2009 ranged from 10% to 26.5%. In national aged care facilities the prevalence has been reported to range from 15.2% to 26%, while in the community setting the reported prevalence ranges from 6% to 42%. However, survey methodologies differ between agencies and researchers and this makes for difficult data comparisons nationally. A standardised approach was adopted for prevalence surveys conducted by WoundsWest across all 86 Western Australian public hospitals in 2007, 2008, 2009 and 2011, and demonstrated 9.5% to 12.5% prevalence (Mulligan et al., 2011).

Pathophysiology

Pressure injuries develop from external pressure that compresses blood vessels or from friction and shearing forces that tear and injure vessels. The primary cause of pressure injuries is a sustained mechanical load that is applied to soft biological tissues, generally near a bony prominence. Pressure gradients that induce sustained deformation of skin and subdermal tissues must be present in order for tissue damage that characterises a pressure injury to occur. The magnitude of the mechanical load that will lead to tissue damage depends on the duration of time for which the pressure is applied. High pressure applied for a short period or low pressure applied for a longer period can lead to tissue damage. Recent evidence suggests that there are two physiologically events leading to tissue damage. One is a lower threshold leading to occlusion of blood vessels which results in ischaemic-induced tissue damage, and the other is a higher threshold leading to direct-pressure-induced damage.

An increasing body of evidence suggests that the microclimate between skin and the supporting surface plays a role in the development of Stage I and II pressure injuries. Microclimate refers to the humidity and temperature between the person's skin and the surface they are lying on (NPUAP, EPUAP & PPPIA, 2014). The increase in humidity and temperature can make the skin less tolerant to pressure and shear force. The greater the body surface area in contact with the support surface, the lower the uniform pressure. The lesser the body surface area in contact with a support surface, the higher the point pressure (Lachenbruch et al., 2013). This principle can be clearly appreciated when one rests the knuckle of the index finger on a table and compares the level of discomfort to that experienced when one rests the whole palm against the same surface. When the body is in the supine position, the body's weight applies pressure to the sacrum, heels, scapula and occiput. The same amount of pressure causes more damage when it is applied to a small area such as the heels than when it is distributed over a large surface.

External pressure that is greater than capillary closing pressure and arteriolar pressure interrupts blood flow in capillary beds. Capillary closing pressure is frequently quoted to be 32 mmHg, as determined more than 80 years ago by Landis (1930). However, capillary closing pressure will differ greatly between individuals and be subject to underlying co-morbidities or factors that impact on vascularity and perfusion, body mass index, nutrition and hydration status.

Shearing forces result when one tissue layer slides over another. The stretching and bending of blood vessels cause injury and thrombosis. People in bed are subject to shearing forces when the head of the bed is elevated and the torso slides down towards the foot of the bed. Pulling the person up in bed or against a chair also subjects the person to shearing forces (for this reason, repositioning devices such as hoists and slide sheets should be used). Friction, particularly in the presence of moisture, causes the skin and superficial fascia to remain fixed to the bed sheet or chair, while the deep fascia and bony skeleton slides in the direction of body movement.

When a person lies or sits in one position for an extended length of time without moving, pressure on the tissue between a bony prominence and the external surface of the body distorts capillaries and interferes with normal blood flow. The healthy, sensate and mobile individual will respond to the discomfort and alter their position. The immediate physiological response to relieved pressure is reactive hyperaemia. Reactive hyperaemia is a compensatory physiological response to tissue hypoxia and is evident when there is a transient increased blood flow or erythematous flush to the tissue. An area of reactive hyperaemia will blanch when pressure is applied (Pieper, 2007). However, if the pressure continues, the capillaries become more permeable and metabolic wastes and oedema accumulate in the interstitial spaces. Oedema in the interstitial spaces inhibits perfusion to the skin. Platelets aggregate in the endothelial cells surrounding the capillaries and form microthrombi. These microthrombi further impede blood flow, resulting in ischaemia and hypoxia of tissues. The skin may appear red; in darkly pigmented people, bluish or purple hues are evident. If the area of skin does not blanch when pressure is applied after relieving the pressure for 30 minutes, a Stage 1 pressure injury is present (see Box 15.10). Eventually, the cells and tissues of the immediate area of pressure and the surrounding area become necrotic. The 2014 *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline* outlines the pressure injury staging system most recommended internationally (NPUAP, EPUAP & PPPIA, 2014), and is found in the accompanying 'Translation to practice' box.

Risk factors

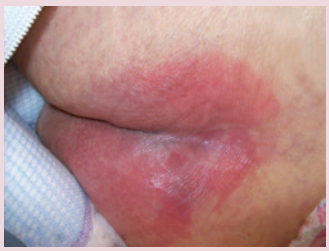
Predisposing factors for pressure injuries are both intrinsic (pertaining to the individual) and extrinsic (pertaining to extracorporeal factors). Intrinsic factors include extremes in age (the very young or elderly), immobility, inactivity, malnutrition, dehydration, poor skin condition, impaired sensory perception and co-morbidities that exacerbate these conditions, such as diabetes mellitus, malignancy, and renal, respiratory, vascular, lymphatic and hepatic disorders. Extrinsic factors include pressure, shear and friction forces, moisture (incontinence, wound exudate or perspiration) and contact with surfaces or devices such as plastic or vinyl which increase skin temperature.

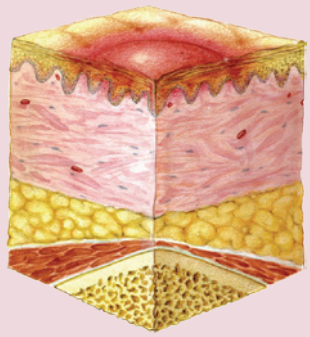
The older immobile adult is at increased risk of the development of pressure injuries because of age-related skin changes that reduce the skin's tolerance to pressure, shear and friction. The skin changes in the older adult include: a thinner dermis with decreased vascularity, decreased sebaceous gland activity,

BOX 15.10 The International NPUAP/EPUAP Pressure Ulcer Classification System

Stage 1: Non-blanchable erythema

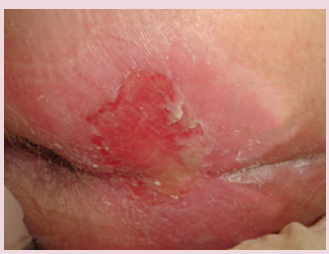
- Intact skin with non-blanchable redness of a localised area usually over a bony prominence.
- Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area.
- The area may be painful, firm, soft, warmer or cooler compared to adjacent tissue.
- May be difficult to detect in individuals with dark skin tones.
- May indicate 'at-risk' people (a heralding sign of risk).

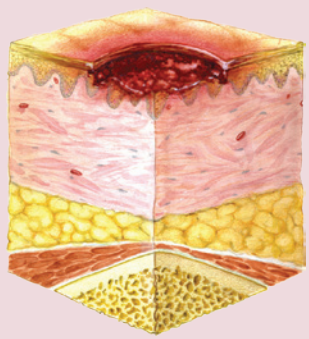




Stage 2: Partial-thickness skin loss

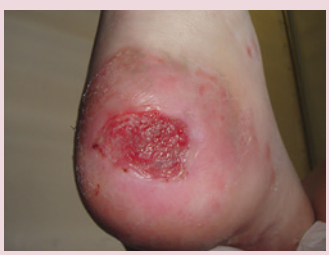
- Partial-thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed, without slough.
- May also present as an intact or open/ruptured serum-filled blister. Presents as a shiny or dry shallow ulcer without slough or bruising (bruising indicates suspected deep tissue injury).
- Stage 2 should not be used to describe skin tears, tape burns, perineal dermatitis, maceration or excoriation.

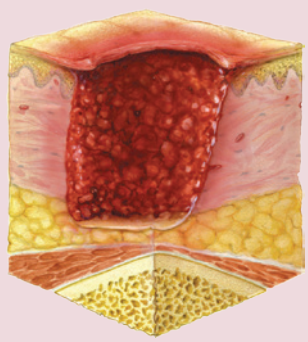




Stage 3: Full-thickness skin loss

- Full-thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunnelling.
- The depth of a Stage 3 pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and Stage 3 ulcers can be shallow. In contrast, areas of significant adiposity can develop extremely deep Stage 3 pressure injuries. Bone/tendon is not visible or directly palpable.

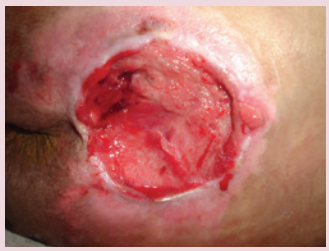


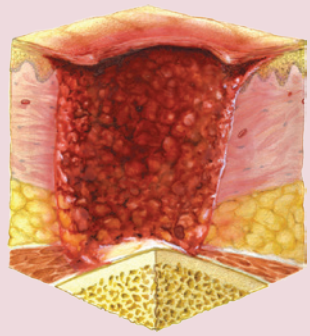


BOX 15.10 The International NPUAP/EPUAP Pressure Ulcer Classification System (continued)

Stage 4: Full-thickness tissue loss

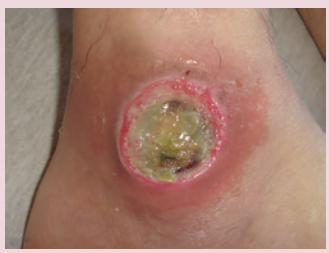
- Full-thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunnelling.
- The depth of a Stage 4 pressure injury varies by anatomical location. The bridge of the nose, ear, occiput and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Stage 4 injuries can extend into muscle and/or supporting structures (e.g. fascia, tendon or joint capsule) making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable.

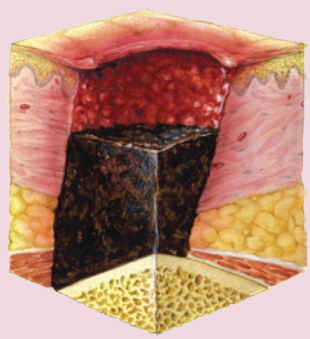




Unstageable: Depth unknown

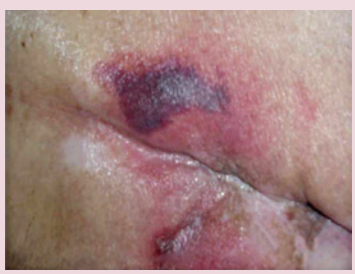
- Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, grey, green or brown) and/or eschar (tan, brown or black) in the pressure injury bed.
- Until enough slough and/or eschar is removed to expose the base of the wound, the stage cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as 'the body's natural (biological) cover' and should not be removed.

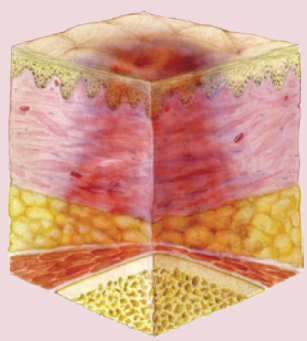




Suspected deep tissue injury: Depth unknown

- Purple or maroon localised area of discoloured intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue.
- Deep tissue injury may be difficult to detect in individuals with dark skin tones.
- Evolution may include a thin blister over a dark wound bed. The pressure injury may further evolve and become covered by thin eschar. Evolution may be rapid exposing additional layers of tissue even with optimal treatment.





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(continued)

TRANSLATION TO PRACTICE Pressure injury risk assessment

The WoundsWest wound prevalence surveys conducted in 2007, 2008, 2009 and 2011 found pressure injuries to be the second-largest group of wounds, following acute (surgical and traumatic) wounds. The majority of these pressure injuries were found to be hospital acquired (Mulligan et al., 2011). Furthermore, there were found to be significant deficits in the documentation of pressure injuries and the use of appropriate support surfaces.

The *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline* (NPUAP, EPUAP & PPPIA, 2014) outlines six recommendations for the risk assessment of pressure injuries. These recommendations are supported and upheld by the Australian Commission on Safety and Quality in Health Care's NSQHS Standards. These recommendations are:

1. All patients should undergo a pressure injury risk assessment within 8 hours of admission. The risk assessment should be structured and incorporate the clinical judgment of the clinician.
2. The risk assessment should be performed as often as clinically required.
3. Risk should be reassessed if the patient's condition changes.
4. A comprehensive skin assessment should form part of every risk assessment.
5. Each risk assessment should be documented accurately so that changes to skin integrity can be detected and treated early.
6. A risk-based prevention plan should be developed for patients identified as being at risk of pressure injuries.

In addition, the Guideline makes evidence-based recommendations for optimising nutritional status, repositioning and early mobilisation of individuals, and the use of support surfaces in the prevention and treatment of pressure injuries (NPUAP, EPUAP & PPPIA, 2014).

and decreased strength and elasticity. As a result, the thinner and less nourished dermal layer is more prone to shear and friction forces. Furthermore, wound healing is inhibited in the older adult due to a myriad of factors such as increased co-morbidities, potential deficits in nutrition and hydration, polypharmacy and the effects of certain medications.

Since the early 1960s an increasing number of pressure injury risk prediction instruments have been developed to assist healthcare providers identify and assess the recognised clinical variables that contribute to pressure injury formation. The most commonly used instruments in Australia include the Braden Scale (1987), Norton Scale (1962), Waterlow Score (1985) and Braden Q Scale (1996) for paediatric populations (Australian Wound Management Association, 2012). Among these prediction instruments there is no consensus on subscales assessed, scoring systems, methodological testing for reliability, sensitivity, specificity or predictive value rating, nor specific populations for use. However, when used in conjunction with informed clinical judgment they can increase the effectiveness of pressure prevention interventions.

INTERPROFESSIONAL CARE

For the person at risk of pressure injuries, the goal is prevention. An interprofessional and collaborative approach to the prevention and management of pressure injuries is required. The most contemporary evidence for prevention and treatment of pressure injuries which should be used by all health professionals is presented in the 2014 *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guidelines* available at www.internationalguideline.com.

Table 15.4 summarises contemporary pressure injury prevention recommendations described in the 2014 *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guidelines*.

Pressure injury management

Pressure injuries are generally considered to be chronic wounds, as healing may be complicated by intrinsic and extrinsic factors that impact on the person, their wound and their healing environment. The principles of wound bed preparation were outlined in the original 'TIME' acronym (Schultz et al., 2003) and updated in 2012 (Leaper et al., 2012) (see Box 15.11).

TIME: TISSUE Wound debridement is the required intervention when assessment reveals the presence of non-viable or necrotic tissue. Chronic wounds may require ongoing debridement to remove necrotic tissue and reduce the levels of microbes and excessive proteases.

The methods of debridement include surgical sharp, conservative sharp, autolytic, mechanical, chemical, enzymatic and parasitic larvae. Surgical sharp debridement may be performed by the medical practitioner in an aseptic environment when there is infection or extensive slough or eschar in the wound. Conservative sharp wound debridement (CSWD), which is the removal of loose avascular tissue without pain or bleeding, may be performed by the competent nurse when there are no contraindications such as:

- lack of access to sterile sharp instruments (Adson toothed forceps, scalpel or iris scissors)
- densely adherent necrotic tissue when the interface between viable and non-viable tissue cannot be clearly identified
- impaired clotting mechanism or the person is on anticoagulant or antiplatelet medications
- increased risk of bleeding—for example, malignant wounds
- a non-infected ischaemic ulcer covered with dry eschar when tissue oxygenation is insufficient to support infection control and wound healing—for example, a diabetic person with a dry or gangrenous foot ulcer (Carville, 2012).

TABLE 15.4 Pressure injury prevention

STRATEGY	DESCRIPTION
Skin care	• Avoid positioning patients on reddened areas of skin (erythema). • Keep the skin clean (using a pH-balanced skin cleanser) and dry. • Do not massage or rub skin at risk of pressure injuries. • Develop and implement an individualised continence plan. • Use a barrier method to protect skin from prolonged exposure to excessive moisture. • Use skin moisturisers judiciously to hydrate the skin (do not use dimethyl sulfoxides).
Microclimate control	• Use correct materials to alter the moisture absorption and heat dissipation depending on the client's need. • Do not apply heating devices directly on the skin or on a pressure injury.
Prophylactic dressings	• Apply polyurethane foam dressings to bony prominences on clients identified as being at high risk of pressure injuries. • Select the above dressings paying consideration to the microclimate needs, ease of application and removal, ability to easily and regularly assess skin, anatomical location and correct sizing.
Fabrics and textiles	• Use silk fabrics over cotton-based fabrics to reduce sheer and friction.
Electrical stimulation	• Consider using electrical stimulation to areas at risk of pressure injuries on spinal cord injury patients.
Nutrition	• Screen all patients at risk of pressure injury with a validated screening tool and refer those identified as at risk of malnutrition to a dietician. • Assess each patient's weight status, ability to eat independently and adequacy of nutrient intake. • Develop an individualised care plan. • Ensure the patient's energy intake has been calculated according to their underlying pathology, weight needs and pressure injury risk. • Provide adequate protein for clients identified as being at risk of pressure injuries. • Provide adequate hydration and monitor for signs of dehydration. • Encourage a vitamin-rich intake or supplementation in patients at risk of pressure injuries.
Repositioning and early mobilisation	• Reposition all patients at risk of pressure injuries, unless contraindicated. • Take into consideration the patient's pressure redistribution support surface when determining the frequency of turns. • Reposition patients so that pressure is relieved or redistributed, avoiding bony prominences and shearing forces.
Support surfaces	• Use high-specification reactive foam mattress for all patients at risk of pressure injuries. • Use an active support surface for all patients at *high* risk of pressure injuries.

BOX 15.11 TIME acronym

T = Tissue, non-viable or deficit
I = Infection or inflammation
M = Moisture imbalance
E = Edge of wound on-advancing or undermined.

The extent of debridement required and patient risk are outside the scope of practice.

Other methods of debridement such as autolytic, 'safe' chemical (using antimicrobial agents), mechanical or parasitic debridement, may be employed when CSWD is inappropriate. At the time of publication there are no licensed enzymatic debridement agents available in Australia.

Dressings that hydrate the wound or maintain wound exudate at the wound interface and thus promote autolysis of eschar are used to promote autolytic debridement (see Table 15.5). Certain modern antiseptic dressings provide a means of autolytic and less toxic chemical debridement and are commonly used when infection or the risk of infection is present (see Table 15.5). Mechanical debridement is the use of wet to dry dressings for the physical removal of necrotic or infected tissue or exudate, and this method may be preferred when delayed primary intention is employed.

Low frequency ultrasound debridement (LFUD) is another form of mechanical debridement. It uses low ultrasonic frequencies of 20 kHz to 100 kHz and converts electrical current to vibrations. As ultrasound does not travel easily through air, saline solution is used as a transducing media, which allows the ultrasound waves to travel from the probe into the tissues. The mechanical energy produced converts into acoustic energy, or a cavitation phenomenon, which is the creation and destruction of small bubbles within the saline fluid surrounding the probe (Shannon, Williams & Bloomer, 2012). During cavitation, the bubbles oscillate and expand and rapidly collapse, causing shockwaves and selective fragmentation or debridement of devitalised tissues (Conner-Kerr et al., 2010). An available example of an LFUD device is the SONOCA-185® (Soring).

The use of fly larvae therapy or maggots for debridement is attracting increased interest in Australia, and laboratory-raised 'sterile' *Lucilia sericata* (greenbottle fly) larvae are being produced at Westmead Hospital in Sydney for this purpose. Parasitic or larval debridement is also known as biosurgical or myasitic

TABLE 15.5 Dressings for autolytic and 'safe' chemical debridement

AUTOLYTIC DEBRIDEMENT	AUTOLYTIC AND CHEMICAL DEBRIDEMENT
Hydrogel dressings	**Cadexomer iodine dressings**
Amorphous hydrogels	Iodosorb™ powder, paste and dressing
Intrasite™	**Wound honey (combines both autolytic and chemical properties)**
SoloSite™	MediHoney™
DuoDerm gel™	Activon™
Purilon gel™	Hypertonic-impregnated dressings
Aquaform™	Curasalt™ gauze
Solugel™	Mesalt™
Gel sheet hydrogels	**Polyhexamethylene Biguanide (PHMB)**
Hydrosorb™	Prontosan™ solution (with Betaine)
Curagel	Prontosan™ gel
Nu-Gel™	AMD™ foam and gauze
Gel-impregnated gauze	
IntraSite Conformable™	
Hydrocolloid dressings	
Comfeel™	
DuoDerm™	
Hydrocoll™	
Nu-Derm™	

larval therapy and involves the deliberate infestation of laboratory-raised fly maggots into a necrotic wound. The *L. sericata* species is deemed to be the most suitable larvae as it secretes collagenases and trypsin enzymes and limits its interest to necrotic rather than healthy tissue (Polat et al., 2014). The enzymes facilitate liquidification of the necrotic tissue which is then digested by the larvae. Prior to application of the larvae, the periwound skin is best protected with a hydrocolloid dressing and the larvae entrapped under a semi-permeable film dressing which requires the insertion of small pin-pricked holes to facilitate air entry and survival of the maggots. Subject to the amount of necrotic tissue and the number of larvae, they are generally allowed to remain in the wound for 2 days or until they are engorged.

TIME: INFECTION OR INFLAMMATION The classic signs and symptoms of inflammation were noted by Celsus in the first century to be *tumor* (swelling), *rubor* (erythema), *calor* (heat) and *dolor* (pain) (Haeger, 1988). Inflammation can occur as a normal response to wound healing. It can also occur in response to wound infection with the added sign of purulence or increased malodorous exudate. Contamination, which is defined as the presence of non-replicating bacteria in a wound, does not inhibit wound healing (Bullock & Hales, 2013). However, tissue hypoxia or necrosis is conducive to colonisation, which is defined as the presence of replicating bacteria, but with no host reaction. Skin commensals such as *Staphylococcus epidermis* and *Corynebacterium* flora are to be expected in the wound and, in fact, have been found at low levels to demonstrate a positive effect on healing (Moffatt, 2004). Critical colonisation (also known as covert infection, local infection, occult infection) is defined as an increase in the bacterial burden of the wound in which healing is inhibited (World Union of Wound Healing Societies, 2008). Critically colonised wounds do not portray the classic signs of infection, although the wound may demonstrate one or more of the following signs: static healing; increased exudate; hypergranulated, bright-red, friable granulation tissue that bleeds easily; tissue bridging; granulation pocketing in the base of the wound; and rolled edges (Swanson, Grothier & Schultz 2014).

There is a lack of international consensus as to whether critical colonisation is a transitional stage between colonisation and overt infection, or is indicative of chronic inflammation in the presence of a biofilm in the wound. Bacteria react to threat in two ways: the development of resistance and the formation of biofilms. **Biofilms** are polymicrobial microbial communities which proliferate and are encased in a protective glycocalax matrix. The bacteria secrete glycocalyx which forms a biofilm (an extracellular polysaccharide (ESP)) that protects the organisms from immune responses by phagocytes and from topical and systemic antibiotics. Biofilms have been found in 60% of chronic wounds compared to 6% of acute wounds (James et al., 2008).

Biofilms form in the following manner:

1. Planktonic (free-floating) organisms attach to the surface of the wound.
2. Sessile (firmly attached) organisms communicate between microorganisms of the same or different species via a process referred to as quorum sensing. Quorum sensing is responsible for phenotypic diversity and some of the genotypic diversity seen in wound biofilms, and enhances the community's nutrient-gathering capacity, defence and reproductive abilities.
3. Firmly attached microorganisms secrete an extracellular polymeric substance (EPS) or protective matrix.
4. The EPS is comprised of polysaccharides, proteins, glycolipids and bacterial DNA.
5. The mature biofilm releases planktonic bacteria which disperse and attach to other parts of the wound, and the cycle is repeated (James et al., 2008; Phillips et al., 2010).

The prudent use of topical antiseptic dressings can restore the bacterial balance in the wound and there is some evidence that polyhexamethylene biguanide (PHMB) with a surfactant (betaine) (Andriessen et al., 2008; Forstner et al., 2013; Kaehn & Eberlein, 2009), cadexomer iodine (Phillips et al., 2010) and silver (Incani et al., 2015) can denature a biofilm. Because bacterial imbalance usually results in increased amounts of wound exudate, maintenance of moisture balance is an aligned goal (Carville, 2012). Examples of antiseptic dressings available for restoration of bacterial balance are outlined in Box 15.12.

TIME: MOISTURE IMBALANCE Desiccation of the wound inhibits epithelialisation, and excessive moisture leads to maceration and further breakdown of the tissues. Chronic wound fluid contains increased levels of matrix metalloproteases which have the potential to degrade much-needed extracellular matrix proteins such as fibronectin and vitronectin. Excessive fluid in chronic wounds can interfere with the activities of important cell mediators, such as growth factors (Dowsett, 2011). The goal is to maintain moisture balance while avoiding desiccation and maceration. Thus, dressing selection will be

BOX 15.12 Antiseptic dressings

Cadexomer iodine dressings
Iodosorb™ paste, powder and dressing
Povidone iodine impregnated tulle gras
Inadine™
Chlorhexidine impregnated tulle gras
Bactigras™
Wound honey
MediHoney™
Activon™
Silver-impregnated dressings
Acticoat™ 3 and 7 day
Acticoat Absorbent™
Aquacel Ag™
Contreet Foam™
Atrauman AG™
Silvercel™
Hypertonic-impregnated dressings
Curasalt™ gauze
Mesalt™
Polyhexamethylene biguanide (PHMB)
Prontosan™ solution (with Betaine)
Prontosan™ gel
AMD™ foam and gauze

influenced by the assessment outcome, and the goal of care will be either to hydrate the wound bed or absorb excessive exudate. Cavity or sinus wounds such as Stage 3 and 4 pressure injuries will require a cavity-filling dressing to eliminate the dead space in the wound and facilitate controlled wound closure. Most generic groups of dressings (e.g. calcium alginates, hydrofibre, foams and hydrogel-impregnated gauzes) now come in forms suitable for filling cavity defects.

Dressing options for maintaining moisture balance in pressure injuries are outlined in Figure 15.20.

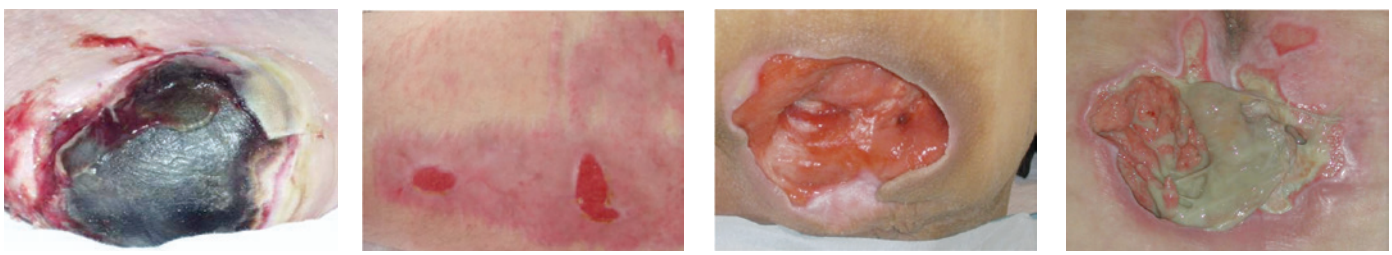
TIME: EDGES ADVANCING Assessment of wound edges is frequently overlooked, but coloured, raised, rolled or undermined wound edges can indicate delays in healing, abnormal pathologies or unrelieved pressure. Desiccation of the wound bed, hypergranulation and periwound debris (scale, scab or dried exudate) will inhibit epithelisation across the wound surface. Hypergranulation commonly results from bacterial imbalance or ongoing wound irritant trauma. Rolled or undermined edges can also indicate bacterial imbalance. Raised edges may indicate unrelieved trauma. However, further diagnostic investigations such as a wound biopsy may be indicated if the wound edge appearance indicates potential malignant changes. The use of negative pressure wound therapy (NPWT) for advancing wound closure is now a common intervention.

Surgical treatment

Surgical debridement may be performed by a medical practitioner when there is infection or extensive slough or eschar. Extensive pressure injuries may also require skin grafting or flap reconstruction to facilitate complete closure.

Nursing care

The person with a pressure injury not only has impaired skin integrity but also is at increased risk of other pressure injuries, infection, pain, decreased mobility and death. Pressure injuries prolong treatment for other health problems, increase healthcare costs and diminish the person's quality of life. Therefore, pressure injury prevention is the optimal principle of care (see Box 15.13).



Dry
Dressing options:
- Hydrogels
- Hydrocolloids
- Interactive wet dressings

Low exudate
Dressing options:
- Semi-permeable films
- Hydrocolloids
- Calcium alginates

Moderate exudate
Dressing options:
- Calcium alginate
- Hydrofibre
- Foams

Heavy exudate
Dressing options:
- Hydrofibre dressing
- Foam sheet/cavity
- Super-absorbent dry dressings
- Wound/ostomy bag
- Topical negative-pressure therapy systems

FIGURE 15.20 ■ Dressings for maintaining moisture balance in pressure injuries

Source: Wound care manual (6th ed.) by K. Carville (2012). Osborne Park, WA: Silver Chain Foundation. Images reproduced with permission K. Carville.

BOX 15.13 Nursing care of the person at risk of a pressure injury and the person with a pressure injury

Comprehensive recommendations for pressure injury prevention and management are to be found in the *Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline* (NPUAP, EPUAP & PPPIA, 2014) at www.internationalguideline.com.

The principle interventions for pressure injury prevention and management are as follows:

Risk assessment

1. Conduct a comprehensive assessment for all people to identify pressure injury risk factors. A comprehensive assessment should include:
   - clinical history
   - pressure injury risk scale
   - skin assessment
   - mobility and activity assessment
   - nutritional assessment
   - continence assessment
   - cognitive assessment
   - assessment of extrinsic risk factors.
2. Use a validated pressure injury risk assessment scale in conjunction with a comprehensive risk assessment to determine the person's risk of pressure injury and to inform the development of a prevention plan.
3. Inspect the skin of all people on admission and at each repositioning to identify indications of pressure injury including:
   - erythema
   - blanching response
   - localised heat
   - oedema
   - induration
   - skin breakdown.

Prevention of pressure injuries

1. Implement preventative strategies to protect the person's skin.
2. Provide high-protein oral nutritional supplements in addition to an appropriate diet for people at a high risk of pressure injury who have been identified at risk of having malnutrition.
3. Use a high-specification, constant-low-pressure support mattress on beds, trolleys, operating theatre tables and chairs for people at high risk of pressure injury (refer to the Guideline for information on characteristics of high-specification support surfaces).
4. Any device used to prevent heel pressure injuries should be selected and fitted appropriately to offload heel pressure.
5. Frequency of repositioning should consider the person's risk of pressure injury, comfort, functional level, medical condition and the support surface used.
6. Skin protection should include:
   - Individuals at risk of developing pressure injuries should have a comprehensive skin inspection at least daily for signs of impaired skin integrity.
   - The skin should be kept clean and free from all potentially irritating substances or those that substantially alter the skin pH.
   - All intrinsic and extrinsic factors that result in dryness or maceration of the skin should be eliminated or minimised by:
     a. treating dry, flaky or scaling skin with a topical moisturiser
     b. avoiding sustained or excessive contact with body fluids, and/or
     c. encouraging continence by employing interventions such as continence training or the use of continence aids.
7. Avoid extremes in skin temperature by avoiding skin contact with plastic support surfaces and ensuring that turning schedules do not exceed 2-hourly intervals for people at high risk.

Mechanical offloading and support surfaces

1. Alternating-pressure mattress replacements and overlays provide similar benefits to high-specification, constant-low-pressure support mattresses and could be used as an alternative in people at high risk of pressure injury.
2. Support surfaces should be used in conjunction with a comprehensive prevention strategy based on frequent observation and assessment, individualised turning regimens and measures to increase the tissues' tolerance to pressure.
3. Pillows and foam wedges can be used to avoid direct contact between bony prominences.
4. Avoid prolonged uninterrupted sitting in a chair or wheelchair. Repositioning or shifting of pressure points should occur as frequently as every 15 minutes to hourly depending on the tissues' tolerance to pressure.
5. Exposure to shear and friction should be reduced by:
   a. employing correct lifting and manual handling techniques
   b. protecting skin constantly exposed to friction with protective dressings or padding or medical-grade sheepskin
   c. elevating the foot of the bed to 20 degrees when sitting to prevent sliding
   d. maintaining the head of the bed at the lowest possible elevation consistent with the individual's medical condition and comfort.
6. Individuals who are bed-bound or have immobilised lower extremities should have total relief of pressure from their heels.
7. Individuals should be encouraged to maximise their activity and mobilisation consistent with their medical condition, ability and energy level.

Documentation

All individuals at risk of developing pressure injuries should have the following details recorded in the person's record on a regular, ongoing basis: risk assessment status (low, moderate

BOX 15.13 Nursing care of the person at risk of a pressure injury and the person with a pressure injury (continued)

or high); identified risk factors; management plan which includes interventions used such as turning schedules, support surface, referrals and the individual's response to treatment.

Evaluation

1. Any pressure injury risk management program should be based on a demonstrable need, relevant to the healthcare setting and supported by a policy and protocol based on the best available research.
2. All pressure injury risk management programs should include the individual at risk and significant family members. The individual should be considered an active participant in the management plan and should be informed of the relevant risk factors and the strategies employed to minimise or eliminate the risk of pressure injury development.
3. A pressure injury risk management program should be supported by a continuing educational program and a multidisciplinary continuous improvement process that is able to monitor and compare the impact of interventions over time.

Source: (NPUAP, EPUAP & PPPIA, 2014).

NURSING CARE PLAN A person with a pressure injury

Mrs Sibutu is 85 years old and lives in a nursing home. Mrs Sibitu has been unable to walk since suffering a dense cerebral vascular accident (CVA) 2 years ago. She requires full assistance to transfer and change her position in bed. She has a history of type II diabetes which is reported to be diet controlled, chronic obstructive pulmonary disease (COPD) and osteoarthritis. Her medications include preventative inhaled corticosteroids (beclomethasone) twice daily and non-steroidal anti-inflammatory agents (naproxen 500 mg) twice daily. In addition, she takes aspirin (Cardiprin) 100 mg daily as a preventive anticoagulant. Her weight is average, but she has had a recent weight loss of 4 kg. While her appetite was previously good, over the past month she has required a lot of assistance and encouragement with fluid and foods. A deterioration in her general condition has been noted and she is reportedly increasingly drowsy and reluctant to be repositioned regularly. She is incontinent of faeces and urine. Her incontinence is managed with incontinent pads. She normally sits out of bed for 4 to 6 hours a day in a recliner chair but otherwise spends the remainder of the day in bed.

ASSESSMENT

On routine skin inspection Mrs Sibitu was assessed to have a pressure injury measuring 3 cm by 4 cm over her sacrum. The wound bed was covered in a layer of yellow slough that inhibited assessment of the depth of the wound. There was a moderate amount of yellow-stained, slightly malodorous exudate. On palpation the surrounding skin is noted to be indurated and dark red. The edges of the wound are level with the surrounding skin, which is macerated due to contact with wound exudate and urine. Although she cannot articulate her pain level, Mrs Sibitu appears restless when the wound is touched or she is repositioned. A pressure injury risk assessment using the Braden Scale was performed and she was assessed as being at high risk of pressure injury.

DIAGNOSIS

- *Impaired skin integrity* related to pressure, friction and shear—an Unstageable sacral pressure injury.
- *Infection* risk due to contamination of pressure injury with faeces and urine.
- *Pain* related to the pressure injury.
- *Anxiety* related to alterations in cognition, mobility and activity.

PLANNING

- Assess the person, their wound and their healing environment.
- Eliminate pressure, shear and friction.
- Control wound infection and manage the wound according to best evidence.
- Debride the slough.
- Manage urinary and faecal incontinence.
- Optimise nutritional status with supplements.
- Manage pain.

Expected outcomes

- Mrs Sibitu's pressure injury will be assessed daily, managed and progress to healing.
- Mrs Sibitu will be free of further pressure injuries or extension of her existing injury with appropriate assessment and intervention.
- Mrs Sibitu will be free of wound infection from faecal and urine contamination based on appropriate infection prevention and control strategies.
- Mrs Sibitu will be free of pain based on accurate assessment and appropriate interventions.

IMPLEMENTATION

- Assess pain and request a medical review for pain management medications.
- Perform a skin assessment and a pressure injury risk assessment on admission and on altered change of condition, or as directed by her risk score and the organisation's protocol.
- Conduct a skin inspection daily and document outcomes.
- Increase repositioning times and use pillows or wedges to secure position and elevate heels from bed and position off sacrum.

(continued)

NURSING CARE PLAN A person with a pressure injury (continued)

- Optimise pressure offloading—utilise an active alternating-pressure replacement mattress on her bed for sleeping.
- Optimise pressure offloading—utilise a constant-low-pressure cushion when seated. The occupational therapist will be requested to review Mrs Sibitu's seating.
- Request a dietitian to review Mrs Sibitu's nutritional intake and determine a plan of care for regular nutritional supplements and increased oral fluids.
- Keep Mrs Sibitu's skin clean and dry and use protective moisturisers and barrier films to protect against maceration.
- Review incontinence management strategies and quality of incontinence pads used. Implement a regular toileting regimen and review her bowel management plan.
- Request the physiotherapist to review her activity and mobility and implement a program of passive and active exercise as appropriate.
- Document wound assessment outcomes including stage of pressure injury, wound bed appearance and dimensions, type and amount of exudate, presence of malodour, status of surrounding skin and wound edges, and pain score, using a validated instrument.
- Document an individualised care plan for pressure injury management with the aim of reducing pain and discomfort, debridement of the slough, the need to maintain a bacterial balance in the wound, promote wound healing and prevent further pressure injury development.
- Observe for clinical signs of overt infection (increased pain, heat, redness, swelling or purulence, elevated temperature, malaise) and report to the medical practitioner.

Evaluation

Mrs Sibitu will require daily skin inspections and regular ongoing risk assessments for pressure injury risk. Ongoing wound assessments will be evaluated for healing progress and successful care planning. Her pain will be controlled. The occupational therapist will review Mrs Sibitu's seating on a regular basis and adapt it as appropriate. The alternating-pressure mattress will be checked each shift to ensure the correct pressure offloading is provided under her bony prominences. The dietitian will review the effectiveness of the nutritional plan. The physiotherapist will review the effectiveness of the exercise plan.

CRITICAL THINKING IN THE NURSING PROCESS

1. Consider Mrs Sibitu's risk factors for pressure injury development and why daily skin inspection of an 'at-risk' individual is of the utmost importance.
2. Use a validated risk assessment tool such as the Braden Scale, Norton Score or Waterlow Score to determine Mrs Sibitu's risk score.
3. What primary dressing would you most likely select to manage Mrs Sibitu's wound if your goal of care is bacterial balance and debridement?
4. What secondary dressing would you most likely select to control the moderate amount of exudate and protect against further contamination by body waste?

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from Mrs Sibitu's situation that you will apply to your future practice.
2. What education strategies would you give to Mrs Sibitu and her family to prevent her developing further pressure injuries?

THE PERSON WITH A SKIN TEAR

A **skin tear** is defined as 'a traumatic wound occurring principally on the extremities of older adults, as a result of friction alone or shearing and friction forces which separate the epidermis from the dermis (partial-thickness wound) or which separate both the epidermis and the dermis from underlying structures (full-thickness wound)' (Payne & Martin, 1993).

Skin tears are the most common wound found in elderly populations. Reasons for this are related to the changes that occur in ageing skin and the increased incidence of knocks, falls and manual handling requirements (Carville, 2012). The risk factors associated with acquiring skin tears are poorly studied but anecdotally reported to be visual impairment, impaired mobility or balance, dementia and the use of certain medications such as steroids or anticoagulants (Crisp et al., 2013). Skin tears are all too frequently considered to be minor wounds, yet they can become extremely problematic and their treatment can be costly.

Until recently, skin tear prevalence was poorly reported in Australia. However, skin tears were found to be the third-largest group of wounds in Western Australia during surveys of all public hospitals conducted in 2007, 2008, 2009 and 2011 (Mulligan et al., 2011). Of particular concern was the fact that the majority of these injuries were hospital-acquired. In a Western Australian community setting, skin tears were found to comprise 20% of wounds in a population aged 70 years or older (Carville & Smith, 2004). Although dated data, in that same state, skin tears were found to be 41.5% of known wounds in an aged care agency (Everett & Powell, 1994).

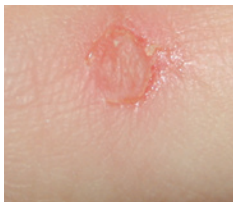
In 1990, Payne and Martin outlined a system for classification of skin tears. They refined this system in 1993, although the classification is poorly utilised in Australia. The Payne–Martin Skin Tear Classification (1993) lists three categories and two subcategories of injury; however, it does not take into account the presence of haematoma or ischaemic changes in the skin or flap (see Box 15.14).

BOX 15.14 Payne–Martin Skin Tear Classification (1993)

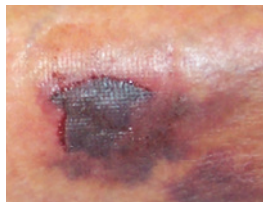
Category 1: Able to approximate the wound borders within 1 mm, no tissue loss
A—linear tear
B—flap

Category 2: Varying amounts of tissue loss
Type A <25% loss—scant
Type B <25% loss—moderate

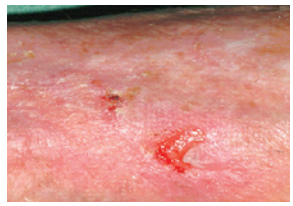
Category 3: Total loss of flap



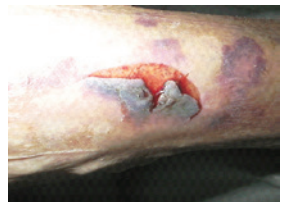
Category 1a
A skin tear where the edges can be realigned to the normal anatomical position (without undue stretching) and the skin or flap colour is not pale, dusky or darkened.



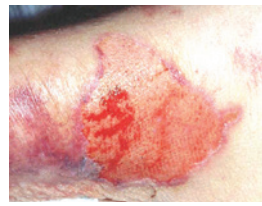
Category 1b
A skin tear where the edges can be realigned to the normal anatomical position (without undue stretching) and the skin or flap colour is pale, dusky or darkened.



Category 2a
A skin tear where the edges cannot be realigned to the normal anatomical position and the skin or flap colour is not pale, dusky or darkened.



Category 2b
A skin tear where the edges cannot be realigned to the normal anatomical position and the skin or flap colour is pale, dusky or darkened.



Category 3
A skin tear where the skin flap is completely absent.

FIGURE 15.21 ■ STAR Skin Tear Classification System

Source: Skin Tear Audit Research (2007). Silver Chain Foundation and Curtin University. Reprinted with permission.

The STAR Skin Tear Classification System (Carville et al., 2007) also lists three categories and two subcategories of skin injury and takes into account skin or flap colour changes that occur with haematoma or hypoxia (see Figure 15.21).

Preventing skin tears

At this time there is no validated risk assessment tool for predicting skin tears. However, individuals who require personal assistance with transfer or mobility, such as the frail, elderly or disabled, are at increased risk. Skin tears can also occur on any individual with the removal of adhesive agents such as tapes, dressings and devices, and due care must be taken during these procedures. A large randomised control trial found that twice-daily moisturising of extremities of aged care residents with a pH-friendly, perfume-free moisturiser reduced skin tear incidence by 50% (Carville et al., 2014). Prevention of skin tears is a fundamental aspect of caring for these populations (see Box 15.15).

BOX 15.15 Skin tear prevention

- Assess for falls risk and implement prevention strategies.
- Use gentle and timely handling on transfer and repositioning.
- Use devices that reduce shear and friction.
- Moisturise the skin twice a day with a pH-friendly, perfume-free moisturiser.
- Cover vulnerable skin surfaces with protective clothing or devices.
- Maintain position with pillows and foam wedges to prevent shear. (Satin or silk covers will further reduce shear forces.)
- Avoid perfumed soaps that dry and alter the skin's pH.
- Cease smoking.
- Maintain adequate hydration and optimal nutrition.
- Avoid adhesive tapes and dressings on fragile skin in favour of roller or tubular bandages.
- Review medications and eliminate if possible those that alter the skin's integrity.
- Control any co-morbidities that alter the skin's condition or risk of injury.
- Provide person and carer education on skin health and injury prevention.

Source: Wound care manual (6th ed.) by K. Carville (2012). Osborne Park, WA: Silver Chain Foundation.

Skin tear management

The principles of skin tear management focus on control of any bleeding, restoration of skin flaps if present and provision of an environment that is conducive to healing and protection from further injury. Skin tears are commonly managed by dressings alone; stabilisation of the skin flap can be achieved via the use of silicone dressings (Prentice, Morey & Rodriguez, 2014). If the skin tear is deep or extensive, primary closure using sutures or tissue adhesives such as Histacryl™ may be required. The use of skin closure strips such as Steri-Strips™ are not recommended across flexures as they can compromise perfusion on flexing of tissues. If tape is required, the use of skin-protective barrier film prior to application and an adhesive remover prior to removal is advised (Prentice et al., 2014).

Selection of an appropriate dressing is subject to the ideal features and benefits, which should include the following:

- easy to apply
- provides a protective anti-shear barrier
- tissue friendly, moulds to contours, flexible
- maintains wound physiological balance—moisture, temperature, pH
- secure, but not aggressive retention
- extended wear time
- non-traumatic removal
- optimises quality of life and cosmesis
- cost-effective.

THE PERSON WITH A DISORDER OF THE HAIR

Racial characteristics and gender influence the amount and type of hair. Caucasians typically have more facial and body hair than do Asians. The latter usually have straight hair, those of African descent have wavy to curly hair and Caucasians have straight to curly hair. In addition, male hair growth characteristics (such as facial hair and hair on the lower extremities) are normal in certain women of some races and families.

The hair grows at various rates. Facial hair grows the most rapidly, followed by the hair of the scalp, axillae, thighs and eyebrows. Normally, an adult's hair grows at a rate of 10 to 12 mm per month; however, the growth rate is influenced by both the person's state of health and the environment. (Hair grows faster in hot climates, more slowly in cold climates.)

Pathophysiology

Hair colour, growth and pattern vary from person to person and are determined largely by genetic inheritance. However, changes do occur. For example, in some instances, hair loss recurs in successive generations of males in a family; in other cases, hair loss may be the result of chemotherapy. Excessive facial hair may be a response to certain endocrine disorders or to the loss of oestrogen after menopause. These changes may seem minor, but they may create psychosocial problems for the person experiencing the changes.

Hirsutism

Hirsutism, also called hypertrichosis, is the appearance of excessive hair in normal and abnormal areas of the body in women. Hirsutism most often occurs in a male distribution (that is, on the upper lip, chin, abdomen and chest) in women. The excess hair is primarily the result of an increase in androgen levels (especially testosterone), which may be due to any of the following:

- familial predisposition (considered normal)
- polycystic ovary syndrome
- ovarian, adrenal or pituitary tumours
- Cushing's syndrome
- central nervous system disorders
- medications, such as minoxidil, cyclosporin, phenytoin, certain progestins and anabolic steroids.

The manifestations of hirsutism include increased male pattern hair growth, acne and menstrual irregularities. If the androgen excess is great, defeminisation (a decrease in breast size and loss of normal adipose tissue) and virilisation (frontal balding, increased muscle mass, deepening of the voice and enlargement of the clitoris) may occur. Virilisation indicates the presence of an androgen-producing tumour.

Alopecia

Alopecia is loss of hair or baldness (see Figure 15.22). Alopecia can affect both men and women and may result from scarring, various systemic diseases or genetic predisposition. Scarring from trauma, radiation and severe bacterial, fungal or viral infections causes permanent and irreversible hair loss over the scarred area. Systemic diseases that may cause alopecia include systemic lupus erythematosus, thyroid disorders and pituitary insufficiency. The hair loss from these disorders may be reversible. Hair loss from androgenic causes may also occur in postmenopausal woman. Alopecia may be drug induced and is a side effect of a variety of medications (see Box 15.16).

FIGURE 15.22 ■ Alopecia (baldness) may be the result of scarring, disease or genetic predisposition

Source: © Fresnel/Shutterstock.com.

Types of alopecia follow:

- Male pattern baldness is the most common cause of alopecia in men and is genetically predetermined. The hair loss begins at the temples, with recession of the hairline and baldness of the crown.
- Female pattern alopecia begins in women in their twenties and thirties, with progressive thinning and loss of hair over the central part of the scalp. Unlike men, women do not lose hair from the frontal hairline. Many of these women have elevated adrenal androgens.
- Alopecia areata is characterised by round or oval bald patches on the scalp, as well as on other hairy parts of the body. The cause is unknown. This type of alopecia is usually self-limiting and reverses without treatment, although it often recurs.

BOX 15.16 Medications causing alopecia

- Thallium
- Retinoids
- Anticoagulants
- Antimitotic agents
- Antithyroid drugs
- Oral contraceptives
- Trimethadione
- Excessive use of vitamin A
- Allopurinol
- Propranolol
- Indomethacin
- Amphetamines
- Salicylates
- Levodopa
- Gentamicin
- Chemotherapy

- Alopecia totalis is the loss of all hair on the scalp. This rare condition is irreversible.
- Alopecia universalis is the total loss of hair on all parts of the body.

INTERPROFESSIONAL CARE

Alopecia is diagnosed by assessing the appearance of the hair and hair loss, and by assessing the person for other systemic diseases and the use of medications that may cause hair loss. Various treatments are used to restore hair.

The person with hirsutism is examined for hormone levels and indications of other systemic illnesses. Hirsutism is treated by addressing the underlying systemic disorder and stopping medications that may be causing the problem.

Diagnosis

Diagnostic tests that may be ordered for the woman with hirsutism include serum testosterone levels and an adrenal CT scan. Testosterone levels greater than 200 ng/dL indicate the need for further tests, such as a pelvic examination and tests of ovarian function. Adrenal tumours, a possible cause of hirsutism, are identified with an adrenal CT scan.

Medications

Hirsutism is treated with medications specific to the underlying cause. Oral contraceptives containing oestrogen decrease ovarian androgen production and decrease free testosterone levels. Dexamethasone may be prescribed for people with high cortisol levels. Ketoconazole inhibits androgen production. Anti-androgenic medications cause congenital abnormalities in male infants and are therefore given only to non-pregnant women, who are cautioned to avoid pregnancy while taking the medications.

Male pattern baldness has been successfully treated with topical minoxidil. These drugs, which are vasodilators, stimulate vertex hair growth, probably by stimulating the epithelium of the hair follicle. These agents have been most successful in people who have a recent onset of alopecia or are less than 50 years of age. About 40% of people treated two times a day for a year will have moderate to dense regrowth of hair at the temples (Bullock & Hales, 2013).

Surgery

Hair transplant techniques are used to restore hair or reduce the size of areas of alopecia. Other types of surgical procedures include scalp reduction and flaps.

- Transplanting hairs as small hair plugs or single hairs taken from the back or sides of the scalp is an effective means of replacing hair to areas of alopecia. This procedure is done in an outpatient office or clinic.
- Scalp reduction is done by excising a portion of the affected scalp. In some cases, a tissue expander (such as a silicone balloon) is first implanted under the scalp to enlarge the hair-bearing scalp so that larger areas of alopecia can be removed.
- Flaps from hair-bearing areas of the scalp can be surgically transplanted from adjacent areas into areas of alopecia. This procedure may be done in stages.

Nursing care

The person with either hirsutism or alopecia is often self-conscious about their appearance and tries a variety of over-the-counter treatments before seeking medical care. Nursing care for the person with hair disorders focuses on teaching them self-care and providing support during long-term care. Women with hirsutism are taught to use various means of removing unwanted hair, such as shaving, applying depilatories, waxing, undergoing electrolysis or having laser treatments. Women with mild hirsutism may bleach facial hair to make it less obvious. People with alopecia may wear hair pieces or wigs.

THE PERSON WITH A DISORDER OF THE NAILS

Nail disorders may be due to systemic diseases, trauma, allergies or irritants. They may also be congenital or genetic. Nails may be discoloured, multicoloured, malformed, infected or separated from underlying tissue.

Pathophysiology

The nail disorders discussed here are separation of the nail, infection and ingrown toenails:

- *Onycholysis* is the separation of the distal nail plate from the nail bed. It occurs most often in the fingernails. This disorder may result from many different factors, including excessive or prolonged exposure to water, soaps, detergent, alkalines and industrial keratolytic agents; *Candida* infections; nail hardeners; and thyroid disorders. Prolonged application of false fingernails may also cause this disorder.
- A *paronychia* is an infection of the cuticle of the fingernails or toenails. The disorder often follows a minor trauma and secondary infection with staphylococci, streptococci.
- or *Candida*. The acute form begins with a painful inflammation that may progress to an abscess. The chronic form is seen most often in people who have frequent exposure to water. In the chronic form, the skin around the nail is painful, oedematous and infected. The nail plate may become ridged and discoloured.
- An *onychomycosis* is a fungal or dermatophyte infection of the nail plate. The nail plate elevates and becomes yellow or white. Psoriasis infections of the nail plate cause the nails to pit.
- An *ingrown toenail (unguis incarnatus)* results when the edge of the nail plate grows into the soft tissue of the toe. Pain and infection may occur. The infection, if untreated,

may spread to the bone. This disorder is especially dangerous for the person with diabetes mellitus or peripheral vascular disease.

INTERPROFESSIONAL CARE

The treatments of disorders of the nail vary from pharmacological treatment to surgical removal. Infections of the nails are treated, depending on the causative agent, with antifungal or antibiotic medications. If the causative agent is a fungus or chronic dermatological disorder, treatment is difficult and may not be effective. Persistently painful and/or infected nails are in some cases surgically removed.

Nursing care

Nursing care of the person with a disorder of the nail focuses on teaching self-care. People with nail disorders that are caused by frequent exposure to water are taught to protect the hands or feet by wearing rubber gloves or boots, and to keep the nails as clean and dry as possible. People with ingrown toenails are cautioned not to cut into the lateral nail bed, but rather to soak the nail twice a day and insert a piece of cotton or gauze under the softened nail until the nail has grown out enough to trim.

CHAPTER HIGHLIGHTS

- Pruritus (itching) accompanies dry skin (xerosis) and many skin disorders and may result in excoriation and infection as a result of scratching.
- Cysts, hypertrophic scars, keloids, naevi, angiomas, skin tags and keratoses are benign skin lesions. However, naevi should be monitored for changes indicating transformation into a malignant lesion.
- Psoriasis is a chronic immune skin disorder arising from keratinocytes. A variety of medications and treatments are used, with ultraviolet light therapy being most effective for generalised lesions.
- Skin disorders may be caused by a variety of bacterial infections, fungal infections, parasitic infestations and viral infections. The disorders are treated with a specific antibiotic, fungicide, antiviral agent or agents that kill the parasites. Herpes zoster, believed to follow a childhood infection with chickenpox, causes acute pain.
- Inflammatory disorders of the skin range from mild dermatitis to potentially lethal toxic epidermal necrolysis. Acne, a disorder of the hair and sebaceous glands opening to the skin surface, is characterised by comedones, pustules and cysts.
- Malignant skin disorders include actinic keratosis, non-melanoma skin cancer (basal cell carcinoma and squamous cell carcinoma) and malignant melanoma skin cancer. Skin cancer is the most common malignancy found in fair-skinned Australians. Prevention by avoiding sunburn, using sunscreen and maintaining monthly skin self-examination is critical in preventing loss of tissue or metastasis and death.
- Skin trauma may be intentional (as in the case of cutaneous and plastic surgery) or unintentional (as from trauma and pressure). Older adults with limited mobility, as well as people who are unable to move or are in critical care units, are at greater risk of pressure injuries and skin tears. Prevention is the goal of both interprofessional and nursing care.
- Disorders of the hair include alopecia (loss of hair) and hirsutism (excess hair in women). Nails may be discoloured, multicoloured, malformed, infected or separated from underlying tissue.

CONCEPT CHECK

1 You are caring for an elderly person with severe xerosis. What topic should be included in your teaching plan?
1. Take a hot bath every day.
2. Use fabric softeners when laundering clothing.
3. Apply skin lotions after a bath.
4. Maintain a warm environment.

2 Which of the following common skin lesions has the potential of becoming malignant?
1. naevi
2. angiomas
3. skin tags
4. keloids

3 You have been asked to teach a woman with generalised psoriasis about ultraviolet light therapy (UVB). What should be included in teaching?
1. 'The exact effect of UVB is unknown, but it decreases severe itching.'
2. 'When combined with hot baths, UVB is very effective.'
3. 'Treatments with UVB have to be given in the hospital to be safe.'
4. 'UVB slows the growth of epidermal cells and decreases keratosis.'

4 Which of the following people is at risk of the development of a candidiasis infection?
1. an older adult with pruritus
2. a young woman who is pregnant
3. an older man with a premalignant skin condition
4. a young man with multiple naevi

5 What question should be included in a health history of a person with a linear pattern of painful vesicles over the left thorax?
1. Do you remember being sunburned as a child?
2. Are you a regular patron of tanning booths?
3. Have you ever been diagnosed with acne?
4. Did you have chickenpox when you were young?

6 Which of the following statements is true of an infestation with lice?
1 Only dirty people have lice.
2 Anyone can have lice.
3 Lice do not like to live on humans.
4 Lice are a form of fungus.

7 Which assessments would indicate a greater risk to develop a non-melanoma skin cancer?
1 blonde hair, freckles, fair skin
2 alopecia, thin hair, itching
3 dark hair, dark skin, dry skin
4 tanned skin, dark hair, oedema

8 Of the following, which is most significant to the development of a malignant melanoma?
1 a change in the colour or size of a naevus
2 sexual contact with a person who has a herpesvirus infection
3 inadequate knowledge about infection prevention
4 a dietary intake of high-kilojoule foods

9 The rationale for lifting, rather than pulling, a person up in bed is that:
1 lifting a person allows a brief period of increased capillary circulation
2 lifting a person prevents tissue injury from shearing forces
3 pulling a person up in bed decreases tissue ischaemia and hypoxia
4 pulling a person up in bed promotes capillary blood flow

10 What STAR Skin Tear Classification would you assign to a skin tear where the edges can be aligned to the normal anatomical position without undue stretching and the skin or flap is pale, dusky or darkened?

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CHAPTER 16

NURSING CARE OF PEOPLE WITH BURNS

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LEARNING OUTCOMES

- Discuss the types and causative agents of burns.
- Explain burn classification by depth and extent of injury.
- Describe the pathophysiology of a minor burn.
- Describe the pathophysiology of a major burn.
- Outline the role of the nurse as part of the interprofessional team in the provision of first aid, acute care and rehabilitation of a burned person.

CLINICAL COMPETENCIES

- Assess the functional health status of peoples with burns and monitor, document and report abnormal manifestations.
- Use evidence-based research to plan and implement nursing care for people with burns.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for the person with burns.
- Administer medications knowledgeably and safely to people with burns.
- Integrate interprofessional care into care of people with burns.
- Provide teaching appropriate for prevention of burns.
- Revise plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to people with burns.

KEY TERMS

A **burn** is an injury resulting from exposure to heat, chemicals, radiation, cold injuries or electric current. A transfer of energy from a source of heat to the human body initiates a sequence of physiological events that in the most severe cases leads to irreversible tissue destruction. Burns range in severity from a minor loss of small segments of the outermost layer of the skin to a complex injury involving all body systems. Treatments vary from simple application of a topical wound dressing in an outpatient clinic or general practice to an invasive, multisystem, interprofessional team approach in the aseptic environment of a burn centre.

Over the past two decades there have been worldwide improvements in the overall care of burns victims and, ultimately, an increased survival rate. These improvements include nutrition, management of hypermetabolism, resuscitation, understanding of the post-burn immune response and technological advances in surgery and wound care products. All are a result of the continuous research into thermal injury.

The data presented in Tables 16.1 and 16.2 are excerpts from the Australian and New Zealand Burn Association (ANZBA) (2011) *Bi-National Burns Registry Annual Report Year 2, 1 July 2010–30 June 2011*. This report shows that 69.5% of burn cases were adults, with those 20 to 29 years of age making up 27% of adult cases. Children 13 to 24 months made up 34% of paediatric cases. The majority of admissions to burn centres were males in both adult and paediatric populations.

Flame injuries are the most common burn injury in the Australian adult population (see Table 16.1). Scalds are the greatest cause of burns injury in the paediatric population (see Table 16.2).

Older adults and young children are susceptible to deep burns from scalds because of their thinner skin. Burns in the older adult are complicated by co-morbidities, lower threshold for infection, higher risk of pulmonary problems, a decrease in lean body mass, micronutrient deficiency or malnutrition. These factors all lead to an increased recovery time, and rebuilding of muscle tissue is slow. There is a thinning of the dermis in older adults and epidermal replacement is slowed. When combined with a decreased blood flow, the result is slower healing of partial-thickness burns and the occurrence of deep burns in older people. Scalds are the most common cause of burns in children, typically from hot tap water or from pulling a hot beverage onto themselves. Children are inquisitive and do not necessarily have a concept of danger, particularly if something looks interesting. Children and older people have decreased reserves and there is little room for error in the treatment of a burned child or older person.

People in occupations involving work with chemicals, petrol and electricity, and those in military service, are at higher risk of burn injury. Burn and scald injuries caused by physical abuse to children or disabled or older people are also a possibility. Abuse is suspected when scald burns show a clear line of demarcation, as this could indicate deliberate immersion. The presence of small, circular burns may be the result of cigarette burns. Abused individuals may also present with poor personal hygiene status, evidence of malnutrition or behavioural problems. There may be associated soft tissue injury or fractures and old scars. In older adults the burn may be a result of self-neglect, rather than neglect by others (Wong et al., 2007). Healthcare workers should be careful when using heat packs, heat lamps and electrical equipment when caring for older people or the young.

Older people are vulnerable to fire and burn injury because of potentially impaired visual acuity, depth perception, sense of smell, hearing and mobility. All of these factors increase the risk of accidental burn injury and increased mortality.

Many burns can be prevented. Nurses can play a part in educating the public about prevention of burns, burns first aid and care of minor burn injuries at home. Box 16.1 identifies some common tips for preventing burns that nurses can advocate to the wider public.

FAST FACTS

High-risk populations

- Children
- Disabled
- Older adults
- Military

TABLE 16.1 Adults—primary cause of burn

PRIMARY CAUSE OF BURN	TOTAL	%
Flame	757	44.0
Scald	494	28.2
Contact	259	15.0
Chemical	80	4.7
Friction	63	3.7
Electrical	30	1.7
Radiant heat (no contact to source)	34	2.0
Other	12	0.7
Total	**1719**	

Source: Australian and New Zealand Burn Association Ltd (ANZBA) (2011). *Bi-National Burns Registry Annual Report Year 2, 1 July 2010–30 June 2011.* © Australian and New Zealand Burn Association Ltd. Reproduced with permission.

TABLE 16.2 Children—primary cause of burn

PRIMARY CAUSE OF BURN	TOTAL	%
Scald	413	55.0
Contact	161	21.3
Flame	106	14.0
Friction	42	5.6
Chemical	16	2.1
Radiant heat (no contact to source)	9	1.2
Electrical	6	0.7
Other	*	0.1
Total	**754**	

*Denotes fewer than five cases

Source: Australian and New Zealand Burn Association Ltd (ANZBA) (2011). *Bi-National Burns Registry Annual Report, Year 2, 1 July 2010–30 June 2011.* © Australian and New Zealand Burn Association Ltd. Reproduced with permission.

FAST FACTS

The aims of early burn management are to:

1. Preserve life
2. Preserve quality of life: functional and cosmetic.

BOX 16.1 Burn prevention tips

- Keep matches and lighters out of reach of children.
- Never leave children unattended around fire or in the bathroom or bathtub.
- Install and maintain smoke detectors in the home.
- Develop and practise a home-exit fire drill with all members of the household.
- Set the water heater temperature no higher than 50°C.
- Do not smoke in bed.
- Do not throw flammable liquids onto an already burning fire.
- Do not use flammable liquids to start fires.
- Do not remove a radiator cap from a hot engine.
- Watch for overhead electric wires and underground wires when working outside.
- Never store flammable liquids near a fire source, such as a pilot light.
- Use caution when cooking.
- Keep a working fire extinguisher and fire blanket in your home and workplace (Farrell, 2005, p. 1713).

For further information on injury prevention for children, contact www.kidsafe.com.au.

TYPES OF BURN INJURY

The types of burn injury are thermal, chemical, electrical, radiation, cold injury and friction. The extent of the injury is determined by the causative agent, length of time the tissue is exposed, body part involved and depth of burn. Although all burns can lead to generalised tissue damage and multisystem involvement, the causative agents and priority treatment measures are unique to each.

Thermal burns

Thermal burns result from exposure to dry heat (flames) or moist heat (steam and hot liquids). Direct exposure to the source of heat causes cellular destruction, resulting in damage to the skin and underlying structures.

Immediate management of thermal burns

In the case of flames, the person should stop, drop and roll to extinguish the flames. Clothing should be removed where possible and first aid commenced. First aid is the application of cool running water for at least 20 minutes. Management of thermal burns is discussed in detail later in this chapter.

Chemical burns

Chemical burns are caused by direct skin contact with acids, alkaline agents or toxic organic compounds (see Box 16.2 for specific household agents). The chemical denatures tissue protein, leading to necrosis. Chemical elements may also have a systemic effect as their elements circulate through the injured person.

BOX 16.2 Household cleaning agents that may cause burns

- Drain cleaners
- Lye
- Industrial-strength ammonia
- Household ammonia
- Oven cleaners
- Toilet bowl cleaners
- Dishwasher detergents
- Bleach

Chemical burn severity is determined by the:

- strength or concentration of the agent
- type of agent
- duration of contact
- amount of body surface area involved
- mechanism of action of the agent.

Chemical agents are further classified according to the manner by which they structurally alter proteins. Chemical burns are classified according to six mechanisms of action: reduction, oxidation, corrosive agents, protoplasmic poison, vesicants and desiccants. Chemicals can also cause eye injuries, as well as other systemic problems. Burning chemicals and substances give off toxic gases that cause inhalation injuries (Elijah, Sanford & Lee, 2012).

Alkalis

Alkalis cause liquefaction of the skin, which allows the alkali to move deeper into the tissue. Examples of alkalis include lime, sodium hydroxide and potassium hydroxide. These are present in many household cleaning products and workplace chemicals. Cement is both an alkali and a desiccant. It can penetrate clothing and when combined with sweat causes an exothermic reaction (Elijah et al., 2012).

Acids

Acids are found in many household agents and include oxalic acid, hydrofluoric acid and hydrochloric acid. Acids often cause hard, dry eschar to form. However, hydrochloric acid fumes can also cause inhalation injury. Hydrofluoric acid is a common industrial acid which causes coagulation necrosis and cellular death. The resultant chelating of calcium and magnesium causes a depletion of intracellular calcium, leading to death (Elijah et al., 2012).

Organic compounds

Organic compounds such as petroleum distillates cause damage by dissolving the lipid wall of cell membranes and may also cause renal and liver failure if absorbed. Some organic compounds such as phenols and petroleum products cause both a contact burn and systemic toxicity. Protoplasmic poisons, such as organic compounds, form salts with proteins, inhibiting calcium and other ions needed for cell viability.

Immediate management of chemical burns

Following a chemical burn the most important first aid intervention is to remove the chemical from the skin. This should

include removing all contaminated clothing and irrigation of the area with large amounts of tepid water. Care should be taken to prevent spread of the chemical on unaffected skin or on the person giving assistance. If there is dry alkali residue, it should be brushed off carefully prior to irrigation with cool running water that should be directed 'to the floor' or down a suitable drain, not in a bath where the chemical will spread to other areas of the body. The pH of the affected skin can be monitored with the use of pH indicator sticks to determine the efficacy of first aid measures. Remember to monitor for hypothermia as irrigation periods may be from 20 minutes to 2 hours.

Irrigation should not be performed with elemental metals such as lithium, potassium, sodium and magnesium. Combining these metals with water produces an exothermic reaction causing thermal injury. The area should be soaked in mineral oil and the metallic pieces removed with forceps, or the area should be covered in gauze soaked in mineral oil (e.g. liquid paraffin).

The use of neutralising agents is generally unreliable as there may be a large number of chemicals present in the workplace. During an emergency, errors in selecting the correct neutralising agent can occur, which can result in further injury. Irrigation with tepid water is the safest, and still the most effective, option.

Wound management principles for chemical burns are the same as for thermal burns. General advanced trauma life support principles—that is, the ABCs of basic life support—should always be followed and include maintenance of haemodynamics (Elijah et al., 2012; Tan & Wong, 2015).

Electrical burns

The severity of electrical burns depends on the type (alternating or direct current), duration of current, amount of voltage, resistance at point of contact and individual susceptibility. In Australia, electrical injuries are classified as low or high voltage. Low-voltage injuries are caused by electric currents below 1000 volts, and high-voltage injuries are caused by over 1000 volts or up to 33 000 volts in high-tension cables. However, less than 12 volts—for instance, that produced by a car battery—can cause a burn due to a short circuit if the person is wearing a ring or a watch (ANZBA, 2013). A high-voltage burn is associated with deep, underlying tissue damage similar to a crush injury. Low-voltage burns are usually more localised. Immediate death can occur as a result of current-induced ventricular fibrillation or asystole, or from respiratory arrest resulting from paralysis of the central respiratory control system and respiratory muscles (Aghakhani et al., 2014; Arnoldo et al., 2012.)

Electric current exists in two forms: alternating current (AC), in which the electrons move back and forth through a conductor in a cyclical fashion; and direct current (DC), in which the electrons flow in one direction. An AC current, like that found in conventional households, produces repeated electrical surges that lead to tetanic muscle contractions that can cause further injury through prolonged contact with the source or if the person is thrown. Loss of consciousness can also result in longer contact exposure. Any differences in potential injuries caused by AC and DC currents are in relation to low-voltage injuries, as high-voltage currents have a similar effect. The heart can be affected either by direct necrosis of the heart tissue or by arrhythmias. Cardiac complications are the most serious injuries associated with electrical burns and are fatal unless resuscitation measures are commenced. Respiratory arrest can occur as a result of injury to the respiratory control centre in the brain or as a result of muscle contractions causing a form of suffocation.

In addition to the injury caused by the electric current passing through vital organs, the person can receive an arc injury and a flame injury if clothing catches fire. Electric current follows the line of least resistance, and in the body this is along blood vessels and through organs, rather than through bone and fat. Deeper tissues retain the heat, and structures between bones sustain more damage than superficial structures (Arnoldo et al., 2012; Aghakhani et al., 2014).

Direct current injury—for example, caused by lightning—exposes the body to very high voltage for a short period of time. Lightning is the result of the electrical difference between a thundercloud and the ground overcoming the insulating properties of the surrounding air. Lightning injuries can range from minor to major burns. Formation of cataracts are the most common permanent injury resulting from lightning strikes. Cardiac and respiratory arrest can occur, as can damage to tympanic membranes in the middle and inner ear (Vogt, Niederbichler & Jokuszies, 2012).

Immediate management of electrical burns

Advanced trauma life support principles should be followed as soon as possible. The person should be transferred to a hospital with a burns unit. Initially, an electrical injury can look less severe than it actually is, as most of the damage is beneath the skin surface. A thorough history of the event may assist in determining the pathway of the current and the areas of injury. The person should be checked for any fractures that may have occurred and cervical spine precautions should be adhered to, particularly if the person was thrown or fell. Cardiac arrest, arrhythmias, metabolic acidosis and myoglobinuria are complications of electrical injury and need specialist medical treatment at a burn centre. The person should be monitored for cardiac complications and have an ECG performed. Pigments from muscle damage (myoglobin) and red blood cell damage (haemoglobin) can cause the urine to become dark. If untreated, myoglobinuria can lead to acute renal failure (Vogt et al., 2012). Urine output should be maintained at 1–2 mL/kg/hr (ANZBA, 2013).

Radiation burns

Ionising radiation is mediated by energy transference. It can, for example, be the result of exposure to electromagnetic radiation, x-ray or particulate radiation (alpha and beta particles or neutrons). Exposure can be from small accidents occurring in a laboratory or x-ray in a hospital. Major accidents may stretch hospital resources, as in the case of multi-trauma from military detonation of nuclear devices.

The overall extent of damage depends on the dose of radiation exposure. Skin changes usually range from erythema to dry desquamation of epidermal cells. Partial-thickness burns may

occur and are known as moist desquamation. Full-thickness burns can occur a few weeks to months after exposure. The most severe radiation injuries result from nuclear weapons and are a combination of thermal and radiation injuries (Milner & Feldman, 2012).

Sunburn tends to be superficial, involving only the epidermis; however, sunburned neonates, infants, children and older people can have significant injuries. Associated sunburn symptoms can include local discomfort, oedema, heat stroke and dehydration.

Immediate management of radiation burns

The management of radiation burns will differ subject to the cause (sunburn or exposure to radioactive substances) and local and systemic effects experienced. Management of relatively minor radiation burns involves removing patient from source, ABC of resuscitation procedures, removing contaminated clothing and copious irrigation to neutralise particles.

Management of radiation burns that occur following treatment for cancer tends to be subject to the policies of each radiotherapy unit.

Long-term effects of exposure to radiation include cancer and slow wound healing. Larger exposures or incidents can overwhelm hospital resources (Milner & Feldman, 2012).

Cold injury

Cold-induced injury (also called frostbite or localised hypothermia) is more common among homeless people, those with an interest in winter outdoor activities or individuals with alterations in cognition or perception due to dementia, high alcohol consumption or psychiatric disorders. Frostbite results when tissue temperatures fall below freezing and decreased blood flow causes tissue necrosis. Depth of injury from frostbite is classified similarly to burn injury.

If the exposure to freezing temperatures is limited, only the skin and subcutaneous tissues become involved; however, as exposure increases, deeper structures freeze. Frostbite is most common on exposed or peripheral areas of the body, such as the nose, ears, feet and hands.

As human tissues freeze, ice crystals form and increase intracellular sodium content. Small blood vessels initially vasoconstrict, but then vasodilate and become more permeable, causing cellular and tissue swelling. Continued exposure increases vasoconstriction, and increased viscosity of the blood causes infarction and necrosis of the affected tissue.

Superficial frostbite causes numbness, itching and prickling. The skin appears cyanotic, reddened or white. Deeper frostbite causes stiffness and paraesthesias. As the skin and tissues thaw, the skin becomes white or yellow and loses its elasticity. The person experiences burning pain. Oedema, blisters, necrosis and gangrene may present (Cochran, Morris & Saffle, 2012).

Immediate management of cold injuries

The biggest challenge in the field is to not cause further injury. Remove jewellery from affected area. Protect affected area from injury as the area is likely to be insensate.

Rapid thawing may significantly decrease tissue necrosis. General guidelines for re-warming areas of frostbite are:

- If outdoors, treat superficial frostbite by applying firm pressure with a warm hand or by placing frostbitten hands in the axillae. If the feet are frostbitten, remove wet footwear, dry the feet and put on dry footwear. Re-warming should not be commenced in the field unless there is certainty that the re-warming can continue uninterrupted. Refreezing of affected parts causes further damage. Monitor person for hypothermia as well (Cochran et al., 2012).
- Do not rub the areas with snow.
- In the hospital, rapidly re-warm affected areas in circulating warm water—40°C to 40.5°C—for 20 to 30 minutes. Do not rub or massage the areas.

Following re-warming, the person is kept on bed rest with the affected parts elevated. Pain medications and anti-inflammatory agents are administered. Topical wound management is instigated subject to assessment outcomes. Recovery from frostbite is usually complete if the involved area has not become necrotic. Necrotic tissue may require debridement or amputation of affected digits or limbs (Cochran et al., 2012).

Inhalation injury

An inhalation injury occurs as a result of inhalation of toxic by-products of combustion or smoke inhalation. This is discussed later in this chapter.

Friction burns

Friction burns occur as a result of the heat caused when the body is moved across a hard surface, such as a road or grass. Depth can vary from an abrasion to a wound that has blistered. Examples of injuries that may cause a friction burn are a rope that is pulled through a person's hand, or a fall from a bike onto a hard surface.

BURN CLASSIFICATION

Tissue damage following a burn is determined primarily by two factors: depth of the burn (the layers of underlying tissue affected) and extent of the burn (the percentage of body surface area involved). The assessment of the total body surface area and depth is then used to guide resuscitation and management protocols.

FAST FACTS

Factors to be considered when determining the depth of burn include:

- How the injury occurred
- Causative agent (flame, chemical, electricity, radiation)
- Temperature of burning agent
- Duration of contact
- Age-related skin thickness
- Anatomical location of burn
- First aid measures employed

Depth of the burn

The depth of a burn injury is determined by the elements of the skin that have been damaged or destroyed. Burn depth results from a combination of the temperature of the burning agent and the length of contact. Burns are classified as either superficial, partial-thickness or full-thickness burns. Characteristics of burns are outlined in Table 16.3 and illustrated in Figure 16.1.

TABLE 16.3 The characteristics of burns

	BURN DEPTH			
	SUPERFICIAL	SUPERFICIAL PARTIAL	DEEP PARTIAL	FULL THICKNESS
Wound appearance	Involves the epithelium • Painful • Red or pink	Epidermis and superficial (papillary) dermis destroyed • Painful • Often blistered • Pink, moist • Blanches	Involves epidermis and reticular dermis • May blister • Mottled pink or white • Fairly dry > day 2 • Discomfort rather than pain • Slow or no capillary refill	Involves epidermis and dermis, and may include fat • Does not blanch • May be mottled, dry, translucent, black or pale in appearance • Full-thickness scalds may have red non-blanching appearance
Healing and scarring	Complete scarless healing within 7–10 days	Heals by epithelial migration within 2 weeks • Can convert to a deeper burn	Will take more than 3 weeks to heal • Will leave a scar if > 3 weeks to heal	Will require surgery unless very small wound • Will have a scar if left to heal without surgery • Early excision and grafting reduce scarring and contracture

Sources: T. McWilliams, Princess Margaret Hospital for Children; and S. Rowe, Fiona Stanley Hospital.

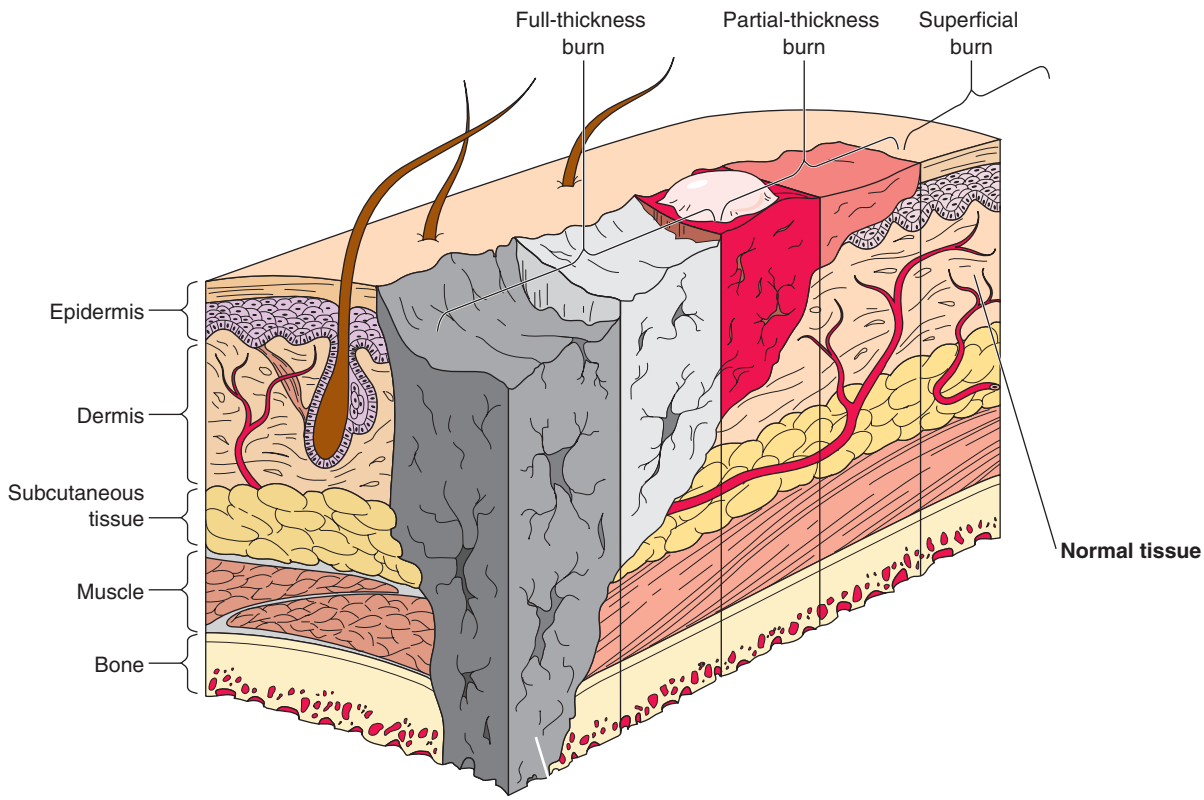


FIGURE 16.1 ■ Burn injury classification according to the depth of the burn

Superficial burns

A **superficial burn** involves only the epidermal layer of the skin (see Figure 16.2). This type of burn most often results from damage from sunburn, ultraviolet light, minor flash injury (from a sudden ignition or explosion) or mild radiation burn associated with cancer treatment. Because the skin remains intact, this degree of burn is not calculated into the estimates of burn injury. Due to the increased blood supply to the area, the erythema ranges from pink to bright red and there may be slight oedema over the burned area. Superficial burns involving large body surface areas may be accompanied by chills, headache, nausea and vomiting. The injury usually heals in 3 to 6 days, with dryness and peeling of the outer layer of skin. There is no scar formation. Superficial burns are treated with analgesia and the application of non-perfumed moisturising ointments and lotions. Extensive superficial burns, especially in infants and older

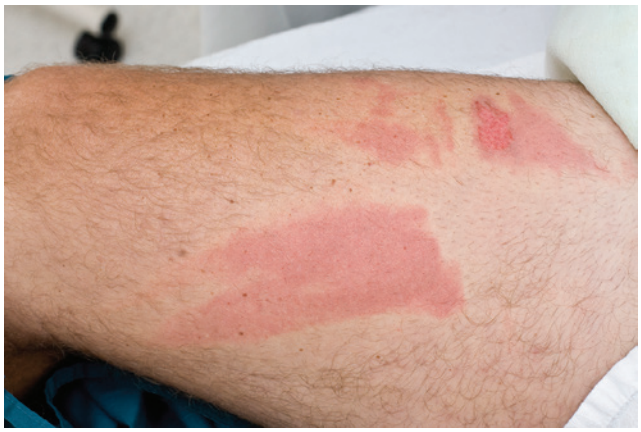


FIGURE 16.2 ■ Superficial burn injury

Source: Royal Perth Hospital.

people, may require intravenous fluid treatment. Superficial burns can convert to partial-thickness wounds if inappropriate treatment is administered or infection and swelling are untreated.

Partial-thickness burns

Partial-thickness burns may be subdivided into superficial partial-thickness and deep partial-thickness burns. The classification depends on the depth of the burn.

A *superficial partial-thickness burn* involves the entire epidermis and the papillae of the dermis. Causes may include such injuries as brief exposure to a flash flame or dilute chemical agents, or contact with a hot surface. This burn is often bright red, but has a moist, glistening appearance with blister formation (see Figure 16.3). The burned area will blanch on pressure, and touch and pain sensation remains intact. Pain in response to temperature and air is usually severe. These injuries heal within 14 days with minimal or no scarring, but pigment changes are common. Analgesics are administered and the wound will require a dressing. Superficial partial-thickness burns can convert to a deep partial if inappropriate treatment is administered or infection and oedema go untreated.

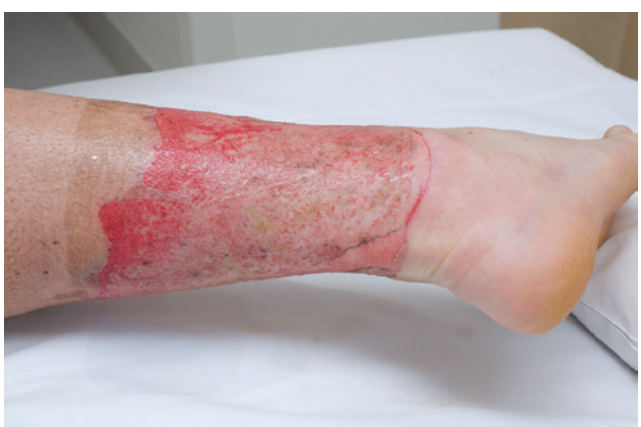


FIGURE 16.3 ■ Mid-dermal or partial-thickness burn injury

Source: Royal Perth Hospital.

A *deep partial-thickness burn* also involves the entire dermis, but extends further into the dermis than a superficial partial-thickness burn. Hair follicles, sebaceous glands and epidermal sweat glands remain intact. Hot liquids or solids, flash flame, direct flame, intense radiant energy or chemical agents may cause this level of burn injury. The surface of the burn wound can appear pale, blotchy red or mottled, and may be moist or dry. Large, easily ruptured blisters may be present or the blisters may look like flat, dry tissue paper. Capillary refill is decreased, and sensation to deep pressure is present. The burn wound is less painful than a superficial partial-thickness burn, but areas of pain and areas of decreased sensation may be present. Deep partial-thickness burn wounds will take more than 21 days for healing and may convert to a full-thickness injury if necrosis extends the depth of the injury. **Contractures** are possible, as are hypertrophic scarring and functional impairment (see Figure 16.4). Excision and grafting may be necessary to decrease scarring and loss of function.

Full-thickness burns

A **full-thickness burn** involves all layers of the skin, including the epidermis, the dermis and the epidermal appendages (see Figure 16.5). The burn wound may extend to the subdermal level involving the subcutaneous fat, connective tissue, muscle

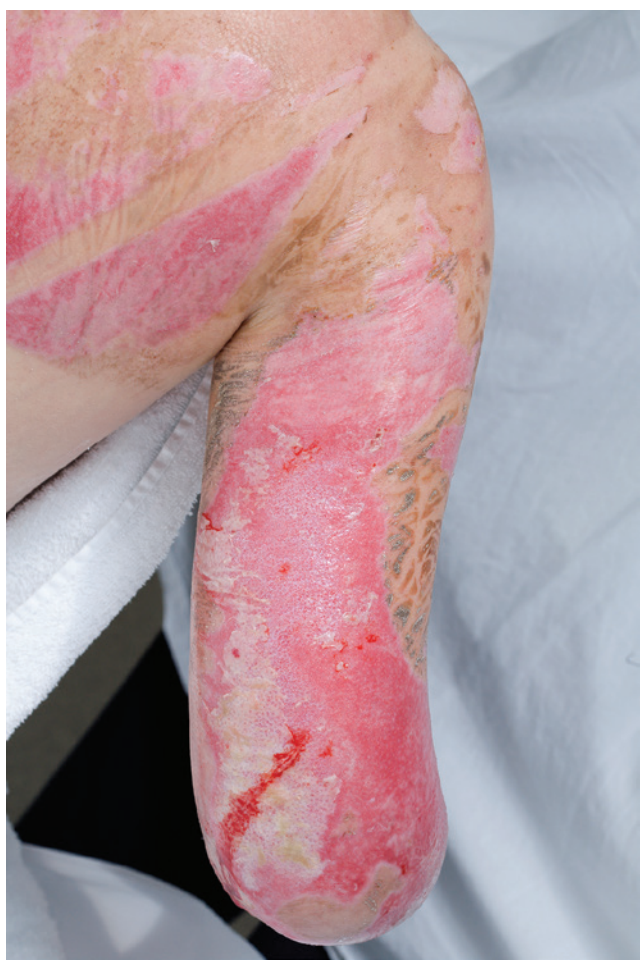


FIGURE 16.4 ■ Deep partial-thickness burn injury

Source: Royal Perth Hospital.

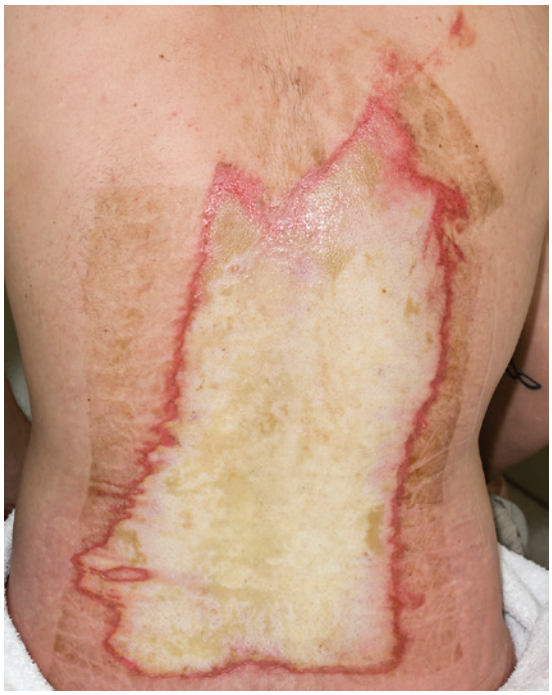


FIGURE 16.5 ■ Full-thickness burn injury

Source: Royal Perth Hospital.

and bone. Full-thickness burns are caused by prolonged contact with flames, steam, chemicals or high-voltage electric current.

Depending on the cause of injury, the burn wound may appear pale, waxy, yellow, brown, mottled, charred or non-blanching red, and the wound surface is dry, leathery and firm to the touch. Thrombosed blood vessels may be visible under the surface of the wound. There is no sensation of pain on light touch, because pain and touch receptors have been destroyed, although there may be deeper discomfort or pain felt. Full-thickness burns require skin grafting to heal. In dark-skinned people, assessment of burned area should be done with gloved hands assessing for skin turgor and pain. The dead skin of the full-thickness burn wound is called **eschar** (Lewis, Heimbach & Gibran, 2012).

Extent of the burn

Accurate assessment of the extent of burn injury is one of the most important aspects of initial care in the emergency treatment of burn injury.

The extent of the burn injury is expressed as a percentage of the total body surface area (TBSA) or body surface area (BSA) and is determined according to validated guides.

Two of the most commonly used guides to estimate TBSA and extent of burn wound are the Wallace (1951) rule of nines (see Figure 16.6) and the Lund and Browder (1944) burn assessment chart (see Figure 16.7).

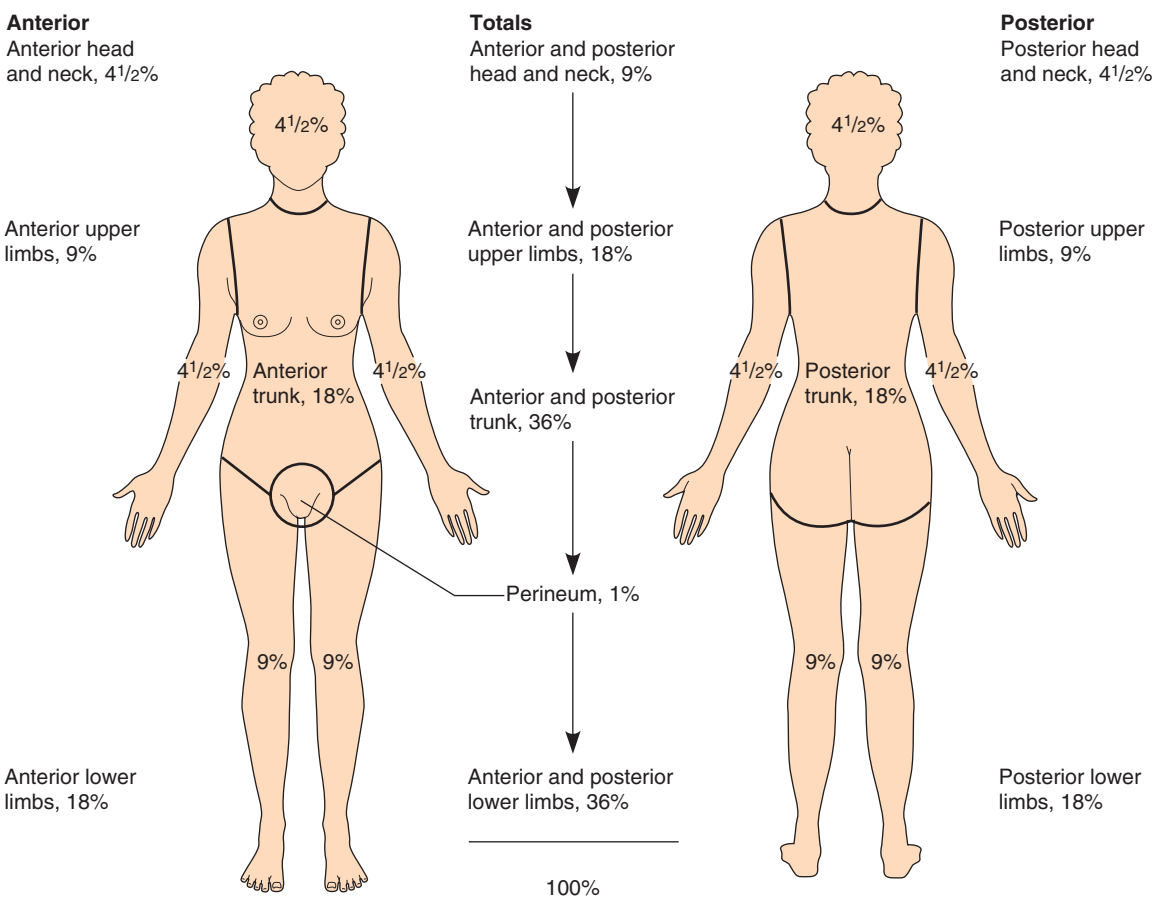


FIGURE 16.6 ■ The rule of nines (Wallace, 1951) is one method for quickly estimating the percentage of TBSA affected by a burn injury. Although useful in emergency care situations, the rule of nines is not accurate for estimating TBSA for adults who are short, obese or very thin, or for children

Area	Age (years)					% 1∞	% 2∞	% 3∞	% Total
	0–1	1–4	5–9	10–15	Adult				
Head	19	17	13	10	7				
Neck	2	2	2	2	2				
Ant. trunk	13	13	13	13	13				
Post. trunk	13	13	13	13	13				
R. buttock	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$				
L. buttock	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$				
Genitalia	1	1	1	1	1				
R.U. arm	4	4	4	4	4				
L.U. arm	4	4	4	4	4				
R.L. arm	3	3	3	3	3				
L.L. arm	3	3	3	3	3				
R. hand	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$				
L. hand	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$	$2\frac{1}{2}$				
R. thigh	$5\frac{1}{2}$	$6\frac{1}{2}$	$8\frac{1}{2}$	$8\frac{1}{2}$	$9\frac{1}{2}$				
L. thigh	$5\frac{1}{2}$	$6\frac{1}{2}$	$8\frac{1}{2}$	$8\frac{1}{2}$	$9\frac{1}{2}$				
R. leg	5	5	$5\frac{1}{2}$	6	7				
L. leg	5	5	$5\frac{1}{2}$	6	7				
R. foot	$3\frac{1}{2}$	$3\frac{1}{2}$	$3\frac{1}{2}$	$3\frac{1}{2}$	$3\frac{1}{2}$				
L. foot	$3\frac{1}{2}$	$3\frac{1}{2}$	$3\frac{1}{2}$	$3\frac{1}{2}$	$3\frac{1}{2}$				
					Total				

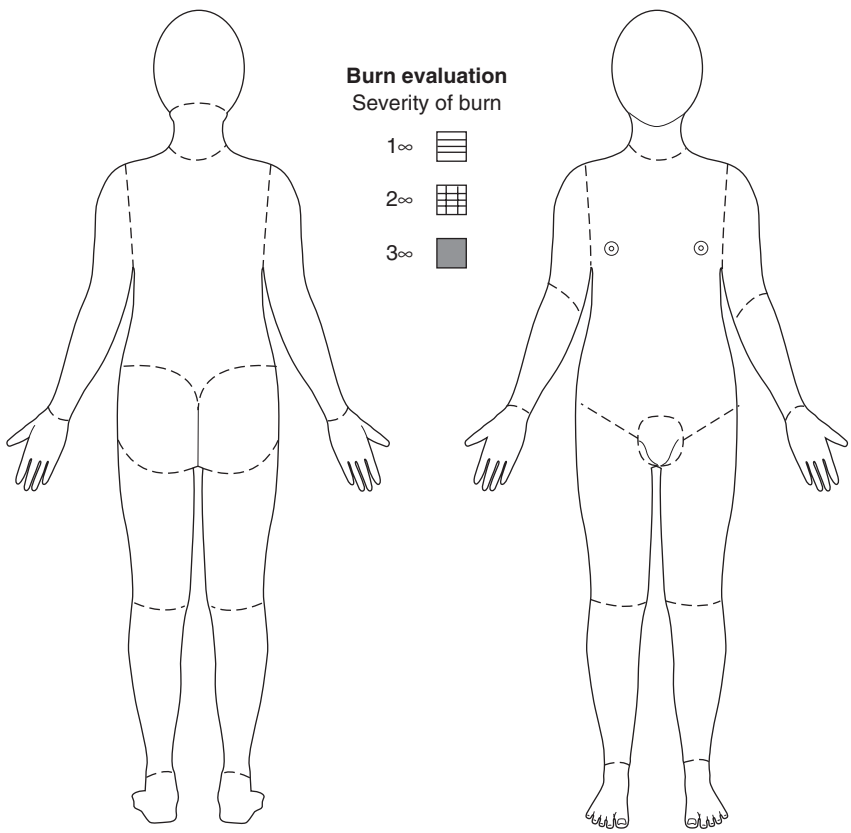


FIGURE 16.7 ■ The Lund and Browder (1944) burn assessment chart. This method of estimating TBSA affected by a burn injury is more accurate than the rule of nines because it accounts for changes in body surface area across the lifespan. As individual burn units may use a modified Lund and Browder chart, practitioners are advised to familiarise themselves with local assessment guidelines

Only partial-thickness and full-thickness burns are included in the estimation; superficial erythema is not classified as a percentage of TBSA. The rule of nines is adequate for initial assessment, but for irregularly shaped burns the palmar surface of the patient's hand (including fingers) is considered 1% TBSA. The percentage of TBSA is reassessed after oedema has subsided and demarcation of zones of injury has occurred. The rule of nines is relatively accurate in adults, but is not suitable for children as children's body surface area proportions differ. Children have relatively large heads and smaller lower bodies than adults. Using the rule of nines on a child could lead to overestimation of TBSA and subsequent inaccurate **fluid resuscitation**. The Lund and Browder burn assessment chart is used for paediatric burn injuries.

The Wallace rule of nines

The Wallace rule of nines divides the surface area of the body into segments of 9%, 18% and 1%.

Head and neck	9% TBSA
Anterior torso	18% TBSA
Posterior torso	18% TBSA
Legs	18% TBSA
Arms	9% TBSA
Groin	1% TBSA

See Figure 16.6 for an example.

Lund and Browder burn assessment chart

Fluid loss in children is proportionally greater than in adults due to their body weight to body surface area ratio. Fluid loss is directly proportional to the burned surface area, so the rule of nines is inadequate in children under 15 years of age (Lee, Norbury & Herndon, 2012). Resuscitation fluid formulae for children are based on body surface area and take into account age, height and weight. This formulae was initially proposed by Lund and Browder (1944) and an assessment chart that takes these factors into account has been named after these practitioners (see Figure 16.7).

For example, a person with burns to the face, anterior right arm and anterior trunk has the following burn injury:

Face = 4.5%
Arm = 4.5%
Trunk = 18%
= 27% TBSA.

Phases of burn management

Management of burn injuries is divided into the resuscitative, acute and rehabilitative phases, although there is overlap of all phased interventions, particularly those involving wound management and rehabilitation:

1. Resuscitative phase:
   - First aid
   - Primary survey
   - Secondary survey.
2. Acute-wound healing phase.
3. Rehabilitative phase.

The resuscitative phase begins at the time of injury and lasts until the fluid resuscitation phase is completed, which usually lasts 48 to 72 hours. The acute-wound healing phase commences as part of the secondary survey. The rehabilitative phase commences on day one; all interventions from first aid through the continuum potentially impact on rehabilitation. The acute-wound healing phase and the rehabilitative phase are discussed in depth later in the chapter (see p. 494).

Resuscitative phase

During this phase, first aid is administered, the size of the burn is calculated, fluid resuscitation is initiated and lifesaving measures are implemented. Of primary concern is the onset of hypovolaemic or **burn shock** and the onset of oedema. The phase ends with the diuresis phase of burn fluid mobilisation. The person is assessed for signs of respiratory distress and shock. Intravenous access is obtained and the person may be prophylactically intubated. A decision should be made as to whether the person requires transfer to a specialist burn centre for complex care (see Box 16.3).

Any concurrent injuries may need to take priority over the burn wound. Communication is vital to the wellbeing of the injured person. All relevant information should be relayed to the receiving hospital, including circumstances surrounding the accident. This is especially important if the person was trapped in an enclosed space or there were chemicals involved or possible trauma (ANZBA, 2013; Knighton & Fong, 2008; Warden, 2012).

FIRST AID At the scene of the burn, priority is given to removing the person from any danger, managing life-threatening conditions and cooling the burn. Rescuers must take care not to be injured as well. Table 16.4 outlines the procedures that must be undertaken at the scene of a burn. For large burns it is not advisable to immerse the whole person in water for cooling, as this will cause too much heat loss. Never put ice in the water as

BOX 16.3 The Australian and New Zealand Burn Association referral criteria for transfer to a burns unit

- Burns greater than 10% of total body surface area (TBSA)
- Burns greater than 4% of TBSA in children
- Burns of specialised areas—face, hands, feet, genitalia, perineum and major joints
- Full-thickness burns greater than 5% of TBSA
- Electrical burns
- Chemical burns
- Burns associated with inhalation injury
- Circumferential burns of the limbs and chest
- Burns in children and older adults
- Burns in people with pre-existing medical disorders that could complicate management, prolong recovery or increase risk of mortality
- Burn injury in pregnant women
- Burns with associated trauma
- Non-accidental burns

Source: Australian and New Zealand Burn Association Ltd (ANZBA) (2013). *Emergency Management of Severe Burns.* Course manual (version 17). © Australian and New Zealand Burn Association Ltd. Reproduced with permission.

TABLE 16.4 First aid procedures at the scene of a burn

Electrical burn	Switch off power. Remove the person from the source of electricity using a non-conducting object; preferably by a trained person. Put out flames if the person has sustained a flame injury as well. Begin primary survey. Protect cervical spine. Ensure rescuer safety. Remember high-voltage electricity will discharge through air.
Chemical burn	Brush residual dry chemical from person. Copious irrigation of area 'to the floor' with water. Ensure rescuer safety. Observe for signs of hypothermia. Begin primary survey.
Flame burn	Stop, drop and roll. Extinguish flames, remove clothing if possible and restrictive jewellery. Irrigate under cool, clean water for at least 20 minutes. If water for copious cool-water irrigation is not available, apply cool wet towels and change when they become warm. Observe for hypothermia. Ensure rescuer safety. Begin primary survey.
Scald	Remove clothing where possible. Irrigate with copious cool, clean water for at least 20 minutes. If water for copious cool-water irrigation is not available, apply cool wet towels and replace when they become warm. Observe for signs of hypothermia.

Source: Australian and New Zealand Burn Association (ANZBA) (2013). *Emergency Management of Severe Burns.* Course manual (version 17). © Australian and New Zealand Burn Association Ltd. Reproduced with permission.

this can cause cold injuries. Remove as much burned clothing as possible and replace with a clean sheet. Cover the person with a blanket to keep them warm.

PRIMARY SURVEY Life-threatening issues are identified and commencement of emergency management is done in this phase. The ABCDEF acronym (see Table 16.5) provides a guide to emergency resuscitative interventions (ANZBA, 2013).

SECONDARY SURVEY This phase is a head-to-toe examination commencing after any life-threatening conditions are dealt with. Secondary survey commences after the primary survey has been completed and the person is stabilised. It includes a comprehensive assessment of the person and records their allergies, medications, health history, the last time they ate and the events surrounding the incident. Identification of the mechanism of injury is vital, and the presence of any penetrating wounds or blunt trauma needs to be ascertained.

CONSIDERATION FOR PRACTICE

Narcotics are always administered intravenously, rather than orally, subcutaneously or intramuscularly in the resuscitative or acute phase of a burn due to decreased circulation and absorption of medications.

PREPARATION FOR TRANSFER TO A BURNS UNIT

Once the referral criteria for transfer to a specialised burns unit have been met (see Box 16.3), preparation of the stabilised person for transfer to that unit involves the following measures and interventions. The following points are covered as part of the primary and secondary survey:

1. *Respiratory system.* Establish and maintain airway; administer humidified 100% oxygen. Consider the need for endotracheal intubation (before transfer) if there is a possibility of upper airway obstruction. Transfer

TABLE 16.5 Emergency resuscitative interventions—ABCDEF

PROBLEM	INTERVENTION
Airway	Check for patency, soot around mouth and nares, singed nasal hairs. Use cervical spine precautions at all times. Injuries above the clavicles or facial injuries can indicate spinal injuries.
Breathing and ventilation	Assess chest movement to ensure chest is rising equally. Administer humidified 100% oxygen. Ventilate with bag and mask if necessary; prepare for possible intubation. Assess for carbon monoxide poisoning (i.e. decreased conscious level, cherry red appearance). Respiratory rate of over 20/minute is not necessarily a good thing. Circumferential chest burns can restrict air entry. Non-circumferential full-thickness torso burns can also restrict air entry.
Circulation	Check for strength, regularity and presence of pulses. Capillary refill of nail beds should be 2 seconds. Longer capillary refill times could indicate hypovolaemia, hypothermia or the need for escharotomies. Stop any bleeding. Elevate any burned or oedematous limb above the level of the heart if not contraindicated. Electrical burns should be monitored for cardiac complications.
Disability and neurological status	Establish level of consciousness using Glasgow Coma Scale. Examine pupil reaction to light. Hypoxia and shock can cause restlessness and decreased level of consciousness.
Exposure and environment	Remove all jewellery and burned clothing. Prevent hypothermia. If clothing is stuck, trim loose pieces.
Fluid resuscitation	Insert two large-bore cannulae into non-burned tissue. Take blood specimen for crossmatching. Commence intravenous fluids as per formula.

Sources: Australian and New Zealand Burn Association (ANZBA) (2013). *Emergency Management of Severe Burns.* Course manual (version 17); Burns: Nursing management by J. A. Knighton & J. Fong (2008). In D. Brown & H. Edwards (eds), *Lewis's Medical–Surgical Nursing: Assessment and Management of Clinical Problems* (2nd ed.) (pp. 533–559). Sydney: Elsevier; Pre-hospital management, transportation and emergency care by M. R. Mlcak, M. C. Buffalo & C. J. Jimenez (2012). In D. Herndon (ed.). *Total Burn Care* (4th ed.) (pp. 93–102). Philadelphia: Saunders.

the person with their head elevated unless contraindicated (for instance, associated spinal injuries) as this will help decrease swelling and aid breathing.

2. *Circulation.* Insert two large-bore intravenous cannulae (size 16 for adults, size 20 for children) through non-burned skin where possible. Maintain resuscitation fluid as per formula for burn resuscitation. All limbs that are burned should be elevated where not contraindicated and cervical spine precautions maintained at all times.
3. *Urinary output.* Insert an indwelling catheter for burns above 10% TBSA in children and above 15% to 20% TBSA in adults or in burns of genitalia. Maintain urine output at:
   - adults 0.5 mL/kg/hr = 30–50 mL/hr
   - children (< 30 kg) 1.0 mL/kg/hr (range 0.5–2 mL/kg/hr).
   - Accurate fluid balance monitoring is the most effective way of determining adequacy of fluid resuscitation measures. In the case of an electrical injury, urine should be maintained at 1.0–2.0 mL/kg/hr. Weighing of nappies for children is not an accurate assessment of urine output.
4. *Wound management.* Wash the burn area with chlorhexidine 4% or normal saline and then cover with a clean dry sheet or, if possible, a silver-impregnated dressing. In remote Australian regions, time delays in transferring people to a burns unit may be prolonged. The use of cling wrap products is not advised as they can cause retention of heat in the burn; the application of a silver-impregnated dressing (e.g. Acticoat®) is preferred if transfer is to be delayed over 2 hours from the time of injury.
5. *Analgesia.* Liaise with burns unit as to the preferred protocol for analgesia administration, particularly if children are involved. Oral analgesia is not recommended except in minor burns. Narcotic analgesia given intravenously is essential for the person's comfort. Monitoring of level of consciousness, respiratory status and effectiveness of analgesia should be maintained.
6. *Gastrointestinal system.* Insert a nasogastric or orogastric tube in a person who has sustained a burn of >15% TBSA in adults and >10% TBSA in children. This is to decrease the risk of vomiting and potential aspiration. If the transfer of the person is delayed, this intervention also allows for enteral feeding, if required and following consultation with the burns unit team.
7. *Tetanus status.* Tetanus prophylaxis is recommended for all people who sustain a burn injury. A correct history should be taken where possible to ascertain if a booster or immune globulin is required (Allison & Porter, 2004; Mlcak, Buffalo & Jimenez, 2012).

Acute wound healing phase

The acute wound healing phase commences as part of the secondary survey and is discussed in depth later in the chapter.

Rehabilitative phase

The rehabilitative phase also commences on day one. All interventions from first aid through the continuum potentially impact on rehabilitation. This is discussed later in the chapter.

BURN WOUND HEALING

Minor burns heal via spontaneous regeneration and repair, while major burns will require surgical debridement, skin grafts or flaps. The healing process involves three phases: inflammation, reconstruction and remodelling. The degree of injury will influence the nature of healing and the amount of regeneration and repair that will occur (Porth & Matfin, 2009).

- *Inflammation*. Immediately following the injury, platelets coming in contact with the damaged tissue aggregate. Fibrin is deposited, trapping further platelets and a thrombus is formed. The thrombus, combined with local vasoconstriction, leads to haemostasis, which inhibits bleeding.

  Local vasodilation and an increase in capillary permeability follow haemostasis. Neutrophils infiltrate the wound and peak in about 24 hours, and then monocytes predominate. The monocytes are converted into macrophages, which consume pathogens and dead cells and also secrete various growth factors. These growth factors stimulate the proliferation of fibroblasts and the deposit of a provisional wound matrix.
- *Reconstruction.* Within 2 to 3 days post burn, fibroblasts are the major cell within the wound. Their number peaks at about 14 days after the injury. Granulation tissue begins to form, with complete re-epithelialisation occurring during this phase. Epithelial cells cover the wound, as each cell stretches across the wound surface to join with other epithelial cell sheets on the other side of the wound. The proliferation phase lasts until complete re-epithelialisation occurs, by epithelial cell migration, surgical intervention or a combination of the two.
- *Remodelling or maturation*. This phase may last for years. Collagen fibres, laid down during the proliferative phase, are reorganised to improve the tensile strength of the wound. Scars contract and fade in colour. In normal healing following a superficial minor burn injury, the newly formed skin closely resembles its neighbouring tissue. However, when a burn injury extends into the dermal layer of skin, two types of excessive scar may develop. A **hypertrophic scar** is an overgrowth of dermal tissue that remains within the boundaries of the wound. A **keloid** is a scar that extends beyond the boundaries of the original wound. People with dark skin, such as Indigenous Australians, Pacific Islanders, Asian, African and Middle Eastern populations, are at greater risk of hypertrophic scars and keloids.

The length of time a wound takes to heal influences scar formation. Time to healing is an outcome measure for treatment effectiveness, regardless of treatment choice. Healing after a 2-week period is known to be a predictor of hypertrophic scarring. Surgery may be needed to speed up healing time to minimise scarring. All wound management decisions should focus on achieving re-epithelialisation within 2 weeks of the burn injury.

Post-burn itch

Although the exact cause of post-burn itch is not known, it is considered to be due to both peripheral and central mechanisms. Mediators which relay itch sensations peripherally are released when keratinocytes are damaged. These mediators include

histamine, interleukins, protease-activated receptors and nerve growth factors. Itch is a common and disruptive part of burn injury. It can start in the first 2 weeks after the burn and can last for a long time. Itch can lead to further damage to fragile skin as the person scratches in an attempt to relieve the itch. Consensus on successful treatment of the pruritus is scarce in the literature; however, there are a number of pharmacological and non-pharmacological treatments employed. Oral and topically applied antihistamines are commonly used. Non-pharmacological treatments include massaging the scar or healed burned area. Colloidal oatmeal baths and shower washes are also recommended, along with ice packs and instructions for keeping the area cool. Treatment of itch should be as much a priority as treatment of pain. Further research into the causes and treatment of post-burn itch is required (Bell & Gabriel, 2009).

THE PERSON WITH MINOR BURNS

Minor burn injuries consist of superficial burns that are not extensive, and superficial partial-thickness burns that involve less than 10% of TBSA, excluding the special care areas (eyes, ears, face, hands, feet, perineum and joints). Minor burns can also be a small area of full-thickness eschar that is to be treated conservatively rather than with surgical debridement. Minor burn injuries are not associated with immunosuppression or hypermetabolism.

People with a minor burn injury are usually treated as an outpatient. The goal of therapy is to promote wound healing, eliminate discomfort, maintain mobility and prevent infection.

Pathophysiology

Sunburn

Sunburns result from exposure to ultraviolet light. Such injuries, which tend to be superficial, are more commonly seen in people with lighter skin. Because the skin remains intact, the manifestations in most cases are mild and are limited to pain, nausea, vomiting, skin redness, chills and headache. Treatment is performed on an outpatient basis and generally consists of applying non-perfumed lotions, increasing fluid intake, administering mild analgesics and maintaining warmth. Older people should be monitored for evidence of dehydration. Proper use of sunscreen and limiting sun exposure to the less hazardous hours of the day can prevent sunburn.

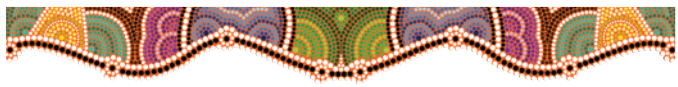
Scald burn

Scald burns result from exposure to moist heat, and the depth of a scald depends on the temperature of the water, the thickness of the skin and the duration of contact. Water at 60°C from fluids such as hot coffee or soup can cause deep partial-thickness burns.

For scalds that are minor, the person can be treated as an outpatient with appropriate dressings and analgesia to allow for a full range of movement and to keep them comfortable. Tetanus toxoid prophylaxis is administered as appropriate.

Contact burn

Contact burns usually result from hot metals, plastics, hot footpaths or motorbike exhaust pipes. They are usually small in circumference, but can be deep partial thickness. Treatment may include dressings and/or surgery. Tetanus toxoid prophylaxis is administered as appropriate. Burns from sun-heated surfaces are not uncommon. Children and adults with diabetes are particularly susceptible. The focus for these kinds of burns should be on prevention and education of vulnerable people (Asquith, Kimble & Stockton, 2014).



Nursing care

Management of a minor burn

Regardless of the care setting, the principles of management are the same. The aim is to promote wound healing and prevent infection while maintaining a full range of movement. Adequate analgesia should be prescribed to keep the person comfortable and to enable the person to maintain full range of movement.

Nursing care includes taking a comprehensive history of the incident and health history, administration of prescribed analgesics, ongoing wound assessments, wound care planning, referrals for physiotherapy and/or occupational therapy and arranging follow-up appointments. Initially, the wound is washed using chlorhexidine liquid soap, or a mild soap if this isn't available. Remove any loose, non-viable skin with sterile scissors.

Deroof blisters where necessary and in accordance with hospital policy as blisters that extend over a joint can decrease function and movement. Blister fluid can also cause pressure over the wound bed, which compromises perfusion and increases pain. If blisters are on the pads of fingers or toes they may be debrided on day 3 or 4 post burn injury, but should be slit to release fluid and decrease pressure on first presentation (Sargent, 2006).

Once the burn has been cleaned and the non-viable tissue removed a more thorough assessment can be made of the wound. Wounds with a pink wound bed, free of slough or eschar, are likely to be superficial or superficial partial thickness and should heal within 7 to 14 days and leave no permanent scarring. It should be noted that infection, poor dressing choice and oedema can cause the burn to convert to a deeper burn. Superficial burns where there is no skin loss may only require non-perfumed moisturising creams. A plethora of wound dressings are used in the treatment of burns, and individual health agencies and burn health professionals will have preferred protocols.

Hydrocolloids are a suitable choice for the low-to-moderate exudating superficial/partial-thickness wounds. They are not suitable for wounds with a large amount of exudate as maceration may occur with the accumulation of exudate. Hydrocolloids are waterproof and for this reason a good choice for an outpatient. Dressing changes are required every 3–4 days depending on the amount of exudate.

Calcium alginates are useful for moderate-to-highly exudating wounds but require a secondary dressing. Alginates require dressing changes every 2–4 days, depending on the amount of exudate, and can be removed in the shower or bath if they have dried out.

Foams are a suitable choice for superficial/partial and deep partial burns if there are moderate-to-high amounts of exudate. Foams are used in conjunction with a hydrogel on full-thickness burns in order to assist debridement of eschar. Foams come in adhesive and non-adhesive forms.

Hydrogels are a suitable choice for low-exudating wounds or wounds with dry eschar as they help to maintain a moist wound environment and assist in debridement of slough and eschar. Hydrogels are available in amorphous gels, or gel-impregnated gauzes or sheets.

Antimicrobial dressings will be required if infection is suspected or confirmed.

Community-based care

The nurse should address the following topics to facilitate self-care at home of minor burns.

- Educate the person and carer about the signs and symptoms of infection—that is, increase in pain, swelling and redness of the wound area. Provide instructions on:
  - how to care for the wound and dressing
  - how to take prescribed analgesia correctly and effectively
  - contact details for a clinic or a burns unit in case of any problems or concerns
  - how to change dressings if needed and equipment to do so
  - non-pharmacological pain-relieving measures—for example, elevation of the limb when sitting.

THE PERSON WITH MAJOR BURNS

The Australian and New Zealand Burn Association (ANZBA) identifies a major burn as above 15% to 20% TBSA in adults (in older adults or the infirm 15% TBSA) or above 10% TBSA in children. In addition, burns of certain anatomical sites, such as the perineum, or those that occur as a result of certain agents, require specialised burn treatment and consultation should be sought (see Box 16.3).

Pathophysiology

There is a local and a general response to a burn injury. The local response is explained by Jackson's zones of burn injury model (1950) (see Figure 16.8). The general response is a multi-system response that requires specialised care in order to preserve life and result in the best possible outcome for the person.

The pathophysiological changes that result from major burn injuries involve all body systems. Extensive loss of skin (the body's protective barrier) can result in infection, fluid and electrolyte imbalances, and hypothermia. Cytokines and other mediators are released into the systemic circulation causing a systemic inflammatory response. If the person inhales the products of combustion, an inhalation injury can result, thus compromising respiratory function. Cardiac arrhythmias and circulatory failure are potential complications of serious burn injuries. A hypermetabolic state dramatically increases kilojoule expenditure and nutritional deficiencies. The increase in metabolic rate can last for up to 1 year post injury (Bourdeaux & Manara, 2008). An alteration in gastrointestinal motility predisposes the person to developing paralytic ileus, and hyperacidity can lead to gastric and duodenal ulcerations. Translocation of gut bacteria due to increased permeability can lead to infection. The person is centrally dehydrated, so glomerular filtration rates and renal clearance of toxic wastes are less effective; this can result in acute tubular necrosis and renal failure. Systemic responses to burns are outlined below.

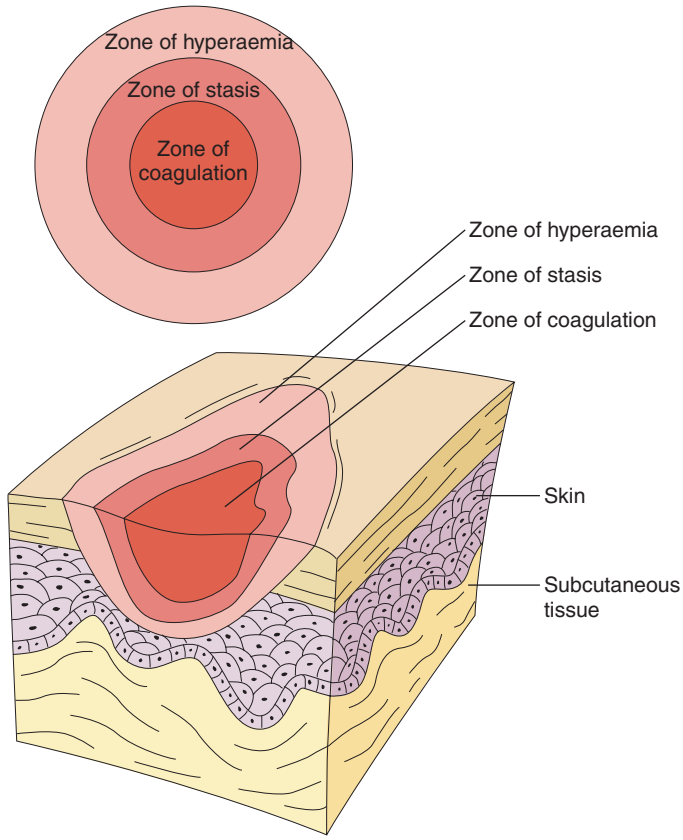


FIGURE 16.8 ■ The Jackson zones of burn injury (Jackson, 1950)

Source: Adapted from D. M. Jackson (1950). The diagnosis of the depth of burning. *British Journal of Surgery*, *40*(164), 588–596.

Integumentary system

The loss of skin in burn injuries interrupts normal skin functions and its protective mechanisms (see Chapter 14). Key mechanisms lost in burn injuries include the prevention of evaporative water loss, protection against bacteria and the maintenance of body warmth.

Heat transfer to skin is a complex phenomenon. If the microcirculation of the skin remains intact during burning, it cools and protects the deeper portions of the skin and cools the outer surface once the heat source is removed. When extensive burn injury occurs, the integrity of the microcirculation is lost and the burning process continues even after the heat source is removed.

JACKSON'S ZONES OF BURN INJURY Jackson (1950) proposed three concentric zones of burn injury. The zones are three dimensional and damage to the zone of stasis can lead to conversion to a deeper burn.

It is important to understand the pathophysiology of a burn in order to treat the person effectively (see Figure 16.8). Different causes of burns require different treatment.

- The outermost zone of hyperaemia affects only the epithelium; there is no skin loss. In this zone, tissue perfusion is increased, giving rise to the reddened erythema appearance. This zone usually heals within 7 days unless there is trauma to the area, infection or oedema, which can cause a conversion to a deeper burn.
- The medial zone of stasis is initially moist, red and blistered, and blanches on pressure. Tissue perfusion is decreased in this zone. It may recover or become pale and necrotic as a result of inadequate fluid resuscitation, infection, trauma, oedema or poor wound management.
- The inner zone of coagulation is the area of most damage. There can be no reversal of necrosis. Proteins are coagulated and the burn has a 'leathery' appearance (Bourdeaux & Manara, 2008; Singh et al., 2007).

The overall thickness of the dermis and epidermis varies considerably from one area of the body to another and is subject to the individual's age. Similar temperatures produce different depths of injury to different body parts. For example, in the adult, skin covering the medial aspect of the forearm is thinner and more easily damaged than the skin covering the back of the same person. Skin dissipates heat maximally in areas of greatest vascularisation. When heat absorption exceeds the rate of dissipation, cellular temperatures rise and skin tissue is destroyed.

The deep burn injury results in the formation of necrotic skin and damage to subcutaneous tissue. During the acute phase of the injury a hard crust (eschar) forms which covers the wound. The eschar is characteristically leathery and rigid. Removal of the eschar is required if healing is to be facilitated. In a partial-thickness and deep partial-thickness wound, slough (moist non-viable tissue) covers the wound surface, which also needs to be debrided to facilitate healing.

Hypovolaemic shock

Fluid and electrolyte shifts in burn injury result in the movement of large amounts of fluid from the intracellular and intravascular compartments into the interstitial spaces. This results in hypovolaemia and oedema of the burn area. It is this fluid shift that causes hypovolaemic shock, which is also referred to as 'burn shock' (see Figure 16.9). Inflammatory mediators and stress hormones are released and start a cascade of events that, if left untreated, can lead to multi-organ failure.

Shock is the inability of the body to adequately deliver oxygen and nutrients to the body and remove cellular waste. Symptoms of hypovolaemic shock are similar to those of shock following haemorrhage: decreased blood pressure, cardiac output, urine output and plasma volume, and an increase in pulse rate along with an increase in systemic vascular resistance leading to decreased peripheral blood flow. Haematocrit and haemoglobin are elevated despite adequate fluid resuscitation due to the haemoconcentration of intravascular fluid. Red blood cells are haemolysed as a result of the burn and inflammatory mediators are released after a major burn injury. After fluid balance is restored, haematocrit levels usually return to normal.

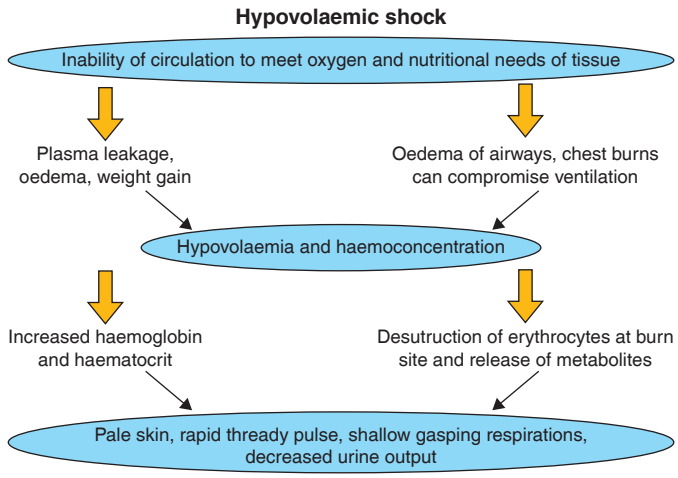


FIGURE 16.9 ■ Hypovolaemic shock algorithm

Source: Royal Perth Hospital.

Water, sodium and protein (albumin) leak through the less selectively permeable membranes of vessels to the interstitial spaces, causing oedema in non-burned tissue. There is also insensible fluid loss through the body surfaces that have been burned. Blisters and wound exudate are a result of this fluid shift. Potassium and sodium also move from the vessels and remain until the fluid shift resolves. Haemolysed red blood cells release potassium into the extracellular spaces (Knighton & Fong, 2008).

Towards the end of the resuscitative phase, as long as fluid replacement has been adequate, the membranes become selectively permeable again and leakage stops. Fluid is reabsorbed from the interstitial compartments into the intravascular compartment. The blood pressure rises as cardiac output increases and urinary output improves. This is usually around 24 hours post burn. This stage is known as the diuresis phase, with low specific gravity of urine. Potassium is slow to return to normal and hypokalaemia can occur, depending on the amount of potassium lost to the vascular space. Sodium can remain low due to sodium loss via the urine. The person is discouraged from drinking water and further depleting their sodium levels. During the diuresis phase the extra cardiac workload may predispose the older person or the person with cardiovascular disease to fluid volume overload. It is essential that the person is monitored and intravenous fluid is decreased and titrated to urine output and specific gravity.

Cardiovascular system

Major burns are manifested by widespread effects on the cardiovascular system. Increased vascular permeability and myocardial dysfunction as well as hypovolaemic shock, if left untreated, can result in a hyperdynamic/hypermetabolic state with an increased oxygen consumption. Improving cardiac output and oxygen delivery during the fluid resuscitation phase greatly improves the chances of survival (Jandziol & Hayes, 2008). Circumferential burns and oedema reduce blood flow to the extremities and if not treated can lead

to necrosis of a limb or digit. An escharotomy may be performed to a limb or the torso when there are circumferential burns. This should be done in consultation with the burns surgeon. Blood viscosity is initially increased due to cell destruction and fluid shifts within the body. Damage to skin structures results in microcirculation impairment.

CARDIAC RHYTHM ALTERATIONS Burns of more than 40% TBSA cause significant myocardial dysfunction, with a decrease in myocardial contractibility and cardiac output. These changes, which occur prior to a decrease in plasma volume, are believed to be due to the release of mediators, hormones and oxygen free radicals as a result of the burn and from ischaemic myocardial cells. Electrical burns often result in cardiac arrhythmias or cardiopulmonary arrest caused by heat damage to the myocardium or electrical interference with cardiac electrical activity (Jandziol & Hayes, 2008).

PERIPHERAL VASCULAR COMPROMISE Circumferential burns to the torso or extremities can result in **compartment syndrome** as a result of progressive development of arterial compression resulting in decreased blood supply to the area. Circulation to the extremities may be further impaired by oedema formation, inadequate fluid resuscitation and constrictive dressings or bandages. Limbs with circumferential burns should be elevated when not contraindicated and people with torso burns should be monitored for respiratory difficulties. Escharotomy may be required after consultation with the burn surgeon.

Respiratory system

Pulmonary damage may result from either direct inhalation injury or as part of the systemic response to the injury. The injury may range from mild respiratory inflammation to massive pulmonary failure, such as acute respiratory distress syndrome. Exposure to heat, toxic by-products of burned substances—for example, plastics—and smoke initiates the pathophysiological process associated with inhalation injury.

Inflammation occurs at localised sites within the airway and is manifested as hyperaemia. As a result, cells are destroyed and the bronchial cilia are rendered inactive. Because the mucociliary transport mechanism no longer functions, the person may develop bronchial congestion and infection.

Interstitial pulmonary oedema develops secondary to the escape of fluid from the pulmonary vasculature into the interstitial compartment of the lung tissue. Surfactant is inactivated, resulting in atelectasis and alveolar collapse. Sloughing of the damaged and dead lung tissue occasionally produces debris that may lead to complete airway obstruction.

Upper airway (above the level of the larynx) thermal injury results from the inhalation of hot gases, steam or vapours from chemicals dissolved in water. Generally, they occur when the person has been in an enclosed space. Inhalation injury is suspected when the person has singed facial, scalp or nasal hair. Physical findings include the presence of soot, charring, oedema, blisters and ulcerations along the mucosal lining of the oropharynx and larynx. The changes to tissue are the same as for thermal burn and severity is proportional to the length of exposure, type of chemical or temperature/pressure of steam. The resulting oedema in the airway may peak within 12 to 36 hours post injury (Bourdeaux & Manara, 2008).

Signs of increasing respiratory distress include hoarseness, laboured breathing and stridor. Inflammatory mediators cause oedema and respiratory obstruction can develop. The oedema will occur despite adequate fluid resuscitation measures. If there is also skin burn to the neck the swelling from this burn can further impede breathing.

The lower airway is protected very efficiently by laryngeal reflexes, so thermal injury below the larynx is not common and is usually only after exposure to extreme heat. However, when it does occur, it is typically associated with the inhalation of the products of combustion. Products containing sulfur, phosphorus or nitrogen, and compounds containing carbon, produce toxic by-products such as hydrogen chloride, hydrogen fluoride and ammonia. Acids and alkalis are released when these compounds come into contact with the moist respiratory mucosa, resulting in chemical burns. A classic finding is sputum containing soot or carbon particles which can damage alveoli (ANZBA, 2013; Bourdeaux & Manara, 2008).

Carbon monoxide, a common asphyxiate, is a colourless, tasteless, odourless gas that has a 200 times greater affinity for haemoglobin than does oxygen. It displaces oxygen to bind with haemoglobin, forming carboxyhaemoglobin. As a result, the decrease in arterial oxyhaemoglobin produces tissue hypoxia. Carbon monoxide impairs both oxygen delivery and cellular oxygen use. The clinical manifestations of carbon monoxide poisoning range from mild visual impairment to coma and death (see Table 16.6). Carbon monoxide poisoning should be suspected if the burn was in an enclosed space and the person is unconscious (Bourdeaux & Manara, 2008; Kealey, 2009).

Cyanide gas is released when plastics, polyurethane, nylon or silk are burned. The resultant production of cyanide gas affects cellular respiration. The brain and heart are most vulnerable to cyanide poisoning. Cyanide directly interferes with a cell's ability to utilise oxygen in metabolism. Cyanide poisoning should be suspected when the person shows signs of lactic acidosis regardless of adequate fluid resuscitation.

TABLE 16.6 Carbon monoxide intoxication

CARBOXYHAEMOGLOBIN (%)	SYMPTOMS
0–15	None (smokers, long-distance truck drivers)
15–20	Headache, confusion
20–40	Nausea, fatigue, disorientation
40–60	Hallucination, ataxia, syncope, convulsions, coma
> 60	Death

Source: Australian and New Zealand Burn Association (ANZBA) (2013). *Emergency Management of Severe Burns.* Course manual (version 17). © Australian and New Zealand Burn Association Ltd. Reproduced with permission.

FAST FACTS

Manifestations of cyanide poisoning

- Headache
- Dizziness
- Seizures
- Tachycardia
- Fatal arrhythmias

Gastrointestinal system

Major burns cause a generalised response in the body that affects all organs, including the gastrointestinal tract. Blood flow to the gut is decreased as part of the effect of hypovolaemic shock. Translocation of gut bacteria due to the disruption of the mucosa increases the risk of sepsis in the person with burns. This process is believed to be one of the mechanisms causing systemic sepsis and multiple-organ dysfunction syndrome (Magnotti & Deitch, 2005).

Other effects include malnutrition, hypermetabolism and splanchnic hypoperfusion as a result of intravascular fluid loss. Early enteral feeding can influence some of the gastrointestinal problems, including prevention of paralytic ileus and stress gastritis. The hypermetabolic response can lead to a catabolic state, which further decreases wound healing and increases the risk of infection. Maintaining blood flow to the gut is vital in preventing multi-organ failure. Maintaining the body's haemodynamics through fluid resuscitation is imperative to support gut blood flow (Singh et al., 2007).

Stress ulcers, also known as **Curling's ulcers**, are acute ulcerations of the stomach or duodenum that may form following the burn injury. Abdominal pain, acidic gastric pH levels, haematemesis and malaena may indicate a gastric ulcer. However, this is a less common presentation in contemporary medicine with the advances in burn management and the implementation of fluid resuscitation, early enteral feeding and antacids (Colon, Schlegel & Chung, 2012).

Urinary system

During the early stages of the burn injury, renal blood flow and glomerular filtration rates are greatly reduced as a result of the decreased intravascular blood volume and the release of antidiuretic hormone by the posterior pituitary. Urine output decreases and serum creatinine and blood urea nitrogen increase.

Dark-brown concentrated urine may indicate myoglobinuria or haemoglobinuria, the result of underlying muscle damage or the release of large amounts of dead or damaged erythrocytes after a major burn injury, particularly following an electrical injury. When large amounts of these pigments are released, the liver cannot keep pace with conjugation and the pigments pass through the glomeruli. The pigments can occlude the renal tubules and cause renal failure, especially when dehydration, acidosis or shock is also present. Adequate fluid resuscitation aims to prevent renal tubule damage as well as renal failure. An indwelling catheter is inserted to enable accurate monitoring of urine output.

Immune system

Major burn injury can cause impairment of the immune system. The protection function of the skin is compromised, particularly in burns where there is a large TBSA. The amount of circulating immunoglobulin is decreased and the WBC function is also impaired. Part of the cascade of events that occurs following a major burn injury is the release of inflammatory cytokines. This release impedes the normal function of neutrophils, monocytes and lymphocytes. This series of events increases the person's risk of infection.

Metabolism

Major burns induce high energy expenditure, mediated by increased production of catecholamines, glucocorticoids and glycagon (Bonet, Marquez & Seron, 2011). The body's high demand for energy cannot be fully reversed despite adequate nutrition and medical management. Hyperglycaemia is also common, with evidence suggesting that pharmacological management of elevated blood glucose levels is required.

Two distinct phases characterise the body's metabolic response to the burn injury. The ebb phase is manifested by decreased oxygen consumption, fluid imbalance, shock and inadequate circulating volume. The gut function also slows down.

A second phase, the flow phase, occurs when adequate burn resuscitation has been accomplished and is dependent on adequate metabolic response mediated by adrenal cortical steroids.

This phase is characterised by increases in cellular activity and protein catabolism, lipolysis and gluconeogenesis. Hypermetabolism persists until after wound closure has been accomplished and may reappear if complications occur (Cochran, Jeschke & Collins, 2013).

CONSIDERATION FOR PRACTICE

An increased body temperature, without other manifestations of infection, is not indicative of infection in people with large burn wounds (in which the hypermetabolic response resets the core temperature to a higher level).

INTERPROFESSIONAL CARE

The burn team is composed of an interprofessional group of healthcare providers who together plan and implement the treatment of the burn-injured person during the acute and rehabilitative phases. The burn team consists of the nurse, physician and/or surgeon, physiotherapist, dietitian, occupational therapist, clinical psychologist and social worker. The team members meet regularly to discuss person progress and to determine collaboratively the most effective regimen of care and psychosocial support.

Phases of interprofessional care

As outlined earlier in this chapter, the clinical course of treatment for the burn person is divided into three phases, which overlap and begin on the day of the burn injury; they are the resuscitative phase, the acute wound healing phase and the rehabilitative phase.

Although these phases are useful predictors of the clinical needs of the burned person, it is important to recognise that the process of burn injury is dynamic and that the clinical phase is not clearly delineated. Assessment and management of the

burn-injured person are ongoing processes determined by the clinical picture; they last throughout the course of treatment. During each phase, different groups of nurses, medical practitioners and other healthcare specialists collaborate to manage the person's recovery.

Although many burn injuries are treated in local healthcare facilities, in many healthcare organisations throughout Australia there are guidelines for determining whether the person should be transported to a burns centre for interdisciplinary approaches to treatment and rehabilitation (see Box 16.3).

The resuscitative phase has already been discussed on previous pages. It includes pre-hospital care.

ACUTE WOUND HEALING PHASE The acute phase begins with the start of diuresis and has no defined end point as each person requires individual goal settings to determine progress. During this phase, wound care management, nutritional therapies and measures to control infectious processes are initiated. Excision and grafting of any wounds that require surgery is performed as soon as possible after injury. Enteral nutritional feeding to address kilojoule needs resulting from extensive energy expenditure are started in the resuscitative phase and continue through to discharge—and beyond, for some people. Measures to combat infection are implemented from the day of the burn injury and must continue until the wounds are healed.

Pain management constitutes a significant segment of the care plan throughout the clinical course of the burn-injured person. Adequate analgesia must be prescribed and given on a regular basis to maximise person comfort and to reduce the anxieties associated with wound debridement and intensive physiotherapy (Patterson, Tininenko & Ptacek, 2006).

REHABILITATIVE PHASE The rehabilitative phase begins on the day of the burn injury and ends when the person returns to the highest level of health and function they can achieve. During this phase, the primary focus is the biopsychosocial adjustment of the person, specifically the prevention of contractures and scars. The person's successful resumption of work, family and social roles is facilitated through physical, vocational, occupational and psychosocial rehabilitation. The person is taught to perform range-of-motion exercises to enhance mobility and support injured joints.

It is imperative to remember that while there have been three phases of interprofessional care identified, collaborative discharge planning commences on the day the person is admitted to the burns unit. No interprofessional team role is more important than another.

Fluid resuscitation formula

Fluid resuscitation is the administration of intravenous fluids to restore the circulating blood volume during the first 24 hours post burn injury. The ANZBA guidelines (2013) state that a burn injury of 20% TBSA and over results in a generalised sequestration of large amounts of fluid from the intravascular space to the interstitial spaces. Combined with oedema and insensible loss, plasma volume is depleted, resulting in intravascular hypovolaemia; left untreated, this can end in multi-organ failure.

Several formulas may be used to replace fluid loss. The ANZBA (2013) recommends the use of the Parkland (Baxter) formula (see Box 16.4).

Respiratory management

On admission to the emergency department, several baseline assessments of respiratory status must be obtained: chest x-ray study, arterial blood gases (ABGs), vital signs and carboxyhaemoglobin (COHb) levels, which should be ascertained from arterial blood. A portable breath analyser can be us d to detect carbon monoxide levels. It is important to remember that high oxygen concentrations have usually been administered in transit and so accurate levels of COHb are difficult to assess. Preparation for possible intubation should always be done in case of airway obstruction. The primary treatment plan is oriented towards preventing

BOX 16.4 Calculation of Parkland formula

Formula

2–4 mL Hartman's solution × kg (body weight) × %TBSA = total fluid requirements for first 24 hours after burn (commencing from time of injury)

Give:

½ (50%) total over first 8 hours

¼ (25%) total over next 8 hours

¼ (25%) total over next 8 hours (makes a total of a 24-hour period)

Example

For a 70-kg person with a 50% TBSA burn

4 mL x 70 kg x 50% TBSA burn = 14 000 mL

= 14 L in 24 h

50% of total in first 8 h = 7000 mL (875 mL/h)

25% of total in second 8 h = 3500 mL (436 mL/h)

25% of total in third 8 h = 3500 mL (436 mL/h)

- For adults the usual fluid maintenance of 2 L/24 hours will also be required (e.g. 2000 mL ÷ 24 hours = 83 mL/h).
- This requires a total of 436 mL + 83 mL = 519 mL/h over the first 8 hours.
- Fluid resuscitation commences as soon as possible following injury.

(Paediatric fluid maintenance requirements are determined subject to the child's weight.)

Enteral feeding volumes are taken into account when determining maintenance fluid volumes.

Formulas are guidelines. Fluid is administered at a rate to produce 0.5–1.0 mL/kg/h—that is, 30–50 mL of urine output per hour. In cases of an electrical burn, then urine output is maintained at 1–2 mL/kg/h.

Blood pressure and pulse rates should not be used in isolation to measure fluid resuscitation adequacy as an adult with a major burn is in a hypermetabolic state and will probably be tachycardic, anxious and in pain.

All formula guidelines are used as starting points and fluids are titrated according to the person's physical response. Hourly urine output is often used as one indicator of effective fluid replacement. Specific gravity of the hourly urine should be monitored.

Source: Fiona Stanley Hospital Burn Service.

atelectasis and maintaining alveolar oxygen exchange. The following interventions should be initiated:

- Maintain the head of the bed at 30 degrees or greater to maximise the person's ventilatory efforts (once cervical spine is cleared). Turn the person side to side every 2 hours to prevent hypostatic pneumonia.
- To keep airway passages clear, encourage the person to use incentive spirometry hourly and help them perform coughing and deep-breathing exercises regularly. Encourage expectoration of sputum. Monitor and document respiratory rate hourly depending on the person's condition. Measure oxygen saturation using pulse oximetry. In a person with high COHb levels the oxygen saturation will be inaccurate as no determination between oxygen and COHb can be made by the pulse oximeter.
- If respiratory status declines, prepare the person for intubation. Endotracheal or nasotracheal intubation may be used. If the person has suffered nasolabial burns, the endotracheal route may be preferred. Intubation is used for short-term ventilatory management. For long-term ventilatory management (i.e. greater than 3 weeks), a tracheostomy is performed.
- Humidified oxygen is administered to help prevent the drying of mucosa and tracheal secretions. Ambient air or oxygen flow is based on ABG results. The person may be placed on a face mask, steam collar, T-piece, mechanical ventilation with positive end-expiratory pressure, pressure-support ventilation or high-frequency jet ventilation. The person's room will be maintained at an ambient temperature at all times because hypothermia will delay wound healing, cause discomfort and affect consciousness. Breathing in warm air will assist the person to breathe comfortably. The goal of all therapies is to maintain adequate tissue oxygenation with the least amount of administered oxygen necessary.
- Medications to dilate constricted bronchial passages may be administered intravenously or by inhalation to control bronchospasms and wheezing. Mucolytic agents liquefy tenacious sputum and aid in expectoration.
- An arterial line may be inserted for continuous assessment of ABGs. Pulmonary artery pressure catheters may be inserted to measure pulmonary vascular resistance (PVR), pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP) and mixed venous oxygen saturation (SvO_2). The PVR and PAP rise in the presence of hypoxia. The SvO_2 is the average percentage of haemoglobin bound with oxygen in the venous blood and reflects overall tissue utilisation of oxygen. Pulse oximetry monitors arterial oxygen saturation levels. The person requiring this level of invasive monitoring may be in a critical care unit.
- Administer intravenous analgesia as ordered.

Intravenous analgesia is the preferred route of administration because of decreased gastric absorption. Intramuscular analgesia will not be absorbed from burned or oedematous areas and results in a build-up of analgesia in the tissues. In the diuresis phase the analgesia is then released and the person could receive too much analgesia at once. Adjunct medications such as anxiolytics should be used to help calm the person.

After stabilisation in the emergency department, the person is transferred to the critical care unit or a specialised burns unit.

Diagnosis

The following diagnostic tests are used to evaluate the person's progress and to modify intervention strategies. The monitoring process should depend on the extent and depth of the burn, the presence of inhalation burn, other injuries, co-morbidities and age.

- *Urinalysis* indicates the adequacy of renal perfusion and the person's nutritional status. In catabolic states, nitrogen is excreted in large amounts into the urine. Nitrogen balance is a measure for nutritional support required and should be done in conjunction with serum protein measurements. Inactivity causes muscle wasting and an increased nitrogen excretion. An indwelling catheter is inserted and should be maintained until the person is medically stable and able to use a urinal or bedpan. During the resuscitative phase, hourly specific gravity tests should be done as a clinical indicator of fluid replacement adequacy.

  Monitoring of urine output should continue until after the diuresis phase and in conjunction with hospital protocols. Loss of plasma protein and dehydration lead to proteinuria and elevated urine specific gravity. Glycosuria is a transient development following major burn injury; it can indicate a need to adjust the nutritional program.
- *Myoglobinuria*, which manifests as a dark-brown or wine-coloured urine, signals the development of acute tubular necrosis. It usually occurs following an electrical injury, and intravenous fluid should be maintained to assist in clearing the myoglobinuria.
- *The full blood count* is monitored regularly. Haematocrit is elevated secondary to haemoconcentration, haemolysis of RBCs and fluid shifts from the intravascular compartment. Haemoglobin is decreased secondary to haemolysis. Changes to leucocyte numbers occur in relation to the burn and further changes in response to medications or sepsis.
- *Serum electrolytes* are monitored regularly as clinically indicated during the resuscitation phase. They are a more accurate assessment of successful fluid resuscitation attempts than urine output. Frequency of tests should be done in accordance with the hospital policy. Sodium levels are decreased secondary to massive fluid shifts into the interstitium. Potassium levels initially are elevated during burn shock, as a result of cell lysis and fluid shifts into the extracellular space. Potassium levels decrease after burn shock resolves, as fluid shifts back to intracellular and intravascular compartments.
- *Renal function* test results are closely monitored. Blood urea nitrogen (BUN) is elevated secondary to dehydration and if the enteral feeding is high in protein. Creatinine is elevated in the presence of renal insufficiency. It is used as an indicator of rhabdomyolysis following electrical injury.
- *Total protein, albumin, transferrin, prealbumin, retinol binding protein, alpha-1-acid glycoprotein* and *C-reactive protein* indicate protein synthesis and nutritional status.

Because of the fluid shifts that occur during the early stages of the burn injury, they are more useful markers during the rehabilitative phase of care.

- *Creatine phosphokinase (CPK)* is elevated following an electrical burn, secondary to extensive muscle damage.
- *Serial ABGs* indicate the presence of hypoxia and acid–base disturbances and indicate the person's responses to changes in oxygen therapies. The burn-injured person may demonstrate elevated or lowered pH, decreased PCO_2 and PO_2, and low to normal bicarbonate levels. Their ABGs are a more accurate estimation of COHb as pulse oximetry cannot distinguish between oxygen and carbon monoxide.
- *Pulse oximetry* allows continuous assessment of oxygen saturation levels.
- *Serial chest x-ray studies* document changes within the first 24 to 48 hours that may reflect the presence of atelectasis, pulmonary oedema or acute respiratory distress syndrome (ARDS), also referred to as severe pulmonary congestion.
- *Serial 12-lead electrocardiograms (ECGs)* are necessary to monitor the development of arrhythmias, especially those associated with hypokalaemic and hyperkalaemic states.

Medications

PAIN CONTROL Burns are painful. In the resuscitative phase of care, intravenously administered narcotics are the best means of managing pain. Intravenous narcotics must be administered subject to hospital policies. Burn treatments can also produce high levels of anxiety, necessitating the use of anxiolytic agents such as midazolam and lorazepam (Montgomery, 2004). Anxiolytics are especially useful when administered 1 hour before wound care. During the acute phase, narcotics are administered around the clock on a regular basis to decrease pain that occurs at rest.

Patient-controlled analgesia (PCA) enhances the person's ability to cope with pain. The oral, subcutaneous or intramuscular route of administration should be avoided until haemodynamic stability and unimpaired tissue perfusion return. Some hospitals have access to specialty pain services and these should be involved to ensure the person receives the most appropriate analgesia to suit their needs. If the person has adequate analgesia at all times, they will be less anxious and more receptive to all care provided by occupational therapists, physiotherapists and nurses.

Alternative forms of pain and anxiety relief should be taught as an adjunct to pharmacological measures. Distraction, self-hypnosis, guided imagery and relaxation techniques are helpful adjuncts in managing pain and coping with loss. See Chapter 8 for a discussion of strategies for managing pain.

On admission it is important to ascertain medications, either prescribed or illicit, taken by the person prior to the burn injury, as these may further complicate the treatment regimen. Drugs that affect any of the major body systems or cause mood alterations will need to be factored into the treatment plan. As part of the early assessment, obtain and document blood levels of therapeutic pharmaceutical agents and mood-altering substances (Gamst-Jensen et al., 2014).

TETANUS PROPHYLAXIS If the person's immunisation status is in doubt, tetanus toxoid is administered intramuscularly early in the acute phase of care to prevent *Clostridium tetani* infection. A thorough health history should be taken to ensure the person has had the primary three doses of tetanus vaccination. If not, or the last booster was 20 years or more ago, then they may require immunoglobulin, not a booster.

ANTIMICROBIAL AGENTS Systemic infection is a leading cause of death in people with major burns. Gram-positive organisms such as *Staphylococcus* and *Streptococcus* colonise the burn surface during the first week post burn; gram-negative enteric organisms become more common with longer periods of hospitalisation. Prophylactic systemic antibiotics are not used in controlling burn wound flora because there may be poor delivery to the burn due to decreased blood flow. To aid in the prevention of infection the person is washed in chlorhexidine liquid soap (Main, 2008). Topical antimicrobial therapy in the form of wound dressings is used to manage the wound bioburden. Of the many antimicrobial agents available, bacteriocidal silver-impregnated dressings are recommended—for example, nanocrystalline dressings or impregnated dressings may be used. Despite antimicrobial therapy, people with major burns have a greater risk of sepsis and septic shock. Systemic antibiotics are commenced when a definitive diagnosis of infection is made. A biopsy of the area may be taken to identify infective organisms. Surface swabbing for microbiology may not be accurate in determining the organism involved (World Union of Wound Healing Societies, 2008).

FAST FACTS

Local signs of burn wound infection

- Increased redness greater than 2 cm erythema around wound
- Increase in pain
- Increased sloughing of burn tissue
- Increased oedema
- Conversion of burn to a deeper burn
- Black or brown areas of discolouration

Systemic antimicrobial therapy is used on induction for surgery and may be administered postoperatively. The therapy is discontinued as soon as the person's haemodynamic status returns to normal, usually within the first 24 hours. In the long-term treatment of identified infectious processes, drug administration is limited to the least amount of time required to eradicate the infection.

Topical antimicrobial agents *Silver* Silver has a long history of use in wound care and is documented in use as early as the 19th century. It has a broad antibacterial activity. With the advent of antibiotics its use declined. It had a resurgence in the 1960s and again in current medicine, particularly in the use of burns. While there is a potential for resistance to silver in wounds, there has not been any definitive research to confirm resistance to silver-impregnated dressings (Vermeulen et al., 2009; Vlachou et al., 2007).

Silver sulfadiazine Silver sulfadiazine (Flamazine™) is a silver and sulfonamide cream and has been used since the 1960s as a topical antimicrobial for burns. Originally called SSD, the product no longer has the chlorhexidine component and the name has changed to reflect this. It has been shown to be effective against anaerobic and aerobic bacteria. The silver ion binds with the DNA of the bacteria. It is bactericidal and is easy to apply, and for some people it is soothing. However, it is contraindicated in instances of sensitivity to silver and sulfur drugs (Fuller, 2009). It is not sustained release and therefore requires daily dressing changes (Vermeulen et al., 2009).

Silver-impregnated dressings There is an increasing variety of silver-impregnated dressings and they all differ in the type and amount of silver they contain. In the management of burns, high-dose, sustained-release nanocrystalline silver formulations (Acticoat®) are preferred. Nanocrystalline silver dressings are bactericidal and have been shown to be effective against anaerobic and aerobic bacteria. Nanocrystalline silver dressings can leave a temporary blue staining of the skin. They are applied moistened with sterile water (not saline). Water compresses are then placed over the dressing and they are secured with a roller bandage or tubular stretch bandage.

Research shows that the most effective topical agents are those that act against the major pathogens responsible for causing burn wound infection, achieve levels of concentration sufficient to decrease microbial colonisation, are rapidly excreted or metabolised, are non-toxic and are easy to use (Fong & Wood, 2006).

Role of the nurse

The role of the nurse in the care of the burn-injured person encompasses all aspects of general nursing care and, in particular, wound management, during which the nurse will focus on the following:

- administration of analgesia prior to treatment
- adherence to hospital protocol regarding wound cleansing and wound treatments
- ongoing assessment of the wound and healing status
- education for the person and their family to promote optimal wound healing outcomes.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Discuss procedure with the burned person and strategies for maximising comfort at dressing changes.
- Advise the person to take analgesia prior to each dressing and on a regular basis to maintain comfort.
- Educate the person about the importance of physiotherapy and performing given range-of-motion exercises while wounds are healing.
- Educate the person about the importance of good nutrition—in particular, protein and carbohydrates to aid healing.

Role of the surgeon

The goal of any wound closure surgery is to provide the best possible cosmetic and functional outcome for the burned person. The current practice of early excision and grafting has improved the outcome for burns and has decreased mortality. Surgery is done once the person is cardiovascularly stable and after the resuscitation period. Not all wounds require the same surgical intervention. A small burn may be excised and directly closed, requiring only suture line care. Surgical management of burn injuries includes escharotomy, debridement of non-viable tissue and harvesting of autologous (person's own) split skin grafts (SSGs). Full-thickness skin grafts (FTGs) are used on functional areas if skin is available. The FTGs contract less than SSGs and in some of the deeper burns a tissue flap may be required to repair the defect.

Surgeons are mindful of the scar left by the donor site and will select an area of low exposure and good colour match when possible. The scalp may be used when there is little skin available for harvesting as it heals quickly and hair regrowth covers the donor site. The development of the dermal regeneration template Integra® has allowed earlier debridement of large areas of full-thickness eschar. Cultured epithelial autografts (CEAs) are also used by some surgeons when there is limited skin available for harvesting or to improve the meshed appearance of the scar from a meshed graft (Sood et al., 2015).

ESCHAROTOMY

When the burn eschar forms circumferentially around the torso or extremities, it acts as a tourniquet, impairing circulation. Left unchecked, the affected body part becomes gangrenous.

To prevent circumferential constriction of the torso or extremity, an **escharotomy** is performed by the physician with a scalpel or by electrocautery (see Figure 16.10). A sterile surgical incision is made longitudinally along the extremity or the trunk to release taut skin and allow for expansion caused by oedema formation. In the first 24 hours following the procedure, the incision should be gently packed with a calcium alginate dressing and covered with a nanocrystalline silver compress. After 24 hours, the site may be treated with a direct application of a topical bactericidal agent. See Box 16.5 for nursing implications for care of the person undergoing escharotomy.

SURGICAL DEBRIDEMENT

Surgical debridement is conducted when there are full-thickness burns, extensive necrosis, infection or prior to skin grafting. Other methods of debridement are outlined in Chapter 15. The surgical technique is dictated by the depth of burn. The wound is excised to the

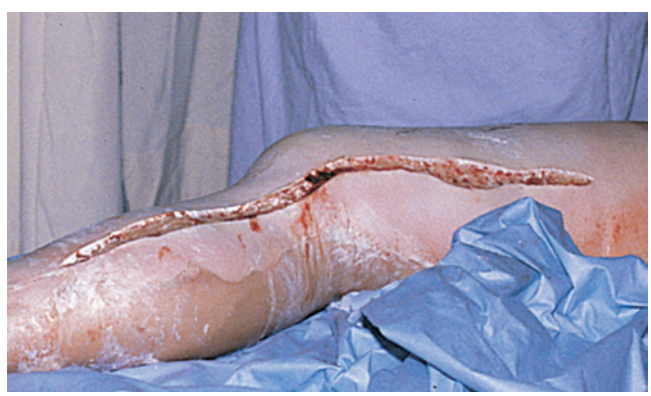


FIGURE 16.10 ■ Escharotomy. The surgical procedure consists of making an incision through inelastic burned skin to relieve compartment pressure

Source: Courtesy of *Dr* William Dominic.

BOX 16.5 Nursing implications for circumferential wound management

Care of the escharotomy wound

- Assess the extremity for reduced or absence of blood flow:
  a. Assess the extremity hourly for warmth, colour, sensation and capillary refill.
  b. Observe for evidence of numbness or tingling.
  c. Elevate the limb unless contraindicated.
  d. When dressing the escharotomy wound or limb with circumferential burns, leave fingertips and toes exposed for assessment purposes.
  e. Any applied bandages should not be tight.
- For circumferential burn wounds of the torso, assess for evidence of respiratory distress: elevate head of bed at least 30 degrees unless contraindicated.
- For circumferential burn wounds of the neck, assess for evidence of respiratory distress. Prepare the person for prophylactic intubation.
- Pack the escharotomy wound with calcium alginate dressings and dress the open wound with topical bactericidal silver dressing as ordered.

level of fascia (**fascial excision**) in full-thickness skin injury or sequential removal of thin slices of the burn wound to the level of viable tissue (tangential excision) is undertaken in partial-thickness skin injury. Because fascial excision sacrifices potentially viable fat and lymphatic tissue, its use is reserved for people with extensive or full-thickness burns. The most common technique used is electrocautery with cutting and coagulating current capabilities. Tangential excision is performed with the use of a dermatome. Shallow burns and some of moderate depth bleed briskly after one slice. If bleeding does not occur, the procedure is repeated until a viable bed of dermis or subcutaneous fat is reached. Following surgical debridement, haemostasis is vital to facilitate the repair of the wound surface. The debrided wound can be closed using traditional skin grafts in isolation or in association with tissue-engineered solutions. Once the wound has been dressed the person is returned to the burns unit and appropriately positioned and splinted, subject to the location of the injury and donor sites.

TYPES OF SKIN GRAFTS Autograft is the surgical removal of skin from one site on the individual (known as the donor site) and relocated to another site on the same individual. The donor site is allowed to heal by secondary intention.

Cultured epithelial autograft (CEA) Cultured epithelial **autografting** is a removal of autologous keratinocytes from a skin biopsy of approximately 2 cm from an unburned site on the person's body. The biopsy should be taken within 24 hours of the burn. The separated keratinocytes are then placed in a medium containing epidermal growth factor. Originally the technique involved placing the cells in a medium where, over a 5- to 7-day period, they expand to 50 to 70 times the size of the initial biopsies. The cells are again separated out and placed in a new culture medium for continued growth. With this technique, enough skin can be grown over a period of 3 to 4 weeks to cover an entire human body. The cells may be prepared as sheets attached to petroleum gauze backing, or in a suspension that is applied to the burn wound site. Problems with infection and lack of attachment have occurred. These problems have been reduced by using cells harvested at the time of the operation, and immediately applying them to the wound. The skin cells undergo no laboratory expansion and adhere directly to the wound bed. Using this more recent technique, the skin biopsy is placed in a ReCell® kit, separated and placed in a culture medium for growth. The cells are ready to use within 15–20 minutes. The CEA results in a smooth finish but is prone to shearing and blistering as cell layers are 10–15 cells thick (Knighton & Fong, 2008; Wood et al., 2012).

Homograft, or **allograft**, is a temporary skin substitute and has some advantages when wound coverage is needed and for the body to behave as if it has skin. Human skin that has been harvested from cadavers is stored in a 'skin bank' until required. The development of methods to achieve prolonged storage of frozen, viable skin has increased the use of this dressing; however, its short supply, infection risk and expense still pose problems. It is manufactured as strips cut to the pattern of the burn and applied using sterile technique. Under normal circumstances, a homograft is rejected within 14 to 21 days following application.

Heterograft, or **xenograft**, is another kind of temporary wound coverage. Only heterograft from porcine dermis is available. Although fresh porcine heterograft is available at some centres, frozen heterograft is much more commonly used. Once applied, heterograft appears to undergo early softening and lysis from enzymatic action from the wound. As a result, frequent changes of the heterograft dressing are necessary. Because of the high infection rates associated with this dressing, silver-nitrate-treated porcine heterograft has been developed to retard microbial growth. Temporary skin coverage does not vascularise but the underlying wound bed may epithelialise. Amniotic membranes are also used, particularly for face burns, but they do not reduce healing times (Lee et al., 2012).

Bioengineered tissue substitutes The multiple problems associated with the use of allograft and heterograft have driven the development of synthetic materials and composite material biological dressings. One such material is Biobrane®, a composite material consisting of nylon mesh bonded to silicone coated with bovine collagen, which has proved successful in the temporary coverage of partial- and full-thickness burns. Whereas Biobrane® adheres well to moderately clean wounds, it cannot adhere to grossly contaminated wounds. Biobrane® dressing is supplied in various sizes and is cut to fit the wound site and secured with staples or tape or skin closure strips (e.g. Steri-Strips®). It spontaneously separates from the wound when the underlying tissue heals (Pham et al., 2007).

If dermal thickness is lost, as in deep partial-thickness or full-thickness burns, several products can serve as a dermal replacement. Integra® is a synthetic dermal substitute made of bovine collagen and shark glycosaminoglycans, and Alloderm®

is human cadaver allograft dermis processed such that it is non-immunogenic. These products are placed in the wound, and split-thickness autografts are then placed over the dermal replacement. These products are used to provide wound coverage, reduce pain and facilitate healing.

Dermal regeneration template (Integra®) Dermal regeneration template provides both dermal and epidermal characteristics. This is a manufactured template that, when placed on to a viable wound bed following debridement of full-thickness or deep partial-thickness burns, forms a matrix resembling dermis. It is of greatest use when autografting is not a possibility and is also used in scar revision. It has two layers and is secured with staples. The dermal layer is biodegradable and causes the body to regenerate a new dermal layer. The silicone layer stays intact for 3 weeks and then is removed and autologous grafting is done. This is a two-stage process. Integra® becomes infected easily, resulting in a non-viable product that must be removed immediately. Because of this, meticulous nursing care of Integra®-covered wounds is required in order to prevent infection and loss of graft. Integra® is dressed daily with a silver-impregnated dressing (e.g. Acticoat®) in some hospitals while the silicone layer is separating. The silicone layer is removed prior to grafting. In some cases, cultured epithelial autograft (CEA) or ReCell® (in conjunction with split skin grafts) will be used after the silicone layer is removed. Nursing care is specific for this template and shearing must be prevented, particularly in the first week post application (Chester & Papini, 2004; Pham et al., 2007).

Split skin grafts Skin grafts can be split thickness (SSGs)—thin, intermediate or thick. They all contain epidermis and variable depths of dermis. Thin SSGs contract within the first few months. They contain no skin appendages, and vascularisation occurs readily; while thick SSGs have less contraction and usually contain some hair follicles.

The SSG can be laid in sheet form or meshed to allow for a greater area to be covered. These grafts leave a 'meshed' scar pattern (see Figure 16.11) and in some instances surgeons will use cultured epithelial autograft (CEA) or ReCell® to reduce the meshed look. Initially the graft is supplied by nutrients from the wound bed: the phase of imbibition. The graft will not take if the wound bed is insufficiently debrided, or slough, haematoma or excess exudate are present. Dressings must prevent shearing of the graft. Next, neovascularisation takes place; during this phase, blood vessels connect with capillaries in the graft. This is followed by the maturation phase when collagen forms between wound bed and graft. This last phase can take months to complete.

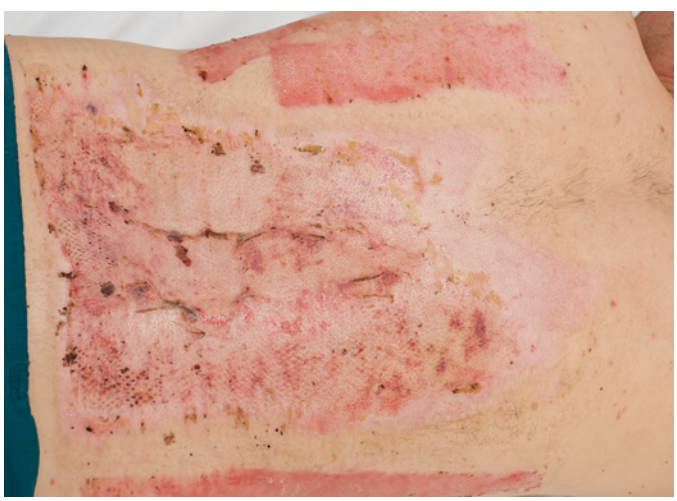


FIGURE 16.11 ■ Split skin graft on a back

Full-thickness skin grafts Full-thickness skin grafts (FTGs) involve removal of the whole epidermis and the dermis. The thickness of the FTG depends on the thickness at the donor site from where it is harvested. The graft is sutured to the recipient wound bed. Care must be taken to ensure the graft is in contact with wound bed or 'graft take' will not occur. Sometimes a 'tie over' dressing will be sutured over the top of the FTG to maintain the contact with the wound bed. The tie over is usually removed at the time of dressing change on about day 5 postoperatively or on the surgeon's instructions.

GRAFT CARE Grafts can be managed open with graft care attended to 1- to 4-hourly, depending on the surgeon's preferences or, more commonly, sealed under a bulky dressing for the first few days. The aim of graft care is to remove haematomas or exudate from beneath the graft to facilitate adherence. Small blisters are slit and exudate is expressed. Any wrinkles are flattened and edges prevented from rolling. Grafts can also be covered with paraffin gauze or silicone meshed dressings, which are then covered with a fluffed gauze filler to reduce any dead space in the wound defect and secured with a bandage system. This dressing is usually left sealed for at least 48 hours then removed. Ongoing burn wound management regimens are subject to the graft appearance. Grafts may also be sealed for up to 5 days with a negative pressure dressing.

DONOR SITE CARE A thin layer of donor skin is taken from the person with the use of a dermatome. Each surgical team has its preferred procedures for caring for donor sites and a range of dressings are used. Calcium alginate dressings are frequently used as they provide haemostatic and absorbent features. Donor sites generally take 2 weeks to re-epithelialise, and dressings are applied until healing.

FLAPS This procedure involves using a portion of skin with its intrinsic structures to repair a defect that is too extensive for an SSG or FTG. Flaps leave a scar at the point of injury, as well as the donor site scar. In burns, some flaps become non-viable due to decreased blood flow. There are many types of flaps; the surgeon decides which one to use depending on the defect and where it is located on the body.

Burn wound management

The outcomes of care for the person with a major burn depend in part on the prevention and treatment of infection. The goals of burn wound management are as follows (Honari, 2004):

- alleviate pain
- control microbial colonisation and prevent burn wound infection

- prevent burn wound conversion to a deeper burn
- achieve burn wound coverage as early as possible
- promote function of healing skin
- preserve function of the body part.

Burn wound management decisions are delayed until primary survey and lifesaving measures have been established. After first aid, burn wound management involves washing and covering the burn with a clean sheet for transfer. Care decisions are dependent on assessment of the depth and appearance of the wound and the dressing products available for the healthcare professional to use. Various dressing products suitable for burn wounds are discussed later in the chapter.

DEBRIDING THE BURN WOUND Burned tissue releases chemical mediators that stimulate phagocytosis in an attempt to digest necrotic tissue. Necrotic tissue that remains despite phagocytic action slows healing and prolongs inflammation. **Debridement** is the process of removing all loose tissue, wound debris and eschar (dead tissue) from the wound. The methods of debridement commonly employed in burn care are surgical (see above), mechanical (irrigation), and conservative sharp wound debridement (removal of loose non-viable tissue without pain or bleeding). Dressings that hydrate the wound will promote autolytic debridement.

During showering, non-viable tissue and slough can be removed while washing the burned area. Showers also promote a feeling of wellness for the person (Betts, 2003; Main, 2008). The person should be encouraged to do as much of the showering themselves as possible as this promotes independence and is good physical therapy. It may reduce the anticipation anxiety that arises when someone else is washing their burns. Hydrotherapy (in an immersion tank, a shower or on a spray table) is also used to cleanse the burn injury using a mild, non-perfumed, antimicrobial soap, to remove dead skin and separate eschar. The solution is then rinsed off with warm saline or tap water (Fernandez, Griffith & Ussia, 2006).

Body hair (except for eyebrows) may be shaved within the burn and to within 2.5 cm of the wound edges. This prevents debris building up around hair follicles, increasing the risk of infection. Shaving also allows for better washing of the burn and adherence of any dressing products. The edges of blisters or eschar are trimmed with sharp scissors. The wound is then covered with a topical antimicrobial agent according to hospital policy.

DRESSING THE BURN WOUND Once the burn wound has been cleaned and debrided, it will require a dressing. Moist wound healing principles and hospital policy on topical antimicrobials should be adhered to. Covering the burn helps to reduce the risk of infection and decreases pain.

All fingers and toes are wrapped separately. Dressings are held in place with roller bandages, retention tapes or tubular net. All care is taken not to restrict range of movement or circulation and to encourage person independence with activities of daily living.

TOPICAL NEGATIVE PRESSURE DEVICES Topical negative pressure devices consist of a foam or gauze dressing that is cut or folded to the wound shape and used to fill any defects. An occlusive, adhesive dressing seals the wound and tubing connects the dressing with the pump. Negative pressure systems reduce wound oedema, remove exudate and promote granulation and epithelialisation (DeSanti, 2005).

Role of the physiotherapist

Physiotherapists are primarily concerned with ensuring the respiratory system is not compromised after a burn injury. Lung compromise may impede oxygen delivery to the wound, slow wound healing and increase the risk of wound infection. To further facilitate oxygen transfer during the acute burn period, physiotherapists will focus on the reduction of oedema in order to improve the speed of wound healing and prevent burn conversion. Therapists recognise that impaired wound healing may lead to the development of hypertrophic scar, which may contribute to contractures after a burn injury. To prevent such long-term complications and assist with oedema reduction, people should be encouraged to exercise their burn-injured and non-burn-injured areas. As soon as possible following admission, the physiotherapist should prescribe and display active and self-assisted passive range-of-movement (ROM) exercises. The physiotherapist will perform passive ROM exercises for the person who is unable to do them independently. Ideally, an active exercise program is initiated and reinforced by the multidisciplinary team members. The program should involve a warm up and simple functional tasks, as well as specific exercises. Early ambulation is also part of the plan of care to reduce the risk of respiratory and circulatory complications. It is imperative that the person receives regular multimodal analgesia and pressure bandage support to facilitate these activities (van Baar et al., 2006).

Burned people must be maintained in positions that prevent or reduce the risk of contractures. Because flexion is the natural resting position of joints and extremities, early physiotherapy includes maintaining and correcting anti-deformity positions. Splints immobilise body parts and may have a role to play in preventing contractures of the joints, particularly if the person is unconscious in the critical care unit. Splints, if required, should be applied soon after the injury and removed according to schedules established by the therapist.

Physiotherapists facilitate postoperative recovery of the burned patient to prevent long-term loss of movement. The person should be encouraged to move and walk normally unless contraindicated following surgery. It is better that the person continues to move while the wounds are healing, rather than leaving it until their wounds are healed (Falder et al., 2009). Postoperative movement changes depending on the surgery type and specialist's preferences.

Role of the occupational therapist

Occupational therapists who work with individuals who have sustained a burn injury have two core areas of focus: function and scar management. These two areas are closely entwined, as a deficit in one area usually goes 'hand-in-hand' with the other, and vice versa.

The International Classification of Functioning, Disability and Health (ICF) represents the *functional impact* of illness

and injury. It defines function and disability in terms of the whole person rather than by illness or injury and looks beyond the medical to include other aspects of disability. It specifically addresses body structure and function, the activities people engage in, the interests they have and the environmental and personal factors that affect those experiences. This framework has a very useful application to the person who has sustained a burn injury as return to function must include the physical, psychological, relational and social impacts of the injury.

The return to functional independence is a journey that starts the day after injury. The contractile properties of the healing burn dictate early mobilisation to avoid scar contracture and this is achieved both through specific exercise programs and through the early resumption of self-care tasks (showering, dressing, feeding and hand function). The occupational therapist will design a functional retraining program which graduates the resumption of these tasks to enable the individual to experience success and gradually improve in independence. Standardised assessments may be used to measure a person's ability to personally care for themselves, but also include return to work, return to leisure and the resumption of social and relational roles (Murgatroyd & Karimi, 2015).

One of the greatest long-term impacts of a burn injury is the subsequent scarring. If healing is delayed beyond 21 days post injury, evidence suggests that this will result in an adverse scar outcome (Deitch et al., 1983), as will an injury that has required surgical intervention. Hypertrophic scarring is an aberrant form of the normal processes of wound healing but is commonly associated with burn injury (Aarabi, Longaker & Gurtner, 2007) and is defined as red, raised, firm and itchy. It carries the very real risk of joint contracture which has the potential to severely impact function, as well as being aesthetically unacceptable (Gabriel, 2011).

Since the 1970s, compression therapy has been the main form of treatment for hypertrophic scarring (Atiyeh, 2007). This compression therapy is primarily delivered through the form of tight-fitting elastic garments (pressure garments). Compression should be commenced as soon as the healing skin is able to tolerate the pressure (Bloeman et al., 2009) and withstand the shearing forces of donning and removing the garment. Occupational therapists 'prescribe' the most appropriate pressure garment for the patient, selecting from a range of ready-to-wear and custom-made garments, and taking into account the patient's individual needs and lifestyle. Pressure garments are to be worn 23 hours a day (Berman et al., 2008) for a period of up to 18 months.

A number of other modalities that are employed to assure a positive scar outcome include:

- daily scar massage—undertaken in conjunction with the application of moisturisers. Massage therapy aims to discourage the formation of fibrotic tissue and to realign the collagen fibres (Edwards, 2011)
- contact media—silicone products (gel sheets, gels 'in a tube', putty) and hydrocolloid dressings which are used in conjunction with compression therapy to improve hydration in the scar, reduce pain and itch, reduce vascularity and scar height, and improve pliability
- splinting—contracting scars may need either dynamic splinting (splints with moving components to apply a pull against the scar) or static splints to be worn at night to maintain an anti-contracture position.

As the occupational therapist is concerned with both function and scar, there are two areas of the body that require additional specialist input. These areas, the hands and the face, carry the risk of significant psychosocial impact given their visibility and significant potential impact on independence and function. Specialised programs include splinting exercise and a range of scar management techniques unique to these areas; most notably, transparent face masks (Parry et al., 2013; Serghiou, Holmes & McCauley, 2004).

Like many in the burns team, the intervention of the occupational therapist is prolonged. It starts at day 1 and accompanies the patient through the acute phase of burn injury, the stages of scar maturation and reintegration to life, work, independence and community.

Role of the dietitian

The role of the dietitian is to optimise the nutritional status of the burned person. Goals may include assisting the patient to maintain weight within 5% to 10% of pre-injury weight, prescription of an enteral feed that meets estimated nutritional needs, coordination of a diet that meets the patient's nutritional and individual needs, prevention of signs and symptoms of micronutrient deficiency, and minimisation of the risk of complications such as hyperglycaemia, hyponatraemia and hypertriglyceridaemia.

THE METABOLIC RESPONSE

The person with a major burn (> 15% to 20% TBSA in adults and > 10% TBSA in children) is in a hypermetabolic and catabolic state. Resting energy expenditure after severe burn injury can be elevated to twice normal levels (Shields et al., 2013). The mediators of the hypermetabolism include elevated levels of catecholamines, glucocorticoids and glucagon (Bonet et al., 2011). The extent of a person's hypermetabolism is directly proportionate to the size of the burn injury (Rousseau et al., 2013).

Recent advances in medical and nursing care have blunted hypermetabolic response to burn injury; however, it cannot be fully reversed.

NUTRITION SUPPORT

Careful nutrition assessment, planning, intervention and monitoring is required in order to ensure burns patients meet nutritional requirements. Adult patients with < 20% TBSA burns and paediatric patients with < 10% TBSA burns may be able to maintain nutritional status via a high-energy/high-protein diet and oral nutritional supplementation.

Early initiation of enteral nutrition improves outcomes in adult patients with > 20% TBSA burns (Kovacic-Vicic, Radman & Kovacic, 2013). Current recommendations suggest commencement of enteral nutrition (within 24–48 hours) for all adults with > 20% TBSA burns and children with > 10% TBSA burns (ANZBA, 2014). Early enteral feeding is

promoted to offset hypermetabolism, improve nitrogen balance, decrease infection, maintain intestinal barrier function, prevent bacterial translocation and decrease length of hospital stay. As nasogastric feeding is usually well tolerated by people with burns, post-pyloric feeding is rarely required. People on enteral feeds are also encouraged to eat and drink, if possible. Total parenteral nutrition (TPN) is appropriate only for those patients who have prolonged intolerance to enteral feeds and who are unable to attain adequate protein and calorie intake with enteral nutrition (Cochran et al., 2013). Examples where TPN may be instigated include pancreatitis, bowel obstruction, paralytic ileus or intestinal perforation.

MONITORING AND EVALUATION As a patient's burn injury heals, they are usually able to tolerate larger amounts of oral diet and nutritional supplements. Patients with major burns will usually progress from complete nutrition via enteral feeds to overnight feeds, and then enteral feeding will cease. The decision as to when to progress the patient is made by the team. Considerations include adequacy of oral intake, consumption of oral nutritional supplements and healing stage of the wounds. Nutrition-related monitoring may include food-intake charts, weekly weights, biochemical indices (renal function, liver function, Mg, PO_4^{3-}, BSL), maintenance of muscle stores (e.g. handgrip strength), bowels and fluid balance.

DISCHARGE PLANNING Hypermetabolism related to a major burn can persist for up to 2 years after the injury (Rodriguez et al., 2011). Patients with major burns should receive education prior to discharge about the importance of weight maintenance, adequate protein and following a general healthy diet. Evidence is scant about the long-term requirement for vitamins and minerals of burns patients. A daily multivitamin/multimineral supplement until wound healing is demonstrated may be useful to optimise vitamin and mineral intake.

Nursing care

Health promotion

Although treatments have improved significantly during the past several decades, prevention remains the primary goal. The nursing profession is currently well positioned to collaborate with other disciplines to develop initiatives to reduce the number of burn injuries. For example, as advocates, nurses can alert political leaders to the need to pass legislation aimed at reducing the incidence of burns. Appropriate legislative themes might centre on safety in the workplace (e.g. requirements for smoke alarms and sprinkler systems), on the highways (e.g. regulations regarding the transportation of flammable liquids) and in the home (e.g. requirements for safety devices for water heaters and wood-burning stoves and for self-extinguishing cigarettes). As educators, nurses can develop teaching plans for families and communities to heighten awareness of the problem. As researchers, nurses can investigate conditions leading to burn injury and suggest methods to reduce its prevalence. Working together with healthcare policy makers and community leaders, nurses can join the effort to lower the number of annual burn cases.

Nursing diagnoses and interventions

A major burn affects virtually every body system, as well as social, cultural, economic, psychological and spiritual wellbeing. Acute care is combined with ongoing rehabilitation that continues post discharge. Many nursing diagnoses are appropriate for the person with a major burn injury; they include *Impaired skin integrity*, *Deficient fluid volume*, *Acute pain*, *Risk of infection*, *Impaired physical mobility*, *Imbalanced nutrition: less than body requirements* and *Powerlessness*. The nursing care within a multidisciplinary team has been discussed with regard to the first six of these nursing diagnoses and the concept of *Powerlessness* is discussed below.

NURSING CARE PLAN A person with a major burn

Craig Howard is a 35-year-old coal miner from Central Queensland. Craig was driving home after a late shift when his 4-wheel drive veered off the road, rolled and caught on fire. He was freed from his vehicle by a passing motorist, who in turn summoned help and called police. The passing motorist stayed with Craig until the aerial rescue team arrived and transported him to the nearest metropolitan hospital emergency department. The hospital has a burns unit. Mr Howard's wife, Mary, and twin daughters, Jessica and Jane, aged 10, were notified of the accident by police.

ASSESSMENT

On his admission to the emergency department, Craig was diagnosed with deep partial-thickness and full-thickness burns of the anterior chest, and circumferential full-thickness burns of the arms and hands. An initial quick assessment based on the rule of nines estimates the extent of his burn injury at 36% of TBSA. His vital signs were as follows: T 35.6°C, P 140, R 40 and BP 98/60. At the scene, the paramedics inserted two large-bore cannulae and started the rapid infusion of Hartmann's solution. Craig is receiving 100% humidified oxygen via a face mask. Initial ABGs are: pH 7.49, PO_2 60 mmHg, PCO_2 32 mmHg and bicarbonate 22 mEq/L. Lung sounds indicate inspiratory and expiratory wheezing, and a persistent cough reveals sooty sputum production. An indwelling catheter was inserted into his bladder and initially drained a moderate amount of dark, concentrated urine. A nasogastric tube was inserted. Craig is alert and oriented and complains of severe pain associated with the burn injuries. The burns unit is notified and Craig is transferred there.

NURSING CARE PLAN A person with a major burn (continued)

Craig's condition is serious. There are several nursing diagnoses that could be developed, as noted below.

DIAGNOSIS

- *Impaired skin integrity* related to major burns injury.
- *Acute pain* related to major burns injury.
- Potential for *Infection* related to *Impaired skin integrity.*
- *Deficient fluid volume* related to major burns injury.
- *Impaired physical mobility* related to major burns injury.
- Potential for pressure injuries related to *Impaired physical mobility.*
- Potential for *Imbalanced nutrition* related to excess body requirements associated with burns injury.

PLANNING

- Ensure room is set up prior to the person's arrival, i.e. assemble all equipment required to weigh the person, shower and dress the person, administer IV fluids and nasogastric fluids, measure and test urine, correct documentation, analgesia ordered.
- Ensure all staff are aware a major burn is being admitted and that an appropriate staff member is allocated to this person.
- Warm the room.
- Prepare for possible prophylactic nasotracheal or endotracheal intubation to maintain airway patency.
- Prepare the person and family when they arrive with an explanation of protocols and procedures. This may help reduce anxiety and help with compliance.

Expected outcomes

- The person maintains an effective airway and vital signs are within normal limits. The person has clear breath sounds, and no evidence of cyanosis. Mental status is within normal limits.
- The person does not suffer from hypothermia as evidenced by warm peripheries, conscious level and vital signs.
- The person demonstrates adequate fluid volume by maintaining appropriate urine output and urea and electrolytes and other laboratory findings within normal limits.
- The person demonstrates understanding of instructions and orientation to person, time and place through conversation and compliance with requests and treatment.

IMPLEMENTATION

- *Maintain effective airway clearance,* as there is potential for increased lung congestion secondary to smoke inhalation and torso burns.
- *Maintain adequate fluid volume*, as abnormal fluid loss is possible secondary to burn injury.
- *Monitor for adequate tissue perfusion* (peripheral), as peripheral constriction secondary to circumferential burn wounds of the arms is a possibility.
- Weigh the person.
- Once cervical spine injury has been cleared, elevate head of bed to improve lung expansion and ventilation.
- Administer humidified 100% oxygen at prescribed amount.
- Educate the person on the importance of deep breathing and sitting upright to improve his breathing.
- Monitor for expectoration of sooty sputum, stridor, increase in wheezing.
- Elevate both arms on one or two pillows to reduce swelling.
- Perform neurovascular observations ½–1 hourly depending on neurovascular status and hospital protocol—that is, colour, warmth, movement, sensation and capillary refill.
- Remove dressings from fingertips to enable neurovascular observations to be done.
- Administer intravenous analgesia as ordered for pain.
- Observe for any signs of respiratory distress or decrease in respiratory status.
  - Observations to be performed ½–1 hourly depending on the person's condition and hospital protocol—that is, respiratory rate, blood pressure and pulse. Temperature 1 hourly as the person is hypothermic on admission. Oxygen saturations should be monitored continuously.
- Explain to the person all procedures and protocols to help reduce anxiety and encourage compliance with treatment. This may need to be done on more than one occasion.
- Initiate fluid resuscitation therapy using the Parkland (Baxter) formula to calculate intravenous fluid rate for the first 24 hours post burn. Review original calculations based on real weight, not estimated weight, and ensure fluid resuscitation time is from time of injury.
- Insert nasogastric tube and commence enteral feeding as instructed. Discourage the person from drinking water.
- Measure urine output 1-hourly and record specific gravity hourly.
- Document all observations, input and output accurately.
- Arrange for blood tests to be taken according to hospital burn resuscitation protocol and ensure the results are reviewed.
- Craig maintained his respiratory status and did not require intubation. He was continued on humidified oxygen sitting in an upright position in bed or chair with his arms elevated.
- Hourly urine outputs indicate adequate fluid resuscitation. Urine output has been maintained at 50 mL/h and is a straw colour; specific gravity is 1020. Blood pressure has increased to 100/64. He has remained tachycardic at 100, and his respiratory rate on humidified oxygen is 28. His temperature is now 37°C.
- To improve tissue perfusion of both arms, the physician has performed bilateral escharotomies and the wounds are packed with alginate and covered in a silver antimicrobial dressing, such as Acticoat®. Colour, warmth, movement, sensation and capillary return have improved, indicating escharotomies have been successful.

EVALUATION

- Craig Howard demonstrated a patent airway, as evidenced by clear breath sounds; absence of cyanosis; and vital signs, chest x-ray findings and ABGs within normal limits.

(continued)

NURSING CARE PLAN A person with a major burn (continued)

- Adequate fluid volume and electrolyte balance demonstrated, as evidenced by urine output, vital signs, mental status and laboratory findings within normal limits.
- Adequate tissue perfusion demonstrated, as evidenced by palpable pulses, warm extremities, normal capillary refill and absence of paraesthesia.

CRITICAL THINKING IN THE NURSING PROCESS

1. Explain the rationale for the immediate insertion of an indwelling catheter and nasogastric tube.
2. An escharotomy was performed on both arms. Why was this procedure necessary in Craig Howard's case?
3. What is the rationale supporting the intravenous administration of narcotics to control Craig's pain?
4. Explain the sequence of events that led to a fluid and electrolyte shift during the first 24 to 48 hours after Craig sustained his injury.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Which communication and person-centred education strategies would you use when caring for people with major burns such as Craig's?

CONSIDERATION FOR PRACTICE

An increased body temperature, without other manifestations of infection, is not indicative of infection in people with large burn wounds (in which the hypermetabolic response resets the core temperature to a higher level).

CONSIDERATION FOR PRACTICE

Assess all people, but especially the older person, for indications of pressure injury formation under a splint.

CONSIDERATION FOR PRACTICE

Narcotics are always administered intravenously, rather than orally, subcutaneously or intramuscularly in the resuscitative or acute phase of a burn due to decreased circulation and absorption of medications.

Powerlessness

Usually, the person with a major burn injury endures a lengthy hospital stay involving many treatments and care protocols that are beyond their control. During the early stages, much of the care regimen involves pain. Further, the foreign environment of the burns unit makes it difficult for the people to relate to the immediate surroundings. The person's body image is often altered, depending on the extent and location of the burn injury.

- Involve the person in their own care. Encourage independence with activities of daily living. Encourage the person to participate in showering and toilet and physiotherapy. Encourage the person to verbalise their feelings. *Powerlessness derives from the belief that one is unable to influence the outcome of a situation.*
- Keep needed items within reach, such as call bell, urinal, water pitcher and tissues, *to reinforce the person's feelings of control.*
- Encourage the person to express feelings. *The nurse can help the person cope by therapeutically listening, displaying a caring presence, clarifying misconceptions and providing positive feedback.*

NURSING CARE OF THE OLDER PERSON Burns in the older person

Older people are at greater risk of burns of all degrees of severity. Most burns are accidental, the result of slower reaction times, decreased mobility, visual deficits, a decreased sense of smell, forgetfulness and impaired sensation. Many older people are burned by stoves, hot water, hot food, irons, cookware and heating pads. Older people with cognitive impairments or dementia may start fires by leaving cooking foods unattended.

The care of older people with burns often presents unique challenges. They may delay seeking treatment, thus increasing the risk of infection and burn wound conversion. Their care may be complicated by the presence of other chronic illnesses. They may live alone and have no one to care for them during rehabilitation. Even small burns have the potential to become serious in older people.

Burn prevention strategies for older adults are as follows:

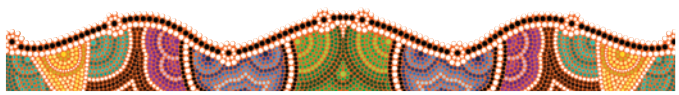
- Check the smoke detector battery once a month.
- Wear close-fitting clothing when cooking.
- Use a cooking timer with a loud alarm.
- Never lay anything over a heating device.
- Install anti-scald devices in bathroom plumbing.
- Encourage no smoking in the house.

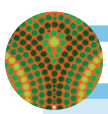
- Involve the person in setting realistic goals to suit their level of recovery, but also encouraging them to aim for improvement, not stasis, in rehabilitation. *Rehabilitation starts the day the person is admitted, not when healed. Small incremental gains are easier to achieve and allow for frequent positive reinforcement.*

Community-based care

Education for the person with burns and their family is an important component of all phases of burn care. As treatment progresses, the nurse encourages family members to assume more responsibility in providing care. From admission to discharge, the nurse teaches the person and family to assess all findings, implement therapies and evaluate progress. The following topics should be addressed when preparing the person and family for home care:

- The long-term goals of rehabilitation care, which are to prevent soft tissue deformity, protect skin grafts, maintain physiological function, manage scars and return the person to an optimal level of independence.
- Avoiding exposure to people with colds or infections, and following aseptic techniques meticulously when caring for the wound.
- The need for progressive physical activity.
- How to apply splints, pressure support garments and other assistive devices.
- Dietary requirements with required kilojoules.
- Alternative pain control therapies, such as guided imagery, relaxation techniques and diversional activities.
- Care of the graft and donor sites.
- Referral for social service, clergy, and/or psychiatric services as appropriate.
- Helpful resources and websites:
  - Australian and New Zealand Burn Association (ANZBA): www.anzba.org.au
  - Australian Bureau of Statistics, *Australian Social Trends. Health—Mortality Morbidity*: www.ABS.gov.au
  - Australian Wound Management Association Inc: http://www.awma.org.au/
  - Burn Foundation Australia: www.burnfoundation.org.au
  - Burns Clinical Practice Guidelines, Royal Children's Hospital Melbourne Victoria, Australia: www.rch.org.au/clinicalguide/guideline_index/Burns
  - Burn Prevention: www.burnprevention.org
  - Burns Support Foundation Incorporated New South Wales: www.burnssupportfoundation.com
  - Burn Survivors Network: www.kidsfoundation.org.au/media1/burns-survivors/
  - Burnsurgery.org: www.burnsurgery.org
  - Changing Faces: www.changingfaces.org.uk
  - International Society for Burn Injuries: www.worldburn.org
  - Fiona Wood Foundation: www.fionawoodfoundation.com
  - K.I.D.S Foundation: www.kidsfoundation.org.au
  - WoundsWest Burns Module: www.health.wa.gov.au/woundswest
  - Integra® www.ilstraining.com/idrt/idrt/brs_it_00.html





TRANSLATIONTO PRACTICE Evidence-based practice: a person with a major burn

People with major burn injuries are at risk of pressure injuries due to deficient fluid volume, inadequate nutrition, pain and immobility. Healthy, intact skin can experience damage from friction and shearing forces, posing a second area of risk of pressure injuries.

In an investigation of pressure injury prevention in burned people (Gordon et al., 2004), a multidisciplinary team of burn care specialists reviewed and evaluated the current evidence of practice for preventing pressure injuries. Pressure injuries result from continuous, unrelieved pressure that causes reduced blood flow to vulnerable areas of the skin, resulting in tissue damage. The results of the study confirmed that contributing factors to the development of pressure injuries in people with burn injuries include inadequate dietary intake, malnutrition, decreased albumin levels, moisture and incontinence, reduced sensation, prolonged immobility, increased age and altered level of consciousness.

IMPLICATIONS FOR NURSING

Nurses and all members of the burn team assume an integral role in preventing pressure injuries. To prevent pressure injuries, nurses must constantly monitor the person with major burn injuries for signs of skin damage and conduct regular and frequent skin inspections. Chapter 15 discusses interventions for pressure injury prevention, assessment and management.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. The person's head must be elevated to 30 degrees when nasogastric tube feedings are implemented. What strategies would you recommend to prevent inhalation of nasogastric feeds?
2. How could splints that are being used to prevent contractures be modified as the person's amount and extent of oedema fluctuates?
3. Develop a teaching plan for instructing nursing assistants on how to move, position and transfer people in a burns centre unit. Include in the teaching plan the proper technique for cleansing unburned skin.

CHAPTER HIGHLIGHTS

- Four types of burn injuries are thermal, chemical, electrical and radiation. The depth of the burn injury determines whether it is classified as a superficial, partial-thickness or full-thickness burn.
- The Wallace (1951) rule of nines, age-dependent burn graphs and the palmar surface assessment are all used to estimate the extent of a burn by assigning percentages to different parts of the body.
- Major burns involve multi-organ pathophysiological alterations. Most critical is the fluid shift from the intracellular and intravascular compartments into the interstitium, resulting in a type of hypovolaemic shock called burn shock. Other pathological processes include an impaired immune system, disturbed functions of the skin, inhalation injury, gastrointestinal ulcerations and ileus, renal failure and hypermetabolism.
- Interprofessional care focuses on managing the person during the resuscitative, acute and rehabilitative phases. To counter the effects of burn shock, fluid resuscitation using guidelines such as the Parkland (Baxter) formula are initiated to replace fluid and electrolyte losses.
- Additional management for the person with major burns includes preventing atelectasis, maintaining respiratory function, controlling pain, preventing infection and Curling's ulcer, promoting nutrition and providing wound care.
- Extensive eschar of an extremity or the torso, called circumferential wounds, can potentially occlude arterial flow or decrease respiratory function. An escharotomy is used to release tension, preventing additional complications.
- Surgical management of burn wounds includes debridement and skin grafting. Biological and biosynthetic dressings provide temporary covering and prepare the wound for permanent autografts.
- Continual psychological support of the person and family is essential throughout convalescence and rehabilitation.

CONCEPT CHECK

1 During the resuscitative phase of burn management, what diagnostic test result should the nurse expect to find?
1 increased haematocrit
2 increased serum albumin
3 decreased serum potassium
4 decreased blood urea nitrogen (BUN)

2 A person is admitted with severe burns to the face and chest. The injured skin is dry and leathery, with no pain sensations present. The nurse recognises that this burn is classified as:
1 superficial
2 superficial partial-thickness
3 deep partial-thickness
4 full-thickness

3 Which of the following people is at greatest risk of developing burn shock?
1 a 21 year old with 90% superficial burn from a tanning bed
2 a 30 year old with 10% TBSA from a gasoline explosion
3 a 39 year old with radiation burns following treatment for cancer
4 a 48 year old with >50% TBSA from a high-voltage electrical accident

4 A person with full-thickness burns over 50% of the body arrives in the emergency department. The person weighs 70 kg. Using the Parkland (Baxter) formula, calculate the amount of fluid replacement that the nurse should deliver in the first 8 hours.
1 3500 mL
2 7000 mL
3 10 500 mL
4 14 000 mL

5 For a person with a major burn, which of the following evaluation criteria indicate that fluid resuscitation is effective during the first 24 hours of care?
1 urine output of 30 to 50 mL/h
2 central venous pressure of 18
3 heart rate of 130 beats per minute
4 blood pressure 96/70

6 A person has deep partial-thickness burns to the entire left arm and left side of the back. What finding should be reported to the physician immediately?
1 fluid-filled vesicles on the left arm
2 pain in the left arm
3 blanching when pressure is applied to the left hand
4 decreased left radial pulse

7 A person received deep partial-thickness burns to the anterior trunk, perineum, and left arm anterior and posterior. Using the Wallace (1951) rule of nines, what is the percentage of total body surface area (TBSA) that was burned?
1 18%
2 28%
3 36%
4 40%

8 Which of the following topics should be included in a presentation on burn prevention at a senior citizens centre? (Select all that apply.)
1 Wear close-fitting clothing when cooking.
2 Use a solar-powered night light.
3 Check smoke detectors annually.
4 Install anti-scald devices in bathroom plumbing.
5 Have a neighbour routinely check for the odour of gas.

9 A person has possible carbon monoxide poisoning secondary to smoke inhalation. What manifestation should the nurse expect to find in a person with a 15% carbon monoxide level?
1 dark red skin colour
2 drowsiness
3 dizziness
4 hypotension

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UNIT 4 BUILDING CLINICAL COMPETENCE

Responses to altered integumentary structure and function

CLINICAL SCENARIO

You have been assigned to work with the following four people for the 0700 shift on a medical–surgical unit. Significant data obtained during report are as follows:

- Mr Johnson is a 46 year old who is hospitalised for surgery to release contractures at his elbows which resulted from a flash burn from a grill fire 3 years ago. He is scheduled for surgery at 0800 and needs vital signs, preoperative medication and the preoperative checklist completed.
- Mrs Carter is a 35 year old who was hospitalised 2 days ago with cellulitis in the right calf. Vital signs are T 38°C, P 80, R 20, BP 116/76. She is complaining of a headache and pain in the right calf. She was last medicated for pain at 0300.
- Mr Jenkins is an 86 year old with herpes zoster. He was admitted 4 days ago with lesions on his left neck and trunk. Vital signs are T 37.2°C, P 88, R 26, BP 158/90. He is complaining of burning pain across his back and is requesting a nurse to check his back for new lesions.
- Mr Ugandi is a 34-year-old Indigenous Australian male who has AIDS. He was transferred from the burn critical care unit to the medical–surgical unit at 0600 after being treated for toxic epidermal necrolysis for the past month. He is ready to begin discharge teaching.

Critical thinking questions

1 In what order would you visit these people after report?

1.
2.
3.
4.

2 What top two priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Mr Johnson		
Mrs Carter		
Mr Jenkins		
Mr Ugandi		

3 Mr Johnson received partial-thickness and full-thickness flash burns on both anterior and posterior arms and his anterior trunk from the grill fire. Using the rule of nines, what is the percentage of total body surface (TBSA) burned? _____%

4 Morphine sulfate is the drug of choice for pain. Which manifestation requires immediate nursing intervention following morphine administration?

1. vomiting once after medication is administered
2. respiratory rate below 8 breaths per minute
3. blood pressure of 110/70 after a baseline blood pressure of 120/80
4. peripheral pulse of 68 after a baseline pulse of 78

5 A diet high in protein and iron may help prevent pressure injuries on the person who is on prolonged bed rest. Which foods should the nurse encourage the person to eat to help prevent pressure injuries?

1. eggs and chicken
2. broccoli and oranges
3. oatmeal and bananas
4. wholegrain bread and kidney beans

6 Mr Jenkins is being discharged to home. Which statement does the nurse need to teach Mr Jenkins about herpes zoster on discharge?

1. 'Continue taking the antiviral medication to cure the herpes zoster.'
2. 'You can attend church functions because herpes zoster is not contagious.'
3. 'Use narcotic pain medications only for severe pain so you do not become addicted to the medication.'
4. 'Wear cotton clothing and keep room temperatures cool to decrease the pain and itching from the herpes zoster lesions.'

7 Mrs Carter was admitted with cellulitis in the right calf. Which manifestations would the nurse assess on admission?

1. redness, oedema, and pain in the right calf
2. purulent drainage, pale skin, and pain in right calf
3. rash, redness, and swelling in right calf
4. itching, rash, and pain in right calf

8 The nurse applies mafenide acetate (Sulfamylon®) to Mr Ugandi's open skin wounds caused by toxic epidermal necrolysis. Which hypersensitivity reactions would require the nurse to discontinue the drug?

1. tachycardia and tachypnoea
2. nausea and vomiting
3. facial oedema and pruritus
4. diarrhoea and candidiasis

9 Which serum laboratory values are decreased with inadequate nutritional intake, such as in a person with severe burns? (Select all that apply.)

1. protein
2. potassium
3. iron
4. full blood cell count
5. glucose
6. calcium

10 When a person is admitted to the burns unit for treatment of severe burns, it is most important for the nurse to monitor the person for:

1. acute pain
2. nausea and vomiting
3. hypothermia
4. fluid and electrolyte imbalance

11 Which of these people is most at risk of skin breakdown leading to pressure injuries?

1. a 70 year old who had a stroke with left-sided paralysis
2. a quadriplegic admitted to the hospital with pneumonia

3. a 56 year old on dialysis three times a week
4. an 84 year old in traction for a hip fracture

12 Health teaching for skin cancer includes which interventions? (Select all that apply.)

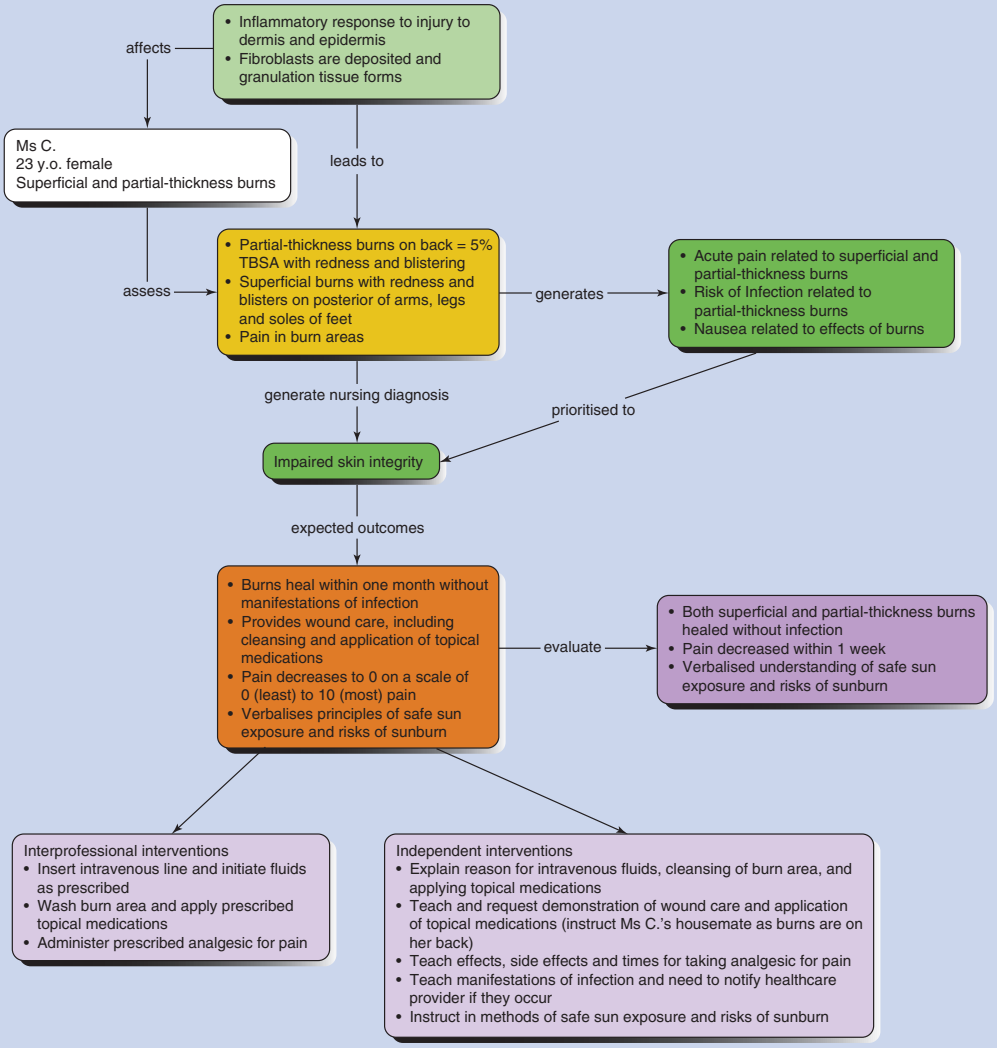
1. Wear long-sleeved shirts and a wide-brimmed hat in the sun.
2. Apply sunscreen once a day.
3. Avoid tanning booths or prolonged exposure to the sun.
4. Minimise exposure to the sun between 1 pm and 4 pm.
5. Apply sunscreen before and after swimming.

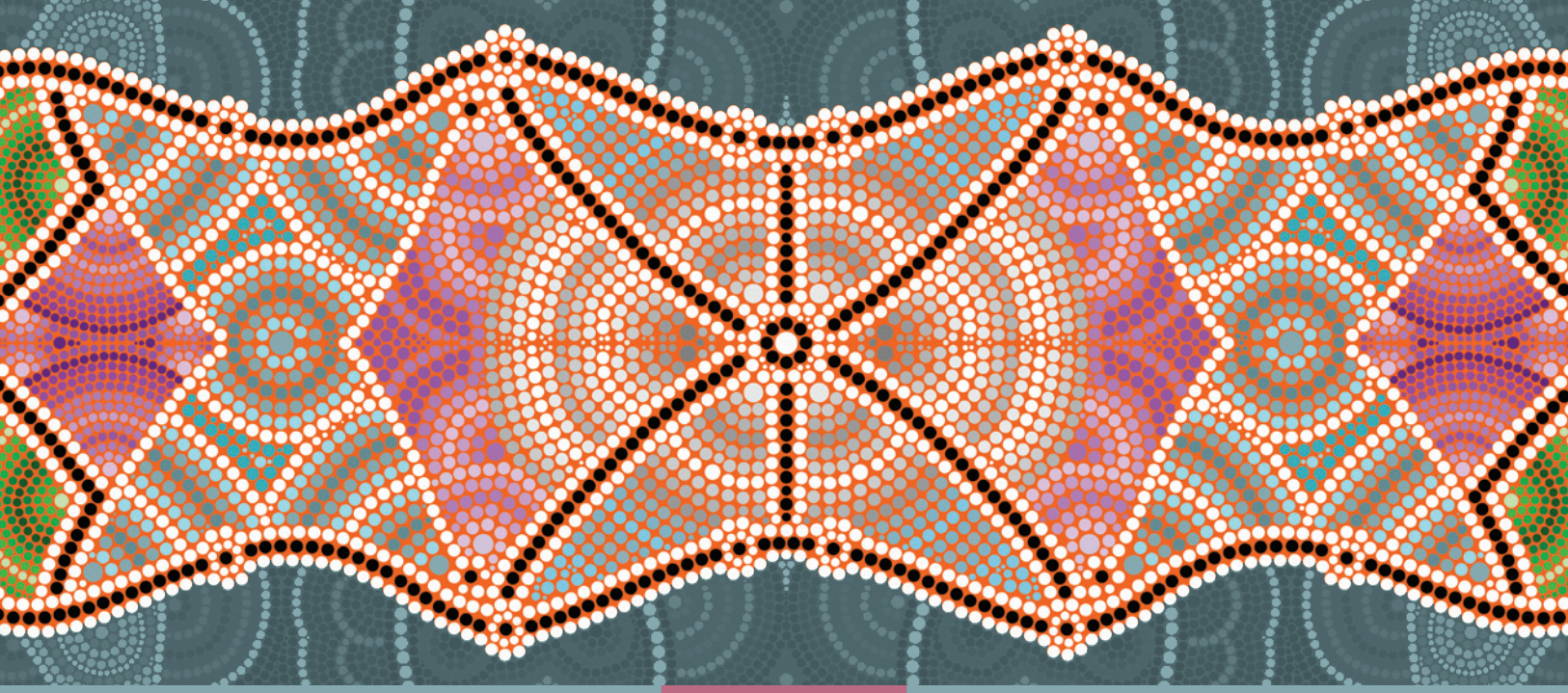
CASE STUDY

Ms Rachel Chelen is a 23 year old who is admitted to the medical–surgical unit for observation and treatment after being exposed to the sun for a prolonged period of time. On admission, Ms Chelen stated she fell asleep lying on her stomach while sunbathing at the beach. Her vital signs are T 39°C, P 94, R 26, BP 116/76. Her height is 167 cm and weight is 87 kg. Assessment findings are that her back is red and slightly oedematous with an area approximately 6 cm by 3 cm between the scapula that is beginning to develop blisters. The backs of her arms and legs, and soles of her feet are red and slightly oedematous. She is complaining of pain on her back, arms, legs and feet. She stated that she feels chilled, is nauseated and has a headache. Blood is drawn for a full blood count and electrolytes for baseline assessment. IV therapy is instituted to maintain hydration. The burn areas are washed with soap and water and an antibiotic ointment is applied. A mild analgesic is administered as ordered. Ms Chelen is covered to prevent further chilling and to prevent burned areas being exposed to air.

She is diagnosed with superficial and superficial partial-thickness burns. Using the rule of nines, the partial-thickness burn on her back is classified as approximately 5% total body surface area. The superficial burns over the rest of the reddened areas are not classified. Superficial burns involve the epidermis layer of the skin. Superficial partial-thickness burns involve the entire dermis and the papillae of the dermis. The pathophysiological effects of the superficial and partial-thickness burns are the result of exposure to the sun for a prolonged time. Inflammation occurs in response to tissue injury. Platelets aggregate at the burn injury site, fibrin is deposited, and a thrombus is formed. The thrombus, along with vasoconstriction, walls off the burn injury site. Then vasodilation occurs with increased capillary permeability, which leads to redness and oedema. Injury to the dermis results in a moist, glistening appearance as blisters form. The burned area will blanch on pressure. There is pain in response to touch and temperature changes. The burned area should heal within 14 to 21 days with minimal or no scarring, but may have pigment changes. Manifestations of superficial and superficial partial-thickness burns are skin redness, blister formation, local pain, headache, chills, nausea and vomiting. Complications of superficial and superficial partial-thickness burns are infection, hypothermia, dehydration and fluid and electrolyte imbalances.

Due to the severity of tissue injury from the burns, the priority nursing diagnosis of *Impaired skin integrity* is appropriate for guiding nursing care.





UNIT 5

RESPONSES TO ALTERED ENDOCRINE FUNCTION

CHAPTER 17

A PERSON-CENTRED APPROACH TO ASSESSING THE ENDOCRINE SYSTEM

NICOLE KNOX

KEY TERMS

LEARNING OUTCOMES

- Describe the anatomy and physiology of the endocrine glands.
- Explain the functions of the hormones secreted by the endocrine glands.
- Identify specific topics to consider during a health history interview of the person with health problems involving endocrine function, incorporating an understanding of age-related changes in assessment findings.
- Explain the nursing implications for a range of diagnostic tests associated with the endocrine glands.

CLINICAL COMPETENCIES

- Conduct and document a health history for the person who has or is at risk of alterations in the structure or function of the endocrine glands.
- Monitor the results of diagnostic tests and report abnormal findings.
- Conduct and document a physical assessment of the structure of the thyroid gland and the effects of altered endocrine function on other body structures and functions.

EQUIPMENT NEEDED

- Reflex hammer
- Safety pin, cotton ball, containers with hot and cold water, tuning fork
- Blood pressure cuff
- Stethoscope

The endocrine system plays an integral part in the regulation of the body's internal environment. Through hormones secreted by its glands, the endocrine system regulates such varied functions as growth, reproduction, metabolism, fluid and electrolyte balance, and gender differentiation. It also helps the body adapt to constant alterations in the internal and external environment.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE ENDOCRINE SYSTEM

The major endocrine organs are the pituitary gland, thyroid gland, parathyroid glands, adrenal glands, pancreas and gonads (reproductive glands). The locations of these glands are illustrated in Figure 17.1. Table 17.1 summarises the functions of the endocrine organs and their hormones. Specific information about the ovaries and testes is found in Chapters 46 to 48.

PITUITARY GLAND

The pituitary gland (hypophysis) is located in the skull beneath the hypothalamus of the brain (see Figure 17.2). It often is called the 'master gland' because its hormones regulate many body functions. The pituitary gland has two parts: the anterior pituitary (or adenohypophysis) and the posterior pituitary (or neurohypophysis). The anterior pituitary is glandular tissue, whereas the posterior pituitary is actually an extension of the hypothalamus.

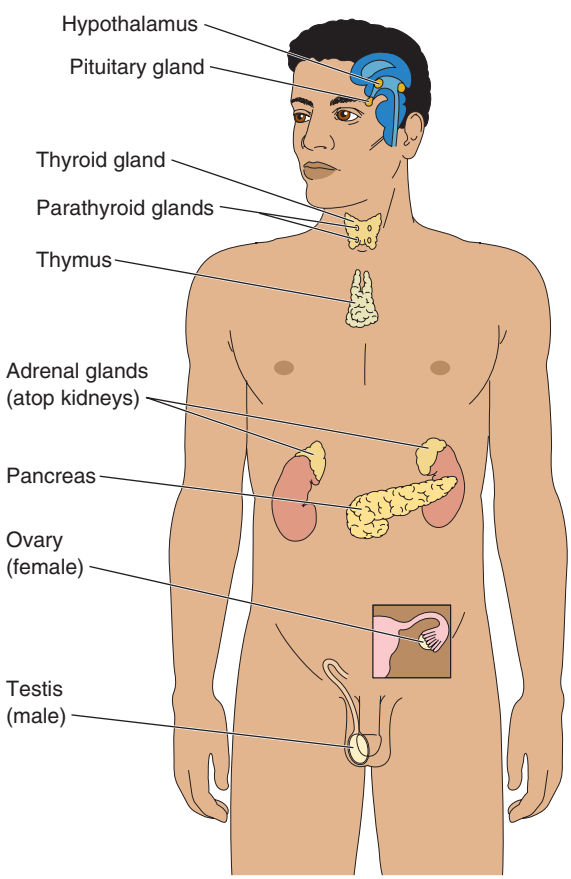


FIGURE 17.1 ■ Location of the major endocrine glands

Anterior pituitary

The anterior pituitary has several types of endocrine cells and secretes at least six major hormones (see Figure 17.3).

- Somatotropic cells secrete growth hormone (GH) (also called *somatotropin*). GH stimulates growth of the body by signalling cells to increase protein production and by stimulating the epiphyseal plates of the long bones.
- Lactotropic cells secrete prolactin (PRL). Prolactin stimulates the production of breast milk.
- Thyrotropic cells secrete thyroid-stimulating hormone (TSH). TSH stimulates the synthesis and release of thyroid hormones from the thyroid gland.
- Corticotropic cells secrete adrenocorticotropic hormone (ACTH). ACTH stimulates release of hormones, especially glucocorticoids, from the adrenal cortex.
- Gonadotropic cells secrete the gonadotropin hormones, follicle-stimulating hormone (FSH) and luteinising hormone (LH). These hormones stimulate the ovaries and testes (the gonads). In women, FSH stimulates the development of ovarian follicles and induces the secretion of oestrogenic female sex hormones. Increasing levels of LH work together with FSH to lead to ovulation and the formation of the corpus luteum from an ovarian follicle. In men, FSH is involved in the development and maturation of sperm. LH in men is called the interstitial cell-stimulating hormone (ICSH), which stimulates the interstitial cells of the testes to produce male sex hormones.

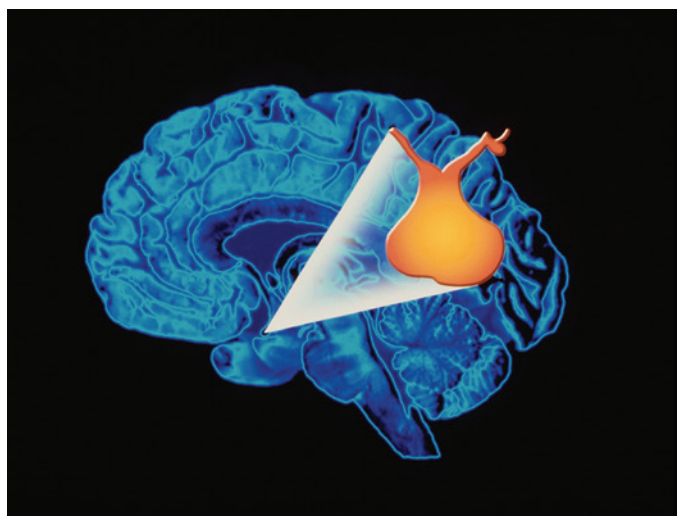


FIGURE 17.2 ■ Location of the pituitary gland

Source: Alfred Pasieka/Science Source.

TABLE 17.1 Organs, hormones, functions and feedback mechanisms of the endocrine system

ENDOCRINE ORGAN	HORMONE SECRETED	TARGET ORGAN AND FEEDBACK MECHANISM
Thyroid gland	Thyroid hormone (TH): thyroxine (T_4) is the major hormone secreted by the thyroid gland. It is converted to triiodothyronine (T_3) at the target tissues.	Maintains metabolic rate and the growth and development of all tissues. T_3 and T_4 are secreted in response to thyroid-stimulating hormone (TSH).
	Calcitonin	Maintains serum calcium levels by decreasing bone resorption and decreasing resorption of calcium in the kidneys whenever levels of plasma calcium are elevated. Works together with parathyroid gland to regulate calcium levels.
Parathyroid gland	Parathyroid hormone (PTH)	Maintains serum calcium levels by stimulating bone resorption and formation and by stimulating kidney resorption of calcium in response to falling levels of plasma calcium.
Adrenal cortex	Mineralocorticoids (e.g. aldosterone)	Promotes renal tubule reabsorption of sodium and water and excretion of potassium in response to elevated levels of potassium and low levels of sodium, thereby increasing blood pressure and circulating blood volume.
	Glucocorticoids (e.g. cortisol)	Help to regulate metabolism of carbohydrates, fats and proteins. Activate anti-inflammatory responses to stressors. Low cortisol levels stimulate hypothalamic secretion of corticotropin-releasing hormone (CRH), which stimulates the anterior pituitary gland to release ACTH, which in turn stimulates the adrenal cortex to secrete cortisol.
	Gonadocorticoids (androgens and small amounts of oestrogen and progesterone)	The quantity of sex hormones produced here is minimal and the mechanism is not well understood.
Adrenal medulla	Catecholamines (adrenaline and noradrenaline)	Secreted in response to physical or psychological stress; catecholamines stimulate the heart, constrict blood vessels, inhibit visceral muscles, dilate bronchioles, increase respiration and metabolism, and promote hyperglycaemia.
Anterior pituitary (adenohypophysis)	Growth hormone (GH)	Promotes growth of body tissues by enhancing protein synthesis and promoting use of fat for energy and thus conserving glucose. Release is stimulated by growth-hormone-releasing hormone (GHRH) in response to low GH levels, hypoglycaemia, increased amino acids, low fatty acids and stress.

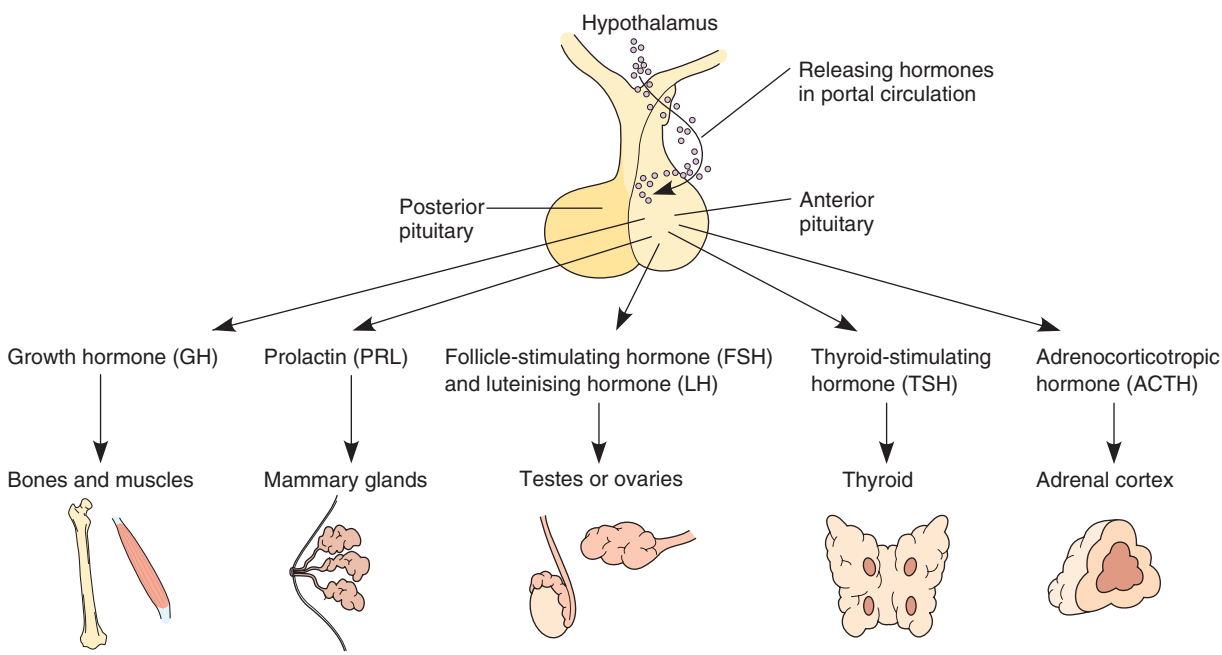


FIGURE 17.3 ■ Actions of the major hormones of the anterior pituitary

Posterior pituitary

The posterior pituitary is made of nervous tissue. Its primary function is to store and release antidiuretic hormone (ADH) and oxytocin, produced in the hypothalamus.

- ADH, also called *vasopressin*, decreases urine production by causing the renal tubules to reabsorb water from the urine and return it to the circulating blood volume.
- Oxytocin induces contraction of the smooth muscles in the reproductive organs. In women, oxytocin stimulates the myometrium of the uterus to contract during labour. It also induces milk ejection from the breasts.

THYROID GLAND

The **thyroid gland** (see Figure 17.4) is anterior to the upper part of the trachea and just inferior to the larynx. This butterfly-shaped gland has two lobes connected by a structure called the isthmus.

The glandular tissue consists of follicles filled with a jelly-like colloid substance called thyroglobin, a glycoprotein–iodine complex. Cells within the follicles secrete thyroid hormone (TH), a general name for two similar hormones: thyroxine (T_4) and triiodothyronine (T_3). The primary role of thyroid hormones in adults is to increase metabolism. TH secretion is initiated by the release of TSH by the pituitary gland and is dependent on an adequate supply of iodine.

The thyroid gland also secretes calcitonin, a hormone that decreases excessive levels of calcium in the blood by slowing the calcium-releasing activity of bone cells, serves as a marker for sepsis and is believed to be a mediator of inflammatory responses. However, when the thyroid gland is totally removed and thyroid hormone is replaced, calcium homeostasis and bone density remain relatively unchanged without replacing calcitonin.

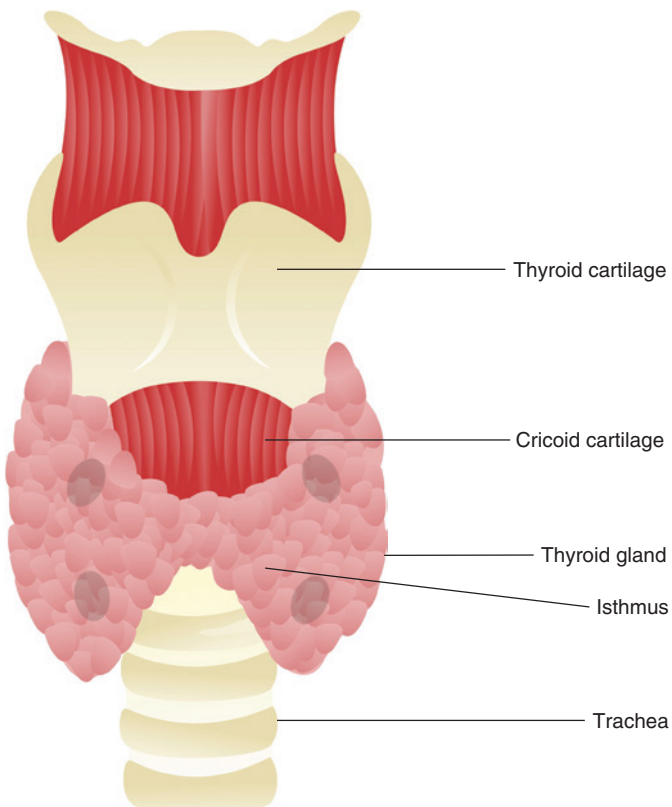


FIGURE 17.4 ■ The thyroid gland

Source: Zuzanae/Shutterstock.

Parathyroid glands

The parathyroid glands (usually four to six in number) are embedded on the posterior surface of the lobes of the thyroid gland. They secrete parathyroid hormone (PTH), or *parathormone*. When serum calcium levels fall, PTH secretion increases. PTH also controls phosphate metabolism. It acts primarily by increasing renal excretion of phosphate in the urine, by decreasing the excretion of calcium and increasing bone reabsorption to cause the release of calcium from bones. Normal levels of vitamin D are necessary for PTH to exert these effects on bone and the kidneys.

ADRENAL GLANDS

The two adrenal glands are pyramid-shaped organs that sit on top of the kidneys (see Figure 17.5). Each gland consists of two parts, which are distinct organs: an inner medulla and an outer cortex.

The adrenal medulla produces two hormones (also called catecholamines): adrenaline (epinephrine) and noradrenaline (norepinephrine). These hormones are similar to substances released by the sympathetic nervous system and thus are not essential to life. Adrenaline increases blood glucose levels and stimulates the release of ACTH from the pituitary; ACTH in turn stimulates the adrenal cortex to release glucocorticoids. Adrenaline also increases the rate and force of cardiac contractions; constricts blood vessels in the skin, mucous membranes and kidneys; and dilates blood vessels in the skeletal muscles, coronary arteries and pulmonary arteries. Noradrenaline increases both heart rate and the force of cardiac contractions. In addition it causes vasoconstriction of blood vessels throughout the body.

The adrenal cortex secretes several hormones, all corticosteroids. They are classified into two groups: mineralocorticoids and glucocorticoids.

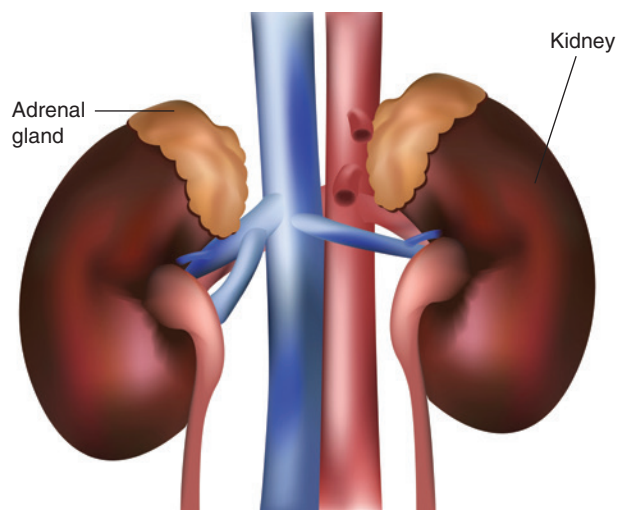


FIGURE 17.5 ■ Location of the adrenal glands

Source: Zuzanae/Shutterstock.

An enzyme named renin primarily controls the release of mineralocorticoids. When a decrease in blood pressure or sodium is detected, specialised kidney cells release renin to act on a substance called angiotensinogen, manufactured by the liver. Renin modifies angiotensinogen to form angiotensin I which is converted to angiotensin II by the angiotensin-converting enzyme. This stimulates the release of aldosterone from the adrenal cortex, which prompts the distal tubules of the kidneys to release increased amounts of water and sodium back into the circulating blood volume to increase blood pressure. This system (the renin–angiotensin–aldosterone system (RAAS)) is illustrated in Chapter 9 with the discussion of body fluid regulation.

The glucocorticoids include cortisol and cortisone. These hormones affect carbohydrate metabolism by regulating glucose use in body tissues, mobilising fatty acids from fatty tissue and shifting the source of energy for muscle cells from glucose to fatty acids. Glucocorticoids are released in times of stress. An excess of glucocorticoids in the body depresses the inflammatory response and inhibits the effectiveness of the immune system.

PANCREAS

The pancreas, located behind the stomach between the spleen and the duodenum, is both an endocrine gland (producing hormones) and an exocrine gland (producing digestive enzymes). The digestive enzymes produced by the pancreas are discussed in Chapter 20. The content in this chapter focuses on pancreatic hormones.

The endocrine cells of the pancreas produce hormones that regulate carbohydrate metabolism. They are clustered in bodies called pancreatic islets (or islets of Langerhans) scattered throughout the gland. Pancreatic islets have at least four different cell types:

1. Alpha cells produce glucagon, which decreases glucose oxidation and promotes an increase in the blood glucose levels by signalling the liver to release glucose from glycogen stores.
2. Beta cells produce insulin, which facilitates the uptake and use of glucose by cells and prevents an excessive breakdown of glycogen in the liver and muscle; thus, insulin decreases blood glucose levels. Insulin also facilitates lipid formation, inhibits the breakdown and mobilisation of stored fat and helps amino acids move into cells to promote protein synthesis. In general, the actions of glucagon and insulin oppose one another, helping to maintain a stable blood glucose level.
3. Delta cells secrete somatostatin, which inhibits the secretion of glucagon and insulin by the alpha and beta cells.
4. F cells secrete pancreatic polypeptide, which is believed to inhibit the exocrine activity of the pancreas.

GONADS

The gonads are the testes in men and the ovaries in women. These organs are the primary source of steroid sex hormones in the body. The hormones of the gonads are important in regulating body growth and promoting the onset of puberty.

In men, androgens (primarily testosterone) produced by the testes maintain reproductive functioning and secondary sex characteristics. Androgens also promote the production of sperm. In women, the ovaries secrete oestrogens and progesterone to maintain reproductive functioning and secondary sex characteristics. Progesterone also promotes the growth of the lining of the uterus to prepare for implantation of a fertilised ovum.

AN OVERVIEW OF HORMONES

Hormones are chemical messengers secreted by the endocrine organs and transported throughout the body, where they exert their action on specific cells called target cells. Hormones do not cause reactions directly, but rather regulate tissue responses. They may produce either generalised effects or local effects.

Hormones are transported from endocrine gland cells to target cells in the body in one of four ways:

1. Endocrine glands release most hormones, such as TH and insulin, into the bloodstream. Some hormones require a protein carrier.
2. Neurons release some hormones, such as adrenaline, into the bloodstream. This is called the neuroendocrine route.
3. The hypothalamus releases its hormones directly to target cells in the posterior pituitary by nerve cell extension.
4. With the paracrine method, released messengers diffuse through the interstitial fluid. This method of transport involves a number of hormonal peptides that are released throughout various organs and cells and act locally. An example is endorphins, which act to relieve pain.

Hormones that are released into the bloodstream circulate as either free, unbound molecules or as hormones attached to transport carriers. Hormone receptors are complex molecular structures, located on or inside target cells. They act by binding to specific receptor sites located on the surfaces of the target cells. These receptors recognise a specific hormone and translate the message into a cellular response. The receptor sites are structured so that they respond only to a specific hormone; for example, receptors in the thyroid gland are responsive to TSH but not to LH. Drugs that compete with a hormone for binding with transport carrier molecules increase hormone action by increasing the availability of the free, unbound hormone. Hormone levels are controlled by the pituitary gland and by feedback mechanisms. Although most feedback mechanisms are negative, a few are positive. Negative feedback is controlled in much the same way that the thermostat in an air conditioner regulates temperature. Sensors in the endocrine system detect changes in hormone levels and adjust the level of hormone secretion to maintain normal body levels. When the sensors detect a decrease in hormone levels, they begin actions to cause an increase in hormone levels; when hormone levels rise above normal, the sensors cause a decrease in hormone production and release. For example, when the hypothalamus or anterior

pituitary gland senses increased blood levels of TH, it releases hormones, causing a reduction in the secretion of TSH, which in turn prompts a decrease in the output of TH by the thyroid gland (see Figure 17.6).

In positive feedback mechanisms, increasing levels of one hormone cause another gland to release a hormone. For example, the increased production of oestradiol (a female ovarian hormone) during the follicular stage of the menstrual cycle in turn stimulates increased FSH production by the anterior pituitary gland. Oestradiol levels continue to increase until the ovarian follicle disappears, eliminating the source of the stimulation for FSH, which then decreases.

Stimuli for hormone release may also be classified as hormonal, humoral or neural (see Figure 17.7). In hormonal release, hypothalamic hormones stimulate the anterior pituitary to release hormones. Fluctuations in the serum level of these hormones in turn prompt other endocrine glands to release hormones. In humoral release, fluctuations in the serum levels of certain ions and nutrients stimulate specific endocrine glands to release hormones to bring these levels back to normal. In neural release, nerve fibres stimulate the release of hormones.

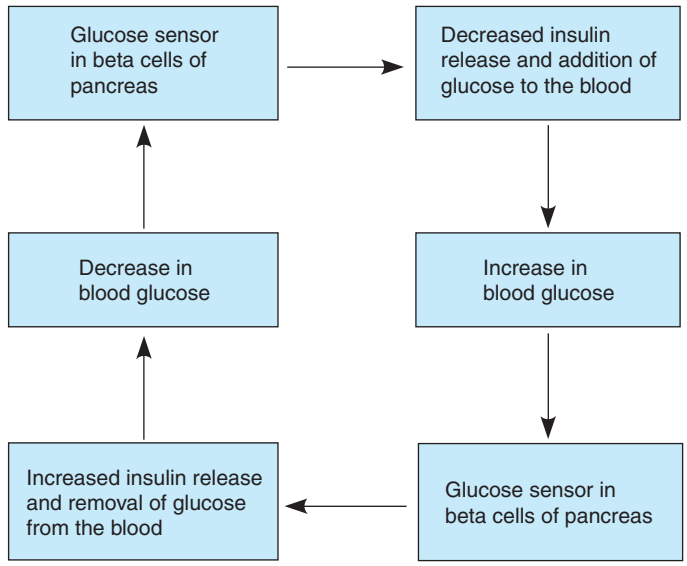


FIGURE 17.6 ■ Negative feedback

ASSESSING ENDOCRINE FUNCTION

The function of the endocrine glands is assessed by findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests. Hormones affect all body tissues and organs and manifestations of dysfunction are often non-specific, making assessment of endocrine function sometimes more difficult than assessment of other body systems.

Health assessment interview

A health assessment interview to determine problems with the endocrine system may be part of a health screening or total health assessment, or it may focus on a chief complaint (such as increased urination or changes in energy levels). If the person has a problem with endocrine function, the nurse analyses its onset, characteristics and course, severity, precipitating and relieving

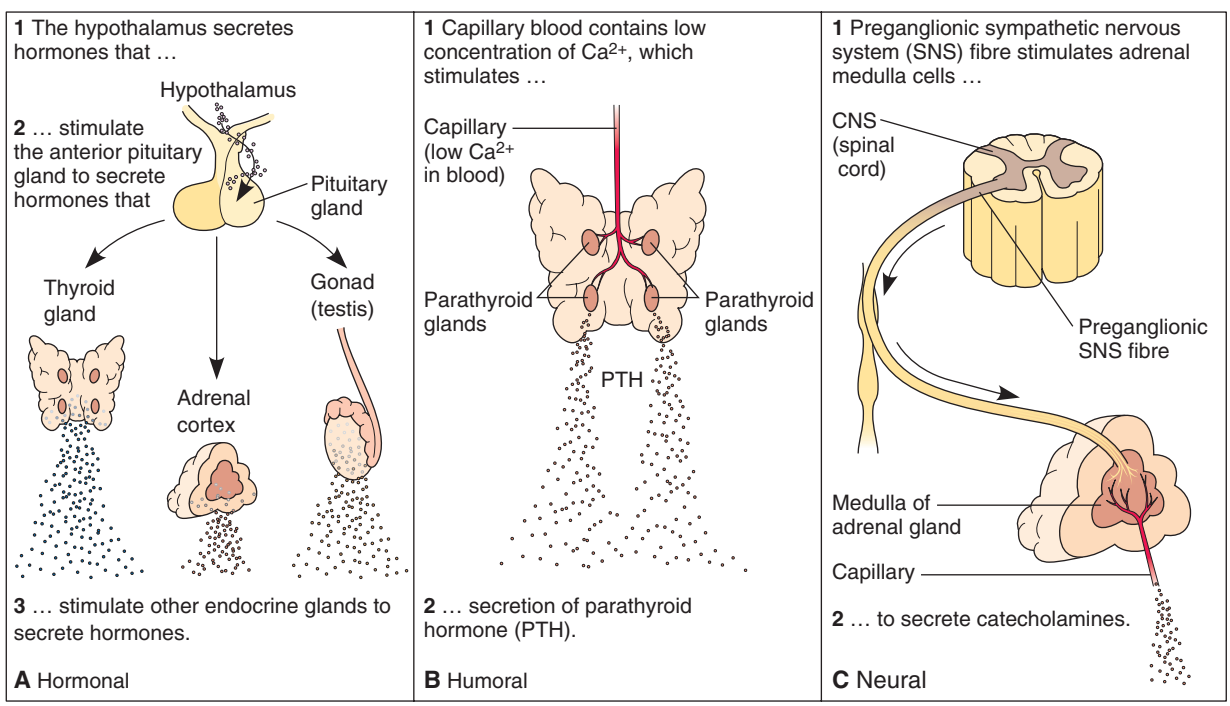


FIGURE 17.7 ■ Examples of three mechanisms of hormone release: *A*, hormonal; *B*, humoral; *C*, neural

factors, and any associated symptoms, noting the timing and circumstances. For example, the nurse may ask the person:

- Describe the swelling you noticed in the front of your neck. When did it begin? Have you noticed any changes in your energy level?
- When did you first notice that your hands and feet were getting larger?
- Have you noticed that your appetite has increased even though you have lost weight?

The health history includes information about the person's medical history, family history and social history. Ask the person about any changes in normal growth and development, as well as in height and weight. Changes in the size of extremities can often be detected by asking whether the person has had to have rings enlarged or buy increasingly larger shoes. Enlargement of the neck may be identified by asking whether the person has difficulty finding shirts or blouses with a collar that fits. Also explore changes such as difficulty swallowing; increased or decreased thirst, appetite and/or urination; visual changes; sleep disturbances; altered patterns of hair distribution (such as increased facial hair in women); changes in menstruation; changes in memory or ability to concentrate; and changes in hair and skin texture. Ask the person about any blow to the head, as well as previous hospitalisations, chemotherapy, radiation (especially to the neck) and the use of medications (especially hormones or steroids).

The nurse also asks about the person's occupational and social history. Include questions about the person's satisfaction with occupation, personal relationships and lifestyle. Other areas of assessment include the person's usual means of coping; use of alcohol, smoking or drugs; diet (including weight gain or loss); and exercise and sleep patterns. Although the person may not recognise changes in behaviour, family members may be able to provide important information.

Interview questions categorised by functional health patterns are listed below.

FUNCTIONAL HEALTH PATTERN INTERVIEW Endocrine system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Describe your overall state of health, rating it on a scale of 1 to 10 with 10 being the best health you have had. ■ Describe any problems you have had with an endocrine gland (pituitary, thyroid, parathyroid, adrenal, pancreas, ovaries, testes). ■ If you had a problem with any of these glands, how was it treated (medications, surgery, diet, hormone replacement)? ■ Has any relative experienced any problems with an endocrine gland (pituitary, thyroid, parathyroid, adrenal, pancreas, ovaries, testes)? ■ Do you smoke, drink alcohol, and/or use recreational drugs? If so, how much and what kind? ■ Have you ever been tested for high or low blood sugar?
Nutritional–Metabolic	■ Describe what you eat and how much (and what type of) fluid you drink in a 24-hour period. ■ Do you take any nutritional supplements, herbs or vitamins? ■ Have you noticed any change in your hunger or thirst? ■ Has your weight changed? If so, by how many kilograms and over what time period? ■ Have you noticed any change in your energy level? If so, explain. ■ Have you noticed any change in your ability to tolerate heat or cold? ■ Have you noticed any difficulty swallowing? Explain. ■ Have you noticed any changes in the texture of your skin? If so, what were they?
Elimination	■ Have you noticed any change in urine colour, odour or amount or in the frequency of urination? If so, describe it. ■ Have you ever had kidney stones? If so, how were they treated? ■ Has there been a change in your bowel elimination (such as diarrhoea or constipation)? If so, explain the change.

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Activity–Exercise	■ Describe your physical activities in a usual day. ■ Has your energy level increased or decreased? Explain. ■ Do some activities make you very tired? Explain how you feel.
Sleep–Rest	■ How many hours of sleep do you get each night? ■ Do you feel nervous and unable to rest? ■ Do you sweat at night?
Cognitive–Perceptual	■ Have you noticed any problem with your memory? ■ Do you feel restless, anxious or confused? ■ Have you noticed any change in your voice? ■ Have you noticed any change in the colour or condition of your skin and hair (colour, dryness, oiliness, bruises)? ■ Have you had any headaches, memory loss, changes in sensation, depression? If so, describe them. ■ Have you noticed any change in your vision? If so, describe them. ■ Have you had any heart palpitations? ■ Have you had any abdominal pain? What is it like and where is it located? ■ Have you had any pain or stiffness in your muscles and joints?
Self-perception–Self-concept	■ How does this condition make you feel about yourself? ■ How do you feel about taking medications?
Role–Relationships	■ How does this condition affect your relationships with others? ■ Does anyone in your family have an endocrine disorder? If so, when did it begin and how does it affect them? What family member is affected and at what age did it begin?
Stress tolerance	■ Does stress seem to make your condition worse? Explain. ■ Has this condition created stress for you? ■ Describe what you do when you feel stressed.
Value–Belief	■ Tell me how specific relationships or activities help you cope with this condition. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this condition. ■ Are there any specific treatments that you would not use to treat this condition?

Physical assessment

Physical assessment of the endocrine system may be performed as part of a total health assessment or it may be a focused assessment of the person with known or suspected problems with endocrine function. Sample documentation of an assessment of the thyroid gland is included in the accompanying box.

The only endocrine organ that can be palpated is the thyroid gland; however, other assessments that provide information about endocrine problems include inspection of the skin, hair, nails, facial appearance, reflexes and musculoskeletal system. Measuring and monitoring trends in height and weight and vital signs also provide clues to altered endocrine system function.

SAMPLE DOCUMENTATION
Assessment of the thyroid gland

A 37-year-old female presents at a community clinic with complaints of 'always feeling so hot', 'always hungry but losing weight' and 'can't sleep at night—too jittery'. Weight 53 kg (loss of 6 kg in last 3 months). BP 90/78, P 96. Skin very warm and moist. Anterior neck has diffuse enlargement. Thyroid gland enlarged bilaterally on palpation. Referral made to endocrine clinic for further evaluation.

The person may sit during the examination. A reflex hammer is used to test deep tendon reflexes. Prior to the examination, the nurse collects the necessary equipment and explains the

TABLE 17.2 Age-related endocrine changes

AGE-RELATED CHANGE	SIGNIFICANCE
Pituitary: ↓ production of ACTH, TSH, FSH	• Decreased secretion of glucocorticoids, 17-ketosteroids, progesterone, androgen and oestrogen (and thus lower levels on diagnostic tests)
Thyroid: ↑ in fibrosis and nodularity, ↓ in gland activity	• Lower basal metabolic rate • Increased incidence of hypothyroidism • Palpable nodules on palpation
Adrenal medulla: ↑ secretion and level of noradrenaline, increased plasma noradrenaline level, however ↓ beta-adrenergic response to noradrenaline	• Decreased response to beta-adrenergic and receptor blockers medications • May contribute to increased incidence of hypertension
Pancreas: calcification of blood vessels and distension and dilation of pancreatic ducts	• Decreased production of lipase with reduced fat absorption and digestion, leading to intolerance of fatty foods and indigestion • Decreased absorption of fat-soluble vitamins
Pancreas: delayed and decreased insulin release; believed accompanied by decreased sensitivity to circulating insulin	• Decreased ability to metabolise glucose with higher and more prolonged blood glucose levels may contribute to increased incidence of type 2 diabetes mellitus with ageing (however, higher than normal blood glucose levels are not unusual in non-diabetic older adults)

techniques to the person to decrease anxiety. Additional techniques for assessing hypocalcaemic tetany, a complication of endocrine disorders or surgery, are included in the examination sequence. Normal age-related changes in assessment findings are described in Table 17.2.

Diagnostic tests

The results of diagnostic tests of the endocrine system are used to support the diagnosis of a specific disease, to provide information to identify or modify the appropriate medication or therapy used to treat the disease, and to help nurses monitor the person's response to treatment and nursing care interventions. Diagnostic tests to assess the structure and function of the glands of the endocrine system are described in the box below and are summarised in the bulleted list that follows. Please note: normal reference values for all serum/urine tests vary slightly from laboratory to laboratory due to specific test kits used in each laboratory. More information is included in the discussion of specific disorders in Chapters 18 and 19.

- As a result of the many hormones produced by the pituitary gland and the number of target organs for those hormones, many direct and indirect diagnostic tests are used to determine pituitary function. The diagnostic tests described in this chapter include those of growth hormone (GH), somatomedin C, and causes of polyuria and pituitary tumours.
- Although a substantial number of diagnostic tests are used to identify and monitor thyroid function, the most accurate is TSH. Other tests of thyroid structure and function include thyroxine, triiodothyronine, thyroid antibodies, radioactive iodine uptake and thyroid scan.
- Diagnostic tests of the parathyroid hormone, which regulates serum calcium and phosphate levels, include PTH and serum calcium.
- Diagnostic tests for the adrenal glands assess the glucocorticoids, mineralocorticoids and androgens through both blood and 24-hour urine studies. The tests are of cortisol, aldosterone, ACTH, 17-ketosteroids and CT of the abdomen to identify adrenal gland tumours.
- Diagnostic tests of the pancreas are performed primarily to identify, confirm and monitor glucose levels in the person with diabetes mellitus. Those described are the oral glucose tolerance test (OGTT), fasting plasma glucose (FPG), glycosylated haemoglobin (HbA1c) and CT of the abdomen to identify pancreatic tumours or cysts.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, for assessing for medication use that may affect the outcome of the tests, for supporting the person during the examination as necessary, for documenting the procedures as appropriate and for monitoring the results of the tests.

DIAGNOSTIC TESTS The endocrine system

PITUITARY TESTS

NAME OF TEST Growth hormone (GH), Human growth hormone (hGH)

PURPOSE AND DESCRIPTION In this blood test, GH levels (affected by food, stress and activity) are measured to identify GH deficiency (dwarfism) or GH excess (gigantism, acromegaly).

Normal value:
Men: < 5 μg/L
Women: < 10 μg/L

RELATED NURSING CARE Inform the person not to eat or drink 8–10 hours prior to having blood taken. Have the person sit down for 30 to 60 minutes before blood is taken.

DIAGNOSTIC TESTS The endocrine system (continued)

NAME OF TEST IGF-1 (Somatomedin C or Insulin-like growth factor)

PURPOSE AND DESCRIPTION The results of this blood test are used to identify diseases or conditions caused by hyper/hyposecretion of growth hormone and to evaluate the function of the pituitary gland.

Normal value:
Adult: 42–110 ng/mL
Results vary by age for children

RELATED NURSING CARE None; overnight fasting is preferred but not necessary.

NAME OF TEST Water deprivation test (Anti-diuretic hormone stimulation)

PURPOSE AND DESCRIPTION This combination blood and urine test is used to identify causes of polyuria (increased urine output), including central diabetes insipidus (DI), neurogenic diabetes insipidus, syndrome of inappropriate antidiuretic hormone (SIADH) and psychogenic polydipsia. ADH or vasopressin is given intramuscularly (IM) or subcutaneously (SC). In people without pathology, there is no change in urine and plasma osmolality. Urine osmolality increases in central diabetes insipidus and decreases in nephrogenic diabetes insipidus.

RELATED NURSING CARE The recommended fluid restriction prior to this test is dependent on the person's current urine output. Inform the person that the test will take up to 8 hours. Every hour for ordered length of test: assess weight, take postural BP (lying and standing measures separated by 2 minutes), assess urine for volume and specific gravity, and send samples of urine to the lab for osmolality. Blood samples for osmolality are taken when urine osmolality shows an increase of less than 30 mOsm/kg for 3 consecutive hours. Vasopressin is administered and urine osmolality is measured 30 to 60 minutes later.

Following the test the person needs to be rehydrated with oral fluids and have vital signs monitored.

NAME OF TEST Magnetic resonance imaging (MRI)

PURPOSE AND DESCRIPTION This radiographic study is done to identify tumours of the hypothalamus or pituitary gland.

RELATED NURSING CARE Inform the person of need to lie still during the examination. Remove any metallic objects (such as hair clips, jewellery) and assess for any metallic implants (such as pacemakers, body piercings, shrapnel). If present, the test is not performed due to the danger caused by the magnetic field from the machine.

THYROID TESTS

NAME OF TEST Thyroid-stimulating hormone (TSH)

PURPOSE AND DESCRIPTION In this blood test, TSH and T_4 levels are measured to differentiate pituitary from thyroid causes of hypothyroidism. A decreased T_4 level and a normal or increased TSH level can indicate a thyroid disorder. A decreased T_4 level and a decreased TSH level can indicate a pituitary disorder.

Normal value:
0.5–4.5 mIU/L

RELATED NURSING CARE Inform the person to avoid shellfish for several days prior to the test. Evaluate medications: TSH value may be increased by aspirin, steroids, dopamine and heparin; and decreased by lithium and potassium iodide.

NAME OF TEST Thyroxine (Free T_4)

PURPOSE AND DESCRIPTION This blood test is done to determine thyroid function and aid in the diagnosis of hyperthyroidism and hypothyroidism.

Normal value:
Adult: 10–36 pmol/L

RELATED NURSING CARE Assess medications: value may be decreased by cortisone, chlorpromazine (Largactil), phenytoin (Dilantin), heparin, lithium, sulfonamides (Serpasil), testosterone and propranolol (Inderal). Values may be increased by aspirin, iodine, oral contraceptives and oestrogens.

NAME OF TEST Triiodothyronine (Total T_3)

PURPOSE AND DESCRIPTION This blood test is used to diagnose hyperthyroidism and to compare T_3 with T_4 for diagnosis of thyroid disorder. It is useful in monitoring the effectiveness of thyroid replacement and suppressive therapy.

Normal value:
Adult 20–50 years: 1.2–3.4 mmol/L
Adult >50 years: 0.6–2.8 mmol/L

RELATED NURSING CARE Evaluate medications: value can be decreased by propylthiouracil, lithium, phenytoin (Dilantin), propranolol (Inderal), large doses of aspirin, steroids and sulfonamides. Value can be increased by oestrogens, progestins, oral contraceptives, T_3 and methadone; it will also be increased during pregnancy.

(continued)

DIAGNOSTIC TESTS The endocrine system (continued)

NAME OF TEST Triiodothyronine resin uptake (T_3RU)

PURPOSE AND DESCRIPTION This blood test is an indirect measure of free thyroxine (T_4). The person's blood is mixed with radioactive T_3 and synthetic resin, and the radioactive T_3 will bind with available thyroid-binding globulin sites. The unbound radioactive T_3 is added to resin for T_3 uptake. In hyperthyroidism there are few binding sites left; more T_3 is taken up by the resin and a high T_3 resin uptake results. The opposite occurs in hypothyroidism.

Normal value:
24–34% uptake

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Thyroid antibodies (TA)

PURPOSE AND DESCRIPTION A blood test used to identify thyroid immune disease (Graves' disease, chronic thyroiditis, Hashimoto's thyroiditis).

Normal values:
Antithyroglobulin: negative to titre <1:20
Antimicrosomal: negative to titre <1:100

RELATED NURSING CARE Assess for family history of thyroid disease and ask about recent viral infection (which could trigger autoimmune disease).

NAME OF TEST Radioactive iodine uptake (Radioimmunoassay or RIA)

PURPOSE AND DESCRIPTION This test provides a direct measure of thyroid activity and is useful in evaluating the activity of solitary thyroid nodules. Based on the rationale that the thyroid gland takes up iodine in any form, radioactive iodine is given orally or intravenously and the thyroid gland uptake is measured with a scanner at several hourly intervals and at 24 hours.

Normal value for uptake:
2–4 hours: 3–19%
24 hours: 11–30%

RELATED NURSING CARE The person should not eat or drink for 6 to 8 hours before the test, but can have food 1 hour after the oral dose is given. Advise the person not to take supplemental iodine several weeks before the test. Thyroid medications and amiodarone interfere with the test.

NAME OF TEST Thyroid scan

PURPOSE AND DESCRIPTION This radiological study evaluates thyroid nodules. Radioactive isotopes are given orally or intravenously and a scanner is passed over the thyroid to make a graphic record of the radiation emitted. A normal thyroid scan has a homogeneous pattern of radiation with symmetric lobes. Benign lesions appear as warm spots (take up more radiation); malignant tumours appear as cold spots (less radiation taken up).

RELATED NURSING CARE No special preparation is needed.

PARATHYROID TESTS

NAME OF TEST Parathyroid hormone (PTH)

PURPOSE AND DESCRIPTION A blood test done to identify hypoparathyroidism or hyperparathyroidism; also used to monitor response to PTH therapy.

Normal value:
Intact PTH: 10–65 ng/L
C-terminal PTH: 50–330 ng/L
N-terminal PTH: 8–24 ng/L

RELATED NURSING CARE Advise the person not to eat or drink for 8 hours before the test.

NAME OF TEST Calcium (Ca)

PURPOSE AND DESCRIPTION This blood test is used to check for serum calcium excess or deficit in parathyroid and bone disorders, and to monitor calcium levels.

Normal value:
2.3–2.8 mmol/L

RELATED NURSING CARE Assess for manifestations of tetany, including positive Chvostek's and Trousseau's signs, if hypocalcaemia is present.

ADRENAL TESTS

NAME OF TEST Cortisol

PURPOSE AND DESCRIPTION A blood test is done to measure amount of total cortisol in the serum and evaluate adrenal cortex function. It is decreased in Addison's disease and hypothyroidism; increased in Cushing's syndrome and hyperthyroidism.

Normal value:
8 am–10 am: 138–635 nmol
4 pm–6 pm: 83–359 nmol

A 24-hour urine test may be conducted to measure free (unbound) cortisol.

Normal value:
<276 nmol/24 hours

DIAGNOSTIC TESTS The endocrine system (continued)

RELATED NURSING CARE Bloods are usually taken at 0800 hours and again at 1600 hours. The results are compared with the 1600 hour level and should be between one- and two-thirds the level of the 0800 hour blood level. Evaluate medications: cortisol is decreased by androgens and phenytoin (Dilantin), and increased by oral contraceptives, oestrogen, cortisone and spironolactone (Aldactone).

Instruct the person how to save urine for a 24-hour period, to eat a low-sodium diet before the test and to avoid stressful situations and physical activity for at least 24 hours prior to the test. You do not need to measure each urine specimen. Ensure the urine specimen is kept on ice or refrigerated. Assess medications; values may be increased by spironolactone (Aldactone), hydrocortisone and oral contraceptives.

NAME OF TEST Aldosterone

PURPOSE AND DESCRIPTION This blood test is done to identify hyperaldosteronism and to compare blood and urine levels with other lab data to evaluate overhydration with increased sodium and adrenal malfunction.

Normal value:
Men: 0.17–0.61 nmol/L
Women: 0.14–0.80 nmol/L

A 24-hour urine test is considered a more reliable measure of aldosterone than a random aldosterone test.

Normal value:
6–72 nmol/L/24 hours

RELATED NURSING CARE Usually the person must sit upright for a period of 2 hours prior to the test. The person should be instructed to follow a diet with normal levels of sodium for 2 weeks prior to the test. Assess diet and lab results: levels are increased by hyponatraemia, hyperkalaemia and a low-salt diet. Assess medications: values are increased by diuretics, hydralazine (Apresoline), nitroprusside and oral contraceptives. Values are decreased by propanolol (Inderal), ACE inhibitors and licorice.

NAME OF TEST Adrenocorticotropic hormone (ACTH)

PURPOSE AND DESCRIPTION This blood test is done to determine if a decreased plasma level of cortisol is due to adrenal cortex hypofunction or pituitary hypofunction.

Normal value:
7 am–10 am: <18 pmol/L
4 pm: <11 pmol/L

RELATED NURSING CARE Advise the person that food and fluids may be restricted and to eat a low-carbohydrate diet for 24 hours prior to the test. Assess medications: ACTH values may be increased by metyrapone, vasopressin and insulin; and decreased by steroids, oestrogen amphetamines and alcohol.

NAME OF TEST ACTH stimulation

PURPOSE AND DESCRIPTION Performed to check for pituitary hypofunction. The drug metyrapone (Metopirone) is given to block the production of cortisol, thus causing an increased ACTH secretion.

If the ACTH level does not increase, the problem is pituitary insufficiency.

RELATED NURSING CARE Assess medications as for ACTH test.

NAME OF TEST ACTH suppression

PURPOSE AND DESCRIPTION Performed to check the origin of the condition. The drug dexamethasone (Dexmethsone) is given to suppress ACTH production. If an extremely high dose is needed, the cause is of pituitary origin; if the plasma cortisol continues to be high with ACTH suppression, the cause could be adrenal cortex hyperfunction (Cushing's syndrome). Normally, the plasma cortisol level should double in 1 hour.

RELATED NURSING CARE Advise the person to avoid caffeinated drinks and chocolates; no other food or fluid restriction is needed. Assess medications: false positives may be caused by phenytoin, barbiturates, meprobamate and carbamazepine. If dexamethasone causes gastric irritation, milk or antacids may be required.

NAME OF TEST 17-Ketosteroids

PURPOSE AND DESCRIPTION This 24-hour urine test is done to measure metabolites in urine and evaluate adrenal cortex function.

Normal value:
Men: 20–70 µmol in 24 hours
Women: 20–60 µmol in 24 hours

RELATED NURSING CARE Teach the person how to save urine. (Urine must contain a preservative and be refrigerated.) Assess medications and refer to information about the test. Levels are affected by a variety of medications; if possible, these should be discontinued for 48 hours before the test. Women cannot have the test while menstruating because blood can cause a false positive finding.

(continued)

DIAGNOSTIC TESTS The endocrine system (continued)

NAME OF TEST Computed tomography (CT) of the abdomen

PURPOSE AND DESCRIPTION This radiological study is used to assess for tumours (including size and metastasis).

RELATED NURSING CARE Determine if contrast medium will be used; if so, assess the person for an allergy to iodine (shellfish).

PANCREATIC ENDOCRINE TESTS

NAME OF TEST Fasting plasma glucose (FPG)

PURPOSE AND DESCRIPTION This blood test is used to identify or confirm a diagnosis of type 2 diabetes. It is also used to monitor treatment of diabetes mellitus. A finding of 7.0 mmol/L or above is now considered to be consistent with a diagnosis of type 2 diabetes. When looking to diagnose a person with type 2 diabetes, an oral glucose tolerance test should also be performed when a person has received a FPG result of 5.5–6.9 mmol/L. Additionally, diagnosis needs to be confirmed by two positive FPG levels on separate days.

Value:
Normoglycaemia: <6.1 mmol/L
Impaired glucose tolerance: 6.1–7 mmol/L
Diabetes: >7 mmol/L
(World Health Organization & International Diabetes Federation, 2006).

RELATED NURSING CARE Advise the person not to eat or drink anything other than water for at least 8 hours before the test. Do not administer insulin or oral hypoglycaemic medications until blood specimen is taken. Assess medications: FPG may be increased by cortisone, diuretics, ACTH, levodopa, anaesthetics and phenytoin (Dilantin).

NAME OF TEST Oral glucose tolerance test (OGTT or GTT)

PURPOSE AND DESCRIPTION Performed to diagnose diabetes mellitus if prior fasting plasma glucose findings are increased or inconsistent. It is also commonly used to diagnose gestational diabetes.

NURSING IMPLICATIONS The tests will not be done if the person's FPG is consistently high (>11 mmol/L). The person drinks a solution of 75 to 100 g of glucose, and samples of blood and urine are taken immediately and at 30, 60 and 120 minutes (or it may extend from 3 to 4 hours).

Reference values:

Time	Serum glucose level
Fasting	3.9–6.1 mmol/L
0.5 hour	<11.1 mmol/L
1 hour	<11.1 mmol/L
2 hours	<7.8 mmol/L
3 hours	Fasting level

RELATED NURSING CARE Advise the person that food, fluids (except water) and smoking are not allowed during the test or 12 hours prior to the test. Advise the person they must not exercise during the test. Assess medications: drugs that may increase OGTT levels are steroids, oral contraceptives, oestrogens, thiazide diuretics and salicylates. Explain to the person that they may feel weak and may perspire during the test and that they should report these symptoms to the nurse. Although they usually are transitory, these symptoms may be manifestations of hyperinsulinism.

NAME OF TEST Glycated haemoglobin (HbA1c)

PURPOSE AND DESCRIPTION This blood test is used to measure the effectiveness of treatment of diabetes mellitus. The results represent an average blood glucose level during a 2- to 3-month period; an elevated level indicates uncontrolled diabetes mellitus and increased risk of complications. It is not currently recommended for diagnosis of diabetes.

Good diabetic control: 2.5–5.9% of total haemoglobin

RELATED NURSING CARE Monitor findings: decreased levels can be caused by anaemias, long-term blood loss and chronic renal failure. Increased levels may result from hyperglycaemia, alcohol ingestion, pregnancy, haemodialysis and prolonged cortisone intake.

Genetic considerations

When conducting a health assessment interview and physical assessment, it is important for the nurse to consider genetic influences on the health of the adult. During the health assessment interview, ask about endocrine disorders in immediate family members, including the family member's age of onset and gender. Ask the person about a family history of such diseases as diabetes mellitus, diabetes insipidus, thyroid disorders, growth problems, hypertension and obesity. Ask women about problems with pregnancy, menstruation and/or menopause.

During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see box below). If data indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

GENETIC CONSIDERATIONS
Endocrine system

- Type 1 and type 2 diabetes mellitus are classified as multifactorial inheritance disorders because both genetic and environmental factors are necessary for onset of the disorder.
- Pendred syndrome is an inherited disorder in which people have hereditary deafness and a thyroid goitre.
- Hashimoto's disease (chronic thyroiditis) is believed to have a genetic component.
- Multiple endocrine neoplasia is a group of rare diseases caused by genetic defects leading to hyperplasia and hyperfunction of two or more components of the endocrine system (especially the parathyroid, pancreas and pituitary glands).

ENDOCRINE ASSESSMENTS

Technique/normal findings	Abnormal findings
Skin assessment	
Inspect skin colour. *Skin colour should be even and appropriate to the person's age and race.*	■ Hyperpigmentation may be seen in peoples with Addison's disease or Cushing's syndrome. ■ Hypopigmentation may be seen in people with diabetes mellitus, hyperthyroidism or hypothyroidism. ■ A yellowish cast to the person's skin might indicate hypothyroidism. ■ Purple striae over the abdomen and bruising may be present in the person with Cushing's syndrome.
Palpate the skin, assessing texture, moisture and the presence of lesions. *Skin should be appropriate to the person's race, smooth, warm, dry and intact without abnormal lesions.*	■ Rough, dry skin is often seen in people with hypothyroidism, whereas smooth and flushed skin can be a sign of hyperthyroidism. ■ Lesions (such as ulcerations) on the person's lower extremities might indicate diabetes mellitus.
Nails and hair assessment	
Assess texture, distribution and condition of nails and hair. *Hair should be of normal texture, appropriately distributed for gender; nail surfaces should have even colour with smooth surfaces.*	■ Increased pigmentation of the nails is often seen in people with Addison's disease. ■ Dry, thick, brittle nails and hair may be apparent in people with hypothyroidism; thin, brittle nails and thin, soft hair may be apparent in people with hyperthyroidism. ■ Hirsutism (excessive facial, chest or abdominal hair) may be seen in people with Cushing's syndrome.
Facial assessment	
Inspect the symmetry and form of the face. *Face should be bilaterally symmetrical.*	■ Variations of form and structure may indicate growth abnormalities such as **acromegaly** (continued growth of bone from growth hormone hypersecretion).
Inspect position of eyes. *Eyes should be equal in position on both sides of the face. Eyelids should close over eyes.*	■ **Exophthalmos** (protruding eyes) may be seen in people with hyperthyroidism.

ENDOCRINE ASSESSMENTS (continued)

Technique/normal findings	Abnormal findings
Thyroid gland assessment	
Palpate the thyroid gland for size and consistency. Stand behind the person and place your fingers on either side of the trachea below the thyroid cartilage (see Figure 17.8). Ask the person to tilt their head to the right. Now ask the person to swallow. As the person swallows, displace the left lobe while palpating the right lobe. Repeat to palpate the left lobe. *Thyroid gland is not usually palpable. If it is, lobes should feel smooth, rubbery and free of nodules.*	■ The thyroid may be enlarged in people with Graves' disease or a **goitre** (enlarged thyroid gland). ■ Multiple nodules may be seen in metabolic disorders, whereas the presence of only one nodule may indicate a cyst or a benign or malignant tumour. ■ One enlarged nodule suggests malignancy.
Motor function assessment	
Assess the deep tendon reflexes. Deep tendon reflexes are assessed with the reflex hammer and include the biceps reflex, brachioradialis reflex, triceps reflex, patellar reflex and Achilles reflex. *Normal values range from 1+ (present, but decreased) to 2+ (normal) to 3+ (increased). See Chapter 40 for guidelines and illustrations of deep tendon reflex assessment.*	■ Increased reflexes may be seen in people with hyperthyroidism; decreased reflexes may be seen in people with hypothyroidism.
Sensory function assessment	
Test the person's sensitivity to pain, temperature, vibration, light touch and stereognosis (the ability to identify an object merely by touch). Compare symmetrical areas on both sides of the body and compare the distal to the proximal regions of the extremities. Ask the person to close their eyes. *Sensory function should be bilaterally intact.* ■ To test pain, use the blunt and sharp ends of a new safety pin. Discard the pin after use. ■ To test temperature, use cups or other containers of cold and hot water. ■ To test vibration, use a tuning fork over one of the person's finger or toe joints. ■ To test light touch, use a cotton tip. ■ To test stereognosis, place in the person's hand a simple, familiar object, such as a rubber band, cotton ball or button. Ask the person to identify the object.	■ Peripheral neuropathy and paresthesias (altered sensations) may occur in people with diabetes, hypothyroidism or acromegaly.
Musculoskeletal assessment	
Inspect the size and proportions of the person's body structure. *Size and proportion of body structures should be bilaterally equal.*	■ Extremely short stature may indicate **dwarfism**, which is caused by insufficient growth hormone. ■ Extremely large bones may indicate acromegaly, which is caused by excessive growth hormone.

FIGURE 17.8 ■ Palpating the thyroid gland from behind the person

Technique/normal findings	Abnormal findings
Assessing for hypocalcaemic tetany	
Assess for **Trousseau's sign** (a test for hypocalcaemia) with resulting tetany (tonic muscle spasms) by inflating a blood pressure cuff above the antecubital space to a point greater than systolic blood pressure for 2–5 minutes. Trousseau's sign is discussed in relation to hypocalcaemia in Chapter 9. *A normal finding would be no carpal spasm in response to compression of the arm by the blood pressure cuff.*	■ Decreased calcium levels cause the persons hand and fingers to contract (**carpal spasm**).
Assess for **Chvostek's sign** (a test for hypocalcaemia) by tapping your finger in front of the person's ear at the angle of the jaw. A positive Chvostek's sign causes facial grimacing due to repeated contractions of the facial muscle. Chvostek's sign is discussed and illustrated in relation to hypocalcaemia in Chapter 9. *A normal finding would be no facial grimacing in response to tapping the person's face in front of the ear.*	■ Decreased calcium levels cause the person's lateral facial muscles to contract.

CONCEPT CHECK

1 What physiological response is expected if the pituitary gland produces an increased amount of ADH?
1 increased output of urine
2 decreased output of urine
3 increased facial hair growth in women
4 decreased production of testosterone

2 What assessment might be made to identify low calcium levels?
1 Save urine to measure 17-ketosteroids.
2 Palpate turgor of skin.
3 Conduct a Trousseau's sign test.
4 Observe colour of skin.

3 Excessive amounts of glucocorticoids, produced by the adrenal cortex, result in what pathophysiological health problem?
1 inhibited immune response
2 increased response to glucagon
3 delayed onset of puberty
4 decreased metabolic rate

4 When conducting a health history focused on the endocrine system, which of the following questions should be included?
1 'When did you first notice the pain in your abdomen?'
2 'Do your children have problems with urination?'
3 'Have you noticed a change in your thirst?'
4 'How did you get this scar on your leg?'

5 What assessments are made when palpating the thyroid gland?
1 oedema and movement
2 size and consistency
3 character and texture
4 pain and pulse rate

6 How many times does a FPG test showing an elevated level need to be performed to confirm a diagnosis of diabetes?
1 once only
2 twice, once in the morning and then in the afternoon
3 never, a glycated haemoglobin test is more accurate
4 twice, on two separate days

7 Which of the following tests is the most accurate indicator of thyroid function?
1 GH
2 FPG
3 aldosterone
4 TSH

8 Which is the only endocrine organ that can be palpated during physical assessment?
1 pancreas
2 liver
3 thyroid
4 pituitary

9 You are caring for a person with newly diagnosed hyperthyroidism. What might you find in an assessment?
1 increased thick hair growth
2 exophthalmos
3 decreased reflexes
4 rough, dry skin

10 What endocrine disorder might be assessed by testing deep tendon reflexes?
1 Cushing's syndrome
2 acromegaly
3 tetany
4 hyperthyroidism

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CHAPTER 18

NURSING CARE OF PEOPLE WITH ENDOCRINE DISORDERS

SANDRA CAMPBELL-CROFTS

LEARNING OUTCOMES

- Compare and contrast the manifestations of disorders that result from hyper- and hypofunction of the thyroid gland.
- Describe the pathophysiology, manifestations and nursing care of the person with hyper- and hypofunction of the parathyroid glands.
- Discuss common disorders of the adrenal glands, incorporating pathophysiology, manifestations and associated nursing care.
- Compare and contrast common disorders of the pituitary gland and the resulting manifestations.

CLINICAL COMPETENCIES

- Assess the functional health status of people with endocrine disorders, and monitor, document and report abnormal manifestations.
- Use evidence-based research to provide appropriate teaching for self-medicating with thyroid hormone.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with endocrine disorders.
- Provide education to ensure the person knows that hormone replacement is for life and how to take medications efficiently and effectively.
- Monitor respiratory function after a thyroidectomy.
- Monitor for latent tetany following parathyroid removal—planned or inadvertent.
- Anticipate and recognise the effects of adrenal hormones.
- Revise the plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to the person with endocrine disorders.

KEY TERMS

The thyroid, parathyroid, adrenal and pituitary glands are part of the endocrine system. Disorders of the structure and function of these glands alter normal hormone levels and the way body tissues use those hormones. When hormone production increases or decreases, people experience alterations in health.

People with disorders of the endocrine glands discussed in this chapter require nursing care for multiple problems. Nursing care is directed towards meeting physiological needs, providing education and ensuring psychological support for the person and family. A holistic approach to the complex needs of people with these endocrine disorders is an essential component of nursing care.

DISORDERS OF THE THYROID GLAND

The thyroid uses iodine to secrete thyroid hormone (TH). TH is of vital importance for both the development and the maintenance of brain function and other major organ systems (Warner & Mittag, 2012).

Thyroid disorders—both hyperthyroidism and hypothyroidism—are among the most common endocrine disorders. Thyroid disease is the second most common endocrine condition in women of childbearing age (Medenica et al., 2015). In Australia, thyroid diseases affect about 850 000 people (7.5% of women and 1.5% of men) and the prevalence of these conditions rises with age. Fortunately, thyroid conditions respond well to treatment (Thyroid Australia, 2011a).

PEOPLE WITH HYPERTHYROIDISM

Hyperthyroidism or **thyrotoxicosis** is a disorder caused by excessive functional activity of the thyroid gland. Because the primary effect of TH is to increase metabolism and protein synthesis, hyperthyroidism is characterised by increased basal metabolism, weight loss despite an increased food intake and alterations of the autonomic nervous system. The increase in metabolic rate and the alterations in cardiac output include sinus tachycardia and increased peripheral blood flow, oxygen consumption and body temperature (Molina, 2009; Porth & Matfin, 2009).

Elevated TH levels also increase carbohydrate, protein and lipid metabolism. Lipids are depleted and glucose tolerance decreases (Wang, 2013).

Pathophysiology and manifestations

Hyperthyroidism results from many different factors, including autoimmune thyroid stimulation as in Graves' disease (Segni et al., 2014); excess secretion of thyroid-stimulating hormone (TSH) by the pituitary gland; thyroiditis; non-malignant neoplasms such as toxic multinodular goitre; and an excessive intake of thyroid medications. The most common aetiologies of hyperthyroidism in Australia are Graves' disease and toxic multinodular goitre.

The person with hyperthyroidism typically has an increased appetite, yet loses weight, and may have increased bowel motility without diarrhoea. Additional manifestations related to hypermetabolism include increased nervousness or irritability, heat intolerance, insomnia, palpitations and increased sweating. There is an increased risk of fractures (Blum et al., 2015). The skin is smooth and warm, hair may become fine and hair loss in the scalp, eyebrow, axillary or pubic areas of the body is common. Emotional lability is also common. The 'Multisystem effects of hyperthyroidism' are shown opposite.

Graves' disease

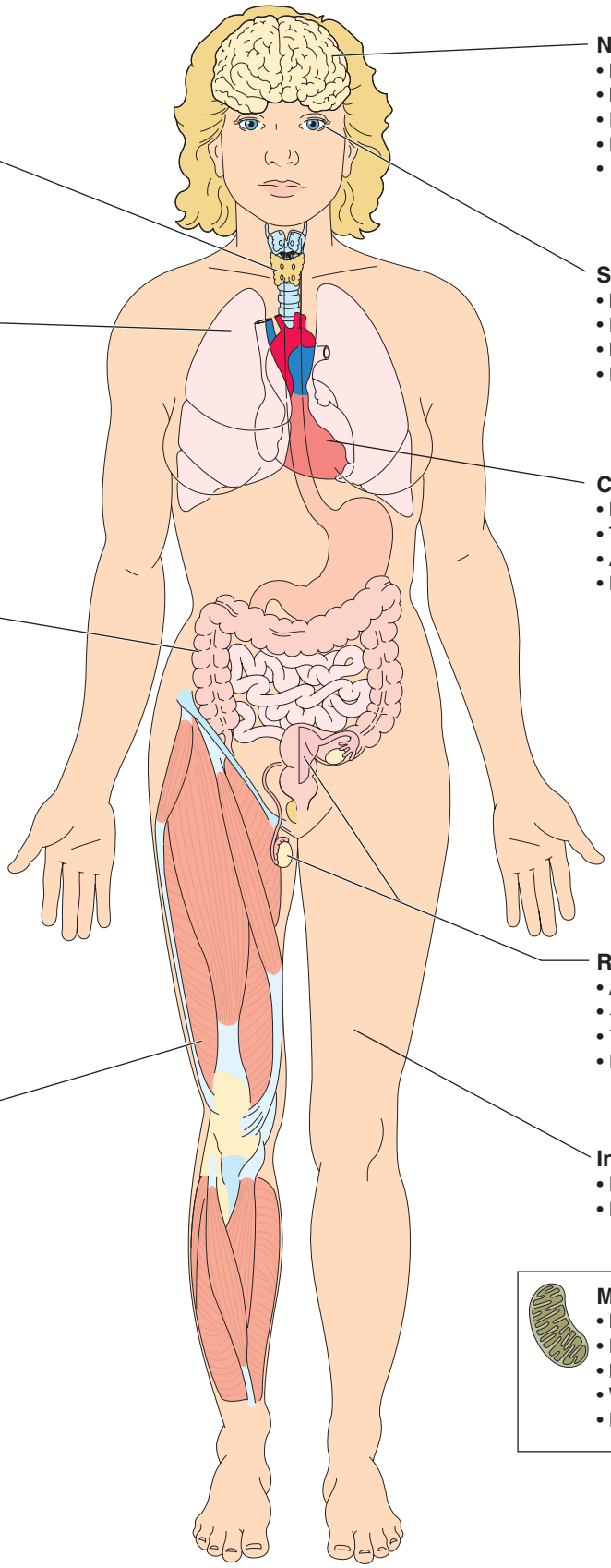
Graves' disease is the most common cause of hyperthyroidism and one of the most common autoimmune disorders in Australia, affecting 0.5% of the population (Campbell & Doogue, 2012). It is caused by a defect in immunoregulation in genetically predisposed individuals, leading to thyroid hyperplasia and an increased production of thyroid-stimulating hormone receptor antibodies (Campbell & Doogue, 2012). It is sometimes associated with the presence of other autoimmune disorders such as polyglandular autoimmune syndrome (PGA), myasthenia gravis and pernicious anaemia (Porth & Matfin, 2009; Pouye et al., 2014).

Graves' disease is seen five times more often in women than in men and occurs most frequently between the ages of 20 and 40. It is seen worldwide, with the incidence often correlated with the amount of iodine in the diet. Increased iodine intake (such as from radiocontrast dyes used in diagnostic tests, ingestion of supplement iodine tablets or medications such as amiodarone that contains 39% iodine by weight) have been associated with this disorder. Smoking, psychological stress and the postpartum period are also associated with Graves' disease.

People with Graves' disease have an enlarged diffuse thyroid gland (**goitre**) and manifestations of hyperthyroidism (as shown in Table 18.1). Goitre may be present as a consequence of both iodine deficiency and excess, and is evident in both hyperthyroidism and hypothyroidism (Zimmermann & Boelaert, 2015).

The common ophthalmopathy of Graves' disease is manifested as proptosis and visual dysfunction. **Proptosis** (forward displacement) of the eyeball occurs in about one-third of cases (Porth & Matfin, 2009). The forward protrusion (**exophthalmos**) results from an accumulation of inflammation by-products in the retro-orbital tissues. Often the sclera is visible above the iris. The upper lids are often retracted and the person has a characteristic unblinking stare (see Figure 18.1). In some cases proptosis may involve only one eye. Due to stretching or compression of the optic nerve, the person may experience blurred vision, diplopia, eye pain, lacrimation and photophobia. The inability to close the eyelids completely over the protruding eyeballs increases the risk of corneal dryness, irritation, infection and ulceration. Infiltration of the muscles that move the eye and of the optic nerve leads to

MULTISYSTEM EFFECTS OF HYPERTHYROIDISM



Endocrine
- Goitre

Respiratory
- Dyspnoea

Gastrointestinal
- Nausea
- Vomiting
- Diarrhoea
- Abdominal pain

Musculoskeletal
- Muscle wasting
- Weakness
- Fatigue

Neurological
- Hand and eye tremors
- Nervousness
- Insomnia
- Emotional lability
- ↑ reflexes

Sensory
- Blurred vision
- Photophobia
- Lacrimation
- Exophthalmos (Graves' disease)

Cardiovascular
- Hypertension
- Tachycardia
- Arrhythmias
- Palpitations

Reproductive
- Amenorrhoea (female)
- ↓ fertility (female)
- ↑ libido (male)
- Impotence (male)

Integumentary
- Fine, thin hair
- Flushed, moist skin

Metabolic processes
- Hyperthermia
- Diaphoresis
- Hunger
- Weight loss
- Fluid volume deficit

TABLE 18.1 Laboratory findings in hyperthyroidism

TEST	NORMAL VALUES	FINDINGS
Serum TA	≤ 1:10 titre	Increased
Serum TSH (sensitive assay)	0.27–4.2 mU/L	Decreased in primary hyperthyroidism
Serum T_4	Total 12–22 pmol/L	Increased
Serum T_3	Free 3–7.8 pmol/L	Increased
T_3 uptake (T_3RU)	25% to 35%	Increased
Thyroid suppression		Increased RAI uptake and T_4 levels

Source: South-Western Sydney Area Health Service (SSWAHS) Liverpool Hospital Pathology Department (2012). Reproduced with permission.
mU: microunits; pmol: picomoles.

paralysis and vision loss. The treatment of Graves' disease generally does not reverse these changes in the eyes.

A rare characteristic dermopathy of Graves' disease is **pretibial myxoedema** which occurs in 1% to 2% of people. Plaques and nodules develop bilaterally over the shins and dorsal surface of the feet. These plaques are oedematous, erythematous and sometimes hyperpigmented. Like the ophthalmopathy, the skin changes often persist despite successful treatment (Fatourechi, 2005).

Other manifestations include anaemia, vomiting, jaundice, fatigue, difficulty sleeping, hand tremors, increased bowel movements and changes in menstruation ranging from decreased flow to amenorrhoea. Twenty per cent of older people may present with atrial fibrillation, angina or congestive heart failure as a result of pulmonary hypertension.

Toxic multinodular goitre

Toxic multinodular goitre (see Figure 18.2) is a non-malignant tumour characterised by small, discrete, independently functioning nodules in the thyroid gland that secrete excessive amounts of TH. The aetiology includes a suspected genetic mutation of follicle cells as well as a deficiency of iodine (Zimmermann & Boelaert, 2015). People with this type of hyperthyroidism are usually women in their sixties or seventies who have had goitre for a number of years.

Excess TSH stimulation

Overproduction of TSH by the pituitary usually stimulates the thyroid gland to produce excess TH. The elevation in TSH

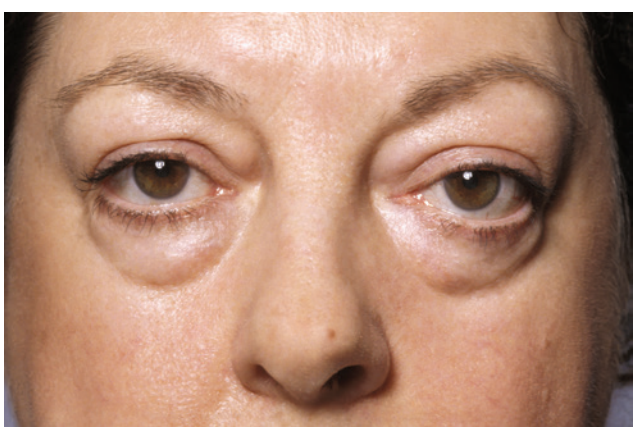


FIGURE 18.1 ■ Exophthalmos in a person with Graves' disease. The disorder causes oedema of fat deposits behind the eyes and inflammation of the extraocular muscles. The accumulating pressure forces the eyes outward from their orbits

Source: © Medical-On-Line/Alamy.

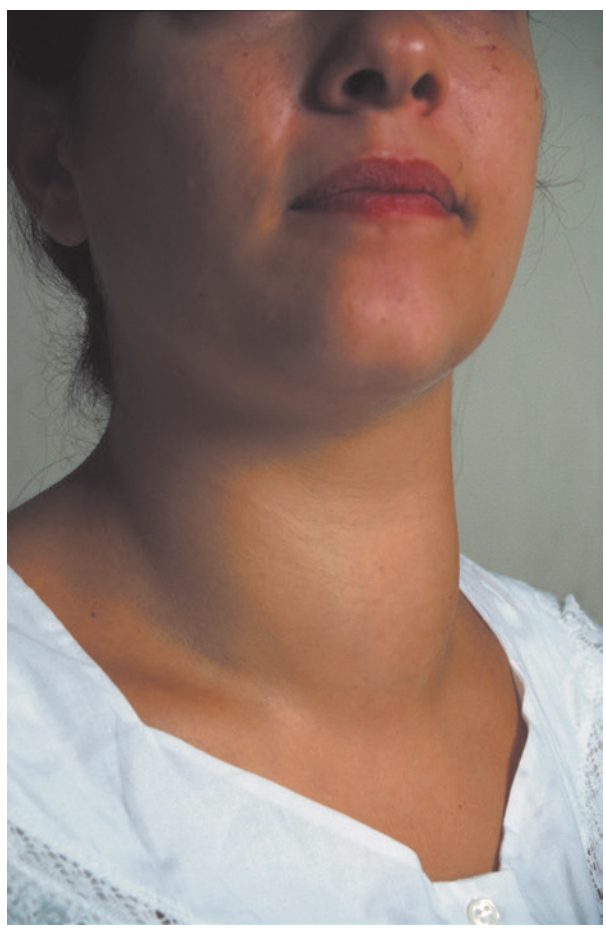


FIGURE 18.2 ■ Toxic multinodular goitre. The formation and growth of numerous nodules in the thyroid gland cause the characteristic massive enlargement of the neck

Source: Marka/Custom Medical Stock Photo.

secretion often results from a pituitary adenoma. This secondary form of hyperthyroidism is rare.

Subacute thyroiditis

Subacute thyroiditis (inflammation of the thyroid gland) is most often the result of a viral infection of the thyroid gland such as influenza or mumps. The symptoms of subacute thyroiditis are those of inflammation and the effects of increased TH. Subacute thyroiditis may become chronic, resulting in a hypothyroid state as repeated infections destroy gland tissue. See the discussion of Hashimoto's thyroiditis later in this chapter.

Thyroid crisis

Thyroid crisis (also called **thyroid storm**) is an extreme state of hyperthyroidism that is rare today because of improved diagnosis and treatment (Carroll & Matfin, 2010). Those affected usually have had untreated hyperthyroidism (most often Graves' disease) or have been receiving therapy but have experienced an acute stressor, such as an infection, trauma, myocardial infarction, diabetic ketoacidosis (DKA) or manipulation of the thyroid gland during surgery. Newer biological agents such as interleukin-2 and interferon alpha have been reported to induce thyroid storm (Carroll & Matfin, 2010). Thyroid crisis is a life-threatening condition.

The rapid increase in metabolic rate results in manifestations including hyperthermia, with body temperatures ranging from 39°C to 41°C; tachycardia; systolic hypertension; and gastrointestinal symptoms (abdominal pain, vomiting and increased bowel motions). Agitation, restlessness and tremors are common, progressing to confusion, psychosis, delirium and seizures. The mortality rate is high. Rapid treatment of thyroid crisis is essential to preserve life. Intensive care treatment includes cooling without aspirin (which increases free TH) or inducing shivering; replacing fluids, glucose and electrolytes; relieving respiratory distress via mechanical ventilation; stabilising cardiovascular function; and reducing TH synthesis and secretion.

INTERPROFESSIONAL CARE

Treatment of hyperthyroidism focuses on reducing the production of TH, thus establishing a **euthyroid** (normal thyroid) state, and preventing or treating complications. Depending on the person's age and physical status, oral antithyroid medications, radioactive iodine therapy or surgery may be used.

Diagnosis

Hyperthyroidism is diagnosed according to the manifestations of the specific disorders causing excessive TH and by diagnostic test results. Elevated levels of TH (both T_3 and T_4) and increased radioactive iodine (RAI) uptake are diagnostic criteria of hyperthyroidism. Laboratory findings in hyperthyroidism were shown earlier in Table 18.1.

The following diagnostic tests may be ordered:

- *TSH receptor antibodies*. This is useful to establish the diagnosis of Graves' disease, especially when a radionuclide thyroid scan is not able to be performed, as in pregnancy or lactation. Thyroid peroxidase and thyroglobulin autoantibodies may be useful in the diagnosis of subacute thyroiditis or autoimmune chronic lymphocytic thyroiditis.
- *TSH test (sensitive assay)*. Serum TSH levels are measured and compared with thyroxine (T_4) levels to differentiate pituitary from thyroid dysfunction. The best indicator of primary hyperthyroidism (such as in Graves' disease) is suppression of TSH below 0.27 mU/L. When the sensitive TSH is not suppressed, the hyperthyroidism is caused by a TSH-secreting pituitary tumour.
- *T_4 test, total*. Serum tetraiodothyronine (thyroxine) levels are measured to determine TH concentration and to test thyroid gland function. Levels are elevated in hyperthyroidism and acute thyroiditis.
- *T_3 test, free*. Serum triiodothyronine (T_3) is the active form of TH. T_3 is measured by radioimmunoassay (T_3RIA), which measures bound and free forms of this hormone. T_3 levels may be elevated in hyperthyroidism and thyroiditis. T_3 decreases in acute illness and starvation and is affected by medications such as amiodarone, propranolol and steroids (Molina, 2009).
- *T_3 uptake test*. T_3 uptake (T_3RU) is measured by an in vitro test in which the person's blood is mixed with radioactive T_3; the results are elevated in hyperthyroidism and in metastatic neoplasms.
- *RAI uptake test*. A radioactive iodine uptake test (thyroid scan) measures the absorption of ^{131}I or ^{123}I by the thyroid gland. A calculated dose of radioactive iodine is given orally or intravenously and the thyroid is then scanned (often after 24 hours). The distribution of radioactivity in the gland is recorded (increased uptake of radioactive iodine is seen in Graves' disease). In addition, the scan reveals the size and shape of the gland.
- *Thyroid suppression test*. RAI and T_4 levels are measured first. The person then takes TH for 7 to 10 days, after which the tests are repeated. Failure of hormone therapy to suppress RAI and T_4 indicates hyperthyroidism.
- *Fine needle biopsy*. Any suspicious enlargement of the thyroid gland should undergo biopsy with expert cytological examination.

Medications

Hyperthyroidism can be treated by administering antithyroid medications that reduce TH production. See 'Medication administration' box below.

Radioactive iodine therapy

Because the thyroid gland takes up iodine in any form, radioactive iodine (^{131}I) concentrates in the thyroid gland and damages or destroys thyroid cells so that they produce less TH. The person is placed on a low-iodine diet for up to six weeks prior to therapy to facilitate uptake (Ngyuen et al., 2015).

Results typically occur in 6 to 8 weeks. In most instances, the person is not hospitalised during treatment and does not require radiation precautions. Women of childbearing age should delay becoming pregnant for 6 months and men should allow 4 months for sperm turnover production. RAI has recently been shown to worsen the ophthalmopathy associated with Graves' disease (Hershman, 2013).

MEDICATION ADMINISTRATION Hyperthyroidism

IODINE SOURCES

Strong iodine solution (sodium iodide (^{131}I) solution BP)

The Wolff–Chaikoff effect has demonstrated that large doses of iodine for a short term inhibit TH synthesis and release (Carroll & Matfin, 2010).

Nursing responsibilities

- Assess for hypersensitivity to iodine before giving medication; for example, ask the person about allergies to shellfish.
- Dilute liquid iodine sources in water or orange juice to disguise bitter taste.
- Monitor for increased bleeding tendencies if the person is also taking anticoagulants; iodine increases their effect.

Health education for the person and family

- Administer at least 1 hour after antithyroid medications.
- The maximum effect of iodine in large doses usually occurs in 10 to 15 days.
- Long-term iodine therapy is not effective in controlling hyperthyroidism.

ANTITHYROID DRUGS

Carbimazole (Neo-Mercazole; Carbimazole Aristo)
(Propylthiouracil or PTU)

Antithyroid drugs, also known as thionamide medications, inhibit the incorporation of iodine into thyroglobulin, thus lowering TH production. They do not affect already formed hormones; therefore, several weeks may elapse before the person experiences therapeutic effects. Carbimazole is converted to methimazole in the body. This oral medication is given in divided doses according to the severity of the hyperthyroidism, from 15 mg/day up to 60 mg/day in severe cases. Once symptoms are controlled the dosage can be tapered to a long-term maintenance dose. Due to the high risk of foetal gastrointestinal defects, carbimazole is contraindicated during the first trimester of pregnancy (Campbell & Doogue, 2012).

Propylthiouracil is taken in divided doses three or four times daily until the person is euthyroid. Propylthiouracil is the preferred medication in the first trimester of pregnancy (Campbell & Doogue, 2012).

Nursing responsibilities

- Monitor for side effects: life-threatening agranulocytosis evidenced by reduced neutrophil count and fever, hypothyroidism, pruritus rash, periorbital oedema, anorexia or vomiting, loss of taste, menstrual irregularities.
- Severe hepatocellular injury occurs with propylthiouracil in 0.1% of patients. Monitor for evidence of jaundice.
- Administer drugs at the same time each day with meals to maintain stable blood levels.
- Monitor for symptoms of hypothyroidism: fatigue, weight gain, bradycardia.

Health education for the person and family

- Watch for unusual bleeding, redness, swelling, nausea, loss of taste or epigastric pain.
- If you are also taking warfarin for atrial fibrillation, report any signs of bleeding.
- It may take up to 12 weeks before you experience the full effects of the drugs. Take the medication regularly and exactly as prescribed. Do not discontinue abruptly.

Because the amount of gland destroyed is not readily controllable, the person may become hypothyroid and require lifelong TH replacement. Adverse reactions include radiation thyroiditis and cardiac instability due to liberation of stored thyroid hormone in the gland (Campbell & Doogue, 2012), sialadenitis (inflammation of the salivary gland) and xerostomia (dry mouth) (Charalambous, Frangos & Talias, 2014).

Surgery

Surgery is recommended when other treatments are not effective in young individuals, people with large or suspicious nodules, obstructive symptoms when the pressure on the oesophagus or trachea causes breathing or swallowing problems, for rapid resolution of symptoms. A *subtotal thyroidectomy* is usually performed. This procedure leaves enough of the gland in place to produce an adequate amount of TH. A total **thyroidectomy** is performed to treat thyroid cancer; the person then requires lifelong hormone replacement (Quérat et al., 2015).

Before surgery, the person should be in as nearly a euthyroid state as possible. Antithyroid drugs may be given to reduce hormone levels and iodine preparations to decrease the vascularity and size of the gland, which also reduces the risk of haemorrhage during and after surgery. Postoperative complications include laryngeal nerve palsy or hypocalcaemia due to removal of or damage to the parathyroid glands (Quérat et al., 2015).

Nursing care of the person having a subtotal thyroidectomy is discussed in the box below.

Nursing care

Health promotion

Although hyperthyroidism is not preventable, it is vital to teach the importance of regular healthcare provider visits and maintaining regular medication intake.

Assessment

The following data are collected through the health history and physical examination (see Chapter 17). Further focused assessments are described with nursing interventions.

NURSING CARE OF THE PERSON Having a subtotal thyroidectomy

PREOPERATIVE CARE

- Administer ordered antithyroid medications and iodine preparations and monitor their effects. *Antithyroid drugs are given before surgery to promote a euthyroid state. Iodine preparations are given before surgery to decrease vascularity of the gland, thereby decreasing the risk of haemorrhage.*
- Teach the person preoperatively to support the neck by placing both hands behind the neck when sitting up in bed, while moving about and while coughing. *Placing the hands behind the neck postoperatively provides support for the suture line.*
- Answer questions and allow time for the person to verbalise concerns. Because the incision is made at the base of the throat, people (especially women) are often concerned about their appearance after surgery. *Explain that the scar will eventually be only a thin line and that jewellery or scarves may be used to cover the scar.*
- Teach the person to expect hoarseness due to generalised swelling at the suture line. *This is expected to diminish with healing and is not caused by laryngeal nerve damage.*

POSTOPERATIVE CARE

- Perform focused assessments to monitor for complications: maintain patent airway and observe for respiratory distress. Assess respiratory rate, rhythm, depth and effort. Maintain humidification as ordered. Assist the person with deep breathing and coughing. Have suction equipment, oxygen and a tracheostomy set available for immediate use. *Respiratory distress may result from haemorrhage and oedema, which may compress the trachea; from tetany and laryngeal spasms resulting from removal or damage to the parathyroid glands; and from damage to the laryngeal nerve, causing spasms of the vocal cords. Stridor is heard in acute obstructions. This is a high-pitched, squeaky sound and is a sign of airway obstruction.*
- *Haemorrhage*. Assess dressing (if present) and the area under the person's neck and shoulders for drainage. Check volume drains. Monitor blood pressure and pulse as well as other signs and symptoms of hypovolaemic shock. Assess tightness of dressing (if present). *The vascularity of the gland increases the risk of haemorrhage. The location of the incision and the position of the person may cause the drainage to run back and under the person's neck. The danger of haemorrhage is greatest in the first 12 to 24 hours after surgery.*
- Provide comfort measures: administer analgesia as ordered and monitor their effectiveness; place the person in a semi-Fowler's position after recovery from anaesthesia; support head and neck with pillows. *Analgesic medications reduce acute pain and physical stress during the postoperative period. Positioning the person in a semi-Fowler's position and supporting the head and neck decreases strain on the suture line.*
  a. *Laryngeal nerve damage*. Assess for the ability to speak aloud, noting quality and tone of voice. *The location of the laryngeal nerve increases the risk of damage during thyroid surgery. Although hoarseness may be due to oedema or the endotracheal tube used during surgery and will subside, permanent hoarseness or loss of vocal volume is a potential danger.*
  b. *Tetany*. Assess for signs of latent tetany due to decreased circulating calcium levels, including tingling of toes, fingers and lips; muscular twitches; positive Trousseau's sign (carpal spasm elicited by compression of the upper arm (King, Hawley & Weller, 2008). Keep calcium gluconate or calcium chloride available for immediate intravenous use. *The parathyroid glands are located in and near the thyroid gland; surgery of the thyroid gland may injure or remove parathyroid glands, resulting in hypocalcaemia and tetany. Tetany may occur up to 7 days after a thyroidectomy.*

- *Health history:* other medical conditions, family history of thyroid disease, when symptoms began, severity of symptoms, intake of iodine or thyroid medications, menstrual history, changes in weight, bowel elimination.
- *Physical assessment:* muscle strength, tremors, vital signs, cardiovascular and peripheral vascular systems, integument, size of thyroid, presence of bruit over thyroid, eyes and vision.

Nursing diagnoses and interventions

In planning and implementing nursing care for the person with hyperthyroidism, the nurse considers the person's responses to the systemic effects of the disorder. Although each person may have different needs, nursing diagnoses discussed in this section focus on the most common problems: cardiovascular problems, visual deficits, altered nutrition and body image disturbance. See the accompanying 'Nursing care plan' below.

CONSIDERATION FOR PRACTICE

Teach the person to cover or tape the eyelids shut at night if they do not close and to sleep with the head of the bed elevated.

Risk of decreased cardiac output

Excess TH directly affects the heart, resulting in increased rate and stroke volume. Increases in the metabolic demands and oxygen requirements of peripheral tissues increase the demands on the heart, and systolic hypertension, angina, arrhythmias or cardiac failure may occur. The person often has shortness of breath and is easily fatigued. The risk of complications is greater in people with pre-existing cardiovascular disorders.

- Monitor blood pressure, pulse rate and rhythm, respiratory rate, oxygenation and breath sounds. Assess for peripheral oedema, jugular vein distension and increased activity intolerance. *Increased TH increases heart rate, stroke volume and tissue demand for oxygen, causing stress on*

the heart. This may result in hypertension, arrhythmias, tachycardia and congestive heart failure.

- Suggest keeping the environment as cool and free of distraction as possible. Decrease stress by explaining interventions, providing reassurance and teaching relaxation procedures. *A physically comfortable and psychologically calm environment can reduce stimuli and stressors. Stress increases circulating catecholamines, which further increase cardiac workload.*

Disturbed visual perception related to exophthalmos

- Monitor visual acuity, photophobia, integrity of the cornea and lid closure. *The cornea is at risk of dryness, injury,*

NURSING CARE PLAN A person with Graves' disease

Mrs Juanita Martin is a 33-year-old mother of four small children. She is a second-year student completing the requirements for an associate degree in childcare. For the past 3 months, Juanita has been constantly hungry and has eaten more than usual, but she has still lost 6.8 kg. She has multiple bowel movements each day and often feels nauseated. Her hands shake, she can feel her heart beating rapidly and she finds herself laughing or crying for no apparent reason.

Mrs Martin makes an appointment with her family physician. The nurse at the office completes a health history and physical assessment. When asked how she has been feeling, Mrs Martin replies, 'Well, I don't know what's wrong with me—but I keep losing weight and I cry at the drop of a hat. I am also just so hot all the time and I've never had that problem before. I hope I find out what's wrong and it's nothing serious.'

ASSESSMENT

The health history indicates that although her appetite has increased, Mrs Martin has lost 6.8 kg. She states that she has had increased bowel movements, nausea, palpitations, heat intolerance and mood changes. Physical assessment findings include the following: T 38.3°C, P 110, R 24 and BP 162/86. Her skin is moist and warm, her hair thin and fine. She has visible tremors in her hands. Her eyeballs protrude and she is unable to close her eyelids completely. Her thyroid is enlarged and palpable. Diagnostic tests reveal the following abnormal results: Free T_3, 18 pmol/L (normal range: 3–7.8 pmol/L), Total T_4, 30 pmol/L (normal range: 12–22 pmol/L). A thyroid scan demonstrates an enlarged thyroid with increased iodine uptake. The medical diagnosis of Graves' disease is made and Mrs Martin is commenced on the antithyroid medication propylthiouracil, 150 mg orally every 8 hours.

DIAGNOSES

- *Risk of imbalanced nutrition: less than body requirements* related to hyperthyroid state and increased metabolism as evidenced by weight loss of 6.8 kg with present weight 10% less than normal for her height.
- *Increased bowel movements* related to increased food intake and peristalsis as evidenced by more than four loose stools per day.
- *Risk of disturbed visual perception* and/or *Risk of eye infection* related to an inability to close the eyelids completely.
- *Anxiety* related to a lack of knowledge about disease process.

PLANNING

- Request that she keep a record of weekly weight.
- Discuss adopting a high-kilojoule diet. Identify food likes and dislikes, before instituting a plan to increase food intake.
- Request that she keep a stool chart, noting the time, type and precipitating factors for stools. Teach comfort measures for irritated anal area (clean washcloth and soap, no irritating ointment).
- Teach how to apply eye drops (artificial tears).
- Explain the need to elevate the head of the bed to 45 degrees at night and tape eye shields over eyes before sleep.
- Teach about Graves' disease, the medication's effects and side effects, and the need for continued medical care.

Expected outcomes

- Gain at least 0.45 kg every 2 weeks.
- Regain normal bowel elimination patterns.
- Maintain normal vision (with no evidence of corneal damage and/or infection) and verbalise measures to protect her eyes.
- Verbalise medical treatment and self-care needs.
- Verbalise a decrease in anxiety.

IMPLEMENTATION

- Mrs Martin will adopt a high-kilojoule diet and increase the food intake.
- Maintain a weekly record of her weight.
- Maintain a stool chart and adopt measures for anal comfort and hygiene.
- Regular application of eye drops and/or lubrication.
- Maintain head elevation and use of eye shields at night.
- Give Mrs Martin contacts for support or if she has any queries or concerns.

EVALUATION

By her next visit, Mrs Martin has gained 0.45 kg and has discussed her dietary needs with the nurse and her husband. She is having fewer bowel motions per day. She has safely applied the eyedrops and states that she uses the eye shields and elevates the head of her bed at night. The office nurse reviews the written and verbal information about Graves' disease and the medication prescribed. Mrs Martin verbalises her understanding, stating, 'I'll always take my medicine—I never want to feel like that again!' She also says that she feels much less anxious now that she understands what has happened.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the pathophysiological basis for Mrs Martin's abnormal vital signs?
2. What is the rationale for having the person with exophthalmos elevate the head of the bed at night?

REFLECTION ON THE NURSING PROCESS

1. Can you think of at least three other strategies that you would add to the plan of education for Mrs Martin and her family? Write the corresponding Expected outcomes and Implementation for your strategies.
2. Design an educational strategy to help teach Mrs Martin about her nutritional requirements.

conjunctivitis and corneal infections. Injury and infection of the cornea can result in further loss of visual acuity.

- Teach measures for protecting the eye from injury and maintaining visual acuity:
  - Use tinted glasses or shields as protection.
  - Use artificial tears to moisten the eyes.
  - Use cool, moist compresses to relieve irritation.
  - Cover and tape eyelids with an eye shield at night if they do not completely close.
  - Elevate the head of the bed to 45 degrees to promote periorbital fluid decrease.
  - Promptly report any pain or changes in vision.
  - Encourage the person to quit smoking which exacerbates exophthalmos.

Imbalanced nutrition: less than body requirements related to gastrointestinal hypermotility

The hypermetabolic state that occurs in hyperthyroidism causes gastrointestinal hypermotility. Although the person may have an increased appetite and eat more than usual, weight loss continues.

- Ask the person to weigh themselves weekly and keep a record of results. *The inability to meet metabolic demands results in loss of body weight. Regular monitoring detects continued weight loss.*
- In collaboration with a dietitian, teach the person the need for a diet high in carbohydrates and protein and including between-meal snacks. Six small meals a day may be more desirable than three large meals. Energy intake may need to be increased to 16 700 kilojoules/day if weight loss exceeds 10% to 17% for height and frame. *Increased nutrients as part of a well-balanced diet are necessary to meet metabolic demands. People are often better able to increase food intake by eating frequent, small meals. A 0.45 kg weight gain requires approximately 14 650 extra kilojoules.*
- Monitor nutritional status through results of laboratory data. Serum albumin, transferrin and total lymphocyte counts are commonly lower than normal in nutritional deficits. *A negative nitrogen balance signifies a catabolic state in which protein is lost and metabolic demands are not being met.*

Potential for disturbed body image related to physical changes common in hyperthyroidism

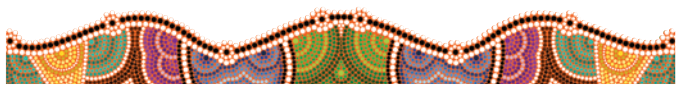
Physical changes common in hyperthyroidism include exophthalmos, goitre, tremors, hair loss, increased perspiration, loss of strength, fatigue, weight loss and changes in reproductive and sexual function (amenorrhoea in women, impotence in men and increased libido in both men and women). In addition, the person often has mood changes and insomnia and is constantly nervous and anxious. There may even be periods of psychosis. These changes are frightening not only for the person but also for family members.

- Establish a trusting relationship; encourage the person to verbalise feelings about self and to ask questions about the illness and treatment. Provide reliable information and clarify misconceptions. *Establishing trust facilitates open sharing of feelings and perceptions.*

Community-based care

People with hyperthyroidism primarily provide self-care at home. Teaching is individualised to meet the person's needs. Address the following topics:

- The person taking oral medications must understand the need for lifelong treatment.
- The person who has a thyroidectomy requires information about postoperative wound care.
- The person having radioactive iodine therapy needs to know the symptoms of hypothyroidism.
- Depending on the age of the person and the support systems available, referral to community healthcare agencies may be necessary.
- In addition, suggest the following online resources:
  - Thyroid Australia: www.thyroid.org.au
  - Australian Thyroid Foundation Limited: www.thyroid foundation.com.au
  - Thyroid Federation International: www.thyroid-fed.org.



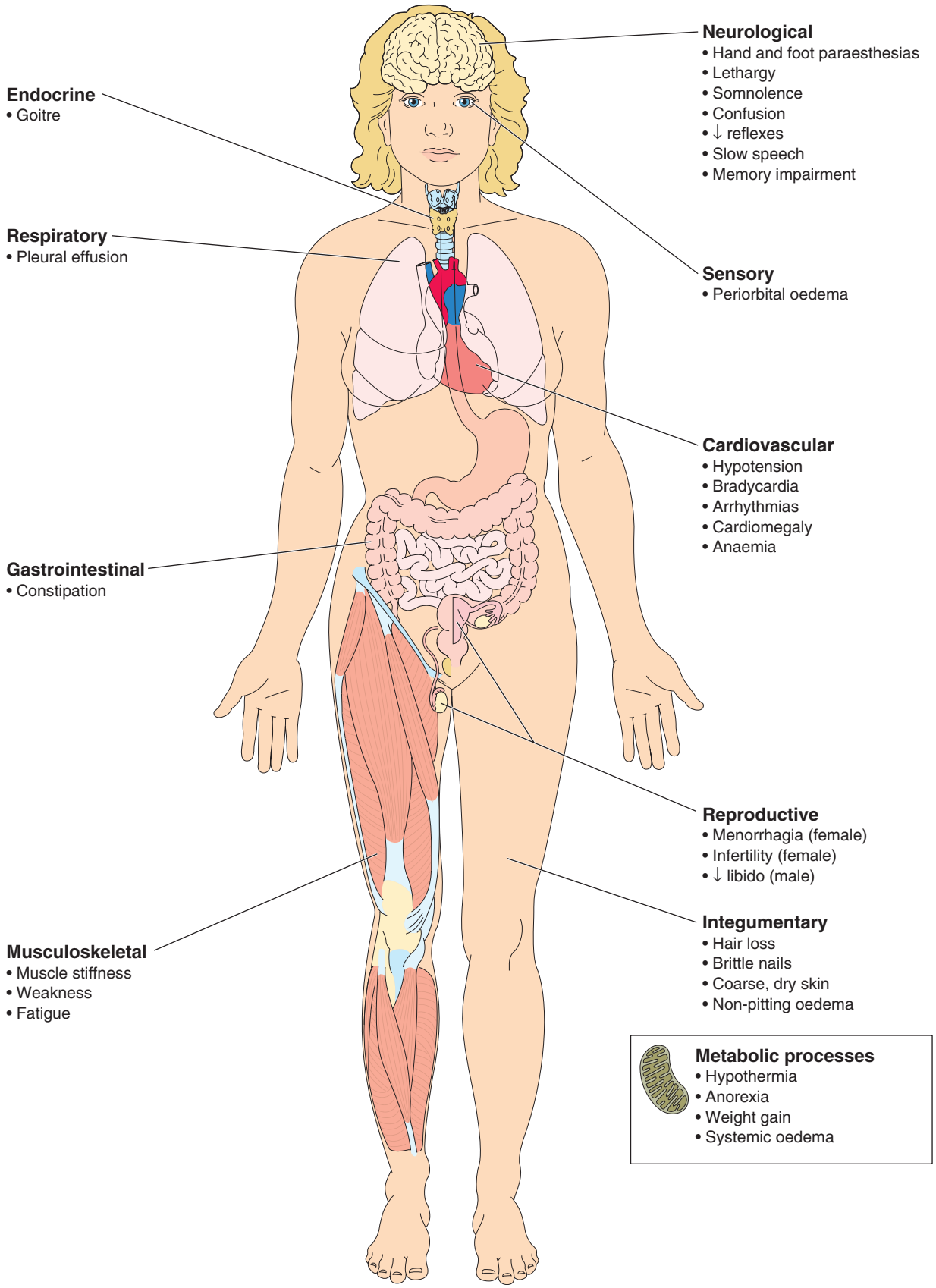
THE PERSON WITH HYPOTHYROIDISM

Hypothyroidism is a disorder that results when the thyroid gland produces an insufficient amount of TH. Because a decrease in TH levels decreases metabolic rate and heat production, hypothyroidism affects all body systems (see 'Multisystem effects of hypothyroidism' below). Hypothyroidism is most common in women between ages 30 and 60; the incidence rises with age. It is also associated with an increase in morbidity and mortality due to cardiovascular involvement (McQuade et al., 2011). Careful evaluation of symptoms is important in the older adult because manifestations of hypothyroidism are often thought to be the result of ageing instead of a pathological process.

Hypothyroidism occurs in about 5% of the adult population; most present with mild thyroid failure, characterised by raised serum TSH but normal free thyroxine or T_4. Worldwide, the most common cause of hypothyroidism is iodine deficiency. However, in Australia the most common cause of hypothyroidism is autoimmune chronic lymphocytic thyroiditis characterised by raised levels of thyroid peroxidase antibody, also known as Hashimoto's thyroiditis (Akamizu, Amino & DeGroot, 2013; Thyroid Australia, 2011b).

The hypothyroid state in adults is sometimes called **myxoedema**. The term reflects the characteristic accumulation of non-pitting oedema in the connective tissues throughout the body. The face of a person with myxoedema appears puffy, the tongue is enlarged, and the voice is hoarse and husky (So, MacIsaac & Grossman, 2012). Proteinaceous fluid can also accumulate within body cavities. The most common sites are the pericardial, pleural and peritoneal cavities. The reported incidence of pericardial effusion in newly diagnosed mild hypothyroid patients is 3%, increasing to 80% in myxoedema (Motabar et al., 2011).

MULTISYSTEM EFFECTS OF HYPOTHYROIDISM



Pathophysiology and manifestations

Hypothyroidism may be either primary or secondary. Primary hypothyroidism, which is more common, may be caused by congenital defects in the gland, loss of thyroid tissue following treatment for hyperthyroidism with surgery or radiation, antithyroid medications, thyroiditis or endemic iodine deficiency. Secondary hypothyroidism may result from pituitary TSH deficiency or peripheral resistance to thyroid hormones. Hypothyroidism has a slow onset, with manifestations occurring over months or even years. With treatment, the mental and physical symptoms rapidly reverse.

When TH production decreases, the thyroid gland enlarges in a compensatory attempt to produce more hormones. The goitre that results is usually a simple diffuse or non-toxic form. People living in certain areas of the world where the soil is deficient in iodine are more prone to become hypothyroid and develop simple goitre. (Iodine deficiency is discussed below.) The geriatric person has a decrease in T_4 production of approximately 30%, but serum levels are usually maintained because of the age-related decrease in T_4 degradation (Weissel, 2006).

The person with hypothyroidism characteristically has manifestations of goitre, fluid retention and oedema, decreased appetite, weight gain, constipation, dry skin, dyspnoea, pallor, hoarseness and muscle stiffness. Many also have a decreased sense of taste and smell, menstrual disorders, anaemia and cardiac enlargement. The pulse is typically bradycardic (Warner & Mittag, 2012). Deficient amounts of TH cause abnormalities in lipid metabolism, with elevated serum cholesterol and triglyceride levels. As a result, the person is at increased risk of atherosclerosis and cardiac disorders. Decreased renal blood flow and glomerular filtration rate reduce the kidneys' ability to excrete water, which may cause hyponatraemia. Sleep apnoea is more common in people with hypothyroidism. A severe state of hypothyroidism is called *myxoedema coma.*

Iodine deficiency

Iodine deficiency may result from certain goitrogenic drugs (which block TH synthesis); lithium carbonate, used to treat bipolar disorders; and antithyroid drugs. Lack of iodine can cause hypothyroidism and goitre, but this is rare in developed countries. It can also have a devastating effect on the development of the foetus and newborn child (Medenica et al., 2015). Insufficient iodine and thus TH during gestation and early childhood is known as the iodine deficiency disorder of congenital hypothyroidism, or cretinism.

In the last few years there has been an increase in iodine deficiency in Australia. This deficiency has been due to changes in milk sterilisation processes, which no longer use iodides, and less consumption of iodised salt due to concerns about salt intake in general (Brand-Miller, 2008). Around 150 Gk mug (millionths of a gram, or micrograms) of dietary iodine is needed each day to produce adequate levels of thyroid hormones. Seafoods such as oysters are particularly rich in iodine (Thyroid Australia, 2011a). Pregnant and breastfeeding women need to include an oral iodine supplement so that they are taking 250 μg in total per day of iodine (World Health Organization (WHO), 2007).

Hashimoto's thyroiditis

Hashimoto's thyroiditis is the most common cause of goitre and primary hypothyroidism in Australia. In this autoimmune disorder, antibodies develop that destroy thyroid tissue. Functional thyroid tissue is replaced with fibrous tissue and TH levels decrease. In addition, decreasing levels of TH in the early stages of the disorder prompt the gland to enlarge to compensate, causing goitre. However, as the disease progresses, the thyroid gland becomes smaller. This disorder is more common in women and has a familial link.

Myxoedema coma

Myxoedema coma is a life-threatening complication of long-standing, untreated primary hypothyroidism usually triggered by an acute infection, the administration of thyroid-hormone-reducing medications or trauma. Five per cent occur as a result of hypothalamic or pituitary causes. It is characterised by severe metabolic disorders (hyponatraemia, hypoglycaemia and lactic acidosis), hypothermia (usually below 32.2°C), cardiovascular complications (hypotension, bradycardia, cardiac tamponade), and altered mental state such as confusion and coma (due to cerebral oedema, hypoxia and hypercarbia). Seizures may precede coma in 25% of people (Gupta, 2013).

The treatment of myxoedema coma addresses the precipitating factors and manifestations and involves maintaining a patent airway; maintaining fluid, electrolyte and acid–base balance; maintaining cardiovascular status; increasing body temperature; and increasing TH levels with intravenous thyroxine and corticosteroids. Despite aggressive treatment the mortality rate for myxoedema coma remains up to 60% (Gupta, 2013).

INTERPROFESSIONAL CARE

The treatment of the person with hypothyroidism focuses on diagnosis, prevention or treatment of complications, and replacement of the deficient TH. With early and continued treatment, both appearance and mental function return to normal.

Diagnosis

Hypothyroidism is diagnosed by the clinical manifestations and by a decrease in TH, especially T_4 (see Table 18.2). TSH concentration often is increased, because the negative hormonal feedback from TH is lost. The same laboratory and diagnostic tests used to diagnose hyperthyroidism are also used to diagnose hypothyroidism, with opposite results in most cases.

Medications

Hypothyroidism is treated with medications that replace TH. Thyroxine, T_4 (Levothyroxine) is the preferred therapy, 1.6 μg/kg lean body weight daily (The Thyroid Society for Education and Research cited by Thyroid Australia, 2012). Medications commonly used to treat hypothyroidism and their nursing implications are shown in the 'Medication administration' box below. In the geriatric person, an age-related decrease in serum albumin and renal excretion can increase the amount of available drug and cause an exaggerated pharmacological effect. Therefore, the older person may require less thyroid medication than a younger person.

TABLE 18.2 Laboratory findings in hypothyroidism

TEST	NORMAL VALUES	FINDINGS
Serum TA	≤ 1:10 titre	Normal
Serum TSH	0.27—4.2 mU/L	Increased in primary hypothyroidism Decreased, secondary hypothyroidism
Serum T_4	Total 12–22 pmol/L	Decreased
Serum T_3	Free 3–7.8 pmol/L	Decreased
T_3 uptake (T_3RU)	25–35 %	Decreased
Thyroid suppression		No change in RAI uptake or T_4 levels

Source: South-Western Sydney Area Health Service (SSWAHS) Liverpool Hospital Pathology Department (2012). Reproduced with permission.
mU: microunits; pmol: picomoles.

Surgery

If the hypothyroid person has goitre large enough to cause respiratory difficulties or dysphagia, a subtotal thyroidectomy may be performed (see the 'Nursing care of the person' box on p. 535).

Nursing care

Health promotion

One of the most critical factors in preventing hypothyroidism is education of the public about the necessity of an adequate dietary intake of iodine. Since 2006, the mandatory fortification of bread with iodised salt has redressed the prevalence of hypothyroidism in Australia.

Assessment

Collect data through the health history and physical examination (see Chapter 17). Further focused assessments are described with nursing interventions below. When assessing the older person, be aware of normal changes with ageing, outlined in the box below.

- *Health history:* pituitary diseases, when symptoms began, severity of symptoms, treatment of hyperthyroidism with medications or radioactive iodine, thyroid surgery, treatment of head or neck cancer with radiation, diet, use of iodised salt, bowel elimination, respiratory difficulties.
- *Physical assessment:* muscle strength, deep tendon reflexes, vital signs, cardiovascular and peripheral vascular systems, integument, palpation of thyroid gland, weight.

MEDICATION ADMINISTRATION Hypothyroidism

THYROID PREPARATIONS

Thyroxine sodium (T_4) (Oroxine, Eutroxsig)
Liothyronine sodium (T_3) (Tertroxin)

Replacement oral thyroxine is the preferred therapy for hypothyroidism. A dose of 1.6 µg per kg body weight daily is the average required in adults. The dose is adjusted in cases of hypopituitarism and adrenal insufficiency due to risk of adrenal crisis if glucocorticoid replacement is not given simultaneously. The presence of cardiovascular disorders also requires a dose adjustment due to the risk of worsening ischaemic symptoms and arrythmias. In diabetes, the dose of insulin or hypoglycaemic drugs may need to be adjusted. Combined thyroxine (T_4) and liothyronine (T_3) therapy is being promoted for a small subset of people whose symptoms persist despite apparently adequate thyroxine therapy (McDermott, 2012).

Specific responsibilities include:

- Give first thing in the morning on an empty stomach with water, at least 30 to 60 minutes before breakfast and 3 to 4 hours before taking other medications.
- Be aware of interactions with vitamins, minerals and other herbal extracts.
- Thyroid preparations potentiate the effect of anticoagulant drugs. If taking an anticoagulant, monitor for bruising, bleeding gums and blood in the urine.
- Thyroid medications potentiate the effect of digitalis; monitor for signs of digitalis toxicity.
- Monitor for symptoms of coronary insufficiency: chest pain, dyspnoea and tachycardia.
- During dose adjustment, take pulse before administering drug. Report pulse > 100.

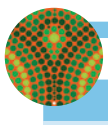
HEALTH EDUCATION OF THE PERSON AND FAMILY

- Opened and unopened packets of Oroxine and Eutroxsig tablets should remain both refrigerated and in their original foil packaging until administration.
- Do not substitute brands of drugs or use generic equivalents.
- These medications are lifelong therapy.
- Take thyroid preparation each morning to decrease the possibility of insomnia.
- Report symptoms of excess thyroid hormone: excess weight loss, palpitations, leg cramps, nervousness or insomnia.
- Thyroid preparations increase the risk of iodine toxicity. Do not use iodised salt or over-the-counter drugs containing iodine.
- If you are also taking an anticoagulant, report any signs of bleeding.
- Report any changes in menstrual periods.
- Closely monitor blood pressure and pulse (older people).
- Avoid excessive intake of raw goitrogenic foods that are known to inhibit TH utilisation, such as turnips, cabbage, carrots, spinach and peaches. The heat in cooking partially destroys the goitrogenic enzymes.

NURSING CARE OF THE OLDER ADULT Variations in assessment findings—hypothyroidism

NORMAL CHANGES WITH AGEING

- The thyroid gland undergoes some degree of atrophy, fibrosis and nodule formation.
- Hair growth decreases.
- Nails are often thick, brittle and yellow.
- Facial skin sags and bones become more prominent.
- Deep tendon reflexes decrease.
- Response to questions may be slower.



TRANSLATION TO PRACTICE Evidence-based practice: thyroid nodules, benign or malignant? Common causes and a simple approach to diagnosis and management in the clinical setting

Thyroid nodules are common with up to 50% of adults having nodules visible on ultrasound. About 5% of thyroid nodules are malignant (Mackenzie & Mortimer, (2004).

Thyroid nodules are more common in the older person, women, people with iodine deficiency and those who have had prior exposure to radiation. Significant symptoms, if present, may include dysphagia due to impingement of the goitre on the oesophagus, shortness of breath due to tracheal impingement and, less commonly, hoarseness of the voice due to possible laryngeal nerve compression (Brennan & French, 2007). Other suspicious features for malignant nodules include people younger than 20 years old or older than 70 years, male, nodule size more than 4 cm, rapid growth or family history. Pain may or may not be present.

IMPLICATIONS FOR NURSING

Clinical assessment of the person and recognising abnormal thyroid signs and symptoms is within the scope of nursing. Clinical assessment of the thyroid gland includes *Inspection* for scars, goitre, movement when swallowing, prominent veins; *Palpation* by (i) positioning the person and examining from behind with the neck slightly flexed to relax sternomastoid muscles—feeling each lobe and the isthmus of the gland for characteristics of goitre or nodules and examining cervical lymph nodes, and (ii) in front of the person examining characteristics of the nodules and position of the trachea which may be displaced; *Percussion* over the manubrium, dullness may indicate goitre, although CT scan is a more common procedure; *Auscultation* as bruit may occur in Graves' disease. *The Pemberton sign* is used to evaluate venous obstruction from goitres. Ask the person to raise the arms as high as possible and wait a few moments; signs of venous congestion such as redness of the face and breathlessness occur from retrosternal extension of the thyroid gland into the thorax (De Filippis et al., 2014). Also look for signs of hyper- or hypothyroidism.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. What is the prevalence of malignant thyroid nodules and who has a higher risk of having them?
2. Most people with thyroid nodules do not have any significant symptoms. But, if present, which symptoms are important to notice and what do they indicate?
3. Which clinical assessments need to be attended when examining the thyroid gland of a person with suspected nodules? Which instructions would be given to a person when assessing for the Pemberton sign?
4. Outline what have you learned from the article that you will apply to your nursing practice.

Nursing diagnoses and interventions

In planning and implementing care for people with hypothyroidism, the nurse needs to take into account that the disorder affects all organ systems. Although many nursing diagnoses might be valid, this section focuses on person problems with cardiovascular function, elimination and skin integrity. See the accompanying 'Nursing care plan' below.

Decreased cardiac output related to decreased stroke volume, bradycardia and possible pericardial effusion

A TH deficiency causes a reduction in heart rate and stroke volume, resulting in decreased cardiac output. There may also be pericardial effusion and coronary artery disease may be present, further compromising cardiac function.

- Monitor blood pressure, heart rate and rhythm, respiratory rate and lung sounds. *Monopolysaccharide deposits in the pericardial sac causing pericardial effusion decreases the intensity of heart sounds and can cause a variety of ECG changes. Pleural effusions are associated with dyspnoea* (McCance & Huether, 2009).
- Administer supplemental oxygen as ordered. *Reduced cardiac output results in less oxygen to the tissues.*
- Avoid cool temperatures, increase room temperature if necessary, use additional bed covers and avoid drafts. *Chilling increases metabolic rate and puts increased stress on the heart.*
- Explain the need to alternate activity with rest periods. Ask people to report any breathing difficulties, chest pain, heart palpitations or dizziness. *Activity increases demands on the heart and should be balanced with rest. Symptoms of cardiac stress include dyspnoea, chest pain, palpitations and dizziness.*

Constipation

The hypothyroid person is likely to have a reduced appetite and decreased food intake, a diminished activity level because of muscle aches and weakness, reduced water absorption and reduced peristalsis to the point that faecal impaction may occur.

NURSING CARE PLAN A person with hypothyroidism

Jane Lee is a 60-year-old retired nurse living with her husband and daughter on a farm that has been in her Chinese family for four generations. Mrs Lee has gained 4.5 kg in the past few months, even though she is rarely hungry and eats much less than normal. She is always tired and weak—so tired that she has not even been able to help with the chores on the farm or do housework. She is concerned about her appearance and the way she sounds when she talks. Her face is puffy and her tongue always feels thick. Mr Lee convinces his wife to make an appointment at a health centre in a nearby town.

ASSESSMENT

Brian Henning, Nurse Practitioner (NP), completes the health assessment for Mrs Lee at the health centre. He finds that she now weighs 68 kg, an increase of 4.5 kg over her weight at her last visit 6 months earlier. Mrs Lee states that she always feels cold, tired and weak. She also states that she is constipated, has difficulty remembering things and looks different. Physical assessment findings include a palpable and bilaterally enlarged thyroid; dry, yellowish skin; non-pitting oedema of the face and lower legs; and slow, slurred speech. Diagnostic tests revealed the following abnormal findings: Free T_3, 1 pmol/L (normal range: 3–7.8 pmol/L); Total T_4, 8 pmol/L (normal range: 12–22 pmol/L); TSH increased. The medical diagnosis of hypothyroidism is made and Mrs Lee is started on levothyroxine 0.05 mg daily.

DIAGNOSES

- *Constipation* related to decreased peristalsis, as evidenced by hard, formed stools every 4 days.
- *Impaired verbal communication* related to changes in speech patterns and enlarged tongue.
- *Low self-esteem* related to changes in physical appearance and activity intolerance.

PLANNING

- Teach to increase fluids, bulk and fibre in the diet to help regain a normal bowel elimination pattern of a soft, formed stool every other day.
- Take medication as prescribed and do not expect immediate reversal of symptoms affecting speech. It may take some time but need to continue with the treatment. Store medication as required by manufacturer.
- Plan activities around rest periods. Encourage husband and daughter to help with house cleaning and cooking.

Expected outcomes

- Regain normal bowel elimination patterns, having a soft, formed stool at least every other day.
- Experience improvement in verbal communication.
- Regain positive self-esteem as medication reduces physical changes and fatigue.

IMPLEMENTATION

- Mrs Lee will start consuming a diet with bulk and fibre as well as increase her fluid intake immediately.
- She will start and continue to take the prescribed medications and understand that it may take some time for some for the symptoms to improve.
- Mrs Lee will be resting and taking naps when required.
- She will seek appropriate advice from a healthcare practitioner if there are any issues or concerns.

EVALUATION

On return to the health centre 2 months later, Mrs Lee reports that she is no longer constipated and that she has increased her fluid intake and eats oatmeal every day. She no longer feels cold, is regaining her normal energy, and even feels well enough to plant her garden. Her speech is clear and easy to understand. As she leaves the examining room, Mrs Lee says: 'It's hard to believe that I have changed so much—now I look and feel like the "old" me!'.

CRITICAL THINKING IN THE NURSING PROCESS

1. Which physical changes that normally occur with ageing are similar to the manifestations of hypothyroidism?
2. The person taking oral thyroid medications may become hyperthyroid. List the manifestations you would include in a teaching plan to signal this condition.

REFLECTION ON THE NURSING PROCESS

1. Which points have you learned from the case study that you will implement in your future nursing practice?
2. How will you plan an appropriate diet for Mrs Lee, taking into consideration her Chinese background as well as her likes and dislikes? Reflect on other people with different ethnic backgrounds in Australia.

- Encourage a free fluid intake. If kilojoule intake is restricted, ensure that liquids have no or low kilojoules. Sufficient fluid intake assists stool consistency.
- Discuss ways to maintain a high-fibre diet. Diets high in fibre and fluid produce soft stools. *Fibre that is not digested absorbs water, which adds bulk to the stool and assists in the movement of faecal material through the intestines.*
- Encourage activity as tolerated. *Activity influences bowel elimination by improving muscle tone and stimulating peristalsis.*

CONSIDERATION FOR PRACTICE

High-fibre foods include lentils, chickpeas and sweet potatoes, apples with skin, grain breads, bran cereals, prunes, broccoli, porridge, popcorn and peas (Brand-Miller, Foster-Powell & McMillan-Price, 2006; Cancer Council NSW, 2015).

Risk of impaired skin integrity related to oedema, dry and rough skin

The person with hypothyroidism is at risk of impaired skin integrity related to the accumulation of fluid in the interstitial spaces and to dry, rough skin. Decreased peripheral circulation,

decreased activity levels and slow wound healing further increase the risk. These interventions are outlined for the older person who is hospitalised for surgery or severe hypothyroidism.

- Monitor skin surfaces for redness or lesions, especially if the person's activity is greatly reduced. Use a pressure injury risk assessment scale to identify people at risk. *Hypothyroidism causes dry, rough, oedematous skin conditions that increase the risk of skin breakdown.*
- Provide or teach the immobile person measures to promote optimal circulation:
  - Use a turning schedule if the person is on bed rest, or teach the person to change position every 2 hours.
  - Limit the time for sitting in one position; shift weight or lift the body using arm rests every 20 to 30 minutes.
  - Use pillows, pads or sheepskin or foam cushions for bed and/or chair.
- Teach and implement a schedule of range-of-motion exercises. *Prolonged pressure, especially in people with oedema and circulatory impairment, can occlude capillaries and cause hypoxic tissue damage.*
- Provide or teach the person measures to maintain skin integrity:
  - Take baths only as necessary; use warm (not hot) water.
  - Use gentle motions when washing and drying skin.
  - Use alcohol-free skin oils and lotions.

  Dry skin and oedema increase the risk of skin breakdown. Hot water, rough massage and alcohol-based preparations may increase skin dryness, further impairing the body's ability to maintain skin integrity.

CONSIDERATION FOR PRACTICE

When moving, positioning or transporting, lift the person up in bed to prevent tissue damage from shearing forces.

Community-based care

People with hypothyroidism require lifelong care, primarily at home. Address the following topics:

- the need to take medications for the rest of their life
- the need for regular periodic dosage reassessments (e.g. every 6 months)
- if the person is older or does not have a support system, advise about helpful community resources.

THE PERSON WITH CANCER OF THE THYROID

The Australian Institute of Health and Welfare (AIHW, 2014) reported that the incidence of thyroid cancer increased by 281% between 1982 and 2014. In 2014, 1890 Australian women and 630 Australian men were diagnosed with thyroid cancer. Thyroid cancer in Australian females is the third most common cancer after breast and melanoma of the skin.

Risk factors associated with thyroid cancer are age, usually affecting people over the age of 35; gender, females having approximately three times the incidence of thyroid cancer than males; diet, either chronic iodine deficiency or chronically high iodine intake; history of thyroid disease; ionising radiation exposure; and family history and ethnicity, with a higher incidence in first-degree relatives (AIHW, 2014; Brennan & French, 2007). Thyroid cancer is more aggressive in men than in women (Gesing et al., 2012).

There are several types of thyroid cancer:

- Papillary thyroid carcinoma (PTC) is the most common thyroid malignancy. The risk is higher in those with a history of nodular goitre (Smith et al., 2013). The average age of diagnosis is 42, with 70% of cases occurring in women. Recently, Cinamon, Levy and Marom (2015) suggested that primary hyperparathyroidism is a possible risk factor for the development of PTC.

  PTC is the least aggressive type, but does metastasise to the local and regional lymph nodes and lungs. The mortality rate of PTC is usually higher in the elderly.
- Follicular thyroid carcinoma (FTC) is the second most common thyroid malignancy and may be associated with iodine deficiency. The average age of diagnosis is 50, with 75% of cases occurring in women. This form is more aggressive, with potential for vascular invasion and spread to lung and bone (Nguyen et al., 2015).

The diagnosis is made by measuring thyroid hormones, performing thyroid scans and fine-needle biopsy. The usual treatment is subtotal or total thyroidectomy. TSH suppression therapy with levothyroxine may be conducted prior to surgery. Radioactive iodine therapy (^{131}I) and chemotherapy are additional therapeutic options. The 5-year survival rate, if the tumour has not metastasised, is 95% for males and 97% for females (AIHW, 2014). Nursing care for the person with cancer is discussed in Chapter 13.

DISORDERS OF THE PARATHYROID GLANDS

Disorders of the parathyroid glands—hyperparathyroidism and hypoparathyroidism—are not as common as those of the thyroid gland. Hypercalcaemia and hypocalcaemia (the primary results of alterations in parathyroid function) are discussed in Chapter 9.

THE PERSON WITH HYPERPARATHYROIDISM

Hyperparathyroidism results from an increase in the secretion of parathyroid hormone (PTH), which regulates normal serum levels of calcium and phosphate. The four parathyroid

glands which secrete PTH are located on the thyroid gland. The two upper parathyroid glands are located on the posterior aspect of the upper thyroid lobes and the two lower parathyroid glands are located in the lower thyroid lobes. Two per cent of people have parathyroid glands situated within the thyroid gland (Sung, 2015).

Pathophysiology and manifestations

Hyperparathyroidism occurs more often in older adults and is three times more common in women. The disorder itself is not common. The three types of hyperparathyroidism are as follows:

1. Primary hyperparathyroidism occurs when there is hyperplasia or an adenoma in one or more of the parathyroid glands, resulting in the unregulated overproduction of parathyroid hormone. The coexistence of thyroid nodules and primary hyperparathyroidism has been reported to range between 12% and 52% (Cinamon et al., 2015). This disorder interrupts the normal regulatory mechanism between serum calcium levels and PTH secretion and increases the absorption of calcium through the gastrointestinal tract.
2. Secondary hyperparathyroidism is a compensatory response by the parathyroid glands to chronic hyperphosphataemia and hypocalcaemia. It is characterised by an increased secretion of PTH. Secondary hyperparathyroidism is found in people with early chronic kidney disease and vitamin D deficiency.
3. Tertiary hyperparathyroidism results from hyperplasia of the parathyroid glands and a loss of response to serum calcium levels. This disorder is most often seen in people with long-standing chronic kidney disease. Due to routine blood analysis of calcium, many people with hyperparathyroidism are identified prior to the development of symptoms. When symptoms occur, they are related to hypercalcaemia and various musculoskeletal, renal and gastrointestinal manifestations. Bone resorption results in pathological fractures, while elevated calcium levels alter neural and muscular activity, leading to muscle weakness and atrophy. Proximal renal tubule function is altered and metabolic acidosis, renal calculi formation and polyuria occur.

Manifestations of the effect of hypercalcaemia on the gastrointestinal tract include abdominal pain, constipation, anorexia and peptic ulcer formation. Hypercalcaemia also affects the cardiovascular system, causing arrhythmias, hypertension and increased sensitivity to cardiotonic glycosides (e.g. digitalis preparations). The manifestations of hyperparathyroidism are summarised in the accompanying box.

INTERPROFESSIONAL CARE

Hyperparathyroidism is diagnosed by excluding all other possible causes of hypercalcaemia; by at least a 6-month history of symptoms; and by laboratory analysis of levels of serum calcium and PTH. Sestamibi nuclear medicine scanning, which is extensively used in cardiac imaging, is also useful as sestamibi accumulates in parathyroid adenomas. Bone mineral density is used to determine the extent, if any, of bone resorption.

Treatment of hyperparathyroidism focuses on decreasing the elevated serum calcium levels. People with mild hypercalcaemia are urged to drink fluids and keep active to prevent dehydration and reduce the risk of kidney stones. They should avoid prolonged immobilisation, thiazide diuretics, large doses of vitamins A and D, antacids containing calcium and calcium supplements. Gastrointestinal illness with vomiting and diarrhoea can cause serum calcium levels to rise. Severe hypercalcaemia requires hospitalisation and intensive treatment with intravenous saline. Medications to inhibit bone resorption and reduce hypercalcaemia or calcimimetic medications such as cinacalcet (Sensipar) are the mainstay of secondary hyperparathyroidism. Calcitonin, a hormone produced by the thyroid gland, helps regulate calcium levels in the body by inhibiting bone resorption and increasing calcium excretion by the kidney (Lehne, 2012). Biphosphinates such as alendronate can be considered for those with symptomatic primary hyperparathyroidism who are unable to undergo surgery. Laser thermal ablation or radiofrequency ablation are other alternative therapies (Sung, 2015).

MANIFESTATIONS Hyperparathyroidism

MUSCULOSKELETAL SYSTEM
- Bone pain (back, joints and shins)
- Pathological fractures
- Muscle weakness
- Muscle atrophy

RENAL EFFECTS
- Renal calculi
- Polyuria
- Polydipsia

GASTROINTESTINAL SYSTEM
- Abdominal pain
- Peptic ulcers
- Pancreatitis
- Insulin resistance
- Nausea
- Constipation

CARDIOVASCULAR SYSTEM
- Arrhythmias
- Hypertension
- Deposition of calcium and phosphate in arterial walls

CENTRAL NERVOUS SYSTEM
- Paraesthesias
- Depression
- Confusion
- Psychosis

METABOLIC EFFECTS
- Acidosis
- Weight loss
- Fatigue

Surgical removal of the parathyroid glands affected by hyperplasia or adenoma treats all forms of hyperparathyroidism. The preoperative and postoperative nursing care is essentially the same as that for the person having a thyroidectomy (see above). In some cases autotransplantation of one parathyroid to the forearm may assist in controlling postoperative calcium homeostasis.

Nursing care

Nursing care of the person with hypercalcaemia is discussed in Chapter 9.

THE PERSON WITH HYPOPARATHYROIDISM

Hypoparathyroidism results from abnormally low PTH levels. The most common cause is inadvertent damage to or removal of the parathyroid glands during thyroidectomy.

Pathophysiology and manifestations

Due to reduced PTH, the ability to resorb calcium from the bone, and to regulate calcium reabsorption from the renal tubules, is impaired. Reabsorption of phosphate is increased in the renal tubules, causing hyperphosphataemia (McCance & Huether, 2009). The low calcium levels cause changes in neuromuscular activity, affecting peripheral motor and sensory nerves.

The neuromuscular manifestations that result from hypocalcaemia include numbness and tingling around the mouth and in the fingertips, muscle spasms of the hands and feet, convulsions and laryngeal spasms. Tetany, a continuous spasm of muscles, is the primary symptom of hypocalcaemia. In severe cases of tetany, death may occur. Assessments for tetany include Trousseau's sign (see Chapters 9 and 17). The manifestations of hypoparathyroidism are summarised in the box opposite.

MANIFESTATIONS Hypoparathyroidism

MUSCULOSKELETAL SYSTEM
- Muscle spasms
- Facial grimacing
- Carpopedal spasms
- Tetany or convulsions

INTEGUMENTARY SYSTEM
- Brittle nails
- Hair loss
- Dry, scaly skin

GASTROINTESTINAL SYSTEM
- Abdominal cramps
- Malabsorption

CARDIOVASCULAR SYSTEM
- Arrhythmias

CENTRAL NERVOUS SYSTEM
- Paraesthesias (lips, hands, feet)
- Mood disorders (irritability, depression, anxiety)
- Hyperactive reflexes
- Psychosis
- Increased intracranial pressure

INTERPROFESSIONAL CARE

Hypoparathyroidism is diagnosed by low serum calcium levels and high phosphorous levels in the absence of renal failure, an absorption disorder or a nutritional disorder.

Treatment of hypoparathyroidism focuses on increasing calcium levels. Intravenous calcium gluconate is given immediately to reduce tetany. Long-term therapy includes supplemental calcium, increased dietary calcium and vitamin D therapy.

Nursing care

Nursing care for the person with hypocalcaemia is discussed in Chapter 9.

DISORDERS OF THE ADRENAL GLANDS

Disorders of the adrenal cortex or adrenal medulla result in changes in the production of adrenocorticotropic hormone (ACTH). Hormones of the adrenal cortex are essential to life and maintain homeostasis in response to stressors. Disorders of the adrenal cortex result in complex physical, psychological and metabolic alterations. The disorders that occur are hyperfunction and hypofunction of the adrenal cortex and hyperfunction of the adrenal medulla.

THE PERSON WITH HYPERCORTISOLISM (CUSHING'S SYNDROME)

Cushing's syndrome is a chronic pathological disorder in which hyperfunction of the adrenal cortex produces symptoms associated with excessive amounts of circulating cortisol or ACTH (see Figure 18.3). Cushing's syndrome is more

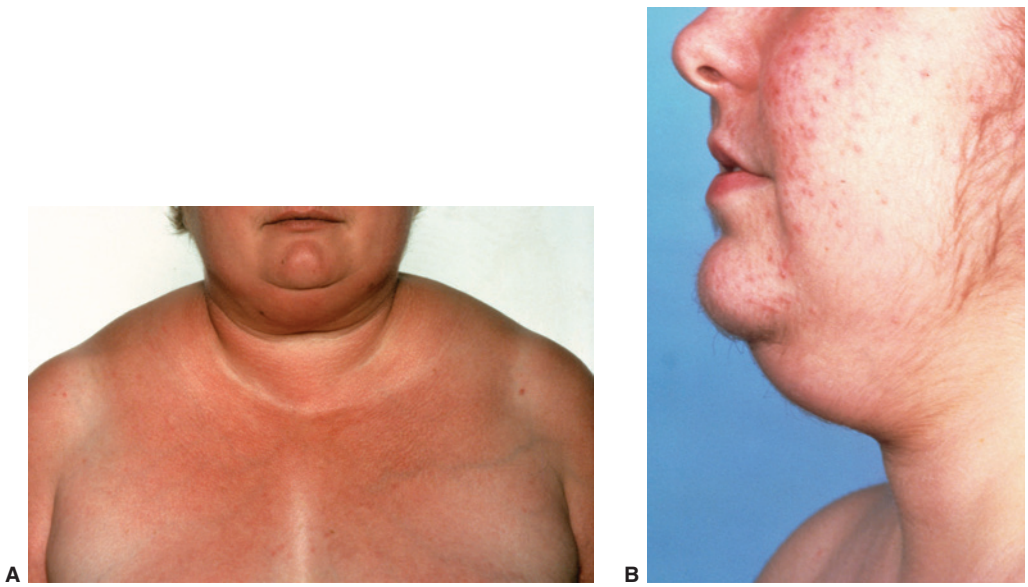


FIGURE 18.3 ■ Among the manifestations of Cushing's syndrome are central obesity; fat deposits around the upper back (*A*), face (*B*) and clavicle; hirsutism; dilation of capillaries; and purple or red striae (due to weight gain)

Sources: A, Wellcome Image Library; *B*, Custom Medical Stock.

common in women, with the average age of onset between 30 and 50 years. However, the disorder may occur at any age, especially as the result of pharmacological therapy. People who take steroids (such as prednisone) for long periods of time are at increased risk of developing the disorder.

Pathophysiology

The most common aetiologies of Cushing's syndrome are as follows:

- The pituitary form, with ACTH hypersecretion by a tumour of the pituitary (called *Cushing's disease*). This is caused by a pituitary adenoma, with persistent but disorderly and random overproduction of ACTH (Lau, Rutledge & Aghi, 2015).
- The ectopic form, caused by ACTH-secreting tumours (usually of the lung or pancreas).
- The adrenal form, resulting from excessive cortisol secretion by a benign or malignant adrenal tumour. The excess secretion suppresses pituitary ACTH production, resulting in atrophy of the uninvolved adrenal cortex. Thirty-two per cent of Cushing's disease is due to excessive autonomous secretion of cortisol by the adrenal glands (McPhee, Papadakis & Tierney, 2008; National Endocrine and Metabolic Diseases Information Service, 2012).
- Iatrogenic Cushing's syndrome, resulting from long-term therapy with potent pharmacological glucocorticoid preparations (steroids).

Manifestations

Excess cortisol results in a redistribution of body fat deposits in the abdominal region (central obesity), fat pads under the clavicle, a 'buffalo hump' over the upper back and a round 'moon' face. Changes in protein metabolism cause peripheral muscle weakness and wasting. Glucocorticoid excess inhibits fibroblasts, resulting in loss of collagen and connective tissue. Thinning of the skin, abdominal striae (reddish purple 'stretch marks'), easy bruising, poor wound healing and frequent skin infections result. Glucose metabolism is altered and type 2 diabetes mellitus may occur. Changes in calcium absorption result in osteoporosis, compression fractures of the vertebrae, fractures of the ribs and renal calculi. Hypokalaemia and hypertension occur as potassium is lost and sodium is retained. Inhibited immune responses increase the risk of infection and increased gastric acid secretion increases the risk of peptic ulcers. Emotional changes range from depression to overt psychosis. In women, increasing androgen levels cause hirsutism (excessive facial hair in particular), acne, menstrual irregularities and infertility. Men could suffer impotence (Lau et al., 2015). The manifestations and effects of Cushing's syndrome are grouped by body system in the box opposite.

If the person undergoes a bilateral adrenalectomy as a treatment for Cushing's syndrome, an acute deficit of cortisol (Addisonian crisis) may result.

INTERPROFESSIONAL CARE

The treatment of Cushing's syndrome includes medications, radiation therapy or surgery, depending on the aetiological origin of the disorder.

Diagnostic tests

Cushing's syndrome is diagnosed through a variety of diagnostic tests. Findings are shown in Table 18.3 and in the following bulleted summary.

- *Plasma cortisol levels* are measured. If Cushing's disease is present, test results show a loss of the normal diurnal variations of higher levels in the morning and lower levels in the afternoon.
- *Plasma ACTH levels* are measured to determine the aetiology of the syndrome. Normally, plasma ACTH levels are

MANIFESTATIONS Cushing's syndrome

MUSCULOSKELETAL SYSTEM
- Weakness
- Osteoporosis
- Peripheral muscle weakness and wasting

INTEGUMENTARY SYSTEM
- Thin, easily bruised skin ('tissue paper' skin)
- Skin infections
- Poor wound healing
- Ecchymosis
- Purple striae (around thighs, breasts, abdomen)
- Hirsutism

CENTRAL NERVOUS SYSTEM
- Emotional lability
- Psychoses

GASTROINTESTINAL SYSTEM
- Peptic ulcers

CARDIOVASCULAR SYSTEM
- Hypertension

RENAL EFFECTS
- Renal calculi
- Polyuria
- Polydipsia
- Glycosuria

METABOLIC EFFECTS
- Hypokalaemia
- Hypernatraemia
- Type 2 diabetes mellitus
- Truncal obesity
- Buffalo hump

REPRODUCTIVE SYSTEM
- Oligomenorrhoea or amenorrhoea
- Impotence
- Decreased libido

highest from 7 am to 10 am and lowest from 7 pm to 10 pm. In secondary Cushing's syndrome, ACTH is elevated; in primary Cushing's syndrome, ACTH is decreased.

- *24-hour urine tests* (17-ketosteroids (17-KS) and 17-hydroxy-corticosteroids) are conducted to measure free cortisol and androgens; these hormones are increased in Cushing's syndrome. Because synthesis and circulation of adrenal hormones are diurnal and episodic, 24-hour urine collections more correctly reflect total hormone than serum levels drawn intermittently.
- *Serum potassium, calcium and glucose levels* are measured to identify electrolyte imbalances.
- *ACTH suppression test* may be conducted to identify the cause of the disorder. A synthetic cortisol (dexamethasone) is given to suppress the production of ACTH and plasma cortisol levels are measured. If an extremely high dose of cortisol is necessary to suppress ACTH, the primary disorder is adrenal cortex hyperplasia. If there is an abnormal release of cortisol after a low-dose suppression test, an adrenal tumour that produces cortisol, a body tumour or a pituitary tumour that produces ACTH is suspected (Stewart & Krone, 2011).

Medications

Cushing's syndrome that results from a pituitary tumour is treated by medications as an adjunct to surgery or radiation. Medical therapies are classified according to their site of action: at the pituitary gland by inhibiting ACTH secretion, at the adrenal gland by inhibiting steroidogenesis or at the target tissue by blocking the glucocorticoid receptor. Examples of some commonly prescribed drugs follow:

- Cabergoline used to suppress lactation or Parkinson's disease is a potent, long-acting dopamine-2 (D2) receptor agonist. Identification of dopamine (D2) receptors in corticotroph tumours has led to clinical trials of cabergoline in Cushing's disease.
- Metyrapone directly suppresses activity of the adrenal cortex and decreases peripheral metabolism of corticosteroids by inhibition of steroidogensis enzyme 11β-hydroxylase. Aminoglutethimide or ketoconazole (or both) inhibit cortisol synthesis by the adrenal cortex and may be administered to people with ectopic ACTH-secreting tumours that cannot be surgically removed. There is growing literature that shows ketoconazole, a well-known antifungal, may benefit by extra-adrenal actions through antagonist activity against glucocorticoid receptors.
- Pituitary adenomas contain receptors for somatostatin (growth hormone release inhibiting factor). Pasireotide diaspartate is an analogue of somatostatin which binds to these receptors. This inhibits the secretion of ACTH.

TABLE 18.3 Laboratory findings in Cushing's syndrome

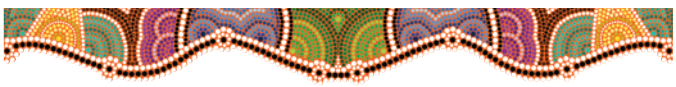
	TEST	NORMAL VALUES	FINDINGS
Serum	Cortisol	am: 82–960 nmol/L	Increased
		pm: 30–550 nmol/L or ≤ 50% of morning peak	
	Blood urea nitrogen	5–25 mg/Dl	Normal
	Sodium	137–146 mmol/L	Increased
	Potassium	3.5–5.0 mmol/L	Increased
	Glucose (serum)	3.0–8.0 mmol/L	Increased
Urine	17-Ketosteroids	Male: 35–76 mmol/day	Increased
		Female: 21–55 mmol/day	

Source: Sydney South West Area Health Service (SSWAHS) Liverpool Hospital Pathology Department (2009). Reproduced with permission.

Surgery

When Cushing's syndrome is caused by an adrenal cortex tumour, an adrenalectomy may be performed to remove the tumour. Only one adrenal gland is usually involved; however, if an ACTH-producing ectopic tumour is involved, a bilateral adrenalectomy is performed. Lifelong hormone replacement is necessary if both adrenal glands are removed. Nursing care of the person having an adrenalectomy is discussed below.

Surgical removal of the pituitary gland (hypophysectomy) is indicated when Cushing's disease is the result of a pituitary disorder. The gland is removed either by a trans-sphenoidal route or by a craniotomy. Immediate cure rates for neurosurgical resection are between 65% and 90% (Lau et al., 2015). Nursing care for the person having cranial surgery is discussed in Chapter 41.



Nursing care

Health promotion

Stress the risk of developing Cushing's syndrome for people taking long-term steroids. The risk of adrenal crisis resulting from the abrupt discontinuation of steroid medications is an essential component of teaching. For a review of glucocorticoid administration, see Chapter 11.

Assessment

Collect the following data through the health history and physical examination (see Chapter 17). Further focused assessments are described with nursing interventions below.

- *Health history:* history of pituitary, adrenal, pancreatic or pulmonary tumour; frequent infections; gastrointestinal bleeding; stress fractures; pain, changes in weight distribution; change in height; fatigue; weakness; change in appearance; bruising; skin infections; menstrual history; sexual function.
- *Physical assessment:* vital signs, behaviour, appearance, fat distribution, face, skin, hair quantity and distribution, muscle size and strength, gait.

Nursing diagnoses and interventions

The nurse caring for the person with Cushing's syndrome must take a holistic approach to plan and implement interventions for a wide variety of responses, including problems related to fluid and electrolyte balance, injury, infection and body image. For additional information about people with alterations in fluid and electrolyte balance, see Chapter 9. See accompanying 'Nursing care plan' below.

Fluid volume excess

The excess cortisol secretion associated with Cushing's syndrome results in sodium and water reabsorption, causing fluid volume excess (Porth & Matfin, 2009). The person will have weight gain, oedema and hypertension.

- Ask the person to weigh themself at the same time each day and maintain a record of results. Body weight is an accurate indicator of fluid status. *One litre of fluid retention corresponds to about 1 kg of body weight.*
- Monitor blood pressure, rate and rhythm of pulse, respiratory rate and lung sounds. Assess for peripheral oedema and jugular vein distension. *Extracellular fluid volume excess resulting from sodium and water retention is manifested by hypertension and a pounding, rapid pulse. There may also be crackles and wheezes on lung auscultation, dependent oedema and venous distension.*
- Teach the person and family the reasons for the importance of limiting fluids as ordered. *Restricting fluid can help decrease the risk of fluid volume excess. Involving the person and family in the plan of care and teaching the rationale for interventions helps to achieve goals and minimise anxiety.*

NURSING CARE OF THE PERSON Having a laparoscopic adrenalectomy

PREOPERATIVE CARE

- Request a dietary consultation to discuss with the person a diet high in vitamins and proteins. If hypokalaemia exists, include foods high in potassium. *Glucocorticoid excess increases catabolism. Vitamins and proteins are necessary for tissue repair and wound healing following surgery.*
- Use careful medical and surgical asepsis when providing care and treatments. *Cortisol excess increases the risk of infection.*
- Monitor the results of laboratory tests of electrolytes and glucose levels. *Electrolyte and glucose imbalances must be corrected before the person has surgery.*
- Teach the person to turn, cough and perform deep-breathing exercises. *Although they are important for all surgical people, these activities are even more important for the person who is at risk of infection. Having the person practise and demonstrate the activities increases postoperative compliance.*

POSTOPERATIVE CARE

- Take and record vital signs, measure intake and output, and monitor electrolytes regularly, especially potassium during the first 48 hours after surgery. *Removal of an adrenal gland, especially a bilateral adrenalectomy, results in adrenal insufficiency. Addisonian crisis and hypovolaemic shock may occur. Cortisol is often given on the day of surgery and in the postoperative period to replace inadequate hormone levels. Intravenous fluids are also administered.*
- Assess body temperature, WBC levels and wound drainage. Change dressings using sterile technique. *Impaired wound healing increases the risk of infection in people with adrenal disorders. Use aseptic technique to decrease this risk.*
- Monitor for pain and administer analgesia as ordered. *Sufficient pain relief assists in early ambulation and reduced length of stay.*

NURSING CARE PLAN A person with Cushing's syndrome

Sara Domico is a 30-year-old solicitor living in a major metropolitan area. She has never been married and she shares her life with her cat, Beau, and her parents, who live nearby. Her doctor recently diagnosed Ms Domico as having Cushing's syndrome and admits her for surgery for an adrenal cortex tumour (adrenalectomy). She has been having increased muscle weakness, so much so that she has difficulty climbing the one set of stairs to her apartment. She has also had difficulty sleeping, irregular menstrual periods and hypertension.

ASSESSMENT

When Ms Domico arrives at the hospital the morning of surgery, she is admitted by the ward nurse, Ann Sprengel, Registered Nurse (RN), Clinical Nurse Specialist (CNS). Ann completes a physical assessment that includes abnormal findings of thin lower extremities, an enlarged abdomen, purple striae over the abdomen and buttocks, a round face and obvious facial hair. Ms Domico's blood pressure is 160/96. She tells Ann that she is always tired and that sometimes it 'just wears me out to walk from the bedroom to the kitchen'. Diagnostic tests conducted prior to admission reveal the following abnormal findings (all except cortisol levels are corrected before surgery):

Glucose: 16 mmol/L (normal range: 3.0–7.0 mmol/L)
Sodium: 152 mmol/L (normal range: 137–146 mmol/L)
Potassium: 3.2 mmol/L (normal range: 3.5–5.0 mmol/L)
Calcium: 2.10 mmol/L (normal range: 2.10–2.60 mmol/L)
Cortisol: 1150 nmol/L (normal for am: 82–960 nmol/L)

(South-Western Sydney Local Health District (SWSLHD) Liverpool Hospital Pathology Department, 2012).

DIAGNOSES

- *Fluid volume excess* related to sodium retention causing oedema and hypertension.
- *Risk of injury* related to generalised fatigue and weakness.
- *Risk of infection* related to impaired immune response and oedema.
- *Disturbed body image* related to physical changes secondary to Cushing's syndrome.

PLANNING

- Organise pre- and post-surgery education for Ms Domico involving an educator, specialist nurse, dietitian and other relevant allied health professionals.
- Develop a plan of pre-op care and recovery post surgery.
- Discuss the importance of monitoring and informing staff about intake and output of fluids as well as maintaining a record.
- Discuss about risks and risk prevention while hospitalised.
- Develop a written schedule of rest and activity periods.

Expected outcomes

- Regain a normal body fluid balance.
- Remain free of injury.
- Remain free of infection.
- Verbalise an understanding of the physical effects of the disease process and have realistic expectations of desired changes in appearance.

IMPLEMENTATION

- Weigh each morning, using the same scale.
- Maintain an accurate record of intake and output.
- Ensure adequate lighting in the room and wear glasses and shoes when getting out of bed.
- If possible, provide a private room and restrict visitors at this time after consulting with the person.
- Use strict medical and surgical asepsis when providing care.
- Provide time for discussion of the disease and treatment; encourage verbalisation of feelings and identify successful coping mechanisms used in the past.
- Encourage turning, coughing and deep breathing, and/or incentive spirometry every 2–4 hours.

EVALUATION

Ms Domico states that she is 'ready to have surgery and start feeling better'. She has not fallen or injured herself and she has remained free of infection. Although oedema is still present, she has lost 3.6 kg and her blood pressure has decreased. Ms Domico has openly discussed her concerns about the way she looks and feels; she understands that symptoms will improve following surgery. She has strong religious beliefs and family support, both of which provide strength and help her cope with the effects of the disorder and the need for any further treatment.

CRITICAL THINKING IN THE NURSING PROCESS

1. When Ms Domico was admitted to the hospital, several of her test results were abnormal. Describe the pathophysiological reason for those results.
2. List the assessments that nurses can make to determine body fluid balance.
3. Develop a plan of care for this person for the nursing diagnosis of *Fatigue*.

REFLECTION ON THE NURSING PROCESS

1. What have you learned from this scenario that you will utilise in your future nursing practice?
2. Develop a plan of education to Ms Domico during her recovery from surgery and plan for her discharge home, taking into consideration her social status such as her profession, living arrangements, infection risk from her pet, etc.

Risk of injury related to decreased bone density

The person with Cushing's syndrome is at risk of injury from several causes. Excess cortisol causes increased absorption of calcium and demineralisation of bones, resulting in an increased risk of pathological fractures, particularly in the vertebra and ribs (Arduc et al., 2014). Muscle weakness and fatigue are common, increasing the potential for accidental falls.

- Teach the person and family to maintain a safe environment:
  - Keep unnecessary clutter and equipment out of the way and off the floor.
  - Ensure adequate lighting, especially at night.
  - Encourage the use of assistive devices for ambulation or to ask for help if needed.
  - If the person wears corrective lenses, be sure they are available and clean.

- Encourage the use of non-slippery slippers or shoes.
- Consider wearing hip protectors when ambulant.
- Monitor for signs of fatigue (increased pulse and respirations); plan rest periods.

  A well-lit environment free of clutter decreases the risk of falls and injury. Sensory and motor deficits increase the risk of falls; corrective lenses, assistive devices and non-slippery footwear can decrease this risk. Hip protectors, although bulky, reduce the risk of hip fractures. Rest relieves fatigue. To reduce energy expenditure, include alternating periods of rest and activity in daily schedules.

Risk of infection

Elevated cortisol levels impair the immune response, increasing the risk of infection. Increased cortisol also affects protein synthesis, causing delayed wound healing, and inhibits collagen formation, which results in epidermal atrophy, further inhibiting resistance to infection. In addition, impaired blood flow to oedematous tissue results in altered cellular nutrition, which increases the potential for infection. The following interventions are outlined for the person with Cushing's syndrome who is hospitalised:

- Place in a private room, if possible and limit visitors. *The person must avoid exposure to environmental infection.*
- Monitor vital signs and verbalisations of subjective manifestations (for example, the person's response to 'How do you feel?') every 4 hours. *Increased body temperature and pulse are systemic indicators of infection; however, because Cushing's syndrome impairs the normal inflammatory response, the usual indicators of inflammation such as fever may not be present.*
- Pat skin dry gently after bathing. *Rough drying with towels increase risk of skin tears and skin infections.*
- Use principles of medical and sterile asepsis when conducting procedures or providing wound care. *Impaired skin and tissues make aseptic techniques even more necessary to decrease the risk of infection. Intact, clean and dry skin is the first line of defence against infection.*
- If wounds are present, assess the colour, odour and consistency of wound drainage and look for increased pain in and around the wound. Use a wound chart to keep a record of the wound progress.
- Teach the importance of increasing oral intake of protein and vitamins C and A. *Protein, vitamin C and vitamin A are necessary for collagen formation; collagen helps support and repair body tissues.*

CONSIDERATION FOR PRACTICE

A generalised feeling of malaise may be the primary manifestation of infection.

Disturbed body image

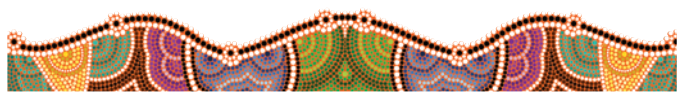
The person with Cushing's syndrome has obvious physical changes in appearance. The abnormal fat distribution, moon face, buffalo hump, thin skin, striae, acne and facial hair (in women) all contribute to disruptions in the way people with this disorder perceive themselves.

- Encourage people to express feelings and to ask questions about the disorder and its treatment. *The loss of one's normal body image may prompt feelings of hopelessness, powerlessness, anger and depression. Understanding the disease and adapting to changes from that disease are the first steps in regaining control of one's own body.*
- Discuss strengths and previous coping strategies. Enlist the support of family or significant others in reaffirming the person's worth. *Disturbances in body image are often accompanied by low self-esteem. Self-esteem derives from one's perception of competence and from appraisals of others.*
- Discuss signs of progress in controlling symptoms; for example, decreased facial oedema or increased activity tolerance. *Many physical changes from cortisol excess disappear with treatment. Clearly communicate this fact, because the person may believe changes are permanent.*

Community-based care

The person with Cushing's syndrome requires education about specific self-care responsibilities at home. Address the following topics:

- Safety measures to prevent falls if fatigue, weakness and osteoporosis are present.
- Taking medications as prescribed, with information about side effects. People often require medications for the rest of their lives and dosage changes are highly likely.
- Having regular health assessments.
- Wearing a MedicAlert® bracelet indicating the person has Cushing's syndrome.
- Helping the older person with referrals to social services or community health services because of the complexity of the treatment and care required.
- Providing helpful resources:
  - Australian Pituitary Foundation Ltd. Tel. 1300 331 807. www.pituitary.asn.au
  - The Endocrine Society of Australia. Tel. (02) 9256 5405. www.endocrinesociety.org.au.



THE PERSON WITH CHRONIC ADRENOCORTICAL INSUFFICIENCY (ADDISON'S DISEASE)

Addison's disease is a rare endocrine disorder that in Australia affects about 4 to 11 people per 100 000. In children, boys have an incidence of 75%; in adults, women constitute 70% of all cases (O'Connell & Siafarikas, 2010). The disease is characterised by low serum cortisol levels which manifest by weight loss, muscle weakness, fatigue, low blood pressure and sometimes darkening of the skin in both exposed and unexposed parts of the body (Australian Addison's Disease Association, 2006).

Pathophysiology

There are many possible causes of Addison's disease:

- Autoimmune destruction of the adrenals. This is the most common cause in the Western world, accounting for about 70% to 80% of all cases (O'Connell & Siafarikas, 2010). It may occur alone or as part of a polyglandular autoimmune syndrome (PGA). Type 2 PGA in adults is often associated with autoimmune thyroid disease, type 1 diabetes, systemic lupus erythematosus, primary ovarian or testicular failure, and pernicious anaemia (Godswill & Odigie, 2014).
- It is also seen in people who are taking anticoagulants, have had major trauma or sepsis, or are having open-heart surgery. Such people may have bilateral adrenal haemorrhage.
- Adrenoleukodystrophy, an X-linked disorder characterised by an accumulation of very-long-chain fatty acids in the adrenal cortex, testes, brain and spinal cord.
- ACTH deficit, resulting from pituitary tumours, pituitary surgery or irradiation, and the use of exogenous steroids.
- People who are abruptly withdrawn from long-term, high-dose steroid therapy. Other people at risk are those with tuberculosis or acquired immune deficiency syndrome (AIDS); the pathogens responsible for either disease can infiltrate and destroy adrenal tissue.

Adrenocortical destruction initially causes a decrease in adrenal glucocorticoid reserves. Basal glucocorticoid secretion is normal, but does not increase in response to stress and surgery. As the destruction of the adrenal cortex continues, even basal secretion of glucocorticoids and mineralocorticoids is deficient. Decreasing plasma cortisol reduces the feedback inhibition of pituitary ACTH and plasma ACTH rises.

Secondary adrenocortical insufficiency occurs when either large doses or prolonged therapy with glucocorticoids are given for their anti-inflammatory and immunosuppressive effects to treat autoimmune diseases such as arthritis and asthma. If the steroid medications are suddenly discontinued, the hypothalamus and pituitary cannot respond normally to the reduced level of circulating glucocorticoids. The person may develop manifestations of chronic adrenocortical insufficiency or, if subjected to stress, adrenal (Addisonian) crisis (McPhee et al., 2008).

Manifestations

The onset of Addison's disease is slow in most cases; the person experiences symptoms after about 90% of the function of the gland is lost. The primary manifestations are the result of elevated ACTH levels and decreased aldosterone and cortisol. Aldosterone deficiency affects the ability of the distal tubules of the nephron to conserve sodium. Sodium is lost, potassium is retained, extracellular fluid is depleted and the blood volume is decreased. Due to sodium loss, the person may have a craving for salt. Postural hypotension and syncope are common and hypovolaemic shock may occur. Hyponatraemia causes dizziness, confusion and neuromuscular irritability. Hyperkalaemia causes cardiac arrhythmias.

Cortisol insufficiency also causes decreased hepatic gluconeogenesis with hypoglycaemia. The person tolerates stress poorly and experiences lethargy, weakness, anorexia, nausea, vomiting and diarrhoea. The increased ACTH levels stimulate hyperpigmentation. In Caucasian people, the skin looks deeply suntanned or bronzed in both exposed and unexposed areas (Godswill & Odigie, 2014).

Addisonian crisis

Addisonian or **adrenal crisis** is a life-threatening response to acute adrenal insufficiency and occurs in about 25% of people (Australian Addison's Disease Association, 2006). Triggers include surgery, acute systemic illness, trauma or abrupt withdrawal of long-term corticosteroid therapy. The disorder is chronic after the acute episode resolves.

This response can occur in any person with Addison's disease; however, it is most commonly precipitated by major stressors, especially if the disease is poorly controlled.

The person with Addisonian crisis may have any of the manifestations of Addison's disease, but the primary symptoms develop rapidly: a high fever; weakness; severe, penetrating pain in the abdomen, lower back and legs; severe vomiting; diarrhoea; hypotension; and circulatory collapse, shock, seizures and coma.

MANIFESTATIONS Addison's disease

INTEGUMENTARY SYSTEM
- Delayed wound healing
- Hyperpigmentation

CARDIOVASCULAR SYSTEM
- Postural hypotension
- Arrhythmias
- Tachycardia

CENTRAL NERVOUS SYSTEM
- Lethargy
- Tremors
- Emotional lability
- Confusion

MUSCULOSKELETAL SYSTEM
- Weakness
- Muscle wasting
- Joint pain
- Muscle pain

GASTROINTESTINAL SYSTEM
- Anorexia
- Nausea and vomiting
- Diarrhoea

REPRODUCTIVE SYSTEM
- Menstrual changes

METABOLIC EFFECTS
- Hyperkalaemia
- Hyponatraemia
- Hypoglycaemia

Treatment of adrenal crisis is emergency resuscitation, restoring and maintaining circulating fluid, intravenous hydrocortisone, management of hypoglycaemia, and identification and treatment of precipitating factors. Admission to an intensive care unit may be necessary in most cases (O'Connell & Siafarikas, 2010).

INTERPROFESSIONAL CARE

The person with Addison's disease requires early diagnosis and treatment. Medical treatment includes cortisol replacement therapy.

Diagnostic tests

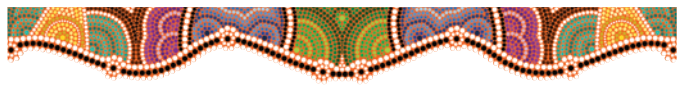
Addison's disease is diagnosed through findings of decreased levels of cortisol, aldosterone and urinary 17-KS. Dehydration may result in increased haematocrit and blood urea nitrogen (BUN). Blood glucose levels are decreased and potassium is increased. A list of laboratory findings in Addison's disease is shown in Table 18.4. The following diagnostic tests are used:

- *serum cortisol levels*, which are decreased
- *blood glucose levels*, which are decreased
- *serum sodium levels*, which are decreased
- *serum potassium levels*, which are increased
- *BUN levels*, which are increased
- *urinary 17-hydroxycorticoids* and *17-KS levels*, which are decreased
- *plasma ACTH levels*, which are increased in primary adrenal insufficiency but decreased in secondary adrenal insufficiency
- *possibly ACTH stimulation test* (cortisol levels rise with pituitary deficiency but do not rise in primary adrenal insufficiency)
- *CT scans* of the head, which identify any intracranial lesion impinging on the pituitary gland.

Medications

Treatment of Addison's disease involves replacing the hormones that the adrenal glands are not making. Cortisol is replaced orally with oral hydrocortisone. If aldosterone is also deficient, it is replaced with oral doses of a mineralocorticoid, fludrocortisone acetate (Florinef), which is taken once a day. Aldosterone replacement therapy is usually accompanied with an increased salt intake. The doses of each of these medications are adjusted to meet the needs of individuals. During an Addisonian crisis, low blood pressure, low blood sugar and high levels of potassium can be life threatening. Standard therapy involves intravenous injections of hydrocortisone, saline (salt water) and dextrose (sugar). When the person can take fluids and medications by mouth, the amount of hydrocortisone is decreased until a maintenance dose is achieved.

Nursing implications in cortisol replacement are given in the following 'Medication administration' box.



Nursing care

Health promotion

Health promotion interventions for the person with or at risk of Addison's disease focus on careful assessments during anticoagulant therapy, open-heart surgery and trauma treatment. If the disease is present, teaching to prevent or treat an Addisonian crisis is essential.

Assessment

Collect the following data through the health history and physical examination (see Chapter 17). Further focused assessments described with nursing interventions follow.

- *Health history:* weight loss, changes in skin colour, nausea and vomiting, anorexia, diarrhoea, abdominal pain, weakness, amenorrhoea, changes in sexual desire, confusion and stress intolerance.
- *Physical assessment:* height and weight, vital signs, skin, hair quality and distribution, muscle size and strength.

Nursing diagnoses and interventions

The person with Addison's disease requires nursing care for a wide variety of responses to the decrease in cortisol levels. Nursing diagnoses discussed in this section are directed towards problems with fluid and electrolyte balance and compliance with lifelong self-care. See the accompanying 'Nursing care plan' below.

TABLE 18.4 Laboratory findings in Addison's disease

	TEST	NORMAL VALUES	FINDINGS
Serum	Cortisol	am: 82–960 nmol/L pm: 30–550 nmol/L or ≤ 50% of morning peak	Decreased
	Blood urea nitrogen	5–25 mg/dL	Increased
	Sodium	137–146 mmol/L	Decreased
	Potassium	3.5–5.0 mmol/L	Increased
	Glucose (serum)	3.0–8.0 mmol/L	Decreased
Urine	17-Ketosteroids	Male: 35–76 mmol/day Female: 21–55 mmmol/L	Low/absent

Source: South-Western Sydney Area Health Service (SSWAHS) Liverpool Hospital Pathology Department (2012). Reproduced with permission.

Deficient fluid volume related to water and sodium loss

Fluid volume deficit in the person with Addison's disease results from loss of water and sodium, as well as from vomiting and diarrhoea. Extracellular fluid volume deficit, decreased cardiac output, hypotension and hypovolaemic shock may occur, especially in crisis situations. Interventions for this diagnosis are outlined for the person who is hospitalised.

- Monitor intake and output and assess for signs of dehydration: dry mucous membranes; thirst; poor skin turgor; sunken eyes; scanty or low, dark urine; increased urine specific gravity; weight loss; and increased haemoconcentration (increased haematocrit and BUN). *Glucocorticoid and mineralocorticoid depletion causes fluid volume deficit. Fluid volume deficit may reach crisis levels if undetected, causing altered tissue perfusion and hypovolaemic shock.*
- Monitor cardiovascular status: take and record vital signs, assess character of pulses, monitor potassium levels and electrocardiogram (ECG). *Fluid volume deficit may lead to hypotension and a rapid, weak or thready pulse. As aldosterone levels fall, renal excretion of potassium decreases and causes hyperkalaemia.*
- Weigh the person daily at the same time and in the same clothing. *Dehydration is manifested by weight loss.*
- Encourage an oral fluid intake of 3000 mL per day and an increased salt intake. Cortisol deficiency increases fluid loss, leading to extracellular fluid volume depletion. Oral fluid replacement is necessary to balance this loss. *An increase in dietary sodium can decrease the hyponatraemia characteristic of adrenal insufficiency.*
- Teach to sit and stand slowly and provide assistance as necessary. *Extracellular fluid volume deficit causes orthostatic hypotension, dizziness and possible loss of consciousness. These manifestations increase the risk of injury from falls.*

MEDICATION ADMINISTRATION Addison's disease

CORTISOL REPLACEMENTS

Cortisone acetate (Cortate, Cortef)
Hydrocortisone (Hysone, Solu-Cortef, Hydrocortisone sodium succinat)
Fludrocortisone acetate (Florinef)
Dexamethasone (Dexmethsone, Celestone chronodose, DBL Dexamethasone sodium phosphate injections, Betamethasone acetate sodium phosphate)
Prednisone (Sone, Predsone)
Prednisolone (Solone, Predsolone, Rediprep oral liquid, Panafcortelone, Panafcort, Predmix oral liquid)
Methylprednisolone (Medrol, Solu-Medrol)

(Australian trade names of medications, Australian Prescriber 2015)

NURSING ALERT

Prednisolone and prednisone are equivalent in potency; dexamethasone is four to five times more potent.

Adrenocorticosteroids are used for replacement therapy in acute and chronic adrenal insufficiency. These drugs have anti-inflammatory and immunosuppressant effects. They also facilitate coping with stress.

When these drugs are administered in small doses for replacement therapy, side effects are uncommon. Large doses or prolonged therapy may cause a Cushing's-like syndrome, with atrophy of the adrenal cortex. Older people, especially postmenopausal women, are more prone to develop hypertension and osteoporosis when undergoing glucocorticoid therapy. These drugs are used with caution in children and the older adult and are not usually administered to pregnant women.

Nursing responsibilities

- Establish baseline data, including mental status, neurological function, vital signs and weight.
- Identify medications that might interact with corticosteroids: hypoglycaemic medications, cardiac glycosides, oral contraceptives, anticoagulants, NSAIDs.
- Document and report increased blood pressure, oedema or weight gain, bleeding or bruising, weakness or manifestations of Cushing's syndrome.
- Administer oral forms of the drug with food to minimise its ulcerogenic effect.
- Monitor electrolyte levels for hypernatraemia and hypokalaemia.
- Monitor capillary blood glucose for hyperglycaemia in the diabetic person.

Health education for the person and family

- Take medications with food or milk and report any gastric distress or dark stools (malaena).
- Most people need to take the medications for the rest of their lives.
- Consume a diet that is low in potassium and higher in sodium and protein.
- Weigh yourself each day at the same time and report any consistent weight gain, which indicates fluid retention.
- Use safety measures in the home to prevent falls and injuries.
- Corticosteroids may impair the effectiveness of oral contraceptives.
- Take the medication regularly and continuously.
- Obtain and carry a MedicAlert® bracelet that says 'Adrenal insufficiency—takes hydrocortisone'.
- Monitor for increased stressors (infection, dental work, personal crisis) and increase the dose as indicated by the doctor.
- Anticoagulant drugs or insulin may decrease the effectiveness of corticosteroids.
- Report signs of dizziness on sitting or standing, nausea and vomiting, pain, thirst, feelings of anxiety, malaise, infections to your doctor.

NURSING CARE PLAN A person with Addison's disease

A 51-year-old unemployed salesman, Mr Don Sardoff, is brought to the emergency department by his wife, Ellen, at 8 am. Mrs Sardoff tells the emergency nurse that her husband has not been feeling well for the last week, but that when he got up this morning he was so weak he couldn't dress himself and didn't know where he was. Mrs Sardoff also tells the nurse that her husband has been taking a cortisone drug for treatment of his rheumatoid arthritis for the past 2 years, but stopped taking the medication as she notes, 'We didn't have the money to buy it this month.'

ASSESSMENT

On admission to the emergency room, Mr Sardoff is dehydrated, with dry oral mucous membranes and tongue, poor skin turgor and sunken eyes. His blood pressure is 94/44 and his pulse is rapid and weak. He is lethargic, dizzy and disoriented of time and place. Diagnostic tests reveal the following abnormal findings at 8:30 am:

- ECG: widening QRS complex and increased PR interval
- Sodium: 129 mmol/L (normal range: 137–146 mmol/L)
- Glucose: 2.9 mmol/L (normal range: 3.0–8.0 mmol/L)
- Potassium: 5.3 mmol/L (normal range: 3.5–5 mmol/L)
- Cortisol: 71 nmol/L (normal for am: 82–960 nmol/L)

The medical orders for Mr Sardoff include intravenous administration of 4% dextrose in 1/5 normal saline at 250 mL/h and hydrocortisone (Solu-Cortef) 200 mg. After the fluids and medication are initiated, Mr Sardoff is admitted to the hospital.

DIAGNOSES

- *Deficient fluid volume* related to hypovolaemia secondary to adrenal insufficiency.
- *Ineffective peripheral tissue perfusion* related to fluid volume deficit.
- *Anxiety* related to lack of knowledge about the effects and treatment of adrenal insufficiency.

PLANNING

- Discuss a diet that is high in sodium, low in potassium and has an increased fluid intake (3000 mL per day). Discuss the types of fluids desired and the best times for intake of increased fluids.
- Discuss and determine the need for and amount of intravenous therapy if the oral intake is not sufficient to restore circulating volume.
- Provide verbal and written instructions and encourage verbal feedback about the causes and effects of the disease, the effects of medications and the effects of not taking long-term cortisone drugs or abruptly stopping this medication.

Expected outcomes

- Regain normal fluid balance.
- Regain normal peripheral perfusion with blood pressure within normal range.
- Verbalise knowledge of the causes and effects of adrenal insufficiency.

IMPLEMENTATION

- Monitor intake and output closely.
- Take and record weight at the same time daily.
- Monitor blood pressure, pulses and skin turgor every 2 hours until stable, then four times a day.
- Monitor electrolytes and report abnormal results.
- Assist during activity to prevent falls.
- Ensure steroid medication is taken regularly or if the medication is to stop, to reduce the dosage in small doses over an extended period of time.

EVALUATION

Following treatment for acute adrenal insufficiency, Mr Sardoff is no longer dehydrated and his blood pressure has returned to his normal reading of 132/88. He is alert and oriented and anxious to learn to care for himself at home. After dietary instructions and teaching for self-care that included his wife, Mr Sardoff verbalises an understanding of his illness and the need to take his medication carefully and accurately.

CRITICAL THINKING IN THE NURSING PROCESS

1. Adrenal insufficiency is often diagnosed only when the person becomes seriously ill in response to a stressor. Explain why this statement is or is not true.
2. Describe the physical assessments that are found in the severely dehydrated person.
3. Outline a teaching plan for Mr Sardoff with foods for a high-sodium, low-potassium diet.

REFLECTION ON THE NURSING PROCESS

1. Outline what have you learned from this case study and how you are going to apply this new knowledge to your future practice.
2. Find out and discuss the financial cost of medications to treat adrenal insufficiency in Australia. What assistance is available to cover the cost of medications and/or treatment?

CONSIDERATION FOR PRACTICE

Hyperkalaemia causes changes in cardiac muscle function, which are reflected in ECG changes.

Community-based care

The person with Addison's disease provides self-care at home. One of the most important components of caring for the person with Addison's disease is teaching both the person and family to provide care. Family stability, an awareness of the serious nature of the disease and the effectiveness of treatment all promote compliance. The length of treatment and the side effects of medications, however, can discourage compliance. In addition to the information in the teaching topics included with nursing diagnoses and interventions, include the following topics:

- the importance of continuing healthcare
- referral to social worker, if appropriate
- referral to community agencies for continued education and support
- helpful resource:
  - Australian Addison's Disease Association Inc. Tel: (02) 6652 4761. www.addisons.org.au

THE PERSON WITH PHEOCHROMOCYTOMA

Pheochromocytomas are tumours of the adrenal medulla. These tumours produce catecholamines (adrenaline or noradrenaline) that stimulate the sympathetic nervous system. A pheochromocytoma tends to have sudden releases of hormones, which cause a sudden 'attack' or onset of symptoms. Symptoms may include hypertension, severe headaches, palpitations, excessive truncal sweating, cold and clammy skin, nausea and vomiting, blurry vision, dyspnoea, severe anxiety or panic attacks. These symptoms may last for a few seconds to several hours (Pheochromocytoma and Paraganglioma Research Support Organization (PRESSOR), 2006). Attacks are often precipitated by physical, emotional or environmental stimuli. Although pheochromocytomas are rare, with an annual incidence of about 10 per million, 10% can be malignant (PRESSOR, 2006).

A pheochromocytoma is diagnosed by increased catecholamine levels in the blood or urine, x-ray studies and surgical exploration. Because catecholamine secretion is episodic, a 24-hour urine test is a better surveillance method than serum catecholamines (Dugdale, Zieve & Black, 2013). Surgical removal of the tumour(s) by adrenalectomy is the treatment of choice.

DISORDERS OF THE PITUITARY GLAND

The pituitary gland produces hormones that affect multiple body systems through regulation of endocrine function. Target tissues include the thyroid, adrenal cortex, ovary, uterus, mammary glands, testes and kidneys. Disorders result from an excess or deficiency of one or more of the pituitary hormones due to a pathological condition within the gland itself or to hypothalamic dysfunction.

Although disorders of the pituitary cause diverse and serious problems, they are not as common as disorders of other endocrine glands. Hyperpituitarism and hypopituitarism are discussed in this section.

THE PERSON WITH DISORDERS OF THE ANTERIOR PITUITARY GLAND

The most common cause of hyperpituitarism is a benign adenoma. The manifestations result from pressure on the optic nerve causing visual changes or an excess of growth hormone (GH), prolactin (PRL), ACTH or TSH. Typically, 70% to 90% of the anterior pituitary is damaged before clinical manifestations develop (Porth & Matfin, 2009).

Conditions causing hypopituitarism include pituitary tumours, surgical removal of the pituitary gland, radiation and pituitary infarction, infection or trauma.

Pathophysiology and manifestations

Growth hormone (also called somatotropin) is produced by cells in the anterior pituitary throughout life. GH is necessary for growth and also contributes to metabolic regulation. GH stimulates all aspects of cartilage growth and one of its major effects is to stimulate the growth of the epiphyseal cartilage plates of the long bones. In addition, other body tissues respond to the metabolic effect of GH with increases in bone width and the growth of visceral and endocrine organs, skeletal and cardiac muscle, skin and connective tissue. Gigantism and acromegaly (discussed next) result from overstimulation of GH. Growth retardation and short stature result from deficient production of GH.

Hypersecretion of PRL affects reproductive and sexual function. Women may have irregular or absent menses, difficulty becoming pregnant and decreased libido. Men may be impotent and have decreased libido. PRL deficiency postpartum causes a failure to lactate.

An excess secretion of ACTH overstimulates the adrenal cortex, which in turn increases secretion of adrenal hormones. The result is Cushing's syndrome. Deficiencies of TSH are uncommon, but cause hypothyroidism.

Gigantism

Gigantism occurs when GH hypersecretion begins before puberty and the closure of the epiphyseal plates. The person becomes abnormally tall, often exceeding 213 cm in height, but body proportions are relatively normal.

Acromegaly

Acromegaly, which literally means 'enlarged extremities', occurs when sustained GH hypersecretion begins during adulthood, most commonly because of pituitary tumours. As a result of constant stimulation, bone and connective tissue continue to grow. The forehead enlarges, the maxilla lengthens, the tongue enlarges and the voice deepens (see Figure 18.4).

Other manifestations include peripheral nerve damage from entrapment of nerves causing pin and needles in the hands, swelling of hands and feet, joint pains, headache, gaps between the teeth, barrel chest, thick and oily skin and strong body odour, hair overgrow, snoring or drooling while asleep, hyperhidrosis (heavy sweating), hypertension, congestive heart failure due to cardiomegaly and visual disturbances. Impaired glucose tolerance and diabetes may also occur. Arthralgia develops secondary to the bone and connective tissue growth and may be relieved by treatment that halts excessive GH production.

INTERPROFESSIONAL CARE

Acromegaly is treated by surgical removal or irradiation of the pituitary tumour. A transsphenoidal or transfrontal surgical procedure is most commonly used. Octreotide (Sandostatin) suppresses the anterior pituitary gland and decreases GH levels. Gastrointestinal side effects are common. About 25% of people develop gallstones (Lehne, 2012).

FIGURE 18.4 ■ In acromegaly progressive alterations in facial appearance include enlargement of the cheekbones and jaw, along with thickening of soft-tissue structures such as the nose, lips, cheeks and the flesh above the brows

Source: © Barcroft Media/Splash News/Corbis.

Nursing care

People with anterior pituitary disorders require interventions to help in coping with physical and emotional changes, as well as to prevent complications involving other organs and functions of the endocrine system. Nursing care for the person having cranial surgery is discussed in Chapter 41.

THE PERSON WITH DISORDERS OF THE POSTERIOR PITUITARY GLAND

Disorders of the posterior pituitary are related primarily to excessive or deficient antidiuretic hormone (ADH) secretion. The disorders discussed here are the syndrome of inappropriate ADH secretion and diabetes insipidus.

Pathophysiology and manifestations

Antidiuretic hormone is secreted in response to serum osmolality, which is monitored by osmoreceptors in the hypothalamus. When a condition of hyperosmolarity occurs, ADH secretion increases and renal water is reabsorbed. Hypo-osmolality causes the suppression of ADH and renal water excretion increases.

Syndrome of inappropriate ADH secretion

The **syndrome of inappropriate ADH secretion (SIADH)** is characterised by high levels of ADH in the absence of serum hypo-osmolality. This disorder is most often caused by the ectopic production of ADH by malignant tumours. A transient form may follow a head injury, pituitary surgery or the use of medications such as barbiturates, anaesthetics or diuretics.

Manifestations of SIADH occur as a result of excess water in relation to sodium in the extracellular fluid (Ellison & Berl, 2007). Blood volume expands, but the plasma is diluted. Aldosterone is suppressed and, as a result, renal excretion of sodium increases. Water moves from the hypotonic plasma and the interstitial spaces into the cells.

Manifestations of SIADH (see Chapter 9, Table 9.6) are usually non-specific but are related to hyponatraemia and water intoxication. Brain cells swell, causing neurological symptoms including headache, changes in mental status or personality, lethargy and irritability. Weight gain results from the retention of fluid (Pillai, Unnikrishnan & Pavithran, 2011). Usually no oedema is present because water is distributed between the intracellular and extracellular spaces.

Treatment addresses the low serum sodium and intracellular swelling. Nursing care involves teaching the person about restricting fluids to 500 to 1000 mL/day. Fluid restriction continues for 3 to 10 days until the source of excessive ADH secretion is addressed. Demeclocycline (Declomycin) is a tetracycline antibiotic with the unique property of creating excessive urine flow. It is used as a treatment for SIADH (Lehne, 2012). In severe or chronic cases, hypertonic saline, loop diuretics with increased salt intake, urea or mannitol may also be utilised to prevent circulatory overload (Thomas & Batuman, 2012).

Diabetes insipidus

Diabetes insipidus is the result of ADH insufficiency. The two types are as follows:

1. *Neurogenic diabetes insipidus* can either result from a disruption of the hypothalamus and pituitary gland (as from trauma, irradiation or cranial surgery) or be idiopathic.
2. *Nephrogenic diabetes insipidus* is a disorder in which the renal tubules are not sensitive to ADH. This may be congenital in origin or the result of renal failure.

A deficit of ADH causes excretion of large amounts of dilute urine (polyuria), in some instances as much as 12 L/day. The person has extreme thirst and drinks large volumes of water (polydipsia). If unable to replace the water loss, the person becomes dehydrated and hypernatraemic. Even though hyperosmolarity is present, the urine is diluted and has a low specific gravity.

If this disorder is caused by cerebral injury, symptoms commonly appear 3 to 6 days after the initial injury and last for 7 to 10 days. If the increased intracranial pressure is relieved, symptoms of diabetes insipidus usually disappear. However, diabetes insipidus may also be a chronic illness requiring lifelong treatment and care. See Table 18.5 for a comparison of posterior pituitary gland disorders.

INTERPROFESSIONAL CARE

SIADH is treated by correcting underlying causes, treating the hyponatraemia with intravenous hypertonic saline and restricting oral fluid intake to less than 800 mL/day.

Diabetes insipidus is also treated by correcting underlying causes, if possible. Other medical interventions include administering intravenous hypotonic fluids, increasing oral fluids and

TABLE 18.5 Comparison of posterior pituitary gland disorders

SIADH	DIABETES INSIPIDUS
Excessive ADH	Deficient ADH
Fluid volume excess	Fluid volume deficit
Restrict fluid intake	Encourage fluid intake
Demeclocycline (Declomycin) (oral agent) causes excessive urination	Desmopressin (DDAVP) (nasal spray) causes increased water reabsorption

replacing ADH hormone. Desmopressin acetate, administered intranasally, orally or parenterally, is the treatment of choice (McPhee et al., 2008).

Nursing care

Nursing care for the person with SIADH and diabetes insipidus focuses on the person's problems with fluid and electrolyte balance, as discussed in Chapter 9.

CHAPTER HIGHLIGHTS

- Hormones regulate growth, development and metabolism. Homeostasis is dependent on a balanced level of each type of hormone. Hormones not only affect organ function; they also interact and, when excesses or deficits occur, signs and symptoms are manifested.
- The pituitary gland, in conjunction with the hypothalamus, is the master gland of the body. Fifteen hormones and regulatory factors are synthesised in the anterior pituitary and hypothalamus; many are trophic hormones that stimulate the release of other hormones.
- Thyroid disorders are the most common endocrine disorders. Occurring mainly among women, these diseases change body image and impose challenges to energy levels, creating fatigue and exhaustion.
- Diagnostic tests and therapies are available to identify and treat thyroid disorders. Surgery, radiation therapy and medications support good quality of life, but the medications must be used throughout the lifetime.
- The parathyroid glands, which are located on thyroid tissues, provide parathyroid hormones that are essential for the maintenance of serum calcium, which is vital for cardiac function, bone stability, nerve conduction and muscle contraction.
- The adrenal glands regulate energy and fluid balance through corticosteroids and mineralocorticoids. Cushing's and Addison's diseases are polar opposites and treatment eliminates the signs and symptoms of one and creates the manifestations of the other. People with these disorders require education until they fully grasp the significance of the condition and the importance of adhering to the treatment plan.

CONCEPT CHECK

1 Graves' disease, the most common cause of hyperthyroidism, is categorised as what type of disorder?
1 autoimmune
2 infectious
3 allergic
4 genetic

2 What principle supports the treatment of hyperthyroidism with radioactive iodine?
1 Radioactive iodine reduces the vascularity of the thyroid gland.
2 Doses of radioactive iodine are too small to be hazardous to other body parts.
3 The thyroid gland takes up iodine in any form.
4 Irradiation of the thyroid gland decreases the risk of hypothyroidism.

3 You assess a person with newly diagnosed hypothyroidism as having an enlarged thyroid gland (goitre). What physiological process causes this enlargement?
1 an excess of TH stimulates thyroid follicles
2 an increased dietary iodine intake
3 a compensatory effort to produce more TH
4 tissue hypertrophy in response to increased TH

4 Mrs Jonah has taken cortisone for her rheumatoid arthritis for several years. What endocrine disorder is she most at risk of developing?
1 hyperthyroidism
2 hypothyroidism
3 acromegaly
4 Cushing's syndrome

5 Which statement illustrates that the person with Addison's disease understands your teaching?
1 'I will be sure to stop taking my medications when I have an infection.'
2 'I have purchased an emergency kit and keep it with me all the time.'
3 'I know I should never alter my dose of medications.'
4 'I wonder why I look suntanned all the time.'

6 Signs and symptoms of hyponatraemia found in SIADH include:
1 weight loss
2 irritability
3 hyperkalaemia
4 constipation

7 A home health nurse is caring for a person with hyperparathyroidism and osteoporosis. Which nursing diagnosis has priority with this person?
1 *Risk of fear*
2 *Risk of injury*
3 *Risk of isolation*
4 *Risk of chronic low self-esteem*

8 A nurse is monitoring a person for signs of hypocalcaemia. Which of the following is a sign of hypocalcaemia?
1 oliguria
2 positive Trousseau sign
3 diminished bowel sounds
4 hyperactive deep tendon reflexes

9 A person with increased ACTH levels and Addison's disease is likely to manifest:
1 tremor
2 hair loss
3 gingival hyperplasia
4 dermal hyperpigmentation

10 People treated with glucocorticoids are at risk of Addisonian crisis due to:
1 rapid withdrawal of glucocorticoids
2 excessive ACTH
3 sodium retention
4 hypokalaemia

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CHAPTER 19

NURSING CARE OF PEOPLE WITH DIABETES MELLITUS

TRACY PARRISH

KEY TERMS

dawn phenomenon 581
diabetes mellitus (DM) 561
diabetic ketoacidosis (DKA) 581
diabetic nephropathy 588
diabetic neuropathies 588
diabetic retinopathy 587
endogenous insulin 564
exogenous insulin 564
gluconeogenesis 562
glucosuria 564
glycogenolysis 562
hyperglycaemia 563
hyperosmolar hyperglycaemic state (HHS) 581
hypoglycaemia 586
insulin 562
insulin reaction 586
ketonuria 567
ketosis 563
lipoatrophy 575
lipodystrophy 575
microalbuminuria 588
polydipsia 564
polyphagia 564
polyuria 564
Somogyi phenomenon 581
type 1 DM 561
type 2 DM 561

LEARNING OUTCOMES

- Discuss the chronic disease diabetes mellitus, identifying the prevalence and incidence of the disease in Australia.
- Explain the pathophysiology, risk factors, manifestations and complications of type 1 and type 2 diabetes mellitus.
- Discuss the nursing implications for insulin and oral hypoglycaemic agents used to treat people with diabetes mellitus.
- Compare and contrast the manifestations and interprofessional care of hyperglycaemia, hypoglycaemia, diabetic ketoacidosis and hyperosmolar hyperglycaemic state.
- Identify and explain the chronic complications associated with the disease diabetes mellitus.

CLINICAL COMPETENCIES

- Assess blood glucose levels and patterns of hyperglycaemia and hypoglycaemia in people with diabetes mellitus.
- Recognise the importance of early diagnosis and control of blood glucose to prevent complications.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with type 1 and type 2 diabetes mellitus.
- Administer oral and injectable medications used to treat type 1 and type 2 diabetes mellitus knowledgeably and safely.
- Provide skilled care to people with diabetic ketoacidosis and hyperosmolar hyperglycaemic states.
- Integrate interprofessional care into care of people with type 1 and type 2 diabetes mellitus, especially foot and eye care.
- Provide appropriate teaching to facilitate blood glucose monitoring, administration of oral and injectable hypoglycaemic medications, diabetic diet, appropriate exercise and foot care.
- Revise plan of care as needed to provide effective interventions to promote, maintain or restore normal glucose levels.
- Teach the relationship of hygiene, neuropathy and impaired microcirculation to infection; teach the principles and procedures of effective foot care.
- Assess the person's ability to read markings on syringes and to identify correct insulin and hypoglycaemics.
- Relate insulin (endogenous and exogenous), dietary intake and exercise to control of blood glucose.

DIABETES MELLITUS

Diabetes mellitus (DM) is a common chronic disease of adults requiring continuing medical supervision and self-care education. However, depending on the type of diabetes and the age of the person, needs and nursing care may vary greatly. Consider the following examples:

- Cheryl Draheim is a 45-year-old school teacher. She developed diabetes at age 34 after a car accident caused severe pancreatic injuries. Cheryl has always been very careful about taking her insulin, following her diet and exercising regularly. However, she is beginning to notice that her vision is getting worse and that she is having increasing pain in her legs, especially after standing for long periods of time.
- Tom Chang is 53 years old. Early in his forties, Tom was diagnosed with type 2 diabetes. Although he was taught about the disease and the importance of taking his oral medications, following his diet plan and getting exercise, he rarely did more than take the medication. Five years ago, he was hospitalised for hyperglycaemia and started taking insulin. Last year Tom had a stroke, leaving him unable to walk. He has now been admitted to the hospital for treatment of gangrene of the big toe on his left foot.
- Grace Staples is an independent 82-year-old woman who lives alone and happily takes care of her two cats. She is slightly overweight. Last year, during Grace's annual eye examination, eye changes typical for diabetes were found. She was referred to her family doctor, who diagnosed type 2 diabetes and started her on oral medications. Grace sticks to her diet, walks a kilometre every day and plans to live to be 100.

As illustrated in these examples, diabetes mellitus is not a single disorder but a group of chronic disorders of the endocrine pancreas, all categorised under a broad diagnostic label. The condition is characterised by inappropriate hyperglycaemia caused by a relative or absolute deficiency of insulin or by a cellular resistance to the action of insulin. Of the several classifications of diabetes, this chapter will focus on the two main types: type 1 and type 2. **Type 1 DM** is the result of pancreatic islet cell destruction and a total deficit of circulating insulin; **type 2 DM** results from insulin resistance with a defect in compensatory insulin secretion.

Diabetes mellitus has been recognised as a disease for centuries. *Diabetes* derives from a Greek word meaning 'to siphon', referring to the increased output of urine. *Mellitus* derives from a Latin word meaning 'sweet'. The two words together identify the disease as an outpouring of sweet urine. It was not until 1921 that techniques were developed for extracting insulin from pancreatic tissue and for measuring blood glucose. At the same time, researchers discovered that insulin, when injected, produces a dramatic drop in blood glucose. This meant that diabetes was no longer a terminal illness because hyperglycaemia could be controlled. Since that time, oral hypoglycaemic drugs, human insulin products, insulin pumps, home blood glucose monitoring and transplantation of the pancreas or of pancreatic islet or beta cells have advanced the treatment and care of people with diabetes.

People with DM face lifelong changes in lifestyle and health status. Nursing care is provided in many settings for the diagnosis and care of the disease and treatment of complications. A major role of the nurse is that of educator in both hospital and community settings.

INCIDENCE AND PREVALENCE

The prevalence of diabetes is increasing worldwide. Between 2011 and 2012, 4.2%—an estimated 999 000 Australians—were diagnosed with diabetes. Of these, around 119 000 people (11.9%) had type 1 and 848 000 (84.9%) had type 2 diabetes. Approximately 23 400 people were diagnosed with unknown type of diabetes. This rate has risen from 1.5% in 1989 (Australian Institute of Health and Welfare (AIHW), 2011). In relation to total Australian health costs, the 4.2% of people who have diagnosed diabetes account for 12% of total health costs. Type 2 diabetes costs Australia $3 billion a year. The cost of diabetes to the community for a person with no complications is $9625 a year, whereas for a person with complications, the cost to the community is $15 850 (AIHW, 2011; Baker IDI Heart and Diabetes Institute, 2006; Vision Australia Foundation, 2009.)

Globally, 171 million people had diabetes in the year 2000, with an estimated rise to 366 million by the year 2030 (World Health Organization (WHO), 2012).

Statistics (Australian Bureau of Statistics (ABS), 2011) reveal that diabetes is the sixth-highest cause of death by disease in Australia. People with diabetes are twice as likely to have cardiovascular disease as well as earlier onset of cardiovascular conditions. Between 2007 and 2008, diabetic people aged less than 45 years were five times as likely to have high cholesterol and 15 times as likely to have hypertension compared with people without diabetes. Furthermore they were more than twice as likely to have mental or behavioural problems as the general population (ABS, 2011).

According to the AIHW (2011), between 2007 and 2008, 4.3% of people born overseas living in Australia had diabetes compared with 4% of people born in Australia for the same period, which shows a similar incidence between these two groups.

OVERVIEW OF ENDOCRINE PANCREATIC HORMONES AND GLUCOSE HOMEOSTASIS

Hormones

The endocrine pancreas produces hormones necessary for the metabolism and cellular utilisation of carbohydrates, proteins and fats. The cells that produce these hormones are clustered in

groups of cells called the islets of Langerhans or pancreatic islets. These islets have three different types of cells:

1. Alpha cells produce the hormone *glucagon*, which stimulates the breakdown of glycogen in the liver, the formation of carbohydrates in the liver, and the breakdown of lipids in both the liver and adipose tissue. The primary function of glucagon is to decrease glucose oxidation and to increase blood glucose levels. Through **glycogenolysis** (the breakdown of liver glycogen) and **gluconeogenesis** (the formation of glucose from fats and proteins), glucagon prevents blood glucose from decreasing below a certain level when the body is fasting or in between meals. The action of glucagon is initiated in most people when blood glucose falls below about 3.9 mmol/L.
2. Beta cells secrete the hormone **insulin**, which facilitates the movement of glucose across cell membranes into cells, decreasing blood glucose levels. Insulin prevents the excessive breakdown of glycogen in the liver and in muscle, facilitates lipid formation while inhibiting the breakdown of stored fats and helps move amino acids into cells for protein synthesis. After secretion by the beta cells, insulin enters the portal circulation, travels directly to the liver and is then released into the general circulation. Circulating insulin is rapidly bound to receptor sites on peripheral tissues (especially muscle and fat cells) or is destroyed by the liver or kidneys. Insulin release is regulated by blood glucose; it increases when blood glucose levels increase and it decreases when blood glucose levels decrease. When a person eats food, insulin levels begin to rise in minutes, peak in 30 to 60 minutes and return to baseline in 2 to 3 hours.
3. Delta cells produce *somatostatin*, which is believed to be a neurotransmitter that inhibits the production of both glucagon and insulin.

Blood glucose homeostasis

All body tissues and organs require a constant supply of glucose; however, not all tissues require insulin for glucose uptake. The brain, liver, intestines and renal tubules do not require insulin to transfer glucose into their cells. Skeletal muscle, cardiac muscle and adipose tissue do require insulin for glucose movement into the cells.

Normal blood glucose is maintained in healthy people primarily through the actions of insulin and glucagon. Increased blood glucose levels, amino acids and fatty acids stimulate pancreatic beta cells to produce insulin. As cells of cardiac muscle, skeletal muscle and adipose tissue take up glucose, plasma levels of nutrients decrease, suppressing the stimulus to produce insulin. If blood glucose falls, glucagon is released to raise hepatic glucose output, raising glucose levels. Adrenaline, growth hormone, thyroxine and glucocorticoids (often referred to as glucose counter-regulatory hormones) also stimulate an increase in glucose in times of hypoglycaemia, stress, growth or increased metabolic demand. The regulation of blood glucose levels by insulin and glucagon is illustrated in Figure 19.1.

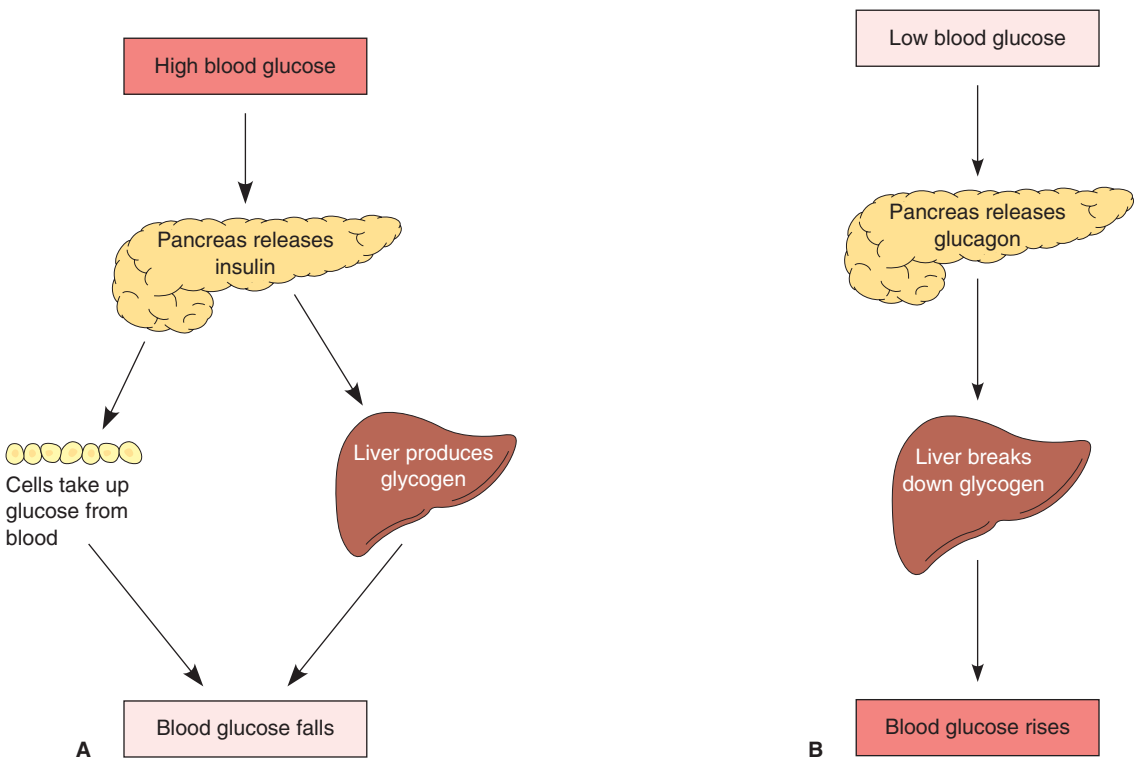


FIGURE 19.1 ■ Regulation (homeostasis) of blood glucose levels by insulin and glucagon. *A*, High blood glucose is lowered by insulin release. *B*, Low blood glucose is raised by glucagon release

PATHOPHYSIOLOGY OF DIABETES

DM is a group of metabolic diseases characterised by hyperglycaemia resulting from defects in the secretion of insulin, the action of insulin or both. There are four main types of DM. Type 1 diabetes (10–15% of all diagnosed cases) was formerly called juvenile-onset diabetes or insulin-dependent diabetes mellitus (IDDM). Type 2 diabetes (85–90% of diagnosed cases) was formerly labelled non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes. The other main types are gestational diabetes (3–8% of all pregnancies); women who have had gestational diabetes are at increased risk of developing type 2 diabetes (Australian Diabetes Council, 2012). A new category called 'impaired fasting glucose' has been introduced. This category is analogous to 'impaired glucose tolerance' in indicating a metabolic stage between normal glucose homeostasis and diabetes. These states are risk factors for the development of diabetes and cardiovascular disease. Impaired fasting glucose is defined by a fasting glucose concentration of between 6.1 and 6.9 mmol/L (diabetesvic.org.au). The classification and characteristics of the types of diabetes are described in Table 19.1.

Type 1 diabetes

Type 1 diabetes most often occurs in childhood and adolescence, but it may occur at any age, even in the eighties and nineties. This disorder is characterised by **hyperglycaemia** (elevated blood glucose levels), a breakdown of body fats and proteins, and the development of **ketosis** (an accumulation of ketone bodies produced during the oxidation of fatty acids). Type 1 DM is the result of the destruction of the beta cells of the islets of Langerhans in the pancreas, the only cells in the body that make insulin. When beta cells are destroyed, insulin

TABLE 19.1 Classification and characteristics of diabetes

	CLASSIFICATION	CHARACTERISTICS
Type 1 diabetes	Immune mediated	Beta cells are destroyed, usually leading to absolute insulin deficiency. Markers to the immune destruction of the beta cells include islet cell autoantibodies (ICAs) and insulin autoantibodies (IAAs). The rate of beta-cell destruction is variable, usually more rapid in infants and children and slower in adults. Destruction of the beta cells has genetic predispositions and is also related to environmental factors as yet undefined.
	Idiopathic	Has no known aetiological causes. Need for insulin may be intermittent.
Type 2 diabetes	—	May range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance. There is no immune destruction of beta cells. Initially, and in some cases for the entire life, insulin is not necessary. Most people with this form are obese or have an increased amount of abdominal fat. Risks for development include increasing age, obesity and a sedentary lifestyle. Occurs more frequently in women who have had gestational diabetes and in people with lipid disorders or hypertension. There is a strong genetic predisposition.
Other specific types	Genetic defects of beta cells	Hyperglycaemia occurs at an early age (usually before age 25). This type is referred to as maturity-onset diabetes of the young (MODY).
	Genetic defects in insulin action	Are genetically determined. Dysfunctions may range from hyperinsulinaemia to severe diabetes.
	Diseases of the exocrine pancreas	Acquired processes causing diabetes include pancreatitis, trauma, infection, pancreatectomy and pancreatic cancer. Severe forms of cystic fibrosis and haemochromatosis may also damage beta cells and impair insulin secretion.
	Endocrine disorders	Excess amount of hormones (e.g. growth hormone, cortisol, glucagon and adrenaline) impair insulin secretion, resulting in diabetes in people with Cushing's syndrome, acromegaly and pheochromocytoma.
	Drug- or chemical-induced	Many drugs impair insulin secretion, precipitating diabetes in people with predisposing insulin resistance. Examples are nicotinic acid, glucocorticoids, thyroid hormone, thiazides and Dilantin.
	Infections	Certain viruses may cause beta-cell destruction, including congenital measles, cytomegalovirus, adenovirus and mumps.
Gestational diabetes mellitus (GDM)	—	Any degree of glucose intolerance with onset or first recognition during pregnancy.
Impaired fasting glucose/impaired glucose tolerance	Metabolic stage between normal glucose homeostasis and diabetes	These states are risk factors for the development of diabetes and cardiovascular disease. Impaired fasting glucose is defined by a fasting glucose concentration of between 6.1 and 6.9 mmol/L (Australian Diabetes Council, 2013).

is no longer produced. Although type 1 DM may be classified as either an autoimmune or idiopathic disorder, 90% of the cases are immune mediated. The disorder begins with insulinitis, a chronic inflammatory process that occurs in response to the autoimmune destruction of islet cells. This process slowly destroys beta-cell production of insulin, with the onset of hyperglycaemia occurring when 80–90% of beta-cell function is lost. This process usually occurs over a long preclinical period. It is believed that both alpha-cell and beta-cell functions are abnormal, with a lack of insulin and a relative excess of glucagon resulting in hyperglycaemia.

Risk factors

Genetic predisposition plays a role in the development of type 1 DM. Although the risk in the general population ranges from 1:400 to 1:1000, the child of a person with diabetes has a 1:20 to 1:50 risk. Genetic markers that determine immune responses—specifically, DR3 and DR4 antigens on chromosome 6 of the human leucocyte antigen (HLA) system—have been found in 95% of people diagnosed with type 1 DM. (HLAs are cell surface proteins, controlled by genes on chromosome 6.) Although the presence of these markers does not guarantee that the person will develop type 1 DM, they do indicate increased susceptibility (Porth & Matfin, 2009).

Environmental factors are believed to trigger the development of type 1 DM. The trigger can be a viral infection (mumps, rubella or coxsackievirus B4) or a chemical toxin, such as those found in smoked and cured meats. As a result of exposure to the virus or chemical, an abnormal autoimmune response occurs in which antibodies respond to normal islet beta cells as though they were foreign substances, destroying them. The manifestations of type 1 DM appear when approximately 90% of the beta cells are destroyed. However, manifestations may appear at any time during the loss of beta cells if an acute illness or stress increases the demand for insulin beyond the reserves of the damaged cells. The actual cause and exact sequence are not completely understood, but research continues to identify the genetic markers of this disorder and to investigate ways of altering the immune response to prevent or cure type 1 DM.

Manifestations

The manifestations of type 1 DM are the result of a lack of insulin to transport glucose across the cell membrane into the cells (see Figure 19.2).

Glucose molecules accumulate in the circulating blood, resulting in hyperglycaemia. Hyperglycaemia causes serum hyperosmolarity, drawing water from the intracellular spaces into the general circulation. The increased blood volume increases renal blood flow and the hyperglycaemia acts as an osmotic diuretic. The resulting osmotic diuresis increases urine output. This condition is called **polyuria**. When the blood glucose level exceeds the renal threshold for glucose—usually about 10 mmol/L—glucose is excreted in the urine, a condition called **glucosuria**. The decrease in intracellular volume and the increased urinary output cause dehydration. The mouth becomes dry and thirst sensors are activated, causing the person to drink increased amounts of fluid (**polydipsia**).

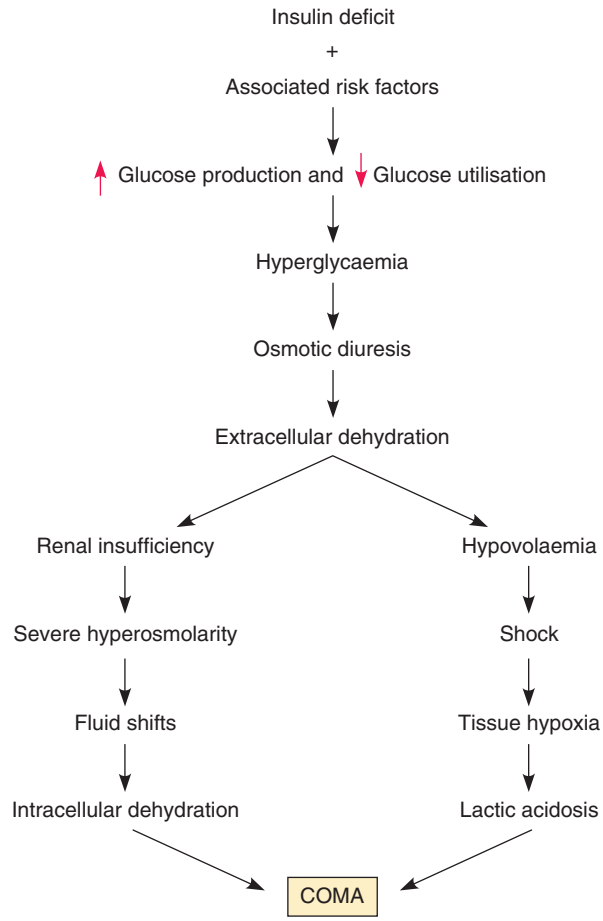


FIGURE 19.2 ■ Pathophysiological results of type 1 DM

Because glucose cannot enter the cell without insulin, energy production decreases. This decrease in energy stimulates hunger and the person eats more food (**polyphagia**). Despite increased food intake, the person loses weight as the body loses water and breaks down proteins and fats in an attempt to restore energy sources. Malaise and fatigue accompany the decrease in energy. Blurred vision is also common, resulting from osmotic effects that cause swelling of the lenses of the eyes.

Thus, the classic manifestations are polyuria, polydipsia and polyphagia, accompanied by weight loss, malaise and fatigue. Depending on the degree of insulin lack, the manifestations vary from slight to severe. People with type 1 DM require **exogenous insulin** to maintain life.

Type 2 diabetes

Type 2 DM is a condition of fasting hyperglycaemia that occurs despite the availability of **endogenous insulin**. Type 2 DM can occur at any age, but it is usually seen in middle-aged and older people. Heredity plays a role in its transmission. The level of insulin produced varies in type 2 DM and despite its availability, its function is impaired by insulin resistance. Insulin resistance forces the pancreas to work harder and produce more insulin, but when demand exceeds supply, DM results (Diabetes

Australia, Victoria, 2012; Saudek & Margolis, 2005). Whatever the cause, there is sufficient insulin production to prevent the breakdown of fats with resultant ketosis; thus, type 2 DM is characterised as a non-ketotic form of diabetes. However, the amount of insulin available is not sufficient to lower blood glucose levels through the uptake of glucose by muscle and fat cells. A major factor in the development of type 2 DM is cellular resistance to the effect of insulin. This resistance is increased by obesity, inactivity, illnesses, medications and increasing age. In obesity, insulin has a decreased ability to influence glucose metabolism and uptake by the liver, skeletal muscles and adipose tissue. Although the exact reason for this is not clear, it is known that weight loss and exercise may improve the mechanism responsible for insulin receptor binding or post-receptor activity (McCance & Huether, 2009). Hyperglycaemia increases gradually and may exist over time before diagnosis. According to the International Diabetes Federation (2009), diabetes remains seriously under-reported due to many people not being aware they have diabetes type 2. Many people do not seek help until they have developed complications, usually many years after diabetes has begun (Shaw, 2012). Treatment usually begins with prescriptions for weight loss and increased activity. If these changes can be sustained, no further treatment will be necessary for many individuals. Hypoglycaemic medications are begun when lifestyle changes are insufficient. Often, a combination of insulin and hypoglycaemic medication is used to achieve the best glycaemic control in the person with type 2 DM.

Risk factors

The main risk factors for type 2 DM are as follows:

- History of diabetes in parents or siblings. Although there is no identified HLA linkage, the children of a person with type 2 DM have a 15% chance of developing type 2 DM and a 30% risk of developing glucose intolerance (the inability to metabolise carbohydrate normally).
- Obesity, defined as being at least 20% over desired body weight or having a body mass index of at least 27 kg/m^2. Obesity, especially of the upper body, decreases the number of available insulin receptor sites in cells of skeletal muscles and adipose tissues, a process called *peripheral insulin resistance*. In addition, obesity impairs the ability of the beta cells to release insulin in response to increasing glucose levels.
- Physical inactivity.
- Race/ethnicity (see the 'Focus on cultural diversity' box below).
- In women, a history of gestational DM, polycystic ovary syndrome or delivering a baby weighing more than 4.1 kg.
- Hypertension ($\geq$ 130/85 in adults), HDL cholesterol of $\geq$ 0.9 mmol/L, and/or a triglyceride level of $\geq$ 2.8 mmol/L.
- Metabolic syndrome is a connection between obesity and the development of diabetes mellitus, and is thought to link cardiovascular disease with insulin resistance (Craft et al., 2011). Hypertension, abdominal obesity, dyslipidaemia, elevated C-reactive protein and fasting blood glucose greater than 6.1 mmol/L increase the risk of DM, coronary heart disease and stroke (Diabetes Australia, 2012; Porth & Matfin 2009).

Manifestations

The person with type 2 DM experiences a slow onset of manifestations and is often unaware of the disease until seeking healthcare for some other problem. The hyperglycaemia in type 2 is usually not as severe as in type 1, but similar symptoms occur, especially polyuria and polydipsia. Polyphagia is not often seen and weight loss is uncommon. Other manifestations are also the result of hyperglycaemia: blurred vision,

FOCUS ON CULTURAL DIVERSITY Diabetes in Indigenous Australians

Type 2 diabetes represents a serious public health problem for Indigenous Australians, occurring at a much higher rate than in the non-Indigenous population, and with a much earlier age of onset of the disease and its micro- and macrovascular complications. It is likely that diabetes is an important contributor to the considerably higher circulatory disease mortality rate among Indigenous Australians at young ages (9–10 times higher in Indigenous men aged 25–44 years and 12–13 times higher in Indigenous women aged 35–54 years). Thus, diabetes imposes significant financial and human costs on Australian society, which are disproportionately borne by Indigenous individuals, families and communities.

Indigenous Australians have the fourth-highest rate of type 2 diabetes (non-insulin-dependent diabetes mellitus, or NIDDM) in the world.

For the period 2003–2007, Indigenous Australians were seven times more likely than non-Indigenous Australians to have diabetes recorded as the cause of death on their death certificate (AIHW, 2011). The incidence of gestational diabetes (diabetes in pregnancy) is also two to three times higher among Indigenous Australian women than in the general Australian population.

As reported in the *National Aboriginal and Torres Strait Islander Health Survey 2004–2005*, 29 874, or 6.3% of, Indigenous Australians self-reported diabetes (ABS, 2012; AIHW, 2011). Craig et al. (2007) highlighted that data analysed from the Australasian Paediatric Endocrine Group NSW Diabetes Register in 2007 showed that type 2 diabetes accounts for 11% of new diabetes cases among 10–18 year olds. The incidence of diabetes in Indigenous children is about six times higher than that in non-Indigenous children (O'Dea, Rowley & Brown, 2007).

For Torres Strait Islanders, there were significant increases in body mass index (BMI)—the major risk factor—between 1999 and 2005 and a very high 5-year incidence of diabetes (O'Dea et al., 2007).

fatigue, paraesthesia and skin infections. If available insulin decreases, especially in times of physical or emotional stress, the person with type 2 DM may develop diabetic ketoacidosis (DKA), but this occurrence is uncommon.

Diabetes in the older adult

Although most older adults with diabetes have type 2 DM, the improved survival rates for people with diabetes have resulted in an increased number of older adults with type 1. The picture is complicated by the fact that blood glucose levels increase with age, beginning in the fifties. For this reason, it is more difficult to diagnose diabetes in the older adult; conversely, the older adult may be mistakenly diagnosed with the disease simply for exhibiting essentially normal age-related changes in glucose. The relationship between normal increases in glucose levels and the presence of diabetes is not yet understood.

The normal physiological changes of ageing may mask manifestations of the onset of diabetes. Signs can be dismissed and be considered as part of 'getting older' (Diabetes Australia, 2012). Signs and symptoms of diabetes in elders may not include the classic symptoms of polyuria and thirst. Conditions such as orthostatic hypotension, periodontal disease, infections, stroke, gastric hypotony, impotence, neuropathy, confusion, lethargy, gradual weight gain, leg cramps and glaucoma should be considered potential indicators of diabetes (Diabetes Australia, 2012) and may also increase the potential for complications from the disease or its treatment. Table 19.2 presents common problems in the older adult that make the diagnosis and management of diabetes more difficult. The older adult with diabetes also has a longer recovery period after surgery or serious illness, often requiring insulin to maintain blood glucose levels. The benefits and risks of treatment to maintain glycaemic control, as well as blood pressure and lipid management, must be carefully balanced.

INTERPROFESSIONAL CARE

Diagnosis

Diagnostic tests are conducted for screening purposes to diagnose diabetes, and ongoing laboratory tests are conducted to evaluate the effectiveness of diabetic management. Definitions of normal blood glucose levels vary in clinical practice, depending on the laboratory that performs the assay.

TABLE 19.2 Implications for nursing care of the older adult with diabetes

HEALTH PROBLEM/ COMPLICATION	IMPLICATIONS FOR NURSING CARE
Urinary incontinence	Polyuria, a classic manifestation of diabetes, often is ignored. This problem also often leads to social isolation.
Increased thirst	Polydipsia, a classic manifestation of diabetes, often is ignored. This further increases the risk of dehydration and electrolyte imbalances.
Decreased hunger and weight loss	Polyphagia, a classic manifestation of diabetes, often is ignored. The ageing process, medications, depression or lack of socialisation may decrease hunger. Weight loss may be gradual and go unnoticed.
Fatigue	Fatigue is a common symptom of diabetes but may be blamed on increased age.
Hypoglycaemia	The older adult may have either very mild manifestations or none at all. As a result, hypoglycaemia is often ignored until it causes serious effects.
Peripheral neuropathy	Manifestations may be thought to be due to arthritis, and over-the-counter drugs often are used to self-medicate. The risk of falls increases, as does the risk of gangrene and amputation.
Peripheral vascular disease	May go undetected if the person does not get enough exercise to cause claudication. May also impair abilities to climb stairs and walk.
Diabetic retinopathy	May be undetected if the person has cataracts. The diabetic person also has an increased incidence of cataracts and glaucoma. Deficits in vision threaten independence, mobility and social interactions. Yellowing of the lens with age makes it difficult to read coloured test strips; numerical meters are preferable. Filling insulin syringes may be impossible for the person with macular degeneration or other causes of visual loss.
Hypertension	Treatment with diuretics may further impair glucose tolerance and result in electrolyte imbalances.
Arthritis	Older adults may believe the pain from arthritis to be more important than the diabetes management. Also, depression from chronic pain as well as inactivity and loss of appetite may interfere with diabetes self-care.
Parkinson's disease	The tremors and rigidity of this disease make self-care involving fine and gross motor skills difficult or impossible.
Medications	Older adults commonly take more than one type of medication and are at increased risk of problems relating to drug interactions.

Sources: Adapted from D. Haire-Joshu (ed.), *Management of diabetes mellitus: Perspectives of care across the lifespan* (2nd ed.), pp. 755–830. St Louis, MO: Mosby; *Gerontological nursing* (6th ed.) by C. Eliopoulos (2005). Philadelphia: Lippincott Williams & Wilkins.

DIAGNOSTIC SCREENING According to the Australian Diabetes Society (ADS) (Twigg et al., 2007) and the National Health and Medical Research Council (NHMRC, 2009), diabetes mellitus is diagnosed if any of the following criteria are satisfied:

1. Symptoms of diabetes (polyuria, polydipsia, unexplained weight loss) and random plasma glucose ≥ 11.1 mmol/L.
2. Fasting plasma glucose ≥ 7.0 mmol/L.
3. Oral glucose tolerance test: 75 g glucose, 2-hour plasma glucose result ≥ 11.1 mmol/L.

In the absence of unequivocal symptoms, or if the glucose level is borderline, diagnosis by criterion 1 or 2 should be confirmed by repeat testing on a separate day.

Impaired glucose tolerance is diagnosed if a plasma glucose result 2 hours after a 75 g glucose load is between 7.8 and 11.0 mmol/L (inclusive).

Impaired fasting glucose is diagnosed if a fasting plasma glucose result is between 6.1 and 6.9 mmol/L (inclusive).

With regard to testing:

- People must fast for at least 8 hours to apply the fasting criteria.
- The criteria apply to people who are otherwise well at the time of diagnosis. Physiological stress from infection, trauma or other illness, or the effect of some drugs, may cause false positive results. In general, the criteria should be applied to ambulant outpatients only.
- Samples for plasma glucose should be separated from red cells as soon as possible after collection. When there may be a delay greater than 1 hour prior to centrifugation, samples must be collected into fluoride-oxalate tubes (grey-top). Even with these tubes a fall in plasma glucose of 0.1–0.2 mmol/L may occur.
- The criteria apply to venous plasma samples. Capillary or whole blood samples may require alternative decision points depending on the analysis method used.
- Haemoglobin A1c and home blood glucose meters are not appropriate methods for the diagnosis of diabetes.
- Fasting plasma glucose less than 5.5 mmol/L indicates a low probability of diabetes.

Who should be tested? Testing should be performed on anyone with symptoms suggestive of diabetes. Additionally, fasting plasma glucose should be performed as a screening test every 5 years in asymptomatic people from the following groups:

- people who are obese, hypertensive or have a first-degree relative with type 2 diabetes (screen from age 50)
- everyone with impaired glucose tolerance or cardiovascular disease
- women with a history of gestational diabetes or with polycystic ovary disease and obesity (screen from time of diagnosis)
- Indigenous Australians and other high-risk groups, including people from the Pacific Islands and the Indian subcontinent, and people of Chinese origin (screen from age 35)
- everyone over 65.

Interpretation of screening test If the fasting plasma glucose is:

- < 5.5 mmol/L—diabetes is unlikely; offer lifestyle advice and re-test in 5 years
- 5.5–6.9 mmol/L—diabetes is uncertain; glucose tolerance testing or more frequent fasting glucose indicated
- ≥ 7.0 mmol/L—diabetes likely; confirm with repeat fasting glucose on a separate occasion.

(Health Options Australia, 2012)

DIAGNOSTIC TESTS TO MONITOR DIABETES MANAGEMENT The following diagnostic tests may be used to monitor diabetes management:

- *Fasting plasma glucose (FPG).* This test is often ordered, especially if the person is experiencing symptoms of hypoglycaemia or hyperglycaemia. In most people, the normal range is 3.9 to 6.1 mmol/L.
- *Glycated haemoglobin (c) (HbA1c or A1c).* This test determines the average blood glucose level over approximately the previous 2 to 3 months. When glucose is elevated or control of glucose is erratic, glucose attaches to the haemoglobin molecule and remains attached for the life of the haemoglobin, which is about 120 days. The American Diabetes Association (2006) and Diabetes Australia (2008) recommend that HbA1c or A1c be performed at the initial assessment and then every 3 to 6 months. The goal for most people with diabetes is an HbA1c of ≤ 7%, although this may need to be adjusted according to individuals.
- *Urine glucose and ketone levels.* These are not as accurate in monitoring changes in blood glucose as blood levels. The presence of glucose in the urine indicates hyperglycaemia. Most people have a renal threshold for glucose of 10 mmol/L; that is, when the blood glucose exceeds 10 mmol/L, glucose is not reabsorbed by the kidney and spills over into the urine. This number varies highly, however. **Ketonuria** (the presence of ketones in the urine) occurs with the breakdown of fats and is an indicator of DKA; however, fat breakdown and ketonuria also occur in states of less-than-normal nutrition.
- *Urine test* for the presence of protein as albumin (*albuminuria*). If albuminuria is present, a 24-hour urine test for creatinine clearance is used to detect the early onset of nephropathy.
- *Serum cholesterol and triglyceride levels.* These are indicators of atherosclerosis and an increased risk of cardiovascular impairments. People with diabetes in general have similar rates of HDL or 'good' cholesterol to the general population but, on average, people with diabetes have higher levels of triglycerides and LDL or 'bad' cholesterol. Diabetes affects the balance between HDL and LDL in a number of ways (Diabetes Australia, 2008).
- *Serum electrolytes.* Levels are measured in people who have DKA or hyperosmolar hyperglycaemic state (HHS) to determine imbalances.

MONITORING BLOOD GLUCOSE There are two ways of monitoring blood glucose levels: self-blood glucose and serum HbA1c level. Self-blood glucose monitoring is a valuable tool for the person with diabetes and for the carer. It enables self-management and alerts carers and health professionals to possible issues and the need to change management. Self-blood glucose monitoring is a simple procedure which gives a result

within seconds. In Australia there are several types of blood glucose meters available for this purpose The HbA1c test is a serum blood test that shows an average blood glucose level over 10 to 12 weeks. (See 'Diagnostic tests to monitor diabetes management', above.)

URINE TESTING FOR KETONES AND GLUCOSE Urine testing is recommended to monitor hyperglycaemia and ketoacidosis in people with type 1 DM who have unexplained hyperglycaemia during illness or pregnancy. Ketones may be detected through urine testing and reflect the presence of DKA. (See 'Procedure 19.1: Testing urine for ketones and glucose'.)

Following is the equipment needed for self-blood glucose monitoring:

- Lancet device and lancets to perform a finger prick for obtaining a drop of blood.
- Test strips.
- A blood glucose meter appropriate for the person's individual needs. The manufacturer's instructions for use and maintenance of the meter must be followed carefully.
- Information on blood glucose monitoring and different blood glucose monitoring devices available in Australia can be found on the websites of Diabetes Australia (www.diabetesaustralia.com.au) or the Australian Diabetes Council (now Diabetes NSW, www.diabetesnsw.com.au).

The Australian Diabetes Educators Association (ADEA) (2010) in a position statement gives recommendations for the reliable and accurate use of blood glucose meters within the healthcare setting and in self-management of diabetes.

HAEMATOCRIT People with higher haematocrit values will usually test falsely low in blood glucose and people with lower haematocrit will test falsely high. Anaemia and sickle cell anaemia are two conditions that can affect haematocrit values.

OTHER SUBSTANCES Overdoses of many medications will cause inaccurate results. Meters and supplies vary in sensitivity to medications. Uric acid (a natural substance in the body that can be more concentrated in some people with diabetes), glutathione (an antioxidant also called *GSH*), ascorbic acid (vitamin C) and a variation in haematocrit are known to interfere (Ginsberg, 2009). Check the package insert for each meter to find what substances might affect its testing accuracy. Furthermore, strips sometimes give inaccurate readings if they have not been stored properly; therefore, it is relevant that the maintenance and storage of blood glucose testing equipment is followed as per instructions (Ginsberg, 2009).

USE OF CORRECT SUPPLIES AND SAMPLE VOLUME Be sure the test strips are compatible with the glucose meter and that they are not outdated and have not been exposed to air and humidity, which can alter strip sensitivity. Insufficient amounts of blood on the testing strip cause inaccurate results. Although a meter may indicate a sufficient amount of blood on the test strip, it is best to observe that the receptacle is full of capillary blood. Other possible reasons for inaccurate reading are wrong positioning of the strip in the meter, dirty hands prior to doing the test, a dirty meter, incorrect calibration, the meter is too cold or too hot, or it has a low or flat battery (Diabetes Australia, 2012).

Pharmacological treatment of diabetes mellitus

The pharmacological treatment for diabetes mellitus depends on the type of diabetes. People with type 1 DM must have insulin; those with type 2 DM are usually able to control glucose levels with an oral hypoglycaemic medication, but they may require insulin if control is inadequate.

INSULIN The person with type 1 DM requires a lifelong exogenous source of the insulin hormone to maintain life. Insulin is not a cure for diabetes; rather, it is a means of controlling hyperglycaemia. Insulin is also necessary in other situations, such as these:

- people with diabetes who are unable to control glucose levels with oral antidiabetic drugs and/or diet. Introduced when beta-cell function declines, insulin maintains glycaemic control and prevents complications (Diabetes Australia, 2012; Funnell, Kruger & Spencer, 2004)
- people with diabetes who are experiencing physical stress (such as an infection or surgery) or who are taking corticosteroids
- women with gestational diabetes who are unable to control glucose with diet
- people with DKA or HHS
- people who are receiving high-kilojoule tube feedings or parenteral nutrition.

Sources of insulin Preparations of insulin are derived from animal (pork pancreas) or synthesised in the laboratory from either an alteration of pork insulin or recombinant DNA technology, using strains of *Escherichia coli* to form a biosynthetic human insulin. Insulin analogues have been developed by modifying the amino acid sequence of the insulin molecule. Although different types are prescribed on an individualised basis, it is standard practice to prescribe human insulin.

PROCEDURE 19.1 Testing urine for ketones and glucose

TO TEST THE URINE FOR KETONES AND/OR GLUCOSE

1 Collect a small amount of urine.
2 Apply this to the test strip by dipping the strip in the urine sample.
3 Read desired test result at the specified time, by comparing the colour change on the test strip with the standard colour range for your brand of test strip. The reference colour chart is usually printed on the container.

Insulin preparations Insulin is classified according to how long it works in the body. There are five different types of insulin, ranging from short to long acting. Some insulins are clear in appearance, while others are cloudy.

Often people need varying amounts of both short- and longer-acting insulin. However, everyone is different and will respond differently to the insulin they take in the management of diabetes.

Types of insulin The five types of insulin are:

1. ultra-short-acting analogues
2. short-acting insulin
3. intermediate-acting insulin
4. long-acting analogue insulin
5. mixed insulin.

Ultra-short-acting analogue insulin Rapid-onset fast-acting insulin has a clear appearance. Its onset of work is within 5 to 10 minutes. Peak effect is 1 to 3 hours and its duration is 5 hours. When people use this type of insulin, they must eat immediately after they inject.

Ultra-short-acting analogue insulin types currently available in Australia are:

- NovoRapid (insulin aspart)
- Humalog (insulin lispro)
- Apidra (insulin glulisine).

Short-acting insulin Short-acting insulins always look clear. The onset is 30 minutes. Peak effect is 2.5 to 5 hours and duration is 8 hours. Short-acting insulin types currently available include:

- Actrapid (insulin neutral, regular)
- Humulin R (insulin neutral, regular).

Intermediate-acting insulin Intermediate-acting insulins appear cloudy. They have either protamine or zinc added to delay their action. These insulins need to be shaken before every use and do not need to be injected with a meal. The onset of work is 1.5 hours. Peak action is 4 to 12 hours. The duration is 12 to 16 up to 24 hours.

Intermediate-acting insulins currently available include:

- Protaphane (isophane)
- Humulin NPH (isophane NPH).

Long-acting analogue insulin Long-acting analogue insulins have a clear appearvvance and do not need to be injected with a meal. They have no pronounced peak action, which means the insulin is released into the bloodstream at a relatively constant rate. One injection can last 24 hours.

Long-acting analogue insulins available in Australia are:

- Levemir (insulin determir)
- Lanctus (insulin glargine).

Mixed insulin Mixed insulin always looks cloudy. It contains a pre-mixed combination of either a rapid-onset fast-acting or a short-acting insulin and intermediate-acting insulin. This makes it easier because two types of insulin can be given in one injection. If the insulin is '30/70', then it contains 30% of quick-acting and 70% of intermediate-acting insulin; '50/50' means 50% of each. Before injecting mixed or other cloudy insulin, the person must gently roll the vial or pen between the palms of their hands and/or rock it slowly to make sure the different strengths of insulin are evenly distributed.

The mixed insulins currently available include:

With ultra-short-acting insulin

- Humalog Mix 25 (25% insulin lispro, 75% lispro protamine suspension)
- Humalog Mix 50 (50% insulin lispro, 50% lispro protamine suspension)
- NovoMix 30 (30% insulin aspart, 70% aspart protamine suspension)

With rapid-acting insulin

- Humulin 30/70 (30% insulin neutral regular, 70% isophane)

Other mixed insulins

- Mixtard 30/70 (30% insulin neutral regular, 70% isophane)
- Mixtard 50/50 (50% insulin neutral regular, 50% isophane).

(Diabetes Australia, Victoria, 2012)

Insulin regimens The appropriate insulin dosage is individualised by achieving a balance between insulin, diet and exercise. While in the past there has been a tendency to try to minimise the number of injections per day, for most people with diabetes two or more injections each day are required, often a mixture of rapid-acting and intermediate-acting insulins. Increasingly more children and teenagers are being treated with multiple daily injections or insulin pumps. This is to try to more closely match the body's insulin needs, similar to the way the pancreas works in people without diabetes. Many children now start on multiple injections; or if started on two injections per day, soon evolve to three or four injections per day (Diabetes Australia, 2007). Timing of the injections depends on blood glucose levels, food consumption, exercise and types of insulin used. The objective is to avoid daytime hypoglycaemia while achieving adequate blood glucose control overnight.

Box 19.1 shows how a combination regimen of insulin three times per day and four times per day is intended to work.

Insulin administration Nursing implications for administering insulin are outlined in the 'Medication administration' box below and further discussion follows in the chapter. The considerations for administering insulin include routes of administration, syringe and needle selection, preparing the injection, sites of injection, mixing insulins and insulin regimens.

Routes of administration Only regular insulin is given by both subcutaneous and intravenous routes; all others are given only subcutaneously. If the intravenous route is not available, regular insulin may also be administered intramuscularly in an emergency situation.

Insulin injection devices There are many different devices available to inject insulin. The main choices include insulin syringes, insulin delivery pens and insulin pumps.

Insulin syringes. It is important to note that only syringes are to be used with insulin vials. Syringes available in Australia are 30 unit (0.3 mL), 50 unit (0.5 mL) and 100 unit (1.0 mL); the size of syringe will depend on the insulin dose. Needles on the syringes are available in different lengths and the syringes

BOX 19.1 Insulin regimens

Three times daily insulin injections

The diagram below shows how a combination is intended to work.

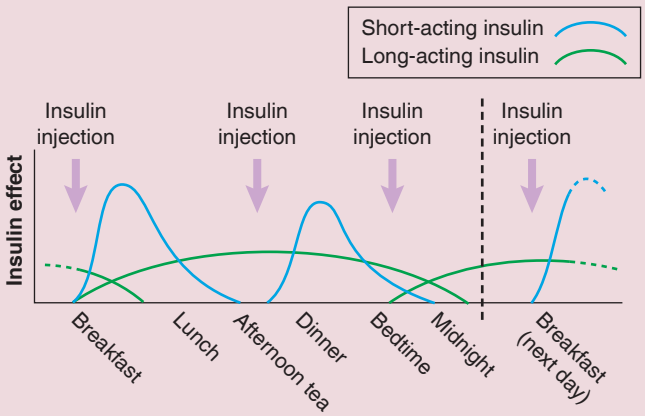


Figure showing an example of a 3 times per day injection pattern with injections at breakfast, afternoon tea and bed

In this routine people have:

- Before breakfast: rapid- or short-acting insulin (meal bolus) plus long-acting insulin (basal insulin)
- Before afternoon tea: rapid- or short-acting insulin (meal bolus)
- Before bed: long-acting insulin (basal insulin).

Four times daily injections (basal-bolus)

The diagram below shows how a combination is intended to work.

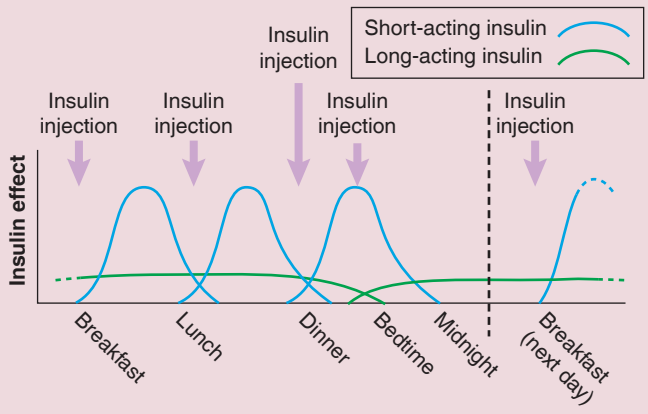


Figure showing a basal-bolus or MDI injection pattern

This routine is often referred to as a basal-bolus injection plan or multiple daily injections (MDI). Commonly about 40–50% of the insulin is given as long-acting insulin and the rest is divided up in the rapid- or short-acting doses. This offers very good flexibility for insulin adjustment and, when part of a comprehensive diabetes management plan, has been shown to have advantages for diabetes control. It is the most common starting insulin plan for older children, adolescents and adults. It is also commonly used in younger children, although they will need assistance with injections, particularly at school.

Source: G. R. Ambler & F. J. Cameron (eds) (2010). *Caring for diabetes in children and adolescents* (3rd ed.), pp. 24–25.

are one use only. Syringes are free for people registered with the National Diabetes Service Scheme (NDSS).

Insulin delivery devices. Many people find pen devices easier and more convenient than syringes. Pen devices come in all shapes and sizes and they can be durable (an insulin cartridge that fits into the pen is inserted and replaced when it is finished) or disposable (the pen device is prefilled with insulin and the whole device is disposed of when finished).

Pen needles vary in length and thickness. The higher the number, the finer the needle. It is recommended that the pen needle be changed with each injection. In Australia, pen needles are free for people registered with the NDSS.

Insulin pumps. The insulin pump is a small programmable device which holds a reservoir of insulin. The pump is programmed to deliver insulin into the body through thin plastic tubing known as the infusion set or giving set. It is worn outside the body in a pouch, on the belt or bra. The infusion set has a fine needle or flexible cannula that is inserted just below the skin (usually on the abdomen) where it stays in place for 2 to 3 days. Only short- or rapid-acting insulin can be used in the pump. Whenever food is eaten, the pump is manually programmed to deliver an amount of insulin into the body, similar to the way the pancreas does in people without diabetes. Between meals, a small and steady rate of insulin is delivered. The insulin pump isn't suitable for everyone. Its use must be discussed first with the diabetes healthcare team.

Storage of insulin Unopened insulin vials or pen cartridges should be kept on their side in the refrigerator (between 2ºC and 8ºC) and away from the freezer or freezing coils. Once opened, insulin should be kept below either 25ºC or 30ºC and thrown away after 30 days, even if there is some insulin left. Always check the expiry date and appearance of insulin before use. Insulin is destroyed when it has been exposed to direct sunlight, heat or allowed to freeze. In these cases, it should not be used.

Disposal of used syringes Used syringes, pen needles and lancets must be disposed of in a sharps container that meets the Australian safety standards, is puncture proof and has a secure lid. These containers are usually yellow in colour and are

MEDICATION ADMINISTRATION Insulin

NURSING RESPONSIBILITIES

- Discard vials of insulin 30 days after opening or where expiration date has passed.
- Refrigerate, but do not freeze, extra insulin vials not currently in use.
- Store insulin in a cool place and avoid exposure to temperature extremes or sunlight.
- Store compatible mixtures of insulin for no longer than 1 month at room temperature or 3 months at 2°C to 8°C.
- Discard any vials with discolouration, clumping, granules or solid deposits on the sides.
- If breakfast is delayed, also delay the administration of rapid-acting insulin.
- Monitor and maintain a record of blood glucose readings 30 minutes before each meal and bedtime (or as prescribed).
- Monitor food intake and notify the doctor if food is not being consumed.
- Monitor electrolytes (especially potassium), blood urea nitrogen (BUN) levels and creatinine.
- Observe injection sites for manifestations of hypersensitivity, lipodystrophy and lipoatrophy.
- If symptoms of hypoglycaemia occur, confirm by testing blood glucose level and administer an oral source of a fast-acting carbohydrate, such as juice, milk or crackers. Hypoglycaemic symptoms may vary but commonly include feelings of shakiness, hunger and/or nervousness, accompanied by sweating, tachycardia or palpitations.
- If symptoms of hyperglycaemia occur, confirm by testing blood glucose level and notify the doctor immediately.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Self-administration of insulin, with a return demonstration (see Boxes 19.2, 19.3 and 19.4).
- Follow instructions for mixing insulins (refer to Box 19.8 later in the chapter).
- Always keep an extra vial of insulin available.
- Always have a vial of regular insulin available for emergencies.
- Be aware of the signs of hypersensitivity responses, hypoglycaemia and hyperglycaemia.
- Keep sweets or a sugar source available at all times to treat hypoglycaemia, if it occurs. Eat within 15 minutes of injecting rapid-acting insulins.
- Vision may be blurred during the first 6 to 8 weeks of insulin therapy; this is the result of fluid changes in the eye and should clear up in 8 weeks.
- Avoid alcoholic beverages, which may cause hypoglycaemia.
- Follow these guidelines for sick days:
  a. Never omit insulin.
  b. Always monitor blood glucose and/or urine ketones at least every 2 to 4 hours.
  c. Always drink plenty of fluids; try to drink at least one glass of water or other kilojoule-free, caffeine-free liquid each hour.
  d. Get as much rest as possible.
  e. Contact the doctor if there is persistent fever, vomiting, shortness of breath, severe pain in the abdomen, dehydration, loss of vision, chest pain, persistent diarrhoea, blood glucose levels above 14 mmol/L or ketones in the urine.
- Establish a plan for rotating injection sites and observe closely for changes in tissues, such as hardness, dimpling or sunken areas.

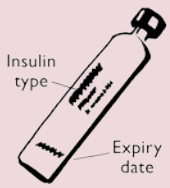
available through pharmacies, councils and Diabetes Australia. Procedures to dispose of sharps containers vary between the states and territories. For information, contact Diabetes Australia in the relevant state or the state government department of health or the local council. These are also excellent sources of information about where to get help with insulin (see Box 19.5).

Sliding-scale insulin Hospitalised people with type 1 and type 2 diabetes require intense blood glucose monitoring and frequent adjustments that are responsive to glycaemic changes secondary to the admitting condition and its treatment, including surgery (Deno & Schaper, 2011). Those with type 2 diabetes cannot manage with oral medications during hospitalisation because of the risk of hypoglycaemia from not eating and the slow response of these medications to correct hyperglycaemia. Hyperglycaemia is a serious and costly healthcare problem in hospitalised people today and is linked to increased morbidity, mortality and length of hospital stay. Hyperglycaemia is even seen in non-diabetic hospitalised people. Diabetic people with home insulin should be treated with an insulin regimen in hospital irrespective whether they have type 1 or type 2 diabetes. It is vital that blood glucose levels are stable prior to elective surgery and ideally people dependent on insulin should be placed first on the morning list of surgery. Following the hospital protocol for medication administration is important in preventing hypoglycaemia or hyperglycaemia. If the person is undergoing major surgery, or hyperglycaemia develops, an insulin–glucose infusion should be commenced. Clinical practice guidelines for the management of perioperative diabetes recommend target blood glucose levels of 5 to 10 mmol/L postoperatively. Clear written instruction regarding medication management should be given preoperatively and again on discharge to prevent complications (Australian Diabetes Council, 2012).

Maintaining normal blood glucose during hospitalisation decreases the risk of postoperative infections and shortens hospital stays. Healing is impaired when haemoglobin is glycosylated (HbA1c); glycosylated haemoglobin has increased affinity for oxygen, putting tissues at risk of ischemia (McCance &

BOX 19.2 How to draw up a mixed dose of insulin from penfill cartridges

1. Wash hands.



2. Check you have the correct insulin types. You will be drawing up the rapid- or short-acting insulin first.

3. If your long-acting insulin is 'cloudy' insulin (e.g. Protaphane, Humulin NPH), mix by tipping the cartridge up and down 10 to 20 times. *Do not shake the cartridge* as this damages the insulin. Clear insulin does not need to be mixed.

4. Open a new syringe. Make sure there is no air in the syringe by first pushing the plunger right down. Insert the needle into the cartridge of rapid- or short-acting insulin (e.g. Actrapid, Humulin R). Pull back the plunger of the syringe to draw up the dose required plus an extra 2 units, which allows you room to get rid of any air bubbles. The rubber stopper in the cartridge will gradually move down as you draw out the insulin and equalise the pressure.

5. Remove the syringe from the bottle, hold it vertically and push the plunger gently to get rid of any air bubbles and any extra insulin to obtain the correct dose. It may help to tap the side of the syringe to remove all air bubbles.

6. Insert the needle into the cartridge of long-acting insulin (e.g. Protaphane, Humulin NPH) and turn it upside down. Pull back the plunger to obtain the correct dose. If you draw back too much, you will have to discard the whole syringe and start again. *Do not push any insulin into the cartridge.*

7. Now you are ready to inject the insulin.

Source: G. R. Ambler & F. J. Cameron (eds) (2010). *Caring for diabetes in children and adolescents* (3rd ed.), p. 30. © Children's Diabetes Services. Reproduced with permission.

Huether, 2009). Further, diabetes leads to small-vessel disease, which impairs circulation and oxygenation of tissue for healing.

Intravenous insulin infusions are preferable for maintaining normal blood glucose during hospitalisation, although their use is dependent on frequent blood glucose monitoring and intensive nursing care. Supplements of regular insulin following sliding-scale prescriptions (relative to monitored blood glucose levels) are ineffective management protocols, risking both hyperglycaemia and hypoglycaemia. These supplements treat hyperglycaemia after it has occurred rather than preventing it. When medical conditions lead to observable periods of hyperglycaemia outside the ICU, it is preferable to provide basal subcutaneous insulin such as insulin glargine or short-acting insulin Actrapid and mealtime

BOX 19.3 Giving insulin with a syringe

To give an injection with a syringe:

1. Draw up insulin as described in Box 19.2.
2. Take a small pinch of skin with the index finger and thumb. The pinch needs to be at least to the depth of the needle. This is especially important in lean people, otherwise the injection may go too deep into the muscle layer and hurt more and the insulin will act differently.
3. Insert the needle straight into the pinched up skin (i.e. at 90 degrees) to its full length and push the plunger slowly all the way down to push in the insulin. In very lean individuals, injecting at a 45-degree angle to the skin may be necessary to avoid the injection going too deep.
4. Leave the needle in for about 5–10 seconds, then gradually let go of the skin and pull out the needle.
5. Dispose of the syringe in an approved sharps container.

Source: G. R. Ambler & F. J. Cameron (eds) (2010). *Caring for diabetes in children and adolescents* (3rd ed.), p. 31. © Children's Diabetes Services. Reproduced with permission.

supplements with rapid-onset fast-acting insulin or aspart insulin. Correctional doses can be added to the mealtime doses to keep serum glucose below 10.0 mmol/L (Diabetes Australia, Victoria, 2008).

Principles of insulin adjustment It is useful to think of three main types of insulin adjustments and to think of them in this order:

1. *Long-term adjustments.* These are changes to regular doses based on patterns in blood glucose readings over several days or longer. This may occur:
   - when coming out of a honeymoon phase
   - as the young person with type 1 diabetes grows, and especially when they have their growth spurt with puberty or reach the end of puberty
   - when there is a general change in activity levels.
2. *Thinking-ahead adjustments.* These are temporary changes to some doses based on what is going to happen that day. Examples of this are:
   - reducing a dose or doses for sporting activity
   - reducing an evening dose of insulin after a very active day to avoid delayed hypoglycaemia
   - adjusting a dose to plan for eating more or less at a meal.
3. *Fix-up adjustments.* This is adjusting a dose or giving an extra dose to 'fix up' a blood glucose reading that is unexpectedly high or low. For example:
   - reducing a short-acting insulin dose after hypoglycaemia near to injection time
   - giving a little extra short-acting insulin when the blood glucose level is found to be high before a meal
   - adjusting for sick days.

BOX 19.4 Giving insulin with a pen

1. Wash hands.
2. Check that you have the correct insulin pen (have your pens clearly marked) and that there is enough insulin remaining in the cartridge for the current injection. It is preferable to use a new needle for each injection.
3. If giving a cloudy long-acting or mixed insulin, be sure to mix the insulin well by inverting the pen 10 to 20 times. The cartridge contains a glass ball which mixes the insulin. Do not shake the pen as this will damage the insulin. Clear insulins do not need to be mixed.
4. Prime the pen (get rid of any air bubbles). Dial up a 2- to 4-unit dose and, holding the pen vertically, inject into the air to expel air bubbles (air shot) and to prime the pen. The pen is primed if drops of insulin without bubbles are coming from the needle. If not, keep repeating this procedure until a bubble-free stream of insulin is achieved with the air shot.
5. Dial up the required dose.
6. Select the injection site.
7. Steady the skin by taking a small pinch of skin with the index finger and thumb at the chosen site. The pinch needs to be at least to the depth of the needle. This is especially important in lean people, otherwise the injection may go too deep into the muscle layer and hurt more and the insulin will act differently. People who are not lean may not need to do a pinch, especially if using short needles (4, 5 or 6 mm), but only use a no-pinch technique if advised by your diabetes team.
8. Insert the needle straight into the pinched-up skin (i.e. at 90 degrees) to its full depth and push the pen button slowly all the way down to push in the insulin. In very lean individuals, injecting at a 45-degree angle to the skin may be necessary to avoid the injection going too deep.
9. Leave the needle in for 5–10 seconds, then gradually let go of the skin and pull out the needle.
10. Remove the needle from the pen after injection and dispose of in an approved sharps container.

Source: G. R. Ambler & F. J. Cameron (eds) (2010). *Caring for diabetes in children and adolescents* (3rd ed.), p. 29. © Children's Diabetes Services. Reproduced with permission.

BOX 19.5 Where to get help with insulin

- Local doctor or general practitioner (GP)
- Diabetes educator
- An accredited practising dietitian (contact the Dietitians Association of Australia)
- Diabetes specialist
- Diabetes Australia Infoline: Tel. 1300 136 588

BOX 19.6 Guidelines for insulin adjustment

- How you adjust insulin will depend on the types of insulin being used and the number of injections each day:
- Insulin doses often need adjustment. General principles are:
  - Pattern adjustment: look for patterns that indicate a need to adjust usual insulin doses because of high or low glucose levels at certain times over a few days or longer periods.
  - Look at this regularly.
  - Day-to-day adjustment (flexible daily adjustment): if you are using a flexible system, think about whether you need to adjust a dose or doses now or today to take into account a variation in carbohydrate intake or activity. Many people on multiple daily injections make these flexible decisions on a daily basis with their pre-meal injections.
  - Corrections: think about the need for a correction adjustment, especially for correcting a blood glucose level that is too high now.
- If hypoglycaemia is occurring frequently, don't delay adjustments.
- Seek help from your diabetes educator or doctor if you are not sure what to do or your adjustments are not working.
- Pump users also need to adjust insulin, but details are different.

Source: G. R. Ambler & F. J. Cameron (eds) (2010). *Caring for diabetes in children and adolescents* (3rd ed.), p. 115. © Children's Diabetes Services. Reproduced with permission.

More detail on these three types of adjustment is given in the following sections. General points include:

- Insulin adjustments are generally based on the recognition of blood glucose patterns over several days, so enough blood glucose readings need to be done to allow this.
- At times of instability or illness it is necessary to do extra blood glucose readings to guide adjustment.
- Cautious adjustments in steps are made in insulin doses until blood glucose levels in the target range are reached.
- Unexplained hypoglycaemia requires thinking about insulin doses without delay, and adjustment without waiting for a pattern to emerge may be justified.
- When adjusting, it is usually unwise to make changes to insulin doses every day, or to change too many doses at once, since this can lead to more instability and confusion.
- Frequent dose changes may be necessary when insulin needs are changing rapidly, in which case your diabetes educator or doctor should be aware and able to help.
- People on insulin are advised to adjust their dose in approximately 10% increments and observe the effects over several days before further changes.
- Half-unit increments can be made in people with small doses of insulin (< 5 units).
- People on insulin pumps need to determine if changes need to be made to the basal or the bolus dose from their monitoring; increments of 5% to 10% are appropriate.
- Rapid changes may be necessary in severe hypoglycaemia. (Australasian Paediatric Endocrine Group for the Department of Health and Ageing, 2005).
- Some children need variations in dose on a day-to-day basis to adjust for activity and exercise. See Box 19.6.
- Insulin adjustment also requires a knowledge of the types of insulin the child is on, particularly when they start to work and how long they work for.

  See Box 19.6 for guidelines for insulin adjustment.

Preparing the injection The vial of insulin in use may be kept at room temperature for up to 4 weeks. Stored vials should be kept in the refrigerator and brought to room temperature prior to administration.

Regular insulin does not require mixing. If the solution is cloudy or discoloured, the vial should be discarded. The other types of insulin must be mixed to disperse the particles evenly throughout the solution. Mix the vial by gently rolling it between the hands; vigorous shaking causes bubble formation and frothing, which makes the dose inaccurate. It is critical that no air bubbles remain in the prepared dose, because even a small bubble can displace several units of insulin.

Sites of injection Although in theory any area of the body with subcutaneous tissue can be used for injections of insulin, certain sites are recommended (see Figure 19.3). The rate of absorption and peak of action of insulin differ according to the site. The site that allows the most rapid absorption is the abdomen, followed by the deltoid muscle, then the thigh and then the hip. Because of the rapid absorption, the abdomen is the recommended site. See Box 19.7 for techniques to minimise painful injections.

When administering insulin, aspiration to check for blood is not necessary. Do not massage the site after administering the injection, because this may interfere with absorption; pressure, however, may be applied for about 1 minute. Rotation of injection sites is recommended for people using pork insulin; rotation within sites is recommended for people using human or purified pork insulin. The distance between consecutive injections should be about 2.5 cm (avoiding the area within a 5 cm radius from the umbilicus). Insulin should not be injected into an area to be exercised (such as the thigh before a vigorous walk) or to which heat will be applied;

BOX 19.7 Techniques to minimise painful injections

- Inject insulin that is at room temperature.
- Make sure no air bubbles remain in the syringe before the injection.
- Although the use of alcohol to clean the skin prior to the insulin injection is not recommended for people who use insulin long term, if there is a need for its use due to a possible contamination risk in the skin then wait until alcohol on the skin completely dries before the injection.
- Relax muscles in the injection area.
- Penetrate the skin with the needle quickly.
- Don't change the direction of the needle during insertion or withdrawal.
- Don't reuse needles. Always use a new needle with each injection.

Sources: Adapted from Hirsch, L., Gibney, M., Albanese, J. et al. (2010). Comparative glycemic control, safety and patient ratings for a new 4 mm x 32G insulin pen needle in adults with diabetes. *Current Medical Research and Opinion*, *26*(6), 1531–1541.

exercise or heat may increase the rate of absorption and cause a more rapid onset and peak of action.

Lipodystrophy **Lipodystrophy** (hypertrophy of subcutaneous tissue) or **lipoatrophy** (atrophy of subcutaneous tissue) may result if the same injection sites are used repeatedly, especially with pork and beef insulins. The tissues become hardened and have an orange-peel appearance. The use of refrigerated insulin may trigger the development of tissue atrophy or hypertrophy.

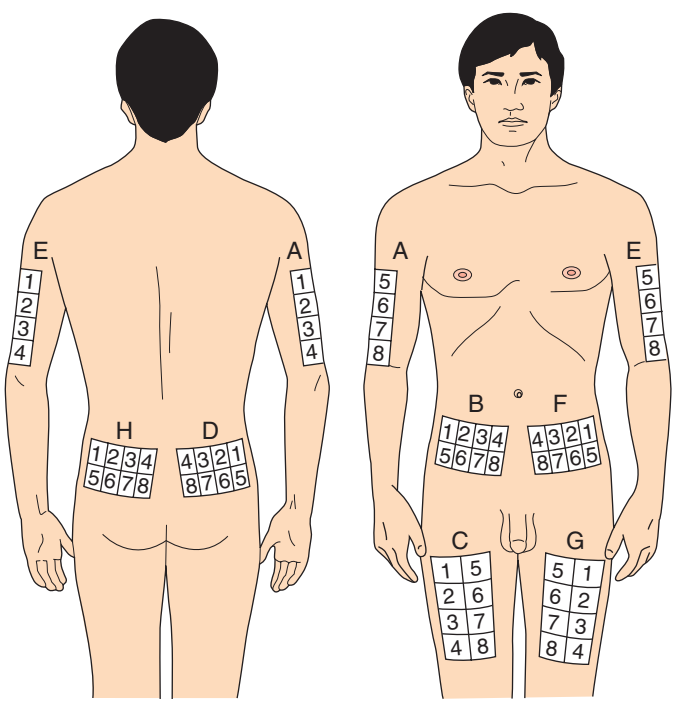


FIGURE 19.3 ■ Sites for insulin injections

These problems rarely occur with the use of human insulins. Lipodystrophy and lipoatrophy alter insulin absorption, delaying its onset or retaining the insulin in the tissue for a period of time instead of allowing it to be absorbed into the body. Lipodystrophy usually resolves if the area is unused for a minimum of 6 months.

Mixing insulins When a person with diabetes requires more than one type of insulin, mixing is recommended to avoid administering two injections per dose. Two different concentrations are administered, because a single dose of intermediate-acting or long-acting insulin rarely provides adequate control of blood glucose levels. The procedure for mixing insulins is described in Box 19.8. Following are some general guidelines:

- Commercially mixed insulins are recommended if the insulin ratio is appropriate for the requirements of the person.
- Regular insulin may be mixed with all types of insulin except long-acting insulin or glargine; it may be injected immediately after mixing or stored for future use.
- Intermediate-acting insulin such as Protophane, Humulin NPH, Hypurin Isophane (bovine or porcine) may be mixed only with regular or short-acting insulin.
- Do not mix human and animal insulins.
- Always withdraw regular insulin first to avoid contaminating the regular insulin with intermediate-acting insulin.

Hypersensitivity responses When injected, insulin may cause local and systemic hypersensitivity responses. Manifestations of local reactions are a hardening and reddening of the area that develops over several hours. Local reactions result from a contaminant in the insulin and are more likely to occur when less purified insulin products are used.

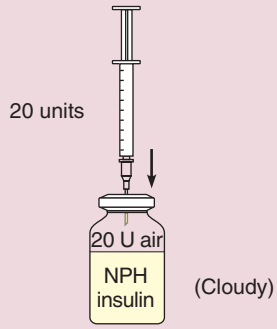
Systemic reactions occur rapidly and are characterised by widespread, red, intensely pruritic welts. Respiratory difficulty may occur if the respiratory system is involved. Systemic responses are due to an allergy to the insulin itself and are most common with beef insulin (not commonly used in Australia). The person can be desensitised by administering small doses of purified pork or human insulin, followed by progressively larger doses.

HYPOGLYCAEMIC AGENTS

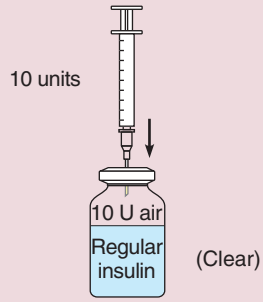
Hypoglycaemic agents are used to treat people with type 2 DM. Nursing implications for this category of drugs are discussed in the 'Medication administration' box below. These medications lower blood sugar by stimulating or increasing insulin secretion, preventing breakdown of glycogen to glucose by the liver and increasing peripheral uptake of glucose by making cells less resistant to insulin. Peripheral uptake refers to uptake by muscles and fat in the arms and legs, rather than in the trunk. Some hypoglycaemics keep blood sugar low by blocking absorption of carbohydrates in the intestines. A new hypoglycaemic agent that is not insulin but is only available as an injectable is exenatide (Byetta), the first synthetic analogue of the incretin hormone glucagon-like peptide 1 (GLP-1). Exenatide is an 'incretin mimetic' which increases glucose-dependent insulin secretion, suppresses inappropriate glucagon secretion, delays gastric emptying and reduces appetite. It is approved for use in

BOX 19.8 Mixing insulins: 10 units of regular or short-acting and 20 units of NPH or intermediate-acting insulin

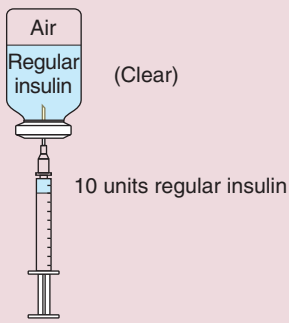
1. Wash hands.
2. Inspect regular insulin for clarity.
3. Gently rotate NPH insulin to mix well.
4. Wipe off the top of both vials with an alcohol pad.
5. Draw 20 units of air into the syringe and inject air into the NPH vial (Figure A). Withdraw needle.
6. Draw 10 units of air into the syringe and inject air into the regular vial (Figure B).
7. Invert the vial and withdraw 10 units of regular insulin (Figure C). Withdraw the needle.
8. Insert the needle into the NPH vial and carefully withdraw 20 units of NPH insulin (Figure D).
9. Administer the insulin.
10. Wash hands and properly dispose of the syringe.



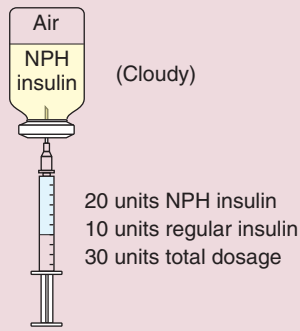
A Injecting air into the NPH vial



B Injecting air into the regular insulin vial



C Withdrawing regular insulin



D Withdrawing NPH insulin

Australia as an adjunctive therapy to improve glycaemic control in people with type 2 diabetes mellitus who are taking metformin, a sulfonylurea, or a combination of metformin and a sulfonylurea, but are not achieving adequate glycaemic control (National Prescribing Service (NPS), 2010).

ASPIRIN THERAPY People with diabetes are up to four times more likely to die from cardiovascular disease. It is recommended by the Royal Australian College of General Practitioners (RACGP) that a once-daily dose of 81 to 325 mg of enteric-coated aspirin be given to reduce atherosclerosis in people with vascular disease or increased cardiovascular risk factors.

Recent concerns have been raised by several experts in Australia with regard to the use of aspirin in people with diabetes for primary prevention of cardiovascular disease. They say that current guidelines for aspirin as primary prevention in these people have been extrapolated from evidence showing efficacy in secondary prevention of CVD events. There is still no clinical trial evidence to support the routine use of aspirin in primary prevention for people with diabetes. Diabetes Australia, which endorses the RACGP recommendations, does not have a position on the use of aspirin as primary prevention (*Medical Observer*, 2012; Song, Gray & Tesfaye, 2009; *The Lancet*, 2009).

Nutrition

The management of diabetes requires a careful balance between the intake of nutrients, the expenditure of energy and the dose and timing of insulin or oral antidiabetic agents. Although everyone has the same need for basic nutrition, the person with diabetes must eat a more structured diet to prevent hyperglycaemias. The goals for dietary management for adults with diabetes as recommended by the Heart and Diabetes Institute of Australia (2009a) are outlined in Box 19.9.

CARBOHYDRATES AND THE GLYCAEMIC INDEX All carbohydrate foods break down to glucose during digestion. Glucose enters the bloodstream, raising the blood glucose level which reaches a peak 30 to 60 minutes later (CSIRO & Baker IDI Heart and Diabetes Institute, 2012). As carbohydrate provides the body with fuel for energy, it should be a part of all diabetic meals and snacks. The quantity and types of carbohydrate-rich foods will affect blood glucose level. A very high carbohydrate intake may raise the blood glucose levels too high. Foods with carbohydrates include bread, legumes, biscuits, rice, fruit, cakes, pasta, milk and yoghurt, sugar, grains, cereals and starchy vegetables such as potato, sweet potato and corn.

MEDICATION ADMINISTRATION Oral hypoglycaemic agents available in Australia

There are six classes of oral medications available in Australia to treat type 2 diabetes. They are prescribed alone or in combination.

1. Sulfonylureas
2. Meglitinides
3. Biguanides
4. Alpha-glucosidase inhibitors
5. Thiazolidinediones (glitazones)
6. Dipeptidyl peptidase 4 (DPP4) inhibitors

SULFONYLUREAS

Glimepiride (Amaryl, Aylide, Diapride, Dimirel, Glimepiride Sandoz)
Glipizide (Minidiab, Melizide)
Gliclazide ER (Diamicron MR, Glyade MR, Oziclide MR)
Glicazide (Glyade, Mellihexal, Nidem, GenRx Gliclazide)
Glibenclamide (Daonil, Glimel)

Sulfonylureas act by stimulating the pancreatic cells to secrete more insulin and by increasing the sensitivity of peripheral tissues to insulin. The most common side effect is hypoglycaemia, therefore it is important that the person eat regular meals while on this medication. A person should only be taking one type of sulfonylurea medication. These drugs are often suspended during hospitalisation. Other side effects that may be seen with these medications are weight gain, stomach upset, jaundice and possibly rash (but is rare) (Diabetes Australia, Victoria, 2012; MIMS Online Australia, 2012).

MEGLITINIDES

Repaglinide (Novonorm)

These drugs lower blood glucose levels by stimulating release of insulin from the pancreatic islet cells. They produce a greater decrease in postprandial glucose (2 hours after starting a meal) and have a decreased risk of hypoglycaemia (Diabetes Australia, Victoria, 2012; MIMS Online Australia, 2012).

BIGUANIDES

Metformin (Glucophage, Diabex, Diaformin, Formet, Metforbell, Glucohexal, Glucomet, Genrx metformin, Genepharm metformin)
Metformin ER (Diabex XR, Diaformin XR, Metex XR)

Metformin is usually prescribed first for people with diabetes type 2. Biguanides decrease the overproduction of glucose by the liver, slow absorption of glucose in the small intestine and make the body more sensitive to insulin. These should be taken with meals, may help with weight loss and have side effects including diarrhoea, nausea and metallic taste. If renal insufficiency develops, metformin must be discontinued. Because of an increased risk of metformin-induced lactic acidosis, metformin is usually suspended during hospitalisation. It should be discontinued temporarily before and after using contrast media for diagnostic imaging and anaesthesia (Diabetes Australia, Victoria, 2012; MIMS Online Australia, 2012).

ALPHA-GLUCOSIDASE INHIBITORS

Acarbose (Glucobay)

These agents inhibit the enzyme alpha-glucosidase, found in the brush-border cells that line the small intestine, which breaks down more complex carbohydrates into sugars. Because alpha-glucosidase inhibitors inhibit the breakdown and subsequent absorption of carbohydrates (dextrins, maltose, sucrose and starch) from the gut following meals, the impact of these drugs is on post-prandial hyperglycaemia. They should not be taken in pregnancy, breastfeeding, inflammatory bowel disease or malabsorption syndrome. Possible side effects are flatulence, bloating and diarrhoea (Complete Medication Services Consultant Pharmacists (CMS), 2006; Diabetes Australia, Victoria, 2012; MIMS Online Australia, 2012).

THIAZOLIDINEDIONES (GLITAZONES)

Rosiglitazone (Avandia)
Pioglitazone (Actos)

This class of drugs acts by sensitising peripheral tissue to insulin, by enhancing insulin activity in both muscle and fat cells and, to a lesser extent, by inhibiting hepatic glucose production. Pioglitazone may also help by reducing cholesterol and triglyceride levels. They do not need to be taken with meals. Weight gain may occur due to fluid retention and increased fat tissue. People with heart failure or liver disease should avoid this medication. Liver function should be monitored regularly while on glitazones. Increased risk of small fractures in the arms, hands and feet have been reported in women taking this medication (Diabetes Australia, Victoria, 2012).

DIPEPTIDYL PEPTIDASE 4 (DDP-4) INHIBITORS

Sitagliptin (Januvia)
Vildagliptin (Galvus)
Saxagliptin (Onglyza)
Linagliptin (Trajenta)

DDP-4 inhibitors reduce glucose levels by inhibiting the enzyme DDP-4 and therefore prolonging the action of the incretin hormones. These hormones work by reducing glucose levels after meals by stimulating insulin production by the pancreas and by reducing the secretion of glucagon which will decrease the release of glucose from the liver. Tablets need to be taken at the same time each day. People may develop headaches and nausea and have an increased risk of getting a cold. These drugs are not to be taken by pregnant or breastfeeding women and people under 18 years old. Caution is to be taken when people have renal impairment, and DDP-4 inhibitors are not recommended for people with liver failure (Diabetes Australia, Victoria, 2012).

COMBINATIONS

Metformin/Glibenclamide (Glucovance)
Metformin/Rosiglitazone (Avandamet)
Sitagliptin/Metformin (Janumet)
Vidagliptin/Metformin (Galvumet)

These medications provide the action of two classes of medications in only one tablet in order to achieve desirable glucose control (Diabetes Australia, Victoria, 2012).

Nursing responsibilities

- Assess people taking oral hypoglycaemic agents closely for the first 7 days to determine therapeutic response.
- Follow their medication(s) instructions strictly.

(continued)

MEDICATION ADMINISTRATION Oral hypoglycaemic agents available in Australia (continued)

- Teach the person the importance of maintaining a prescribed diet and exercise program.
- Monitor for hypoglycaemia if the person is also taking non-steroidal anti-inflammatory agents (NSAIDs), sulfonamide antibiotics, ranitidine, cimetidine or beta-blockers; these drugs intensify the action of sulfonylureas.
- Monitor for hyperglycaemia if the person is also taking calcium channel blockers, oral contraceptives, glucocorticoids, phenothiazines or thiazide diuretics; these drugs decrease the hypoglycaemic responses to sulfonylureas.
- Do not administer these drugs to pregnant or lactating women.
- Assess for side effects.
- If the person is to have a thyroid test, determine whether a sulfonylurea has been taken; sulfonylureas interfere with the uptake of radioactive iodine.
- Monitor for hypoglycaemia with concurrent administration of an oral antidiabetic agent and insulin.

Health education for the person and family

- Maintain prescribed diet and exercise regimen.
- You may need insulin if you have surgery, trauma, fever or infection.
- Follow instructions to monitor blood glucose.
- Report illness or side effects to the healthcare provider.
- Undergo periodic laboratory evaluations as prescribed by your healthcare provider.
- Avoid alcohol intake, which may cause a reaction involving flushing, palpitations and nausea.
- The medication interferes with the effectiveness of oral contraceptives; other birth control measures may be required.
- Mild symptoms of hyperglycaemia may appear if a different agent is begun.
- Take medications as prescribed—for example, once a day at the same time each day.

What does GI mean? The glycaemic index (GI) is a guide to how a carbohydrate food may affect the blood glucose level. A low GI food breaks down to glucose more slowly and causes a slower rise in the blood glucose levels compared to a high GI food. Low GI carbohydrate foods should be chosen for a healthy diet. At least one low GI food should be included in each meal (see Table 19.3).

FIBRE A healthy meal plan contains foods high in dietary fibre. There are two types of fibre—soluble and insoluble. Eating soluble fibre helps to lower blood glucose and cholesterol levels. Insoluble fibre is good for a healthy digestive system. A variety of plant foods to get plenty of insoluble and soluble fibre should be included in the diet. The aim is to eat at least 30 g of fibre each day. Many of the low GI foods listed in Table 19.3 are also good sources of fibre.

SUGAR Small amounts of sugar can be included as part of a healthy meal—for example, 1–2 teaspoons of sugar added to porridge, or a teaspoon of jam or honey on multigrain toast. Savoury foods containing sugar, such as baked beans and tomato sauce, can also be eaten.

Foods which contain large amounts of sugar, such as soft drinks, regular jelly and confectionery, should be avoided. Some artificially sweetened foods are suitable, but some may be high in fat.

FATS Although blood glucose levels are directly affected by carbohydrate, eating too much fat is also a problem. Fats provide energy and some vitamins, so a small amount is essential. However, too much leads to carrying excess body weight, which increases insulin resistance. There are different types of fats in food and some are worse than others. Saturated fat and trans fat increase the risk of heart disease by increasing blood cholesterol levels.

Saturated fat is found in animal products (e.g. butter, full-cream milk, cheese and yoghurt, fatty meats, processed meats, cream, lard), palm oil (often used in commercial snacks and baked products such as biscuits and pastries), takeaway foods and chips, and coconut oil, including coconut cream and milk.

Trans fat is found in a variety of foods manufactured with vegetable oils, such as biscuits, pastries and some margarines.

Ways to reduce saturated and trans fat intake are to eat less fried foods, pies, processed meats, chips, chocolate, biscuits and pastries, limit butter, lard, cream and sour cream, choose lean meat and trim any fat off meat before cooking, remove chicken skin, use low-fat dairy products, and use monounsaturated and polyunsaturated oils and margarine spreads. These foods are still high in kilojoules; the amount should be minimised if weight control or weight loss is desired.

Monounsaturated fat is found in oils such as olive, canola, sunflower and peanut oil; in margarines made from monounsaturated vegetable oils such as olive or canola oil; in nuts such as peanuts, almonds and cashews; and in avocados and olives.

Polyunsaturated fat also helps to improve cholesterol levels. Polyunsaturated fat is found in oils such as sunflower, safflower and soybean oil, in margarines made from polyunsaturated vegetable oils such as sunflower or grapeseed oil, and in walnuts and fish.

Omega-3 fats (a type of polyunsaturated fat) improve heart health and have other health benefits. Eat fish (fresh or canned) at least twice a week to increase the intake of omega-3 fats. Fish such as salmon, gemfish and blue grenadier are good sources of omega-3 fats.

Plant sterols are added to some foods, including margarine spreads such as Flora Proactiv™ and Meadow Lea Logicol™. These help to reduce cholesterol levels. The use of these products should be discussed with the health

BOX 19.9 Goals for dietary management for adults with diabetes

- Control blood glucose levels.
- Control weight.
- Improve cholesterol levels.
- Reduce the risk of heart disease.
- Improve general health and wellbeing.

Dietary guidelines for Australian adults suggest:

- enjoying a wide variety of nutritious foods
- eating plenty of vegetables, legumes and fruits
- eating plenty of cereals (including breads, rice, pasta and noodles), preferably wholegrain
- including lean meat, fish, poultry and/or alternatives
- including milks, yoghurts, cheeses and/or alternatives—reduced-fat varieties should be chosen, where possible
- drinking plenty of water and taking care to:
  - limit saturated fat and moderate total fat intake
  - choose foods low in salt
  - limit alcohol intake if the person chooses to drink
  - consume only moderate amounts of sugars and foods containing added sugars
  - eat regularly during the day and spread food over breakfast, lunch and dinner.

Diabetics should aim to eat the following each day:

- 5 serves vegetables
- 2 serves fruit
- 5 serves grains, breads and cereals
- 2–3 serves low-fat dairy products
- 1 serve lean meat, chicken or fish, or a meat alternative such as lentils, beans or tofu.

People with higher energy needs may require more serves. A dietitian can offer guidance on the quantity of food that the person may need.

Source: Heart and Diabetes Institute of Australia (2009a). *Learning to live with type 2 diabetes.* © 2009 Baker IDI Heart and Diabetes Institute. All rights reserved. Retrieved from www.bakeridi.edu.au/ShopProductList.aspx?CategoryID=4.

TABLE 19.3 Foods with a low GI

Breads	Multigrain breads, pumpernickel, fruit and grains, Bakers Delight low GI™
Breakfast cereals	High-fibre breakfast cereals such as rolled oats, untoasted muesli, All-Bran™, Guardian™
Pasta and noodles	Wheat pasta Most noodles, such as rice vermicelli, soba noodles, mung bean noodles
Rice	Long-grain rice such as Doongara and Basmati; wild, moolgiri low GI, brown, black and red rice
Grains	Quinoa, barley, buckwheat, semolina and bulgur
Fruits	Fresh, dried and canned fruit, such as apples, pears, oranges, grapes, grapefruit, peaches, plums, kiwi fruit and bananas
Starchy vegetables	Sweet potato (orange), yam, corn, Carisma™ and Nicola potato *Note:* Most coloured vegetables are low in carbohydrate and have little effect on blood glucose levels, e.g. salad, greens and orange
Legumes and pulses	All types (canned or dried) such as kidney beans, mixed beans, chick peas, lentils and baked beans
Milk and yoghurt	Low-fat milk and yoghurt, soy milk and yoghurt, and almond milk

Source: Heart and Diabetes Institute of Australia (2015). *Carbohydrates and Glycaemic Index (GI).* Retrieved from http://admin.bakeridi.edu.au/Assets/Files/Carbs%20&%20Glycaemic%20Fact%20SHEET.pdf.

professional, as quantities are important and they are not suitable for everyone.

When used in small quantities, both monounsaturated fat and polyunsaturated fat can help to improve blood cholesterol levels. Monounsaturated fat can help improve cholesterol levels if it is used in place of saturated fat.

Cholesterol in food Cholesterol is found in foods such as eggs, offal (e.g. kidneys, liver and brain) and seafood. Although cholesterol in food can increase the blood cholesterol level, it is more important to limit foods high in saturated fat or trans fat.

PROTEIN Protein is used by the body for growth and repair and is found in foods such as meat, fish, eggs and dairy products. The body's requirements for protein are met by 2–3 serves of dairy foods and 1 serve of meat or fish each day. A higher protein intake is not necessary and many foods high in protein are also high in saturated fat (e.g. dairy foods, meat) so eating more protein may also increase saturated fat intake.

SALT A high salt (sodium) intake contributes to high blood pressure in some people. Reducing the amount of salt that is consumed may help reduce blood pressure.

ALCOHOL Alcohol is high in energy and therefore can contribute to weight gain. If taking insulin or medications for diabetes, alcohol may increase the risk of hypoglycaemia. To reduce the risk of hypoglycaemia, alcohol should not be consumed without having a meal or snack containing carbohydrate.

It is recommended that people with diabetes limit alcohol to no more than 2 standard drinks a day. One standard drink equals: 285 mL beer, 425 mL light beer (less than 3% alcohol), 100 mL wine, 60 mL fortified wine, 30 mL spirit (Diabetes Australia, Victoria, 2012).

SWEETENERS The diet plan for people with diabetes restricts the amount of refined sugars. As a result, many people use non-nutritive sweeteners and foods or drinks made with non-sweeteners. Commercially produced non-nutritive sweeteners are approved for use by the FDA. Although questions have been raised about the safety of these substances in laboratory animal studies, they are considered safe for use by humans.

Included in this category of sweeteners are saccharin (Sweet & Low), aspartame or neotame (Nutrasweet®, Equal®), sucralose (Splenda®) and acesulfame potassium. The non-nutritive sweeteners have negligent amounts of or no kilojoules, do not produce dental caries and produce very little or no changes in blood glucose levels.

People with diabetes also use nutritive sweeteners, including fructose, sorbitol and xylitol. The kilojoule content of these substances is similar to that of table sugar (sucrose), but they cause less elevation in blood glucose. They are often included in foods labelled as 'sugar free'. Sorbitol may cause flatulence and diarrhoea.

Researchers are continuing to study the safety and effectiveness of the sweeteners. When teaching people about diet, the nurse should include information about the kilojoule content of sweeteners and the meaning of such phrases as *sugar free* and *dietetic* on labels (Diabetes Australia, 2012; Heart and Diabetes Institute of Australia, 2009a).

MEAL PLANNING Several different systems for meal planning are available to the person with diabetes. These systems include a consistent-carbohydrate diabetes meal plan, exchange lists, point systems, food groups, carbohydrate counting and kilojoule counting. No matter what system is used, however, it must take into account the person's individualised eating habits, diet history, food values and special needs. Altering foods and meal patterns is often one of the most difficult parts of diabetes management; careful consideration of individualised preferences enhances compliance with the diet. Although dietitians provide nutrition information and advice, nurses must know what is prescribed and be able to reinforce teaching and answer questions.

For information on diabetes and healthy eating, go to the Better Health Channel site at www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Diabetes_and_healthy_eating.

Diet plan for type 1 diabetes Diet and insulin prescription must be integrated for optimal energy metabolism and the prevention of hyperglycaemia or hypoglycaemia. The goals of the diet plan are to achieve optimal glucose and lipid levels, improve overall health and maintain reasonable body weight. To meet these goals, the following strategies must be implemented:

- Glucose regulation requires correlating eating patterns with insulin onset and peak of action.
- Meals, snacks and insulin regimens should be based on the person's lifestyle.
- Meal planning depends on the specific insulin regimen prescribed.
- Snacks are an important consideration in relation to the amount and timing of exercise.
- The diet plan must consider the availability of foods, based on occupational, financial, religious and ethnic constraints.
- Self-monitoring of blood glucose levels helps the person make adjustments for planned and unplanned changes in routines.

Diet plan for type 2 diabetes The goals of the diet plan are to improve blood glucose levels, improve overall health, prevent or delay complications, and attain or maintain reasonable body weight. Because the majority of these people are overweight, weight loss is important and facilitates achieving the other goals.

There are no specific guidelines for the type 2 diet, but in addition to decreasing kilojoules, it is recommended that the person consume three meals of equal size, evenly spaced approximately 4 to 5 hours apart, with one or two snacks. The person with type 2 DM should also decrease fat intake. If the exchange list is difficult to use, kilojoule counting or designing the diet by grams of fat may be more useful.

Sick-day management When the person with diabetes is sick or has surgery, blood glucose levels increase, even though food intake decreases. The person often mistakenly alters or omits the insulin dose, causing further problems. The guidelines for dietary management during illness focus on preventing dehydration and providing nutrition for promoting recovery. In general, sick-day management includes the following:

- monitoring blood glucose at least four times a day throughout an illness
- testing urine for ketones if blood glucose is greater than 13 mmol/L
- continuing to take the usual insulin dose or oral hypoglycaemic agent
- sipping 150 to 250 mL of fluid each hour
- substituting easily digested liquids or soft foods if solid foods are not tolerated (the substituted liquids and foods should be carbohydrate equivalents—e.g. ½ cup sweetened gelatine, ½ cup fruit juice, 1 ice block, ¼ cup sherbet and ½ cup regular soft drink)
- calling the healthcare provider if the person is unable to eat for more than 24 hours or if vomiting and diarrhoea last for more than 6 hours.

Diet plan for the older adult The majority of older adults have type 2 DM and should follow the general guidelines for that diet plan. However, special considerations for the older adult are important if the diet plan is to be followed, including:

- dietary likes and dislikes
- who prepares the meals
- age-related changes in taste perception
- dental health
- transportation to buy foods
- available income.

Other factors to consider in planning the diet for the older adult include the age-related decline in kilojoule requirements, decline in physical activity due to age and/or chronic illnesses, and the onset or progression of other chronic illnesses. The older adult who is overweight should reduce kilojoule intake to ensure weight loss; but at the same time, careful monitoring for malnutrition is necessary. It is possible for the older adult to revert to normal glucose tolerance if ideal body weight is regained.

Exercise

The third component of diabetes management is a regular exercise program. The benefits of exercise are the same for everyone, with or without diabetes: improved physical fitness,

improved emotional state, weight control and improved work capacity. In people with diabetes, exercise increases the uptake of glucose by muscle cells, potentially reducing the need for insulin. Exercise also decreases cholesterol and triglycerides, reducing the risk of cardiovascular disorders. People with diabetes should consult their primary healthcare provider before beginning or changing an exercise program.

The ability to maintain an exercise program is affected by many different factors, including fatigue and glucose levels. It is as important to assess the person's usual lifestyle before establishing an exercise program as it is before planning a diet. Factors to consider include the person's usual exercise habits, living environment and community programs. The exercise that the person enjoys most is probably the one that they will continue with throughout life. People with diabetes should determine the best type of exercise for their individual needs and follow the recommendations and advice of their health care provider, diabetes educator and other professionals when engaging in any exercise program. Considerations include using proper footwear, inspecting the feet daily and after exercise, avoiding exercise in extreme heat or cold, and eating a snack prior to strenuous exercise to prevent hypoglycaemia (Diabetes Australia, 2012; Better Health Channel, 2013).

ACUTE COMPLICATIONS OF DIABETES

The person with DM, regardless of type, is at increased risk of complications involving many different body systems. Alterations in blood glucose levels, alterations in the cardiovascular system, neuropathies, and an increased susceptibility to infection and periodontal disease are common. In addition, the interaction of several complications can cause problems of the feet. The 'Multisystem effects of diabetes mellitus' illustration on the next page shows the progression from cardinal signs to acute and late complications for the person with diabetes. A discussion of each of these complications follows; related interprofessional care and nursing care are discussed later in the chapter.

Acute complications: alterations in blood glucose levels

The following discussion provides additional information about hyperglycaemia and hypoglycaemia. Table 19.4 compares DKA, HHS and hypoglycaemia.

Hyperglycaemia

The main problems resulting from hyperglycaemia in the person with diabetes are DKA and HHS. Two other problems are the dawn phenomenon and the Somogyi phenomenon.

The **dawn phenomenon** is a rise in blood glucose between 4 am and 8 am that is not a response to hypoglycaemia. It is believed to be due to an increase in counter-regulatory hormones such as growth hormone, cortisol and catecholamines which work against the action of insulin and cause morning hyperglycaemia (WebMD, 2012). The **Somogyi phenomenon** is a combination of hypoglycaemia during the night with a rebound morning rise in blood glucose to hyperglycaemic levels. The hyperglycaemia stimulates the counter-regulatory hormones, which stimulate gluconeogenesis and glycogenolysis and also inhibit peripheral glucose use. This may cause insulin resistance for 12 to 48 hours (McCance & Huether, 2009).

DIABETIC KETOACIDOSIS As the pathophysiology of untreated type 1 DM continues, the insulin deficit causes fat stores to break down, resulting in continued hyperglycaemia and mobilisation of fatty acids with a subsequent ketosis. **Diabetic ketoacidosis (DKA)** develops when there is an absolute deficiency of insulin and an increase in the insulin counter-regulatory hormones. Glucose production by the liver increases, peripheral glucose use decreases, fat mobilisation increases and ketogenesis (ketone formation) is stimulated. Increased glucagon levels activate the gluconeogenic and ketogenic pathways in the liver. In the presence of insulin deficiency, hepatic overproduction of beta-hydroxybutyrate and acetoacetic acids (ketone bodies) causes increased ketone concentrations and an increased release of free fatty acids. As a result of a loss of bicarbonate (which occurs when the ketone is formed), bicarbonate buffering does not occur and a metabolic acidosis occurs, called DKA. Depression of the central nervous system from the accumulation of ketones and the resulting acidosis may cause coma and death if left untreated (Porth & Matfin, 2009). See Figure 19.4.

DKA also may occur in a person with diagnosed diabetes when energy requirements increase during physical or emotional stress. Stress states initiate the release of gluconeogenic hormones, resulting in the formation of carbohydrates from protein or fat. The person who is sick, has an infection, or decreases or omits their insulin doses is at a greatly increased risk of developing DKA.

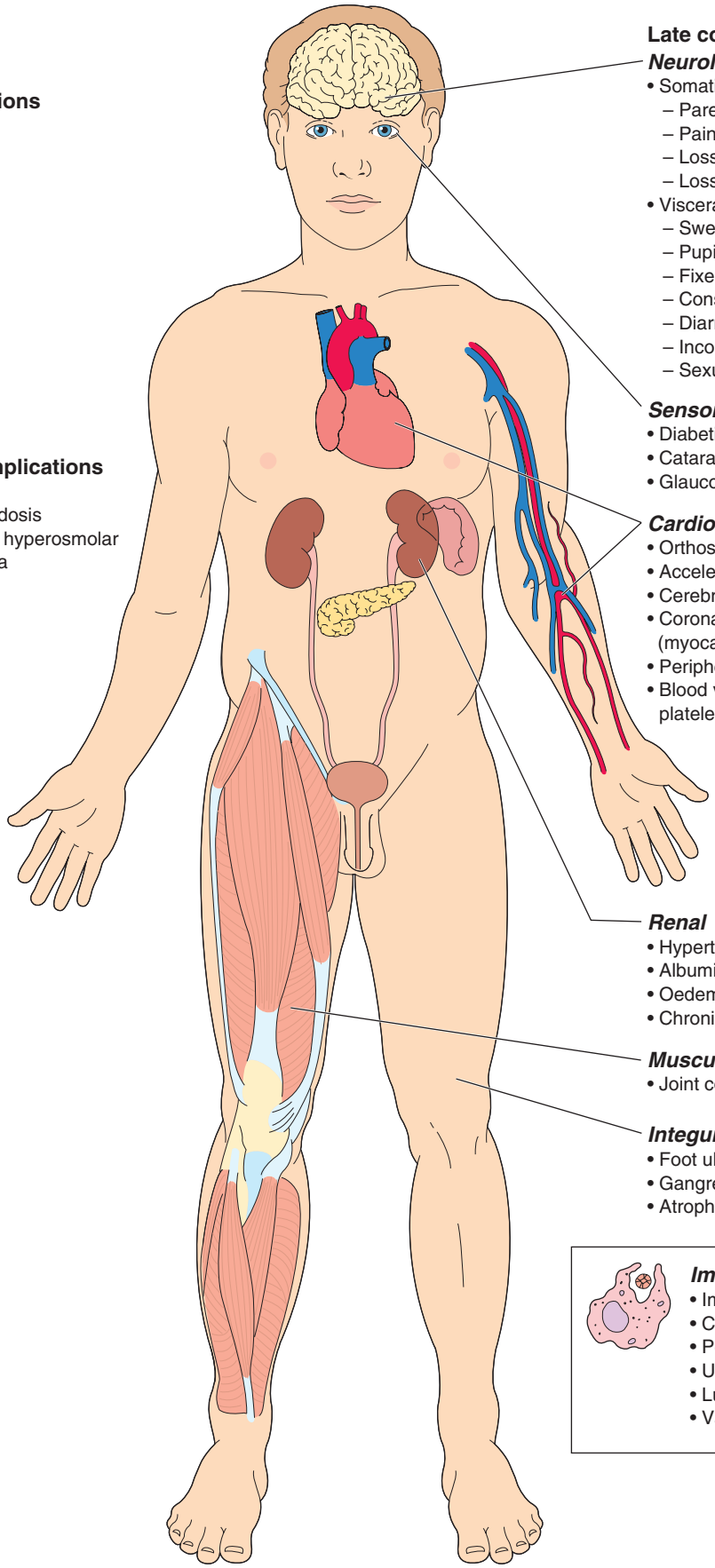
DKA involves four metabolic problems:

1. hyperosmolarity from hyperglycaemia and dehydration
2. metabolic acidosis from an accumulation of ketoacids
3. extracellular volume depletion from osmotic diuresis
4. electrolyte imbalances (such as loss of potassium and sodium) from osmotic diuresis.

Manifestations of DKA result from severe dehydration and acidosis.

HYPEROSMOLAR HYPERGLYCAEMIC STATE (HHS) The metabolic problem called **hyperosmolar hyperglycaemic state (HHS)** occurs in people who have type 2 DM. HHS is characterised by a plasma osmolarity of 340 mmol/kg or greater (the normal range is 280 to 300 mmol/kg (South-Western Sydney Local Health District (SWSLHD), 2012), greatly elevated blood glucose levels (over 33.3 mmol/L and often 55.5 to 111 mmol/L) and altered levels of consciousness. HHS is a serious, life-threatening medical emergency and has a higher mortality rate than DKA. Mortality is high not only because the metabolic changes are serious but also because people with diabetes are usually older and have other medical problems that either cause or are caused by HHS. The precipitating factors associated with

MULTISYSTEM EFFECTS OF DIABETES MELLITUS



Early manifestations
- Type 1 DM
  - Polyuria
  - Polydipsia
  - Polyphagia
  - Weight loss
  - Glycosuria
  - Fatigue
- Type 2 DM
  - Polyuria
  - Polydipsia
  - Blurred vision

Progressive complications
- Hyperglycaemia
  - Diabetic ketoacidosis
  - Hyperglycaemic hyperosmolar non-ketotic coma
- Hypoglycaemia

Late complications

Neurological
- Somatic neuropathies
  - Paresthaesias
  - Pain
  - Loss of cutaneous sensation
  - Loss of fine motor control
- Visceral neuropathies
  - Sweating dysfunction
  - Pupillary constriction
  - Fixed heart rate
  - Constipation
  - Diarrhoea
  - Incomplete bladder emptying
  - Sexual dysfunction

Sensory
- Diabetic retinopathy
- Cataracts
- Glaucoma

Cardiovascular
- Orthostatic hypotension
- Accelerated atherosclerosis
- Cerebrovascular disease (stroke)
- Coronary artery disease (myocardial infarction)
- Peripheral vascular disease
- Blood viscosity and platelet disorders

Renal
- Hypertension
- Albuminuria
- Oedema
- Chronic renal failure

Musculoskeletal
- Joint contractures

Integumentary
- Foot ulcers
- Gangrene of the feet
- Atrophic changes

Immune system
- Impaired healing
- Chronic skin infections
- Periodontal disease
- Urinary tract infections
- Lung infections
- Vaginitis

TABLE 19.4 DKA, HHS and hypoglycaemia comparison

		DKA	HHS	HYPOGLYCAEMIA
Diabetes type		Primary type 1	Type 2	Both
Onset		Slow	Slow	Rapid
Cause		↓ Insulin	↓ Insulin	↑ Insulin
		Infection	Older age	Omitted meal/snack
				Error in insulin dose
Risk factors		Surgery	Surgery	Surgery
		Trauma	Trauma	Trauma
		Illness	Illness	Illness
		Omitted insulin	Dehydration	Exercise
		Stress	Medications	Medications
			Dialysis	Lipodystrophy
			Hyperalimentation	Renal failure
				Alcohol intake
Assessments	Skin	Flushed; dry; warm	Flushed; dry; warm	Pallor; moist; cool
	Perspiration	None	None	Profuse
	Thirst	Increased	Increased	Normal
	Breath	Fruity	Normal	Normal
	Vital signs	BP ↓	BP ↓	BP ↓
		P ↑	P ↑	P ↑
		R Kussmaul's	R normal	R normal
	Mental status	Confused	Lethargic	Anxious; restless
	Thirst	Increased	Increased	Normal
	Fluid intake	Increased	Increased	Normal
	Gastrointestinal effects	Nausea/vomiting; abdominal pain	Nausea/vomiting; abdominal pain	Hunger
	Fluid loss	Moderate	Profound	Normal
	Level of consciousness	Decreasing	Decreasing	Decreasing
	Energy level	Weak	Weak	Fatigue
	Other	Weight loss	Weight loss	Headache
		Blurred vision	Malaise	Altered vision
			Extreme thirst	Mood changes
			Seizures	Seizures
Laboratory findings	Blood glucose	> 16.7 mmol/L	> 33.3 mmol/L	< 2.8 mmol/L
	Plasma ketones	Increased	Normal	Normal
	Urine glucose	Increased	Increased	Normal
	Urine ketones	Increased	Normal	Normal
	Serum potassium	Abnormal	Abnormal	Normal
	Serum sodium	Abnormal	Abnormal	Normal
	Serum chloride	Abnormal	Abnormal	Normal
	Plasma pH	< 7.3	Normal	Normal
	Osmolality	> 340 mmol/kg	> 340 mmol/kg	Normal
Treatment		Insulin	Insulin	Glucagon
		Treatment	Intravenous fluids	Rapid-acting carbohydrate
		Intravenous fluids Electrolytes	Electrolytes	Intravenous solution of 50% glucose

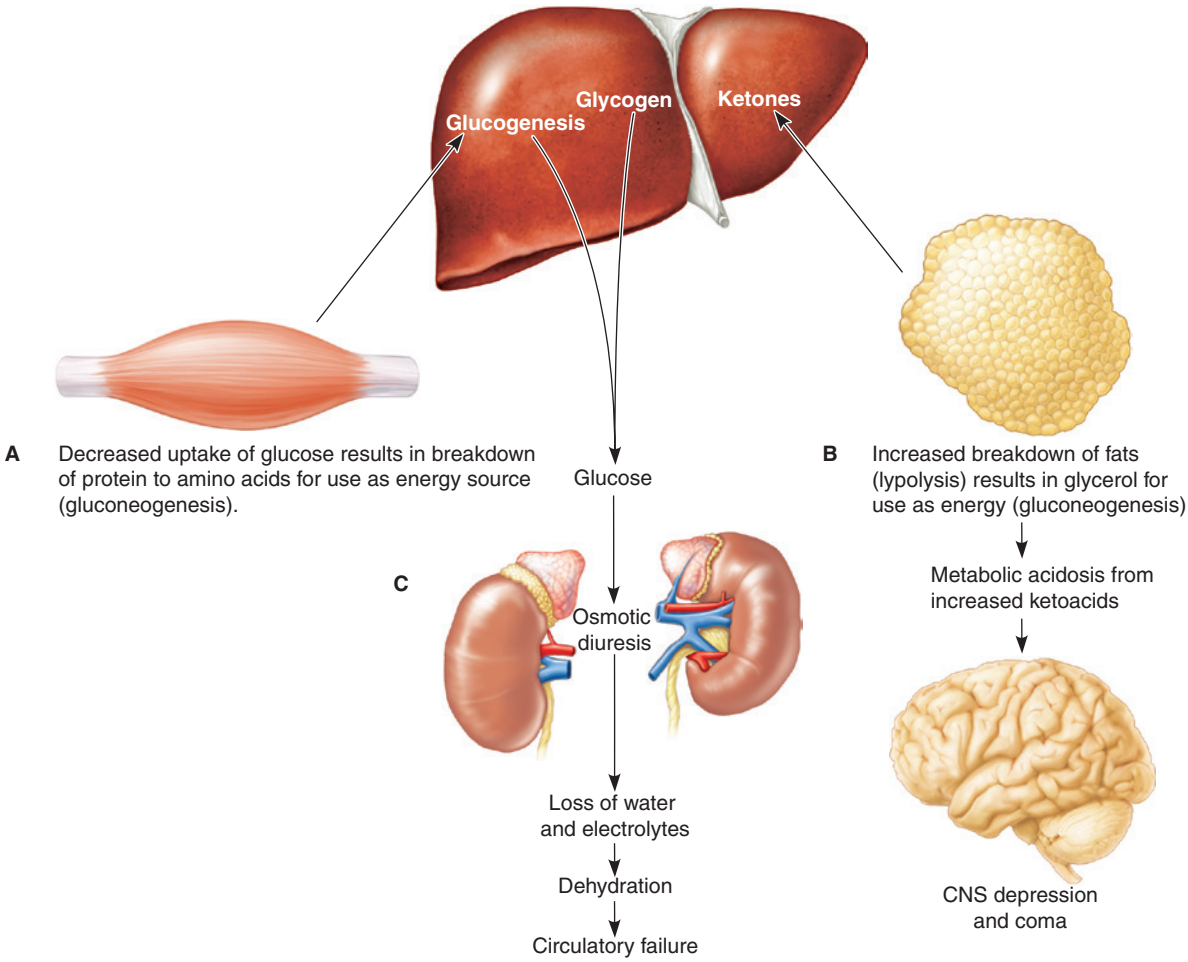


FIGURE 19.4 ■ In type I diabetes mellitus, without adequate insulin, muscle (*A*) and fat (*B*) cells are metabolised to provide sources of energy. Amino acids from skeletal muscle are converted to glucose in the liver; glycerol from fat cells is converted to glucose and fatty acids (ketoacids) which cause CNS depression and coma. Increased glucose (*C*) causes osmotic diuresis, leading to dehydration and decreased circulatory volume. These processes create the symptoms of diabetic ketoacidosis (DKA). The symptoms can be reversed with intravenous insulin to lower blood glucose. Blood pressure is raised to prevent circulatory failure by administering intravenous fluids; electrolytes are monitored and corrected

HHS include infection, therapeutic agents, therapeutic procedures, acute and chronic illness (see Box 19.10). The most common precipitating factor is infection. The manifestations of this disorder may be slow to appear, with onset ranging from 24 hours to 2 weeks. The manifestations are initiated by hyperglycaemia, which causes increased urine output. With increased output, plasma volume decreases and glomerular filtration rate drops. As a result, glucose is retained and water is lost. Glucose and sodium accumulate in the blood and increase serum osmolarity.

Serum hyperosmolarity results in severe dehydration, reducing intracellular water in all tissues, including the brain. The person has dry skin and mucous membranes, extreme thirst and altered levels of consciousness (progressing from lethargy to coma). Neurological deficits may include hyperthermia, motor and sensory impairment, positive Babinski's sign and seizures. *Metabolic acidosis is not part of the pathology; despite elevated blood glucose, sufficient insulin is present to prevent metabolism of fats with the resulting fatty acids and ketones of DKA.* Treatment is directed towards correcting fluid and electrolyte imbalances, lowering blood glucose levels with insulin and treating underlying conditions.

INTERPROFESSIONAL CARE

Treatment of hyperglycaemia

DKA

DKA requires immediate medical attention. Admission to the hospital is appropriate when the person has blood glucose of greater than 14 mmol/L, a decreasing pH and ketones in the urine. Priority management include assessment and management of the person's airway, breathing and circulation. Consider intubation and ventilation if required, administer oxygen if the person is hypoxic and insert a nasogastric tube if the person has low level of consciousness and is vomiting. Early

BOX 19.10 Factors associated with hyperosmolar hyperglycaemic state

Therapeutic agents
- Glucocorticoids
- Diuretics
- Beta-adrenergic blocking agents
- Immunosuppressants
- Chlorpromazine
- Diazoxide

Acute illness
- Infection
- Gangrene
- Urinary infection
- Burns
- Gastrointestinal bleeding
- Myocardial infarction
- Pancreatitis
- Stroke

Therapeutic procedures
- Peritoneal dialysis
- Haemodialysis
- Hyperosmolar alimentation (oral or parenteral)
- Surgery

Chronic illness
- Renal disease
- Cardiac disease
- Hypertension
- Previous stroke
- Alcoholism

assessment and correction of dehydration is essential. In the first 12 hours of treatment, adults usually require 8 to 10 L of intravenous fluid to replace losses from polyuria and vomiting (Lehne, 2012; Oakes & Cole, 2007). The initial fluid replacement may be accomplished by administering 0.9% saline solution at a rate of 500 to 1000 mL/h. After 2 to 3 hours (or when blood pressure and perfusion is returning to normal), the administration of 0.45% saline at 200 to 500 mL/h may continue for several more hours. When the blood glucose levels reach 14 to 16 mmol/L, dextrose is added to prevent rapid decreases in glucose; hypoglycaemia could result in fatal cerebral oedema.

Regular insulin is used in the management of DKA and may be given by various routes, depending on the severity of the condition. Mild ketosis may be treated with subcutaneous insulin, whereas severe ketosis requires intravenous insulin infusion. Nursing responsibilities for the person receiving intravenous insulin are described in the 'Medication administration' box below.

The electrolyte imbalance of primary concern is depletion of body stores of potassium. Initially, serum potassium levels may be normal, but they decrease during treatment. In DKA (and from rehydration), the body loses potassium from increased urinary output, acidosis, catabolic state and vomiting or diarrhoea. Potassium replacement is begun early in the course of treatment, usually by adding potassium to the rehydration fluids. Replacement is essential for preventing cardiac arrhythmias secondary to hypokalaemia. Cardiac rhythms and potassium levels must be monitored every 2 to 4 hours or more often, according to the severity of the person's condition. Often continuous cardiac monitoring is necessary.

HHS

HHS is a serious, life-threatening metabolic condition. The person admitted to the critical care unit for treatment typically manifests blood glucose levels over 40 mmol/L, increased serum osmolarity and altered levels of consciousness or seizures. Treatment is similar to that of DKA: correcting fluid and electrolyte imbalances and providing insulin to lower hyperglycaemia. In general, treatment modalities include the following:
- establishing and maintaining adequate ventilation
- correcting hypovolaemic shock with adequate intravenous fluids
- instituting nasogastric suction or gastric drainage if comatose to prevent aspiration
- maintaining fluid volume with intravenous isotonic or colloid solutions
- administering potassium intravenously to replace losses
- administering insulin to reduce blood glucose, usually discontinuing administration when blood glucose levels reach 14 mmol/L. (Because ketosis is not present, there is no need to continue insulin, as with DKA.)

MEDICATION ADMINISTRATION Intravenous insulin

GENERAL GUIDELINES

- Regular insulin may be given undiluted directly into the vein or through a Y-tube or three-way stopcock.
- Insulin infusion is usually diluted in 0.9% saline.

Nursing responsibilities

- Monitor blood glucose levels hourly.
- Infuse the insulin solution separately from the hydration solution.
- Flush or prime the intravenous tubing with 50 mL of insulin mixed with normal (0.9%) saline solution to saturate binding sites on the tubing before administering the insulin to the person; this step increases the amount of insulin delivered over the first few hours.
- Do not discontinue the intravenous infusion until subcutaneous administration of insulin is resumed.
- Monitor for manifestations of hypoglycaemia.
- Ensure that glucagon and/or dextrose 50% is readily available as an antidote for insulin overdose (also known as severe hypoglycaemia).
- Follow strict guidelines and protocols in relation to the administration, maintenance and titration of intravenous insulin infusions.

Hypoglycaemia

Hypoglycaemia (low blood glucose levels) is common in people with type 1 DM and occasionally occurs in people with type 2 DM who are treated with oral hypoglycaemic agents. This condition is often called insulin shock, **insulin reaction** or 'the lows' in people with type 1 DM. Hypoglycaemia results primarily from a mismatch between insulin intake (e.g. an error in insulin dose), physical activity and carbohydrate availability (e.g. omitting a meal). The intake of alcohol and drugs such as chloramphenicol (Chloromycetin), warfarin (Coumadin), monoamine oxidase inhibitors, probenecid (Benemid), salicylates and sulfonamides can also cause hypoglycaemia.

The manifestations of hypoglycaemia (see box below) result from a compensatory autonomic nervous system (ANS) response coupled with an impaired cerebral function due to a decrease in glucose available for use by the brain. The manifestations vary, particularly in older adults. The onset is sudden and blood glucose is usually less than 2.5 to 3.3 mmol/L. Severe hypoglycaemia may cause death.

People who have had type 1 DM for 4 or 5 years fail to secrete glucagon in response to a decrease in blood glucose. They then depend on adrenaline to serve as a counter-regulatory response to hypoglycaemia. However, this compensatory response can become absent or blunted. The person then develops a syndrome called *hypoglycaemia unawareness*. The person does not experience symptoms of hypoglycaemia even though it is present. Because treatment is not initiated in the absence of symptoms, the person is likely to have episodes of severe hypoglycaemia.

INTERPROFESSIONAL CARE

Treatment of hypoglycaemia

MILD HYPOGLYCAEMIA When mild hypoglycaemia occurs, immediate treatment is necessary. People experiencing hypoglycaemia should take about 15 g of a rapid-acting or simple sugar. This amount of sugar is found, for example, in three glucose tablets, ½ cup of fruit juice or a regular soft drink, 250 mL of skim milk, five Life Savers® sweets, three large marshmallows or 3 teaspoons of sugar or honey. Sugar should not be added to fruit juice. Adding sugar to the fruit sugar already in the juice could cause a rapid rise in blood glucose, with persistent hyperglycaemia.

People with diabetes should have some source of carbohydrate readily available at all times so that hypoglycaemic symptoms can be quickly reversed. A meal, including complex carbohydrates, should be consumed immediately so that hypoglycaemia does not re-occur. If hypoglycaemia occurs more than two or three times a week, the diabetes management plan should be adjusted.

SEVERE HYPOGLYCAEMIA People with diabetes who have severe hypoglycaemia are often hospitalised. The criteria for hospitalisation are one or more of the following:

- Blood glucose is less than 3.0 mmol/L and the prompt treatment of hypoglycaemia has not resulted in recovery of sensorium.
- The person has coma, seizures or altered behaviour.
- The hypoglycaemia has been treated, but a responsible adult cannot be with the person for the following 12 hours.
- The hypoglycaemia was caused by a sulfonylurea drug.

If the person is conscious and alert, 10 to 15 g of an oral carbohydrate may be given. If the person has altered levels of consciousness, parenteral glucose or glucagon is administered.

Glucose is administered intravenously as a 25–50% solution, usually at a rate of 10 mL over 1 minute by intravenous push or bolus, as the most rapid method of increasing blood glucose levels (Diabetes Australia, 2012).

Glucagon is an antihypoglycaemic agent that raises blood glucose by promoting the conversion of hepatic glycogen to glucose. It is used in severe insulin-induced hypoglycaemia and may be given in the recommended dose of 1 mg by the subcutaneous, intramuscular or intravenous routes. Glucagon has a short period of action; an oral (if the person is conscious) or intravenous carbohydrate should be administered following the glucagon to prevent a recurrence of hypoglycaemia. If the person has been unconscious, glucagon may cause vomiting when consciousness returns.

MANIFESTATIONS Hypoglycaemia

MANIFESTATIONS CAUSED BY RESPONSES OF THE AUTONOMIC NERVOUS SYSTEM

- Hunger
- Shakiness
- Nausea
- Irritability
- Anxiety
- Rapid pulse
- Pale, cool skin
- Hypotension
- Sweating

MANIFESTATIONS CAUSED BY IMPAIRED CEREBRAL FUNCTION

- Strange or unusual feelings
- Slurred speech
- Headache
- Blurred vision
- Decreasing levels of consciousness
- Difficulty in thinking
- Inability to concentrate
- Seizures
- Change in emotional behaviour
- Coma

Chronic complications

Alterations in the cardiovascular system

The macrocirculation (large blood vessels) in people with diabetes undergoes changes due to atherosclerosis; abnormalities in platelets, red blood cells and clotting factors; and changes in arterial walls. It has been established that atherosclerosis (clogging and hardening of the blood vessels) has an increased incidence and earlier age of onset in people with diabetes (although the reason is unknown). Other risk factors that contribute to the development of macrovascular disease of diabetes are hypertension, hyperlipidaemia, cigarette smoking and obesity. Alterations in the vascular system increase the risk of the long-term complications of coronary artery disease, cerebral vascular disease and peripheral vascular disease.

Alterations in the microcirculation in the person with diabetes involve structural defects in the basement membrane of smaller blood vessels and capillaries. (The basement membrane is the structure that supports and serves as the boundary around the space occupied by epithelial cells.) These defects cause the capillary basement membrane to thicken, eventually resulting in decreased tissue perfusion. Changes in basement membranes are believed to be due to one or more of the following: the presence of increased amounts of sorbitol (a substance formed as an intermediate step in the conversion of glucose to fructose), the formation of abnormal glycoproteins, or problems in the release of oxygen from haemoglobin (Porth & Matfin, 2009). The effects of alterations in the microcirculation affect all body tissues but are seen primarily in the eyes and the kidneys.

Coronary artery disease

Coronary artery disease is a major risk factor in the development of myocardial infarction in people with diabetes, especially in the middle to older adult with type 2 DM. Coronary artery disease is the most common cause of death for people with diabetes in Australia (ABS, 2015). Indigenous people have a higher incidence of death due to diabetes mellitus and ischaemic heart disease than non-Indigenous Australians (ABS, 2012). People with diabetes who have myocardial infarction are more prone to develop congestive heart failure as a complication of the infarction and are also less likely to survive in the period immediately following the infarction. (Myocardial infarction is discussed in Chapter 29.)

Hypertension

Hypertension (blood pressure ≥ 140/90 mmHg) is a common complication of DM. It affects 20–60% of all people with diabetes and is a major risk factor for cardiovascular disease and microvascular complications such as retinopathy and nephropathy. Hypertension may be reduced by weight loss, exercise, and decreasing sodium intake and alcohol consumption. If these methods are not effective, treatment with antihypertensive medications is necessary.

Stroke (cerebrovascular accident)

People with diabetes, especially older adults with type 2 DM, are 2 to 6 times more likely to have a stroke. Atherosclerosis of the cerebral vessels develops at an earlier age and is more extensive in people with diabetes due to high glucose levels over time leading to increased fat deposits in the blood vessel walls (Porth & Matfin, 2009; National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) and National Institutes of Health (NIH), 2011). Around 75% of people with diabetes will die from cardiovascular disease or stroke (Diabetes NSW, 2015).

The manifestations of impaired cerebral circulation are often similar to those of hypoglycaemia or HHS: blurred vision, slurred speech, weakness and dizziness. People with these manifestations have potentially life-threatening health problems and require constant medical attention.

Peripheral vascular disease

Peripheral vascular disease of the lower extremities accompanies both types of DM, but the incidence is greater in people with type 2 DM. Atherosclerosis of vessels in the legs of people with diabetes begins at an earlier age, advances more rapidly and is equally common in both men and women. Impaired peripheral vascular circulation leads to peripheral vascular insufficiency with intermittent claudication (pain) in the lower legs and ulcerations of the feet. Occlusion and thrombosis of large vessels and small arteries and arterioles, as well as alterations in neurological function and infection, result in gangrene (necrosis or the death of tissue). Gangrene from diabetes is the most common cause of non-traumatic amputations of the lower leg. In people with diabetes, dry gangrene is most common, manifested by cold, dry, shrivelled and black tissues of the toes and feet. The gangrene usually begins in the toes and moves proximally into the foot.

Diabetic retinopathy

Diabetic retinopathy is the name for the changes in the retina that occur in the person with diabetes. The retinal capillary structure undergoes alterations in blood flow, leading to retinal ischaemia and a breakdown in the blood–retinal barrier. Retinopathy is a major long-term complication of diabetes. It affects about 1 in 4 people with diabetes (Vision Australia Foundation, 2009). The development of retinopathy is strongly related to the length of time diabetes has been present and the degree of blood glucose control. Regular eye checks and treatment can help prevent retinopathy.

Retinopathy has three stages:

- *Stage I:* non-proliferative retinopathy. Dilated veins, microaneurysms, oedema of the macula and the presence of exudates characterise this stage.
- *Stage II:* pre-proliferative retinopathy. Retinal ischaemia causes infarcts of the nerve fibre layer, with characteristic 'cotton wool' patches on the retina. Shunts form between occluded and patent vessels.
- *Stage III:* proliferative retinopathy. As fibrous tissue and new vessels form in the retina or optic disc, traction on the vitreous humor may cause haemorrhage or retinal detachment.

After 20 years of diabetes, almost all people with type 1 DM and more than 60% of people with type 2 DM will have some degree of retinopathy, in most cases without vision loss (Wong et al., 2006). If exudate, oedema, haemorrhage or ischaemia

occurs near the fovea, the person experiences visual impairment at any stage. In addition, the person with diabetes is at increased risk of developing cataracts (opacity of the lens) as a result of increased glucose levels within the lens itself. Screening for retinopathy is important, as laser photocoagulation surgery has proven beneficial in preventing loss of vision. Indigenous Australians have an increased incidence and rate of new cases of diabetes-associated retinopathy; therefore, this population needs appropriate resources allocated and to be closely monitored and treated (Landers, Henderson & Craig, 2012).

Diabetic nephropathy

Diabetic nephropathy is a disease of the kidneys characterised by the presence of albumin in the urine, hypertension, oedema and progressive renal insufficiency. Diabetes is the fastest-growing cause of kidney failure. It is the leading cause of end-stage renal disease (ESRD). About 30% of people with diabetes will develop kidney disease (International Diabetes Federation, 2009).

Despite research, the exact pathological origin of diabetic nephropathy is unknown; it has been established, however, that thickening of the basement membrane of the glomeruli eventually impairs renal function. It has been suggested that an increased intracellular concentration of glucose supports the formation of abnormal glycoproteins in the basement membrane and mesangium. The accumulation of these large proteins stimulates glomerulosclerosis (fibrosis of the glomerular tissue). Glomerulosclerosis thickens the basement membrane and simultaneously makes it functionally leaky, allowing large molecules such as protein to be lost in the urine. *Kimmelstiel–Wilson syndrome* is a type of glomerulosclerosis found only in people with diabetes. In advanced nephropathy, tubular atrophy occurs and end-stage renal disease results. (Renal failure is discussed in Chapter 27.)

The first indication of nephropathy is **microalbuminuria**, a low but abnormal level of albumin in the urine. Without specific interventions, people with type 1 DM with sustained microalbuminuria will develop overt nephropathy, accompanied by hypertension, over a period of 10 to 15 years. People with type 2 DM often have microalbuminuria and overt nephropathy shortly after diagnosis, because the diabetes has often been present but undiagnosed for many years. Because the hypertension accelerates the progress of diabetic nephropathy, aggressive antihypertensive management should be instituted. Management includes control of hypertension with angiotensin-converting-enzyme (ACE) inhibitors such as captopril (Capoten), weight loss, reduced salt intake and exercise.

Alterations in the peripheral and autonomic nervous systems

Peripheral and visceral neuropathies are disorders of the peripheral nerves and the autonomic nervous system. In people with diabetes, these disorders are often called **diabetic neuropathies**. The aetiology of diabetic neuropathies involves: (1) a thickening of the walls of the blood vessels that supply nerves, causing a decrease in nutrients; (2) demyelinisation of the Schwann cells that surround and insulate nerves, slowing nerve conduction; and (3) the formation and accumulation of sorbitol within the Schwann cells, impairing nerve conduction. The manifestations depend on the locations of the lesions.

PERIPHERAL NEUROPATHIES

The peripheral neuropathies (also called *somatic neuropathies*) include polyneuropathies and mononeuropathies. *Polyneuropathies*, the most common type of neuropathy associated with diabetes, are bilateral sensory disorders. The manifestations appear first in the toes and feet and progress upward. The fingers and hands may also be involved, but usually only in the later stages of diabetes. The manifestations of polyneuropathy depend on the nerve fibres involved.

The person with polyneuropathy commonly has distal paraesthesias (a subjective feeling of a change in sensation, such as numbness or tingling); pain described as aching, burning or shooting; and feelings of cold feet. Other manifestations may include impaired sensations of pain, temperature, light touch, two-point discrimination and vibration. There is no specific treatment for polyneuropathy.

Mononeuropathies are isolated peripheral neuropathies that affect a single nerve. Depending on the nerve involved, manifestations may include the following:

- palsy of the third cranial (oculomotor) nerve, with headache, eye pain and an inability to move the eye up, down or medially
- radiculopathy, with pain over a dermatome and loss of cutaneous sensation, most often located in the chest
- diabetic femoral neuropathy, with motor and sensory deficits (pain, weakness, areflexia) in the anterior thigh and medial calf
- entrapment or compression of the medial nerve at the wrist, resulting in carpal tunnel syndrome, with pain and weakness of the hand; the ulnar nerve at the elbow, with weakness and loss of sensation over the palmar surface of the fourth and fifth fingers; and the peroneal nerve at the head of the fibula, with foot drop.

VISCERAL NEUROPATHIES

The visceral neuropathies (also called autonomic neuropathies) cause various manifestations, depending on the area of the ANS involved. These neuropathies may include the following:

- Sweating dysfunction, with an absence of sweating (*anhydrosis*) on the hands and feet and increased sweating on the face or trunk.
- Abnormal pupillary function, most commonly seen as constricted pupils that dilate slowly in the dark.
- Cardiovascular dysfunction, resulting in such abnormalities as a fixed cardiac rate that does not change with exercise, postural hypotension, and a failure to increase cardiac output or vascular tone with exercise.
- Gastrointestinal dysfunction, with changes in upper gastrointestinal motility (*gastroparesis*) resulting in dysphagia, anorexia, heartburn, nausea and vomiting, and altered blood glucose control. Constipation is one of the most common gastrointestinal symptoms associated with diabetes, possibly a result of hypomotility of the bowel. Diabetic diarrhoea is not as common, but it does occur and is often

associated with faecal incontinence during sleep due to a defect in internal sphincter function.
- Genitourinary dysfunction, resulting in changes in bladder function and sexual function. Bladder function changes include an inability to empty the bladder completely, loss of sensation of bladder fullness and an increased risk of urinary tract infections. Sexual dysfunctions in men include ejaculatory changes and impotence. Sexual dysfunctions in women include changes in arousal patterns, vaginal lubrication and orgasm. Alterations in sexual function in people with diabetes are the result of both neurological and vascular changes.

Mood alterations

People with DM, both type 1 and type 2, endure the chronic strains of living with complex self-care and are at a somewhat increased risk of depression, which can negatively affect management of DM. Treating depression has been associated with better control of serum glucose, so screening for depression is an important part of assessing the individual's ability to manage the disease. Diabetes Australia (2012) reports the risk of depression more than doubles in people with diabetes. Nurses can assist depressed people by correcting misconceptions about depression, identifying individual strengths in managing diabetes, acknowledging negative feelings that may be expressed and suggesting problem-solving behaviours to better manage the disease.

Increased susceptibility to infection

The person with diabetes has an increased risk of developing infections. A combination of vascular insufficiency, neuropathies and systemic and local immunological dysfunction as well as hyperglycaemia and altered neutrophil function are believed to play a role in higher infection risks in people with diabetes (Porth & Matfin, 2009; Weintrob & Sexton, 2012).

The person with diabetes may have sensory deficits resulting in inattention to trauma and vascular deficits that decrease circulation to the injured area; as a result, the normal inflammatory response is diminished and healing is slowed. Nephrosclerosis and inadequate bladder emptying with retention of urine predispose the person with diabetes to pyelonephritis and urinary tract infections. Bacterial and fungal infections of the skin, nails and mucous membranes are common. Tuberculosis is more prevalent in people with diabetes than in the general population. Hospitalised people with a blood glucose value greater than 12 mmol/L have higher infection rates (AIHW, 2008).

Periodontal disease

Although periodontal disease does not occur more often in people with diabetes, it does progress more rapidly, especially if the diabetes is poorly controlled. It is believed to be caused by microangiopathy, with changes in vascularisation of the gums. As a result, gingivitis (inflammation of the gums) and periodontitis (inflammation of the bone underlying the gums) occur.

Complications involving the feet

The high incidence of both amputations and problems with the feet in people with diabetes is the result of angiopathy, neuropathy and infection. People with diabetes are at high risk of amputation of a lower extremity, with increased risk in those who have had DM for more than 10 years, are male, have poor glucose control or have cardiovascular, retinal or renal complications.

MANIFESTATIONS Peripheral vascular disease

- Loss of hair on lower leg, feet and toes
- Atrophic skin changes: shininess and thinning
- Cold feet
- Feet and ankles darker than leg
- Dependent rubor, blanching on elevation
- Thick toenails
- Diminished or absent pulses
- Nocturnal pain
- Pain at rest, relieved by standing or walking
- Intermittent claudication
- Patchy areas of gangrene on feet and toes

Vascular changes in the lower extremities of the person with diabetes result in arteriosclerosis. Diabetes-induced arteriosclerosis tends to occur at an earlier age, occurs equally in men and women, is usually bilateral and progresses more rapidly. The blood vessels most often affected are located below the knee. Blockages form in the large, medium and small arteries of the lower legs and feet. Multiple occlusions with decreased blood flow result in the manifestations of peripheral vascular disease (see the accompanying box). Peripheral vascular disease is discussed in Chapters 28 and 31.

Diabetic neuropathy of the foot produces multiple problems. Because the sense of touch and perception of pain are absent, the person with diabetes may have some type of foot trauma without being aware of it. The person thus is at increased risk of trauma to tissues of the feet, leading to ulcer development. Infections commonly occur in traumatised or ulcerated tissue (see Figure 19.5).

Despite the many potential sources of foot trauma in the person with diabetes, the most common are cracks and fissures caused by dry skin or infections such as athlete's foot, blisters caused by improperly fitting shoes, pressure from stockings or shoes, ingrown toenails and direct trauma (cuts, bruises or burns). It is important to remember that the person with diabetic neuropathy who has lost the perception of pain may not be aware that these injuries have occurred. In addition, when a part of the body loses sensation, the person tends to dissociate from or ignore the part, so that an injury may go unattended for days or weeks. The injury may even be forgotten entirely.

Foot lesions usually begin as a superficial skin ulcer. In time, the ulcer extends deeper into muscles and bone, leading to abscess or osteomyelitis. Gangrene can develop on one or more toes; if untreated, the whole foot eventually becomes gangrenous. (Care of the feet, an essential part of health education for the person and family, is discussed later in the chapter.)

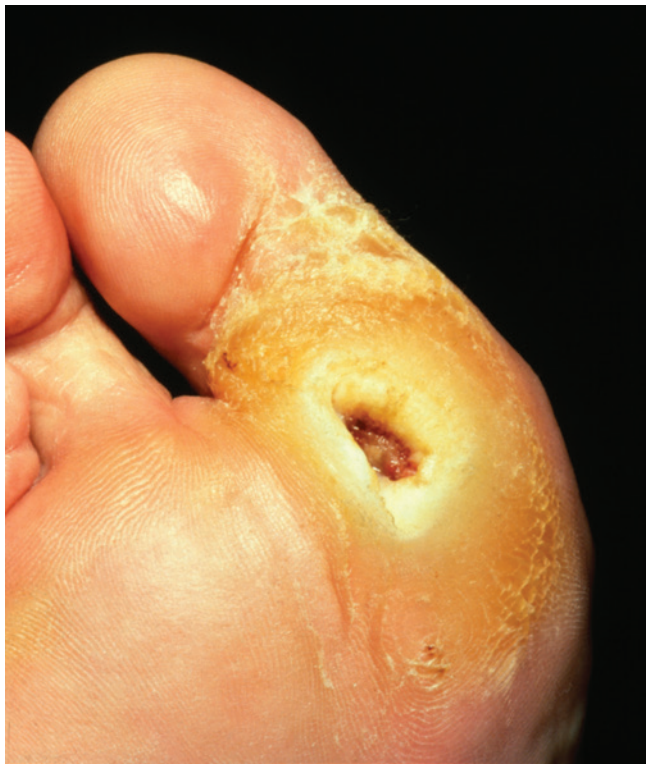


FIGURE 19.5 ■ Ulceration following trauma in the foot of the person with diabetes

Source: Harry Przekop, Medichrome/The Stock Shop, Inc.

Nursing care

The responses of the person with diabetes to the illness are often complex and individual, involving multiple body systems. Assessments, planning and implementation differ for the person with newly diagnosed diabetes, the person with long-term diabetes and the person with acute complications of diabetes. The plan of care and content of teaching also differ according to the type of diabetes, the person's age and culture, and their intellectual, psychological and social resources. However, nursing care often focuses on teaching the person to manage the illness. The 'Translation to practice' box below describes a study of quality of life in people with diabetes.

Health promotion

Health promotion activities primarily focus on preventing the complications of diabetes. Prevention of the disease has not been determined, although it is recommended that all people should prevent or decrease excess weight, follow a sensible and well-balanced diet, and maintain a regular physical exercise program. Blood glucose screening at 1–3-year intervals beginning at age 45 is recommended for those in the high-risk groups. These same activities, when combined with medications and self-monitoring, are also beneficial in reducing the onset of complications.

Assessment

The following data are collected through the health history and physical examination (see Chapter 17). Further focused assessments are described with nursing interventions in the following text. When assessing the older person, be aware of normal ageing changes in all body systems that may alter interpretation of findings.

- *Health history:* family history of diabetes; history of hypertension or other cardiovascular problems; history of any change in vision (e.g. blurring) or speech, dizziness, numbness or tingling in hands or feet; pain when walking; frequent voiding; change in weight, appetite, infections and healing; problems with gastrointestinal function or urination; or altered sexual function.
- *Physical assessment:* height/weight ratio, vital signs, visual acuity, cranial nerves, sensory ability (touch, hot/cold, vibration) of extremities, peripheral pulses, skin and mucous membranes (hair loss, appearance, lesions, rash, itching, vaginal discharge).

Nursing diagnoses and interventions

Although many different nursing diagnoses are appropriate for the person with diabetes, those discussed in this section address problems with skin integrity, infection, injury, sexuality, coping and health maintenance. The goals of care are to maintain function, prevent complications and teach self-management. See the accompanying 'Nursing care plan' for more information.

Risk of impaired skin integrity related to diabetic neuropathies

The person with diabetes is at increased risk of altered skin integrity as a result of decreased or absent sensation from neuropathies, decreased tissue perfusion from cardiovascular complications and infection. Impaired skin and tissue integrity, with resultant gangrene, is especially common in the feet and lower extremities.

Conduct baseline and ongoing assessments of the feet, including:

- musculoskeletal assessment that includes foot and ankle joint range of motion, bone abnormalities (bunions, hammer toes, overlapping digits), gait patterns, use of assistive devices for walking and abnormal wear patterns on shoes
- neurological assessment that includes sensations of touch and position, pain and temperature
- vascular examination that includes assessment of lower extremity pulses, capillary refill, colour and temperature of skin, lesions and oedema
- hydration status, including dryness or excessive perspiration
- lesions, fissures between toes, corns, calluses, plantar warts, ingrown or overgrown toenails, redness over pressure points, blisters, cellulitis or gangrene.

People with diabetes are at significant risk of lower-extremity gangrene. Peripheral neuropathies may result in alterations in the perception of pain, loss of deep tendon reflexes, loss of cutaneous pressure and position sensation, foot drop, changes in the shape of the foot and changes in bones and joints. Peripheral vascular disease may cause intermittent claudication, absent pulses, delayed venous filling on

TRANSLATION TO PRACTICE Evidence-based practice: diabetes: a national health priority

Diabetes is endemic in Australia and this chronic condition creates challenges for the individual, carers and healthcare providers. For the sufferers of diabetes and their families, the condition poses significant emotional, psychological and financial stress (Colagiuri et al., 2009). For the healthcare system, the significance is in the rising costs of treatment and the challenge of prevention of type 2 diabetes mellitus in particular. Sufferers of type 1 diabetes mellitus are generally young people who need support and education to transition to management of the condition as adults.

The consensus among experts is that patient education is pivotal to ensuring effective management of the condition and reducing healthcare costs (Callister, 2011; Colagiuri et al., 2009). The aim is to reduce the stressors to the individual and their carers, and to reduce the cost of healthcare services to both the sufferers of diabetes and the wider Australian community.

The Australian federal government has classified diabetes mellitus, in particular type 2 diabetes mellitus (which constitutes 85% of diagnosed cases), as a national health priority because of its significant impact on the health status and health economy of Australia (AIHW, 2011; Colagiuri, et al., 2009).

The *National Evidenced Based Guideline for Patient Education in Type 2 Diabetes* and the *National Evidence-based Clinical Care Guidelines for Type 1 Diabetes in Children, Adolescents and Adults* are evidence-based tools for healthcare providers in Australia to create a uniform support network for persons with diabetes and their carers.

Literature reviewed in these guidelines agree that the individual's active participation in goal setting and decision making is paramount to the success of management of diabetes and reducing complications which in turn would reduce the burden of cost to the healthcare system (Colagiuri et al., 2009). Dijkstra et al. (2006), Ragucci et al. (2005), Smart et al. (2009) and Wolfe et al. (2004) (as cited in Colagiuri et al., 2009) are examples of studies that identified the input of professionals such as pharmacists and dietitians to discuss medication management and lifestyle changes as a cost-effective method of delivery of information to the patient. The focus for type 1 diabetes mellitus patients was to support psychological and behaviour states of young children and adults in order to improve knowledge and self-management skills so as to transition to adulthood with less stress (Callister, 2011).

Diabetes is a multifaceted condition that requires a multidisciplinary approach to support the individual and carers. The nurse's involvement is to advocate for and support the individual in the management of diabetes. The role of the nurse is also pivotal to the prevention of type 2 diabetes.

IMPLICATIONS FOR NURSING

All teaching plans should be individualised and developed in collaboration with the person. Nurses often focus on diabetes management and control, especially compliance with prescribed regimens. Although people are sometimes termed 'non-compliant', this term conveys judgment about dependence. A focus on individualised education based on assessment of individual needs facilitates higher levels of compliance and improved self-care. It is important also to understand that self-control behaviours can change with variations in life and the disease itself, and that each person with diabetes responds uniquely to situations and interventions. Respecting and valuing what a person has learned from living with diabetes is critical to providing effective care.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. You are caring for two people with diabetes who are receiving home care for complications of long-term diabetes. One person follows the medical regimen faithfully; the other adapts it to his own schedule and needs. What differences can you identify in your own reaction to these two different people? How would these reactions affect your relationship with the people?
2. Imagine you have just been diagnosed with type 1 diabetes. Make a list of the questions you would have and the areas that would cause you the most difficulty in complying with your medical care.
3. How would you respond if the person tells you, 'Sometimes I eat whatever I want to for several days.'? What do you think this behaviour indicates?

elevation, dependent rubor and gangrene. Injuries, lesions and changes in skin hydration potentiate infections, delayed healing and tissue loss in the person with diabetes mellitus.

- Teach foot hygiene. Wash the feet daily with lukewarm water and mild hand soap; pat dry and dry well between the toes. Apply a very thin coat of lubricating cream if dryness is present (but not between the toes). *Proper hygiene decreases the chance of infection. Temperature receptors may be impaired, so the water should always be tested before use.*

CONSIDERATION FOR PRACTICE

Teach the person with diabetes to always test the water temperature in the shower or bath before stepping in.

- Discuss the importance of not smoking if the person smokes. *Nicotine in tobacco causes vasoconstriction, further decreasing the blood supply to the feet.*
- Discuss the importance of maintaining blood glucose levels through prescribed diet, medication and exercise. *Hyperglycaemia promotes the growth of microorganisms.*
- Conduct foot care teaching sessions as often as necessary (see the box below). Include information about proper shoe fit and composition, avoiding clothing or activities that decrease circulation to the feet, foot inspections, care of toenails and the importance of obtaining medical care for lesions. If the person has visual deficits, is obese or cannot reach the feet, teach the caregiver how to inspect and care for the feet. Feet should be inspected daily. *Foot care is a*

NURSING CARE PLAN A person with type 1 diabetes

Jim Meligrito, aged 24, is a third-year nursing student at a large university. Mr Meligrito also works 20 hours a week as an on-campus security guard. His working hours are 8 pm to midnight, 5 nights a week. He lives with his father, who is also a student. Neither of the two men likes to cook and they usually eat 'whatever is handy'. Mr Meligrito has smoked 8 to 10 cigarettes a day for 5 years. He was diagnosed with type 1 diabetes mellitus at age 12. Although his insulin dosage has varied, he currently takes a total of 52 units of insulin each day, 8 units of Humalog (insulin lispro) three times per day, before each meal, and 28 units of Lanctus (glargine insulin) at different times in the evenings or before bed. He monitors his blood glucose about three times a week. He feels that he is too busy for a regular exercise program and that he gets enough exercise at work and in weekend sports activities. He has not seen a healthcare provider for over a year.

One day during a 6-hour clinical laboratory in paediatrics, Mr Meligrito notices that he is urinating frequently, is thirsty and has blurred vision. He is also very tired but blames all his symptoms on drinking a couple of beers and having had only 4 hours' sleep the night before while studying for an examination, and the stress he has been under lately from his studies and work. When he remembers that he had forgotten to take his insulin that morning, he realises he must have hyperglycaemia but decides that he will be all right until he gets home in the afternoon. Around noon, he begins having abdominal pain, feels weak, has a rapid pulse and vomits. When he reports his physical symptoms to his clinical facilitator, she sends him immediately to the hospital emergency department, accompanied by another student.

ASSESSMENT

As soon as Mr Meligrito arrives at the emergency room, his blood glucose level is measured at 17.0 mmol/L. Urine samples and additional blood samples are sent to the laboratory for analysis. Blood glucose is 18.3 mmol/L, urine shows the presence of ketones, electrolytes are normal and pH is 7.1. His vital signs are as follows: T 37.2°C, P 140, R 28 and BP 102/52. An intravenous infusion of 1000 mL normal (0.9%) saline with 40 mmol of KCl is started at a rate of 400 mL/h. Intravenous regular insulin at 5 units/h (diluted in 0.9% saline) is begun. Hourly blood glucose monitoring is initiated. Mr Meligrito is nauseated and lethargic but remains oriented. Five hours later, he has a blood glucose level of 9.0 mmol/L and his pulse and blood pressure are normal. Now there are no ketones in the urine, his vital signs, including his glucose level, are within normal parameters and he has no signs of metabolic acidosis. He is discharged from the hospital after seeing the hospital's diabetes nurse educator. When he meets with the diabetes educator, he says that he no longer feels in control of the diabetes or his future goal to become an anaesthetic nurse.

DIAGNOSES

- *Powerlessness* related to a perceived lack of control of his diabetes due to present demands on time.
- *Deficient or poor knowledge* of self-management of diabetes.
- *Risk of ineffective role performance* related to uncertainty about capacity to achieve desired role as a registered nurse.

PLANNING

- Mutually establish specific and individualised short-term and long-term goals for self-management to control blood glucose.
- Assess his level of knowledge and establish his needs to develop a program of education in different aspects of diabetes.
- Determine the need and plan referrals to appropriate support health allies such as dietitian, educator, psychologist, counsellor, etc.

Expected outcomes

- Identify those aspects of diabetes that can be controlled and participate in making decisions about self-managing care.
- Demonstrate an understanding of diabetes self-management through planned medication, diet, exercise and blood glucose self-monitoring activities.
- Explore and clarify Mr Meligrito's perceptions of his role as a student nurse, verbalising his ability to meet his expectations.

IMPLEMENTATION

- Provide opportunities to express his feelings about himself and his illness.
- Explore perceptions of his own ability to control his illness and his future, and clarify these perceptions by providing information about resources and support groups.
- Facilitate decision-making abilities in self-managing his prescribed treatment regimen.
- Provide positive reinforcement for increasing involvement in self-care activities.
- Provide relevant learning activities about insulin administration, dietary management, exercise, self-monitoring of blood glucose and healthy lifestyle.

EVALUATION

After taking an active part in the weekly educational meetings for 2 months, Mr Meligrito has greatly enhanced his understanding of and compliance with self-management of his diabetes. He states that he finally understands how insulin, food and exercise affect his body, having previously thought they were 'just things I should do when I wanted to'. He decides to perform self-management activities 1 week at a time, rather than think too far into (and thereby feel overwhelmed by) the future. Both son and father have developed a workable meal schedule and weekly grocery list and they have begun eating breakfast and dinner together. Jim and a friend have arranged to walk 5 to 10 km three times a week on a hiking trail. To gain a sense of control over his illness, he has also worked out a schedule that allows time for university, work, healthcare and himself.

CRITICAL THINKING IN THE NURSING PROCESS

1 What is the pathophysiological basis for the changes in temperature, pulse, respirations and blood pressure that

(continued)

occurred on Mr Meligrito's admission to the hospital emergency department?
2 How can poor self-management of diabetes increase the risk of long-term complications?
3 Is powerlessness a common response to a chronic illness? Why or why not?
4 Consider that you are teaching Mr Meligrito and another person, Mr McDaniel (aged 75, newly diagnosed with type 2 DM). What components of your teaching plan would be the same and what components would be different?

REFLECTION ON THE NURSING PROCESS

1 List the aspects that you have learned from this scenario that you will apply to your future clinical practice when caring for a person with poorly controlled diabetes.
2 Detail how you would approach and communicate with a person who has poor management of their diabetes in order to provide advice and education.

priority in diabetes management to prevent serious problems. Many people with diabetes are unaware of lesions or injury until infection and compromised circulation are far advanced. The hows and whys of each component must be included in teaching. A variety of methods may be used, including demonstration, return demonstration, audiovisual aids and written lists. If the person is wearing shoes and socks, ask them to remove them to practise foot care effectively.

Risk of infection

The person with diabetes is at increased risk of infection; this is believed to be due to vascular insufficiency that limits the inflammatory response, neurological abnormalities that limit the awareness of trauma and a predisposition to bacterial and fungal infections.

- Use and teach meticulous handwashing. *Handwashing is the single most effective method for preventing the spread of infection.*
- Monitor for manifestations of infection: increased temperature, pain, malaise, swelling, redness, discharge, cough. *Early diagnosis and treatment of infections can control their severity and decrease complications.*
- Discuss the importance of skin care. Keep the skin clean and dry, using lukewarm water and mild soap. *People with diabetes are more prone to develop furuncles and carbuncles; the infection often increases the need for insulin.*

MEETING INDIVIDUALISED NEEDS Foot care teaching session

Buying and wearing shoes and stockings

- Shoes that allow 1 to 2 cm of toe room are best; there should be room for toes to spread out and wiggle. The lining and inside stitching should be smooth and the insole soft. The sole should be flexible and cushion the foot. The heel should fit snugly and the arch support should give good support.
- Do not wear open-toed shoes, sandals, high heels or thongs; they increase the risk of trauma.
- Buy shoes late in the afternoon, when feet are at their largest; always buy shoes that feel comfortable and do not need to be 'broken in'.
- Shoes made of natural fibres (leather, canvas) allow perspiration to escape.
- Check the shoes before each wearing for foreign objects, wrinkled insoles and cracks that might cause lesions.
- Stockings made of wool or cotton allow perspiration to dry.
- Do not wear garters, knee stockings or pantyhose; they may interfere with circulation.
- Wear insulated boots in the winter.

Inspecting the feet

- Check the feet daily for red areas, cuts, blisters, corns, calluses or cracks in the skin. Check between the toes for cracks or reddened areas.
- Check the skin of the feet for dry or damp areas.
- Use a mirror to check each sole and the back of each heel.
- If you are unable to inspect the feet daily, be sure that someone else does so.

Care of toenails

- Cut the toenails after washing, when they are softer and easier to trim.
- Cut the nails straight across with a nail clipper and smooth edges and corners with an emery board.
- Do not use razor blades to trim the toenails.
- If you are unable to see well or to reach the feet easily, have someone else trim the nails. If the nails are very thick or ingrown, if the toes overlap or if circulation is poor, get professional care from a podiatrist.

General information

- Never go barefoot. Wear slippers when leaving the bed during the night.
- Do not use commercial corn medicines or pads, chemicals (such as boric acid, iodine or hydrogen peroxide) or over-the-counter cortisone medications on the feet.
- Do not put heating pads, hot water bottles or ice packs on the feet. If the feet become cold at night, wear socks or use extra blankets.
- Do not allow the feet to become sunburned.
- Do not put tape on the feet.
- Do not sit with the legs crossed at the knees or ankles.

Clean, intact skin and mucous membranes are the first line of defence against infection.

- Teach dental health measures:
  - Obtain a dental examination every 4 to 6 months.
  - Maintain careful oral hygiene, which includes brushing the teeth with a soft toothbrush and fluoridated toothpaste at least twice a day and flossing as recommended.
  - Be aware of the symptoms requiring dental care: bad breath; unpleasant taste in the mouth; bleeding, red or sore gums; and tooth pain.
  - If dental surgery is necessary, monitor for need to make adjustments in insulin. All people with diabetes need to be taught proper oral hygiene, and about the risk of periodontal disease and the importance of obtaining dental care for symptoms of oral or dental problems.
- Teach women with diabetes the symptoms and preventive measures for vaginitis caused by *Candida albicans*. The symptoms are an odourless, white or yellow cheese-like discharge and itching. Sexual transmission is unlikely, but discomfort may cause the person to avoid sexual activity. *Diabetes is a predisposing factor for* Candida albicans *vaginitis, the most common form of vaginitis. Poor personal hygiene and wearing clothing that keeps the vaginal area warm and moist increase the risk of vaginitis. The infection may spread to the urinary tract, resulting in urinary tract infections; preventing and treating vaginitis decrease this risk.*

CONSIDERATION FOR PRACTICE

Teach women with DM to take preventive measures by maintaining good personal hygiene, wiping front to back after voiding, wearing cotton underwear, avoiding tight jeans and nylon pantyhose, and avoiding douching.

Risk of injury related to neuropathies, visual deficit, hyperglycaemia

The person with diabetes is at risk of injury from multiple factors. Neuropathies may alter sensation, gait and muscle control. Cataracts or retinopathy may cause visual deficits. Hyperglycaemia often causes osmotic changes in the lenses of the eye, resulting in blurred vision. In addition, changes in blood glucose alter levels of consciousness and may cause seizures. The impaired mobility, sensory deficits and neurological effects of complications of diabetes increase the risk of accidents, burns, falls and trauma.

- Assess for the presence of contributing or causative factors that increase the risk of injury: blurred vision, cataracts, decreased adaptation to the dark, decreased tactile sensitivity, hypoglycaemia, hyperglycaemia, hypovolaemia, joint immobility, unstable gait. *A knowledge base is necessary to develop an individualised plan of care. The risk of injury increases with the number of factors identified.*
- Reduce environmental hazards in the healthcare facility and teach the person about safety in the home and in the community.

IN THE HEALTHCARE FACILITY

- Orient the person to new surroundings on admission.
- Keep the bed at the lowest level.
- Keep the floors free of objects.
- Use a night-light.
- Check the temperature of the bath or shower water before the person uses it.
- Instruct the person to wear shoes or slippers when out of bed.
- Monitor blood glucose levels regularly.
- Monitor for side effects of prescribed medications, such as dizziness or drowsiness.

IN THE HOME AND COMMUNITY

- Use a night-light, preferably one with a soft, no-glare bulb.
- Turn the head away when switching on a bright light.
- Avoid directly looking into headlights when driving at night.
- Test the temperature of the bath or shower water before use.
- Conduct a daily foot inspection.
- Wear shoes and slippers with non-skid soles.
- Do not use throw rugs.
- Install hand grips in the tub and shower and next to the toilet.
- Wear a seat belt when driving or riding in a car.

Strange environments and the presence of hazardous environmental factors increase the risk of falls or other accidents. Glare is often responsible for falls in people with visual deficits. The nurse can reduce factors that increase the risk of injury by implementing care and teaching safe practices during the activities of daily life.

- Monitor for and teach the person and family to recognise and seek care for the manifestations of DKA in the person with type 1 DM: hyperglycaemia, thirst, headaches, nausea and vomiting, increased urine output, ketonuria, dehydration and decreasing level of consciousness. *Blood glucose levels increase if the insulin need is unmet or insufficiently met; the cellular use of fats for fuel results in ketosis. Osmotic diuresis increases urinary output, resulting in thirst and dehydration.*

CONSIDERATION FOR PRACTICE

Make frequent assessments to monitor for symptoms of HHS in the older adult who has had major surgery.

- Monitor for and teach the person and family to recognise and seek care for the manifestations of HHS in the person with type 2 DM: extreme hyperglycaemia, increased urinary output, thirst, dehydration, hypotension, seizures and decreasing level of consciousness. *HHS is a life-threatening condition requiring recognition and treatment.*
- Monitor for and teach the person and family to recognise and treat the manifestations of hypoglycaemia: low blood glucose, anxiety, headache, uncoordinated movements, sweating, rapid pulse, drowsiness and visual changes. Teach the person and family to carry some form of rapid-acting sugar source at all times. *Severe hypoglycaemia causes a decrease in the level of consciousness. The decrease in blood glucose most often results from too much insulin, too little food or too much exercise.*

- Recommend that the person wear a MedicAlert® bracelet or necklace identifying self as a person with diabetes. *In case of sudden, severe illness or accident, a medical alert bracelet can allow immediate medical attention for diabetes to be instituted.*

Risk of sexual dysfunction related to peripheral neuropathy

Sexuality is a complex and inseparable part of every person. It involves not only physical sexual activities but also a person's self-perception as male or female, roles and relationships, and attractiveness and desirability. Changes in sexual function and in sexuality have been identified in both men and women with diabetes.

Alterations in erectile ability occur in approximately 50% of all men with diabetes. The incidence of impotence increases with the duration of the diabetes and is often associated with peripheral neuropathy. Libido is usually unaffected, even when impotence is present.

CONSIDERATION FOR PRACTICE

Sexual function is a private matter and people rarely share concerns unless the nurse initiates the discussion.

Women with diabetes also have alterations in sexual function, although the reason is less clear. The problems reported by women involve decreased desire and decreased vaginal lubrication. Women with diabetes are also at increased risk of vaginitis and may avoid sexual intercourse in order to avoid pain.

- Include a sexual history as a part of the initial and ongoing assessment of the person with diabetes. A specific history form may be used that addresses sexual development, personal and family values, current sexual practices and concerns, and changes desired. Ask a non-threatening, open-ended question to elicit information, such as: 'Tell me about your experience with sexual function since you have been diagnosed with diabetes.' *Obtaining accurate information to assess the sexual health of a person is necessary before counselling can begin or referrals can be made.*
- Provide information about the actual and potential physical effects of diabetes on sexual function. Include the effect of poor control of blood glucose on sexual function as part of any teaching plan. *People benefit from basic information about male and female anatomy and the sexual response cycle and how diabetes can affect this part of the body. Changes in blood glucose levels not only may cause changes in desire and physical response but also may alter sexual responses as a result of depression, anxiety and fatigue.*
- Provide counselling or make referrals as appropriate. The nurse is responsible for knowing about sexuality and sexual health throughout the lifespan and provides information based on knowledge of the effects of illness and treatment on sexual function. For example, men who are impotent may regain the ability to have sexual intercourse through penile implants, suction apparatus, the use of sildenafil citrate (Viagra) or injections of medications (such as yohimbine, an alpha-2 adrenergic blocker) that increase vascular blood flow into the corpus of the penis. Women with decreased vaginal lubrication can decrease painful intercourse by using vaginal lubricants (such as K-Y Jelly) or oestrogen creams.

  The nurse may make specific suggestions to facilitate positive sexual functioning, referring the person to the appropriate healthcare provider as necessary for intensive therapy.

Risk of ineffective coping related to the chronicity of diabetes

Coping is the process of responding to internal or environmental stressors or potential stressors. When coping responses are ineffective, the stressors exceed the individual's available resources for responding. The person diagnosed with diabetes is faced with lifelong changes in many parts of their life. Diet, exercise habits and medications must be integrated into the person's lifestyle and be carefully controlled. Daily injections may be a reality. Fear of potential complications and of negative effects on the future is common.

If the person is unable to cope successfully with these changes, emotional stress can interfere with glycaemic control. In addition, unsuccessful coping often results in non-compliance with prescribed treatment modalities, further impairing glycaemic control and increasing the potential for acute and chronic complications.

- Assess the person's psychosocial resources, including emotional resources, support resources, lifestyle and communication skills. *Chronic illness affects all dimensions of a person's life, as well as the lives of family members and significant others. A comprehensive assessment of strengths and weaknesses is the first step in developing an individualised plan of care to facilitate coping.*
- Explore with the person and family the effects (actual and perceived) of the diagnosis and treatment of diabetes on finances, occupation, energy levels and relationships. *Common frustrations associated with diabetes are the disease itself, the treatment modalities and the healthcare system. Effective coping involves maintaining a healthy self-concept and satisfying relationships, emotional balance and handling emotional stress.*
- Teach constructive problem-solving techniques. *Problem-focused behaviours include setting attainable and realistic goals, learning about all aspects of the problem, learning new procedures or skills that increase self-esteem and reaching out to others for support.*
- Provide information about support groups and resources, such as suppliers of products, journals, books and cookbooks for people with diabetes. *Sharing with others who have similar problems provides opportunities for mutual support and problem solving. Using available resources improves the ability to cope.*

Community-based care

Teaching the person and family to self-manage diabetes is a nursing responsibility. Even if a formal teaching plan is developed and implemented by advanced practice nurses, all nurses must be able to reinforce knowledge and answer questions. Teaching is necessary for both the person who is newly diagnosed and for the person who has had diabetes for years. In fact, the latter may need almost as much teaching as the newly diagnosed person. Products for diabetes care, especially insulins, have changed dramatically and knowledge about risk reduction to prevent complications has increased.

For the hospitalised person with diabetes, teaching should begin on admission. Prior to designing the teaching plan, the nurse makes an initial assessment of the person's and family's knowledge and learning needs, outlining past diabetes management practices and identifying physical, emotional and sociocultural needs. Educational level, preferred learning methods and style, life experiences and support systems are also assessed. According to Shrivastava, Shrivastava and Ramasava (2013), individuals who have been diagnosed with diabetes were shown to make a dramatic impact on the progression and development of their disease by being autonomous in their own care.

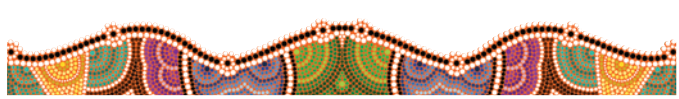
It is important that the nurse and person mutually establish goals based on the assessment data. It is equally important that family members understand that the responsibility for daily management lies with the person and that the primary role of the family is supportive. The person is the one who has the disease and who each day must take medications or inject insulin, test blood or urine, calculate and balance foods, exercise, adjust medications, inspect the body for injury and determine whether and when medical assistance is needed. However, family members require the same knowledge so that they can provide emotional support as well as physical care if necessary.

The following should be included in teaching the person and family about care at home.

- Information about normal metabolism, diabetes mellitus and how diabetes changes metabolism.
- Diet plan: how diet helps keep blood glucose in normal range; number of kilojoules required and why; amount of carbohydrates, meats and fats allowed, and why; and how to calculate the diet, integrating personal food preferences.
- Exercise: how it helps lower blood glucose; the importance of a regular program; types of exercise; integrating personal exercise preferences; how to handle increased activity.
- Self-monitoring of blood glucose: how to perform the tests accurately, how to care for equipment, what to do for high or low blood glucose.
- Medications:
  - Insulin: type, dosage, mixing instructions (if necessary), times of onset and peak actions, how to get and care for equipment, how to give injections, where to give injections.
  - Insulin: oral agents: type, dosage, side effects, interaction with other drugs.
- Manifestations of acute complications of hypoglycaemia and hyperglycaemia; what to do when they occur.
- Hygiene: skin care, dental care, foot care.
- Sick days: what to do about food, fluids and medications.
- Helpful resources:
  - Diabetes Australia: www.diabetesaustralia.com.au
  - Juvenile Diabetes Research Foundation Australia: www.jdrf.org.au
  - Koori Diabetes Services: www.archi.net.au/resources/chronic/disease/koori-diabetes
  - AIHW Diabetes, Australian Facts: www.aihw.gov.au/publication-detail/?id=6442468075
  - International Diabetes Federation: www.idf.org

Teaching may have to be adapted to the special needs of the older adult. Because 40% of all people with diabetes are over the age of 65, considering the special needs of this population is essential. Uncontrolled diabetes in the older adult increases the potential for functional loss, social disengagement and increased morbidity and mortality. Education for self-care allows the older adult to be more actively involved in their diabetes management and decreases the potential for acute and long-term complications from the disease. Considerations for teaching the older adult with diabetes include the following:

- Changes in diet may be difficult to implement for many reasons. Favourite foods are difficult to give up. Balanced meals at regular intervals may not have been part of the person's lifestyle. Purchasing, storing and preparing foods may be a problem. Dentures may not fit well. Changes in taste sensation often cause the person to increase the use of salt and sugar.
- Exercise of any type may not have been part of the activities of daily living. Exercise must be individualised for any physical limitations imposed by other chronic illnesses, such as arthritis, Parkinson's disease, chronic respiratory diseases and/or cardiovascular diseases.
- The diagnosis of a chronic illness threatens independence and self-worth. After years of taking care of self, the older adult with diabetes may now have to depend on others for help in meeting self-care needs. This in turn often leads to withdrawal from social interactions with others.
- Money to purchase medications and supplies often must be taken out of a fixed income.
- Visual deficits make insulin administration difficult or impossible. Visual deficits also interfere with blood glucose monitoring, food preparation, exercises and foot care.



CHAPTER HIGHLIGHTS

- The incidence of type 2 DM is increasing in epidemic proportions in all racial and ethnic groups globally.
- About 1 million Australians are diagnosed with diabetes and it is estimated that one person is diagnosed every 7 minutes.
- Type 2 diabetes represents a serious public health problem for Indigenous Australians, occurring at a much higher rate than in the non-Indigenous population. Aboriginal and Torres Strait Islander people have the fourth-highest rate of type 2 diabetes in the world.
- Type 2 DM has a hereditary link and is characterised by obesity and sedentary lifestyles. Unlike type 1 DM, in which the onset is often sudden, the development of symptoms that bring people to their healthcare providers for evaluation is slow; it is estimated that 50% of newly diagnosed type 2 DM people have already developed complications secondary to hyperglycaemia.
- Tighter, more intensive glycaemic control is increasingly the focus of care for hospitalised people with hyperglycaemia. Correcting hyperglycaemia is considered a benefit to diabetics and non-diabetics alike.
- New products for people with DM include insulins, non-insulin hypoglycaemics, insulin delivery devices such as pens and pumps, and blood glucose and ketone monitoring devices. Nurses must be familiar with these products and help people with diabetes become proficient in their use.
- Motivation for self-care by the person with diabetes continues to be a challenge because treatment commonly includes lifestyle changes. Through education and support, people can achieve control of DM and avoid complications.

CONCEPT CHECK

1 Increased susceptibility to the development of type 1 diabetes is indicated by which of the following?
1. genetic markers that determine immune response
2. persistent obesity throughout the adolescent years
3. delivery of a baby that weighs less than 2.7 kg
4. excessive amounts of plasma glucagon

2 Diabetic ketoacidosis is the result of which pathological process?
1. An excess amount of insulin drives all glucose into the cells.
2. A decreased amount of glucagon causes low protein levels.
3. A deficit of insulin causes fat stores to be used as an energy source.
4. An increase occurs in the breakdown of glucose molecules with hypoglycaemia.

3 Which of the following people would be most at risk of the development of type 2 DM?
1. young adult who is a professional basketball player
2. middle-aged man who maintains normal weight
3. middle-aged woman who is the sole caretaker of her parents
4. woman over age 70 who is overweight and sedentary

4 You are assigned a person who has a nursing diagnosis of *peripheral neurovascular dysfunction* involving both feet. Which of the following assessments would support this diagnosis?
1. normal sensation to touch
2. loss of normal reflexes
3. states, 'I can't feel my feet any more.'
4. states, 'I have been having chest pain.'

5 Which of the following statements would indicate the person understands teaching about foot care at home?
1. 'I will walk barefooted as long as I am in the house.'
2. 'I always buy my shoes as soon as the stores open.'
3. 'I will check my feet for cuts and bruises every night.'
4. 'If I get a blister, I just put alcohol on it and bandage it.'

6 Lantus insulin, a long-acting insulin, has a unique insulin characteristic that increases the risk of administration error. The nurse understands that this long-acting insulin is:
1. combined with glucose to raise energy levels
2. subject to being inactivated by light
3. a clear solution like regular insulin, unlike intermediate and long-acting insulins
4. activated by vigorous agitation

7 The nurse is preparing an insulin infusion for a person in diabetic ketoacidosis (DKA). She is careful to select the only type of insulin that can be administered intravenously, which is:
1. Lantus
2. Actrapid
3. regular
4. Humalog

8 Glycosylated haemoglobin (HbA1c) is useful for evaluating the degree of blood glucose control the person with diabetes has been maintaining for the previous 2 to 3 months. The American Diabetes Association recommended goal for HbA1c in the general population with diabetes is:
1. > 10%
2. > 8%
3. < 7%
4. < 3%

9 Subcutaneous injections of insulin can be made in several locations in the body. The nurse teaches the person that the most predictable absorption occurs in the:
1. hip
2. thigh
3. deltoid
4. abdomen

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UNIT 5 BUILDING CLINICAL COMPETENCE
Responses to altered endocrine function

CLINICAL SCENARIO

In the afternoon shift you are looking after two people that have been admitted to your ward earlier in the day:

- Mrs Kelly Ly is a 47-year-old Vietnamese woman who has limited English. She is accompanied by her husband and her son who is helping with English translation when necessary. Mrs Ly has been admitted from the emergency department with increased confusion, agitation, nausea and vomiting, tachycardia, hypertension and weight loss. She has a history of Graves' disease, diagnosed 2 years ago on her arrival to Australia from Vietnam. On your assessment her observations are T 38.9°C, P 119, R 30, BP 171/83. Her blood glucose level is 3.7 mmol/L. She is anxious, agitated, her hands are shaking and she is complaining of abdominal pain and wanting to vomit. Her son told you that she is 'not herself' and she is 'confused'.
- Colin is 17 years old. He has had diabetes type 1 since he was 10 years old. He was transferred from the High Dependency Unit this morning. He is recovering from diabetic ketoacidosis (DKA) after falling ill with a severe chest infection. His vital signs are T 37.1°C, P 85, R14, BP 112/55. His blood sugar level is 7.9 mmol/L 2 hours after lunch. Colin says that he is feeling better but still coughing. He has two cannulaes for intravenous antibiotics. You performed a urinalysis that showed glucose + and ketones trace. Colin still has an indwelling catheter (IDC) and asks you when he can have it removed.

Critical thinking questions

1 After your assessment and above findings, which person needs your immediate attention? Provide the rationale for your decision.

2 What do you think is occurring for Mrs Ly? Explain the signs and symptoms that she is showing and relate them to her diagnosis of Graves' disease.

3 Mrs Ly may be showing other significant signs and symptoms of hyperthyroidism. List them according to body systems.

4 What are the main nursing diagnoses for Mrs Ly? List your immediate actions.

5 Mrs Ly is very likely to be experiencing a thyroid crisis, which is a medical emergency. Thyroid crisis or thyroid storm can be defined as:

1. extreme hypothyroidism
2. excess production of parathyroid hormone (PTH)
3. low production of thyroid hormone (TH)
4. excess production of TH

6 Mrs Ly has been experiencing difficulty fully closing her eyelids for a few months. What is the possible cause for this?

1. goitre
2. pretibial myxoedema
3. proptosis
4. amenorrhoea

7 Mrs Ly has recovered with medication management and discharge planning is in progress as she may be discharged home tomorrow. Develop a discharge plan, taking into consideration her ethnic background and limited English. Include in your plan some education in relation to follow-up care, management and prevention of complications in Graves' disease.

8 Colin is upset because he feels that he has always done the best to manage his diabetes and he still got DKA. Explain DKA to Colin and how it occurred when he got the chest infection.

9 The doctor has told Colin that he would like to leave the IDC in until there are no ketones present in the urine and this is likely to occur soon. What is the reason for ketones to be present in Colin's urine?

1. Ketones are the product of glucose breakdown that is excreted in the urine.
2. They are the product of fat breakdown excreted in the urine.
3. Polyuria causes dehydration that leads to ketones in the urine.
4. It is normal to have presence of ketones in the urine when there is glucose as well.

10 Colin presented to the hospital with a sugar level of 30 mmol/L. He was rehydrated, electrolyles were replaced and he was commenced on insulin and then dextrose intravenously. Why is it essential to reduce the blood sugar level slowly and gradually at that stage of DKA?

11 Colin's glycosylated haemoglodin or HbA1c come back at 9%. Explain glycosylated haemoglobin and what this result means.

12 If Colin gets an episode of hypoglycaemia with a glucose level of 2.7 mmol/L, which signs and symptoms might he experience?

1. bradycardia, nausea, vomiting
2. polyuria, polydipsia, polyphagea
3. diarrhoea, nausea, thirst
4. hypotension, tachycardia, profuse sweating

13 Colin is preparing for dinner and needs to have his Humalog injection. When does he need to take his insulin?

1. 30 minutes before he starts eating
2. immediately after he finishes his meal
3. immediately before he starts eating
4. 1 hour before his meal

CASE STUDY

Mr Lewis is a 70-year-old Aboriginal male who was admitted with health problems of increased urination, increased thirst, fatigue, blurred vision and numbness in his feet. He states that he retired 9 months ago after 45 years as a construction worker. He now leads a sedentary lifestyle and doesn't have as much energy as he used to have. He has gained 14 kg since retirement. Upon assessment,

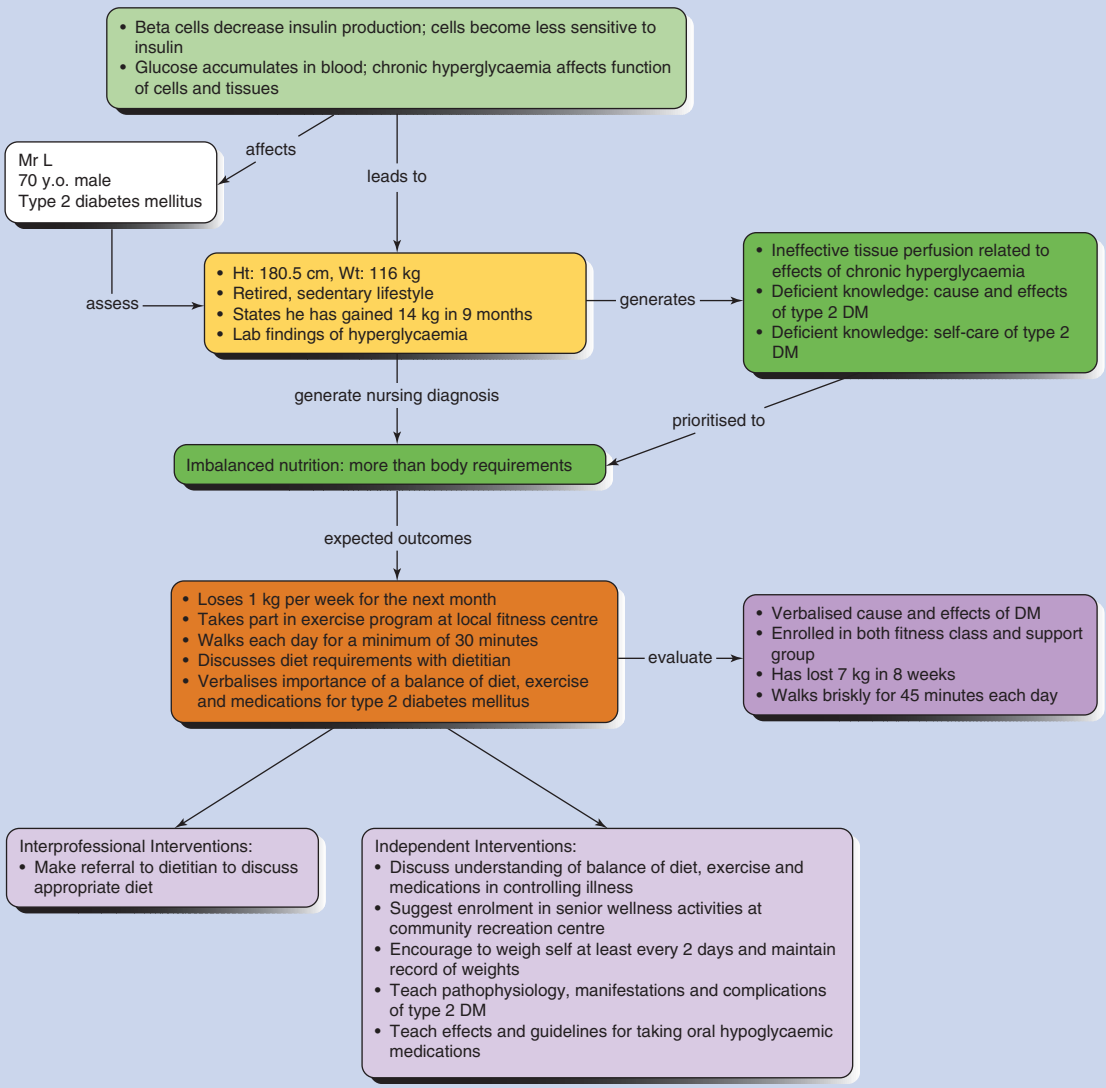
Mr Lewis weighs 116 kg and is 180.5 cm tall. Vital signs are T 37°C, P 88, R 20 and BP 150/90. Decreased pulses are palpated in dorsal pedalis and posterior tibial pulses. Both feet are cool to touch with slow capillary refill in toes. The following laboratory studies are ordered to confirm the diagnosis of type 2 diabetes mellitus: plasma glucose concentration, fasting blood glucose and oral glucose tolerance test.

Type 2 diabetes mellitus is a disease of fasting hyperglycaemia despite the production of some insulin by the beta cells of the pancreas. The level of insulin production varies, affecting the amount available for cellular metabolism.

With increased age, sedentary lifestyle and obesity, cells become resistant to insulin. The uptake of glucose by muscle and fat cells is not sufficient to lower the blood glucose. In obesity, insulin has a decreased ability to influence glucose metabolism and affect the uptake of glucose by cells in the liver, skeletal muscles and adipose tissue, resulting in a high blood glucose level.

The manifestations of type 2 diabetes mellitus include polyuria, polydipsia, blurred vision, fatigue, paraesthesias and skin infections. In times of physical or emotional stress, the person may develop a hyperosmolar hyperglycaemic state (HHS). Complications of type 2 diabetes mellitus include coronary heart disease (myocardial infarction), hypertension, stroke, peripheral vascular disease (atherosclerosis, vascular insufficiency, amputations), end-stage renal disease (nephropathy) and blindness (retinopathy).

Based on Mr Lewis's manifestations and weight gain, a priority nursing diagnosis of *Imbalanced nutrition: more than body requirements* is appropriate for guiding nursing care for this person.



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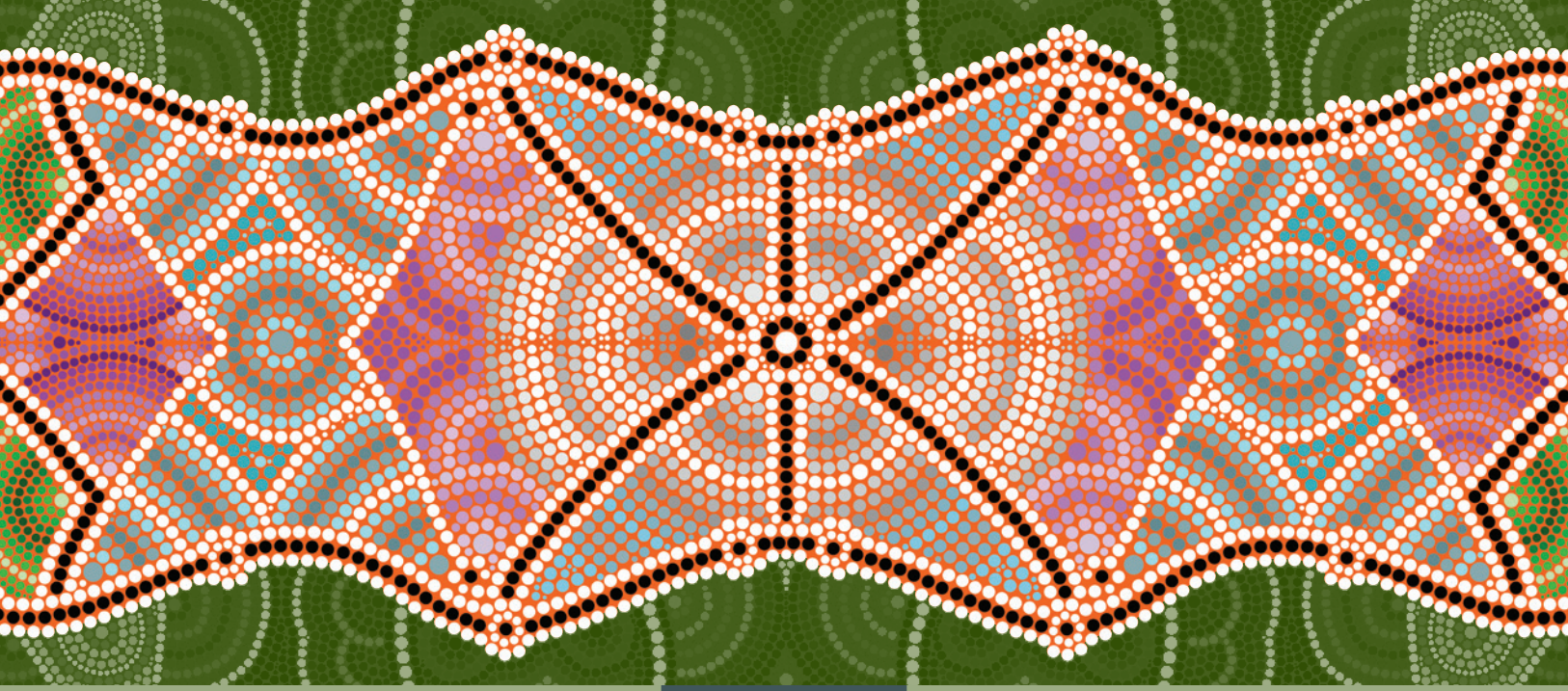


UNIT 6

RESPONSES TO ALTERED GASTROINTESTINAL FUNCTION

CHAPTER 20

A PERSON-CENTRED APPROACH TO ASSESSING THE GASTROINTESTINAL SYSTEM

NICOLE KNOX

KEY TERMS

LEARNING OUTCOMES

- Describe the sources of nutrients and their functions in the human body.
- Describe the anatomy, physiology and functions of the gastrointestinal system and accessory digestive organs.
- Discuss rationales for questions included in a health assessment interview of a person with nutritional and gastrointestinal disorders.
- Explain techniques used for assessing nutritional status and gastrointestinal function.

CLINICAL COMPETENCIES

- Conduct and document a health history for people who have or are at risk of alterations in nutrition and gastrointestinal function.
- Conduct and document a physical assessment of nutritional status and the gastrointestinal system.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Stethoscope
- Weight scale with height measuring attachment
- Tape measure
- Skinfold calipers
- Water-soluble lubricant
- Faecal occult blood test kit (FOBT)
- Disposable gloves

Nutrition is the sum total of the processes involved in the taking in and the utilisation of food substances by which growth, repair and maintenance of the body are accomplished. It involves ingestion, digestion, absorption/assimilation and elimination. Nutrients are stored by the body in various forms and drawn upon when the food intake is not sufficient.

The digestive organs responsible for these processes are the gastrointestinal tract (also called the alimentary canal) and the accessory digestive organs. The gastrointestinal tract consists of the mouth, pharynx, oesophagus, stomach, small intestine and large intestine. The accessory digestive organs include the liver, gallbladder and pancreas (see Figure 20.1). This chapter discusses the assessment of these organs.

NUTRIENTS

Nutrients are substances found in food and are used by the body to promote growth, maintenance and repair. The categories of nutrients are carbohydrates, proteins, fats, vitamins, minerals and water. Dietary guidelines for nutrients specific to Australians are summarised in Table 20.1.

Nutritional deficits are common in people who are obese. All treatments, including bariatric surgery (see Chapter 21), consist of lifelong dietary control, exercise and behaviour change. These procedures contribute to nutritional deficiencies by restricting food intake and/or limiting intestinal absorption. The most commonly described nutritional deficiencies include thiamine (B_1), B_{12}, folate (B_9), vitamin D, vitamin E and copper deficiencies (Becker, Balcer & Galetta, 2012). The incidence of neurological complications in this group of people is estimated at around 16% and includes encephalopathy, optic neuropathy, myelopathy and polyneuropathy. Risk factors for nutritional complications include vitamin non-adherence, protracted vomiting and excessive alcohol consumption.

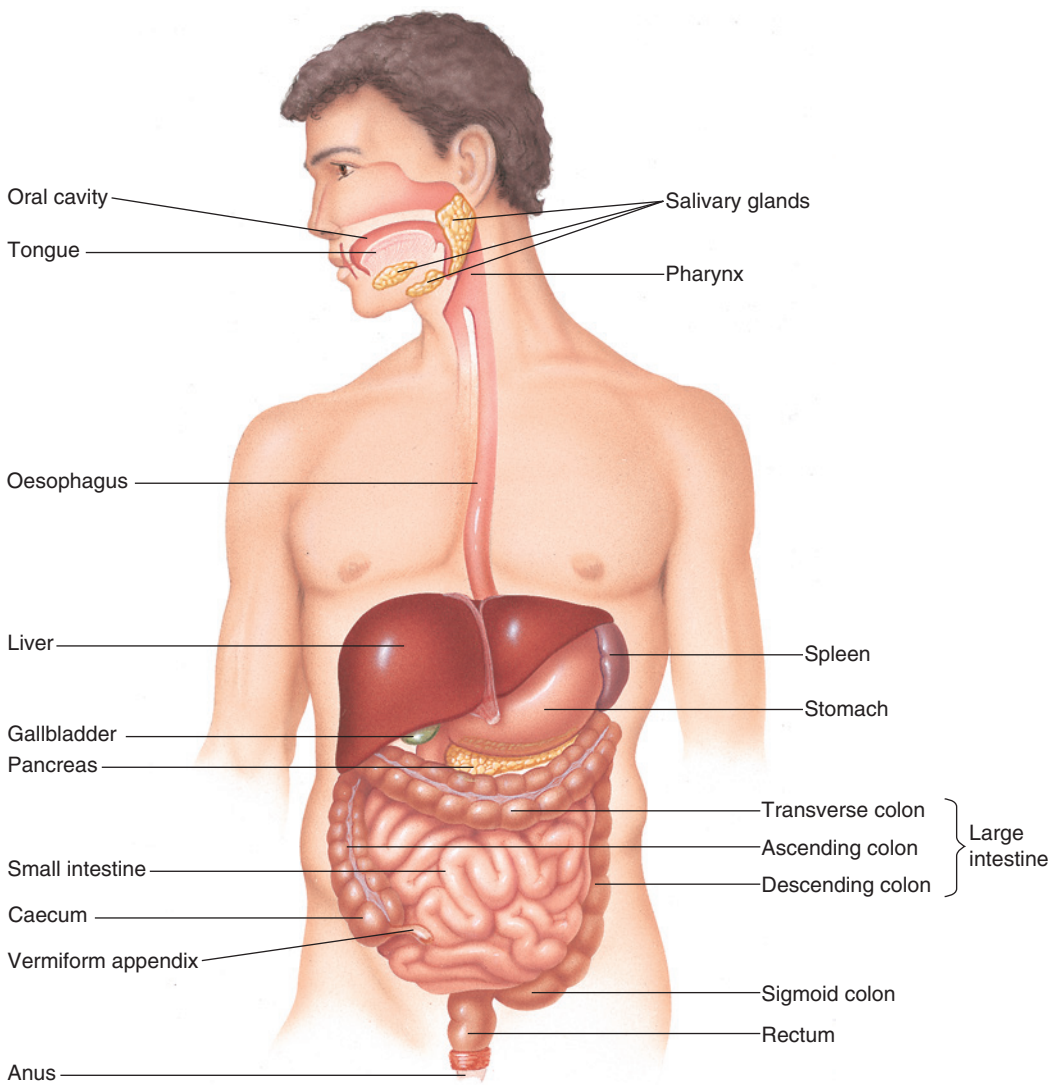


FIGURE 20.1 ■ Organs of the gastrointestinal tract and accessory digestive organs

TABLE 20.1 Dietary guidelines for Australians

FOR ADULTS	FOR CHILDREN AND ADOLESCENTS
Enjoy a wide variety of nutritious foods *Adults should be encouraged to:* • Eat mostly vegetables and legumes/beans • Include grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties (including breads, rice, pasta and noodles) • Include lean meat and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans • Include milks, yoghurts, cheeses and/or alternatives, mostly reduced fat • Include 2 servings of fruit per day • Drink plenty of water *and take care to:* • Limit foods with saturated fat and moderate total fat intake • Limit foods with added salt • Limit your alcohol intake if you choose to drink • Limit foods and drinks containing added sugars **Prevent weight gain: be physically active and eat according to your energy needs** **Care for your food: prepare and store it safely** **Encourage and support breastfeeding**	**Encourage and support breastfeeding** **Children and adolescents need sufficient nutritious foods to grow and develop normally** • Growth should be checked regularly for young children • Physical activity is important for all children and adolescents **Enjoy a wide variety of nutritious foods** *Children and adolescents should be encouraged to:* • Eat plenty of vegetables and legumes/beans • Eat moderate amounts of fruit (whole fruit is preferred to fruit juice) • Eat plenty of cereal foods, mostly wholegrain and/or high cereal fibre varieties (including breads, rice, pasta and noodles) • Include lean meats and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans • Include milks, yoghurts, cheese and/or alternatives. Reduced-fat milks are not suitable for young children under 2 years, because of their high energy needs, but reduced-fat varieties should be encouraged for older children and adolescents • Choose water as a drink. Alcohol is not recommended for children *and care should be taken to:* • Limit foods with saturated fat and moderate total fat intake. Low-fat diets are not suitable for infants • Limit foods high in salt • Limit foods and drinks containing added sugars **Care for your child's food: prepare and store it safely**

Source: Based on material provided by the National Health and Medical Research Council (NHMRC) (2013). *Eat for Health Educator Guide: Information for nutrition educators*, www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n55b_educator_guide_140321.pdf. Used by permission of the Australian Government.

Carbohydrates

The primary sources of carbohydrates (which include sugars and starches) are plant foods. Monosaccharides and disaccharides come from milk, sugar cane, sugar beets, honey and fruits. Polysaccharide starch is found in grains, legumes and root vegetables. Following ingestion, digestion and metabolism, carbohydrates are converted primarily to glucose, the molecule body cells use to make adenosine triphosphate (ATP). Excess glucose in the healthy person is converted to glycogen or fat. Glycogen is stored in the liver and muscles; fat is stored as adipose tissue. Carbohydrate use by the body is shown in Figure 20.2A.

Regardless of the source, all carbohydrates supply 16.7 kilojoules (kJ) per gram. The recommended dietary intake is 125 to 175 g, most of which should be complex carbohydrates (such as fruits, starchy vegetables and whole grains). Excessive intake of carbohydrates over time can result in obesity, tooth decay and elevated plasma triglycerides. Over extended periods of time, carbohydrate deficiencies lead to tissue wasting from protein breakdown and metabolic acidosis from an excess of ketones as a by-product of fat breakdown.

Proteins

Proteins are classified as either complete or incomplete. Complete proteins are found in animal products such as eggs, milk, milk products and meat. They contain the greatest amount of amino acids and meet the body's amino acid requirements for tissue growth and maintenance. Incomplete proteins are found in legumes, nuts, grains, cereals and vegetables. These sources are low in or lack one or more of the amino acids essential for building complete proteins.

The body uses proteins to build many different structures, including skin keratin and the collagen and elastin in connective tissues and muscles. They also are used to make enzymes, haemoglobin, plasma proteins and some hormones. Protein use by the body is shown in Figure 20.2B.

Proteins provide 16.7 kJ per gram. The recommended dietary intake of protein is 64 g for men and 46 g for women. (This is higher for adults over 70 years of age.) Healthy people with adequate energy intake have an equal rate of protein synthesis and protein breakdown and loss, reflected as nitrogen balance. If the breakdown and loss of proteins exceed intake, a negative nitrogen balance results. This may be due to starvation, altered physical states (e.g. from injury or illness) and altered emotional states (such as depression or anxiety). A positive nitrogen balance, which results when protein intake exceeds breakdown, is normal during growth, tissue repair and pregnancy. Anabolic steroids affect the rate of protein use; for example, the adrenal corticosteroids are released in times of stress to increase protein breakdown and conversion of amino acids to glucose. Excessive intake of proteins may lead to obesity, whereas deficits cause weight loss and tissue wasting, oedema and anaemia.

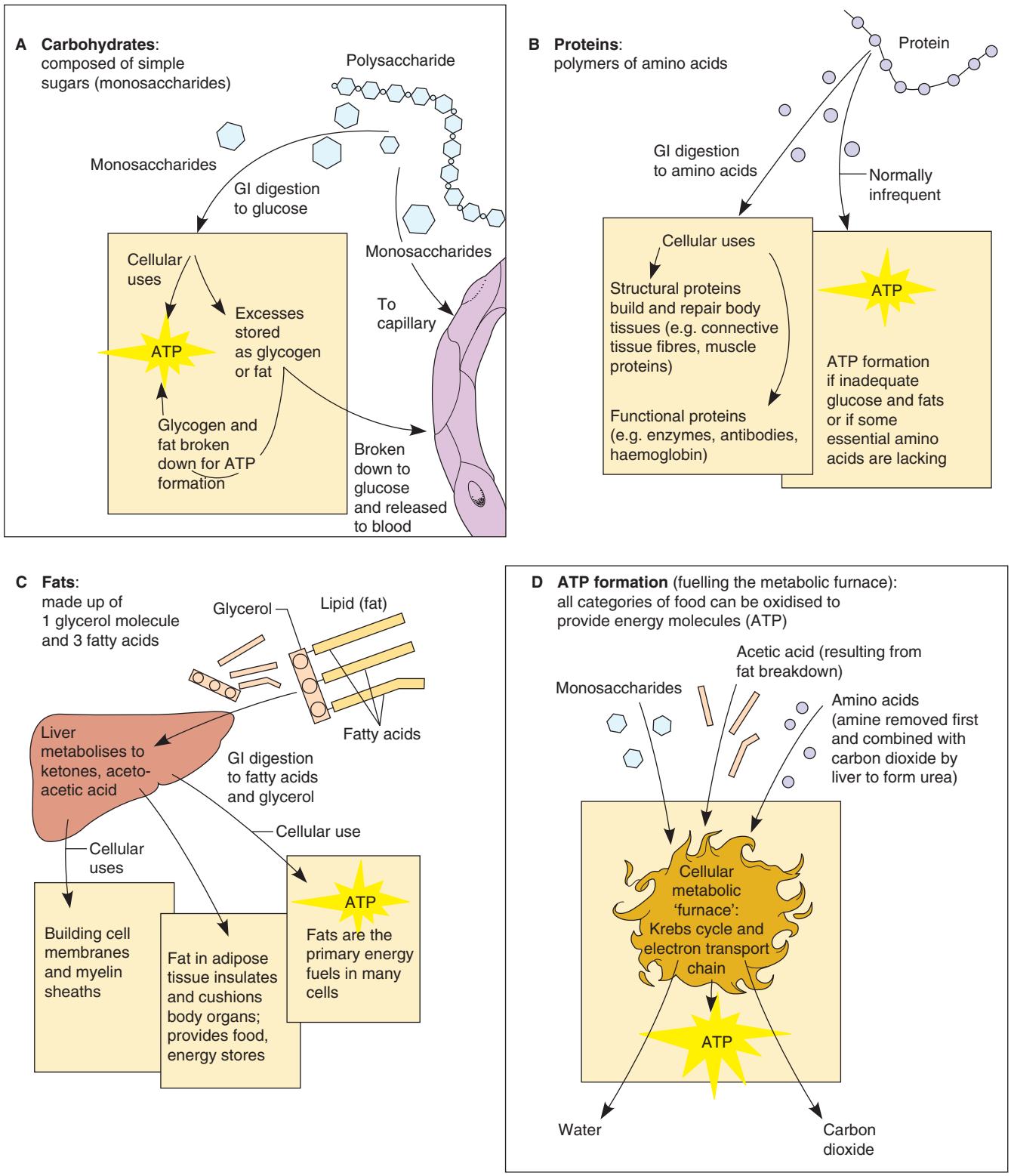


FIGURE 20.2 ■ A schematic overview of nutrient use by body cells, including *A*, carbohydrates; *B*, proteins; *C*, fats; and *D*, ATP formation

Fats (lipids)

Fats, or lipids, include phospholipids; steroids, such as cholesterol; and neutral fats, more commonly known as triglycerides. Neutral fats are the most abundant fats in the diet. They may be either saturated or unsaturated. Saturated fats are found in animal products (milk and meats) and in some plant products (such as coconut). Unsaturated fats are found in seeds, nuts and most vegetable oils. Sources of cholesterol include meats, milk products and egg yolks. Fat use by the body is shown in Figure 20.2C.

Fats supply 37.7 kJ per gram. When a person consumes more than the body requires, the excess is stored as adipose tissue, increasing the risk of obesity and heart disease. A deficit of fats may cause excessive weight loss and skin lesions.

Fats are a necessary part of the structure and function of the body. For example:

- Phospholipids are a part of all cell membranes.
- Triglycerides are the major energy source for hepatocytes and skeletal muscle cells.
- Dietary fats facilitate absorption of fat-soluble vitamins.
- Linoleic acid, an essential fatty acid, helps form prostaglandins, regulatory molecules that assist in smooth muscle contraction, maintenance of blood pressure and control of inflammatory responses.
- Cholesterol is the essential component of bile salts, steroid hormones and vitamin D.
- Adipose tissue serves as a protection around body organs, as a layer of insulation under the skin and as a concentrated source of fuel for cellular energy.

Vitamins

Vitamins are organic compounds that facilitate the body's use of carbohydrates, proteins and fats. All the vitamins except vitamins D and K must be ingested in foods or taken as supplements. Vitamin D is made by ultraviolet irradiation of cholesterol molecules in the skin and vitamin K is synthesised by bacteria in the intestine.

Vitamins are categorised as either fat soluble or water soluble. The fat-soluble vitamins (A, D, E and K) bind to ingested fats and are absorbed as the fats are absorbed. Water-soluble vitamins (the B complex and C) are absorbed with water in the gastrointestinal (GI) tract. (However, vitamin B_{12} must become attached to the intrinsic factor (a protein) to be absorbed.) Fat-soluble vitamins are stored in the body and excesses may cause toxicity; water-soluble vitamins in excess of body requirements are excreted in the urine.

The recommended dietary intake (RDI) or adequate intake (AI) and the source and function of vitamins, as stated by the National Health and Medical Research Council (NHMRC), are listed in Tables 20.2 and 20.3.

Minerals

Minerals work with other nutrients to maintain the structure and function of the body. An adequate supply of calcium, phosphorus, potassium, sulfur, sodium, chloride and magnesium—as well as other trace elements such as iron, iodine, copper and zinc—is necessary to health. Most minerals in the body are found in body fluids or are bound to organic compounds. The best sources of minerals are vegetables, legumes, milk and some meats. Dietary sources for the major minerals are discussed in Chapter 9. The recommended dietary intake for minerals is outlined in Table 20.4.

TABLE 20.2 Recommended dietary intake/adequate intake of fat-soluble vitamins

NAME	SOURCE	FUNCTION	MINIMUM RECOMMENDED DIEATRY INTAKE/ADEQUATE INTAKE (AI) (M = MEN, W = WOMEN)
Vitamin A (retinol)	• Fish liver oils • Egg yolk • Liver • Fortified milk • Margarine	Necessary for vision, integrity of skin and mucous membranes, cell membrane function and reproductive function	M = 900 mcg W = 700 mcg
Vitamin D	• The action of sunshine on cholesterol in the skin	Necessary for blood calcium homeostasis (in turn, necessary for blood clotting), bone formation and neuromuscular function	M and W = 5 mcg M and W 51–70 = 10 mcg M and W > 70 = 15 mcg
Vitamin E (as ∝-tocopherol)	• Vegetable oils • Margarine • Whole grains • Dark green leafy vegetables	As an antioxidant, helps prevent the oxidation of vitamins A and C in the intestines and decreases the oxidation of unsaturated fatty acids to facilitate cell membrane integrity	M = 10 mg W = 7 mg
Vitamin K	• Synthesised by coliform bacteria in the large intestine • Green, leafy vegetables • Cabbage • Cauliflower • Pork	Essential for the formation of clotting proteins in the liver	M = 70 mcg W = 60 mcg

TABLE 20.3 Recommended dietary intake of water-soluble vitamins

NAME	SOURCE	FUNCTION	MINIMUM RECOMMENDED DIETARY INTAKE/ ADEQUATE INTAKE (A1) (M = MEN, W = WOMEN)
Vitamin B_1 (thiamine)	• Lean meats • Liver • Eggs • Green leafy vegetables • Legumes • Whole grains	An essential coenzyme for carbohydrate metabolism and use; also for healthy function of nerves, muscles and the heart	M = 1.2 mg W = 1.1 mg
Vitamin B_2 (riboflavin)	• Liver • Egg white • Whole grains • Meat • Poultry • Fish • Milk	Involved in the catabolism and use of carbohydrates, fats and proteins; the use of other B vitamins; and is important for the production of adrenal hormones	M = 1.3 mg M > 70 years = 1.6 mg/day W = 1.1 mg W > 70 years = 1.3 mg/day
Vitamin B_6 (pyridoxine)	• Meat • Poultry • Fish • Potatoes • Tomatoes • Sweet potatoes • Spinach	Necessary for amino acid metabolism, and formation of antibodies and haemoglobin	M and W < age 51 = 1.3 M > age 51 = 1.7 mg W > age 51 = 1.5 mg
Vitamin B_{12} (cyanocobalamin)	• Liver • Meat • Poultry • Dairy foods (except butter) • Eggs	Essential for the production of nucleic acids and red blood cells in the bone marrow; also plays an important role in the use of folic acid and carbohydrates and in healthy function of the nervous system	M and W = 2.4 mcg
Vitamin C (ascorbic acid)	• Citrus fruits • Potatoes • Tomatoes • Green leafy vegetables	Acts as an antioxidant and vasoconstrictor; also serves in the formation of connective tissue, conversion of cholesterol to bile salts, iron absorption and use and conversion of folic acid to an active form	M and W = 45 mg
Vitamin B_3 (niacin; nicotinamide)	• Meat • Poultry • Fish • Liver • Peanuts • Green leafy vegetables	Plays an important role in the metabolism of carbohydrates and fats; inhibits cholesterol synthesis; important for integumentary, nervous and digestive system health; assists in the manufacture of reproductive hormones	M and W = 35 mg (as nicotinic acid)
Biotin (B_7)	• Liver • Eggs • Nuts • Legumes	Essential for the catabolism of fatty acids and carbohydrates and helps dispose of the waste products of protein catabolism	M = 30 mcg W = 25 mcg
Pantothenic acid (B_5)	• Meats • Whole grains • Egg yolk • Liver • Yeast • Legumes	Assists in the synthesis of steroids and of the haem in haemoglobin; is essential for the metabolism of carbohydrates and fats, and for the manufacture of reproductive hormones	M = 6 mg W = 4 mg
Folic acid (folate; B_9)	• Liver • Dark green vegetables • Lean beef • Eggs • Veal • Whole grains • Synthesised by bacteria in the intestine	The basis of a coenzyme necessary to the manufacture of nucleic acids and so is essential for the formation of red blood cells, growth and development, and nervous system health	M and W = 400 mcg

TABLE 20.4 Recommended dietary intake of minerals

NAME	MINIMUM RECOMMENDED DIETARY INTAKE/ADEQUATE INTAKE (A1) (M = MEN, W = WOMEN)	NAME	MINIMUM RECOMMENDED DIETARY INTAKE/ADEQUATE INTAKE (A1) (M = MEN, W = WOMEN)
Calcium	M and W = 1000 mg M > 70 and W > 51 = 1300 mg	Chromium	M = 35 mcg W = 25 mcg
Phosphorus	M and W = 1000 mg	Iodine	M and W = 150 mcg
Iron	M = 8 mg W = 18 mg W > 51 = 8 mg	Selenium	M = 70 mcg W = 60 mcg
Zinc	M = 14 mg W = 8 mg	Magnesium	M = 400–420 mg W = 310–320 mg
Manganese	M = 5.5 mg W = 5 mg	Copper	M = 1.7 mg W = 1.2 mg
Molybdenum	M and W = 45 mcg	Sodium	M and W = 460–920 mg

Dietary fibre

Food Standards Australia New Zealand (FSANZ) defines dietary fibre as:

> *that fraction of the edible parts of plants or their extracts, or synthetic analogues, that are resistant to the digestion and absorption in the small intestine, usually with complete or partial fermentation in the large intestine and promote one or more of the following beneficial physiological effects—(i) laxation; (ii) reduction in blood cholesterol; (iii) modulation of blood glucose.* (FSANZ, 2012, p. 2)

Adequate intake of dietary fibre has been linked to a reduction in the occurrence of several chronic diseases and is essential to maintain functioning of the GI tract. An adequate intake for women is 25 g/day and for men is 30 g/day.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE GASTROINTESTINAL SYSTEM

The gastrointestinal tract is a continuous hollow tube, extending from the mouth to the anus. Foods are placed in the mouth, then they are subjected to a variety of processes that move them and break them down into usable elements, and nutrients are absorbed from the lumen of the small intestine into the blood or lymph, while indigestible materials are eliminated. Bowel elimination is the end process in digestion. The digestive processes are as follows:

- ingestion of food
- movement of food and wastes
- secretion of mucus, water and enzymes
- mechanical digestion of food
- chemical digestion of food
- absorption of nutrients from digested food
- elimination of wastes.

The mouth

The mouth, also called the oral or buccal cavity, is lined with mucous membranes and is enclosed by the lips, cheeks, palate and tongue (see Figure 20.3).

The lips and cheeks are skeletal muscle covered externally by skin. Their function is to keep food in the mouth during chewing. The palate consists of two regions: the hard palate and the soft palate. The hard palate covers bone in the roof of the mouth and provides a hard surface against which the tongue forces food. The soft palate, extending from the hard palate and ending at the back of the mouth as a fold called the uvula, is primarily muscle. When food is swallowed, the soft palate rises as a reflex to close off the oropharynx.

The tongue, composed of skeletal muscle and connective tissue, is located in the floor of the mouth. It contains mucous and serous glands, taste buds and papillae. The tongue mixes food with saliva during chewing, forms the food into a mass (called a *bolus*) and initiates swallowing. Some papillae provide surface roughness to facilitate licking and moving food; other papillae house the taste buds.

Saliva moistens food so it can be made into a bolus, dissolves food chemicals so they can be tasted and provides enzymes (such as amylase) that begin the chemical breakdown of starches. Saliva is produced by salivary glands, most of which lie superior or inferior to the mouth and drain into it. The salivary glands include the parotid, the submaxillary and the sublingual glands.

The teeth chew (masticate) and grind food to break it down into smaller parts. As the food is masticated, it is mixed with saliva. Adults have 32 permanent teeth. The teeth are embedded in the gingiva (gums), with the crown of each tooth visible above the gingiva.

The pharynx

The pharynx consists of the oropharynx and the laryngopharynx (see Figure 20.3). Both structures provide passageways for food,

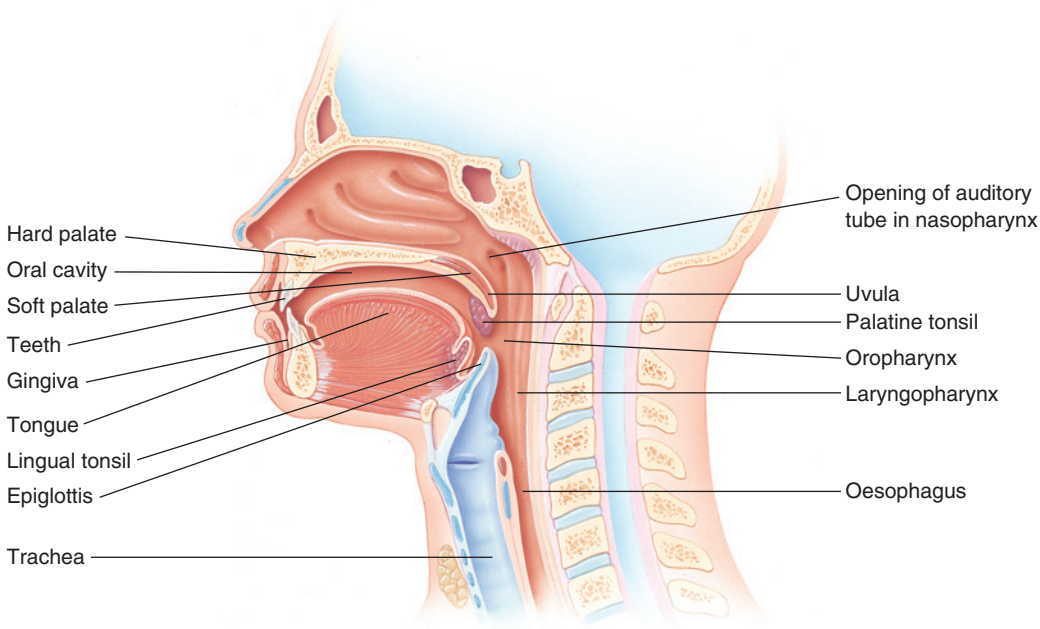


FIGURE 20.3 ■ Structures of the mouth, the pharynx and the oesophagus

fluids and air. The pharynx is made of skeletal muscles and is lined with mucous membranes. The skeletal muscles move food to the oesophagus via the pharynx through **peristalsis** (alternating waves of contraction and relaxation of involuntary muscle). The mucosa of the pharynx contains mucus-producing glands that provide fluid to facilitate the passage of the bolus of food as it is swallowed.

The oesophagus

The oesophagus, a muscular tube about 25 cm long, serves as a passageway for food from the pharynx to the stomach (see Figures 20.1 and 20.3). The epiglottis, a flap of cartilage over the top of the larynx, keeps food out of the larynx during swallowing. The oesophagus descends through the thorax and diaphragm, entering the stomach at the cardiac orifice. The gastro-oesophageal sphincter surrounds this opening. This sphincter, along with the diaphragm, keeps the orifice closed when food is not being swallowed.

For most of its length, the oesophagus is lined with stratified squamous epithelium; simple columnar epithelium lines the oesophagus where it joins the stomach. The mucosa and submucosa of the oesophagus lie in longitudinal folds when the oesophagus is empty.

The stomach

The stomach, located high on the left side of the abdominal cavity, is connected to the oesophagus at the upper end and to the small intestine at the lower end (see Figure 20.4). Normally about 25 cm long, the stomach is a distensible organ that can expand to hold up to 4 L of food and fluid. The concave surface of the stomach is called the lesser curvature; the convex surface is called the greater curvature. The stomach may be divided

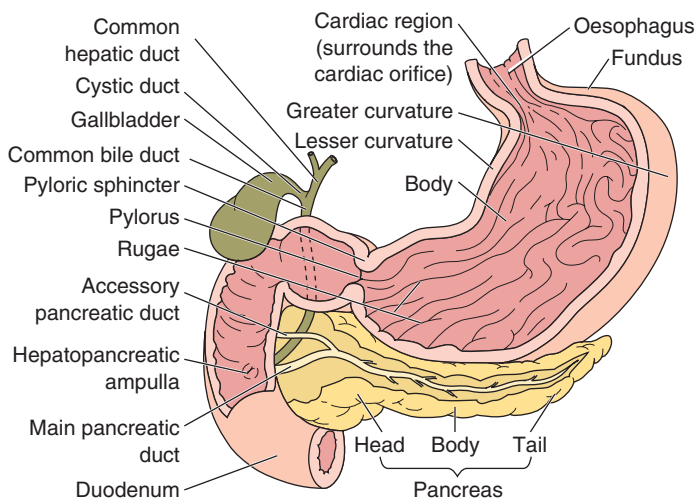


FIGURE 20.4 ■ The internal anatomical structures of the stomach, including the pancreatic, cystic and hepatic ducts, the pancreas and the gallbladder

into regions extending from the distal end of the oesophagus to the opening into the small intestine. These regions are the cardiac region, fundus, body and pylorus (see Figure 20.4). The pyloric sphincter controls emptying of the stomach into the duodenal portion of the small intestine. The stomach is a storage reservoir for food, continues the mechanical breakdown of food, begins the process of protein digestion and mixes the food with gastric juices into a thick fluid called chyme.

The stomach is lined with columnar epithelial, mucus-producing cells. Millions of openings in the lining lead to

gastric glands that can produce 4 to 5 L of gastric juice each day. The gastric glands contain a variety of secretory cells, including the following:

- Mucous cells produce alkaline mucus that clings to the lining of the stomach and protects it from being digested by gastric juice.
- Zymogenic cells produce pepsinogen (an inactive form of pepsin, a protein-digesting enzyme).
- Parietal cells secrete hydrochloric acid and intrinsic factor. Hydrochloric acid activates and increases the activity of protein-digesting cells and also is bactericidal. Intrinsic factor is necessary for the absorption of vitamin B_{12} in the small intestine.
- Enteroendocrine cells secrete gastrin, histamine, endorphins, serotonin and somatostatin. These hormones or hormone-like substances diffuse into the blood. Gastrin is important in regulating secretion and motility of the stomach.

The secretion of gastric juice is under both neural and endocrine control. Stimulation of the parasympathetic vagus nerve increases secretory activity; in contrast, stimulation of sympathetic nerves decreases secretions. The three phases of secretory activity are the cephalic phase, the gastric phase and the intestinal phase.

- The cephalic phase prepares for digestion and is triggered by the sight, odour, taste or thought of food. During this initial phase, motor impulses are transmitted via the vagus nerve to the stomach.
- The gastric phase begins when food enters the stomach. Stomach distension (stimulating stretch receptors) and chemical stimuli from partially digested proteins initiate this phase. Gastrin-secreting cells produce gastrin, which in turn stimulates the gastric glands (especially the parietal cells) to produce more gastric juice. Histamine also stimulates hydrochloric acid secretion.
- The intestinal phase is initiated when partially digested food begins to enter the small intestine, stimulating mucous cells of the intestine to release a hormone that promotes continued gastric secretion.

Mechanical digestion in the stomach is accomplished by peristaltic movements that churn and mix the food with the gastric juices to form chyme. Gastric motility is enhanced or retarded by the same factors that affect secretion—namely, distension and the effect of gastrin. After a person eats a well-balanced meal, the stomach empties completely in approximately 4 to 6 hours. Gastric emptying depends on the volume, chemical composition and osmotic pressure of the gastric contents. The stomach empties large volumes of liquid content more rapidly, while solids and fats slow gastric emptying.

The small intestine

The small intestine begins at the pyloric sphincter and ends at the ileocaecal junction at the entrance of the large intestine (see Figure 20.1). The small intestine is about 6 m long but only about 2.5 cm in diameter. This long tube hangs in coils in the abdominal cavity, suspended by the mesentery and surrounded by the large intestine. The small intestine has three regions: the duodenum, the jejunum and the ileum. The duodenum begins at the pyloric sphincter and extends around the head of the pancreas for about 25 cm. Both pancreatic enzymes and bile from the liver enter the small intestine at the duodenum. The jejunum, the middle region of the small intestine, extends for about 2.4 m. The ileum, the terminal end of the small intestine, is approximately 3.6 m long and meets the large intestine at the ileocaecal valve.

Food is chemically digested, and most of it is absorbed, as it moves through the small intestine. Circular folds (deep folds of the mucosa and submucosa layers), villi (finger-like projections of the mucosa cells) and microvilli (tiny projections of the mucosa cells) increase the surface area of the small intestine to enhance absorption of food. Although up to 10 L of food, liquids and secretions enter the GI tract each day, less than 1 L reaches the large intestine.

Enzymes in the small intestine break down carbohydrates, proteins, lipids and nucleic acids. Pancreatic amylase acts on starches, converting them to maltose, dextrins and oligosaccharides; the intestinal enzymes dextrinase, glucoamylase, maltase, sucrase and lactase further break down these products into monosaccharides. Pancreatic enzymes (trypsin and chymotrypsin) and intestinal enzymes continue to break down proteins into peptides. Pancreatic lipases digest lipids in the small intestine. Triglycerides enter as fat globules and are coated by bile salts and emulsified. Nucleic acids are hydrolysed by pancreatic enzymes and then broken apart by intestinal enzymes. Both pancreatic enzymes and bile are excreted into the duodenum in response to the secretion of secretin and cholecystokinin, hormones produced by the intestinal mucosa cells when chyme enters the small intestine.

Nutrients are absorbed through the mucosa of the intestinal villi into the blood or lymph by active transport, facilitated transport and passive diffusion. Almost all food products and water, as well as vitamins and most electrolytes, are absorbed in the small intestine, leaving only indigestible fibres, some water and bacteria to enter the large intestine.

The large intestine

The large intestine, or colon, begins at the ileocaecal valve and terminates at the anus (see Figure 20.5). It is about 1.5 m long. The large intestine frames the small intestine on three sides and

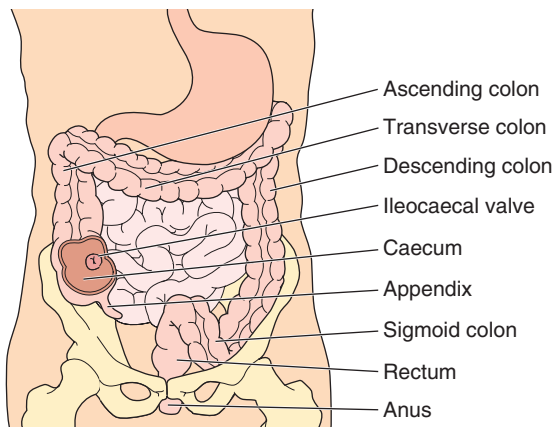


FIGURE 20.5 ■ Anatomy of the large intestine

includes the caecum, the appendix, the colon, the rectum and the anal canal.

The first section of the large intestine is the caecum. The appendix is attached to its surface as an extension. The appendix, a twisted structure in which bacteria can accumulate, may become inflamed.

The colon is divided into ascending, transverse and descending segments. The ascending colon extends along the right side of the abdomen to the hepatic flexure, where it makes a right-angled turn. The next segment, called the transverse colon, crosses the abdomen to the splenic flexure. At this juncture, the descending colon descends down the left side of the abdomen and ends at the S-shaped sigmoid colon. The sigmoid colon terminates at the rectum.

The rectum is a mucosa-lined tube approximately 12 cm in length (see Figure 20.6). The rectum has three transverse folds (valves of Houston) that retain faeces yet allow flatus to be passed through the anus. The rectum ends at the anal canal, which terminates at the anus.

The anus is a hairless opening at the end of the digestive system, from which bowel motions (stools) are passed. It has both an internal involuntary sphincter and an external voluntary sphincter. The sphincters are usually open only during defecation. The anorectal junction separates the rectum from the anal canal and may be the site of internal haemorrhoids (clusters of dilated veins in swollen anal tissue).

The major function of the large intestine is to eliminate indigestible food residue from the body. The large intestine absorbs water, salts and vitamins formed by the food residue and bacteria. The semiliquid chyme that passes through the ileocaecal valve is formed into faeces as it moves through the large intestine. Faeces are moved along the intestine by peristalsis**,** waves of alternating contraction and relaxation. Goblet cells lining the large intestine secrete mucus that facilitates the lubrication and passage of faeces.

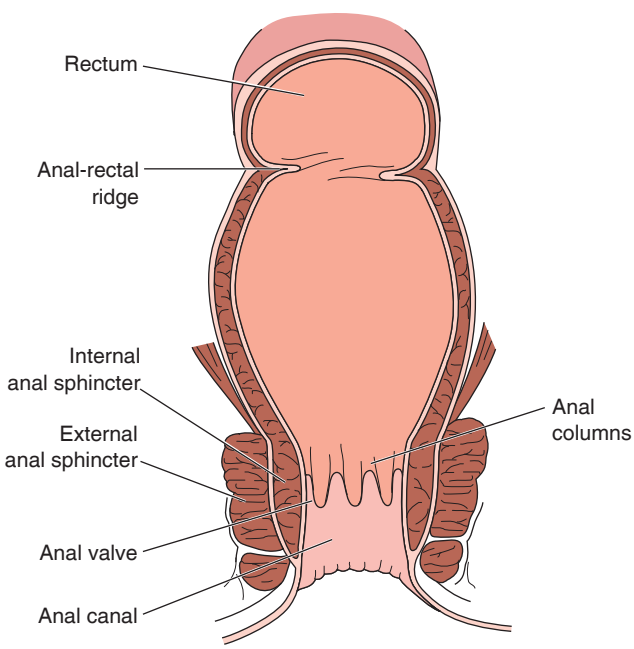


FIGURE 20.6 ■ Structure of the rectum and anus

The defecation reflex is initiated when faeces enter the rectum and stretch the rectal wall. This spinal cord reflex causes the walls of the sigmoid colon to contract and the anal sphincters to relax. This reflex can be suppressed by voluntary control of the external sphincter. Closing the glottis and contracting the diaphragm and abdominal muscles to increase intra-abdominal pressure (**Valsalva manoeuvre**) facilitate expulsion of faeces. Prolonged suppression of defecation can result in a weakened reflex that may in turn lead to **constipation** (infrequent and often uncomfortable passage of hard, dry stool). Frequent bouts of constipation may lead to external haemorrhoids at the area of the external haemorrhoidal plexus.

The accessory digestive organs

The liver, gallbladder and exocrine pancreas secrete substances necessary for the digestion of chyme. The liver produces bile, necessary for fat digestion and absorption, and stores it in the gallbladder. The liver also receives nutrients absorbed by the small intestine and metabolises or synthesises these nutrients so they are in a form that can be used by the cells of the body. The exocrine pancreas produces enzymes necessary for digestion of fats, proteins and carbohydrates.

The liver and gallbladder

The liver weighs about 1.4 kg in the average-size adult. It is located in the right side of the abdomen, inferior to the diaphragm and anterior to the stomach (see Figure 20.1). The liver has four lobes: right, left, caudate and quadrate. A mesenteric ligament separates the right and left lobes and suspends the liver from the diaphragm and anterior abdominal wall. The liver is encased in a fibroelastic capsule, called the Glisson capsule. This capsule contains blood vessels, lymphatics and nerves. When the liver is diseased or swollen, distension causes pain and the lymphatics may ooze fluid into the peritoneal cavity.

Liver tissue consists of units called lobules, which are composed of plates of hepatocytes (liver cells). A branch of the hepatic artery, a branch of the hepatic portal vein and a bile duct communicate with each lobule. Sinusoids, blood-filled spaces within the lobules, are lined with Kupffer cells. These phagocytic cells remove debris from the blood.

Bile production is the liver's primary digestive function. **Bile** is a greenish, watery solution containing bile salts, cholesterol, bilirubin, electrolytes, water and phospholipids. These substances are necessary to emulsify and promote the absorption of fats. Liver cells make from 700 to 1200 mL of bile daily. When bile is not needed for digestion, the sphincter of Oddi (located at the point at which bile enters the duodenum) is closed and the bile backs up the cystic duct into the gallbladder for storage.

Bile is concentrated and stored in the gallbladder, a small sac cupped in the inferior surface of the liver. When food containing fats enters the duodenum, hormones stimulate the gallbladder to secrete bile into the cystic duct. The cystic duct joins the hepatic duct to form the common bile duct, from which bile enters into the duodenum (see Figure 20.4).

The major digestive and metabolic functions of the liver are outlined in Box 20.1. These functions require a large amount of blood, with the liver receiving blood from both venous and arterial blood vessels. The hepatic artery, branching from the abdominal aorta, provides oxygenated blood at the rate of 400 to 500 mL/min. The hepatic portal vein delivers about 1000 to 1200 mL/min of deoxygenated blood to the liver from the inferior and superior mesenteric veins and the splenic vein.

The exocrine pancreas

The pancreas, a gland located between the stomach and small intestine, is the primary enzyme-producing organ of the digestive system. It is a triangular gland extending across the abdomen, with its tail next to the spleen and its head next to the duodenum (see Figure 20.4). The body and tail of the pancreas are retroperitoneal, lying behind the greater curvature of the stomach. The pancreas is actually two organs in one, having both exocrine and endocrine structures and functions. The exocrine portion of the pancreas, through secretory units called acini, secretes alkaline pancreatic juice containing many different enzymes. The acini, clusters of secretory cells surrounding ducts, drain into the pancreatic duct. The pancreatic duct joins with the common bile duct just before it enters the duodenum (so that pancreatic juice and bile from the liver enter the small intestine together). The pancreas also has endocrine functions, discussed in Chapter 17.

The pancreas produces from 1 to 1.5 L of pancreatic juice daily. Pancreatic juice is clear and has a high bicarbonate content. This alkaline fluid neutralises the acidic chyme as it enters the duodenum, optimising the pH for intestinal and pancreatic enzyme activity. The secretion of pancreatic juice is controlled by the vagus nerve and the intestinal hormones secretin and cholecystokinin. Pancreatic juice contains enzymes that aid in the digestion of all categories of foods: lipase promotes fat breakdown and absorption; amylase completes starch digestion; and trypsin, chymotrypsin and carboxypeptidase are responsible for half of all protein digestion. Nucleases break down nucleic acids.

BOX 20.1 Major digestive and metabolic functions of the liver

- Secretes bile.
- Stores fat-soluble vitamins (A, D, E and K).
- Metabolises bilirubin.
- Stores blood and releases blood into the general circulation during haemorrhage.
- Synthesises plasma proteins to maintain plasma oncotic pressure.
- Synthesises prothrombin, fibrinogen and factors I, II, VII, IX and X, which are necessary for blood clotting.
- Synthesises fats from carbohydrates and proteins to be either used for energy or stored as adipose tissue.
- Synthesises phospholipids and cholesterol necessary for the production of bile salts, steroid hormones and plasma membranes.
- Converts amino acids to carbohydrates through deamination.
- Releases glucose during times of hypoglycaemia.
- Takes up glucose during times of hyperglycaemia and stores it as glycogen or converts it to fat.
- Alters chemicals, foreign molecules and hormones to make them less toxic.
- Stores iron as ferritin, which is released as needed for the production of red blood cells.

METABOLISM

After nutrients (carbohydrates, fats and proteins) are ingested, digested, absorbed and transported across cell membranes, they must be metabolised to produce and provide energy to maintain life. **Metabolism** is the process of biochemical reactions occurring in the body's cells. Metabolic processes are either catabolic or anabolic. Catabolism involves the breakdown of complex structures into simpler forms—for example, the breakdown of carbohydrates to produce ATP, an energy molecule that fuels cellular activity. In the process of anabolism, simpler molecules combine to build more complex structures—for example, amino acids bond to form proteins.

The biochemical reactions of metabolism produce water, carbon dioxide and ATP (see Figure 20.2D). The energy value of foods is measured in calories or joules; in Australia the preferred unit of measure is kilojoules (kJ). One kilojoule is equivalent to 0.24 kilocalorie. A kilocalorie is defined as the amount of heat energy needed to raise the temperature of 1 kilogram (kg) of water 1 degree Celcius (1°C).

ASSESSING NUTRITIONAL STATUS AND GASTROINTESTINAL FUNCTION

Nutritional status and the function of the gastrointestinal system are assessed by findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests. The obesity epidemic has put **bariatric care** in the forefront for all healthcare providers. Bariatric nurse specialists reveal that obese individuals are often the target of prejudice, bullying and ridicule, and that misunderstanding by the community, media and health professionals is common. People affected in this way are often reluctant to seek care or to follow medical advice. Person-centred care and collaboration with the affected person and their family is essential to positive long-term outcomes. Sensitive attention to health assessment interviews and physical assessment of overweight and obese people is required to identify those at risk of nutritional and neurological deficits and in need of specialist support.

See the box below for sample documentation of a nutritional status assessment.

Health assessment interview

A health assessment interview to determine problems with nutrition and GI function may be conducted during a health screening, may focus on a chief complaint (such as nausea or unexplained weight loss) or may be part of a total health assessment. People may feel embarrassed to talk about weight, digestion and bowel elimination patterns. Remember to request permission to conduct the interview. Due to the sensitive nature of many of the questions, ask about the presence of others during the assessment. To promote effective rapport, ask about less personal information first. It is important to identify whether the problem is acute (less than 3 months) or chronic (more than

SAMPLE DOCUMENTATION

Assessment of nutritional status

28/02/2016
NURS
1100 hrs

A 22-year-old female visiting her GP for a regular check-up. Height 165 cm; weight 58 kg. BMI: 24. MAC: 28 cm. Waist-to-hip ratio: 0.6. Skin is warm, moist and smooth without lesions other than well-healed scar on RLQ of abdomen from appendectomy, age 15. Oral mucosa and tongue pink and moist. No breath odour. All teeth present with evidence of dental care. Abdomen slightly concave when lying on back, bowel sounds present in all four quadrants, liver non-palpable, tympany over lower abdomen on percussion.——————— RN Brown

(S BROWN RN)

3 months). If there has been a change in the person's normal GI function lasting for 4 weeks or longer, ask if this is related to any change in their lifestyle. Questions could cover diet, fluids, medication (prescribed, complementary and over-the-counter medicines), personal stresses such as a change in job, recent foreign travel (especially if a person has diarrhoea) and surgery.

If the person has a health problem involving nutrition and GI function, analyse its onset, characteristics and course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, the nurse may ask the person:

- Have you had any episodes of indigestion, nausea, vomiting, diarrhoea or constipation? If so, describe the appearance of what was vomited or the stools and anything that makes these problems better or worse. How long have you had these problems?
- What do you usually eat and drink (include alcohol) during a 24-hour period? Has your dietary pattern changed recently? If so, please describe.
- Are you generally happy with your diet? If necessary, ask person to describe what they believe to be a 'healthy' diet.

Also consider red-flag symptoms for bowel cancer, such as blood mixed in with stools, an increase in mucus and wind, weight loss without dieting, feeling tired and a family history. For example, ask the person:

- Do you have cramping or abdominal pain? If so, please ask for a description of type frequency and quality.
- Have you ever had any bleeding from your rectum? If so please describe the amount and colour of the blood (for example, was it bright red or dark?).

When collecting information about the person's current health status, ask about any changes in weight, appetite and the ability to taste, chew or swallow. What is the person's perception of the role of nutrition in maintaining health? Who buys and prepares the food? What medications (prescribed, over-the-counter or complementary) is the person currently taking? Does the person take any vitamins, herbal supplements or other 'health food' items? Does the person consume alcohol or recreational drugs (how much, what type, how often)? If the person has experienced nausea or vomiting, ask whether the vomitus contains bright red blood, dark (old) blood, bile or faecal material. If the person is very thin or expresses concerns about body size incongruent with the ratio of height to weight, ask whether the person induces vomiting or uses laxatives to control weight. Ask whether the person has dental appliances, such as braces, bridges or dentures, and what self-care measures are used for them, as well as oral hygiene practices and frequency of dental visits.

CONSIDERATION FOR PRACTICE

Culture influences all aspects of one's life. It is a learned system of beliefs, values and rules that people use to guide their activities. Culture shapes behaviours, attitudes and beliefs about being fat, being thin, eating, food nutrition, activity and exercise. Culture provides the foundation for how you view your own body as well as someone else's body. The term 'fat' is influenced by culture. Body weight is looked at differently from one culture to another.

Ask the person to describe any heartburn, indigestion, abdominal discomfort or pain. Explore the location of the pain, the type of pain, the time it occurs, foods that aggravate or relieve it, and how it is relieved. Abdominal pain is often referred to other sites (see Chapter 8 for further information on pain assessment). For example, a person with a liver disorder may experience pain over the right shoulder (Kehr's sign). Epigastric (middle upper abdominal) pain is experienced in cases of acute gastritis, obstruction of the small intestine and acute pancreatitis. Pain in the right upper quadrant is associated with cholecystitis. Pain in the left upper quadrant may be related to a gastric ulcer. Sudden onset of lower abdominal cramping often occurs in obstruction of the colon. Left lower abdominal pain may be associated with diverticulitis. Rectal pain may occur with stool retention and/or haemorrhoids. Determine whether the person has had any lower abdominal pain or rectal pain, which may be associated with a distended colon filled with gas or fluid. Crampy, colicky pains occur with diarrhoea and/or constipation.

Ask about any medical conditions that may influence the person's bowel elimination pattern, such as stroke or spinal cord impairment, inflammatory gastrointestinal diseases, endocrine disorders, cancer and allergies. Note any recent travel to other countries. Information about the person's psychosocial history and cultural background is also important. Assess the person's lifestyle for any patterns of psychological stress and/or depression, which may alter bowel elimination. Depression may be associated with constipation, whereas **diarrhoea** (frequent passage of loose, watery stools) may occur in situations of high stress and anxiety. Explore the person's activities of daily living (ADLs), including exercise, sleep–rest patterns and dietary and fluid intake. Changes in ADLs can influence bowel elimination patterns. More women than men seek help for constipation, suggesting that hormonal changes may play a role. Ask about history of diarrhoea, constipation or bleeding from the rectum, and collect information about the use of medications, laxatives, suppositories or enemas. For example, anticholinergic drugs, antihistamines, tranquillisers or narcotics may cause constipation.

The health history should include questions about any prior surgeries or trauma of the GI tract. Explore the history (personal

FUNCTIONAL HEALTH PATTERN INTERVIEW — Nutritional status and gastrointestinal system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Have you had any illness or surgery that affects your nutrition and gastrointestinal (GI) function? If so, how were these treated? ■ Describe your current problem; how long has it lasted; what have you done to treat it? ■ What medications do you take? Do you take antacids? If so, what do you use them for and how often do you take them? ■ Are you currently taking alternative or complementary medicines, such as herbs, homeopathics, traditional medicines? If so, what do you use them for and how often do you take them? ■ Do you have allergies to foods? What are they and how do you react? How do you manage them? ■ Do you have your own teeth? If not, please describe any gaps, replacement teeth/dentures. ■ Do you have tooth or gum problems or pain that interferes with your ability to eat? ■ When was your last dental examination? ■ Describe what you do each day to take care of your teeth.
Nutritional–Metabolic	■ Describe what you eat and how much (and type) of fluids you drink in a 24-hour period. ■ Describe dietary supplements, such as vitamins, minerals, tonics, herbs, that you currently are taking. ■ Have you noticed any change in your appetite recently? Explain. ■ What is your current weight? What do you feel your ideal weight would be? Have you had a recent gain or loss? Explain. ■ Describe your food likes and dislikes. ■ Do you have any of the following: indigestion, belching, nausea, vomiting, difficulty swallowing? If so, what causes this and how do you treat it? ■ Do you drink alcohol? If so, what type? Describe your average number of drinks each day. ■ Questions specific to the person's culture and ethnic group would be included in this functional health pattern area, such as what types of food are preferred or not eaten, what types of foods are never eaten together and what types of foods are eaten to remain healthy.
Elimination	■ What is the pattern of your bowel movements? Has it been affected by your condition/illness? ■ Ask women of reproductive age if their menstrual cycle affects their bowel movements. If so, how? ■ Do you use laxatives, suppositories, enemas or other substances to help the regularity of your bowel movements? Please describe. ■ Are your bowel movements affected by what you eat? Explain. ■ Have you noticed any change in the colour of your urine or bowel movements? Explain. ■ Have you ever used laxatives or made yourself vomit to control your weight? Explain.
Activity–Exercise	■ Describe your activities on a typical day. ■ What type of exercise do you get, and how often? ■ Do you smoke? If so, what and how many cigarettes per day? ■ Do you use recreational drugs? If so, what type, amount and how often?
Sleep–Rest	■ Do you wake up hungry during the night? ■ Does abdominal pain, cramping, nausea or diarrhoea ever interfere with your sleep? Explain.
Cognitive–Perceptual	■ Describe the amount and type of foods you should eat each day. ■ Rate your ability to taste and smell foods on a scale of 1 to 10 (with 10 being excellent). ■ Describe any pain you have had in your mouth, stomach or abdomen. What type of pain was it (dull, crampy, achy, burning)? What seems to cause it? What do you do to relieve it? ■ How do you prefer to receive your health information? Written, verbal, internet, mixture? Please explain. ■ Do you have any difficulties reading and understanding health information? If so, what or who helps you to understand and feel well informed?

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Self-perception–Self-concept	■ How does this problem/condition make you feel about yourself? ■ Are you satisfied with your appearance in terms of weight? If not, why? ■ Have you tried gaining or losing weight? If so, what worked or didn't work for you?
Role–Relationships	■ How does this condition affect your relationships with others? ■ Who normally buys food and prepares your meals? ■ Do you eat with others regularly? If so, who? Where do you normally eat your meals?
Sexuality–Reproductive	■ Has this condition affected your usual sexual activities? Can you describe your concerns and what you do to manage? ■ Ask women of reproductive age if their menstrual cycle is affected by this condition? If so, please describe what happens, and how you manage it.
Coping–Stress tolerance	■ Have you experienced any type of stress that may have worsened this condition? ■ Has having this condition created stress for you? ■ Describe what you do when you feel stressed.
Value–Belief	■ Tell me how specific relationships or activities help you cope with this problem. ■ Do you have specific cultural beliefs or practices that affect how you care for and feel about this condition? Please describe. ■ Are there any specific treatments that you would not use to treat this condition? ■ Are your spiritual beliefs, needs or practices affected by this condition/illness? Please describe.

and family) of any medical condition that may affect the person's ingestion, digestion and/or metabolism (e.g. Crohn's disease, diabetes mellitus, irritable bowel syndrome, peptic ulcers or pancreatitis). Other areas significant to the assessment of nutritional status and the gastrointestinal system are food allergies (especially to milk, which is evidenced as lactose intolerance with abdominal cramping, excessive flatus and loose stools), and a family history that may provide clues to increased risk of health problems.

If the person has an **ostomy** (surgical opening into the bowel), ask about skin care problems, consistency of stool, foods that cause problems with diarrhoea or **flatus** (intestinal gas), the number of times that the person empties the appliance bag each day and irrigation habits. It is also important to explore the person's feelings about the appliance.

To obtain information about the person's nutritional status, ask about changes in weight, appetite, food preferences, fibre content, food intolerances (wheat or dairy products—possible intolerance), special or supplementary diets (may cause diarrhoea), spicy foods (often increase gut motility) and any cultural or ethnic influences on dietary intake. The person could complete a food diary before attending for a clinic or outpatient visit. Ask about regular fluid intake—for example, amount of coffee, diet drinks, sports drinks, excess alcohol; these may increase gut motility, giving a loose stool. Ask whether the person experiences nausea and vomiting; if so, determine any relation to food intake and ask the person to describe the character of the emesis. In addition, ask about indigestion, the use of antacids or other over-the-counter medications, herbal preparations and episodes of diarrhoea and its character. Also ask about the use of analgesic drugs prescribed or other drugs; for example, narcotics slow bowel motility and can lead to chronic constipation, and anti-inflammatory drugs can irritate the gut and bowel.

Explore any family history of colon cancer, colitis, gallbladder disease or malabsorption syndromes, such as lactose intolerance and coeliac sprue. Assess the person's risk factors for cancer, including age greater than 50; family member with colon cancer; history of endometrial, ovarian or breast cancer; and previous diagnoses of colon inflammation, polyps or cancer.

People with cognitive problems require careful assessment. For example, if a person is unable to retain information or change behaviour, there is little point in teaching anal exercises to improve the strength of the pelvic floor muscles as these are unlikely to be carried out. An assessment of cognitive function should include the person's ability to understand what is being explained, retain information and learn, change behaviour and carry out instructions at home.

In addition to other factors assessed in the health history, culture and ethnicity are important components of nutritional status and gastrointestinal health. Australia is culturally and ethnically diverse and, as such, numerous cultural food customs can be seen throughout the country. Nutritional diversity is common among cultural and ethnic groups, and questions should be included to identify specific customs, food likes and dislikes, and how foods are prepared and served. In some ethnic groups, for example, dietary substances are used to protect health, such as eating raw garlic or onions (Spector, 2012). In Indigenous Australian culture, food is often depicted in art and stories, highlighting how important food and the collecting and sharing of it are in this culture. In other cultures, dietary balance is believed to be necessary to keep the body in balance or harmony. It is therefore necessary for nurses to be mindful of specific culturally related nutritional values and practices and to ask questions to identify health-related concerns specific to individualised dietary intake. If English is not

the person's first language it is important to assess oral and written language fluency, ask which language they prefer to receive their health information in and use interpreters where necessary.

Interview questions categorised by functional health patterns are listed in the box above.

Physical assessment

Begin by explaining what will happen during the physical examination, and encourage the person to take deep, regular breaths to increase relaxation. If a rectal examination is included, explain that it may be uncomfortable may feel as though they might be about to have a bowel movement, and sometimes flatus (gas) is passed. Assure the person that this is normal. Ensure that the examination area is private and the person is draped properly to prevent unnecessary exposure. Remember to ask the person's permission to proceed with a physical examination.

Physical assessment of gastrointestinal and nutritional status may be performed as part of a total health assessment, as a focused assessment of people with known or suspected health problems, in combination with assessment of the urinary and reproductive systems (problems that may cause manifestations similar to those of the gastrointestinal system), or alone for people with known or suspected health problems. The techniques of inspection, auscultation, percussion and palpation are used. Palpation is the last method used in assessing the abdomen.

CONSIDERATION FOR PRACTICE

When assessing the abdomen, use palpation last, because pressure on the abdominal wall and contents may interfere with bowel sounds and cause pain, ending the examination.

Collect objective data by obtaining anthropometric measurements (height, weight, triceps skin folds and midarm circumference) and by examining the mouth and abdomen. Prior to the examination, collect all necessary equipment and explain techniques to the person to decrease anxiety. The person may be seated during assessment of the mouth, but is supine during the abdominal assessment. Older adults or those with limited mobility may need assistance with positioning.

Physical assessment of the integumentary system, nervous system, musculoskeletal system, cardiovascular system and respiratory system may also reflect the person's nutritional status. Table 20.5 summarises abnormal nutritional assessment findings related to these body systems. Normal age-related findings for the older adult are summarised in Table 20.6.

Diagnostic tests

The results of diagnostic tests of nutritional status and gastrointestinal function are used to support the diagnosis of a specific disease, to provide information to identify or modify the appropriate medication or therapy used to treat the disease and to help nurses monitor the person's responses to treatment and nursing care interventions. More information, including specific laboratory tests, is included in the discussion of disorders in Chapters 21, 22, 23 and 24.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, for supporting the person during the examination as necessary, for documenting the procedure as appropriate and for monitoring the results of the test.

Genetic considerations

When conducting a health assessment interview and physical assessment, it is important for the nurse to consider genetic influences on the health of the adult. During the health assessment interview, ask about family members with known abnormalities of copper accumulation in the body, hypercholesteraemia, abnormal cholesterol or fat metabolism, obesity or cancer of the pancreas. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the accompanying 'Genetic considerations' box). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

TABLE 20.5 Assessment findings due to malnutrition

BODY SYSTEM	ASSESSMENT FINDINGS
Nails	Soft and spoon shaped in iron deficiency. Splinter haemorrhages in vitamin C deficiency.
Hair	Dry, dull and scarce in zinc, protein and linoleic acid deficiencies.
Skin	Flaky and dry in vitamin A, vitamin B and/or linoleic acid deficiency. Cracks and/or hyperpigmentation in niacin deficiency. Bruising in vitamin C or vitamin K deficiency.
Eyes	Eyes become dry and soft with decrease in vitamin A. Conjunctiva is pale with a decrease in iron and red with a decrease in riboflavin.
Nervous system	Reflexes are decreased and person may have peripheral neuropathies with thiamine deficiency. Person may be irritable and/or disoriented with thiamine deficiency.
Musculoskeletal system	Muscle wasting is seen with deficits in protein, carbohydrate and fat metabolism. Calf pain occurs with thiamine deficiency; joint pain may occur with vitamin C deficiency.
Cardiovascular system	Heart size and rate may increase with thiamine deficiency. Diastolic blood pressure may be increased with a high intake of fat. Lowered cardiac output and decreased blood pressure may occur with kilojoule deficiencies over a long time period.
Gastrointestinal system	Cheilosis (sores at corner of mouth) seen in vitamin-B-complex deficiencies, especially riboflavin. Stomatitis and spongy, bleeding gums may also be seen in malnutrition.

TABLE 20.6 Age-related gastrointestinal changes

AGE-RELATED CHANGE	SIGNIFICANCE
Teeth: ↑ number of root cavities and cavities around existing dental work; tooth enamel harder and more brittle; dentin is more fibrous; tooth cusps flatten; root pulp shrinks; ↑ loss of bone supporting teeth	Increase in periodontal disease and tooth loss Increase in fractures of teeth Increased incidence of dentures, caps, tooth implants
Gums: gingiva retracts	Increase in periodontal disease
Taste: less acute as tongue atrophies, especially for sweet sensations	Excessive seasoning of foods
Saliva: ↓ amount is produced (one-third of that produced in younger years)	Decreased ability to break down starches Swallowing may take longer
Oesophageal motility: ↓ intensity of propulsive waves and slower emptying time, weaker gag reflex	Discomfort when swallowing food Increased risk of aspiration
Stomach: mucosa atrophies, ↓ production of hydrochloric acid and pepsin leading to higher pH in stomach	Increase in incidence of gastric irritation
Liver: less efficient handling of cholesterol	Increased incidence of gallstones

DIAGNOSTIC TESTS Gastrointestinal disorders

OESOPHAGEAL AND STOMACH TESTS

NAME OF TEST Oesophageal acidity, Oesophageal manometry, Acid perfusion (Bernstein test)

PURPOSE AND DESCRIPTION Oesophageal acidity is measured to diagnose problems of the lower oesophageal sphincter and chronic reflux oesophagitis. A catheter with a pH electrode is inserted into the oesophagus through the mouth. The measurement may be one time, or over a 24-hour period.

Oesophageal manometry is done to measure oesophageal sphincter pressure and peristaltic contractions for diagnosis of oesophageal motility problems, such as achalasia. A manometric catheter with a pressure transducer is inserted into the oesophagus through the mouth and oesophageal pressure is measured before and after swallowing.

Acid perfusion (Bernstein test) tests are performed to distinguish between gastric acid reflux and cardiac involvement. A nasogastric tube is inserted through the nose into the oesophagus. A saline solution, followed by a hydrochloric acid (HCl) solution, is dripped into the catheter and the person is asked to indicate when pain occurs. Normal oesophageal pH is 5 to 6.

RELATED NURSING CARE Advise the person to be nil by mouth (NBM) and to avoid alcohol intake for 8–12 hours prior to the exam. Assess medications: results of the tests may be affected by antacids, anticholinergics and cimetidine-like drugs, which increase the pH, reducing acidity and causing false test results.

NAME OF TEST Barium swallow or Upper GI series (see Figure 20.7)

PURPOSE AND DESCRIPTION To diagnose oesophageal varices, inflammation, ulcerations, hiatal hernia, foreign bodies, polyps, diverticula and tumours of the oesophagus, stomach and duodenal bulb. These radiological studies are done by observing the movement of a contrast medium with a fluoroscope.

RELATED NURSING CARE Advise the person to be NBM and avoid smoking for 8–12 hours before the exam; person will drink 450–550 g of barium sulfate (a non-water-soluble chalky liquid) or meglumine diatrizoate (Gastrografin, a water-soluble liquid) before the exam. Withhold medications for 8 hours before the exam, according to the medical officer's directions. Following the exam, ensure the person eliminates the barium by giving laxatives and forcing fluids as appropriate.

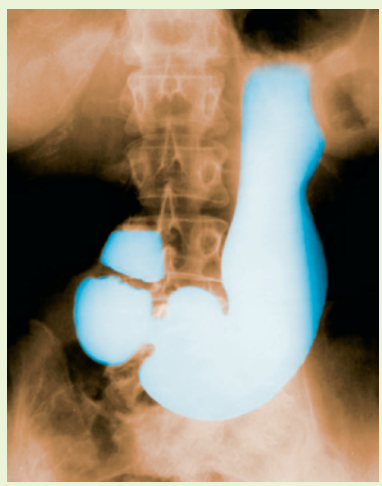


FIGURE 20.7 ■ A barium x-ray of a healthy stomach

Source: Biophoto Associates/Science Source.

(continued)

DIAGNOSTIC TESTS Gastrointestinal disorders (continued)

NAME OF TEST Barium enema

PURPOSE AND DESCRIPTION An x-ray examination of the lower gastrointestinal (GI) tract, it is used to diagnose inflammation, ulcerations, structural changes, diverticula and tumours of the lower GI and investigations of pain and anaemia. These radiological studies are done by observing the movement of a contrast medium, which is inserted through a tube in the rectum into the colon.

RELATED NURSING CARE Preparation for the test requires emptying of the large intestine. The nurse will advise the person to follow a clear fluid diet for 1–3 days prior to the examination. The day prior to the test the person will be required to take a combination of oral laxatives and an enema (an additional enema may be required on the day). Following the exam, the person will resume a normal diet. If the person has not eliminated the barium a laxative may be required.

NAME OF TEST Colonoscopy

PURPOSE AND DESCRIPTION An examination of the lower GI tract, used to diagnose cancer, inflammation, ulcerations, structural changes and investigations of pain, bowel changes and anaemia. The colonoscope is used to directly visualise the colon and rectum; a biopsy can be taken during the examination if an abnormal growth is visualised.

RELATED NURSING CARE Preparation for the test requires emptying of the large intestine. The nurse will advise the person to follow a clear fluid diet for 1–3 days prior to the examination. The day prior to the test the person will be required to take a combination of oral laxatives and an enema (an additional enema may be required on the day). Inform the person a light sedative is given during the exam. Following the exam, the person will resume a normal diet; inform the person flatus is quite common post procedure. The person will require someone to drive them home following the test due to the sedatives used during the examination.

NAME OF TEST Upper GI endoscopy (Oesophagogastroduodenoscopy (OGD))

PURPOSE AND DESCRIPTION To directly visualise mucous membrane lining of the oesophagus, stomach and duodenum. A flexible fibre-optic endoscope is used to visualise inflammations, ulcerations, tumours or varices; and video imaging may illustrate gastric motility. May also be combined with an ultrasound examination by attaching an ultrasound transducer to the endoscope.

RELATED NURSING CARE Schedule at least 2 days after barium swallow or upper gastrointestinal series. Ensure the informed consent is signed prior to premedication. Encourage questions and provide answers and support.

Keep the person NBM for 6–8 hours before the procedure. Remove dentures and eyewear. Follow routine preoperative checklist.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Do not eat or drink anything for 6–8 hours before the procedure.
- The procedure is somewhat uncomfortable but requires only 20–30 minutes to complete.
- A local anaesthetic will be used in your throat and you will be given a sedative during the procedure.
- After the procedure, you will be allowed to eat and drink as soon as your gag reflex returns and you are able to swallow.
- You may experience mild bloating, belching or flatulence following the procedure.
- Contact your doctor immediately if you develop any of the following: difficulty swallowing; epigastric, substernal or shoulder pain; vomiting blood; black tarry stools; or fever.

NAME OF TEST Magnetic resonance imaging (MRI)

PURPOSE AND DESCRIPTION A scan that uses magnetic and radio waves to identify a source of gastric bleeding, tumours or cysts.

RELATED NURSING CARE Inform the person of need to lie still during the examination. Remove any metallic objects (such as hair clips, jewellery) and assess for any metallic implants (such as pacemakers, body piercings, shrapnel). If present, test is not performed due to the danger caused by the magnetic field from the machine. Explain the need to be NBM for at least 6 hours prior to the exam.

NAME OF TEST Gastroscopy

PURPOSE AND DESCRIPTION See Upper GI endoscopy.

RELATED NURSING CARE See Upper GI endoscopy.

DIAGNOSTIC TESTS Gastrointestinal disorders (continued)

NAME OF TEST Gastric analysis
Normal values:
Fasting: 1.0–5.0 mEq/L/per hour
Stimulation: 10–25 mEq/L/per hour

PURPOSE AND DESCRIPTION To evaluate gastric secretions and detect an increase or decrease of free hydrochloric acid. To conduct the gastric analysis, a nasogastric tube is inserted into the stomach and specimens are aspirated to evaluate gastric acidity. A stimulation gastric analysis may follow, with a gastric stimulant (such as Histalog or pentagastrin) administered and several gastric samples aspirated.

RELATED NURSING CARE Advise person to remain NBM with no smoking for 8–12 hours prior to the exam. Assess medications and fluid intake: anticholinergics, cholinergics, adrenergic blockers, antacids, steroids, alcohol and coffee can alter results. Remove loose dentures. Insert nasogastric tube. Aspirate gastric contents at 15- to 20-minute intervals as ordered.

NAME OF TEST Gastric emptying studies

PURPOSE AND DESCRIPTION To evaluate the ability of the stomach to empty liquids or solids. In this nuclear imaging study, the person is asked to eat a cooked egg containing Tc^{-99m} (solids) or to drink orange juice with Tc^{-99m} (liquids). Sequential images are recorded with a gamma camera every 2 minutes for up to an hour. Emptying should occur within 70–125 minutes.

RELATED NURSING CARE Explain to person that the substances contain only very small amounts of radioactivity and are not hazardous.

GALLBLADDER AND PANCREAS TESTS

NAME OF TEST Abdominal ultrasound, Hepatobiliary ultrasound, Gallbladder ultrasound

PURPOSE AND DESCRIPTION Abdominal ultrasound is used to detect abdominal tumours, cysts and ascites.

Hepatobiliary ultrasound is used to visualise the biliary ducts and to detect subphrenic abscesses, cysts, tumours and cirrhosis of the liver.

Gallbladder ultrasound is used to detect gallstones.

These non-invasive procedures record ultrasound waves as they are reflected off body structures. A conductive gel is applied to the skin and a transducer placed on the area.

RELATED NURSING CARE Advise the person to remain NBM for 8–12 hours prior to the test.

NAME OF TEST Cholecystography (oral) (GB Series)
If the gallbladder cannot be visualised with an oral contrast substance, an IV cholangiography may be ordered. *If the person is also having GI x-rays with barium, the GB tests should be done first, because barium would interfere with the test.*

PURPOSE AND DESCRIPTION To detect gallbladder stones, inflammation or tumours and obstruction of the cystic duct. The evening before the test, radiopaque tablets (e.g. iopanoic acid (Telepaque), sodium ipodate (Oragrafin), iodoalphionic acid (Priodax) or iodipamide meglumine (Cholografin)) are given; the following morning x-rays are taken. A high-fat meal may be given after the fasting x-rays are completed and further x-rays taken to determine how fast the GB expels the dye.

RELATED NURSING CARE Advise person to eat a fat-free diet 24 hours prior to the test. No food or fluids except sips of water should be taken 12 hours before the test. Assess person for allergy to iodine, seafood or x-ray dye (many contain iodine).

NAME OF TEST Cholangiography

- Percutaneous transhepatic cholangiogram (PTC)
- Surgical cholangiogram

PURPOSE AND DESCRIPTION A PTC is done to evaluate filling of the hepatic and biliary ducts. Using local anaesthesia, the liver and bile duct is entered with a long needle (using fluoroscopy), bile is withdrawn and a contrast medium is injected into the bile duct.

During a surgical cholangiogram with general anaesthesia, contrast medium is injected into the common bile duct to evaluate filling of the common bile duct.

RELATED NURSING CARE Assess person for allergy to iodine, seafood or x-ray dye (many contain iodine). Monitor for bile leakage or haemorrhage following the tests. Normal preoperative routine is required for the surgical procedure.

NAME OF TEST Magnetic resonance cholangiopancreatography (MRCP)

PURPOSE AND DESCRIPTION This non-invasive MRI study is done to evaluate the biliary and pancreatic ducts.

RELATED NURSING CARE Assess for metal implants or pregnancy. (Test will not be done if present.)

(continued)

DIAGNOSTIC TESTS Gastrointestinal disorders (continued)

NAME OF TEST Computed tomography (CT)

PURPOSE AND DESCRIPTION A non-invasive procedure, using radiofrequency waves and a magnetic field; used to evaluate disorders of the gallbladder, pancreas, biliary tract and liver.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Endoscopic retrograde cholangiopancreatography (ERCP)

PURPOSE AND DESCRIPTION To directly visualise gastrointestinal structures and retrieve gallstones from the distal common bile duct, dilate structures and biopsy tumours. A fibre-optic endoscope is inserted (under fluoroscopy) through the mouth down the oesophagus, stomach and descending duodenum, and the common bile ducts and pancreatic ducts are cannulated. Contrast medium is injected into the ducts and structures are visualised.

RELATED NURSING CARE Advise the person to be NBM for 8 hours before the test. Following the test, assess vital signs and gag reflex and monitor for complications (such as pancreatitis).

NAME OF TEST Serum lipase

PURPOSE AND DESCRIPTION This blood test is used to measure the secretion of lipase by the pancreas.

Normal value: 0–160 Unit/L

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Serum amylase

PURPOSE AND DESCRIPTION This blood test is used to measure the secretion of amylase by the pancreas. It is used to diagnose acute pancreatitis, when amylase level peaks in 24 h and then drops to normal in 48 to 72 h.

Normal value: 0–130 Unit/L

RELATED NURSING CARE No special preparation is needed.

LIVER TESTS

NAME OF TEST Liver biopsy

PURPOSE AND DESCRIPTION To rule out metastatic cancer or to detect a cyst or cirrhosis of the liver. Using ultrasound, a biopsy needle is inserted into the liver and guided to the pathological site. See Figure 20.8.

RELATED NURSING CARE Related nursing care of the person having a liver biopsy is described below.

PREPARATION OF THE PERSON

- Review chart for signed consent form.
- Keep the person NBM as per policy, usually 4–6 hours pre-procedure.
- Assess and record baseline vital signs.
- Review prothrombin time (PT) and platelet count; administer vitamin K as ordered.
- Instruct to empty bladder immediately before the biopsy.
- Place in supine position on far right side of bed; turn head to left and extend right arm above head to improve access to the biopsy site.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Discuss preparation for the biopsy and expected sensations during the procedure.
- Hold your breath following expiration during needle insertion to keep diaphragm and liver high and stabilised in the abdominal cavity.
- Obtaining the tissue sample usually requires only 10–15 seconds; there may be some pain or discomfort during this time.
- Direct pressure is applied to the site immediately after the needle is removed; you will be placed on your right side to maintain site pressure.
- You may develop pain in the right shoulder as the anaesthetic loses effect.
- You will be monitored for bleeding after the procedure.
- Food and fluids are withheld for 2 hours after the biopsy; you then can resume your usual diet.
- Avoid coughing, lifting or straining for 1–2 weeks.

Note: A wide variety of blood tests are used to diagnose and monitor liver disease. These are discussed in appropriate interprofessional care sections in Chapter 24.

DIAGNOSTIC TESTS Gastrointestinal disorders (continued)

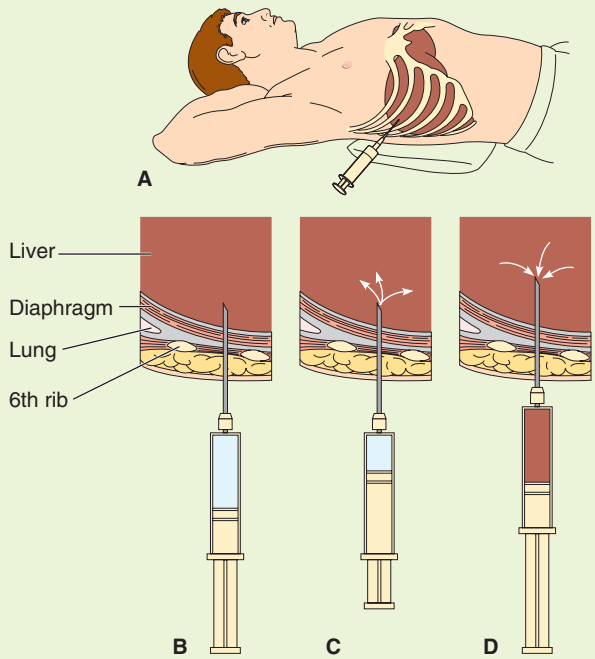


FIGURE 20.8 ■ Liver biopsy. *A*, The person exhales completely, and then holds their breath. This brings the liver and diaphragm to their highest position. *B*, The biopsy needle is inserted into the liver. *C*, Approximately 1 mL of saline is injected to clear the needle of blood and tissue. *D*, The needle is advanced and a tissue sample is aspirated. Pressure is applied to the site immediately after the needle is withdrawn. The specimen is sent to the laboratory for analysis

GENETIC CONSIDERATIONS Nutritional and gastrointestinal system

- Cleft lip and/or palate are influenced by genetic factors.
- An autosomal recessive disorder, Wilson's disease is an abnormality of copper transport, resulting in copper accumulation and toxicity to the liver and brain, resulting in neurological disease in adults.
- Colon cancer is one of the most common inherited cancer syndromes.
- Tangier disease is a disease of cholesterol transport, leading to characteristic orange tonsils, very low levels of high-density lipoprotein and an enlarged liver and spleen.
- Hypercholesterolaemia has a familial tendency.
- About 90% of human pancreatic cancers show a chromosome defect.
- Obesity is believed to result from a variety of factors, including genetics.
- Gaucher disease, more common in descendants of Jewish people from Eastern Europe, results in the lack of an enzyme to break down fats. Fats accumulate in the liver, spleen and bone marrow, causing pain, fatigue, jaundice, bone damage, anaemia and even death.
- Coeliac disease is a genetic, inherited disease responsible for the malabsorption of nutrients resulting in malnutrition. If people with coeliac disease eat certain types of proteins (glutens, found in wheat, barley, rye, and oats) an autoimmune response causes damage to the small intestine, so that nutrients are not absorbed.
- Due to a rapid change in diet, Indigenous Australians are prone to a group of conditions identified as 'insulin resistant syndrome' (also known as syndrome X). It includes renal disease, heart disease, obesity and type 2 diabetes (AIHW, n.d.).

NUTRITIONAL AND GASTROINTESTINAL ASSESSMENTS

Technique/normal findings	Abnormal findings

Anthropometric assessment

Weigh the person and measure the person's height. Compare the person's actual weight to ideal body weight (IBW) (see Table 20.7). *Weight should be appropriate to height as indicated on a standardised table.*

- A weight 10–20% less than ideal body weight indicates malnutrition.
- A weight 10% above ideal body weight is considered overweight.
- A weight 20% above ideal body weight is considered obese.

TABLE 20.7 Example of a height and weight table (IBW)

HEIGHT (CM)	HEIGHT (FT, IN)	WEIGHT (KG)	HEIGHT (CM)	HEIGHT (FT, IN)	WEIGHT (KG)
148	4'10"	44–55	173	5'8"	59–74
150	4'11"	45–56	175	5'9"	61–76
152	5'0"	46–58	178	5'10"	63–79
155	5'1"	48–60	180	5'11"	65–81
158	5'2"	50–62	183	6'0"	66–83
160	5'3"	51–64	185	6'1"	68–85
162	5'4"	52–66	188	6'2"	71–88
165	5'5"	54–68	190	6'3"	72–90
168	5'6"	66–71	193	6'4"	74–92
170	5'7"	58–72	196	6'5"	77–96

Calculate the person's percentage of ideal body weight (%IBW). Use the formula in Table 20.8 to determine the presence of obesity and/or malnutrition based on %IBW. *Ideal body weight should be within normal range.*

TABLE 20.8 Indications of nutritional status by body weight

%IBW	%UBW	NUTRITIONAL STATUS
> 120	—	Obese
110–120	—	Overweight
80–90	85–95	Mildly undernourished
70–79	75–84	Moderately undernourished
< 70	< 75	Severely undernourished

UBW = usual body weight

- Using %IBW may result in overlooking malnutrition in a very obese person.

Calculate the person's percentage of usual body weight (%UBW) to determine weight change, using this formula:

$$\frac{\text{Current weight}}{\text{Usual weight}} \times 100$$

Refer to Table 20.8 to determine nutritional status based on %UBW.

Measure body mass index (BMI). Determine BMI by using one of the following formulas. *BMI should be between 20 and 25.*

$$\frac{\text{Weight in kilograms}}{\text{Height in metres}^2} = \text{BMI}$$

$$\frac{\text{Weight in pounds} \times 705}{\text{Height in inches}^2} = \text{BMI}$$

- A BMI of 25–29.9 kg/m^2 indicates overweight.
- A BMI of 30 kg/m^2 and above indicates obesity.

Technique/normal findings	Abnormal findings
Measure triceps skinfold thickness (TSF). Find the midpoint between the person's olecranon and acromion processes. Grasp the skin and fat and pull it away from the muscle. Apply skinfold calipers for 3 seconds and record reading (see Figure 20.9). Repeat three times and average the three readings. Compare the person's reading with the standard values shown in Table 20.9. *TSF should be within normal range as compared to standard values.*	■ Triceps readings are 10% or more below standards in malnutrition and 10% or more above standards in obesity or overnutrition.

FIGURE 20.9 ■ Measuring TSF with calipers

Source: 3660 Group Inc./Getty Images.

TABLE 20.9 Values for anthropometric measurements

	STANDARD VALUE	
MEASUREMENT	MALE	FEMALE
Triceps skinfold thickness	12.5 mm	16.5 mm
Midarm circumference	29.3 cm	28.5 cm
Midarm muscle circumference	25.3 cm	23.2 cm

Measure midarm circumference (MAC). Find the midpoint between the person's olecranon and acromion processes. Wind tape measure around arm (see Figure 20.10). Compare the person's reading to the standard values shown in Table 20.9. *MAC should be within normal range as compared with standard values.*	■ MAC decreases with malnutrition and increases with obesity.

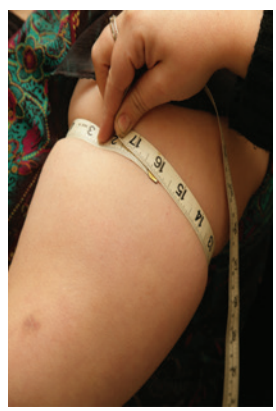


FIGURE 20.10 ■ Measuring MAC with a tape measure

Source: Bubbles Photolibrary/Alamy.

Calculate midarm muscle circumference (MAMC). Use the person's triceps skinfold measurement and midarm circumference readings to calculate the person's MAMC: $MAMC = MAC - (0.314 \times TSF)$ Compare the result to the standard values shown in Table 20.9. *MAMC should be within normal range as compared to standard values.*	■ In mild malnutrition, the MAMC is 90% of the standard; in moderate malnutrition, 60–90%. In severe malnutrition (muscle wasting), the MAMC is less than 60% of the standard.

Technique/normal findings	Abnormal findings
Determine waist-to-hip ratio. With the person standing, measure the waist and then measure the hips midway between the iliac crest and the greater trochanter. Use the formula below to calculate the waist-to-hip ratio. *Normal findings: females, waist ratio less than or equal to 0.80; males, waist ratio less than or equal to 1.0.*	■ Females with a ratio greater than 0.80 and males with a ratio greater than 0.9 have a greater risk of mortality from a cardiac-related death.

$$\frac{\text{Waist circumference}}{\text{Hip circumference}} = \text{waist-to-hip ratio}$$

Oral assessment

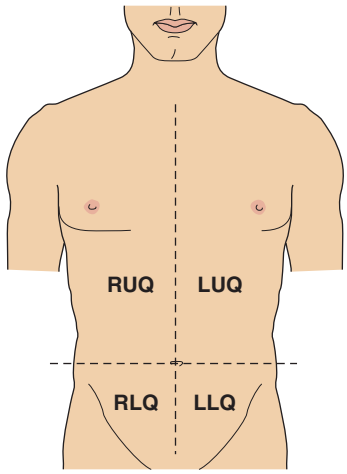
Inspect and palpate the lips. *Lips should be of normal colour for race without lesions.*	■ **Cheilosis** (painful lesions at corners of mouth) is seen with riboflavin and/or niacin deficiency. ■ Cold sores or clear vesicles with a red base are seen in herpes simplex 1.
Inspect and palpate the tongue. *Tongue should be pink, smooth and have good turgor.*	■ Atrophic smooth **glossitis** is characterised by a bright red tongue. It is seen in B_{12}, folic acid and iron deficiencies. ■ Vertical fissures are seen in dehydration. ■ A white or pale coated tongue during or after treatment with oral antibiotics can be caused by *Candida albicans* (the same pale fungus responsible for most vaginal yeast infections). ■ Black hairy tongue, also known as lingua villosa nigra, results from hyperkeratosis of the tongue. It may have different colours varying from white, yellow or brown to black depending upon the involved extrinsic factors (i.e. tobacco, coffee, tea, food or drugs) and intrinsic factors (i.e. chromogenic organisms in the normal flora).
Inspect and palpate the buccal mucosa. *Mucosa should be moist, without lesions and of appropriate colour.*	■ **Leucoplakia** (small white patches) may be a sign of a premalignant condition. ■ A reddened, dry, swollen mucosa may be seen in stomatitis. ■ Candidiases (white cheesy patches that bleed when scraped) may be seen in immunosuppressed people receiving antibiotics or chemotherapy, and in terminally ill people.
Inspect and palpate the teeth. *Teeth should be in a state of good hygiene without caries.*	■ Cavities and excessive plaque are seen with poor nutrition and/or poor oral hygiene.
Inspect and palpate the gums. *Gums should be of even colour without swelling.*	■ Swollen, red gums that bleed easily (**gingivitis**) are seen in periodontal disease, vitamin C deficiencies or with hormonal changes.
Inspect the throat and tonsils. *Tonsils (if present) should be of appropriate colour and size.*	■ In acute infections, tonsils are red and swollen and may have white spots.
Note the person's breath. *Breath should not have unusual or foul odours* (***halitosis***).	■ Sweet, fruity breath (like the smell of apples which are 'past their prime' or even downright rotten) is noted in diabetic ketoacidosis. A person on a high-protein diet can suffer from halitosis. ■ Acetone breath (smells like nail polish remover) may be a sign of uraemia or diabetes. ■ Foul breath may result from respiratory infections, postnasal drip, liver disease and anorexia. ■ Poor dental/oral health (e.g. untreated cavities, oral candidiasis and poor oral hygiene—inadequate brushing and flossing) may result in bad breath.

CONSIDERATION FOR PRACTICE
Always wear gloves when assessing the oral cavity.

Technique/normal findings	Abnormal findings

Abdominal assessment

The quadrants of the abdomen, with related internal structures, are illustrated in Figure 20.11. Box 20.2 provides guidelines for abdominal assessment.



Right Upper Quadrant
Liver and gallbladder
Pylorus
Duodenum
Head of pancreas
Right adrenal gland
Portion of right kidney
Hepatic flexure of colon
Portions of ascending and transverse colon

Left Upper Quadrant
Left lobe of liver
Spleen
Stomach
Body of pancreas
Left adrenal gland
Portion of left kidney
Splenic flexure of colon
Portions of transverse and descending colon

Midline
Aorta
Bladder
Uterus

◯ = Umbilicus

Right Lower Quadrant
Lower pole of right kidney
Caecum and appendix
Portion of ascending colon
Bladder (if distended)
Right ovary and salpinx
Right spermatic cord
Right ureter

Left Lower Quadrant
Lower pole of left kidney
Sigmoid colon
Portion of descending colon
Bladder (if distended)
Left ovary and salpinx
Uterus (if enlarged)
Left spermatic cord
Left ureter

FIGURE 20.11 ■ The four quadrants of the abdomen, with anatomical location of organs within each quadrant

BOX 20.2 Guidelines for assessing the abdomen

Ask the person to empty their bladder before beginning the examination. Assist the person to the dorsal recumbent (supine) position, with a small pillow under the head, a pillow under the knees (if desired) and the arms at the sides of the body. Warm the stethoscope before applying it to the person's skin. Ask the person to point to areas that are painful and explain that those areas will be examined last. Expose the abdomen from below the breasts to the pubic symphysis and drape the person's thoracic and genital areas. When you document your findings, specify the location by abdominal quadrant.

General guidelines for abdominal assessment are as follows:

1. Inspect the abdomen under a good light source that is shining across the abdomen. Sit at the right side of the person and note symmetry, distension, masses, visible peristalsis and respiratory movements. If masses are detected, ask the person to take a deep breath, which decreases the size of the abdominal cavity and makes any abnormality more visible.
2. Auscultate each quadrant of the abdomen, using the diaphragm of the stethoscope. Listen for bowel sounds, arterial bruits, venous hums and friction rubs.
3. Percuss several areas within each quadrant of the abdomen, using a systematic path (e.g. always begin in the lower left quadrant, then proceed to the lower right quadrant, upper right quadrant and upper left quadrant, respectively). The predominant percussion tones for the entire abdomen are tympany and dullness. Tympany is present over gas-filled intestines. Dullness is present over the liver, the spleen, an enlarged kidney or a full stomach. Percuss for fluid, gaseous distension and masses.
4. Palpate each quadrant of the abdomen for shape, position, mobility, size, consistency and tenderness of the major abdominal organs. Begin this part of the assessment with light palpation and increase the depth of palpation to elicit tenderness or better identify organ size and shape. Deep palpation should be conducted only by nurses with considerable experience. Remember to palpate areas of indicated tenderness last and to use gentle pressure. Palpation may be difficult or impossible if the person exhibits muscle guarding from pain or is ticklish. The gallbladder and the spleen are normally not palpable.

Technique/normal findings	Abnormal findings
Inspect abdominal contour, skin integrity, venous pattern and aortic pulsation. *Abdomen should be slightly concave or rounded with intact skin. There should not be distended veins or obvious aortic pulsations.*	■ Generalised abdominal distension may be seen in gas retention or obesity. ■ Lower abdominal distension is seen in bladder distension, pregnancy or ovarian mass. ■ General distension and an everted umbilicus are seen with ascites and/or tumours. ■ A scaphoid (sunken) abdomen is seen in malnutrition or when fat is replaced with muscle. ■ **Striae** (whitish silver), commonly called stretch marks, may result from rapid growth at puberty, during or after pregnancy, or significant weight gain. Striae may also occur as a result of abnormal collagen formation or a result of medications or chemicals that interfere with collagen formation. ■ Spider angiomas may be seen in liver disease. ■ Dilated veins are prominent in cirrhosis of the liver, ascites, portal hypertension or venocaval obstruction. ■ Pulsation is increased in aortic aneurysm.
Auscultate all four quadrants of the abdomen with the diaphragm of the stethoscope (see Figure 20.12). Begin in the lower right quadrant, where bowel sounds are almost always present. Normal bowel sounds (gurgling or clicking) occur every 5 to 15 seconds. *Listen for at least 5 minutes in each of the four quadrants to confirm the absence of bowel sounds.*	■ **Borborygmus** (hyperactive high-pitched, tinkling, rushing or growling bowel sounds) is heard in diarrhoea or at the onset of bowel obstruction. ■ Bowel sounds may be absent later in bowel obstruction, with an inflamed peritoneum and/or following surgery of the abdomen.
Auscultate the abdomen for vascular sounds with the bell of the stethoscope (see Figure 20.13). *No sounds (bruits, venous hum or friction rub) other than bowel sounds should be auscultated.*	■ **Bruits** (blowing sound due to restriction of blood flow through vessels) may be heard over constricted arteries. A bruit over the liver may be heard in hepatic carcinoma. ■ A venous hum (continuous medium-pitched sound) may be heard over a cirrhotic liver. ■ Friction rubs (rough grating sounds) may be heard over an inflamed liver or spleen.

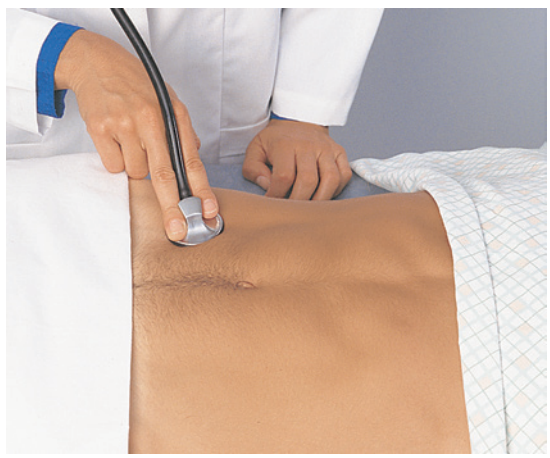


FIGURE 20.12 ■ Auscultating the abdomen with the diaphragm of the stethoscope

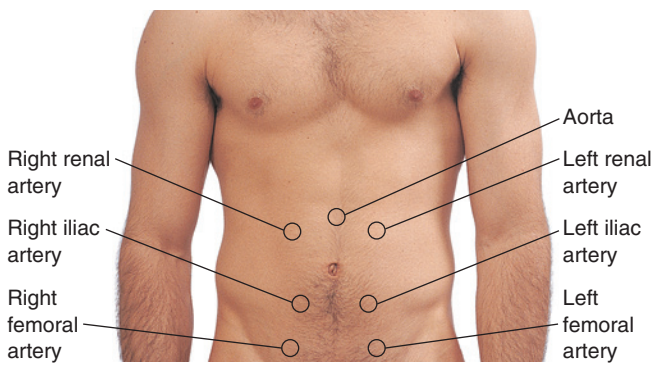


FIGURE 20.13 ■ Location of placement of the stethoscope for auscultation of arteries of the abdomen

Technique/normal findings	Abnormal findings
Percuss the abdomen in all four quadrants (see Figure 20.14). *Normally, tympany is heard over the stomach and gas-filled bowels.*	■ Dullness is heard when the bowel is displaced with fluid or tumours, or filled with a faecal mass.

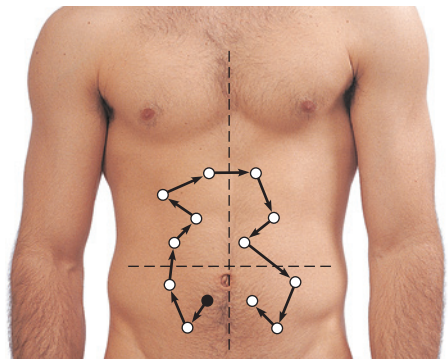


FIGURE 20.14 ■ Location of sites for systematic percussion of all four quadrants

Percuss the liver (see Box 20.3 for guidelines for liver percussion and palpation; see Figure 20.15 for landmarks). *The lower border of liver dullness is located at the costal margin to 1–2 cm below.*	■ In cirrhosis and/or hepatitis, the liver span is greater than 6–10 cm in the MCL and greater than 4–8 cm in the MSL.

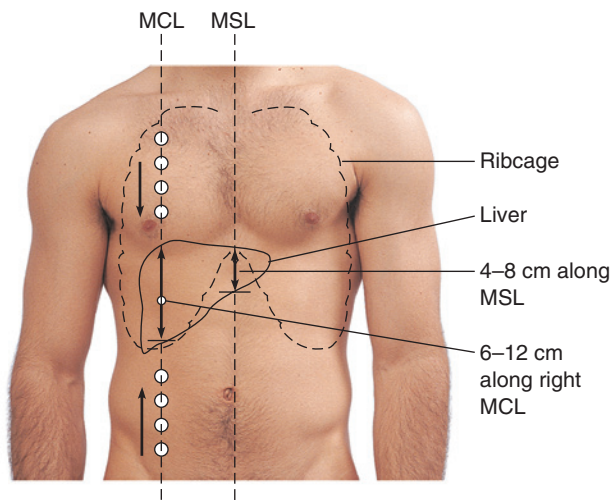


FIGURE 20.15 ■ Anatomical location of the liver, with the midclavicular line (MCL) and midsternal line (MSL) superimposed. The normal liver span is 6–12 cm at the MCL

BOX 20.3 Guidelines for percussing and palpating the liver

The size of the liver may be determined by percussion and palpation, as follows:

1. Percuss in the midclavicular line (MCL), beginning below the umbilicus (see Figure 20.15). Begin to percuss over a region of tympany and move upwards. The first dull percussion tone occurs at the lower border of the liver. Determine the upper liver border by beginning percussion over an area of lung resonance (in the MCL) and percussing downwards to the first dull tone, usually at the 5th to 7th interspace. Mark each of these locations and measure the distance from one mark to the other to determine liver size. The normal liver size is 6–12 cm at the MCL; however, men have larger livers than women.
2. Conduct bimanual palpation of the liver (see Figure 20.19) by placing your left hand under the person at the level of the 11th to 12th ribs and applying upward pressure. Place your right hand below the costal margin, ask the person to take a deep breath and palpate for the liver border. The liver is not normally palpable in a healthy adult, although it may be in very thin people.

Technique/normal findings	Abnormal findings
Percuss the spleen for dullness posterior to the midaxillary line at the level of the 6th to 11th rib (see Figure 20.16). *The spleen is percussed as an oval area of dullness approximately 7 cm wide near the left 10th rib and slightly posterior to the midaxillary line.*	■ A large area of dullness that extends to the left anterior axillary line on inspiration is associated with an enlarged spleen and may be related to trauma, infection or mononucleosis.

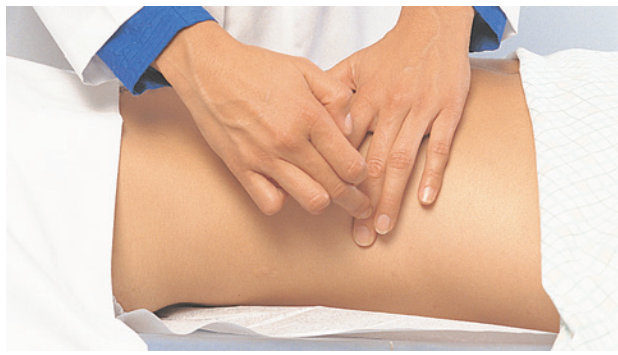


FIGURE 20.16 ■ Percussing the spleen

Percuss for shifting dullness (see Figure 20.17). *If ascites is not present, the borders between tympany and dullness remain relatively constant despite position changes.*	■ In a person with ascites, the level of dullness increases when the person turns to the side.

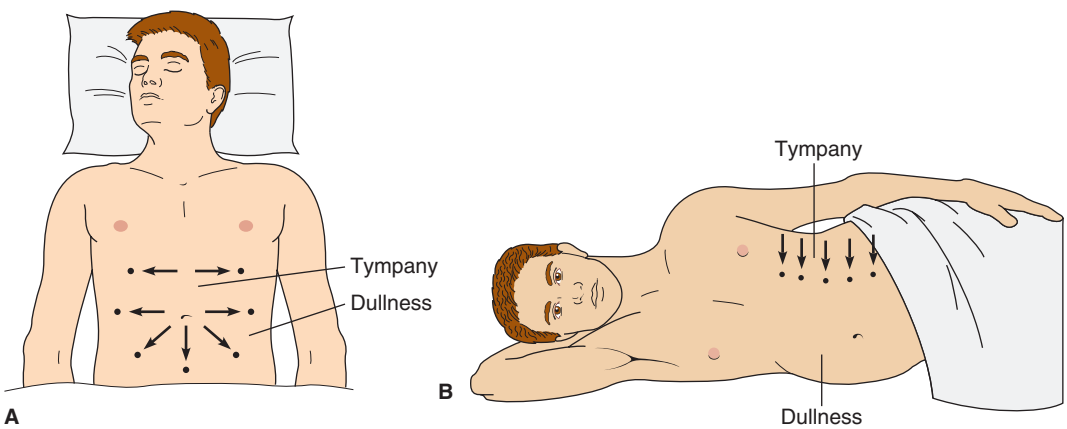


FIGURE 20.17 ■ Percussing for shifting dullness in ascites. *A*, Common percussion tones when the person is lying supine; *B*, changes in percussion tones (shifting dullness) when the person turns to the side

Palpate the abdomen in all four quadrants. *There should be no abdominal masses or pain on palpation.*	■ In cases of peritoneal inflammation, palpation causes abdominal pain and involuntary muscle spasms. ■ Abnormal masses include aortic aneurysms, neoplastic tumours of the colon or uterus, and a distended bladder or distended bowel due to obstruction. ■ A rigid, board-like abdomen may be palpated when the person has a perforated duodenal ulcer.

CONSIDERATION FOR PRACTICE

Never use deep palpation in a person who has had a pulsatile abdominal mass, renal transplant or polycystic kidneys, or is at risk of haemorrhage

Technique/normal findings	Abnormal findings
Use a circular motion to move the abdominal wall over underlying structures (see Figure 20.18). Feel for masses and note any tenderness or pain the person may have during this part of the exam. Palpate lightly at first (1.3–2 cm), then deeply (4–5 cm) with caution. If a mass is palpated, ask the person to raise the head and shoulders. A mass in the abdomen may become more prominent with this manoeuvre, as will a ventral abdominal wall hernia. If the mass is no longer palpable, it is deeper in the abdomen.	

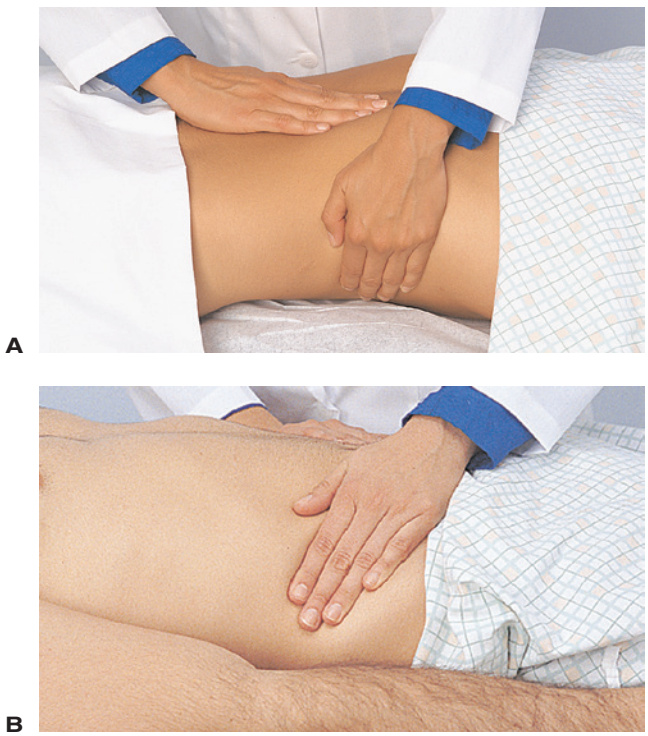


FIGURE 20.18 ■ Light to moderate palpation of the abdomen. *A*, In light palpation, the examiner, keeping the fingers approximated, gently depresses the abdominal wall about 1 cm to assess for large masses, slight tenderness and muscle guarding. *B*, The examiner performs moderate palpation by using the palm or the side of the hand to depress the abdominal wall to a slightly greater depth than in light palpation. This technique is useful for assessing abdominal organs that move with respiration (such as the liver and the spleen)

Palpate for rebound tenderness. Press the fingers into the abdomen slowly and release the pressure quickly. *Releasing pressure should not cause or increase pain.*	■ In peritoneal inflammation, pain occurs when the fingers are withdrawn. ■ Right upper quadrant pain occurs with acute cholecystitis. ■ Upper middle abdominal pain occurs with acute pancreatitis. ■ Right lower quadrant pain occurs with acute appendicitis. ■ Left lower quadrant pain is seen in acute diverticulitis.
Palpate the liver (see Figure 20.19). Note whether the person guards the abdomen or reports any sharp pain, especially on inspiration. *The abdomen should be non-tender and the liver is usually non-palpable.*	■ An enlarged liver with a smooth, tender edge may indicate hepatitis or venous congestion. ■ An enlarged, non-tender liver may be felt in a malignant condition. ■ The person with inflammation of the gallbladder feels sharp pain on inspiration and stops inspiring. This is called Murphy's sign.

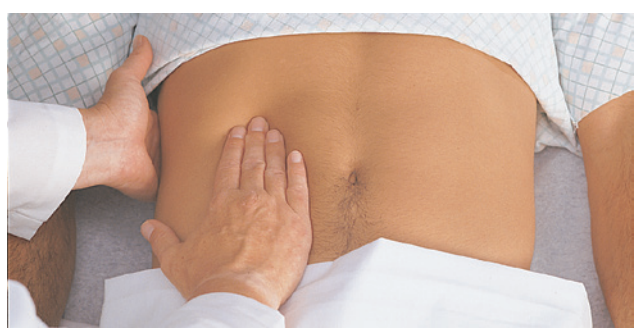


FIGURE 20.19 ■ Palpating the liver with the bimanual method

Inguinal area assessment

Inspect the inguinal area for bulges after asking the person to bear down. *The inguinal area is normally free of bulges.*	■ Bulges that appear in the inguinal area when the person bears down may indicate a **hernia** (a defect in the abdominal wall that allows abdominal contents to protrude outward).

Technique/normal findings	Abnormal findings
Palpate the inguinal area with a gloved hand. Ask the person to shift weight to the left to palpate the right inguinal area and vice versa. Place your right index finger upward into the inguinal area and ask the person to bear down or cough. *Bulging or masses are normally not palpable.*	■ A bulge or mass may indicate a hernia.

CONSIDERATION FOR PRACTICE

Nurses need to be culturally alert and competent when working with people with disorders of the bowel, bowel cancer and undergoing faecal testing. Ways of raising issues and talking about them will depend on a person's cultural worldview about health, bodies and elimination practices. Being well informed and engaging in ongoing cultural education, together with close collaboration with Indigenous or migrant health professionals, will assist nurses to provide relevant, timely and culturally safe care.

Perianal assessment (performed only by experienced nurses or under supervision)

Inspect the perianal area. Wearing gloves, spread the person's buttocks apart. Observe the area and ask the person to bear down as if trying to have a bowel movement. *The perianal area should be intact, without obvious lesions.*	■ Swollen, painful, longitudinal breaks in the anal area may appear in people with anal fissures. (These are caused by the passing of large, hard stools or by diarrhoea.) ■ Dilated anal veins appear with haemorrhoids. ■ A red mass may appear with prolapsed internal haemorrhoids. ■ Doughnut-shaped red tissue at the anal area may appear with a prolapsed rectum. ■ Movable, soft masses may be polyps.
If accredited to conduct a rectal examination, palpate the anus and rectum. Lubricate the gloved index finger and ask the person to bear down. Touch the tip of your finger to the person's anal opening. Flex the index finger and slowly insert it into the anus, pointing the finger towards the umbilicus (see Figure 20.20). Rotate the finger in both directions to palpate any lesions or masses. *There should be no masses in the anus or rectum.*	■ Hard, firm, irregular embedded masses may indicate carcinoma.

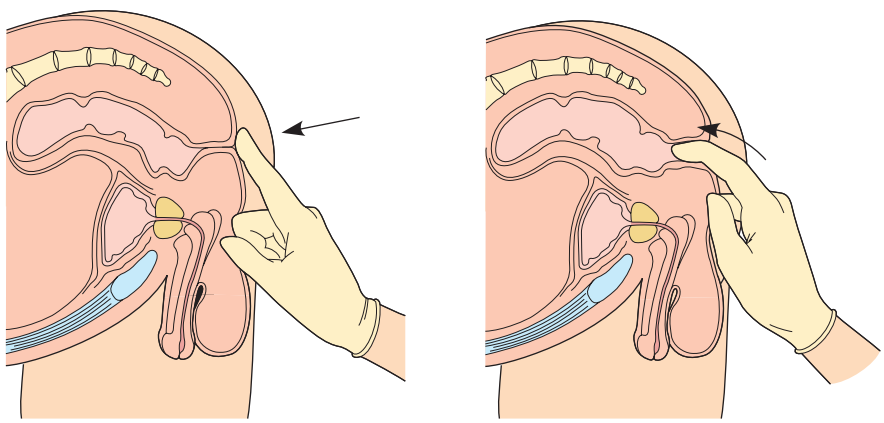


FIGURE 20.20 ■ Digital examination of *A*, the anus; and *B*, the rectum

Faecal assessment

Inspect the person's faeces. After palpating the rectum, withdraw your finger gently. Inspect any faeces on the glove. Note colour and/or presence of blood. Also use gloved fingers to note consistency. *Stool should be soft with no blood present, either on the stool or as occult blood.*	■ See Box 20.4 for information about stool characteristics.
Test the faeces for occult blood. Use a testing kit such as Occultest or Hemoccult II. *There should be no blood in the faeces.*	■ A positive occult blood test requires further testing for colon cancer or gastrointestinal bleeding due to peptic ulcers, ulcerative colitis or diverticulosis.
Note the odour of the faeces. *No distinctly foul odours should be present.*	■ Distinctly foul odours may be noted with stools containing blood, infections, parasites or extra fat, or in cases of colon cancer.

BOX 20.4 Assessing stool characteristics

Inspect faeces for colour, odour and consistency after the rectal exam or after defecation. Both hands are gloved.

Colour

- Blood *on* the stool results from bleeding from the sigmoid colon, anus or rectum. Blood *within* the stool indicates bleeding from the colon due to ulcerative colitis, diverticulosis or tumours. Black, tarry stools, called **melaena**, occur with upper gastrointestinal bleeding. Oral iron may turn stools black and mask melaena.
- Greyish or whitish stools can result from biliary tract obstruction due to lack of bile in stool.
- Greasy, frothy, yellow stools, called **steatorrhoea**, may appear with fat malabsorption.

Odour

- Distinct, foul odours may be noted with stools containing blood or extra fat, or in cases of colon cancer.
- Consistency:
  - Hard stools or long flat stools may result from a spastic colon or bowel obstruction due to a tumour or haemorrhoids. Hard stools may also result from dehydration or ingestion of oral iron.
  - Mucusy, slimy faeces may indicate inflammation and occur in irritable bowel syndrome.
  - Watery, diarrhoea stools appear with malabsorption problems, irritable bowel syndrome, emotional or psychological stress, ingestion of spoiled foods or lactose intolerance.

CONCEPT CHECK

1 A person you are caring for asks you what type of foods are complete proteins. What would be your best response?
1 none
2 eggs and milk
3 fruits and vegetables
4 butter and oils

2 Following minor surgery, a nurse would assess a person who is deficient in vitamin K for what possible complication?
1 infection
2 blood clotting
3 keloid formation
4 slow peristalsis

3 On monitoring a person's lab results, you notice a greatly elevated serum amylase level. What disease does this indicate?
1 cheilosis
2 gastric reflux
3 gallstones
4 acute pancreatitis

4 While assessing an older adult, you notice her teeth have obvious cavities and she has difficulty swallowing. She says, 'My mouth is so dry.' What health problem might result from these findings?
1 nutritional deficit
2 acute pain
3 altered elimination
4 risk of infection

5 What percussion sound would a nurse expect to hear when assessing the abdomen of a person with ascites?
1 inaudible bowel sounds
2 resonance
3 alternating amplitude
4 shifting dullness

6 A person asks you to tell her what internal haemorrhoids are. What would you say?
1 'They are part of the arteries of the body.'
2 'They are just bits of tissue that occur for no reason.'
3 'They are swollen veins in the anal canal.'
4 'They are part of the lymphatic system.'

7 Which of the following questions or statements would be appropriate for the person with an ostomy?
1 'Have you had any bleeding from your haemorrhoids?'
2 'Has your appetite changed lately?'
3 'Tell me about your family.'
4 'Describe the consistency of your stools.'

8 Why is removal of polyps from the colon during a colonoscopy important?
1 to identify genetic disorders
2 to prevent the development of cancer
3 to facilitate further examination of the bowel
4 to decrease future problems with constipation

9 What term is used to describe black, tarry stools?
1 occult blood
2 haematemesis
3 melaena
4 steatorrhoea

10 You are caring for a person the first day following bowel surgery. You do not hear bowel sounds during your initial assessment. What would you do?
1 Immediately call the doctor and report this abnormal finding.
2 Repeat the assessment in 30 minutes to ensure accuracy of findings.
3 Document the assessment as normal following abdominal surgery.
4 Ask another nurse to check your assessment before reporting it.

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CHAPTER 21

NURSING CARE OF PEOPLE WITH NUTRITIONAL DISORDERS

SANDRA CAMPBELL-CROFTS

LEARNING OUTCOMES

- Describe the complications and interprofessional care associated with the nutritional disorder of obesity.
- Describe the pathophysiology, complications and interprofessional care associated with the eating disorder malnutrition.
- Compare and contrast the three nutritional eating disorders of anorexia nervosa, bulimia nervosa and binge-eating disorder.

CLINICAL COMPETENCIES

- Assess the functional health status of the person with nutritional disorders.
- Monitor nutritional status and responses to care; document and report abnormal or unexpected responses.
- Use assessment data to determine priority nursing diagnoses and select and implement nursing interventions.
- Administer medications and enteral and parenteral nutrition knowledgeably and safely.
- Integrate interprofessional care in the plan of care.
- Adapt cultural values and variations into the plan of care for the person with nutritional disorders.
- Plan and provide family teaching to restore, promote and maintain functional health status.
- Evaluate responses to care and use data to revise a plan of care as needed.

KEY TERMS

Obesity and malnutrition, the major nutritional disorders in the world today, affect many systems and organs. They often cause serious health problems, such as hypertension, cardiovascular disease, fluid and electrolyte imbalances, disability and even death.

A person with nutritional disorders requires complex, skilled nursing care. Developmental, sociocultural, psychological and physiological factors may play a role in these disorders: a holistic approach to nursing care is vital. Nursing care focuses on identifying causes, meeting nutritional and physiological needs, providing education and meeting the psychological needs of the person and their family. Before proceeding with the discussion of obesity and malnutrition, review the sections on metabolism and nutrients in Chapter 20.

THE PERSON WITH OBESITY

Obesity, an excess of adipose tissue, is one of the most common, preventable non-communicable health problems worldwide, with 1.6 billion adults classified as overweight and 400 million considered obese (World Health Organization, 2006). The contribution of obesity to a poor health-related quality of life is greater than alcohol abuse and similar to smoking, with 7% of the total burden of disease in Australia being attributed specifically to obesity (National Preventative Health Taskforce (NPHT), 2009).

Health-related problems associated with obesity are listed in Table 21.1.

While obesity is often defined by body weight, it is more accurately measured by the **body mass index (BMI)**, the international standard measurement of the amount of body fat or adipose tissue compared to height, and the waist-to-hip ratio. (See Box 21.1 to calculate BMI.) A BMI of 25 to 29.9 kg/m^2 is classified as *overweight but not obese*; class 1 obese is a BMI between 30 and 35 kg/m^2, class 2 obese is a BMI between 35 and 40 kg/m^2 and class 3 is a BMI greater than 40 (Australian Institute of Health & Welfare (AIHW), 2015). Class 3 obesity is also known as morbid obesity.

Incidence and prevalence

Australia ranks seventh among high-income countries for rates of obesity (National Health Performance Authority, 2013). According to the Australian Bureau of Statistics (ABS) (2012), in 2011–2012, 10.8 million Australian adults were estimated to be either overweight or obese and, of these, 4.7 million were estimated as being obese. The prevalence of overweight and obese Australian adults aged 18 years and over has continued to rise to over half the population, at 63.4% in 2011–2012. The prevalence of overweight and obese children aged 5–17 has stabilised at 25.3%. One challenge in obtaining accurate prevalence data is the tendency for people to underestimate their self-reported weight (Magnusson, 2010).

Table 21.2 shows estimations of overweight and obesity in Australia. It indicates that in 2011–2012 South Australia had the greatest percentage of Australian adults who were overweight but Queensland had the greatest percentage who were obese.

The ABS (2008) identified that adults with higher qualifications were less likely to be obese, while those in low-income households were more likely to be obese. Many overweight and obese individuals suffer from sarcopenic malnutrition (high fat mass, low muscle mass) due to the overconsumption of nutrient-poor high-fat diets.

The NPHT (2009) has reported that 11% of the burden of disease in Indigenous Australians can be attributed to overweight and obesity. Kondalsamy-Chennakesavan et al. (2008) found that Indigenous Australians have a lower BMI than Australians of European origin, but central fat deposition as measured by waist-to-hip-ratios was common in all Indigenous Australian communities.

TABLE 21.1 Health-related problems associated with obesity

BODY SYSTEM	OBESITY-RELATED PROBLEMS
Cardiovascular	Atherosclerosis, hypercholesterolaemia, dyslipidaemia Coronary artery disease Congestive cardiac failure Hypertension Stroke Varicosities Venous thrombosis, hypercoagulopathy, pulmonary embolism
Respiratory	Sleep disorders Asthma Sleep apnoea
Gastrointestinal	Gallbladder disease Diverticular disease Hiatal hernia Non-alcoholic fatty liver (steatohepatitis) Colorectal cancer Oesophageal cancer
Genitourinary	Cancer of the prostate Kidney cancer Obesity-induced glomerulopathy Stress incontinence
Musculoskeletal	Chronic lower back pain Muscle strains and sprains Osteoarthritis of hips and knees
Endocrine and reproductive	Metabolic syndrome Insulin sensitivity and/or resistance Diabetes mellitus, type 2 Pancreatic cancer Postmenopausal breast cancer Ovarian, uterine and endometrial cancers Amenorrhoea and infertility Complications of pregnancy, gestational diabetes mellitus Polycystic ovarian syndrome
Other	Depression Binge-eating disorder Postoperative complications, infection Obesity-related sarcopenia

TABLE 21.2 Estimations of overweight and obesity by Australian state or territory

STATE/TERRITORY	OVERWEIGHT	OBESE
South Australia	37%	30%
Western Australia	37%	28%
Tasmania	37%	28%
Australian Capital Territory	37%	25%
Victoria	35%	26%
New South Wales	35%	26%
Northern Territory	35%	27%
Queensland	30%	35%

Sources: Adapted from National Health Performance Authority (2013). *Overweight and obesity rates across Australia 2011–12*, p. 4, Table 1. Retrieved from www.nhpa.gov.au. © National Health Performance Authority 2015.

Risk factors

Adipose tissue is created when energy consumption exceeds energy expenditure. Research has identified that adipose tissue is not a passive store of energy but plays a complex role in endocrine, metabolic and immune regulation which can contribute to the development of cancer, insulin resistance and type 2 diabetes mellitus (Booth, Magnuson & Fouts, 2015). Genotype technologies associated with the Human Genome Project have found specific genes related to the development of obesity, which has led to the view that some individuals are genetically predisposed to obesity (Locke et al., 2015).

Physical inactivity is probably the most important factor contributing to obesity. Inactive people may consume fewer kilojoules than active people but continue to gain weight due to a lack of energy expenditure. Contemporary cultural and environmental factors such as increased work and time pressures encourage the use of labour-saving devices and reliance on the car for transportation. Increased sedentary time spent watching television and using the computer also contribute to decreased energy expenditure (Dunstan et al., 2012). Environmental influences, such as an abundant and readily accessible food supply, fast-food restaurants, advertising and inappropriately stocked vending machines contribute to increased food intake. Magnusson (2010) has suggested that the imperative within the Australian food industry is the maintenance of profits through unhealthy purchasing patterns.

Sociocultural influences that contribute to obesity in Australia include the social appetite, which is the social, cultural, political, religious and economic contexts in which food is eaten. Examples are overeating at family meals, rewarding behaviour with food, religious and family gatherings that promote increased food intake, a tendency to eat meals away from home or eat takeaway meals from fast-food restaurants, and sedentary lifestyles (Germov & Williams, 2009). Economic pressures encourage low-income consumers towards higher-calorie energy-dense food options (Magnusson, 2010).

Low self-esteem may precipitate unhealthy eating behaviours such as seeking comfort foods (e.g. chocolate) and the resulting weight gain in turn may diminish self-image even further. A person may overeat as a result of anxiety, depression, guilt or boredom, or as a means of getting attention. Some experts characterise overeating as a food addiction and as a coping mechanism for stressful life events (Fraser, 2013).

Overview of normal physiology

All body activities require energy, including activities of daily living and those necessary to maintain cell and tissue function. **Nutrients** in food (or enteral or parenteral feedings) provide this energy and are the building blocks for growth and tissue repair. The body stores excess nutrients and energy (measured as kilojoules) to meet its needs when required nutrients are unavailable. More than 70% of the energy expended each day goes to maintaining the **basal metabolic rate (BMR)**, essentially the 'cost' (in kilojoules) of being alive. Physical activity accounts for only 5–10% of the energy spent daily.

Fat cells store excess energy as **triglycerides**, formed from dietary fats and carbohydrates. The body breaks down the triglycerides in fat cells when needed to provide energy (Porth & Matfin, 2009).

Pathophysiology

Obesity occurs when excess kilojoules are stored as fat. It can result from excess energy intake, decreased energy expenditure or a combination of both.

Appetite, which affects food intake, is regulated by the central nervous system and by emotional factors (Mayer, 2011). The hunger centre in the hypothalamus stimulates appetite in response to stimuli such as hypoglycaemia. As nutrient levels rise, the satiety centre (also in the hypothalamus) sends the message to stop eating. Gastrointestinal filling and hormonal factors also signal *satiety* (a sensation of fullness). Several hormones are involved in regulating obesity and body fat distribution including thyroid hormone, insulin, leptin (a peptide produced by adipocyte tissue that suppresses appetite and increases energy expenditure) and ghrelin (a peptide that originates within the lining of the stomach to induce hunger) (Klok, Jacobsdottir & Drent, 2007). The two main types of body fat distribution are central (visceral) obesity and peripheral (subcutaneous) obesity. Each type of body fat distribution is characterised by a different metabolic profile and degree of cardiovascular risk (Ibrahim, 2010).

Central (or visceral) **obesity** (apple shaped) is identified by a waist circumference greater than 80 cm in women and 94 cm in men or, alternatively, a waist-to-hip ratio of greater than 1 in men or 0.8 in women. (See Chapter 20 for a method to calculate the waist-to-hip ratio.) People with central body obesity tend to have more intra-abdominal fat within the mesentery and omentum which surrounds the abdominal organs, generate higher levels of circulating free fatty acids and have a greater uptake of glucose due to insulin resistance (Ibrahim, 2010). As a result, central obesity is associated with a greater risk of complications such as hypertension, abnormal blood lipid profile, heart disease, stroke and elevated insulin levels. Men tend to have more intra-abdominal fat than women, although women tend to develop a central fat distribution pattern after menopause due to oestrogen deficiency (Ibrahim, 2010).

Peripheral (or subcutaneous) **obesity** (pear shaped), in which the waist-to-hip ratio is less than 0.8, is more commonly seen in women. The risk of hyperinsulinaemia, abnormal lipids and heart disease is lower in people with peripheral obesity than in those with central obesity.

Complications of obesity

Obesity is a significant risk factor for cardiovascular disease, including hypertension, coronary heart disease (CHD) and heart failure. The prevalence of hypertension and hypercholestrolaemia in obese men and women is higher than in people with a BMI of less than 25 (Booth, Prevost & Gulliford, 2015). The increases in blood pressure seen with obesity increase the risk of CHD and stroke. Approximately 60% of obese individuals have **metabolic syndrome**, including three or more of the following: increased waist circumference; hypertension; elevated blood triglycerides and fasting blood glucose; and low HDL cholesterol (Ibrahim, 2010; McPhee, Papadakis & Tierney, 2008). The metabolic syndrome is an identified risk factor for atherosclerosis and CHD. Obesity-associated obstructive sleep apnoea also contributes to the risk of CHD and heart failure.

Obesity increases the risk of insulin resistance and type 2 diabetes mellitus. Both weight gain in adulthood and abdominal (central) obesity are positively correlated with the risk of developing insulin resistance and type 2 diabetes mellitus (Gallagher et al., 2009). Obesity affects reproductive function in both men and women. Androgen (male sex hormone) levels are reduced in obese men; menstrual irregularities and polycystic ovarian syndrome (PCOS) are more common in obese women. PCOS is an additional risk factor for hyperinsulinaemia and insulin resistance. Increased weight also increases the risk of developing gallstones in both men and women. The risk of developing several types of cancer, including colon, breast and endometrial, increases in obesity (AIHW, 2015). A link between obesity and chronic kidney disease has been attributed to the increased metabolic demands of the kidney, leading to higher glomerular capillary pressures with resultant glomerular hypertrophy (Singh & Kari, 2013). Increased weight places abnormal stress on joints, increasing the prevalence of joint pain and osteoarthritis, particularly in the weight-bearing joints of the knees and hips. Other health-related problems associated with obesity are listed in Table 21.1.

INTERPROFESSIONAL CARE

Because obesity has many contributing factors, its treatment is far more complex than just reducing the amount of food consumed. Most experts recommend an individualised program of moderate exercise, diet and behaviour modification designed to meet that person's specific capabilities and needs.

Diagnosis

Although body weight may be used to identify obesity, measures of body fat are more accurate. Males at ideal body weight have 10–20% body fat, whereas females at ideal body weight have 20–30% body fat.

BOX 21.1 Calculating body mass index (BMI)

BMI = weight (kg)/height^2 (m^2)
Normal = BMI 18.5–24.9 kg/m^2
Overweight = BMI 25–29.9 kg/m^2
Obese = BMI > 30 kg/m^2
Morbid obesity = BMI > 40 kg/m^2

- *Body mass index* is used to identify excess adipose tissue (National Health and Medical Research Council (NHMRC), 2013a). BMI is calculated by dividing the weight (in kilograms) by the height in metres squared (m^2) (see Box 21.1). BMI calculations may not as accurately reflect the extent of adipose tissue in people who are highly muscular (e.g. body builders) or in those who have lost muscle mass (e.g. older adults). Table 21.3 provides a tool for determining adult BMIs.
- *Anthropometry* includes measurements of height, weight, bone size and skinfold to estimate subcutaneous fat. See Chapter 20 for more information about anthropometric measurements.
- *Waist circumference* is an additional measurement to determine body fat distribution. The measurement is taken halfway between the last rib and the top of the iliac crest. Men with a waist measurement of 94 cm or greater and women with a waist measurement of 80 cm or greater have a higher risk of complications of obesity.

Other diagnostic tests may be done to help identify a physiological cause of obesity, as well as complications of obesity.

- *A thyroid profile*, including a total T_3 and T_3 uptake, free T_4 (FT_4) and total T_4, free thyroxine index (FTI) and thyroid-stimulating hormone (TSH), rules out thyroid disease (see Chapter 17).
- *Serum glucose* is measured to identify coexisting diabetes mellitus.
- A *lipid profile* is ordered; high-density lipoprotein (HDL) ('good cholesterol') levels may be reduced in obese people, whereas low-density lipoprotein (LDL) ('bad cholesterol') levels are elevated.
- An *electrocardiogram* (ECG) is performed to detect effects of obesity on the heart, such as rate or rhythm disruptions, myocardial infarction or ventricular hypertrophy.

Medications

When used in combination with diet and moderate exercise, medications can assist in promoting weight loss. Their long-term efficacy, however, is questionable; rebound weight gain following the cessation of drug use is common. In addition, tolerance, addiction and side effects may occur. These products are usually recommended only as an adjunct to therapy and only when the traditional therapies of diet and exercise have been unsuccessful.

Sibutramine hydrochloride (Reductil) is a prescription-only medication that acts on the CNS as an appetite suppressant. Orlistat (Xenical) inhibits fat absorption from the GI tract, leading to weight loss. It has the added benefit of lowering blood

TABLE 21.3 Body mass index table for BMIs

Height (cm)	Weight (kg)																	
200	11	13	14	15	16	18	19	20	21	23	24	25	26	28	29	30	31	33
195	12	13	14	16	17	18	20	21	22	24	25	26	28	29	30	32	33	34
190	12	14	15	17	18	20	21	22	24	25	26	28	29	30	32	33	35	36
185	13	15	16	18	19	20	22	23	25	26	28	29	31	32	34	35	37	38
180	14	15	17	19	20	22	23	25	26	28	29	31	32	34	35	37	39	40
175	15	16	18	20	21	23	24	26	28	29	31	33	34	36	38	39	41	42
170	16	17	19	21	22	24	26	28	29	31	33	35	36	38	40	42	43	45
165	17	18	20	22	24	26	28	29	31	33	35	37	39	40	42	44	46	48
160	18	20	21	23	25	27	29	31	33	35	37	39	41	43	45	47	49	51
155	19	21	23	25	27	29	31	33	35	37	40	42	44	46	48	50	52	54
150	20	22	24	27	29	31	33	36	38	40	42	44	47	49	51	53	56	58
145	21	24	26	29	31	33	36	38	40	43	45	48	50	52	55	57	59	62
	45	50	55	60	65	70	75	80	85	90	95	100	105	110	115	120	125	130

Underweight
Healthy
Overweight
Obese
Morbid obesity

glucose and cholesterol. See the 'Medication administration' box below for the nursing implications of these drugs.

Methylcellulose and other bulk-forming products may decrease appetite by producing a sensation of fullness. A person taking these products may experience flatulence or diarrhoea and may need to increase fluid intake to prevent constipation.

Treatments

Successful treatment of obesity (sustained achievement of normal body weight without adverse consequences) is rarely achieved. Treatment focuses on reducing the health risks associated with obesity by changing lifestyle in both eating and exercise habits. A combination of treatments including a reduction in sedentary behaviour and an increase in physical activity, dietary therapy, behaviour modification, pharmacology and, in some cases, surgery is required to achieve and maintain weight loss.

EXERCISE Exercise is a critical element in weight loss and maintenance. Physical activity improves physical fitness, decreases appetite, promotes self-esteem and increases the basal metabolic rate. An exercise or activity program should reflect the person's physical condition, interest, lifestyle and abilities (NHMRC, 2013b). Evaluation by a healthcare practitioner is important before beginning an exercise program. The practitioner instructs the person to progressively increase the duration and intensity of activity but to stop exercising and report symptoms if chest pain or shortness of breath occurs. A moderate aerobic exercise program of 30–40 minutes, 5 or more days a week promotes weight loss while reducing adipose tissue, increasing lean body mass and promoting long-term weight control. Table 21.4 presents examples of kilojoule use per hour for moderate physical activities.

TABLE 21.4 Kilojoule use per hour for moderate physical activities

TYPE OF EXERCISE	KILOJOULES/HOUR	TYPE OF EXERCISE	KILOJOULES/HOUR
Sleeping	230	Walking	1170
Eating	360	Table tennis	1210
Sewing	360	Gardening	1470
Knitting	360	Tennis	1470+
Sitting	360	Water aerobics	1670
Standing	420	Skating	1760+
Driving	460	Dancing, aerobic	1760+
Office work	590	Aerobics	1880+
Housework, moderate	670+	Bicycling, moderate	1880+
Golf, with trolley	750	Jogging	2090
Golf, without trolley	1000	Gardening, digging	2090
Gardening, planting	1050	Swimming, active	2090+
Dancing, ballroom	1090		

MEDICATION ADMINISTRATION Drugs to treat obesity

APPETITE SUPPRESSANTS

Sibutramine (Reductil)

Sibutramine reduces hunger and increases sensations of satiety by inhibiting the uptake of serotonin, noradrenaline and dopamine. The medication should be used in conjunction with a low-kilojoule, low-fat diet and regular moderate exercise.

Nursing responsibilities

- Assess for contraindications, such as pregnancy or lactation, use of other appetite suppressants, impaired liver or kidney function, history of CHD or alcohol abuse.
- Regularly monitor blood pressure and heart rate during treatment. Increases may indicate need to reduce dose or discontinue treatment.

Health education for the person and family

- Take as directed; do not exceed recommended dose. Do not take if you may be pregnant or are breastfeeding.
- Take your last dose no later than 4 pm to avoid insomnia.
- You may experience difficulty sleeping, nervousness or palpitations while taking this drug.
- Increase your fluid intake to reduce possible side effects of dry mouth and constipation.
- This drug does not replace diet and exercise for weight loss; continue to follow your prescribed regimen.

LIPASE INHIBITOR

Orlistat (Xenical)

Orlistat inhibits lipases necessary for the breakdown and absorption of fat, thus decreasing the absorption of dietary fat. Its action is primarily local, within the GI tract, with few systemic effects.

Nursing responsibilities

- Administer with meals or up to 1 hour following a meal.
- Provide a fat-soluble vitamin supplement (A, D, E and K) daily. Separate administration time from orlistat by at least 2 hours.

Health education for the person and family

- Take as directed; do not increase dose. You may skip a dose if you do not consume a meal containing fat.
- Use in conjunction with a low-kilojoule, low-fat diet.
- Common gastrointestinal side effects include oily or fatty stools, flatulence, oily discharge or frequent stools with difficulty controlling defecation. These side effects may diminish with time or increase if a meal high in fat is consumed.
- Notify your healthcare provider if you become pregnant while taking this medication.

NUTRITION The diet is planned to create a daily 2000 to 4000 kJ deficit off the recommended 8700 kJ/day currently recommended in Australia. Ideally, the diet should be low in kilojoules and fat, and contain adequate nutrients, minerals and fibre. The person should eat regular meals with small servings. A gradual, slow weight loss of no more than 0.5 kg per week is recommended. For most people, this means a diet of 4186 to 5020 kJ/day for women and 5020 to 6700 kJ/day for men. Fewer than 5020 kJ each day may lead to loss of lean tissue and nutritional deficiencies. 'Yo-yo' dieting (repeated cycles of weight loss and gain) may lead to a metabolic deficiency that makes subsequent weight loss efforts increasingly difficult. Therefore, it is critical that dieters take any weight loss effort seriously and include plans for long-term maintenance. The best approach is to modify dietary intake without severe restrictions, eating a well-balanced diet and developing improved eating habits.

Very-low-kilojoule diets (VLKDs), otherwise known as very-low-energy diets (VLEDs), are generally reserved for people who have a BMI greater than 30 (Sumithran & Proietto, 2008). This type of program requires meals to be replaced with nutritionally complete but protein-sparing and modified fat sachets of powder which are dissolved in a glass of water to create a 'milkshake'. Between 1674 and 3349 kJ/day is consumed. This type of weight loss regimen must be undertaken for any length of time under close medical supervision. Exercise, nutrition and behaviour modification counselling should accompany the diet (Snel et al., 2012). Adverse effects generally are minor, but could include fatigue, constipation, nausea and diarrhoea. Recently, the development of gallstones formation requiring cholecystectomy has been attributed to VLKDs (Gudzune et al., 2015).

BEHAVIOUR MODIFICATION People who wish to lose weight quickly readily accept dangerous regimens to achieve rapid weight loss. Behaviour modification is the critical component of a successful long-term weight loss management program. Strategies such as keeping food records of the type and amount of foods eaten, eliminating cues that precipitate eating such as television advertisements, and changing the type of food eaten and the speed of eating are often helpful.

Researchers have found that most overweight people are stimulated to eat by external and emotional cues, such as the proximity to food and the time of day. In contrast, hunger and satiety are the cues that regulate eating in adults of normal weight. Strategies to control food cues include keeping food out of view, controlling portion size, reducing or eliminating the habit of consuming snack foods, and eating only in designated areas. See Box 21.2 for a list of behavioural modification strategies.

Other behaviour modification approaches focus on helping a person examine factors that affect their eating behaviours (Magnusson, 2010). The goal is to empower the person who is stimulated to eat to choose other activities that are not related to food.

Weight loss services such as Weight Watchers, Jenny Craig Weight Loss Centres, SureSlim and Lite N' Easy promote weight loss success through coaching and peer support. Most organised programs require participants to pay a fee, which may improve compliance.

BOX 21.2 Behavioural modification strategies for the obese person

Controlling the environment

- Encourage participation in reading and analysing food labels so as to purchase nutritious low-kilojoule foods.
- Shop from a prepared list and on a full stomach.
- Keep all foods in the kitchen.
- Store all foods in the refrigerator or the cabinets in opaque containers.
- Prepare exact portions of food to eliminate leftovers.
- Limit portion size of foods.
- Ensure 2 serves of fresh fruit and 5 serves of vegetables are eaten each day (Department of Health and Ageing, 2008).
- Eat all foods in the same place; avoid eating in the kitchen or in the car.
- Avoid eating when watching television or reading.
- Reduce frequency of eating out at fast-food restaurants.

Controlling physiological responses to food

- Have set times for meals; avoid grazing behaviour.
- Eat slowly by taking small bites, allowing 20 minutes for a meal.
- Eat a salad or drink a hot beverage before a meal.
- Put eating utensils or food down between bites.
- Concentrate on the eating process; savour the food.
- Stop eating with the first feelings of fullness.

Controlling psychological responses to food

- Appreciate the aesthetic experience of eating.
- Use attractive dinnerware and prepare a formal setting for eating.
- Use small plates and cups to make servings of food look larger.
- Concentrate on conversations and socialisation during the meal.
- Use non-food rewards for meeting a goal.
- Acknowledge small successes and improvements in all behaviour.
- Substitute other activities for eating (e.g. reading, exercise, hobbies).

SURGERY Surgical treatment of obesity, otherwise known as *bariatric surgery* or *metabolic surgery*, is limited to **morbidly obese** people (BMI of over 40 kg/m^2 or 200% ideal body weight) who have a documented failure of non-surgical weight loss or those with a BMI over 35 kg/m^2 who have serious obesity-related health problems such as metabolic syndrome, type 2 diabetes mellitus, hypertension or heart disease (Korda et al., 2012). In addition, a person must be able to tolerate surgery and be free of addiction to alcohol or other drugs such as nicotine. The benefits of surgery include major weight loss, improved blood pressure, improved blood lipid profile, a remission of type 2 diabetes mellitus and reduced risk of sleep apnoea, angina and heart failure (Diabetes Australia, 2011). However, bariatric surgery is not without risk, and some people regain some of the lost weight over time. Surgical problems, such as a stretched pouch or separated stitches, may also affect the amount of weight loss). Although bariatric surgery procedures have been listed on the Australian Medicare Benefits Schedule (MBS) since 1992, Korda et al. (2012) found that this procedure is predominantly performed in private hospitals with significant out-of-hospital expenses, thereby exacerbating existing Australian health inequalities in obesity due to the socioeconomic gradient.

Bariatric surgery takes two forms: malabsorptive and restrictive. The most common procedures in Australia are all performed laprascopically: adjustable gastric banding; sleeve gastrectomy; and Roux-en-Y gastric bypass (Shannon, Gervasoni & Williams, 2013). In restrictive banding procedures such as adjustable gastric banding (AGB) (see Figure 21.1), a hollow band of silicone rubber is placed around the upper (proximal) portion of the stomach. The band is inflated with saline solution to create a small stomach pouch with a narrow passage through to the rest of the stomach which restricts the amount of food that can be consumed. The amount of band inflation can be adjusted using a port implanted under the skin. Sleeve gastrectomy is performed by resecting the stomach to the size of a tubular sleeve in the lesser curvature of the stomach (Rosenthal et al., 2012). The Roux-en-Y gastric bypass involves the stomach being made smaller, with the duodenum being bypassed to reduce absorptive capacity. This procedure was found to achieve a greater total weight loss and for up to 5 years but had a higher complication rate (Colquitt et al., 2014).

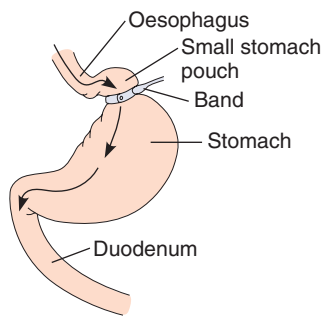


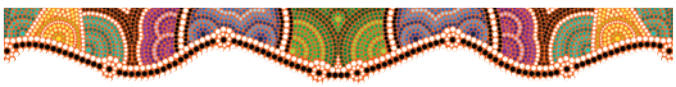
FIGURE 21.1 ■ Adjustable gastric banding

Few nutritional deficiencies are associated with restrictive bariatric procedures. Postprandial vomiting is a common postoperative complication (Shannon et al., 2013). The band may slip or break, necessitating a return to surgery.

Although the risk of postoperative complications is high, the mortality rate for bariatric procedures is low; possible postoperative complications include anastomosis leak with peritonitis, haemorrhage, abdominal wall hernia, gallstones, wound

infections, deep venous thrombosis, nutritional deficiencies and gastrointestinal symptoms (McPhee et al., 2008). In dumping syndrome, stomach contents move rapidly through the small intestine, drawing fluid into the intestine by osmosis. The person experiences nausea, bloating, abdominal pain, weakness, sweating and possibly syncope. See Chapter 22 for more information about dumping syndrome.

Nursing care for the person who has undergone bariatric surgery is substantially the same as for a person who has undergone a gastric resection. See Chapter 22 for more information about gastric resection and associated nursing care. People undergoing bariatric surgery have some additional nursing care needs related to the effects of the surgery on gastrointestinal function (see 'Meeting individualised needs', below).



Nursing care

Health promotion

Maintaining a healthy weight throughout the lifespan begins in childhood. Obese children and teenagers become obese adults. Promote healthy eating with education to reduce consumption of highly processed high-sugar, high-fat, high-salt foods and increase the exposure to low-processed nutrient-rich foods such as high-fibre whole grains, lean protein-rich meats, fresh fruits and a variety of vegetables (NHMRC, 2013b). The Health at Every Size (HAES) movement shifts the focus from weight loss to increasing health-promoting behaviours (Bacon & Aphramor, 2011). This healthy eating approach to health and thus weight loss has been supported by the Dietary Approaches to Stop Hypertension (DASH) diet which has been repeatedly demonstrated to protect against cardiovascular disease, stroke and heart failure (Salehi-Abargouei et al., 2013). The Healthy Living Pyramid (Nutrition Australia, 2015) provides visual guidance for appropriate food choices to maintain a healthy, well-balanced diet.

Adults commonly gain about 10 kg between early and middle adulthood. Encourage the person to reduce the number of kilojoules consumed as energy needs change.

Assessment

Collect the following data through the health history and physical examination (see Chapter 20):

- *Health history:* risk factors; current and usual weight; recent weight gains or losses; perception of weight and effect on health; usual diet and food intake; exercise/activity patterns; prior weight loss efforts and results; current medications; coexisting disorders such as cardiovascular disease and type 2 diabetes mellitus; tobacco use; family history of overweight and weight-related morbidity.
- *Physical examination:* vital signs; weight and height; anthropometric skinfold measurements; waist circumference; waist-to-hip ratio; BMI; inspect skin under the breasts and abdominal folds.

Nursing diagnoses and interventions

Nursing care for overweight and obese people is community based and holistic, focusing on both physiological and psychological responses to weight and appearance. See below for a nursing care plan for the person with obesity.

MEETING INDIVIDUALISED NEEDS Recommended diet following bariatric surgery

A person undergoing bariatric surgery to restrict stomach capacity and/or nutrient absorption has unique learning needs to prevent discomfort and nutritional complications after surgery:

- Ensure slow, progressive transitions in food texture from liquids to solid foods over the first 8 postoperative weeks. A restricted liquid diet is prescribed in the first 2 weeks of the postoperative period. Fruit juices and other concentrated sugars are avoided. *The person is at risk of developing dumping syndrome in the early postoperative period; simple carbohydrates increase the risk.*
- When clear liquids are tolerated, non-fat or low-fat milk is added to the diet. *Milk provides a protein source. Lactose-free or soy milk is recommended to reduce the risk of diarrhoea. People with a lactase deficiency may not be able to digest the natural lactose sugar in milk.*
- Pureed foods are introduced into the diet in approximately 1–2 weeks and the diet is advanced to include soft foods within about 2 months after surgery. *Gradual increases in food textures preserve the staple line and allow the restricted stomach to adapt.*
- Increasing fluid intake and maintaining protein intake are priorities during the healing process. *Fluid intake is necessary to maintain fluid balance and protein for nitrogen balance and tissue healing.*
- Instruct the person to eat regularly, consume smaller portions, cut food into small pieces, chew food well and eat slowly. *Failure to thoroughly chew food and eat slowly can lead to regurgitation, vomiting and abdominal discomfort.*
- Advise the person to take an adult multiple vitamin and mineral supplement after restrictive surgery. *Because the overall quantity of food consumed is reduced, the person may develop a micronutrient deficiency.*
- Advise the person to avoid sugar and concentrated sweets (fruit juice, sugar-containing beverages, honey) and to separate consumption of solid foods and liquids by at least 30 minutes. *Concentrated sugars can precipitate dumping syndrome.*

Source: Adapted from Shannon, Gervasoni & Williams (2013). The bariatric surgery patient: Nutrition considerations. *Australian Family Physician, 42*(8), 547–552.

CONSIDERATION FOR PRACTICE

Use of an inappropriate-sized sphygmomanometer is a common source of error in measuring blood pressure in obese people, resulting in a falsely high blood pressure. Choose a cuff on which the width of the bladder is 40% of the circumference of the arm and the length of the bladder is sufficient to cover at least 65% of the upper arm.

Activity intolerance

Obese people may experience excess fatigue, tachycardia and shortness of breath with activity due to the physiological effects of excess weight as well as a sedentary lifestyle. A medical evaluation may be needed before beginning an exercise program.

- Assess current activity level and tolerance of that activity. Assess vital signs. *This provides baseline information to plan an activity program and assess response to that activity.*
- After medical clearance, plan with the person a program of regular, gradually increasing exercise. Consider a consultation with an exercise physiologist. *An individualised exercise program promotes activities within that person's physical capabilities.*

Imbalanced nutrition: more than body requirements

Although many factors contribute to obesity, it always involves an imbalance of kilojoule consumption to energy expenditure.

- Encourage the person to identify the factors that contribute to excess food intake. *Identification of cues to eating helps the person eliminate or reduce these cues.*
- Establish realistic weight loss goals and exercise/activity objectives. *Small, reasonable goals, such as loss of 0.5 kg per week, increase the likelihood of success.*
- Encourage participation in reading and analysing food labels on processed and packaged foods. *Increased decision making encourages healthier food choices.*
- Assess knowledge and discuss well-balanced diet plans. Provide necessary teaching about the recommended inclusion of two fruit and five vegetable serves in the diet. *Knowledge empowers the person to participate and make appropriate food choices.*
- Discuss behaviour modification strategies, such as self-monitoring and environmental management. *Behaviour modification, diet and exercise are critical to promoting successful, long-term weight loss.*
- Monitor weight loss and blood pressure weekly with annual laboratory data, including blood glucose and lipid levels. *Continuing assessment is important not only to evaluate the safety of weight loss strategies, but also to reinforce positive benefits of weight loss.*

Ineffective therapeutic regimen management

Most overweight or obese people experience some difficulty integrating all the components of a weight loss program into a daily routine. For a weight loss and maintenance program to be successful, the overweight person must modify dietary intake in a world of daily temptations. There may be many obstacles to exercise, including a busy schedule, activity intolerance, impaired physical mobility, lack of equipment and the embarrassment of being fat.

- Discuss ability and willingness to incorporate changes into daily patterns of diet, exercise and lifestyle. *This provides data from which to set realistic goals with the person.*
- Beginning with gentle non-weight-bearing exercise such as swimming may encourage participation. Increase activity levels based on physical ability. *Exercise success increases likelihood of continuation.*
- Establish strategies for dealing with 'stress' eating or interruptions in the therapeutic regimen. *A sense of failure associated with overeating or lack of exercise can lead to further overeating. Identifying positive strategies to deal with these situations promotes self-acceptance and limits self-punishment through overeating.*

Chronic low self-esteem

Although many obese people may have accepted their weight and body appearance on some level, most overweight and obese individuals verbalise the experience of 'fat prejudice' or stigma in their family, workplace or community. Obese people may experience ridicule, prejudice and health problems attributed to being 'fat'. These experiences, coupled with day-to-day problems such as finding attractive clothing or a chair large enough to sit on can affect self-esteem. Many report that 'fat' jokes or comments contribute to a sense of negative self-worth.

- Encourage the person to verbalise the experience of being overweight and validate that experience. *This provides baseline data to use in developing individualised interventions to address self-esteem issues.*
- Set small goals with the person and offer positive feedback and encouragement. Small goals provide more opportunities for success. *Positive feedback and encouragement provide a comfortable environment in which to develop self-esteem.*
- Refer for counselling as appropriate. *Many people benefit from counselling for issues related to self-esteem.*

Community-based care

Weight reduction usually occurs in community-based settings. Weight loss and maintenance require a long-term commitment by the person, their family and supportive environmental systems that encourage healthy choices and lifestyles. Address the following topics with the person and their family:

- Small, subtle lifestyle modifications are more effective than dramatic diets. Fad diets promote rapid weight loss but often are not nutritionally sound or may be difficult to maintain for a lifetime (Magnusson, 2010).
- All household members should also consume a diet that is nutritionally sound. Encourage participation in analysing nutrient food labels prior to purchasing processed or packaged foods.

NURSING CARE PLAN A person with obesity

Sam Elliott, aged 57, has gained 20 kg since his retirement 2 years ago. The most active thing he does each day is 'pottering around the garden'. His diet includes apple juice, oatmeal porridge, 2 slices of white toast with strawberry jam and white coffee with 2 sugars for breakfast; 2 donuts or a blueberry muffin and coffee with friends mid-morning; a ham-and-cheese sandwich with a bag of potato chips and a soft drink for lunch; and cheese, biscuits and wine before a dinner of meat, potatoes, vegetables and dessert. He tells the nurse, 'I have never had to diet. I just don't know how to get this weight off.'

ASSESSMENT

Mr Elliott is 173 cm tall and weighs 91.2 kg. His BMI is 30.1 kg/m^2. His cholesterol is 6.14 mmol/L (normal < 5.13 mmol/L) with an HDL of 1.00 mmol/L (normal 1.5 mmol/L) and an LDL of 4.85 mmol/L (normal < 3.31 mmol/L). His BP is 138/90. His fasting blood glucose is normal at 5.0 mmol/L. His ECG shows normal sinus rhythm. He reports fatigue and shortness of breath with activity. His healthcare provider has advised a weight loss of 13 kg and a regular exercise program.

DIAGNOSES

- *Imbalanced nutrition* related to food intake in excess of nutritional energy requirements.
- *Risk of ineffective therapeutic regimen* related to knowledge deficit.
- *Activity intolerance* related to sedentary lifestyle evidenced by fatigue when attempting gentle exercise.

PLANNING

- Enter an agreement with Mr Elliott to meet once each week to assess progress.

Expected outcomes

- Lose 0.5 kg each week.
- Increase levels of moderate exercise up to walking 30 minutes for 5 days each week.
- Verbalise an understanding of the relationship between weight loss, weight control and exercise.
- Identify support systems for behaviour modification.
- Increase knowledge level of healthier food choices.

IMPLEMENTATION

- Assess weight and blood pressure each week
- Discuss current eating habits and strategies to reduce fat and kilojoule intake.
- Discuss cues such as boredom that promote eating. Identify strategies to eliminate or reduce eating cues.
- Teach to keep a food diary to examine and change eating habits.
- Discuss the role of regular exercise in weight loss and weight control. Instruct to maintain an exercise record to track the intensity and duration of activity.
- Discuss lifestyle and behaviour modification strategies to promote successful weight loss and control.

EVALUATION

Two weeks after changing his diet and beginning to exercise, Mr Elliott has lost 1 kg. He has maintained a food diary. He has identified boredom as a cue to eating. In light of that fact, he has started volunteering at the local hospital. He is walking for 30 minutes, 5 days a week. He plans to increase his activity periods to 45 minutes. He verbalises commitment to a lifelong plan of exercising and eating a high-fibre, low-fat diet. His BP has ranged from 132/76 to 136/84. He plans to have the employee health nurse at the hospital check his weight and BP each week and to join Weight Watchers for ongoing support.

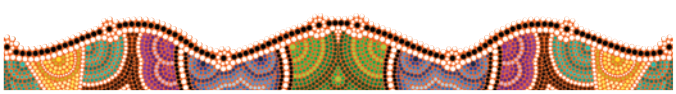
CRITICAL THINKING IN THE NURSING PROCESS

1. What are some possible pathophysiological bases for Mr Elliott's abnormal cholesterol, HDL and LDL levels?
2. Develop a teaching plan for a group of overweight men and women.
3. Identify potential barriers to losing weight and strategies to reduce or eliminate these barriers.

REFLECTION ON THE NURSING PROCESS

1. How achievable is a non-surgical weight loss intervention program over the long term?
2. What other nursing interventions would you consider utilising if Mr Elliott's weight loss does not meet the expected outcome?

- Set realistic goals to increase levels of incidental exercise.
- Establish realistic weight loss goals and a system of non-food rewards for achieving each goal.
- Identify an 'exercise buddy' or support system to promote continued physical activity.
- Expect occasional failures. Resume prescribed diet and exercise routine as soon as possible; the goal is long-term weight management.
- Community resources such as Weight Watchers or healthcare-based programs provide information, strategies and social support for successful weight management.



THE PERSON WITH MALNUTRITION

Malnutrition results from an inadequate intake of nutrients and can be defined as the state of being poorly nourished due to an imbalance in nutrition (Tuso & Beattie, 2015). There may be a lack of macronutrients (kilojoules, carbohydrates, proteins and fats) or micronutrients such as vitamins and/or minerals. Malnutrition is associated with a high burden of illness, depression of the immune system, poor wound healing, bone and muscle weakness, longer lengths of hospital stay with higher treatment costs and increased morbidity and mortality (van der Meij, Wijnhoven & Finlayson, 2015). Malnutrition may be caused by inadequate nutrient intake; impaired absorption and use of nutrients; or loss of nutrients due to diarrhoea, haemorrhage, renal failure or increased

metabolic needs due to an underlying illness (e.g. infection or physiological stressors such as cancer).

Protein energy malnutrition (PEM) is the state of decreased body pools of protein with or without fat depletion or a state of diminished functional capacity, caused at least partly by inadequate nutrient intake relative to nutrient demand and/or which is improved by nutritional repletion (Cant, 2011).

Incidence and prevalence

PEM is a widespread cause of disease and mortality throughout the world. It is endemic in African subtropical regions affected by global warming and famine (Sunyer & Grimalt, 2007). Groups at risk of malnutrition in Australia include children of Indigenous Australian heritage, culturally and linguistically diverse people, families with low socioeconomic status, older adults, the homeless and people undergoing complex medical treatments such as chemotherapy or dialysis. Even when food is plentiful, a person may be undernourished due to poor food choices. Food insecurity in Australia is a risk factor for malnutrition due to limited access, either financially or geographically, to healthy nutritious food. Malnutrition is associated with the anorexia of ageing due to decreased appetite or increased food insecurity (van der Meij et al., 2015). Malnutrition may be present on admission to hospital or may develop as a result of surgery or serious illness. Malnutrition increases both mortality and the incidence of complications in both medical and surgical situations.

See Box 21.3 for conditions commonly associated with malnutrition.

Risk factors

Risk factors for malnutrition include the following:

- age—older adults are at greater risk of malnutrition due to a variety of factors (see the 'Meeting individualised needs' box below)
- poverty, homelessness, inadequate food storage and preparation facilities
- functional health problems that limit mobility or vision
- history of rapid weight loss of more than 10% of usual weight over the last 6 months
- oral or gastrointestinal problems that affect food intake, digestion and absorption
- chronic pain or chronic diseases such as pulmonary, cardiovascular, renal and endocrine disorders, or cancer
- dementia, mental health disorders such as depression
- medications or treatments that affect appetite
- alcohol or drug addition
- acute problems such as infection, surgery or trauma
- dieting behaviours such as anorexia nervosa or bulimia
- inability to obtain food such as would occur in famine or disaster.

BOX 21.3 Conditions associated with malnutrition

- Acute respiratory failure
- Ageing
- AIDS
- Alcoholism
- Burns
- COPD
- Dementia
- Eating disorders
- Ear disease in Aboriginal and Torres Strait Islander children
- Gastrointestinal disorders
- Neurological disorders
- Renal disease
- Short bowel syndrome
- Surgery
- Trauma

Pathophysiology

Carbohydrates and fats in the diet are the body's primary energy sources. Approximately 15–25% of the body is fat, the body's energy reservoir. Fat-free mass includes muscles, bones, skin and organs which are metabolically active tissues. Proteins in the diet primarily are used to maintain these tissues. Glycogen and proteins in this lean body mass also act as energy stores.

When dietary intake of nutrients does not meet the body's energy needs, the body uses glycogen, body proteins and lipids (fats) to support metabolism. Neurones in the brain depend completely on a constant source of glucose for normal function; importantly, glucose is unable to be stored within the brain and needs to be delivered via the bloodstream.

In **starvation** (inadequate dietary intake), glycogen initially is used to provide energy. After the first 24 hours of starvation, gluconeogenesis (formation of new glucose from proteins) is the major source of energy. As starvation continues, the body breaks down fats into free fatty acids and ketones, which provide glucose for the brain. The size of all body compartments is reduced as body fats and muscle proteins are used to meet this glucose demand. As lean body mass is reduced, metabolically active tissue is lost and energy expenditure decreases.

The stress of acute illness or trauma produces a different response. The acute stress response produces a state of hypermetabolism and **catabolism** (cell and muscle breakdown). This hypermetabolic state increases energy expenditure and nutrient needs. If untreated, up to half of the body's protein stores can be used up within 3 weeks.

Many hospitalised people are malnourished (starved) on admission. Surgery or illness promotes a stress response, resulting in protein energy malnutrition. In PEM, both protein and kilojoules are deficient. The intake of adequate kilojoules but with a chronic reduction in protein intake is called *kwashiorkor*. When both proteins and kilojoules are insufficient to meet the body's needs, PEM is also known as *marasmus*.

Manifestations

Weight loss is the most apparent manifestation of malnutrition. The malnourished person may have a body weight of less than 90% of ideal. Body mass also is reduced (see Box 21.1), as is anthropometric skinfold thickness. Other manifestations include a wasted appearance, dry and brittle hair, and pale mucous membranes. Abdominal or bipedal peripheral oedema may be present. Older adults may present with general symptoms of frailty, falls, weakness, slow walking speed, reduced physical activity capacity, pressure ulcers, unintentional weight loss and exhaustion (Malafarina et al., 2012). Manifestations of specific nutrient deficiencies may be present (see accompanying box). See also the following 'Multisystem effects of malnutrition'.

MEETING INDIVIDUALISED NEEDS **Nutrition for the older adult**

Older adults are at greater risk of malnutrition. Body fat mass increases up to about 75 years of age, then begins to decrease. Age-related changes that contribute to malnutrition include a loss of appetite due to changes in taste and smell, a higher incidence of gastrointestinal disease such as dysphagia or delayed gastric emptying, poor oral health, loss of teeth or ill-fitting dentures, anorexia caused by medications, and functional limitations that contribute to food insecurity by impairing the ability to shop and cook. Psychosocial issues such as depression or dementia also contribute to the problem. Older adults living on fixed incomes may not be able to afford to purchase food. Lifestyle factors such as social isolation and loneliness contribute to the issue (van der Meij et al., 2015). Eating is a social event and older adults who eat alone may not eat as well as those who share meals with companions.

Conduct a thorough assessment to determine nutritional status. Assess psychological factors that influence eating habits, such as loneliness, isolation and depression. Note the person's general appearance and obtain a diet history, including information about foods and nutrients the person consumes and any recent weight losses or gain. Review laboratory values, including complete blood count, total protein, albumin and prealbumin.

Health education for the person and family

To maintain nutritional status, the older person should be advised to:

- Eat a well-balanced diet.
- Keep a food diary.
- Eat fresh 2 serves of fruit and 5 serves of vegetables each day.
- Consume soft foods if chewing difficulties are present.
- Increase consumption of micronutrient-dense dairy products if tolerated.
- Shop wisely to get the most value for money.
- Avoid high-fat, high-salt processed foods.
- Drink adequate fluids.
- Take regular gentle exercise to maintain muscle tone.
- Take body weight each week and contact local physician if weight has further decreased.
- Contact local organisations for the availability of congregate meals (e.g. at local senior centres) or home-delivered meals (e.g. Meals on Wheels).

Subcutaneous fat and muscle proteins are broken down in PEM, impairing mobility and increasing the risk of skin and tissue breakdown (pressure ulcers). A lack of physical activity results in further muscle loss known as sarcopenia. Protein synthesis is inhibited in PEM and wound healing is delayed. Serum albumin levels fall, leading to abdominal oedema, diarrhoea and impaired nutrient absorption. Cachexia is characterised by severe muscle loss with increased protein catabolism due to an underlying disease such as chronic kidney disease or cancer (Barker, Gout & Crowe 2011). Immune function is impaired in cachexia with an increased risk of infection. Cardiac output falls and the risk of postural hypotension increases.

INTERPROFESSIONAL CARE

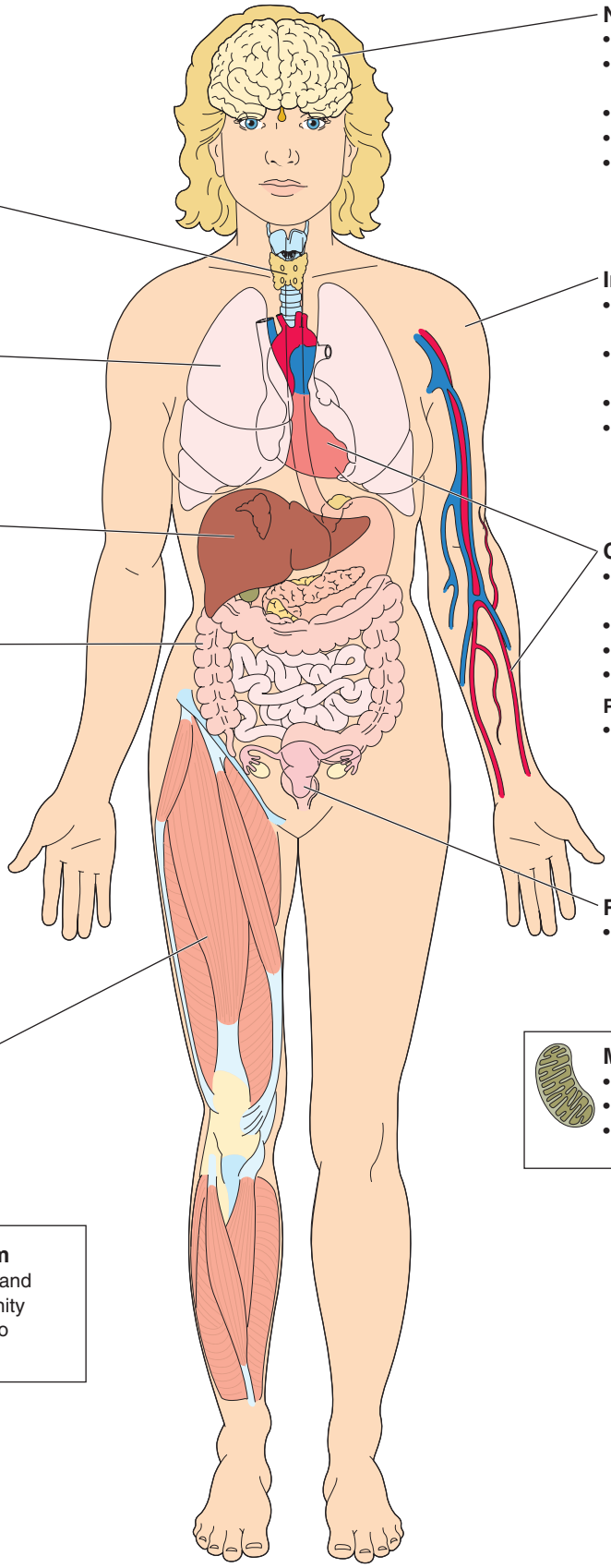
The goal of treatment for the malnourished person is to restore ideal body weight while replacing and restoring depleted nutrients and minerals. The person's age, severity of malnutrition and coexisting health problems help determine interventions. Treatment may include oral supplementation, tube feedings or parenteral nutrition.

Diagnosis

Nutrition screening tools such as those discussed in Box 21.4 can help identify a person at risk of malnutrition. Their use has been suggested as best practice guidelines for all acute hospital settings by the Dietitians Association of Australia. Barker et al. (2011) identified a number of validated screening and assessment tools for malnutrition. These include the Malnutrition Screening Tool (MST), the Malnutrition Universal Screening Tool (MUST), the Mini Nutrition Assessment (MNA), the Nutritional Risk Screening (NRS-2002), the four-item Short Nutrition Assessment Questionnaire (SNAQ) and the Subjective Global Assessment (SGA). As with obesity, the standard measurements to assess for malnutrition include diet history, height, weight, calculation of BMI and anthropometric skinfold measurements. A BMI of less than 18–20 kg/m^2 may indicate malnutrition. Other assessments include questioning on any history of unintentional weight loss and the severity of any illnesses that have impacted on oral food intake. The following laboratory studies also may be ordered:

- *Serum albumin* is reduced in PEM and may be below 32 g/L.
- *Prealbumin* (also known as transthyretin) should be measured for any person at risk of malnutrition and anyone with a serum albumin of less than 30 g/L. Prealbumin, which has a short (2-day) half life, is a better measure of nutritional status than albumin because it is sensitive to acute changes in nutritional status despite the presence of multiple-organ disease (Caccialanza et al., 2013). See Table 21.5 for normal prealbumin levels and the implications of reduced prealbumin levels.
- *Transferrin* is a protein that transports iron. It decreases in the presence of protein energy malnutrition.
- The *total lymphocyte count* is evaluated by multiplying the WBC by the percentage of lymphocytes. The total lymphocyte count is reduced in PEM.
- *Serum electrolytes* are measured. Potassium levels are low in severe malnutrition.

MULTISYSTEM EFFECTS OF MALNUTRITION



Neurological
- ↓ Cognition
- ↓ Consciousness (drowsiness, lethargy)
- Tremors
- Paraesthesias
- Impaired coordination

Endocrine
- ↓ Thyroid hormones
- ↓ Testosterone (male)
- ↓ Oestrogen (female)

Integumentary
- Hair: brittle, dull, dry, loss of colour
- Nails: fragile, brittle, spoon-shaped
- Petechiae
- Poor wound healing

Respiratory
- Respiratory rate
- Vital capacity

Hepatic
- Hepatomegaly
- ↓ Bile synthesis

Cardiovascular
- Arrythmias and conduction disturbances
- ↓ HR
- ↓ BP
- Enlarged heart

Potential complication
- Heart failure

Gastrointestinal

Oral/oesophageal:
- Cheilosis
- Glossitis
- Gingivitis

Stomach/intestines:
- Ascites
- Constipation
- Intestinal atrophy
- Steatorrhoea
- ↓ Gastric and pancreatic secretions

Potential complication
- Malabsorption syndrome

Reproductive
- Amenorrhoea

Musculoskeletal
- Muscle wasting
- Tenderness
- Impaired strength

Metabolic processes
- ↓ Weight
- ↓ Core body temperature
- Oedema

Immune system
- ↓ Cell-mediated and humoral immunity
- ↑ Susceptibility to infections

MANIFESTATIONS Specific nutrient deficiencies

DEFICIENCY	ASSESSMENT DATA	DEFICIENCY	ASSESSMENT DATA
Kilojoules	Weight loss	Dry beriberi	Polyneuritis, convulsions, confusion, apathy, muscle weakness, ataxia, nystagmus, sleeplessness, anxiety
Protein (marasmus*)	Weakness, listlessness Loss of subcutaneous fat Muscle wasting	Riboflavin (vitamin B_2)	Cheilosis (crusting and ulceration at the corner of the mouth), stomatitis Neuropathy, glossitis Normocytic anaemia
Protein (kwashiorkor†)	Thin or sparse hair Flaking skin Loss of cardiac muscle Hepatomegaly Peripheral oedema	Vitamin C (scurvy)	Swollen, bleeding gums Delayed wound healing Weakness, depression Easy bruising
Vitamin A	Night blindness Altered taste and smell Dry, scaling, rough skin Impaired immune function	Iron	Smooth tongue Listlessness, fatigue Dyspnoea Anaemia
Thiamine (vitamin B_1‡) Wet beriberi	Confusion, apathy Cardiomegaly, dyspnoea Muscle cramping and wasting Paraesthesias, neuropathy Ataxia		

* Marasmus is a physiological adaptation to severe protein energy malnutrition (PEM) which involves the gradual breakdown of fat and muscle to provide energy and amino acids for protein synthesis, essential for continued homeostasis (Badaloo et al., 2006).

† Kwashiorkor is a similar response to severe protein energy malnutrition (PEM) but is associated with more complex and pathological changes due to the inefficient mobilisation and utilisation of adipose tissues together with impaired muscle breakdown processes (Badaloo et al., 2006).

‡ Thiamine, vitamin B_1 is a water-soluble vitamin that is absorbed primarily in the jejunum. The thiamine deficiency disease known as beriberi can have neurological or cardiac manifestations. Beriberi may be seen in developed nations where the diet contains excessive carbohydrates as thiamine is a requirement of carbohydrate metabolism. Bariatric beriberi occurs from the rearrangement of the absorptive areas of the intestinal tract resulting in a decreased surface area of the jejunum (Frank, 2011). Diuretics may also contribute to beriberi due to the losses through the urinary system (Misumida, Hisashi Umeda & Iwase, 2014).

Medications

Malafarina et al. (2012) found that vitamin D demonstrated increased strength and was associated with fewer falls. Malnourished people generally require supplemental vitamins and minerals to restore essential micronutrients (Tuso & Beattie, 2015). A multivitamin and mineral supplement may be given or therapy may be tailored to correct specific deficiencies. See the 'Medication administration' box below for nursing implications of vitamin and mineral supplements.

TABLE 21.5 Normal and high-risk prealbumin levels

PREALBUMIN LEVEL	IMPLICATIONS	SUGGESTED INTERVENTIONS
150–360 mg/L	Within normal limits	None
100–150 mg/L	High risk of nutritional deficit	Monitor level biweekly
100–50 mg/L	Significant risk of malnutrition	Aggressive nutritional support (e.g. enteral feedings or parenteral nutrition)
≤ 50 mg/L	Malnourished	

Nutrition

Fluids and nutrients should be carefully reintroduced in severely malnourished people to prevent life-threatening refeeding syndrome (Redgrave et al., 2015). First, fluid and electrolyte imbalances are corrected, with particular attention paid to restoring normal potassium, phosphate, magnesium and calcium levels, as well as acid–base balance. Once fluid and electrolyte imbalances are corrected, oral protein and kilojoules are gradually reintroduced. Initial feedings are in limited amounts (100 mL) of liquid formula to prevent diarrhoea. Vitamin and mineral supplements at about twice the Nutrient Reference Values (NRVs) are provided along with refeeding. Lactose intolerance may develop in severely malnourished people; lactose-free dairy products or yoghurt may be tolerated better.

BOX 21.4 Nutrition screening tools

NUTRITION SCREENING TOOL	SUMMARY/USES	LIMITATIONS
Malnutrition Screening Tool (MST)	• Easy three-question tool which can be used by non-trained staff • Assesses recent weight and appetite losses • Useful in medical, surgical and oncology environments	• Further nutritional assessment is needed for those identified at severe risk of malnutrition
Mini Nutrition Assessment (MNA)	• Used in aged care environments • Has an abbreviated two-step questionnaire which identifies those at risk of malnutrition who then can be referred to the complete 18-step MNA	• Limited scope—developed for use in aged care
Nutritional Risk Screening (NRS-2002)	• Uses BMI, recent weight and appetite losses together with a subjective analysis of disease • Recommended by the European Society for Clinical Nutrition and Metabolism (ESPEN) for hospital use • Is useful in prompting the initiation of nutritional support	• Subjectivity of the tool • Does not allow for definitive diagnosis
The four-item Short Nutrition Assessment Questionnaire (SNAQ)	• Quick and easy screening tool • Developed to diagnose malnutrition in hospitalised people • Does not require calculation of BMI	• Further nutritional assessment is needed for those identified at severe risk of malnutrition

Source: Based on Barker, L. A., Gout, B. S. & Crowe, T. C. (2011). Hospital malnutrition: Prevalence, identification and impact on patients and the healthcare system. *International Journal of Environmental Research and Public Health*, *8*, 514–527.

Gradual refeeding is necessary to prevent electrolyte imbalances from developing as potassium, magnesium, phosphorus and glucose move into the cells. Heart failure may occur due to depressed cardiac function (Abed et al., 2014). Abnormalities in gastrointestinal function can lead to malabsorption and diarrhoea with refeeding. Food intake is gradually increased in increments of 1000 kJ twice a week until the person is able to consume about 10 000 kJ per day and is gaining 1.5–2.0 kg weekly. Commercially available nutritional supplements (such as Carnation Instant Breakfast, Ensure and Sustacal) may supplement protein and kilojoule intake.

ENTERAL NUTRITION

Enteral nutrition, or tube feeding, may be used to meet kilojoule and protein requirements in those unable to consume adequate food. The indication for tube feedings is when the lower gastrointestinal tract is still able to absorb nutrients, such as where there is difficulty swallowing, loss of gag reflex in victims of cerebrovascular accident, unresponsiveness, and oral or neck surgery (Curtis, 2013). Enteral nutrition is also indicated in the presence of severe trauma where the lower gastrointestinal tract is unaffected, anorexia or serious illness such as Crohn's disease, or malignancy. Tube feedings may provide part or all of the person's nutritional needs. Enteral nutrition is the preferable method of feeding when oral intake is not possible, as this method provides nutrients directly to the stomach and other digestive organs, reduces the incidence of enteric pathogens, promotes blood flow to the gastrointestinal tract and supports other functions of the GI tract such as the release of hormones and epidermal growth factors (Preiser et al., 2015). There is a growing trend in Australia to offer enteral nutrition in the home for long-term serious illnesses (Agency for Clinical Innovation Home Enteral Nutrition Network, 2012).

Enteral tube feeding is the delivery of a liquid, nutritionally complete formula through a soft, small-calibre tube such as a nasogastric tube into the stomach, or a nasoduodenal or nasojejunal tube into the small intestine (see Figure 21.2). Large-bore gastric (Salem sump) tubes can be used for the dual purposes of gastric decompression and feeding. Fine-bore gastric tubes are more comfortable for the person and cause less trauma and tissue irritation. Jejunal tubes are associated with much better absorption of feeds and a lower risk of regurgitation and subsequent aspiration compared to gastric tubes. A transgastric–jejunal feeding tube allows for simultaneous jejunal feeding and gastric decompression. Feeds can also be administered through a gastrostomy or jejunostomy tube. Percutaneous endoscopic gastrostomy (PEG) tubes are becoming the method of choice for

MEDICATION ADMINISTRATION Vitamin and mineral supplements

FAT-SOLUBLE VITAMINS

Vitamin A
Vitamin D
Vitamin E
Vitamin K

The fat-soluble vitamins are absorbed in the gastrointestinal tract. Vitamins A and D are stored in the liver. Fat-soluble vitamins A, D, E and K should be taken with food.

All fat-soluble vitamins may become toxic if taken in excess amounts.

Nursing responsibilities

- Monitor for manifestations of vitamin excess as well as for adverse effects from vitamin administration.
- Monitor carefully for hypersensitivity reactions during parenteral administration. Have emergency equipment available.
- Do not administer vitamin K intravenously due to increased risk of anaphylaxis.

Health education for the person and family

- Teach the importance of eating a well-balanced diet. If indicated, provide lists of foods high in specific vitamins.
- Caution that excessive intake of these vitamins may lead to vitamin toxicity.

WATER-SOLUBLE VITAMINS

Vitamin C (ascorbic acid)
Vitamin B complex:
Thiamine (B_1)
Riboflavin (B_2)
Niacin (nicotinic acid)
Pyridoxine hydrochloride (B_6)
Pantothenic acid
Biotin

If the diet is deficient in one vitamin, it is usually deficient in other vitamins as well; therefore, multivitamin preparations are often administered. Most of these water-soluble vitamins are well absorbed from the gastrointestinal tract.

Nursing responsibilities

- Monitor for responses to replacement therapy.
- Monitor for hypersensitivity reactions from parenteral administration. Have emergency equipment available.

Health education for the person and family

- Do not exceed the recommended daily allowances for the specific vitamin.

MINERALS

Copper
Manganese
Phosphorus
Chromium
Iodine
Iron
Selenium
Calcium
Zinc

Minerals are inorganic chemicals that are vital to a variety of physiological functions. Also called trace elements, these minerals are part of a balanced diet. Recommended daily intakes have not been established for all mineral substances. The dosage of prescribed minerals depends on the specific deficiency, route of administration and the person's general health.

Nursing responsibilities

- Monitor for manifestations of mineral imbalance.
- Prior to administration, dilute oral mineral preparations. Administer minerals with fruit juice to increase absorption.
- Prior to the administration of iodine, assess for history of hypersensitivity to iodine or seafood; if hypersensitive, notify the doctor.

Health education for the person and family

- Encourage the person to avoid exceeding the known recommended daily intake of the mineral.
- Instruct the person to take minerals on an empty stomach, which will increase their ability to be fully absorbed. Zinc can be taken with or after meals.

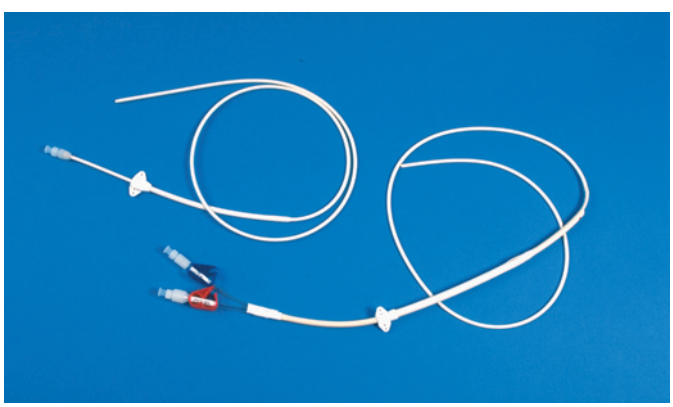


FIGURE 21.2 ■ A nasoduodenal tube and a jejunostomy tube

Source: Courtesy of Michal Heron/Pearson Education/PH College.

long-term enteral feeding regimens, palliative enteral nutrition for terminal malignancies of the upper gastrointestinal tract or for decompression of malignant obstructions (Mobily & Patel, 2015).

Tube placement must be checked by x-ray prior to initiating any fluids or feeds to confirm that the end of the tube is correctly placed within the stomach and not in the lungs (Curtis, 2013). Kim et al. (2012) report that ultrasonography is a low-radiation alternative for determining correct placement. However, if results are inconclusive, an x-ray is mandatory. Periodical aspiration of the tube and checking the pH of aspirated contents is necessary to verify continued correct placement. A pH of < 4 indicates the presence of gastric acid and confirms placement in the stomach; pH > 6 indicates the tube is in the jejunum. See Box 21.5 and the 'Translation to practice' box below.

Most tube feeding formulas provide 4 kJ/mL with approximately 14% of the kilojoules from protein, 60% from

FIGURE 21.3 ■ The nurse secures the feeding tube of a person receiving a continuous enteral feeding

carbohydrates and 25–30% from fat. Administering 1500 mL per day provides the recommended daily intake of all vitamins and minerals. Formulas that provide more kilojoules per millilitre, more grams of protein, added fibre or lower fat are also available (see Table 21.6). Commercial products provide instructions for initiating therapy. Enteral feedings should be started with small volumes of water then small volumes of feed to prevent diarrhoea, with the volume gradually increased to provide the required kilojoules for maintenance and healing. Formulas may be administered as a bolus feeding or as a continuous drip feeding regulated by a kangaroo feeding pump (see Figure 21.3).

Aspiration and diarrhoea are the most common complications of enteral feedings. Continuous infusion of the formula reduces the risk of aspiration. The risk also is reduced by placing the feeding tube in the jejunum rather than the stomach. To avoid aspiration, the nurse elevates the head of the bed at least 30 degrees during feeding and for at least 1 hour after feeding. Formulas that contain fibre can reduce the incidence of

BOX 21.5 Measures to verify feeding tube placement

After inserting the feeding tube and verifying appropriate placement through pH of the aspirate and an x-ray, mark the feeding tube position near the nose with an indelible marker. Prior to each feeding (or every 8 to 12 hours if continuous feedings are being administered), assess the abdomen and tube placement. Use the following steps to assess tube placement:

- Assess the abdomen for distension, bowel sounds and tenderness using the sequence of inspection, auscultation, percussion and palpation.
- Assess tube condition and placement by verifying that the indelible mark remains at the same position and that the feeding tube is securely fastened to the nose. Ask the person to open their mouth and inspect the position of the tube in the oropharynx. Do not administer a feeding if the person is having difficulty speaking or is coughing.
- Using a 60 mL syringe, inject 30 mL of air into the feeding tube, then aspirate a small amount of stomach contents and check the pH of the aspirate.

Reassess tube placement if the person vomits or retches, requires oropharyngeal suctioning, complains of discomfort or reflux into the mouth, or develops signs of respiratory distress (Curtis, 2013).

TRANSLATION TO PRACTICE Evidence-based practice: determining feeding tube placement

The auscultatory (air bolus) method of determining the location for feeding tubes on initial placement and prior to initiating feeding has previously been recommended in nursing textbooks. The premise was that injecting a small amount of air into the tube will produce a distinctive sound if the tube is correctly located, presumably in the stomach. Sounds produced by tubes located in the oesophagus, intestines and respiratory tract were presumed to be different enough to alert the nurse to the improper location. However, the auscultatory method for determining placement is not reliable because the rush of air can be heard irrespective of tube location (i.e. lung or stomach) (Metheny, Stewart & Mills, 2012). This is especially true for small-bore feeding tubes. Testing the pH level of aspirates from newly inserted feeding tubes and regularly during intermittent feedings can distinguish gastric from respiratory and intestinal placement. Based on the pH of a large number of samples of aspirates from feeding tubes, a pH of between 0 and 4 suggests gastric placement and a pH of 7 or greater indicates respiratory placement. As the tube advances through the stomach into the intestine, pH levels increase and the colour of the aspirate changes. If placement in the small intestine is required, a confirmatory x-ray should be taken. Metheny et al. (2012) recommend that routinely the pH method be used rather than the auscultatory method and that protocols be updated to reflect current information.

IMPLICATIONS FOR NURSING

With the advent of small-bore nasogastric and nasointestinal feeding tubes and the increasing use of gastrostomy and jejunostomy feeding tubes, more people are candidates to receive enteral feedings. Aspiration of contents with pH testing prior to x-ray confirmation is mandatory for determining tube placement.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Compare the normal pH of aspirates obtained from the respiratory tract, oesophagus, stomach, duodenum and jejunum.
2. Develop a teaching plan for the person being discharged with a gastrostomy or jejunostomy feeding tube.

TABLE 21.6 Selected enteral feeding formulas

FORMULA TYPE	CONTAINS	EXAMPLES
Complete—suitable for most people requiring enteral feedings	• 4 kJ/mL • Protein: ~ 14% total kJ • Fat: ~ 30% total kJ • Carbohydrate: ~ 60% total kJ • Recommended daily intake of all minerals and vitamins is 1500 mL/day	Compleat, Ensure, Isocal, Nutren, Isolan Sustacal, Resource
High-kilojoule complete—appropriate for those on a fluid restriction	As above; provides 6–8 kJ/mL	Ensure Plus, Sustacal HC, Comply, Nutren 1.5, Resource Plus, Isocal HCN, Magnacal, TwoCal HN
Complete lactose-free, high-residue—used to prevent/treat diarrhoea, constipation	As above; provides fibre	Jevity, Profibre, Nutren 1.0 with fibre, Fibrelan, Sustacal with fibre, Ultracal, Ensure with fibre, Fibresource, Accupep HPF, Reabfin, others
Disease-specific formulas:		
Renal failure	Essential amino acids	Amin-Aid, Travasorb Renal, Aminess
Respiratory failure	Fat: > 50% total kJ	Pulmocare, NutriVent
Liver failure with hepatic encephalopathy	High amounts of branched-chain amino acids	Hepatic-Acid II, Travasorb Hepatic

diarrhoea. Fluid and electrolyte status is monitored carefully and additional water is regularly administered as needed to provide fluid replacement and ensure tube patency.

PARENTERAL NUTRITION **Parenteral nutrition (PN)** is the intravenous administration of carbohydrates (high concentrations of dextrose), protein (amino acids), electrolytes, vitamins, minerals and fat emulsions. These hypertonic solutions usually are administered through a central vein, such as the subclavian or external jugular vein (see Figure 21.4). A peripherally inserted central catheter (PICC) line may be used for short-term total parenteral nutrition (TPN).

PN is initiated when a person's nutritional requirements cannot be met through diet, enteral feedings or peripheral vein infusions. A person who has undergone major surgery or trauma where the lower intestinal tract is non-functioning or who is seriously undernourished is often a candidate for PN. PN is used for both short- and long-term management of nutritional deficiencies. Many people are discharged to home with PN and monitored by home health nurses.

To begin therapy, the physician inserts the central venous catheter under aseptic conditions. The location of the catheter tip is confirmed by x-ray. A triple-lumen catheter is most commonly used. This type of catheter permits concomitant administration of medications, intralipids or blood through other lumens. Parenteral nutrition solutions are either purchased commercially or mixed in the pharmacy using sterile technique under a laminar-flow airhood. A commonly used solution includes 500 mL of 50% dextrose, 500 mL of an 8.5% amino acid solution, electrolytes, minerals and vitamins. The sterility of the solution is maintained and no medication, other than intralipids, is added to the solution after it is mixed or to the lumen through which the PN is being administered. Most hospitals have specific policies and procedures for changing the tubing and the dressing at the insertion site, as well as for hanging new containers. PN solutions are always administered with an infusion pump to ensure the correct rate of infusion.

The person receiving parenteral nutrition is at risk of mechanical, metabolic and infectious complications. Pneumothorax, haemothorax, brachial plexus injury and improper position are possible complications of central venous catheter insertion. Once in place, the catheter may dislodge, leak or break and become an embolus. Clots also may form within or around the catheter.

Fluid overload is a risk with parenteral nutrition, particularly in older adults. The high-glucose formulas can lead to osmotic diuresis or shifts of electrolytes—potassium and phosphorus in particular—into the cells, leading to hyperglycaemia, hypokalaemia or hypophosphataemia. Blood glucose and serum electrolyte levels are carefully monitored during treatment. In addition to electrolyte imbalances, acid–base disturbances may develop, as well as refeeding oedema or heart failure (Fletcher, 2013). Long-term use of parenteral nutrition can lead to gallstone formation and liver disease. Nutrient deficiencies may develop, including deficiencies of vitamins, iron and other minerals when parenteral nutrition is continued for three or more months (Fletcher, 2013).

Disruption of the skin barrier and administration of a solution high in glucose presents a risk of infection in a person receiving PN. Infection may be local, limited to the exit site or surrounding a tunnelled catheter, or may lead to sepsis. The person's temperature and other manifestations of infection must be carefully monitored. Meticulous sterile technique is used for catheter exit site care and container and tubing changes (Fletcher, 2013).

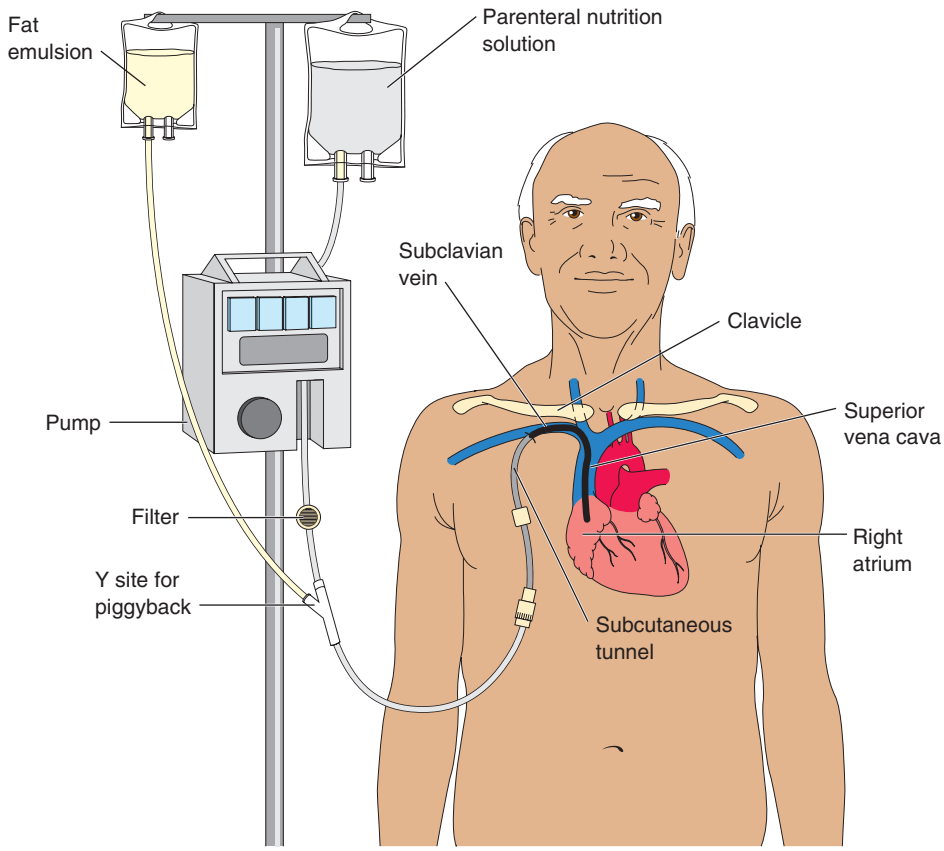


FIGURE 21.4 ■ Parenteral nutrition through a catheter in the right subclavian vein

Nursing care

Health promotion

Aggressive nursing assessment and interventions can help prevent malnutrition associated with hospitalisation or long-term care. In hospitalised people, carefully monitor any food intake (Tuso & Beattie, 2015). Document the nutrition plan and report if nutrition intake does not meet with that plan. When the person is placed on nil by mouth (NBM) status for surgery or tests, ask the doctor to restore diet orders as soon as possible. If allowed, encourage family members to provide favourite or culturally appropriate foods to promote intake. In long-term care settings, promote socialisation during meals. Assess food likes and dislikes and provide foods the person is more likely to eat.

Assessment

Collect nutritional assessment data on admission and periodically (once or twice a week) during long-term institutionalisation.

- *Health history:* usual daily dietary pattern (type and amount of foods consumed); usual weight and recent changes; appetite and food tolerance; specific food likes and dislikes; specific cultural foods; difficulty swallowing; problems such as anorexia, nausea, diarrhoea or constipation; history of surgery and/or chronic diseases (e.g. chronic lung disease) and medications.
- *Physical examination:* height, weight, anthropometric skinfold thickness, BMI; vital signs; general appearance, evidence of muscle wasting, mobility; skin and mucous membranes; bowel sounds; laboratory studies. In children, the mid upper arm circumference (MUAC) has been recently endorsed by the World Health Organization as a validated method to assess malnutrition (Blackwell et al., 2015).

Use of a nutritional assessment tool can help identify a person (older adults in particular) at risk of malnutrition (see Box 21.4). Such tools assess food intake, mobility and BMI, as well as weight loss, psychological or physiological stress, and dementia or psychological conditions to determine the presence of or risk of malnutrition.

Nursing diagnoses and interventions

The complex effects of malnutrition on multiple body systems place the person at high risk of a number of other health issues. This section addresses problems with nutrition, infections, fluid volume and skin integrity. See below for a nursing care plan for the person with malnutrition.

NURSING CARE PLAN A person with malnutrition

Rose Chow is an 88-year-old widow who lives alone. She typically rises early and has a cup of tea before spending her morning pottering in her garden. She consumes her main meal of the day at lunch, which usually includes rice and some vegetables. For dinner, she generally eats a bowl of rice with 'whatever seems to be in the fridge'. She admits to having little interest in cooking or eating since her husband died 10 years ago, and her group of friends has been 'dying off too'.

ASSESSMENT

Mrs Chow weighs 43.1 kg and is 160 cm tall, with a BMI of 16.8.kg/m^2. She reports weighing 53.5 kg 5 years ago. Her triceps anthropometric skinfold thickness measurement is 11 mm. (Normal values for a female: >13 mm.) Her skin is pale and she appears thin and wasted. Her temperature is 36.1°C. Diagnostic test results include serum albumin 30 g/L (normal 38–50 g/L) and serum cholesterol 3.0 mmol/L (normal 3.5–5.4 mmol/L). A diagnosis of protein energy malnutrition is made and a 6280 kJ per day diet is recommended.

DIAGNOSES

- *Imbalanced nutrition* related to food intake less than nutritional energy requirements.
- *Risk of ineffective therapeutic regimen* related to knowledge deficit.
- *Risk of infection* related to protein energy malnutrition.
- *Impaired social interaction* related to widowhood and reduced social support group.

PLANNING

Plan to meet with Mrs Chow once each week to assess progress.

Expected outcomes

- Gain at least 0.5 kg per week.
- Verbalise understanding of nutritional requirements and identify strategies to incorporate requirements into daily diet after discharge.
- Remain infection-free, evidenced by normal vital signs.
- Identify strategies to increase social interaction, such as participating in senior citizens' lunches at local senior centre.

IMPLEMENTATION

- Weigh weekly at a consistent time of day.
- Refer to dietitian for evaluation of nutritional needs.
- Teach about nutritional requirements and plan an eating program that includes high-kilojoule, high-protein foods and supplements and reflects her food preferences.
- Encourage small, frequent meals.
- Encourage keeping a food intake diary.
- Teach strategies to reduce risks for infection.
- Provide information about communal meals available to seniors in the community and help Mrs Chow develop a plan to participate.

EVALUATION

One month later, Mrs Chow has gained 1.5 kg and reports feeling 'more energetic'. A friend is helping her shop to ensure that she purchases foods to maintain her protein, kilojoule and nutrient intake. She has begun attending senior lunches twice a week and is enjoying 'being around people again'. Although she still doesn't enjoy cooking like she used to, she is using prepared foods and supplements to maintain her nutrient intake.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the physiological basis for Mrs Chow's low albumin and cholesterol levels?
2. Mrs Chow asks, 'Can I get better by just taking more vitamins?' How will you respond?
3. Design a teaching plan for a person with protein energy malnutrition.

REFLECTION ON THE NURSING PROCESS

1. What role did culture have on Mrs Chow's risk of protein malnutrition?
2. What role did being an older person have on Mrs Chow's nutritional intake?

Imbalanced nutrition: less than body requirements

The nurse plays a critical role in the ongoing assessment of the malnourished person, while collaborating with the multidisciplinary team to provide nutritional therapies.

- If the person is able to eat, provide an environment and nursing measures that encourage eating. Eliminate foul odours, provide oral hygiene before and after meals, make meals appetising and offer frequent, small meals including preferred cultural foods. Consult with the nutrition support team to provide adequate protein, kilojoules, minerals and vitamins. *Oral hygiene and a pleasant environment make food more appetising. Small, frequent meals are generally more appealing and less overwhelming to a person with anorexia. Many require complicated nutritional therapy, such as enteral or parenteral therapy, to meet nutritional needs.*
- Provide a rest period before and after meals. *Eating requires energy and the malnourished person may have decreased physical strength and energy.*
- Assess knowledge and provide appropriate teaching. *Lack of knowledge often contributes to undernutrition. Education empowers the person to make healthy choices.*

Risk of infection

Malnourished people have a much higher risk of infection than well-nourished people. Malnutrition affects many components of the immune system, including the skin, mucous membranes, and lymph tissue and cells.

- Monitor temperature and assess for manifestations of infection every 4 hours. *Although the baseline temperature may be subnormal in malnourished people, any elevation from baseline may indicate infection. Manifestations of*

infection may include chills, malaise, erythema and leukocytosis. Early detection of infection may prevent complications.

- Maintain medical asepsis when providing care and surgical asepsis when carrying out procedures. *Handwashing is the best strategy to prevent the spread of pathogens. Sterile technique is required for procedures such as inserting central lines and changing dressings.*
- Teach the signs and symptoms of infection, good handwashing technique and factors that increase the risk of infection. *Knowledge empowers the person to participate in self-care, thus reducing exposure to infectious pathogens.*

Risk of fluid volume deficit

The person with malnutrition may also have a fluid volume deficit due to difficulty swallowing fluids. Administration of hyperosmolar nutritional solutions may lead to dehydration or electrolyte disturbances.

- Monitor oral mucous membranes, urine specific gravity, level of consciousness and laboratory findings every 4 to 8 hours. *Dry mucous membranes, increased urine specific gravity, decreased level of consciousness and electrolyte imbalances may indicate dehydration.*
- Weigh daily and monitor intake and output on fluid balance chart. *Daily weights and intake and output measurements help monitor fluid balance.*
- If allowed, offer preferred fluids frequently in small amounts, considering the person's preferences. *Frequent, small amounts of fluids are better tolerated and promote adequate intake.*

Risk of impaired skin integrity

Skin integrity depends on adequate nutrition. Loss of subcutaneous tissue and muscle increase the risk of pressure ulcers. In addition, healing is impaired in malnourished people.

- Assess skin every 4 hours. *Baseline and ongoing assessments allow prompt identification of early skin breakdown.*
- Turn and position at least every 2 hours. Encourage passive and active range-of-motion exercises. *These measures reduce pressure and promote oxygenation of cells.*
- Keep skin dry and clean and minimise shearing forces. Keep linen smooth, clean and dry. Provide therapeutic beds, air mattresses or pads. *These nursing measures promote comfort and reduce the risk of skin breakdown.*

Community-based care

A person with malnutrition may be cared for at home or in the hospital with oral diet, enteral or parenteral therapy. Each year, it is more common to see people managing tube feeding or TPN at home. Health education for the person and their family includes the following topics:

- Diet recommendations and use of nutritional supplements.
- Where to obtain recommended foods and nutritional supplements.
- If continuing enteral or parenteral nutrition, how to (1) prepare and/or handle solutions, (2) add them to either the feeding tube or central line, (3) manage infusion pumps, (4) care for the feeding tube or central catheter, (5) recognise and manage problems and complications, and (6) how and when to notify the healthcare provider of problems.

The person with an eating disorder

Eating disorders are characterised by severely disturbed eating behaviour and weight management. The three most common eating disorders in Australia are anorexia nervosa, bulimia nervosa and binge-eating disorder (BED). Eating disorders are increasing in Australia and are more common in affluent societies that are exposed to mass media advertising and plentiful food. Eating disorders are the third most common chronic illness in adolescents in Australia after obesity and asthma. Eating disorders occur equally in males and females before puberty but females are affected in increasing numbers during adolescence and young adulthood (Gonzales, Kohn & Clarke, 2007). It was estimated in 2012 that eating disorders affected nearly 1 million Australians. Premature death from physical causes and by suicide in young adults who suffer from an eating disorder is higher than in the general population (Eating Disorders Victoria, 2015).

The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) advises that the diagnosis of **Anorexia nervosa** is characterised by: (1) the persistent restriction of oral energy intake leading to a significantly low BMI or BMI-for-age; (2) an intense fear of gaining weight or of becoming fat, or persistent ritualistic behaviour that interferes with weight gain despite having a lower than normal BMI; and (3) a disturbance in the way one's body weight or shape is experienced, undue influence of body shape and weight on self-evaluation, or a persistent lack of recognition of the seriousness of the low BMI (Eating Disorders Victoria, 2015). **Bulimia nervosa** is characterised by recurring episodes of binge eating followed by purge behaviours such as self-induced vomiting, use of laxatives or diuretics, fasting or excessive exercise at least twice a week for a period of greater than 3 months. A third eating disorder, **binge-eating disorder**, is believed to affect many more people than either anorexia or bulimia. Binge-eating disorder is characterised by recurrent episodes of binge eating—eating an excessive amount of food during a defined period of time and a sense of lack of control over eating during binge episodes (Eating Disorders Victoria, 2015).

Anorexia nervosa

Anorexia nervosa typically begins during puberty in an attempt to cope with a perceived stress (Gonzales et al., 2007). People with anorexia nervosa are usually high achievers who have a distorted body image and an irrational fear of gaining weight. Refusal to maintain body weight at or above a minimally normal level for height and body type is a common manifestation of anorexia nervosa. The person will maintain weight loss by restricted kilojoule intake, often accompanied by excessive exercise. Some may exhibit binge–purge behaviour. Although its cause is unknown, a number of risk factors, both biological and psychosocial, have been identified for anorexia nervosa.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 8:
Preventing and Managing Pressure Injuries

'The intention of this Standard is to prevent patients from developing pressure injuries and effectively managing pressure injuries when they do occur.' (Australian Commission on Safety and Quality in Health Care, 2011, p. 54)

Implementing this standard is achieved through organisations establishing systems to assist with prevention and management of pressure injuries. Patients should be screened on admission and appropriate strategies put in place to prevent pressure injury. People with pressure injuries are managed using best practice guidelines and affective communication is undertaken between all individuals involved in a person's care.

Caring for an individual experiencing a nutritional disorder requires the consideration that there is an increased risk of developing a pressure injury. Constant vigilance and adherence to facility guidelines is required to minimise the risk of the development of pressure injuries.

Source: © Australian Commission on Safety and Quality in Health Care.

Abnormal levels of neurotransmitters and other hormones may play a role. Current research suggests genetic factors linked with environmental factors may influence the development of eating disorders (Eating Disorders Victoria, 2015). Women who develop anorexia nervosa tend to be obsessive and perfectionist and often feel inadequate or unable to maintain control in their lives. Family, social or occupational (e.g. a career in modelling, television or ballet) pressures contribute to maintain low body weight. People with anorexia often experience depression or anxiety.

The manifestations and complications of anorexia nervosa are listed in the box below.

MANIFESTATIONS AND COMPLICATIONS Eating disorders

DISORDER	MANIFESTATIONS	COMPLICATIONS
Anorexia nervosa	▪ Weight < 85% of normal; muscle wasting ▪ Fear of weight gain, refusal to eat ▪ Disturbed body image, excessive exercise ▪ Amenorrhoea (loss of menses) ▪ Skin and hair changes (fine hair—lanugo—over body) ▪ Hypotension, bradycardia ▪ Hypothermia ▪ Constipation ▪ Insomnia	▪ Electrolyte and acid–base disturbances ▪ Reduced cardiac muscle mass, low cardiac output, arrhythmias ▪ Anaemia ▪ Hypoglycaemia, elevated serum uric acid levels ▪ Osteoporosis ▪ Enlarged salivary glands ▪ Delayed gastric emptying ▪ Abnormal liver function
Bulimia nervosa	▪ Weight often normal; may be slightly overweight ▪ Binge–purge behaviour ▪ Oligomenorrhoea or amenorrhoea ▪ Lacerations of palate; callus on fingers or dorsum of hand	▪ Enlarged salivary glands ▪ Stomatitis, loss of dental enamel ▪ Fluid, electrolyte and acid–base imbalances ▪ Arrhythmias ▪ Oesophageal tears, stomach rupture

Binge-eating disorder	▪ Usually overweight or obese
	▪ Recurrent episodes of binge eating (2 or more days a week for 6 months)
	▪ Episodes characterised by:
	  ▪ Eating more rapidly than usual
	  ▪ Eating until uncomfortably full
	  ▪ Eating large amounts of food when not physically hungry
	  ▪ Eating alone due to embarrassment over quantity
	  ▪ Disgust, depression or guilt following a binge episode
	  ▪ Marked distress about binging behaviour
		▪ Type 2 diabetes mellitus
		▪ Hypertension, hyperlipidaemia
		▪ Coronary heart disease, heart failure
		▪ Gallbladder disease
		▪ Depression, social isolation

Bulimia nervosa

Bulimia nervosa is an eating disorder in which people binge on food and then, in an attempt to counteract this behaviour, take extreme measures such as making themselves vomit, taking laxatives or starving themselves (Hay et al., 2014). Bulimia nervosa develops in late adolescence or early adulthood, often following failed attempts to lose weight through dieting. Foods consumed during a binge often are high in kilojoules and fat. After binge eating, the person induces vomiting (usually by stimulating the gag reflex) or may take excessive quantities of laxatives or diuretics. In contrast to anorexia, the person's weight is often normal. Fluid and electrolyte balance, in contrast, may be severely disrupted by loss of fluid and gastrointestinal secretions. The complications of bulimia nervosa (see the box below) primarily result from the purging behaviour.

Binge-eating disorder

Binge-eating disorder (BED) was identified as a condition distinct from overeating in 1992 (Eating Disorders Victoria, 2015). While many of the characteristics of bulimia and binge-eating disorder are similar with feelings of guilt, disgust and depression, a person with BED usually binges in private and does not purge. BED commonly affects middle-aged adults and is slightly more common in women than in men. People with BED usually eat when not hungry and are overweight or obese, often morbidly obese. Psychosocial factors contribute: up to half of people with BED either are depressed or have experienced depression in the past. Alcohol abuse and impulsivity are common behavioural traits in a person with BED.

INTERPROFESSIONAL CARE

Eating disorders, anorexia nervosa in particular, are difficult to treat effectively. Because of the intense fear of weight gain and distorted body image, these people strongly resist increasing food intake. In all cases, a comprehensive treatment plan for eating disorders includes medical care and monitoring, psychosocial interventions and nutrition counselling (Gonzales et al., 2007).

Diagnosis

There is no specific diagnostic test for anorexia, bulimia or binge-eating disorder. Laboratory studies of a person with anorexia or bulimia may show anaemia and leucopenia, abnormal serum electrolyte levels and elevated blood urea nitrogen (BUN) and serum creatinine. In people with BED, the blood glucose and lipid levels may be elevated. The BMI is usually above the normal range and may identify the person as obese or morbidly obese.

A mental health evaluation is indicated for people with eating disorders to identify contributing factors and help direct treatment.

Treatment

A multidisciplinary approach that includes psychology, psychiatry, medicine, dietetics, family therapy and social work is used In Australia (Hay et al., 2014). The aims of treatment are to restore weight, reverse malnutrition and relieve the mental anguish associated with eating.

Treatment is usually as an outpatient but some may require hospitalisation, particularly if their weight is less than 75% of normal. Refeeding is gradually introduced to avoid complications such as heart failure. Oral intake is the preferred option in refeeding. Meals must be supervised and a firm but empathetic attitude conveyed about the importance of adequate food intake. Enteral or intravenous feeding may be required to supplement oral intake. Psychological treatment, initiated when malnutrition has been corrected and weight gain begun, focuses on providing emotional support and helping the person base their self-esteem on factors other than weight (e.g. personal relationships, satisfaction with achieving occupational goals) (Hay et al., 2014). Cognitive–behavioural therapy or psychotherapy may be used; families may be included in the treatment program. A selective serotonin reuptake inhibitor (SSRI) such as olanzapine (Zyprexa), quetiapine (Seroquel) or risperidone (Risperdal) may be prescribed to facilitate weight maintenance and reduce anxiety.

The goal of bulimia treatment is to reduce or eliminate binge eating and purging behaviour. A combination of nutritional counselling and therapy, psychosocial interventions and medications may be used. Nutritional counselling is directed at establishing a regular meal pattern and encouraging an appropriate amount of regular exercise. An SSRI may be of benefit. Cognitive–behavioural therapy also is used to treat bulimia, focusing on excessive concerns about weight, persistent dieting and binge–purge behaviours.

Treatment for those with BED focuses on establishing healthy eating patterns, psychosocial therapy (including cognitive–behavioural therapy and group counselling) to address underlying issues and management of obesity and its complications. A person with BED may also benefit from an SSRI or other antidepressant drug.

Nursing care

Nurses can be instrumental in identifying a person with an eating disorder and referring for treatment. It is particularly important to identify these disorders early to prevent adverse effects on growth and increase the success of treatment.

The nurse is an integral part of the eating disorders treatment team. *Imbalanced nutrition: less than body requirements* is a primary nursing diagnosis for a person with anorexia or

bulimia, and *Imbalanced nutrition: more than body requirements* is a priority nursing diagnosis for people with binge-eating disorder (BED). The following nursing diagnoses also should be considered:

- *Ineffective sexuality patterns* related to body image concerns.
- *Chronic low self-esteem* related to tendency to binge eat.
- *Disturbed body image* related to altered eating pattern.
- *Ineffective therapeutic regimen* related to knowledge deficit.

When planning and implementing care, consider the following nursing activities:

- Ensure a commitment from the person that they wish to alter their eating patterns.
- Regularly monitor weight each week using standard conditions. *Weight gain or loss provides information about the effectiveness of care, as well as the person's risk of complications.*
- Monitor food intake during meals, recording percentage of meal and snacks consumed. Maintain close observation for at least 1 hour following meals; do not allow the person to be alone in the bathroom. *Observing the person during and after meals helps prevent disposal of food and purging behaviour after eating. Recording actual food intake allows accurate calculation of kilojoule intake.*
- Serve balanced meals, including all nutrient groups. Increase serving size gradually. The person may find 'normal' food servings overwhelming, reducing the desire to eat. *Kilojoule intake is initially limited to prevent complications associated with refeeding, then gradually increased.*
- Administer a multivitamin and mineral supplement to replace losses.

Involvement of the family and social support persons is vital to success. Encourage family members to participate in teaching and nutritional counselling sessions. Discuss the value of family therapy to address issues that have contributed to the disorder. Emphasise the need to provide consistent messages of support for healthy eating habits. Discuss using rewards for food and kilojoule intake rather than weight gain. Provide referrals to a dietitian, nutritional support team, counselling and support groups for people with eating disorders.

CHAPTER HIGHLIGHTS

- Nutritional disorders are common, affecting people worldwide and contributing significantly to mortality and morbidity. While malnutrition is a serious problem in underdeveloped nations, obesity and its consequences are more prevalent in Australia and other industrialised societies.
- Obesity, defined as excess adipose tissue and a BMI greater than 30 kg/m^2, is linked with many disorders, including type 2 diabetes mellitus, coronary heart disease, gallbladder disease and osteoarthritis.
- Exercise and reduced kilojoule intake are the mainstays of obesity treatment. Drugs that suppress the appetite or interfere with fat absorption in the gut may be used to facilitate weight loss in a person with multiple risk factors for obesity complications or people who have had difficulty achieving weight loss through diet and exercise.
- Bariatric surgery is a viable treatment option for the morbidly obese. The primary types of bariatric surgery used in Australia are restrictive and malabsorptive procedures that limit stomach capacity and nutrient absorption.
- Nursing care for obese people focuses on health promotion, education and support of the prescribed treatment plan.
- In Australia, protein energy malnutrition is a common problem among the elderly and hospitalised people. Malnutrition increases the risk of complications and impairs healing. Early identification and prevention are the primary focuses of treatment; nurses can be instrumental in identifying at-risk people (e.g. the elderly, a person living alone, people on extended NBM status).
- Refeeding of malnourished people is a gradual process. Enteral feedings (oral or by feeding tube) are preferred whenever possible. Parenteral nutrition may be required when enteral feeding is not possible or not tolerated.
- Eating disorders, including anorexia nervosa, bulimia nervosa and binge-eating disorder, can be difficult to effectively treat and maintain in remission. While a person with anorexia typically is underweight and malnourished, resisting efforts to achieve a normal weight, a person with bulimia is more likely to be of normal weight and those with binge-eating disorder tend to be overweight or obese.
- Treatment for eating disorders is multifaceted, including physical care to restore electrolyte balance and treat complications, nutritional counselling and therapy, psychosocial therapy, family support and possibly medications.

CONCEPT CHECK

1 Of the following noted in a person's history, which does the nurse identify as risk factors for obesity?
   1 adopted at 2 months of age
   2 usual diet includes 'fast-food' meals twice a week
   3 does not engage in regular activity
   4 allergic to chocolate and strawberries

2 A person on a reduced-kilojoule diet asks the nurse what she can do to lose weight faster, because most weeks she loses no more than 0.2 kg. 'At this rate, it will take me years to get to my goal!'
1 'Let's re-evaluate your long-term goal. Perhaps it was set too low for you.'
2 'A kilogram of body fat equals 14 650 kJ. Let's re-evaluate your diet and exercise plan for kilojoule intake and expenditure.'
3 'Perhaps we should look into a diet supplement since you are unable to stick with your prescribed diet plan.'
4 'You sound frustrated. Would you like to take some time off from your diet and exercise plan?'

3 An expected finding in a person admitted with a diagnosis of protein energy malnutrition would be:
1 recent 5 kg weight loss
2 increased anthropometric skinfold thickness measurements
3 hyperactive bowel sounds
4 anxiety and agitation

4 Before administering an intermittent enteral feeding, the nurse confirms placement of the small-bore feeding tube in the stomach by:
1 instilling water and listening for the gastric gurgle
2 withdrawing the tube slightly, then reinserting it
3 aspirating gastric contents and checking for a pH of < 4
4 obtaining an x-ray of the chest and stomach

5 The nurse identifies which of the following as realistic goals for a person with anorexia nervosa?
1 will consume 100% of a 10 465 kJ diet
2 will gain 0.5 kg per week
3 will rest alone in room following meals
4 will participate in family counselling

6 The nurse identifies which nursing diagnosis as high priority for a person with a BMI of 30.4 kg/m^2 and a waist-to-hip ratio of 1.1?
1 *Health-seeking behaviours: weight loss*
2 *Risk of impaired cardiovascular tissue perfusion*
3 *Ineffective coping*
4 *Deficient knowledge regarding diet*

7 The nurse teaching a person about sibutramine (Reductil) includes which of the following instructions?
1 You may skip a dose of the drug if you skip a high-fat meal.
2 Do not consume alcohol while taking this drug.
3 Do not drive while taking this drug because the drug may increase sleepiness.
4 Increase your intake of water and other fluids while taking this drug.
5 Continue to follow your prescribed diet while taking this drug.

8 The nurse caring for a home-bound older adult who is losing 1 to 2 kg monthly plans for which of the following?
1 Meals on Wheels deliveries
2 ensure nutritional supplements
3 placement in a residential care facility
4 transportation to congregate senior meals
5 follow-up by primary care physician
6 referral for diagnostic studies

9 Which of the following is a high-priority nursing intervention to prevent malnutrition in the person undergoing surgery?
1 aggressive pain management
2 daily weighing
3 maintaining intravenous flow
4 requesting early restoration of oral intake

10 Three days after gastric bypass surgery, the person complains of increasing abdominal pain. Bowel sounds are absent; the abdomen is firm and very tender. The nurse should:
1 report findings to the surgeon
2 ambulate the person to promote peristalsis
3 chart assessment data and continue to monitor
4 evaluate the effectiveness of analgesia

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CHAPTER 22

NURSING CARE OF PEOPLE WITH UPPER GASTROINTESTINAL DISORDERS

TRACY PARRISH

LEARNING OUTCOMES

- Describe the pathophysiology and complications associated with common disorders of the mouth.
- Discuss the pathophysiology, manifestations and nursing care of a person with a disorder of the oesophagus.
- Describe the pathophysiology, manifestations and complications associated with common disorders of the stomach and duodenum.

CLINICAL COMPETENCIES

- Assess the functional health status of people with upper gastrointestinal disorders.
- Monitor, document and, as needed, report manifestations of upper gastrointestinal disorders and their complications.
- Plan nursing care using evidence-based research.
- Determine priority nursing diagnoses and interventions based on assessed data.
- Administer medications and prescribed care knowledgeably and safely.
- Coordinate and integrate interprofessional care into the plan of care.
- Construct and revise individualised plans of care, considering the culture and values of the person.
- Plan and provide the person and family with teaching to promote, maintain and restore functional health.

KEY TERMS

The upper gastrointestinal (GI) tract includes the mouth, oesophagus, stomach and proximal small intestine. Food and fluids, ingested through the mouth, move through the oesophagus to the stomach. The stomach and upper intestinal tract (duodenum and jejunum) are responsible for the majority of food digestion. When an acute or chronic disease process interferes with the function of this portion of the GI tract, nutritional status can be affected and the person may experience symptoms that interfere with their lifestyle.

Nurses provide both acute care for the hospitalised person and education about the skills and knowledge needed to manage these conditions at home.

DISORDERS OF THE MOUTH

Inflammations, infections and neoplastic lesions of the mouth affect food ingestion and nutrition. Oral lesions may have a variety of causes, including infection, mechanical trauma, irritants such as alcohol, and hypersensitivity. Appropriate treatment of the disorder, any underlying factors and associated symptoms is essential.

THE PERSON WITH STOMATITIS

Stomatitis, inflammation of the oral mucosa, is a common disorder of the mouth. It may be caused by viral (herpes simplex) or fungal (*Candida albicans*) infections, mechanical trauma (e.g. cheek biting) or irritants such as tobacco or chemotherapeutic agents. Stomatitis is a common side effect of cancer treatment, occurring in 40% of people receiving standard dose chemotherapy and more than 85% of people receiving head and neck radiation (Caplinger, Royse & Martens, 2010).

Box 22.1 outlines risk factors for stomatitis.

FAST FACTS

- Specific chemotherapy treatments, high-dose and dose-dense chemotherapy protocols increase the risk of stomatitis.
- Repetitive radiation to the head and neck or radiation directly to the oral mucosa contributes to stomatitis.
- Adherence to an oral healthcare protocol can reduce the severity and duration of stomatitis.
- Healing time can take from 2–3 weeks unless there is a complicated infection or repeated doses of chemotherapy or radiation (Caplinger et al., 2010; Cheng, 2007; Harris et al., 2008).

Pathophysiology and manifestations

Radiotherapy and chemotherapy damage the DNA of basal epithelial cells, resulting in necrosis and death of some cells. This stimulates the release of inflammatory cytokines such as tumour necrosis factor alpha (TNF-α) and interleukins that further damage tissues, causing additional epithelial cells to die and greater injury to the mucosa. The injury to the mucosa is manifested in extremely painful lesions, which are portals for bacteria, viruses and fungi. This may lead to bacteraemia and sepsis; ultimately, however, healing of the mucosa will occur (Niscola et al., 2008).

The clinical manifestations of stomatitis vary according to its cause; however, the main symptom is pain. Pain may vary from a mild burning through to severe pain and may be associated with nausea, hypersalivation and infection (Niscola et al., 2008). Table 22.1 outlines common causes of stomatitis with their manifestations and treatment.

Stomatitis can lead to malnutrition, fluid and electrolyte imbalance, an increased risk of infection (especially for neutropenic patients), a reduction in quality of life and increased hospital stays.

BOX 22.1 Risk factors for stomatitis

- Age > 65 years
- Impaired immune status (HIV disease, cancer, diabetes)
- Chronic renal failure or heart failure
- Chemotherapy, radiation therapy, stem cell transplant
- Oxygen therapy, mouth breathing
- Medications (antibiotics, phenytoin, anticholinergics, corticosteroids)
- Poor oral hygiene, ill-fitting dentures
- Tobacco or alcohol use

INTERPROFESSIONAL CARE

Stomatitis is diagnosed by direct physical examination and, if indicated, cultures, smears and evaluation for systemic illness. Assessment tools such as the World Health Organization Mucositis Assessment Tool are available to assist with diagnosis.

Treatment addresses both the underlying cause and any coexisting illnesses. An undiagnosed oral lesion present for more than 1 week and which does not respond to therapy must be evaluated for malignancy.

Direct smears and cultures of lesions may be obtained to identify causative organisms. If systemic illness is suspected, a variety of diagnostic tests may be ordered to identify the underlying cause.

The best management for mucositis, the oral form of stomatitis, due to chemotherapy or radiation is prevention through education on oral care and diet. Oral care reduces the amount of microbial flora, and reduces pain and bleeding which assists in preventing infection. General treatment measures include providing meticulous oral hygiene four times a day with a soft toothbrush or sponge swab and flossing. Bland oral mouth rinses such as normal saline or water and sodium bicarbonate mixed

TABLE 22.1 Manifestations and treatment of common stomatitis conditions

TYPE	CAUSE	MANIFESTATIONS	TREATMENT
Cold sore, fever blister	Herpes simplex virus	• Initial burning at site • Clustered vesicular lesions on lip or oral mucosa	• Self-limiting • Aciclovir, valaciclovir to shorten course
Aphthous ulcer (canker sore, ulcerative stomatitis)	Unknown; may be type of herpes virus	• Well-circumscribed, shallow erosions with white or yellow centre encircled by red ring • Less than 1 cm in diameter • Painful	• Topical steroid ointment • Oral prednisone
Candidiasis (thrush)	*Candida albicans*	• Creamy white, curd-like patches • Red, erythematous mucosa	• Fluconazole (Diflucan) • Ketoconazole (Nizoral) • Clotrimazole pessaries • Nystatin mouth rinse
Necrotising ulcerative gingivitis (trench mouth, Vincent's infection)	Infection with spirochetes and bacilli or systemic infection	• Acute gingival inflammation and necrosis • Bleeding, halitosis • Fever • Cervical lymphadenopathy	• Correct any underlying disorders • Oral penicillin
Oral mucositis	Damage to epithelial cells and stem cells in the submucosa caused by chemotherapy or radiation therapy	• Erythema and inflammation of oral mucosa • Painful, irregularly shaped ulcerations, initially superficial, progressing to deep ulcers that may be confluent (overlapping with one another) • Pseudomembranes covering ulcers • Tissue necrosis with spontaneous bleeding, potential sepsis	• Regular oral hygiene with brushing and flossing • Sodium bicarbonate solution or normal saline mouth rinses after and between meals • Lignocaine viscous gel before meals for analgesia • Cryotherapy before, during and after chemotherapy administration

together can be used to loosen debris in the mouth, reduce the acidity of oral fluids, dilute accumulating mucus and discourage yeast colonisation. The person should be instructed to take a tablespoon of the rinse, swish it in their mouth for at least 30 seconds, and then expectorate (Harris et al., 2008; Munn, 2008).

Cryotherapy, the use of ice chips or ice-cold water for the prevention of oral mucositis, is also recommended for use in chemotherapy agents with a short half life. The person sucks on ice or holds ice-cold water in their mouth prior to, during and after rapid infusion of the chemotherapy agent. Cryotherapy is based on the theory that vasoconstriction decreases exposure of the oral mucous membranes to the chemotherapy agent (Harris et al., 2008; Weiner & Deeken, 2010).

Medications

Using a topical anaesthetic, such as 2% viscous lignocaine, diphenhydramine (Benadryl) solution or benzocaine spray or gel can promote comfort and the ability to consume oral food and fluids. Lignocaine solution is not swallowed, to avoid impairment of the swallowing mechanism. Orabase, a protective paste, may be applied to oral ulcers to promote comfort. Triamcinolone acetonide may be mixed in Orabase to reduce inflammation and promote healing. Sodium bicarbonate or normal saline mouthwashes may provide relief and promote cleansing, whereas alcohol-based mouthwashes may cause pain and burning and should be avoided.

Fungal infections often are treated with a nystatin oral suspension; the person 'swishes and swallows' the solution. Clotrimazole lozenges also treat oral fungal infections. If the infection does not resolve, oral antifungal medications such as fluconazole or ketoconazole may be used. Antifungals are usually continued for at least 3 days after symptoms disappear.

Herpetic lesions may be treated with topical or oral aciclovir of valaciclovir (Valtrex). Aciclovir ointment provides comfort and lubrication while limiting the spread of the virus. Aciclovir capsules reduce the severity of symptoms and the duration of the lesions.

Bacterial infections are treated with antibiotics based on cultures and smears. Oral penicillin is the treatment of choice if the person is not allergic and the cultured bacteria are sensitive. Nursing implications for selected drugs used to treat stomatitis are outlined below.

Nursing care

Health promotion

Nurses can help prevent stomatitis by identifying people at risk and suggesting measures to reduce the likelihood that stomatitis will develop. Educate and encourage all people to regularly perform mouth care, including teeth brushing and flossing. Written information sheets or posters that can be displayed in

MEDICATION ADMINISTRATION Drugs used to treat stomatitis

TOPICAL ORAL ANAESTHETICS

Viscous lignocaine
Triamcinolone acetonide (Kenalog in Orabase)

These drugs reduce the pain associated with mucous membrane lesions or stomatitis. They provide temporary relief of pain. Any oral lesion that persists longer than 1 week should be evaluated by an oral surgeon.

Nursing responsibilities

- Instruct the person to seek medical attention for any oral lesion that does not heal within 1 week.
- Monitor for local hypersensitivity reactions and discontinue use if they occur.

Health education for the person and family

- Apply every 1 to 2 hours as needed.
- Perform oral hygiene after meals and at bedtime.

TOPICAL ANTIFUNGAL AGENTS

Clotrimazole (Canestan)
Nystatin (Nilstat)

These products help in the topical treatment of candidiasis. Their effects are primarily local rather than systemic.

Nursing responsibilities

- Instruct the person to dissolve lozenges in the mouth.
- Instruct the person to rinse mouth with oral suspension for at least 2 minutes and expectorate or swallow as directed.
- These drugs are contraindicated in pregnancy.

Health education for the person and family

- Take medication as prescribed.
- Do not eat or drink 30 minutes after medication.
- Contact physician if symptoms worsen.
- Perform good oral hygiene after meals and at bedtime; remove dentures at bedtime.

ANTIVIRAL AGENTS

Aciclovir (Zovirax)
Valaciclovir (Valtrex)

Aciclovir and valaciclovir are useful in the treatment of oral herpes simplex virus. They help reduce the severity and frequency of infections. These antiviral agents interfere with the DNA synthesis of herpes simplex virus.

Nursing responsibilities

- Start therapy as soon as herpetic lesions are noted.
- Administer with food or on an empty stomach.

Health education for the person and family

- The virus remains latent and can recur during stressful events, fever, trauma, sunlight exposure and treatment with immunosuppressive drugs.
- Take the medication as ordered and contact the physician if symptoms worsen.

the person's bathroom outlining an oral care regimen, with symptoms that should be reported to clinicians, may promote compliance and reduce risk of oral mucositis occurring. Verify the person's understanding with return explanation and demonstration (Moore et al., 2009; Harris et al., 2008).

Encourage people with ill-fitting dentures or other dental prostheses (such as partial plates) to see a qualified dentist or dental prosthetist. Suggest people taking an extended course of antibiotic therapy or who have impaired immune function consume yoghurt containing live bacterial cultures unless contraindicated. Discuss dietary modifications, such as limiting consumption of highly spiced or acidic foods and avoiding very hot beverages. A high-protein diet and increased oral fluids will assist in mucous membrane regeneration. People undergoing chemotherapy or radiation therapy should avoid use of alcohol and tobacco because these substances further damage oral mucosa, increasing the risk of oral mucositis. Instead suggest the person chew or suck ice during chemotherapy sessions.

Assessment

Oral assessment is important not only for people who have been diagnosed with stomatitis but also for those with risk factors, manifestations or evidence of possible complications (e.g. recent weight loss).

- *Health history:* complaints of mouth pain, altered taste, dysphagia, lack of appetite, malaise; presence of dentures, regularity of dental care; current health status including chronic diseases; current medications; use of alcohol or tobacco.
- *Physical examination:* inspect lips, gums, teeth, interior cheeks, tongue and base of tongue, soft and hard palate; tonsils, oral pharynx and the amount of saliva. Observe and assess general health status including temperature and weight.
- *Diagnostic tests:* WBC, sedimentation rate, serum albumin.

Nursing diagnoses and interventions

Nursing care for the person with stomatitis or oral mucositis focuses not only on the oral inflammation, but also on any underlying systemic diseases, and the effects of the condition on the person's comfort and nutrition.

Impaired oral mucous membrane related to loss of integrity of oral mucous membrane

Regardless of cause, the pain and symptoms must be relieved to promote comfort as well as food and fluid intake.

- Assess and document oral mucous membranes and the character of any lesions every 4 to 8 hours. Baseline and ongoing assessment data provide the basis for evaluation. Teach the person or their caregiver to perform daily oral assessments in the outpatient setting and to report findings to clinicians as required (Harris et al., 2008).
- Assist with thorough mouth care after meals and at bedtime. Brush all tooth surfaces for at least 90 seconds twice daily with a soft toothbrush and floss at least once daily. Allow toothbrush to air dry before storing. If unable to tolerate a toothbrush, offer sponge swabs. Avoid using alcohol-based mouthwashes or lemon-glycerin swabs as

these may dry and irritate mucous membranes, causing pain and further tissue damage. Providing bland rinses such as sodium bicarbonate and normal saline after meals promotes comfort and reduces risk of infection (Maguire et al., 2013).
- Assess knowledge and teach about condition, mouth care and treatments. Instruct to avoid alcohol, tobacco and spicy or irritating foods. Knowledge promotes the person's participation in the plan of care and compliance. Alcohol, tobacco and hot, spicy or rough foods may injure the inflamed mucous membranes.

Risk of imbalanced nutrition (less than body requirements) related to oral pain

Oral lesions and pain may limit oral intake, which may in turn lead to nutritional deficits. Anorexia and general malaise may also contribute to decreased intake.
- Assess food intake as well as the person's ability to chew and swallow. Weigh daily. Provide appropriate assistive devices such as straws or feeding syringes. *Adequate nutrition is essential for healing. Daily weights allow monitoring of the adequacy of food intake. Assistive devices may allow food intake while avoiding irritation of ulcerations or lesions.*
- Encourage a high-kilojoule, high-protein diet considerate of food preferences. Offer soft, lukewarm or cool foods or liquids, such as milk shakes, nutritional supplements, iceblocks and puddings, frequently in small amounts. Obtain nutritional consultation. *Oral intake may be limited and enriched foods and liquids enhance nutrition. A nutritional consultation can help ensure an adequate diet and assist in meeting nutritional needs.*
- Provide analgesics for pain relief as needed. *Significant pain associated with stomatitis or oral mucositis can interfere with effective mouth care and food and fluid intake. Pain management is a vital part of nursing care.*

Community-based care

People with mild stomatitis generally provide self-care. While people with cancer-treatment-related oral mucositis may require more aggressive therapy, the person and caregivers are often able to manage the regimen in home or community-based settings. Include the following topics in teaching for home care:
- management of underlying health conditions and ongoing treatments such as chemotherapy
- the recommended diet and oral hygiene regimen, including foods and substances to avoid (e.g. alcohol, tobacco products)
- nutritional supplements to help meet nutritional requirements
- prescribed medication, its route, side effects, frequency of administration and signs and symptoms to report
- the importance of completing the full course of antibiotic, antiviral or antifungal treatment
- manifestations to report and the importance of follow-up care.

THE PERSON WITH ORAL CANCER

Oral cancer, malignancy of the oral mucosa, may develop on the lips, tongue, floor of the mouth or other oral tissues. It is uncommon, accounting for 2–3% of all malignancies and 0.5% of all deaths in Australia in 2006 (Australian Institute of Health and Welfare (AIHW), 2012; Kademani, 2007). It has, however, a high rate of morbidity and mortality. The incidence of this type of head and neck cancer is three times as high in men as in women, and it is seen more often in men over age 40 (AIHW, 2012). The stage of an oral cancer determines the prognosis, treatment and degree of disability. The primary risk factors for oral cancer are smoking, drinking alcohol and chewing tobacco. Human papillomavirus (HPV) has been associated in 14–22% of oropharyngeal tumours and increases the risk of oral cancer development 3- to 5-fold (Kademani, 2007). Marijuana use and occupational exposures to chemicals also may contribute to the risk of oral cancer.

Pathophysiology and manifestations

Oral cancer is usually a squamous cell carcinoma. Although a cancerous lesion can develop in any area of the mouth, the most common sites are the lower lip, tongue and floor of the mouth. Most early cancers present as inflamed areas with irregular, ill-defined borders. These lesions typically are not painful. More advanced cancers appear as deep ulcers that are fixed to deeper tissues. Early lesions involve the mucosa or submucosa, whereas more advanced tumours may invade and destroy underlying tissues, including muscles and bones of the face. Tumours frequently metastasise to regional lymph nodes. Other cancerous lesions, including lymphoma, malignant melanoma and Kaposi's sarcoma, may also develop in the mouth, although less frequently than squamous cell carcinoma.

The person may be asymptomatic with oral cancer in the early stages and detection is through routine screening or referral when symptoms develop. The earliest symptom of oral cancer is a painless oral ulceration or lesion (see Figure 22.1). Later symptoms vary and may include mass lesions, difficulty speaking, swallowing or chewing, loose teeth, earache, bleeding, swollen lymph nodes and sensory or motor nerve compromise (Kademani, 2007). See the accompanying box for other manifestations of oral cancer. Any oral lesion that does not heal or respond to treatment within 1 to 2 weeks should be evaluated for malignancy.

MANIFESTATIONS Oral cancer

- White patches (leucoplakia)
- Red patches (erythroplakia)
- Ulcers
- Masses
- Pigmented areas (brownish or black)
- Fissures
- Asymmetry of the head, face, jaws or neck

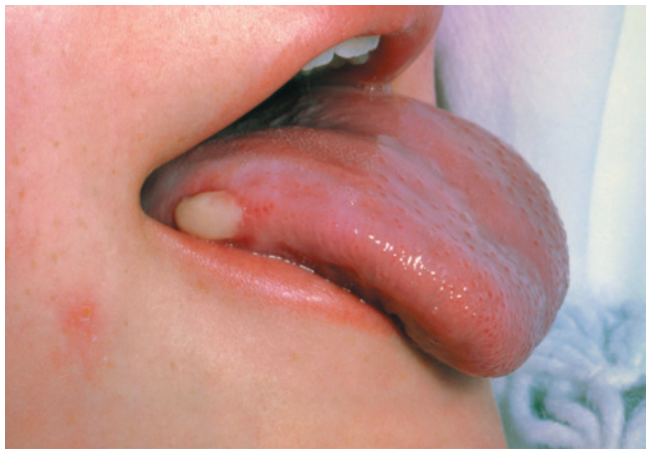


FIGURE 22.1 ■ Oral cancer

Source: © John Radcliffe Hospital/Science Source.

INTERPROFESSIONAL CARE

The first component of treatment is eliminating any causative factors such as chewing tobacco, smoking or drinking alcohol. Tumour staging then determines therapy. The TNM (tumour, nodes, metastasis) classification is used to stage oral cancer (see Box 22.2). A biopsy of the oral lesion allows direct visualisation of cells to determine the presence or absence of cancerous cells. Staging may require additional diagnostic studies such as computed tomography (CT) scans or magnetic resonance imaging (MRI).

Radiation and chemotherapy may be considered based on the person's age, tumour stage, general condition and preferences. Radiation therapy may be used preoperatively to 'shrink' the tumour or postoperatively to limit the risks of metastasis. Chemotherapy may be indicated depending on the stage of the tumour. See Chapter 13 for more information about radiation and chemotherapy to treat cancer.

Following the biopsy and staging of the tumour, surgery is generally indicated, although an advanced or extensive tumour may be considered unresectable. If the tumour involves surrounding tissues, the cosmetic effects of surgery are important considerations. The goal of surgery is removal of the lesion and potentially cancerous surrounding tissue or lymph nodes. Advanced carcinomas may require extensive excision or a *radical neck dissection*, a potentially disfiguring procedure in which the lymph nodes and muscles of the neck are removed. A tracheostomy is performed at the time of surgery. The tracheostomy may be temporary but is often permanent. See Chapter 34 for more information about caring for a person following radical neck dissection and a tracheostomy.

BOX 22.2 Oral cancer staging

Stage 0	Carcinoma in situ
Stage I	Tumour ≤ 2 cm; no regional node involvement
Stage II	Tumour > 2 cm to ≤ 4 cm; no regional node involvement
Stage III	Tumour ≤ 2 cm to > 4 cm; one involved lymph node
Stage IVA & B	Tumour may invade adjacent structures; one or more nodes involved
Stage IVC	Distant metastasis present

Nursing care

Health promotion

Reducing or eliminating tobacco use (smoking and smokeless tobacco) and excess alcohol consumption can significantly reduce the incidence of oral cancer. Teach children and adolescents about the dangers of using tobacco and alcohol. Emphasise the relationship between smokeless tobacco and oral cancer. Discuss strategies to deal with peer pressure to use tobacco and alcohol.

To promote early identification of and intervention for oral cancer, teach people about the risk factors for and manifestations of the disease. With the link between HPV and oral cancer (Kademani, 2007), promoting vaccination against HPV should also be considered.

Assessment

Early precancerous oral lesions are very treatable. Unfortunately, these lesions usually are painless, so diagnosis and treatment often is delayed. Assess the oral cavity of all people, particularly those with risk factors for oral cancer.

- *Health history:* complaints of oral lesions that fail to heal; use (current or past) of tobacco products or excess alcohol.
- *Physical examination:* inspect and palpate lips and oral mucosa (including tongue and floor of mouth under the tongue) for tumours or lesions. Lesions may appear as velvety red or white patches that do not scrape off or as ulcers or areas of necrosis.

Nursing diagnoses and interventions

The mouth allows food ingestion and the lips are integral to verbal and non-verbal expression. The head, mouth and lips are important to self-perception and body image. Nursing diagnoses discussed in this section consider such problems as airway clearance, nutrition, communication and body image. See the accompanying nursing care plan below.

Risk of ineffective airway clearance related to potential oral obstruction by the cancer

The location and the extent of an oral cancer and its excision may compromise the airway. Swelling of adjacent tissues, increased oral secretions or difficulty swallowing may contribute to respiratory distress. If extensive surgery is performed, a tracheostomy is usually performed to maintain airway patency.

CONSIDERATION FOR PRACTICE

In the initial postoperative period, assess airway patency and respiratory status at least hourly. A patent airway is vital to maintain respirations and oxygenation of tissues. Frequent assessment allows early identification of possible airway compromise.

- Unless contraindicated, place in high-Fowler's position, supporting arms. Assist the person to turn, cough and deep breathe at least every 2 to 4 hours. *High-Fowler's position promotes lung expansion. Turning, coughing and deep breathing help maintain a patent airway by preventing pooling of secretions.*
- Maintain adequate hydration (2000 to 3000 mL per day unless contraindicated) and humidity of inspired air. *Adequate hydration helps thin and loosen secretions.*

Risk of imbalanced nutrition (less than body requirements) related to inability to tolerate oral intake

Surgery affects oral food and fluid intake. Enteral feedings or total parenteral nutrition may be required. A gastrostomy tube usually is inserted during surgery to maintain nutrition. If an oral diet is permitted, anorexia or pain may affect intake.

NURSING CARE PLAN A person with oral cancer

Gavin Sandford, a married 44-year-old farmer, has two adult children. He and his wife grow and sell fruit and vegetables. Two months ago, Mr Sandford developed a sore on his tongue that would not heal. Mr Sandford tells his admission nurse, Sara Bucklin, 'The doctor says he will have to remove part of my tongue', and anxiously asks, 'Will I look the same? How will I be able to talk?'

ASSESSMENT

Mr Sandford's admission history reveals that he has been healthy, but has smoked two packets of cigarettes a day for more than 20 years and usually drinks two to four beers per day. He admits to being anxious and fearful of surgery and its outcomes. He says he quit smoking and drinking 2 weeks ago. The biopsy report is positive for squamous cell carcinoma of the tongue. Mr Sandford has no enlarged cervical nodes and says he has no bloody sputum or saliva, difficulty swallowing, chewing or talking. His weight is in the normal range for his height. A wide excision of the oral lesion is planned.

DIAGNOSES

- *Risk of ineffective airway clearance* related to oral surgery.
- *Risk of imbalanced nutrition (less than body requirements)* related to oral surgery.
- *Impaired verbal communication* related to excision of a portion of the tongue.
- *Disturbed body image* related to surgical excision of the tongue.

PLANNING

- Teach the importance of activity, turning, coughing and deep breathing prior to the surgery.
- Encourage Mrs Sandford to visit at mealtimes to assist with and encourage oral intake.
- Demonstrate and allow practising using magic slate and flash cards prior to surgery.
- Arrange for a dietician review prior to surgery to assess energy needs and plan appropriate enteral feeding.
- Discuss the grief process with Mr Sandford and his family and encourage expression of feelings.

Expected outcomes

- Maintain a patent airway and remain free of respiratory distress.
- Maintain a stable weight and level of hydration.
- Effectively communicate with staff and family using a magic slate and flash cards.
- Communicate an increased ability to accept changes in body image.

IMPLEMENTATION

- Assess airway patency and respiratory status every hour until stable.
- Maintain semi-Fowler's position, supporting arms. Encourage to turn, cough and deep breathe every 2 to 4 hours.
- Monitor daily weights.
- Assess response to enteral feedings.
- Allow adequate time for communication efforts using magic slate and flash cards where necessary.
- Keep buzzer in reach at all times and answer promptly. Alert all staff of inability to respond verbally.
- Provide emotional support and encourage self-care and participation in decision making.

EVALUATION

At the time of discharge, Mr Sandford has maintained his weight and has started on oral liquids, including supplements and enriched liquids. His airway has remained clear and he is effectively coughing and deep breathing. He has used the magic slate to communicate throughout his hospital stay. He is regaining use of his tongue and can speak a few words. Although initially distressed, he is communicating an increased ability to cope with loss of part of his tongue. He and his wife say they understand his discharge instructions, including diet, activity, follow-up care and signs and symptoms to report.

CRITICAL THINKING IN THE NURSING PROCESS

1. What measures can you, as a nurse, implement to reduce the incidence of oral cancer?
2. Plan a health education program for young men who smoke.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Mr Sandford's wife calls you 2 weeks after discharge. She tells you that he refuses to try to talk and is relying on his magic slate to communicate. Reflect on how you will respond.

- Weigh daily. Assess oral intake for adequacy of protein, kilojoules and nutrients. *Daily weights and nutritional assessments provide information about the adequacy of diet.*
- Offer soft, bland foods with supplements as indicated. Provide small, frequent feedings, making mealtimes pleasant. *Soft, bland foods may be better tolerated following oral surgery. Large meals may be overwhelming; small, frequent meals promote food and nutrient intake.*
- Provide enteral feedings per gastrostomy tube as ordered. Elevate the head of the bed 30 to 45 degrees. *Enteral feedings maintain nutritional status in the person who is unable to consume foods orally. Elevating the head of the bed reduces the risk of regurgitation and aspiration of gastric contents.*
- Assess for gastric residual volume per facility protocol for the type of feeding (intermittent or continuous). See the 'Translation to practice' box below. Notify the doctor of volumes greater than 200 mL or 50% of previous feeding if feeding is intermittent. *Excess residual volume may increase the risk of aspiration.*
- Consider a nutritional consultation to assess diet and plan appropriate supplements. *A registered dietitian can calculate energy requirements and develop an individualised diet plan to meet nutritional requirements.*

Impaired verbal communication

Oral surgery can interfere with communication. Effective communication is vital to postoperative recovery and prevention of complications.

- Before surgery, establish and practise a communication plan such as using a sketch pad or flash cards. *Practising communication techniques reduces fear and anxiety while promoting communication.*
- Provide ample time for communication efforts and do not answer for the person. Be alert for non-verbal communications. Use yes/no questions and simple phrases. *Providing adequate time allows the person opportunity to express ideas and thoughts. Non-verbal communication provides cues regarding comfort or other needs. Simple yes/no questions are easily answered non-verbally.*
- If indicated, refer to or consult with a speech therapist. *A speech therapist can help promote or restore effective communication.*

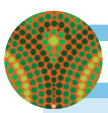
Disturbed body image

Radical surgery of the head or neck seriously affects body image. An altered speech pattern and any disfigurement affect the person's ability to feel attractive or effective in work or social roles. People may defer lifesaving surgery to postpone disfiguring interventions or therapies.

CONSIDERATION FOR PRACTICE

Provide buzzer by the bedside and respond promptly. Make all staff aware that the person cannot verbally respond. Non-verbal people rely on buzzers to summon help. Answering promptly reduces fear and anxiety and maintains safety.

- Assess coping style, self-perception and responses to altered appearance or function. *This information can be used to identify appropriate interventions and care.*
- Encourage verbalisation of feelings regarding perceived and actual changes. *Non-judgmental acceptance of feelings and fears helps establish trust.*



TRANSLATION TO PRACTICE Evidence-based practice for people with enteral feeding tubes

Practices and protocols for caring for enterally fed people and for assessing gastric residual volume have previously been inconsistent. Current recommendations include providing good oral hygiene, elevating the head of the bed 30–45 degrees, administering prokinetic agents such as metoclopramide where indicated, providing continuous feeds, administering the feeds directly to the small bowel via a gastrostomy tube and assessing gastric residual volumes regularly (Lord, 2012).

Gastric residual volumes have been used to determine aspiration risk; when the volume exceeds a predetermined level the tube feed is then withheld. Researchers have come to varying conclusions about the amount of residual volume that is considered safe and does not increase the risk of aspiration. Recent research advocates limits of 200–500 mL as saliva and gastric secretions need to be included in the volume as well as the tube feed (Lord, 2011). In addition, the withholding of enteral feed should be influenced by assessment of the person. Feelings of fullness, nausea, vomiting, abdominal discomfort or distension indicate when the enteral feed should be withheld (Lord, 2012).

IMPLICATIONS FOR NURSING

Gastric motility slows following surgery or trauma, in diabetes, sepsis or electrolyte imbalance, and with medications such as narcotic analgesics. Excessive gastric distension may increase the risk of aspiration of gastric contents; however, when enteral feeds are withheld unnecessarily, the nutritional status of the person is at risk. Impaired nutrition affects healing and recovery and may prolong the person's hospital stay. Therefore it is recommended that a clinical pathway or algorithm is developed which reflects current practice guidelines and is institution-specific to guide nurses with decision making regarding the withholding of feeds (Lord, 2011; 2012).

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Which factors might influence the accuracy of residual volume measurements? Which measures can be taken to obtain accurate measurements?
2. What would be the effect of withholding one bolus feeding of 240 mL of a standard enteral formula? If this is repeated daily for a week, what is the cumulative effect?
3. One reason frequently cited for avoiding checking residual volume is the risk of plugging the feeding tube. Identify measures to prevent this potential problem.

- Provide emotional support, encourage self-care and provide decision-making opportunities. *Self-care promotes self-acceptance and independence. Giving choices empowers the person to participate in care.*

Community-based care

Discharge planning for the person with oral cancer depends on the type of treatment planned and surgery performed. Depending on the person's age, condition and availability of support systems, referral to community healthcare agencies may be an essential component of care. Visits from home health nurses can assist in meeting healthcare needs.

Discuss the following topics with the person and their family members or care providers:

- diagnosis and prescribed care
- monitoring for new lesions or recurrences
- diet, nutrition and activity
- pain management
- airway management, care of incision and signs and symptoms to report.

DISORDERS OF THE OESOPHAGUS

The oesophagus plays an essential role in the ingestion of food and liquids. Disorders of the oesophagus can be inflammatory, mechanical or cancerous. Because of its location and neighbouring organs, the symptoms of oesophageal disorders may mimic those of a variety of other illnesses.

FAST FACTS

- GORD affects up to 20% of people in Western countries
- Up to 10% of people are likely to experience heartburn weekly (Selby, 2010).

THE PERSON WITH GASTRO-OESOPHAGEAL REFLUX DISEASE

Gastro-oesophageal reflux is the backward flowing of gastric contents into the oesophagus. When this occurs, the person experiences heartburn. Many people with gastro-oesophageal reflux have few symptoms, while others develop inflammatory oesophagitis as a result of exposure to gastric juices. **Gastro-oesophageal reflux disease (GORD)** is a common gastrointestinal disorder.

Pathophysiology

Normally, the lower oesophageal sphincter remains closed except during swallowing. Reflux (backflow) of gastric contents into the oesophagus is prevented by pressure differences between the stomach and the lower oesophagus. The diaphragm, the lower oesophageal sphincter and the location of the gastro-oesophageal junction below the diaphragm help maintain this pressure difference (see Figure 22.2).

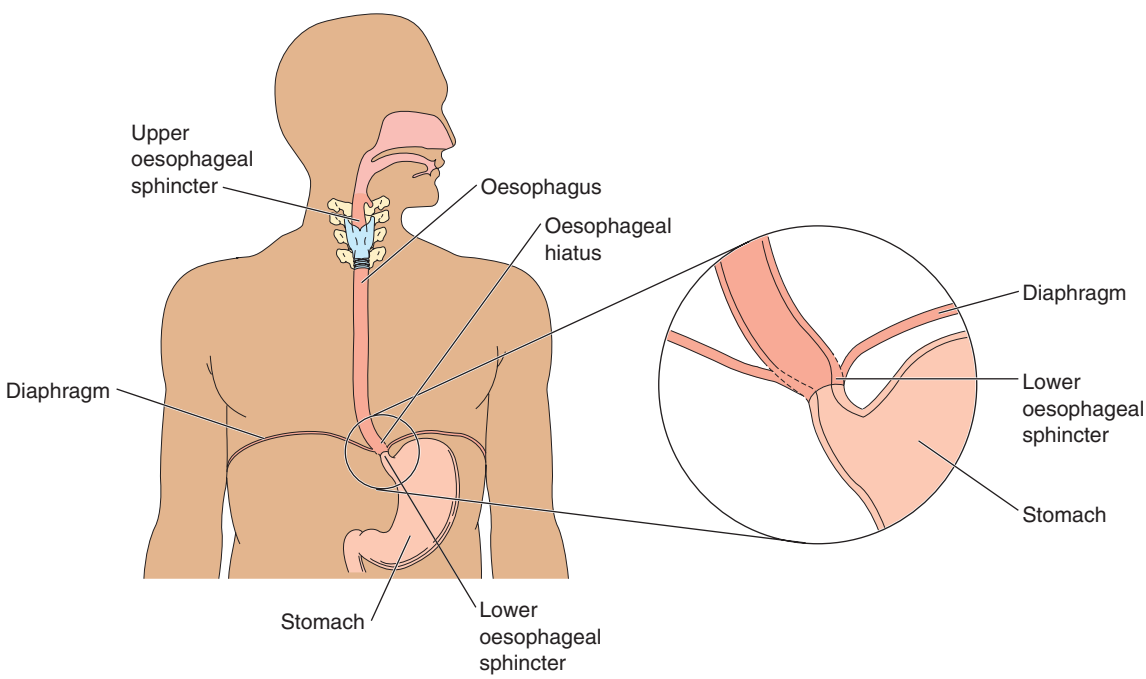
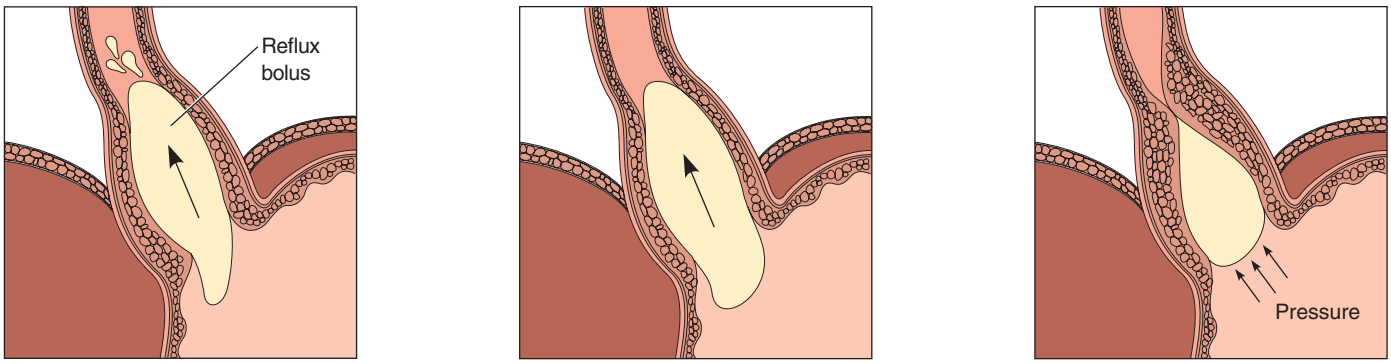


FIGURE 22.2 ■ The oesophagus. The inset shows a closer view of the lower oesophageal sphincter



Transient lower oesophageal sphincter relaxation

Incompetent lower oesophageal sphincter

Increased intragastric pressure

FIGURE 22.3 ■ Mechanisms of gastro-oesophageal reflux

Gastro-oesophageal reflux may result from transient relaxation of the lower oesophageal sphincter, an incompetent lower oesophageal sphincter and/or increased pressure within the stomach (see Figure 22.3). Factors contributing to gastro-oesophageal reflux include increased gastric volume (e.g. after meals), positioning that allows gastric contents to remain close to the gastro-oesophageal junction (e.g. bending over, lying down) and increased gastric pressure (e.g. obesity, pregnancy or wearing tight clothing). A hiatal hernia may contribute to GORD.

Gastric juices contain acid, pepsin and bile, which are corrosive substances. Oesophageal peristalsis and bicarbonate in salivary secretions normally clear and neutralise gastric juices in the oesophagus. During sleep, however, and in people with impaired oesophageal peristalsis or salivation, the oesophageal mucosa is damaged by gastric juices, causing an inflammatory response. With prolonged exposure, oesophagitis develops. Superficial ulcers develop and the mucosa becomes red, friable and may bleed. If untreated, scarring and oesophageal stricture may develop.

Manifestations

GORD causes burning, retrosternal pain which may radiate to the jaw or other areas of the chest. These symptoms can be confused with cardiac pain and therefore thorough assessment, the person's history and investigations are necessary. Regurgitation, or reflux, also occurs in GORD causing gastric acid to enter into the throat leaving a metallic, bitter taste in the mouth. The gastric acid may also irritate the airways causing the person to develop a chronic cough, or exacerbation of their asthma or chronic obstructive pulmonary disease (Selby, 2010).

Complications of GORD include oesophagitis, oesophageal ulcer, peptic stricture, haemorrhage, Barrett's oesophagus and oesophageal cancer. Both symptoms and complications of GORD lead to significant lifestyle disruptions including sleep disturbance, impaired ability to concentrate and inability to undertake physical or social activities (Morcom, 2008).

INTERPROFESSIONAL CARE

Often the diagnosis of GORD is made by the history of symptoms and predisposing factors. Interprofessional care focuses on lifestyle changes, diet modification and, for more severe cases, drug therapy. Surgery is reserved for people who develop serious complications.

Diagnosis

Diagnostic tests that may be ordered for people with manifestations of GORD include:

- *Barium swallow* to evaluate the oesophagus, stomach and upper small intestine.
- *Upper endoscopy* to permit direct visualisation of the oesophagus. Tissue may be obtained for biopsy to establish the diagnosis and rule out malignancy. See Chapter 20 for nursing care of the person undergoing an upper endoscopy.
- *24-hour ambulatory pH monitoring* may be performed to establish the diagnosis of GORD. For this test, a small tube with a pH electrode is inserted through the nose into the oesophagus. The electrode is attached to a small box worn on the belt that records the data. The data are later analysed by computer.
- *Oesophageal manometry* measures pressures of the oesophageal sphincters and oesophageal peristalsis.

Medications

Antacids such as Mylanta relieve mild or moderate symptoms by neutralising stomach acid. Gaviscon, which forms a floating barrier between the gastric contents and the oesophageal mucosa when the person is upright, may also be used.

Omeprazole (Losec), lansoprazole (Zoton), pantoprazole (Somac) and esomeprazole (Nexium) are proton-pump inhibitors (PPIs) that reduce gastric secretions. PPIs are the first-line pharmacological treatment as they promote healing of erosive oesophagitis and also relieve symptoms (Morcom, 2008; Selby, 2010). An 8-week course of treatment is initially prescribed, although some people may require 3 to 6 months of therapy.

MEDICATION ADMINISTRATION Drugs used to treat GORD, gastritis and peptic ulcer disease

PROTON-PUMP INHIBITORS

Esomeprazole (Nexium)
Lansoprazole (Zoton)
Omeprazole (Losec)
Pantoprazole (Somac)
Rabeprazole (Aciphex)

Proton-pump inhibitors are the drugs of choice for GORD. PPIs inhibit the hydrogen-potassium-ATP pump, reducing gastric acid secretion. Initially, the PPI may be given twice a day, with the dose reduced to once daily (at bedtime) after 8 weeks.

Nursing responsibilities

- Administer before breakfast and at bedtime if ordered twice a day; at bedtime if once a day.
- Do not crush tablets.
- Monitor liver function tests for possible abnormal values, including increased AST, ALT, alkaline phosphatase and bilirubin levels.

Health education for the person and family

- Take the drug as ordered for the full course of therapy, even if symptoms are relieved.
- Do not crush, break or chew tablets.
- Avoid cigarette smoking, alcohol, aspirin and NSAIDs while taking this drug because these substances may interfere with healing.
- Report black tarry stools, diarrhoea or abdominal pain to your primary care provider.

H_2-RECEPTOR BLOCKERS

Cimetidine (Tagamet) **Ranitidine (Zantac)**
Famotidine (Pepcidine) **Nizatidine (Nizac)**

H_2-receptor blockers reduce acidity of gastric juices by blocking the ability of histamine to stimulate acid secretion by the gastric parietal cells. As a result, both the volume and concentration of hydrochloric acid in gastric juice are reduced. H_2-receptor blockers are given orally or intravenously. Both prescription and over-the-counter preparations are available.

Nursing responsibilities

- To ensure absorption, do not give an antacid within 1 hour before or after giving an H_2-receptor blocker.
- When administered intravenously, do not mix with other drugs. Administer in 20 to 100 mL of solution over 15 to 30 minutes. Rapid intravenous injection as a bolus may cause arrhythmias and hypotension.
- Monitor for interaction with such drugs as oral anticoagulants, beta-blockers, benzodiazepines, tricyclic antidepressants and others. H_2-receptor blockers may inhibit the metabolism of other drugs, increasing the risk of toxicity.

Health education for the person and family

- Take the drug as directed, even if pain and gastric discomfort are relieved early in the course of therapy.
- Take at bedtime if once-a-day dosing is ordered. If spaced through the day, take before meals. Avoid taking antacids for 1 hour before and 1 hour after taking this drug.
- To promote healing, avoid cigarette smoking (which increases gastric acid secretion) and gastric mucosal irritants such as alcohol, aspirin and NSAIDs.
- Long-term use of these drugs can lead to gynaecomastia (breast enlargement) and impotence in men, and breast tenderness in women. Discontinuing the drug will reverse these effects.
- Report possible adverse effects such as diarrhoea, confusion, rash, fatigue, malaise or bruising to your care provider.

ANTI-ULCER AGENT

Sucralfate (Carafate)

Sucralfate reacts with gastric acid to form a thick paste that adheres to damaged gastric mucosal tissue. It protects gastric mucosa and promotes healing through this local action.

Nursing responsibilities

- Administer on an empty stomach, 1 hour before meals and at bedtime.
- Do not crush tablets.
- Separate administration time from antacids by at least 30 minutes.

Health education for the person and family

- Take as directed, even after symptoms have been relieved.
- Do not crush or chew tablets; shake suspension well.
- Increase your intake of fluids and dietary fibre to prevent constipation.

ANTACIDS

Gavisco Gelusil Gastrogel Mylanta

Antacids buffer or neutralise gastric acid, usually acting locally. Antacids are used in GORD, gastritis and peptic ulcer disease to relieve pain and prevent further damage to oesophageal and gastric mucosa.

Nursing responsibilities

- Antacids interfere with the absorption of many drugs given orally; separate administration times by at least 2 hours.
- Monitor for constipation or diarrhoea resulting from antacid therapy. Notify the physician should either develop; a different antacid may be ordered.
- Although most antacids have little systemic effect, electrolyte imbalances can develop. Monitor serum electrolytes, particularly sodium, calcium and magnesium levels.

Health education for the person and family

- Take your antacid frequently as prescribed, 1 to 3 hours after meals and at bedtime. To be effective, the antacid must be in your stomach.
- Avoid taking an antacid for approximately 2 hours before and 1 hour after taking another medication.
- Shake suspensions well prior to administration.
- Chew tablets thoroughly and follow with a glass of water.
- Report worsening symptoms, diarrhoea or constipation to your primary care provider.

(continued)

MEDICATION ADMINISTRATION Drugs used to treat GORD, gastritis and peptic ulcer disease (continued)

- Continue taking the antacid for the duration prescribed. Although pain and discomfort often are relieved soon after treatment begins, healing takes 6 to 8 weeks.

PROKINETIC AGENT

Metoclopramide (Maxalon)

By acting on the central nervous system, metoclopramide stimulates upper gastrointestinal motility and gastric emptying. As a result, nausea, vomiting and symptoms of GORD are reduced.

Nursing implications

- Do not administer this drug to children or to people with possible gastrointestinal obstruction or bleeding, or a history of seizure disorders, pheochromocytoma or Parkinson's disease.
- Monitor for extrapyramidal side effects (e.g. difficulty speaking or swallowing, loss of balance, gait disruptions, twitching or twisting movements, weakness of arms or legs) or manifestations of tardive dyskinesia (uncontrolled rhythmic facial movement, lip smacking, tongue rolling). Report immediately.
- Give oral doses 30 minutes before meals and at bedtime.
- May be given by direct intravenous push over 1 to 2 minutes.

Health education for the person and family

- Take this drug as directed. If you miss a dose, take as soon as you remember unless it is close to the time for the next dose.
- Do not drive or engage in other activities that require alertness if this drug makes you drowsy.
- Avoid using alcohol or other CNS depressants while you are taking this drug.
- Immediately contact your healthcare provider if you develop involuntary movements of your eyes, face or limbs.

Histamine$_2$-receptor (H_2-receptor) blockers reduce gastric acid production and are effective in treating GORD symptoms. When treating GORD, H_2-receptor blockers are usually given twice a day or more frequently for a prolonged period of time. Cimetidine, ranitidine, famotidine and nizatidine are all approved by the Therapeutic Goods Administration (TGA) for the treatment of GORD.

A prokinetic agent, such as metoclopramide (Maxalon), may be ordered to enhance oesophageal clearance and gastric emptying. Metoclopramide is used to treat people with regurgitation, symptoms of indigestion and night-time symptoms. However, it is not recommended for long-time use due unfavourable side effects and only modest efficacy for symptom relief and healing (Morcom, 2008). See the 'Medication administration' box above for the nursing implications of drugs used to treat GORD.

Nutrition and lifestyle management

GORD is a chronic condition. Dietary and lifestyle changes are important to reduce symptoms and long-term effects of the disorder. Contributing factors to GORD include obesity, smoking, alcohol and certain foods, and therefore lifestyle interventions are an essential adjunct to pharmacological therapy for GORD (Morcom, 2008). Dietary modifications such as the avoidance of acidic or fatty foods, avoiding large meals and waiting 2 hours after dinner before going to bed may be beneficial; however, there is limited clinical evidence to support this (Morcom, 2008). Smoking cessation, alcohol avoidance, stress reduction, weight loss and elevating the head of the bed are recommended for symptom relief (Morcom, 2008; Selby, 2010).

Surgery

Surgery may be used for people who do not respond to pharmacological and lifestyle management. Antireflux surgeries increase pressure in the lower oesophagus, inhibiting gastric content reflux. Laparoscopic fundoplication, a procedure in which the gastric fundus is wrapped around the distal oesophagus, is the treatment of choice for GORD. An open surgical procedure known as Nissen fundoplication may also be done (see Figure 22.4). Other laparoscopic procedures to tighten the lower oesophageal sphincter may include use of an endoscopic suturing system or burning spots on the muscle surrounding the sphincter to create scar tissue. Surgery or ablation therapy also is recommended to reduce the risk of oesophageal cancer in people with persistent cell changes in the distal oesophagus.

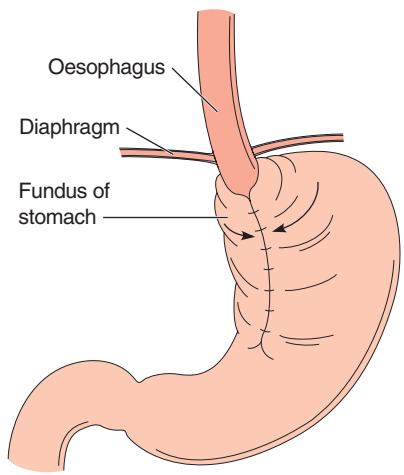


FIGURE 22.4 ■ Nissen fundoplication. The fundus of the stomach is wrapped around the lower oesophagus and the edges are sutured together

Nursing care

Assessment

Assessment data related to GORD include the following:

- *Health history:* manifestations such as frequent heartburn; intolerance of foods that are acidic, spicy or fatty; regurgitation of acidic gastric juice; increased symptoms when bending over, lying down or wearing tight clothing; difficulty swallowing.
- *Physical assessment:* epigastric tenderness, weight.

Nursing diagnoses and interventions

Relieving the discomfort associated with GORD is the priority of nursing care. Education focuses on preventing symptoms and long-term consequences of the disorder.

Pain related to reflux

The epigastric pain associated with GORD can be severe, interfering with rest and causing anxiety.

- Provide small, frequent meals. Restrict intake of fat, acidic foods, coffee and alcohol. *Limiting the size of meals reduces pressure in the stomach, reducing oesophageal reflux. Fatty, acidic foods, coffee and alcohol increase gastric acidity and interfere with gastric emptying, increasing the incidence of gastro-oesophageal reflux.*
- Instruct to stop smoking. Refer to a smoking cessation clinic or program as needed. *Cigarette smoking increases gastric acidity and interferes with healing of damaged mucosa.*
- Administer antacids, H_2-receptor blockers and PPIs as ordered. Instruct the person to continue therapy as prescribed, even after symptoms have been relieved. *These drugs neutralise or reduce gastric acid secretion, relieving symptoms and promoting healing.*
- Discuss the long-term nature of GORD and its management. *Lifestyle changes need to be continued after healing and symptom relief to manage the long-term effects of GORD.*

Community-based care

GORD is a lifelong condition best managed by the person. Teach the person and family about continuing management strategies which may be of benefit, including dietary changes, remaining upright after meals and avoiding eating for at least 3 hours before bedtime. Suggest elevating the head of the bed on 15 to 20 cm wooden blocks placed under the legs. Discuss the need for continued gastric acid reduction using antacids, H_2-receptor blockers or PPIs. All are effective to reduce the acidity of gastric juices. Antacids, the most cost-effective measure, require frequent doses to neutralise gastric acid. H_2-receptor blockers, also available over the counter, are a cost-effective management strategy that requires only twice-a-day dosing.

THE PERSON WITH A HIATAL HERNIA

A **hiatal hernia** occurs when part of the stomach protrudes through the oesophageal hiatus of the diaphragm into the thoracic cavity. Although hiatal hernia is thought to be a common problem, most affected individuals are asymptomatic. The incidence of hiatal hernia increases with age.

In a *sliding hiatal hernia*, the gastro-oesophageal junction and the fundus of the stomach slide upward through the oesophageal hiatus (see Figure 22.5A). Several factors may contribute to a sliding hiatal hernia, including weakened gastro-oesophageal diaphragmatic anchors, shortening of the oesophagus or increased intra-abdominal pressure. Small sliding hiatal hernias produce few symptoms.

In a *paraoesophageal hiatal hernia*, the junction between the oesophagus and stomach remains in its normal position below the diaphragm while a part of the stomach herniates through the oesophageal hiatus (see Figure 22.5B). A paraoesophageal hernia can become incarcerated (constricted) and strangulate, impairing blood flow to the herniated tissue. People with paraoesophageal hernia may develop gastritis or chronic or acute gastrointestinal bleeding. The manifestations of hiatal hernias are listed in the following box.

A barium swallow or an upper endoscopy may be done to diagnose hiatal hernia. Many people with hiatal hernia require no treatment. If symptoms are present, treatment measures such

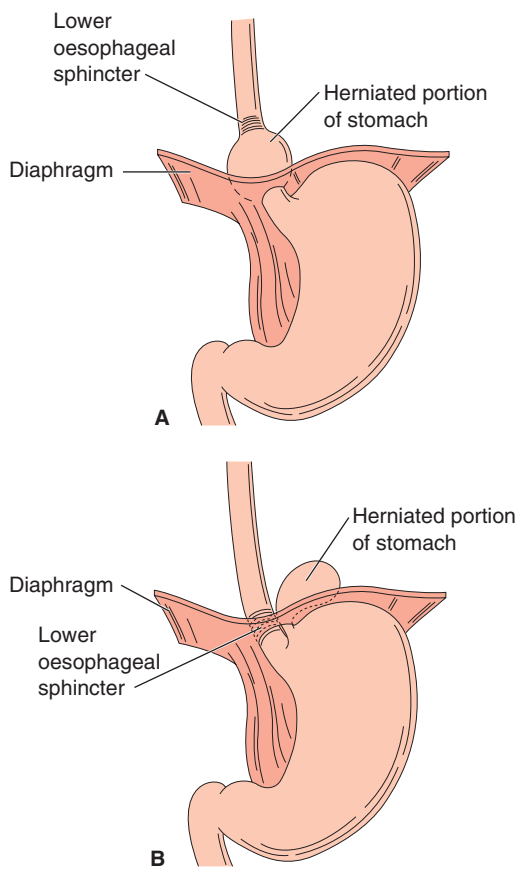


FIGURE 22.5 ■ Hiatal hernias. *A*, Sliding hiatal hernia. *B*, Paraoesophageal hiatal hernia

as those for people with GORD may be ordered. If medical management is ineffective or the hernia becomes incarcerated, surgery may be required. The most common surgical procedure is the Nissen fundoplication (see Figure 22.4). This surgery, which may be done laparoscopically, prevents the gastro-oesophageal junction from slipping into the thoracic cavity.

Nursing care for the person with a hiatal hernia is similar to that for the person with GORD. If surgery is performed, nursing care is similar to that for people undergoing gastric or thoracic surgery (see Chapter 3).

MANIFESTATIONS Hiatal hernia

- Reflux, heartburn
- Feeling of fullness
- Substernal chest pain
- Dysphagia
- Occult bleeding
- Belching, indigestion

THE PERSON WITH IMPAIRED OESOPHAGEAL MOTILITY

Disorders of oesophageal motility can cause **dysphagia** (difficult or painful swallowing) or chest pain. It is estimated that nearly 75% of people hospitalised with stroke experience dysphagia. Other neurological disorders such as Parkinson's disease, amyotrophic lateral sclerosis and Alzheimer's disease also can cause dysphagia.

Primary disorders of swallowing are less common. **Achalasia**, a disorder of unknown aetiology, is characterised by impaired peristalsis of the smooth muscle of the oesophagus and impaired relaxation of the lower oesophageal sphincter (LOS). The person experiences gradually increasing dysphagia with both solid foods and liquids. Fullness in the chest during meals, chest pain and night-time cough are additional manifestations. Other people may experience **diffuse oesophageal spasm** that causes non-peristaltic contraction of oesophageal smooth muscle. This disorder causes chest pain and/or dysphagia. The chest pain can be severe and usually occurs at rest.

Treatment of achalasia may include endoscopically guided injection of botulinum toxin into the lower oesophageal sphincter or balloon dilation of the LOS. Botulinum toxin injection lowers LOS pressure, but may need to be repeated every 6 to 9 months. Balloon dilation tears muscle fibres in the LOS, reducing its pressure (see Figure 22.6). A laparoscopic myotomy (incision into the circular muscle layer of the LOS) also reduces pressure and relieves symptoms.

THE PERSON WITH OESOPHAGEAL CANCER

Cancer of the oesophagus is a relatively uncommon malignancy in Australia. It does, however, have a high mortality rate, with a 5-year survival rate of only 15% (Edmondson & Schiech, 2008) primarily because symptoms often are not recognised until late in the course of the disease.

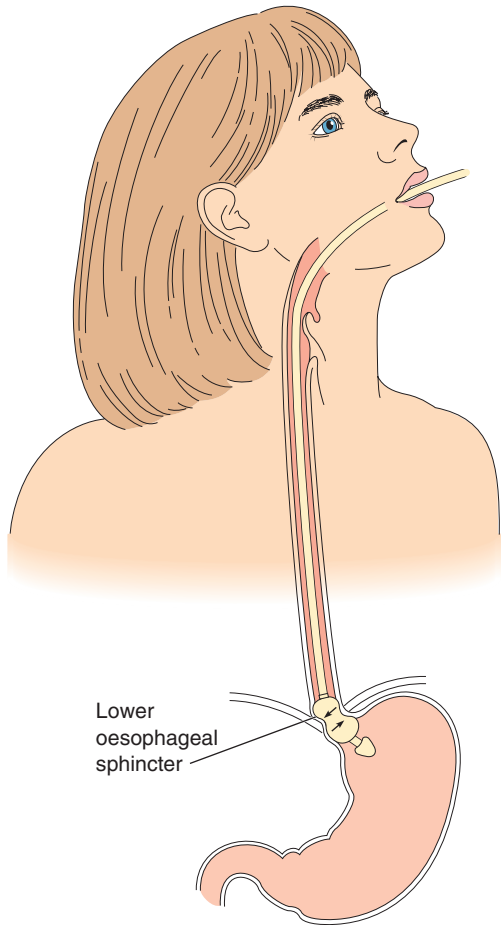


FIGURE 22.6 ■ Balloon dilation of the lower oesophageal sphincter

Pathophysiology

There are two types of oesophageal tumours: adenocarcinoma and squamous cell carcinoma. Cigarette smoking and chronic alcohol use are strong risk factors for squamous cell oesophageal tumours in Western countries. In Asian countries, chewing betel nuts and eating certain pickled vegetables are risk factors. There are investigations currently into the link between HPV and oesophageal cancer (Edmondson & Schiech, 2008).

Adenocarcinoma has been linked to obesity, GORD and Barrett's oesophagus. Combining any of these co-morbidities with tobacco use further increases the risk of oesophageal cancer developing (Edmondson & Schiech, 2008). Box 22.3 lists major identified risk factors for oesophageal cancer.

Only about 15% of oesophageal tumours develop in the upper portion of the oesophagus; about 35% develop in the mid portion. The lower third of the oesophagus is the most common site, accounting for about 50% of tumours. Adenocarcinomas tend to develop in dysplastic (abnormal) columnar epithelium in the distal oesophagus. They are commonly associated with Barrett's oesophagus, a possible complication of chronic GORD and achalasia.

The disease usually spreads to adjacent and supraclavicular lymph nodes, the liver, lungs and the pleura.

BOX 22.3 Risk factors for oesophageal cancer

- Excess alcohol consumption
- Cigarette smoking
- Ingested carcinogens such as nitrates and industrial chemicals
- Smoked opiates
- Physical mucosal damage (e.g. lye ingestion, radiation damage, chronic achalasia)
- Congenital disorders
- Chronic gastric reflux
- Obesity

Manifestations

Dysphagia is the most common symptom of oesophageal carcinoma, typically accompanied by weight loss (Pennathur et al., 2013). Other manifestations are listed in the following box.

Tracheoesophageal fistulas may develop as the disease progresses, leading to aspiration, pneumonia and shortness of breath. Paraneoplastic symptoms such as hypercalcaemia also may accompany advanced oesophageal cancer.

MANIFESTATIONS Oesophageal cancer

- Dysphagia
- Anaemia
- Unintentional weight loss
- GORD-like symptoms
- Regurgitation
- Anorexia
- Chest pain
- Persistent cough
- Hoarseness

INTERPROFESSIONAL CARE

Controlling dysphagia and maintaining nutritional status are essential goals of therapy for people with oesophageal cancer, regardless of the stage of the disease. Treatment may involve surgery, radiation therapy and/or chemotherapy.

Diagnosis

Diagnostic and staging procedures for oesophageal cancer may include endoscopy, bronchoscopy and scans to detect metastasis. The following diagnostic tests may be performed (see Chapter 20):

- *Barium swallow* to identify irregular mucosal patterns or narrowing of the lumen, which suggest oesophageal cancer.
- *Upper endoscopy* to allow direct visualisation of the tumour and obtain tissue for biopsy.
- *Chest x-ray, CT scans* or *MRI* to identify possible tumour metastases to other organs or tissues.
- *Full blood count (FBC)* may indicate anaemia due to chronic blood loss. *Serum albumin* levels may be low due to malnutrition and liver function tests (*ALT*, *alkaline phosphatase*, *AST* and *bilirubin*) are elevated if liver metastases are present.

Treatments

The treatment of oesophageal cancer depends on the stage of the disease, as well as factors such as the person's condition and preference.

People with early oesophageal cancer usually are treated with surgery alone. Surgery involves resection of the affected portion of the oesophagus (*oesophagectomy*) and possible anastomosis of the stomach to the remaining oesophagus. Mediastinal lymph nodes may be resected at the time of surgery. Oesophagectomy is not without risk; potential surgical complications include anastomosis leak, respiratory complications such as pneumonia or acute respiratory distress syndrome, gastric necrosis or bleeding, cardiac arrhythmias, and infection and sepsis. Postoperative recovery is usually in the intensive care unit (ICU) for 12 days before transfer to a surgical ward (Edmondson & Schiech, 2008; Viklund & Lagergren, 2007).

Other approaches to early oesophageal cancer include combined radiation and chemotherapy or radiation therapy or chemotherapy alone prior to surgical resection of the tumour. Although the prognosis of oesophageal cancer is poor, current evidence suggests significant benefit from preoperative chemotherapy or chemoradiotherapy prior to resection of local, operable cancer (Gebski et al., 2007). However, more research in this area is needed.

When the tumour is too advanced or if surgery is considered too risky, palliative measures such as brachytherapy, chemotherapy and oesophageal stenting may be considered (Edmondson & Schiech, 2008). Approximately 50% of people with oesophageal cancer are considered incurable at time of diagnosis, therefore palliative treatments focus on maintaining good quality of life through relief of dysphagia, providing nutritional support, administering pain relief and offering psychological support (Viklund & Lagergren, 2007).

Nursing care

Health promotion

Health promotion measures to reduce the risk and incidence of oesophageal cancer include educating people (especially young people) about the dangers of cigarette smoking and excess alcohol use. Refer to smoking cessation and alcohol treatment programs as indicated. Educate people with GORD about the relationship between chronic damage to the oesophagus due to reflux and oesophageal cancer and stress the importance of effective disease management.

Assessment

Early diagnosis and treatment of oesophageal cancer can make a difference in the person's prognosis. Collect the following assessment data related to oesophageal cancer:

- *Health history:* current symptoms such as chest pain, dysphagia, odynophagia (pain with swallowing), coughing or hoarseness; duration of symptoms; recent weight loss; smoking history; current and past patterns of alcohol consumption.
- *Physical examination:* weight; general health status; skin colour; supraclavicular and cervical lymph nodes for lymphadenopathy.

Nursing diagnoses and interventions

Disruption of the integrity and function of the oesophagus and the discomfort associated with swallowing in people with oesophageal cancer affect the person's ability to maintain adequate nutritional status and, potentially, a patent airway.

Risk of imbalanced nutrition (less than body requirements) related to dysphagia

The person diagnosed with oesophageal cancer may already suffer from some degree of malnutrition because of difficulty and pain with swallowing. Enteral nutrition via nasogastric feeding tube or gastrostomy tube or parenteral nutrition maintains nutritional status after surgery or if the tumour is inoperable and obstruction occurs. See Chapter 21 for nursing interventions related to enteral and parenteral feedings.

Risk of ineffective airway clearance due to surgery

After surgery for oesophageal cancer, the person is at high risk of aspiration and difficulty maintaining a patent airway due to disruption of the oesophagus and incision into the thoracic cavity.

- Assess mental and respiratory status (including rate, depth, breath sounds and oxygen saturation levels) at least every hour during the initial postoperative period. *Altered mental status increases the risk of aspiration. An increased respiratory rate, dyspnoea, diminished breath sounds or decreased oxygen saturation levels may indicate impaired airway clearance or possible aspiration pneumonia.*
- Provide aggressive pulmonary hygiene measures, including endotracheal suctioning and chest physiotherapy as indicated or ordered. Following extubation, encourage frequent coughing, deep breathing and use of the incentive spirometer. *Respiratory complications are a frequent complication of oesophagectomy. Aggressive nursing care helps mobilise secretions and prevent atelectasis and possible pneumonia.*
- If present, monitor chest tube function and drainage. Promptly report drainage that is bright red and excessive in amount (> 70 mL/h) or purulent. Maintain patency of chest tubes per unit protocol or physician's order. If a thoracic incision has been used, chest tubes are placed to promote lung reinflation. *Proper chest tube function is necessary to prevent pneumothorax and impaired lung inflation.*
- Monitor cardiopulmonary status and haemodynamic pressures. Administer intravenous fluids and fluid boluses as ordered. Fluid volume imbalances that compromise cardiopulmonary status may develop following oesophagectomy. *Maintaining adequate fluid intake and preventing fluid overload are important postoperatively. The person also is at risk of acute respiratory distress syndrome, a critical complication that can further compromise ventilation, gas exchange and circulation.*
- Do not move or manipulate the nasogastric tube. Maintain low gastric suction as ordered. *Manipulating or moving the nasogastric tube may disrupt suture lines, resulting in a leak into the mediastinum.*
- Verify enteral tube feeding placement by x-ray (see Chapter 21). Stop enteral feedings if feelings of fullness or nausea occur. Suction gastrointestinal contents as needed, positioning the person on the side. *Overdistension of the stomach or delayed gastric emptying may result in regurgitation of stomach contents. Nausea or a feeling of fullness may indicate stomach overdistension. Suctioning and positioning limit the risk of aspiration.*

Anticipatory grieving

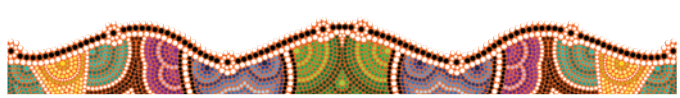
Upon a diagnosis of cancer, the person and family may experience a grief reaction. The pessimistic prognosis associated with oesophageal cancer and the disruptions in relationships may result in an intense sense of loss. Chapter 4 discusses care of the person experiencing grief and loss.

Community-based care

Most care for people with oesophageal cancer is provided in community-based and home settings. Include the following topics in person and family teaching for home care:

- planned treatment options, including the risks, benefits and potential adverse effects of each
- wound and follow-up care following surgery
- prevention and manifestations of complications such as wound or chest infection, anastomosis leak, deep vein thrombosis
- how to prepare, implement and care for tube feedings or home parenteral nutrition.

Based on the person's needs and prognosis, referral to a home health agency and/or hospice may be appropriate.



DISORDERS OF THE STOMACH AND DUODENUM

The stomach and upper small intestine (duodenum and jejunum) are responsible for the majority of food digestion. The major disorders that affect digestion are nausea and vomiting, gastritis, peptic ulcer disease and cancer of the stomach. Nursing roles in managing these disorders include both acute care for the hospitalised person and teaching to give the person the skills and knowledge to manage these conditions at home.

OVERVIEW OF NORMAL PHYSIOLOGY

Normally, the stomach is protected from the digestive substances it secretes—namely, hydrochloric acid and pepsin—by the **gastric mucosal barrier**. The gastric mucosal barrier includes:

- An impermeable hydrophobic lipid layer that covers the gastric epithelial cells. This lipid layer prevents diffusion of water-soluble molecules, but substances such as aspirin and alcohol can diffuse through it.
- Bicarbonate ions secreted in response to hydrochloric acid secretion by the parietal cells of the stomach. When bicarbonate (HCO_3^-) secretion is equal to hydrogen ion (H^+) secretion, the gastric mucosa remains intact. Prostaglandins, chemical messengers involved in the inflammatory response, support bicarbonate production and blood flow to the gastric mucosa.
- Mucous gel that protects the surface of the stomach lining from the damaging effects of pepsin and traps bicarbonate to neutralise hydrochloric acid. This gel also acts as a lubricant, preventing mechanical damage to the stomach lining from its contents.

When an acute or chronic irritant disrupts the mucosal barrier, or when disease alters the processes that maintain the barrier, the gastric mucosa becomes irritated and inflamed. Lipid-soluble substances such as aspirin and alcohol penetrate the gastric mucosal barrier, leading to irritation and possible inflammation. Bile acids also break down the lipids in the mucosal barrier, increasing the potential for irritation (Porth & Matfin, 2009). In addition, aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs) inhibit prostaglandins. Aspirin and NSAIDs also alter the nature of gastric mucus, affecting its protective function.

THE PERSON WITH NAUSEA AND VOMITING

Nausea and vomiting are common gastrointestinal symptoms. **Nausea** is a subjective, unpleasant sensation of sickness or queasiness. It may or may not be accompanied by (and possibly relieved by) vomiting. **Vomiting** is the forceful expulsion of the contents of the upper gastrointestinal tract resulting from contraction of muscles in the gut and abdominal wall. Nausea and vomiting without abdominal pain are commonly associated with food poisoning, infectious gastroenteritis (discussed in Chapter 23), gallbladder disease or ingestion of toxins (such as drugs or alcohol). When associated with severe abdominal pain, they may indicate a serious disorder such as peritonitis, acute gastrointestinal obstruction or pancreatitis.

Pathophysiology

Nausea occurs when the vomiting centre in the medulla of the brain is stimulated. Distension of the duodenum is a common stimulus for nausea. The vomiting centre can be stimulated by input from several different sources:

- the gastrointestinal tract, produced by distension, irritation or infection
- the vestibular system of the ear
- higher central nervous system centres in response to certain sights, smells or emotional experiences
- chemoreceptors outside the blood–brain barrier that are stimulated by drugs, chemotherapeutic agents, toxins, systemic disorders and pregnancy
- disorders such as acute myocardial infarction and heart failure commonly produce nausea and vomiting, possibly due to direct stimulation of the vomiting centre by hypoxia
- increased intracranial pressure (e.g. due to intracranial bleeding or a tumour) produces vomiting that may or may not be accompanied by nausea.

Anorexia—loss of appetite—commonly precedes nausea, just as nausea frequently precedes vomiting. Vomiting, a response that requires coordinated movements of the thorax and abdominal wall, the gut, the pharynx and the muscles of the mouth and face, is coordinated by the brainstem. *Emesis* (or *vomitus*) is produced when inspiratory muscles of the thorax (including the diaphragm) and abdomen contract, increasing intrathoracic and intra-abdominal pressures. The gastro-oesophageal sphincter relaxes and the larynx moves upward to facilitate oral expulsion of gastric contents.

In addition to the subjective sensation of queasiness, nausea frequently is accompanied by autonomic nervous system manifestations such as pallor, sweating, tachycardia and increased salivation. Vomiting, which stimulates the vagus nerve and parasympathetic nervous system, may be accompanied by dizziness, light headedness, hypotension and bradycardia.

Potential complications of vomiting include dehydration, hypokalaemia, metabolic alkalosis (from loss of hydrochloric acid from the stomach), aspiration with resulting pneumonia and rupture or tears of the oesophagus (known as a Mallory–Weiss tear).

INTERPROFESSIONAL CARE

In most cases, nausea and vomiting are self-limited and require no treatment. If vomiting is severe or accompanied by other symptoms, acute care may be required to determine the underlying problem and prevent or treat complications such as dehydration.

Diagnostic tests may include serum electrolytes; pregnancy testing if indicated; liver, pancreatic and renal function studies; and imaging studies (x-ray of the abdomen, abdominal CT scan) to detect gastrointestinal obstruction. An upper endoscopy may be performed. (See Chapter 20 for nursing care of the person undergoing upper endoscopy.) CT scan or MRI of the head may be ordered if an intracranial problem is suspected as the cause. Specialised testing, such as gastrointestinal motility studies, may be indicated when other diagnostic studies are negative for an anatomical cause of nausea and vomiting.

Food is initially withheld, although clear liquids in small quantities are encouraged to prevent dehydration. Dry foods such as crackers may reduce nausea and promote comfort.

Medications

Unless vomiting is associated with pregnancy, anti-emetic medications may be prescribed to prevent or control nausea and vomiting. These drugs fall into several different classes and often are more effective when given in combination.

- Serotonin receptor antagonists are the most effective drugs available for people experiencing nausea and vomiting due to chemotherapy. They are effective when given only once or twice a day, an additional advantage. Drugs within this class include ondansetron (Zofran), granisetron (Kytril) and tropisetron (Navoban).
- Dopamine antagonists include the phenothiazines (e.g. prochlorperazine (Stemetil) and promethazine (Phenergan)), butyrophenones (haloperidol and droperidol) and other drugs such as metoclopramide (Maxalon). These drugs, while effective, can produce extrapyramidal symptoms, sedation and hypotension.
- Antihistamines such as betahistine (Serc) are primarily used to treat nausea and vomiting arising from vestibular centre stimuli (e.g. motion sickness).
- While corticosteroids are not approved as a class for treating nausea and vomiting, two drugs in this class, methylprednisolone (Solu-Medrol) and dexamethasone (Decadron), may be used in combination to treat vomiting associated with cancer treatment.
- Lorazepam (Ativan) is a benzodiazepine drug approved for use as an anti-emetic. It produces a degree of sedation, but can suppress anticipatory vomiting (e.g. before chemotherapy). It also helps control extrapyramidal symptoms associated with the phenothiazine anti-emetics (dopamine antagonists).

Nursing responsibilities and person education for anti-emetic drugs are outlined in the accompanying ‘Medication administration’ box.

Complementary and alternative medicine

Mind–body interventions such as biofeedback, guided imagery, music therapy and hypnosis may be effective for some people with nausea. Biofeedback uses machinery to translate physiological processes into audible or visible signals to teach the person to exert conscious control over those processes. In guided imagery, the person uses imagination to invoke specific images to modify physiological responses. Music therapy involves the person in creating or listening to music to affect physiological and psychological responses. In hypnosis, an altered mind state is induced to make the person receptive to suggestions.

Ginger, an aromatic root frequently used in cooking and also available in capsule form, may also be helpful in relieving nausea and vomiting, particularly when due to pregnancy and chemotherapy (Alparslan et al., 2012; Ozgoli, Goli & Simbar, 2009). Ginger can inhibit platelet aggregation and may increase the risk of bleeding in people taking antiplatelet or anticoagulant drugs (Lehne, 2012).

Nursing care

Assessment of the person is vital to help determine the cause of nausea and vomiting and to rule out underlying systemic disease or acute conditions that require immediate care (e.g. bowel obstruction). When the cause is known or no other acute symptoms are present, nursing interventions can promote comfort and prevent complications.

Nausea

An unpleasant sensation in the epigastric or abdominal region, nausea is a subjective sensation best described by the person.

- Monitor subjective complaints of nausea, vital signs, skin turgor and condition, and weight. Maintain accurate intake and output records. Monitor amount, colour and specific gravity of urine. *Nausea can cause aversion to food and fluids, leading to dehydration even when it is not accompanied by vomiting.*
- Administer anti-emetic medication as ordered, prior to meals and before treatments or procedures known to stimulate nausea. *Preventing nausea is particularly important for people receiving chemotherapy, to avoid the association between the treatment and nausea.*
- Instruct to deep breathe to voluntarily suppress the vomiting reflex. *Controlling vomiting helps prevent dehydration and other complications associated with prolonged or severe vomiting.*
- Instruct to consume small quantities of clear fluids and dry foods at separate times. *Separating the intake of dry foods and fluids helps reduce the nausea stimulus.*

MEDICATION ADMINISTRATION Drugs used to prevent and treat nausea and vomiting

SEROTONIN RECEPTOR ANTAGONISTS

Ondansetron (Zofran)
Granisetron (Kytril)
Tropisetron (Navoban)

The serotonin receptor antagonists suppress nausea and vomiting by blocking the effect of serotonin on vagal afferent nerves that stimulate the vomiting centre. Their primary uses are to prevent vomiting associated with chemotherapy, radiation therapy and surgery.

Nursing responsibilities

- Administer 30 to 60 minutes prior to chemotherapy or surgery as directed.
- May be given orally, sublingually (Ondansetron wafers) or intravenously (push or infusion; follow directions specific to the drug used).
- Monitor liver function and clotting studies; report abnormal levels to the physician.

Health education for the person and family

- Take this drug exactly as directed.
- This drug may be taken without regard to food intake.
- Headache is a common side effect of these drugs. Use paracetamol or another mild analgesic as directed by your physician.

DOPAMINE ANTAGONISTS

Prochlorperazine (Stemetil)
Promethazine (Phenergan)
Haloperidol (Haldol)
Droperidol (Dropletan)
Metoclopramide (Maxalon)

These drugs act by blocking dopamine receptors in the chemoreceptor trigger zone (CTZ). Their primary uses are to suppress nausea and vomiting associated with surgery, cancer chemotherapy and toxins. The major adverse effects associated with these drugs are sedation, hypotension and extrapyramidal reactions. Older adults are more sensitive to the effects of these drugs; a lower dose often is indicated.

Nursing responsibilities

- Administer orally or parenterally as ordered before surgery, or before meals and procedures known to produce nausea and vomiting.
- These drugs may interact with a number of other medications, often increasing their sedative and hypotensive effects.
- Administer with caution to older adults, closely monitoring for adverse effects such as confusion, agitation, changes in vital signs.
- Monitor for evidence of extrapyramidal symptoms, including tremor, restlessness, hyperactivity, anxiety, impaired coordination; notify doctor if symptoms develop.

Health education for the person and family

- Use the drug as ordered; do not increase your dose without consulting your primary care provider.
- These drugs may cause drowsiness. Avoid using other central nervous system (CNS) depressants such as alcohol while taking these drugs.
- Change positions from lying to sitting and sitting to standing slowly because these drugs can cause light headedness or dizziness.
- Promptly report changes in coordination, tremors, difficulty speaking or swallowing, or weakness to your physician.

ANTIHISTAMINES

Betahistane (Serc)

Antihistamines are primarily used to treat nausea and vomiting associated with motion sickness. They act by blocking histamine and acetylcholine (muscarinic) receptors in the neural pathway from the inner ear to the vomiting centre in the brainstem.

Nursing responsibilities

- Do not administer these drugs to people for whom anticholinergic drugs are contraindicated: people with narrow-angle glaucoma, urinary retention, bowel obstruction.
- May be administered orally, parenterally or rectally, depending on the preparation and the person's ability to tolerate oral preparations.
- Use with caution in people who are taking other CNS depressants or antihistamine preparations, tricyclic antidepressants or monoamine oxidase inhibitors.

Health education for the person and family

- These drugs frequently cause drowsiness. Use caution when operating machinery or performing tasks requiring mental alertness.
- Avoid using alcohol or other substances that cause drowsiness or sedation while taking these drugs.
- The medication may cause dry mouth. Sips of water, ice chips, boiled lollies and sugarless gum can be used for comfort.
- Use sunscreen and protective clothing to protect from sunburn while using these drugs.

THE PERSON WITH GASTROINTESTINAL BLEEDING

Because of its constant exposure to the environment, the gastrointestinal tract can be subjected to trauma and exposed to toxins, infection with pathogens such as *Helicobacter pylori* (*H. pylori*), inflammatory processes and insults such as ischaemia due to systemic diseases. While the mucosal lining of the GI tract is remarkably able to withstand these insults and heal rapidly, its rich supply of blood can result in significant bleeding when a vessel is eroded or abnormally distended (*varices*). Gastrointestinal haemorrhage is a relatively common cause of admission and complication of critical illnesses. It is a medical emergency requiring aggressive medical and nursing care.

Although bleeding and haemorrhage can occur anywhere in the GI tract, the upper portion of the tract is more commonly affected. The three primary disorders leading to upper

gastrointestinal (UGI) haemorrhage are erosive gastritis, peptic ulcer disease and oesophageal varices. Erosive gastritis and peptic ulcer disease are discussed in the following sections of this chapter; oesophageal varices, usually seen as a complication of cirrhosis of the liver, are discussed in Chapter 24.

FAST FACTS

- UGI bleeds are associated with increasing non-steroidal anti-inflammatory drug (NSAID) use.
- Approximately 64% are associated with *H. pylori* infection in people with peptic ulcer bleeding.
- UGI bleeding is twice as common in men as in women.
- Prevalence increases with age (Wilkins et al., 2012).

Pathophysiology

Blood in the GI tract has several effects. It is irritating to the stomach and typically leads to nausea and vomiting (**haematemesis**, vomiting blood). If the blood has been present in the stomach for a period of time and is partially digested, it may have a 'coffee-grounds' appearance, rather than presenting as bright red blood. The accumulation of blood in the GI tract stimulates peristalsis, leading to hyperactive bowel sounds and diarrhoea. Stools may be black and tarry (melaena) or frankly bloody (**haematochezia**); stool containing partially digested blood has a characteristic odour. With significant upper GI bleeding, digestion of blood proteins increases blood urea nitrogen (BUN) levels.

Physiological responses to an upper GI bleed depend on the rapidity and magnitude of the blood loss. GI bleeding resulting from erosion of a small vessel typically is slow and may not be identified until the person presents with manifestations of blood loss anaemia due to depletion of iron stores. (See Chapter 32 for further discussion of blood loss anaemia.) Although no visible blood may be present in the stool, **occult** (or hidden) **bleeding** may be detected by chemical means.

GI haemorrhage, with loss of a significant amount of blood within a few hours, rapidly depletes blood volume, producing manifestations of decreased cardiac output: tachycardia, hypotension, pallor and decreased urine output. Peripheral blood vessels constrict to maintain perfusion of vital organs. Unless the blood volume is restored, hypovolaemic shock progresses, leading to acidosis, renal failure, bowel infarction, acute coronary syndrome, coma and death. See Chapter 10 for more information about shock and its management.

INTERPROFESSIONAL CARE

The acuity of the bleed and the person's condition dictate the timing and extent of diagnostic testing and interventions. A person with a massive GI haemorrhage is admitted to the critical care unit and aggressively treated to stem bleeding, restore blood volume and stabilise the cardiovascular system. Identifying the cause of the bleeding is postponed in many cases until the person's condition has been stabilised.

When the bleeding is slow or chronic, diagnostic testing and treatment may be managed in a community-based setting.

Diagnosis

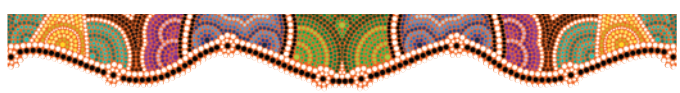
Diagnostic testing focuses on determining the extent and effects of the bleed, as well as its cause.

- A *full blood count (FBC) with haemoglobin and haematocrit* is obtained. In an acute bleed, the FBC, haemoglobin and haematocrit may not initially indicate the extent of blood loss because plasma is lost along with blood cells.
- *Blood type and crossmatch* are performed to prepare for transfusion as necessary.
- *Serum electrolytes, osmolality* and *BUN* are obtained to determine the effects of the blood loss and protein digestion on blood chemistries.
- *Liver function tests* and a *coagulation profile* may be obtained to help determine the cause of the bleeding.
- An *upper endoscopy* is performed as soon as possible to identify and, if possible, treat the source of bleeding. See Chapter 20 for nursing care of the person undergoing upper endoscopy.

Treatments

In acute GI haemorrhage, initial treatment focuses on stemming the bleeding, restoring cardiovascular stability and maintaining the person's airway. Intravenous fluids such as normal saline are administered through a large-bore intravenous catheter. Fresh whole blood, which contains clotting factors, is administered to restore blood volume and components in an acute haemorrhage. In less acute situations, packed red cells may be administered to restore the oxygen-carrying capacity of the blood. Intubation may be required to protect against aspiration of blood into the airways (Wilkins et al., 2012).

Haemostasis is achieved using upper endoscopy whenever possible. A sclerosing agent may be injected into the bleeding vessel or the vessel may be sealed using a heated probe, diathermy or laser. Rarely, emergency surgery is required to stop haemorrhage.



Nursing care

Health promotion

Preventing gastrointestinal bleeding is the most important step in reducing the mortality and morbidity associated with an acute GI haemorrhage. Identifying people at risk and instituting regular gastric pH monitoring and maintenance of drug therapy to reduce gastric acidity are important preventive measures. All critically ill people should be considered to be at risk of stress-related erosive gastritis.

Assessment

Assessment of the person experiencing an acute GI haemorrhage is very focused on the immediate crisis. The ability to obtain subjective information may be limited; however, it is important to identify possible contributing factors such as use of aspirin, NSAIDs, other platelet inhibitors or anticoagulant

medications, and the presence of any acute or chronic conditions that may contribute to bleeding (e.g. hypertension, a clotting disorder, peptic ulcer disease, excessive alcohol use, chronic hepatitis or cirrhosis of the liver). If possible, identify all current medications and their purpose, as well as any allergies to medications or other substances. If possible obtain any history of abdominal pain, black tarry stools, vomiting, dysphagia, rectal bleeding or chest pain (Wilkins et al., 2012).

Physical examination focuses on the effect of the bleeding on cardiovascular status. Obtain vital signs and orthostatic vital signs (an early sign of hypovolaemia). Place the acutely ill person on a cardiac monitor and obtain a rhythm strip. Obtain oxygen saturation level. Assess peripheral pulse strength, as well as colour, temperature and capillary refill of extremities. Evaluate mental status, including level of consciousness and orientation. An indwelling catheter may be inserted to evaluate urine output.

Nursing diagnoses and interventions

Nursing care priorities for the person with an acute GI bleed focus on restoring and maintaining an effective cardiac output and tissue perfusion, and on stopping the haemorrhage and preventing further bleeding.

Risk of decreased cardiac output related to haemorrhage

Significant amounts of blood may be lost in a very short time with an acute GI haemorrhage. Because some of the blood enters the bowel, it may be difficult to accurately estimate the amount of blood lost by measuring emesis, gastric suction return and blood expelled as faeces. As blood volume drops, venous return decreases. The heart rate increases to maintain the cardiac output, and peripheral blood vessels constrict to improve venous return and cardiac output.

- Frequently assess and document vital signs, including blood pressure, pulse rate and cardiac rhythm, respiratory rate and oxygen saturation levels. Obtain haemodynamic pressure measurements as ordered, reporting trends and changes. *The vital signs, oxygen saturation levels and haemodynamic pressure values provide indicators of the effectiveness of peripheral tissue perfusion, oxygenation and fluid replacement.*
- Monitor for and report changes in skin colour, temperature and moisture, or slow capillary refill. *Peripheral vasoconstriction and activation of the sympathetic nervous system typically cause pale, cool and moist or diaphoretic skin. Development of cyanosis or mottling indicates a further decrease in tissue perfusion and oxygenation.*
- Insert an indwelling urinary catheter and measure urine output hourly. Report an output of less than 30 mL for two consecutive hours. *A fall in urine output may indicate further reduction in cardiac output. As cardiac output falls, the kidneys become ischaemic and acute renal failure may develop.*
- Unless contraindicated, insert a nasogastric tube to assess for presence of blood or coffee ground material. *Acute blood in the stomach implies UGI bleeding is likely and predicts that the bleeding is caused by a high-risk lesion* (Wilkins et al., 2012).
- Maintain two peripheral intravenous lines with large-bore catheters or a central venous catheter for fluid and blood administration as ordered. Frequently monitor vital signs, respiratory status and haemodynamic pressure measurements, reporting changes in status. *Rapid administration of isotonic intravenous fluids, blood and blood products can lead to fluid overload and potential heart failure.*

Risk of impaired tissue integrity related to nasogastric tube insertion

- Maintain drainage and patency of nasogastric tube. Connect to low suction if ordered. *Blood is irritating to the GI tract, precipitating vomiting and stimulating peristalsis, leading to diarrhoea. In addition, digested blood can increase BUN levels, potentially leading to confusion and altered mental status.*
- Irrigate the nasogastric tube with room-temperature saline or tap water as ordered. Calculate intake and output, subtracting the amount of irrigant from gastric output. *Irrigation of the nasogastric tube helps remove irritating blood from the gut and produces a degree of vasoconstriction in stomach mucosa, slowing bleeding.*
- Prepare for upper endoscopy or surgery as planned. *Endoscopy or emergency surgery may be performed to repair the bleeding site or sclerose bleeding vessels.*
- Following an acute bleed and in people at risk of GI bleeding, monitor gastric pH as ordered and check vomitus and faeces for the presence of occult blood. Maintain infusions of drugs to reduce gastric acidity as ordered. *The person remains at risk of GI bleeding. Monitoring for occult blood helps identify slow bleeding or recurrent haemorrhage. Reducing the acidity of gastric secretions reduces irritation of the gastric mucosa, reducing the risk of bleeding.*

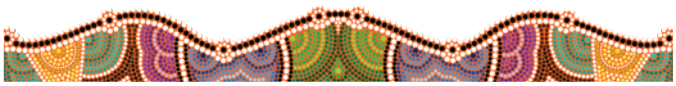
Community-based care

Following an acute GI haemorrhage, continuing care focuses on resolving the underlying disease process if possible and preventing future episodes of GI bleeding. If a bleeding gastric ulcer was identified, testing for *H. pylori* infection will be done and a treatment regimen prescribed to eradicate the infection. (See the section on peptic ulcer disease later in this chapter.) The person who experienced an episode of erosive stress gastritis will often be discharged with instructions to continue taking a gastric-acid-reducing medication and avoid known gastric irritants such as aspirin, NSAIDs and alcohol. The person with oesophageal varices due to cirrhosis or chronic hepatitis needs additional instructions (see Chapter 24).

People with minor or slow GI bleeding often are managed in the community. Provide teaching about the cause of the bleeding and measures to prevent future episodes. Provide verbal and written instructions for prescribed medications such as acid reducers and oral iron supplements. Discuss appropriate nutrition; while a special diet to ‘soothe the stomach’ rarely is

indicated, foods rich in iron may be recommended to treat the resulting anaemia.

Discuss indicators of GI bleeding to be reported to the doctor. If the source of bleeding has not been identified, provide instructions about prescribed follow-up diagnostic testing.



THE PERSON WITH GASTRITIS

Gastritis, inflammation of the stomach lining, results from irritation of the gastric mucosa. Gastritis is common and may be caused by a variety of factors. The most common form of gastritis, **acute gastritis**, is generally a benign, self-limiting disorder associated with the ingestion of gastric irritants such as aspirin, NSAIDs, alcohol, caffeine or foods contaminated with certain bacteria. Manifestations of acute gastritis may range from asymptomatic to mild heartburn to severe gastric distress, vomiting and bleeding with haematemesis (vomiting blood).

Chronic gastritis is a separate group of disorders characterised by progressive and irreversible changes in the gastric mucosa (Porth & Matfin, 2009). Chronic gastritis is more common in older adults, chronic alcoholics and cigarette smokers. When symptoms of chronic gastritis occur, they are often vague, ranging from a feeling of heaviness in the epigastric region after meals to gnawing, burning, ulcer-like epigastric pain unrelieved by antacids.

Pathophysiology

Acute gastritis

Acute gastritis is characterised by disruption of the mucosal barrier by a local irritant. This disruption allows hydrochloric acid and pepsin to come into contact with the gastric tissue, resulting in irritation, inflammation and superficial erosions. The gastric mucosa rapidly regenerates, generally making acute gastritis a self-limiting disorder, with resolution and healing occurring within several days.

The ingestion of aspirin or other NSAIDs, corticosteroids, alcohol and caffeine is commonly associated with the development of acute gastritis. Accidental or purposeful ingestion of a corrosive alkali (such as ammonia, disinfectant and other cleaning agents) or acid leads to severe inflammation and possible necrosis of the stomach. Gastric perforation, haemorrhage and peritonitis are possible results. Iatrogenic causes of acute gastritis include radiation therapy and administration of certain chemotherapeutic agents.

Erosive gastritis

A severe form of acute gastritis, **erosive** or **stress-induced gastritis** occurs as a complication of other life-threatening conditions such as shock, severe trauma, major surgery, sepsis, burns or head injury. When these erosions follow a major burn, they are called Curling's ulcers, after Thomas Curling, a British physician who first described them in 1842. When stress ulcers occur following head injury or CNS surgery, they are referred to as **Cushing's ulcers**, after Harvey Cushing, a US surgeon.

The primary mechanisms leading to erosive gastritis appear to be ischaemia of the gastric mucosa resulting from sympathetic vasoconstriction and tissue injury due to gastric acid. As a result, multiple superficial erosions of the gastric mucosa develop. Maintaining the gastric pH at greater than 3.5 and inhibiting gastric acid secretion with medications help prevent erosive gastritis.

MANIFESTATIONS The person with acute gastritis may have mild symptoms such as anorexia or mild epigastric discomfort relieved by belching or defecating. More severe manifestations include abdominal pain, nausea and vomiting. Gastric bleeding may occur, with haematemesis or melaena (black, tarry stools that contain blood). Erosive gastritis is not typically associated with pain. The initial symptom often is painless gastric bleeding occurring 2 or more days after the initial stressor. Bleeding typically is minimal, but can be massive. Corrosive gastritis can cause severe bleeding, signs of shock and an *acute abdomen* (severely painful, rigid, board-like abdomen) if perforation occurs. See the accompanying 'Manifestations' box.

Chronic gastritis

Unrelated to acute gastritis, chronic gastritis is a progressive disorder that begins with superficial inflammation and gradually leads to atrophy of gastric tissues. The initial stage is characterised by superficial changes in the gastric mucosa and a decrease in mucus. As the disease evolves, glands of the gastric mucosa are disrupted and destroyed. The inflammatory process involves deep portions of the mucosa, which thins and atrophies. There appear to be at least two different forms of chronic gastritis, classified as type A and type B.

Type A gastritis, the less common form of chronic gastritis, usually affects people of Northern European heritage. This type of gastritis is thought to have an autoimmune component. In type A or autoimmune gastritis, the body produces antibodies to parietal cells and to intrinsic factor. These antibodies destroy gastric mucosal cells, resulting in tissue atrophy and the loss of hydrochloric acid and pepsin secretion. Because intrinsic factor is required for the absorption of vitamin B_{12}, this immune response also results in pernicious anaemia. For further discussion of pernicious anaemia, see Chapter 32.

MANIFESTATIONS Acute and chronic gastritis

ACUTE GASTRITIS

Gastrointestinal
- Anorexia
- Nausea and vomiting
- Haematemesis
- Melaena
- Abdominal pain

Systemic
- Possible shock

CHRONIC GASTRITIS

Gastrointestinal
- Vague discomfort after eating; may be asymptomatic

Systemic
- Anaemia
- Fatigue

Type B gastritis is the more common form of chronic gastritis. Its incidence increases with age, reaching nearly 100% in people over the age of 70. Type B gastritis is caused by chronic infection of the gastric mucosa by *H. pylori*, a gram-negative spiral bacterium. *H. pylori* infection causes inflammation of the gastric mucosa, with infiltration by neutrophils and lymphocytes. The outermost layer of gastric mucosa thins and atrophies, providing a less effective barrier against the auto-digestive properties of hydrochloric acid and pepsin.

Infection with *H. pylori* also is associated with an increased risk of peptic ulcer disease. *H. pylori* infection significantly increases the risk of developing gastric cancer. See the sections that follow for more information about these disorders.

MANIFESTATIONS Chronic gastritis is often asymptomatic until atrophy is sufficiently advanced to interfere with digestion and gastric emptying. The person may complain of vague gastric distress, epigastric heaviness after meals or ulcer-like symptoms. These symptoms typically are not relieved by antacids. In addition, the person may experience fatigue and other symptoms of anaemia. If intrinsic factor is lacking, paraesthesias and other neurological manifestations of vitamin B_{12} deficiency may be present. See the 'Manifestations' box above.

INTERPROFESSIONAL CARE

Acute gastritis is usually diagnosed by the history and clinical presentation. In contrast, the vague symptoms of chronic gastritis may require more extensive diagnostic testing.

People with acute and chronic gastritis are generally managed in community settings. The person requires acute care only when nausea and vomiting are severe enough to interfere with their normal fluid and electrolyte balance and nutritional status. If haemorrhage results, surgical intervention may be required.

Diagnosis

Diagnostic tests that may be ordered for the person with gastritis include the following:

- *Haemoglobin, haematocrit* and *red blood cell (RBC) indices* are evaluated for evidence of anaemia. The person with gastritis may develop pernicious anaemia because of parietal cell destruction or iron-deficiency anaemia because of chronic blood loss.
- *Serum vitamin B_{12} levels* are measured to evaluate for possible pernicious anaemia. Normal values for vitamin B_{12} are 200 to 1000 pg/mL, with lower levels seen in older adults.
- *Upper endoscopy* may be done to inspect the gastric mucosa for changes, identify areas of bleeding and obtain tissue for biopsy. Bleeding sites may be treated with electro- or laser coagulation or injected with a sclerosing agent during the procedure. See the 'Diagnostic tests' box in Chapter 20 for preparation and teaching related to an upper endoscopy.

Medications

Drugs such as a PPI, H_2-receptor blocker or sucralfate may be ordered to prevent or treat acute stress gastritis. PPIs and H_2-receptor blockers reduce the amount or effects of hydrochloric acid on the gastric mucosa. Lansoprazole (Zoton), esomeprazole (Nexium) and omeprazole (Losec) are examples of PPIs. H_2-receptor blockers include cimetidine (Tagamet), ranitidine (Zantac), famotidine (Pepcid) and nizatidine (Nizac). These drugs also are available in non-prescription strength. Sucralfate (Carafate) works locally to prevent the damaging effects of acid and pepsin on gastric tissue. It does not neutralise or reduce acid secretion. Nursing implications for drugs commonly used in managing gastritis are included in the 'Medication administration' box on page 671.

The person with type B chronic gastritis may be treated to eradicate the *H. pylori* infection. This generally involves combination therapy consisting of two antibiotics (such as metronidazole and clarithromycin or a tetracycline) and a PPI. In some cases, eradication of the infection is not warranted and the person is treated symptomatically.

Treatments

In acute gastritis, gastrointestinal tract rest is provided by 6 to 12 hours of NBM status, then slow reintroduction of clear fluids (broth, tea, jelly, carbonated beverages), followed by ingestion of free fluids (creamy soups, puddings, milk) and finally a gradual reintroduction of solid food.

If nausea and vomiting threaten fluid and electrolyte balance, intravenous fluids and electrolytes are ordered.

Nursing care

Health promotion

Health promotion aims to educate the person about measures to prevent acute gastritis. Food contaminated with bacteria is a significant cause of acute gastritis. Discuss food safety measures such as fully cooking meats and egg products and promptly refrigerating foods after cooking to avoid bacterial growth. Stress that food contaminated with potential pathogens often looks, smells and tastes good, making it difficult to identify.

Teach people to abstain from eating or drinking anything during an acute episode of vomiting and then to reintroduce clear liquids gradually once vomiting has stopped (2 to 4 hours after the last episode of vomiting). Suggest using a sports drink to replace lost electrolytes and fluid. Instruct people to avoid milk and milk products until they easily tolerate clear liquids and solid foods such as dry toast or crackers.

Assessment

Assessment data to collect for people with acute or chronic gastritis include the following:

- *Health history:* current symptoms and their duration; relieving and aggravating factors; history of ingestion of toxins, contaminated food, alcohol, aspirin or NSAIDs; other medications.
- *Physical examination:* vital signs, including orthostatic vitals if indicated; peripheral pulses; general appearance; abdominal assessment, including appearance, bowel sounds and tenderness.

Nursing diagnoses and interventions

In planning and implementing nursing care for the person with acute or chronic gastritis, consider both the direct effects of the disorder on the gastrointestinal system and nutritional status, as well as its effects on lifestyle and psychosocial integrity. This section focuses on problems of fluid balance and nutrition.

Risk of fluid volume deficit related to gastrointestinal upset

Nausea, vomiting and abdominal distress are the primary manifestations of acute gastritis. The risk of fluid and electrolyte imbalance is high because of inadequate intake of food and fluids and abnormal losses of fluids and electrolytes with vomiting.

- Monitor and record vital signs at least every 2 hours until stable, then every 4 hours. Check for orthostatic hypotension.

CONSIDERATION FOR PRACTICE

Tachycardia, tachypnoea and hypotension, especially orthostatic hypotension, may indicate fluid volume deficit. Electrolyte or acid–base imbalances resulting from vomiting may cause cardiac arrhythmias or changes in respirations.

- Weigh daily. Monitor and record intake and output; record urine output every 1 to 4 hours as indicated. *Daily weights are an accurate indicator of fluid volume. Urine output of less than 30 mL per hour indicates decreased cardiac output and a need for prompt fluid replacement.*
- Monitor skin turgor, colour and condition and status of oral mucous membranes frequently. Provide skin and mouth care frequently. *Skin turgor and mucous membrane assessments indicate hydration status. Good skin and mouth care are necessary to maintain skin and mucous membrane integrity.*
- Monitor laboratory values for electrolytes and acid–base balance. Report significant changes or deviations from normal. *Electrolytes are lost through vomiting, increasing the risk of electrolyte and acid–base imbalances. These imbalances, in turn, affect multiple body systems.*
- Administer oral or parenteral fluids as ordered. *Oral fluids may be withheld until vomiting has ceased, then gradually reintroduced. Intravenous fluids restore or maintain hydration until adequate oral intake is resumed.*
- Administer anti-emetic and other drugs as ordered to relieve vomiting and facilitate oral feeding. Encourage fluids as soon as feasible. *The oral route is preferred for fluid and nutrient intake; medications may be used to allow earlier resumption of feeding.*

CONSIDERATION FOR PRACTICE

Ensure safety: place buzzer within reach, put up the side rails, instruct person to avoid getting up without assistance. Orthostatic hypotension may lead to syncope and to falls if the person attempts to get up without assistance.

Risk of imbalanced nutrition (less than body requirements) related to gastrointestinal upset

Manifestations of chronic gastritis may lead to reduced food intake and malnutrition. The person often associates these unpleasant sensations with eating and may gradually reduce food intake. Associated anorexia also contributes to poor food intake.

- Monitor and record food and fluid intake and any abnormal losses (such as vomiting). *Careful monitoring can help in developing a dietary plan to meet the energy needs of the person. Consultation with a dietitian may prove beneficial.*
- Monitor weight and laboratory studies such as serum albumin, haemoglobin and RBC indices. *Weights and laboratory values provide data regarding nutritional status and the effectiveness of interventions.*
- Arrange for dietary consultation to determine energy and nutrient needs and develop a dietary plan. Consider food preferences and tolerances in menu planning. *A diet high in protein, vitamins and minerals may be prescribed to meet the nutritional needs of the person with chronic gastritis. In addition, specific food intolerances may need to be considered. Planning to include preferred foods in the diet helps ensure consumption of the prescribed diet.*
- Provide nutritional supplements between meals or frequent small feedings as needed. *Many people with chronic gastritis tolerate small, frequent feedings better than three large meals per day.*
- Maintain tube feedings or parenteral nutrition as ordered. Refer to Chapter 21 for further information on enteral and parenteral feedings.

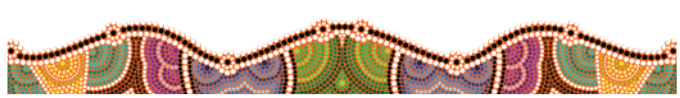
Community-based care

Because acute or chronic gastritis is usually managed in community-based settings, education is vital. For the person with acute gastritis, teaching focuses on managing acute symptoms, reintroducing fluids and solid foods, identifying indicators of possible complications (e.g. continued vomiting, signs of fluid and electrolyte imbalance) and preventing future episodes.

Provide the following information for people with chronic gastritis:

- maintaining optimal nutrition
- helpful dietary modifications
- using prescribed medications
- avoiding known gastric irritants, such as aspirin, NSAIDs, alcohol and cigarette smoking.

Referral to smoking cessation classes or programs to treat alcohol abuse may be necessary.



THE PERSON WITH PEPTIC ULCER DISEASE

Peptic ulcer disease (PUD), a break in the mucous lining of the gastrointestinal tract where it comes in contact with gastric juice, is a chronic health problem. In Australia, people presenting

to hospital with PUD are likely to be male and aged 60 or above (Halland et al., 2011). **Peptic ulcers** occur in any area of the gastrointestinal tract exposed to acid–pepsin secretions, including the oesophagus, stomach or duodenum. **Duodenal ulcers** usually develop between the ages of 60 and 69 and are more common in men than women. **Gastric ulcers** usually develop between the ages of 50 and 59 (Halland et al., 2011).

Risk factors

Chronic *H. pylori* infection and use of aspirin and NSAIDs are the major risk factors for PUD. Contributing risk factors are listed in Box 22.4. Overall, it is estimated that one in six people infected with *H. pylori* develops PUD. A strong familial pattern suggests a genetic factor in the development of PUD. Cigarette smoking is a significant risk factor, doubling the risk of PUD. Cigarette smoking inhibits the secretion of bicarbonate by the pancreas and possibly causes more rapid transit of gastric acid into the duodenum.

Pathophysiology

The innermost layer of the stomach wall, the gastric mucosa, consists of columnar epithelial cells supported by a middle layer of blood vessels and glands and a thin outer layer of smooth muscle. The mucosal barrier of the stomach, a thin coating of mucous gel and bicarbonate, protects the gastric mucosa. The mucosal barrier is maintained by bicarbonate secreted by the epithelial cells, by mucous gel production stimulated by prostaglandins and by an adequate blood supply to the mucosa.

An **ulcer**, or break in the gastrointestinal mucosa, develops when the mucosal barrier is unable to protect the mucosa from damage by hydrochloric acid and pepsin, the gastric digestive juices. See the accompanying 'Pathophysiology illustrated: Peptic ulcer disease'.

H. pylori infection, found in about 70% of people who have PUD, is unique in colonising the stomach. It is spread person to person (oral–oral or faecal–oral) and contributes to ulcer formation in several ways. The bacteria produce enzymes that reduce the efficacy of mucous gel in protecting the gastric mucosa. In addition, the host's inflammatory response to *H. pylori* contributes to gastric epithelial cell damage without producing immunity to the infection. Although the gastric mucosa is the usual site for *H. pylori* infection, this infection also contributes to duodenal ulcers. This is possibly related to increased gastric acid production associated with *H. pylori* infection.

NSAIDs contribute to PUD through both systemic and topical mechanisms. Prostaglandins are necessary for maintaining the gastric mucosal barrier. NSAIDs interrupt prostaglandin synthesis by disrupting the action of the enzyme cyclooxygenase (COX). The two forms of this enzyme are COX-1 and COX-2. The COX-1 enzyme is necessary to maintain the integrity of the gastric mucosa, but the anti-inflammatory effects of NSAIDs are due to their ability to inhibit the COX-2 enzyme. The COX-2-selective NSAIDs may be less damaging to the gastric mucosa because they have less effect on the COX-1 enzyme. In addition to their systemic effect, aspirin and many NSAIDs cross the lipid membranes of gastric epithelial cells, damaging the cells themselves.

The ulcers of PUD may affect the oesophagus, stomach or duodenum. They may be superficial or deep, affecting all layers of the mucosa (see 'Pathophysiology illustrated: Peptic ulcer disease'). Duodenal ulcers usually develop in the proximal portion of the duodenum, close to the pylorus (see Figure 22.7). They are sharply demarcated and usually less than 1 cm in diameter (see Figure 22.8). Gastric ulcers often are found on

BOX 22.4 Risk factors for peptic ulcer disease

- *H. pylori* infection
- Low socioeconomic status
- Crowded, unsanitary living conditions
- Unclean food or water
- Use of aspirin
- Advanced age
- History of PUD
- Concurrent use of other drugs such as glucocorticoids or NSAIDs
- Cigarette smoking
- Family history of PUD

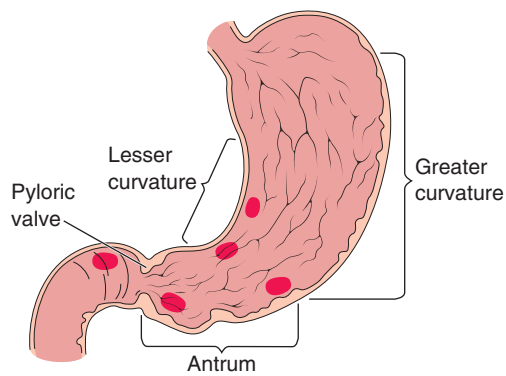


FIGURE 22.7 ■ Common sites affected by peptic ulcer disease

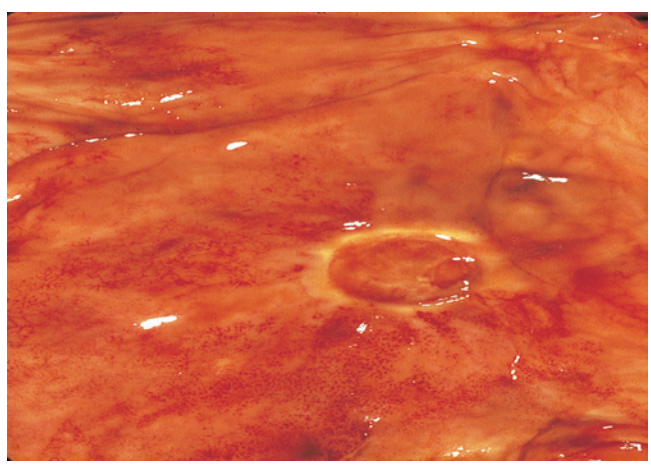


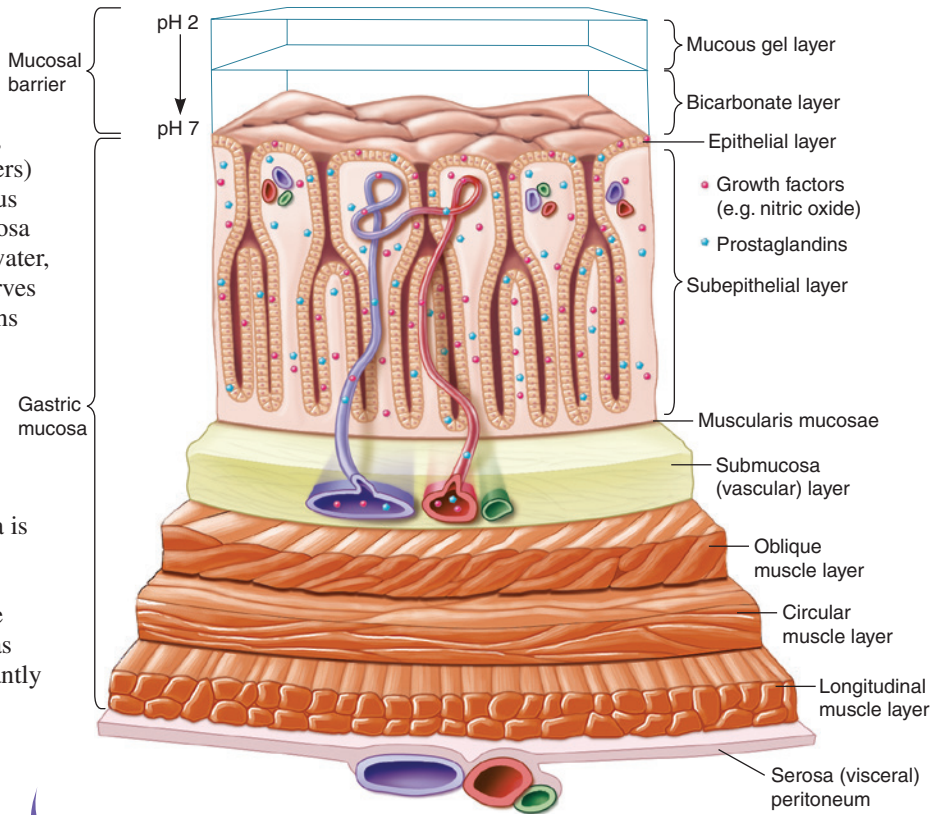
FIGURE 22.8 ■ A superficial peptic ulcer

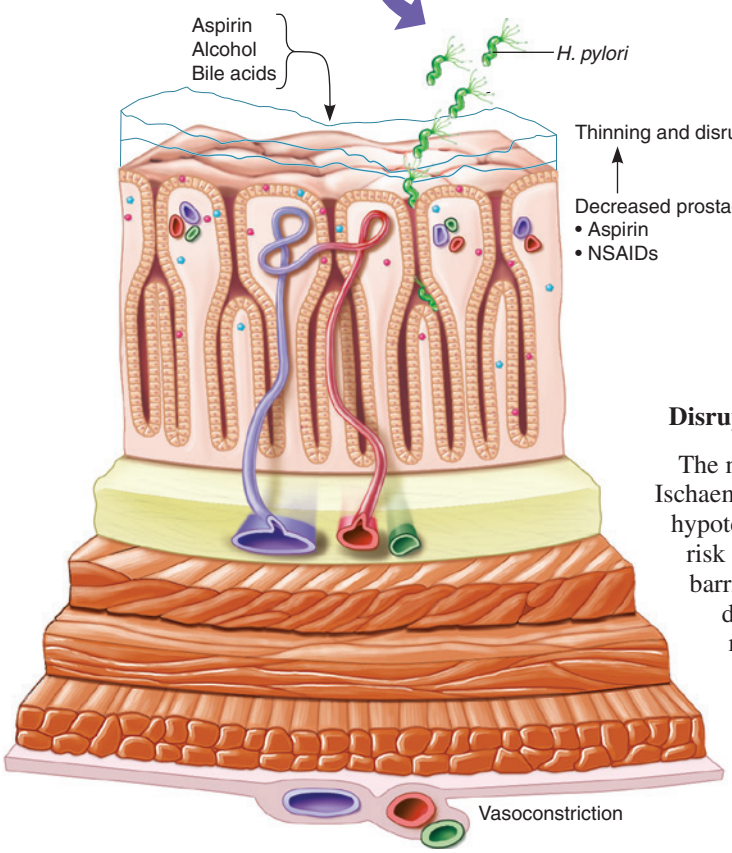
Source: © CNRI/Science Source.

Peptic ulcer disease

Normal gastric mucosa

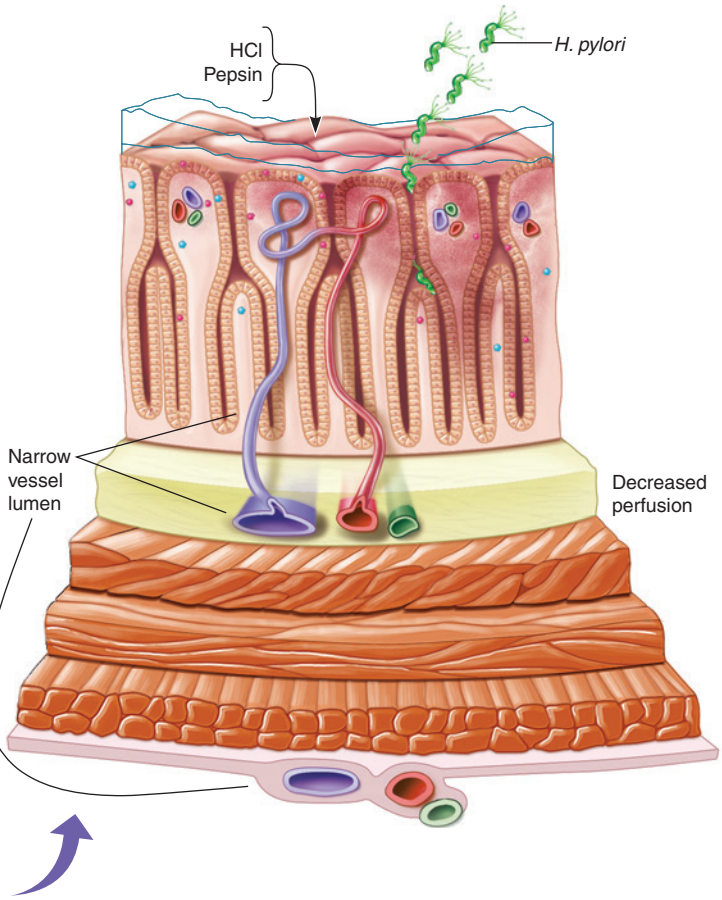
In the stomach and duodenum, the mucosal barrier protects the gastric mucosa (including the epithelial, vascular and smooth muscle layers) from damage. Specialised mucous cells throughout the gastric mucosa produce a mucus (a mixture of water, lipids and glycoproteins) that serves as a barrier to the diffusion of ions (such as hydrogen ion) and molecules (such as pepsin). A thin layer of bicarbonate, secreted by surface epithelial cells, forms between the mucous and cell membranes. Blood flow to the gastric mucosa is vital to maintain this barrier. Prostaglandins and nitric oxide stimulate mucus and bicarbonate production, helping maintain it as well. The mucosal barrier constantly bathes surfaces of the gastric epithelial lining.





Disruption of mucosal barrier

The mucosal barrier can be disrupted by a number of factors. Ischaemia of the gastric mucosa (e.g. due to haemorrhage, hypotension or shock) impairs mucus production, increasing the risk of damage to the mucosa. Aspirin disrupts the mucosal barrier and, along with other non-steroidal anti-inflammatory drugs, inhibits prostaglandins which are necessary to maintain mucous production. Alcohol and bile acids also damage the mucous barrier. *Helicobacter pylori*, a common pathogen to infect the gastric mucosa, disrupts the mucosal barrier.

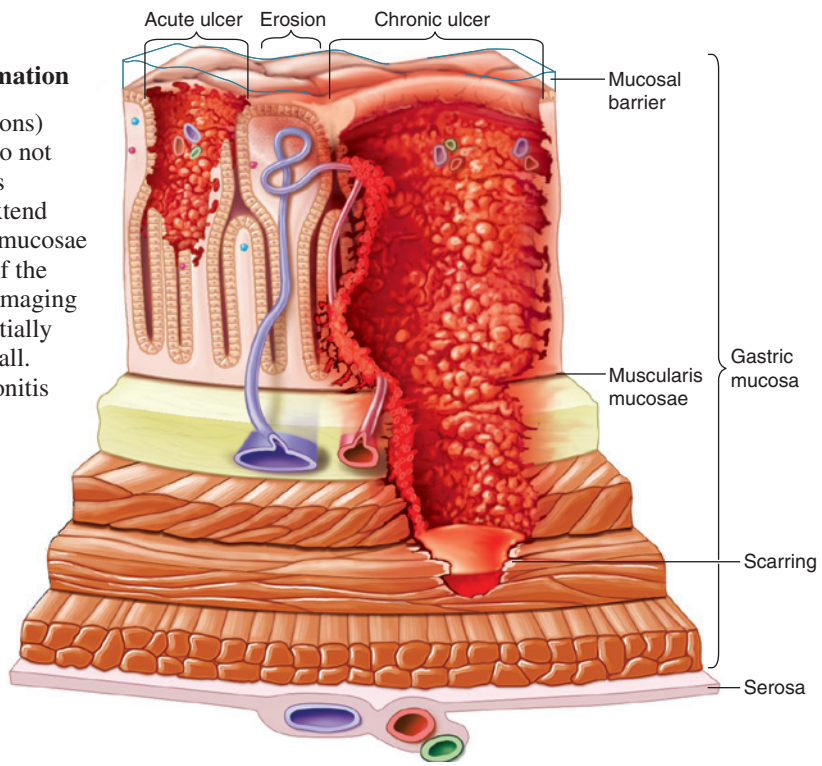


Inflammatory process

When the mucosal barrier is damaged, gastric acid and digestive juices disrupt the epithelial cell membranes, allowing acid to diffuse into cell walls. An acute inflammatory process results. Gastric epithelial cells migrate to the damaged area, a process known as restitution. Adequate blood flow and an alkaline environment are necessary for this repair process. Prostaglandins play an important role in epithelial repair. In the presence of *H. pylori* infection, excess acid production, inadequate blood flow, inhibition of prostaglandins, and other factors that are less clear, the inflammatory process further damages gastric and duodenal epithelial cells, leading to ulceration of the mucosa.

Erosion and ulcer formation

Superficial ulcers (erosions) erode the mucosa, but do not penetrate the muscularis mucosae. True ulcers extend through the muscularis mucosae and into deeper layers of the gastrointestinal wall, damaging blood vessels and potentially penetrating the entire wall. Haemorrhage and peritonitis are potential acute complications of peptic ulcers.



the lesser curvature and the area immediately proximal to the pylorus. Gastric ulcers are associated with an increased incidence of gastric cancer.

Peptic ulcer disease may be chronic, with spontaneous remissions and exacerbations. Exacerbations of the disease may be associated with trauma, infection or other physical or psychological stressors.

Manifestations

Pain is the classic symptom of peptic ulcer disease. The pain is typically described as gnawing, burning, aching or hunger-like and is experienced in the epigastric region, sometimes radiating to the back. The pain occurs when the stomach is empty (2 to 3 hours after meals and in the middle of the night) and is relieved by eating, with a classic 'pain–food–relief' pattern. The person may complain of heartburn or regurgitation and may vomit.

The presentation of peptic ulcer disease in the older adult is often less clear, with vague and poorly localised discomfort, perhaps chest pain or dysphagia, weight loss or anaemia. In the older adult, a complication of PUD, such as upper GI haemorrhage or perforation of the stomach or duodenum, may be the presenting symptom.

Complications

The complications associated with peptic ulcers include haemorrhage, obstruction and perforation. See the box below for the manifestations of these complications.

Among people with PUD, 10–20% experience **haemorrhage** as a result of ulceration and erosion into the blood vessels of the gastric mucosa. In the older adult, bleeding is the most frequent complication. When small blood vessels erode, blood loss may be slow and insidious, with occult blood in the stool the only initial sign. If bleeding continues, the person becomes anaemic and experiences symptoms of weakness, fatigue, dizziness and orthostatic hypotension. Erosion into a larger vessel can lead to sudden and severe bleeding with haematemesis, melaena or haematochezia (blood in the stool) and signs of hypovolaemic shock.

Gastric outlet obstruction may result from oedema surrounding the ulcer, smooth muscle spasm or scar tissue. Generally, obstruction is a gradual rather than an acute process. Symptoms include a feeling of epigastric fullness, accentuated ulcer symptoms and nausea. If the obstruction becomes complete, vomiting occurs. Hydrochloric acid, sodium and potassium are lost in vomitus, potentially leading to fluid and electrolyte imbalance and metabolic alkalosis.

The most lethal complication of PUD is **perforation** of the ulcer through the mucosal wall. When perforation occurs, gastric or duodenal contents enter the peritoneum, causing an inflammatory process and peritonitis. Chemical peritonitis from the hydrochloric acid, pepsin, bile and pancreatic fluid is immediate; bacterial peritonitis follows within 6 to 12 hours from gastric contaminants entering the normally sterile peritoneal cavity. When an ulcer perforates, the person has immediate, severe upper abdominal pain, radiating throughout the abdomen and possibly to the shoulder. The abdomen becomes rigid and board-like, with absent bowel sounds. Signs of shock may be present, including diaphoresis, tachycardia and rapid, shallow respirations. Classic symptoms of perforation may not be present in an older adult. The older adult may instead present with mental confusion and other non-specific symptoms. This atypical presentation can lead to delays in diagnosis and treatment, increasing the associated mortality rate.

MANIFESTATIONS PUD complications

HAEMORRHAGE
- Occult or obvious blood in the stool
- Haematemesis
- Fatigue
- Weakness, dizziness
- Orthostatic hypotension
- Hypovolaemic shock

OBSTRUCTION
- Sensations of epigastric fullness
- Nausea and vomiting
- Electrolyte imbalances
- Metabolic alkalosis

PERFORATION
- Severe upper abdominal pain, radiating to the shoulder
- Rigid, board-like abdomen
- Absence of bowel sounds
- Diaphoresis
- Tachycardia
- Rapid, shallow respirations
- Fever

Zollinger–Ellison syndrome

Zollinger–Ellison syndrome is peptic ulcer disease caused by a gastrinoma or gastrin-secreting tumour of the pancreas, stomach or intestines. Gastrinomas may be benign, although 50–70% are malignant tumours. Gastrin is a hormone that stimulates the secretion of pepsin and hydrochloric acid. The increased gastrin levels associated with these tumours result in hypersecretion of gastric acid, which in turn causes mucosal ulceration.

The peptic ulcers of Zollinger–Ellison syndrome may affect any portion of the stomach or duodenum, as well as the oesophagus or jejunum. Characteristic ulcer-like pain is common. The high levels of hydrochloric acid entering the duodenum may also cause diarrhoea and steatorrhoea (excess fat in the faeces) from impaired fat digestion and absorption. Complications of bleeding and perforation are often seen with Zollinger–Ellison syndrome. Fluid and electrolyte imbalances may also result from persistent diarrhoea, with resultant losses of potassium and sodium in particular.

INTERPROFESSIONAL CARE

Treatment for PUD focuses on eradicating *H. pylori* infection and treating or preventing ulcers related to the use of NSAIDs.

Diagnosis

- *CT scan* using oral contrast is commonly the diagnostic procedure chosen first; it is less costly and less invasive than endoscopy. Small or very superficial ulcers may be missed, however.
- *Upper endoscopy* allows visualisation of the oesophageal, gastric and duodenal mucosa and direct inspection of ulcers. Tissue also can be obtained for biopsy. Nursing care of the person undergoing an endoscopy is outlined in Chapter 20.
- Biopsy specimens obtained during an endoscopy can be tested for the presence of *H. pylori* using several different methods. In the *biopsy urease test*, the specimen is put into a gel containing urea. If *H. pylori* is present, the urease that it produces changes the colour of the gel, often within minutes. Biopsy specimen cells also can be microscopically examined or cultured for evidence of *H. pylori*. Although these tests are highly specific for *H. pylori* infection, their invasiveness, cost and lack of availability in some areas limits their usefulness.
- A non-invasive method of detecting *H. pylori* infection is the *urea breath test*. In this test, radiolabelled urea is given orally. The urease produced by *H. pylori* bacteria converts the urea to ammonia and radio-labelled carbon dioxide, which can then be measured as the person exhales.
- If Zollinger–Ellison syndrome is suspected, *gastric analysis* may be performed to evaluate gastric acid secretion. Stomach contents are aspirated through a nasogastric tube and analysed. In Zollinger–Ellison syndrome, gastric acid levels are very high.

Medications

The medications used to treat PUD include agents to eradicate *H. pylori*, drugs to decrease gastric acid content and agents that protect the mucosa. Nursing responsibilities related to selected drugs to treat GORD, gastritis and PUD are found in the 'Medication administration' box on pages 671–672.

Eradication of *H. pylori* is often difficult. Combination therapies that use two antibiotics with a proton-pump inhibitor (e.g. combinations of a PPI, clarithromycin and amoxicillin, or a PPI, a tetracycline and metronidazole) are necessary. With complete eradication of *H. pylori*, reinfection rates are less than 0.5% per year.

In people who have NSAID-induced ulcers, the NSAID in use should be discontinued if at all possible. If this is not possible, twice-daily PPIs enable ulcer healing.

Medications that decrease gastric acid content include PPIs and the H_2-receptor antagonists.

- Proton-pump inhibitors bind the acid-secreting enzyme (H^+, K^+ ATPase) that functions as the proton pump, disabling it for up to 24 hours. These drugs are very effective, resulting in more than 90% ulcer healing after 4 weeks. Compared with the H_2-receptor blockers, the PPIs provide faster pain relief and more rapid ulcer healing.
- Histamine$_2$-receptor blockers inhibit histamine binding to the receptors on the gastric parietal cells to reduce acid secretion. These drugs are very well tolerated and have few serious side effects; however, drug interactions can occur. These drugs must be continued for 8 weeks or longer for ulcer healing.

Agents that protect the mucosa include sucralfate, bismuth, antacids and prostaglandin analogues.

- Sucralfate binds to proteins in the ulcer base, forming a protective barrier against acid, bile and pepsin. Sucralfate also stimulates the secretion of mucus, bicarbonate and prostaglandin.
- Antacids stimulate gastric mucosal defences, thereby aiding in ulcer healing. They provide rapid relief of ulcer symptoms and are often used as needed to supplement other anti-ulcer medications. Antacids are inexpensive, but people often have difficulty with a regular regimen because the drugs must be taken frequently and may cause either constipation (from the aluminium-type antacids) or diarrhoea (from the magnesium-based antacids). Antacids also interfere with the absorption of iron, digoxin, some antibiotics and other drugs.
- Prostaglandin analogues (misoprostol) promote ulcer healing by stimulating mucus and bicarbonate secretions and by inhibiting acid secretion. Although not as effective as the other drugs discussed, misoprostol is used to prevent NSAID-induced ulcers.

Treatments

NUTRITION In addition to pharmacological treatment, people are encouraged to maintain good nutrition, consuming balanced meals at regular intervals. It is important to teach people that bland or restrictive diets are no longer necessary. Mild alcohol intake is not harmful. Smoking should be discouraged because it slows the rate of healing and increases the frequency of relapses.

SURGERY The identification of *H. pylori* as a cause of PUD and the availability of drugs to treat the infection and heal peptic ulcers has all but eliminated surgery as a treatment option for peptic ulcer disease. Older people, however, may have undergone gastric resection surgery for PUD and may have long-term complications related to the surgery. See the section on gastric cancer for more information about gastric surgery and its potential complications.

Treatment of complications

The person hospitalised with a complication of PUD, such as bleeding, gastrointestinal obstruction or perforation, and peritonitis, requires additional interventions to restore homeostasis.

In haemorrhage associated with PUD, initial interventions focus on restoring and maintaining circulation. Crystalloid solutions such as normal saline are administered intravenously to restore intravascular volume if signs of shock (tachycardia, hypotension, pallor, low urine output and anxiety) are present. Whole blood or packed red blood cells may be administered to restore haemoglobin and haematocrit levels. A nasogastric tube is inserted to prevent aspiration of vomited gastric contents.

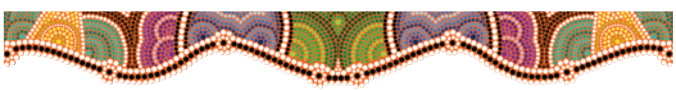
Endoscopy with direct injection of a clotting or sclerosing agent into the bleeding vessel may be performed. Laser photocoagulation, using light energy, or electrocoagulation, which uses electric current to generate heat, can also be done via endoscopy to seal bleeding vessels.

The person is kept NBM until bleeding is controlled. Proton-pump inhibitors are administered intravenously (e.g. 40 mg

of esomeprazole (Nexium) per intravenous push or admixture daily) to reduce the risk of rebleeding. Surgery may be necessary if medical measures are ineffective in controlling bleeding. Older adults who experience bleeding as a complication of PUD are more likely to rebleed or require surgery to control the haemorrhage. See page 695 for the nursing care of the person having gastric surgery.

Repeated inflammation, healing, scarring, oedema and muscle spasm can lead to gastric outlet (pyloric) obstruction. Initial treatment includes gastric decompression with nasogastric suction and administration of intravenous normal saline and potassium chloride to correct fluid and electrolyte imbalance. H_2-receptor blockers are given intravenously as well. Balloon dilation of the gastric outlet may be done via upper endoscopy. If these measures are unsuccessful in relieving obstruction, surgery may be required.

Gastric or duodenal perforation resulting in contamination of the peritoneum with gastrointestinal contents often requires immediate intervention to restore homeostasis and minimise peritonitis. Intravenous fluids maintain fluid and electrolyte balance. Nasogastric suction removes gastric contents and minimises peritoneal contamination. Placing the person in the Fowler's or semi-Fowler's position allows peritoneal contaminants to pool in the pelvis. Intravenous antibiotics aggressively treat bacterial infection from intestinal flora. Laparoscopic surgery or an open laparotomy may close the perforation.



Nursing care

Health promotion

Although it is difficult to predict which people will develop peptic ulcer disease, promote health by advising people to avoid risk factors such as excessive aspirin or NSAID use and cigarette smoking. In addition, encourage people to seek treatment for manifestations of GORD or chronic gastritis, both of which are also associated with *H. pylori* infection.

Assessment

Collect the following subjective and objective data when assessing the person with peptic ulcer disease:

- *Health history:* complaints of epigastric or left upper quadrant pain, heartburn or discomfort; its character, severity, timing and relationship to eating; measures used for relief; nausea or vomiting, presence of bright blood or 'coffee grounds' appearing material in vomitus; current medications, including use of aspirin or other NSAIDs; cigarette smoking and use of alcohol or other drugs.
- *Physical examination:* general appearance, including height and weight relationship; vital signs, including orthostatic measurements; abdominal examination, including shape and contour, bowel sounds and tenderness to palpation; presence of obvious or occult blood in vomitus and stool.

Nursing diagnoses and interventions

The priorities of nursing care for the person with peptic ulcer disease are reducing discomfort, maintaining nutritional status and preventing or rapidly identifying and intervening for potential complications. See the accompanying nursing care plan below.

Pain related to PUD

The pain of PUD is often predictable and preventable. Pain is typically experienced 2 to 4 hours after eating, as high levels of gastric acid and pepsin irritate the exposed mucosa. Measures to neutralise the acid, minimise its production or protect the mucosa often relieve this pain, minimising the need for analgesics.

- Assess pain, including location, type, severity, frequency and duration, and its relationship to food intake or other contributing factors.
- Administer PPIs, H_2-receptor antagonists, antacids or mucosal protective agents as ordered. Monitor for effectiveness and side effects or adverse reactions. *The pain associated with PUD is generally caused by the effect of gastric juices on exposed mucosal tissue. These medications reduce pain and promote healing by reducing acid production, neutralising the acid or providing a barrier for the damaged mucosa.*
- Teach relaxation, stress reduction and lifestyle management techniques. Refer for stress management counselling or classes as indicated. *Although there is no clear relationship between stress and PUD, measures to relieve stress and promote physical and emotional rest help to reduce the perception of pain and may reduce ulcer genesis.*

CONSIDERATION FOR PRACTICE

Avoid making assumptions about pain. Acute pain may indicate a complication, such as perforation (often heralded by sudden, severe epigastric pain and a rigid, board-like abdomen) or it may be totally unrelated to PUD (e.g. angina, gallbladder disease or pancreatitis).

Disturbed sleep pattern

Night-time ulcer pain, which typically occurs between 1 and 3 am, may disrupt the sleep cycle and result in inadequate rest. Anticipation of pain may lead to insomnia or other sleep disruptions.

- Stress the importance of taking medications as prescribed. *The bedtime dose of PPI or H_2-receptor blocker minimises hydrochloric acid production during the night, reducing night-time pain.*
- Instruct to limit food intake after the evening meal, eliminating any bedtime snack. *Eating before bed can stimulate the production of gastric acid and pepsin, increasing the likelihood of night-time pain.*
- Encourage the use of relaxation techniques and comfort measures such as soft music as needed to promote sleep. *Once the pain associated with PUD has been controlled, these measures help to reduce anxiety and re-establish a normal sleep pattern.*

NURSING CARE PLAN A person with peptic ulcer disease

Sean O'Donnell is a 47-year-old police officer who lives and works in a metropolitan area. Mr O'Donnell has had 'heartburn' and abdominal discomfort for years, but thought it went along with his job. Last year, after becoming weak, light headed and short of breath, he was found to be anaemic and was diagnosed as having a duodenal ulcer. He took omeprazole (Losec) and ferrous sulfate for 3 months before stopping both, saying he had 'never felt better in his life'. Mr O'Donnell has now been admitted to the hospital with active upper GI bleeding.

ASSESSMENT

Rachel Clark is Mr O'Donnell's admitting nurse. On initial assessment, Mr O'Donnell is alert and oriented, though very apprehensive about his condition. Skin pale and cool; BP 136/78, P 98; his abdomen is distended and tender with hyperactive bowel sounds; 200 mL bright red blood is obtained on nasogastric tube insertion. Haemoglobin 8.2 g/dL and haematocrit 23% on admission. Mr O'Donnell is taken to the endoscopy lab where his bleeding is controlled using laser photocoagulation. On his return to the ward, he receives two units of packed RBCs and intravenous fluids to restore blood volume. A 5-day course of high-dose oral omeprazole (40 mg bd) is ordered to prevent rebleeding and Mr O'Donnell is allowed to begin a clear fluid diet 24 hours after his endoscopy. Tissue biopsy obtained during endoscopy confirms the presence of *H. pylori* infection.

DIAGNOSES

- *Deficient fluid volume* related to acutely bleeding duodenal ulcer.
- *Risk of injury* related to acute blood loss.
- *Anxiety* related to threat to wellbeing.
- *Ineffective therapeutic regimen management* related to lack of knowledge regarding PUD and its treatment.

PLANNING

- Ensure buzzer is kept by bedside at all times.
- Discuss situation and provide information about all procedures and treatments. Encourage Mr O'Donnell to alert nursing staff immediately if he experiences any signs of blood loss such as dizziness or vomiting.
- Arrange for a pharmacist review to discuss current and planned treatment measures, the importance of completing the prescribed treatment to reduce the risk of further ulcer development and avoiding use of aspirin or NSAIDs in the future.

Expected outcomes

- Maintains normal blood pressure, pulse and urine output (> 30 mL/h).
- Remains free of injury.
- Seeks information to reduce anxiety.
- Identifies and uses coping strategies to manage anxiety.
- Describes prescribed therapeutic regimen.
- Verbalises ability to manage prescribed regimen.

IMPLEMENTATION

- Encourage Mr O'Donnell to ask for help when getting up or ambulating. Remind to rise slowly from lying to sitting and sitting to standing.
- Reassure about the effectiveness of treatment in reducing the risk of further bleeding.
- Discuss stress reduction techniques and refer for stress reduction counselling or workshops as indicated.

EVALUATION

Mr O'Donnell is discharged 48 hours after admission. He has had no further evidence of bleeding and has resumed a regular diet. His haemoglobin and haematocrit remain low and he has a prescription for ferrous sulfate. He will complete the prescribed high-dose omeprazole regimen at home and then begin treatment with omeprazole, amoxicillin and clarithromycin to eradicate the *H. pylori* infection detected during endoscopy. After 2 weeks of this regimen, he will continue taking omeprazole at bedtime for 4 to 8 weeks. He verbalises a good understanding of his treatment and of the importance of completing the entire regimen. Mr O'Donnell expresses concern about his ability to 'keep his cool on the inside' when under stress. Ms Clark, his Registered Nurse, gives him the names of several resources to help with stress management in case he wants help.

CRITICAL THINKING IN THE NURSING PROCESS

1. How does *H. pylori* infection contribute to the development of peptic ulcers?
2. Describe the physiological responses to fear and anxiety. Why is it important to alleviate fear and its physical consequences in people with PUD?
3. What suggestions can you make to help Mr O'Donnell manage his complex treatment regimen during the next 3 months?
4. Develop a teaching plan that includes stress reduction techniques Mr O'Donnell can use while carrying out his duties as a police officer.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Reflect on the lifestyle stressors involved in Mr O'Donnell's life. What strategies could he use to reduce stress while carrying out his duties as a police officer?

Imbalanced nutrition (less than body requirements) related to PUD

In an attempt to avoid discomfort, the person with PUD may gradually reduce food intake, sometimes jeopardising nutritional status. Anorexia and early satiety are additional problems associated with PUD.

- Assess current diet, including pattern of food intake, eating schedule and foods that precipitate pain or are being avoided in anticipation of pain. *The person may not realise the extent of self-imposed dietary limitations, especially if symptoms have persisted for an extended time. Assessment increases awareness and also helps to identify the adequacy of nutrient intake.*
- Refer to a dietitian for meal planning to minimise PUD symptoms and meet nutritional needs. Consider normal eating patterns and preferences in meal planning. *Although*

no specific diet is recommended for PUD, people should avoid foods that increase pain. Six small meals per day often help increase food tolerance and decrease postprandial discomfort.

- Monitor for complaints of anorexia, fullness, nausea and vomiting. *Adjust dietary intake or medication schedule as indicated. PUD and resultant scarring can lead to impaired gastric emptying, necessitating a treatment change.*

CONSIDERATION FOR PRACTICE

Advise the person to report increasing or persistent symptoms of anorexia, nausea and vomiting, or fullness to the healthcare provider.

- Monitor laboratory values for indications of anaemia or other nutritional deficits. Monitor for therapeutic and side effects of treatment measures such as oral iron replacement. Instruct the person taking oral iron replacement to avoid using an antacid within 1 to 2 hours of taking the iron preparation. *Anaemia can result from poor nutrient absorption or chronic blood loss in people with PUD. Oral iron supplements may cause GI distress, nausea and vomiting; if these side effects are intolerable, notify the physician for a possible change of therapy. Antacids bind with oral iron preparations, blocking absorption.*

Deficient fluid volume related to haemorrhage

Erosion of a blood vessel with resultant haemorrhage is a significant risk for the person with PUD. Acute bleeding can lead to hypovolaemia and fluid volume deficit, which can lead to a decrease in cardiac output and impaired tissue perfusion.

CONSIDERATION FOR PRACTICE

Monitor and record blood pressure and pulse every 15 to 30 minutes until stable. Monitor central venous pressure or pulmonary artery pressure as indicated. Insert an indwelling catheter and monitor urinary output hourly. Weigh daily. Continuous monitoring of cardiac output parameters is essential in people with an acute haemorrhage to identify possible shock and intervene at an early stage.

- Monitor stools and gastric drainage for overt and occult blood. Assess gastric drainage (vomitus or from a nasogastric tube) to estimate the amount and rapidity of haemorrhage. *Drainage is bright red with possible clots in acute haemorrhage; dark red or the colour of coffee grounds when blood has been in the stomach for a period of time. Haematochezia (stool containing red blood and clots) is present in acute haemorrhage; melaena (black, tarry stool) is an indicator of less acute bleeding. When small vessels are disrupted, bleeding may be slow and not overtly evident. With chronic or slow gastrointestinal bleeding, the risk of a fluid volume deficit is minimal; anaemia and activity intolerance are more likely.*
- Maintain intravenous therapy with fluid volume and electrolyte replacement solutions; administer whole blood or packed cells as ordered. *Both fluids and electrolytes are lost through vomiting, nasogastric drainage and diarrhoea in an episode of acute bleeding. To prevent shock, it is essential to maintain a blood volume and cardiac output sufficient to perfuse body tissues. Whole blood and packed cells replace both blood volume and red blood cells, providing additional oxygen-carrying capacity to meet cell needs.*
- Unless contraindicated insert a nasogastric tube and maintain its position and patency. Initially, measure and record gastric output every hour, then every 4 to 8 hours. *Nasogastric suction removes blood from the gastrointestinal tract, preventing vomiting and possible aspiration. Gastric output is replaced millilitre for millilitre with a balanced electrolyte solution to maintain homeostasis.*
- Monitor haemoglobin and haematocrit, serum electrolytes, BUN and creatinine values. Report abnormal findings. *Haemoglobin and haematocrit are lower than normal with acute or chronic GI bleeding. In acute haemorrhage, initial results may be within normal range because both cells and plasma are lost. Loss of fluids and electrolytes with gastric drainage and diarrhoea will alter normal levels. Digestion and absorption of blood in the GI tract may result in elevated BUN and creatinine levels.*
- Assess abdomen, including bowel sounds, distension, girth and tenderness every 4 hours and record findings. *Borborygmi or hyperactive bowel sounds with abdominal tenderness are common with acute GI bleeding. Increased distension, increasing abdominal girth, absent bowel sounds or extreme tenderness with a rigid, board-like abdomen may indicate perforation.*
- Maintain bed rest with the head of the bed elevated. Ensure safety. *Loss of blood volume may cause orthostatic hypotension with resultant syncope or dizziness upon standing.*

Community-based care

Peptic ulcer disease is managed in home and community-based settings; only its complications typically require treatment in an acute care setting. Provide the following information when preparing the person for home care:

- prescribed medication regimen, including desired and potential adverse effects
- importance of continuing therapy even when symptoms are relieved
- relationship between peptic ulcers and factors such as NSAID use and smoking. If indicated, refer to a smoking cessation clinic or program
- importance of avoiding aspirin and other NSAIDs; stress the necessity of reading the labels of over-the-counter medications for possible aspirin content
- manifestations of complications that should be reported to the local doctor, including increased abdominal pain or distension, vomiting, black or tarry stools, light headedness or fainting

- stress and lifestyle management techniques that may help prevent exacerbation. Refer to resources for stress management, such as classes, counselling and formal or informal groups.

THE PERSON WITH CANCER OF THE STOMACH

Worldwide, cancer of the stomach is the second most common cancer after skin cancer; but it is less common in Australia.

FAST FACTS

- The incidence of gastric cancer increases with age, with the most common ages of presentation being between 50 and 70 years.
- Men are affected twice as often as women.
- The incidence of gastric cancer is highest in the Republic of Korea followed by Japan, China and Eastern Europe (Ferlay et al., 2014).
- Five-year survival is poor, at approximately 26% (Cancer Australia, 2015).

Risk factors

H. pylori infection is a major risk factor for cancer of the distal portion of the stomach; from 35% to 89% of cases can be attributed to this infection. Other risk factors are a genetic predisposition, chronic gastritis, pernicious anaemia, gastric polyps and carcinogenic factors in the diet (such as smoked foods and nitrates). Achlorhydria, a lack of hydrochloric acid in the stomach, is a known risk factor. The risk of gastric cancer also is increased in people who have had a partial gastric resection. Socioeconomic factors are also linked with gastric cancer, including occupations that involve exposure to certain chemicals such as dust, asbestos, solvents and pesticides; smoking; and a diet poor in fruit and vegetables and lacking antioxidant mechanisms (Santibanez et al., 2012).

Pathophysiology

Adenocarcinoma, which involves the mucus-producing cells of the stomach, is the most common form of gastric cancer. These carcinomas may arise anywhere on the mucosal surface of the stomach but are most frequently found in the distal portion. Fifty to sixty per cent of gastric cancers occur in the antrum or pyloric region (Porth & Matfin, 2009). Gastric cancer begins as a localised lesion (in situ), then progresses to involve the mucosa or submucosa (early gastric carcinoma). Lesions may spread by direct extension to tissues surrounding the stomach—the liver, in particular. The lesion may ulcerate or appear as a polypoid (polyp-like) mass (see Figure 22.9). Lymph node involvement and metastasis occur early due to the rich blood and lymphatic supply to the stomach. Metastatic lesions are often found in the liver, lungs, ovaries and peritoneum.

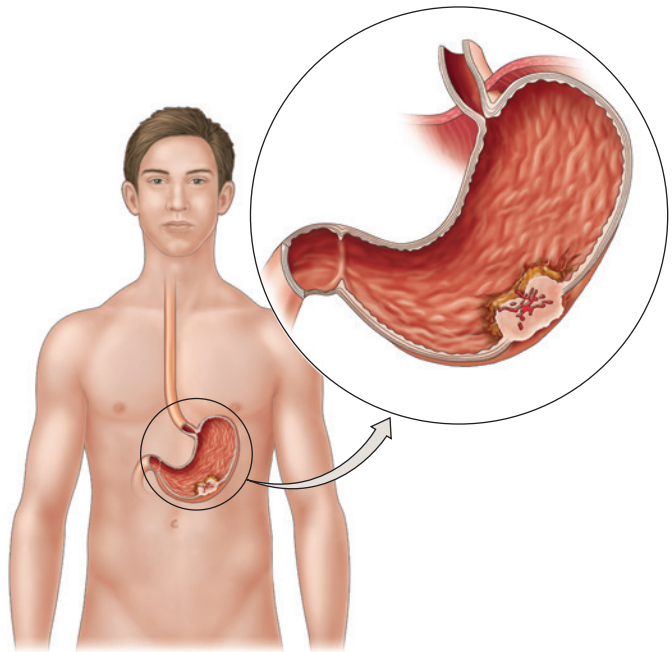


FIGURE 22.9 ■ Gastric cancer affecting the antrum of the stomach

Manifestations

Few symptoms are associated with gastric cancer. Unfortunately, the disease is often quite advanced and metastases are usually present at the time of diagnosis. Early symptoms are vague, including feelings of early satiety, anorexia, indigestion and possibly vomiting. The person may experience ulcer-like pain unrelieved by antacids, typically occurring after meals. As the disease progresses, weight loss occurs and the person may be **cachectic** (in very poor health and malnourished) at the time of diagnosis. An abdominal mass may be palpable and occult blood may be present in the stool, indicating gastrointestinal bleeding.

INTERPROFESSIONAL CARE

Diagnosis

Anaemia detected by a full blood count is often the first indication of gastric cancer. An upper GI x-ray with barium swallow is useful to identify lesions and ultrasound or CT scan may identify a mass. Upper endoscopy with visualisation and biopsy of the lesion provides the definitive diagnosis.

Surgery

When gastric cancer is identified prior to the development of metastasis, surgical removal of part or all of the stomach and regional lymph nodes is the treatment of choice. **Partial gastrectomy** involves removal of a portion of the stomach, usually the distal half to two-thirds. In partial gastrectomy, the surgeon constructs an anastomosis from the remainder of the stomach directly to the duodenum or to the proximal jejunum.

The **gastroduodenostomy**, or **Billroth I**, and the **gastrojejunostomy**, or **Billroth II**, are commonly used partial gastrectomy procedures (see Figures 22.10A and B).

A **total gastrectomy**, removal of the entire stomach, may be done for diffuse cancer that is spread throughout the gastric mucosa but limited to the stomach. In a total gastrectomy, the surgeon constructs an anastomosis from the oesophagus to the duodenum or jejunum. Total gastrectomy with **oesophagojejunostomy** is illustrated in Figure 22.10C.

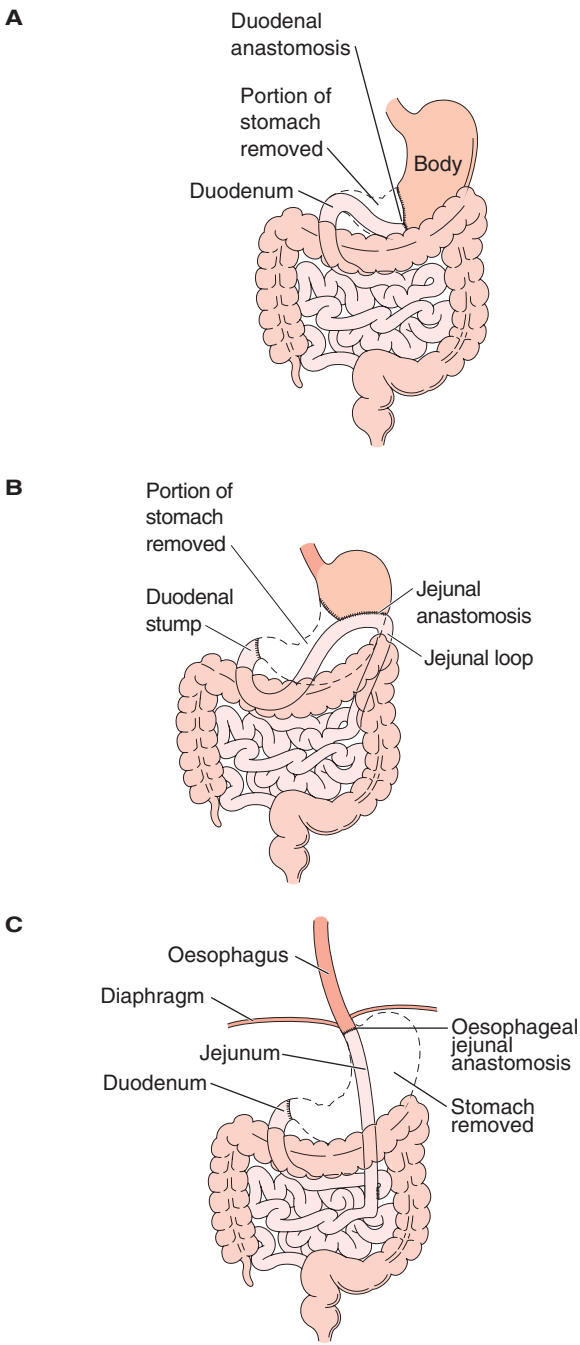


FIGURE 22.10 ■ Partial and total gastrectomy procedures. *A*, Partial gastrectomy with anastomosis to the duodenum. *B*, Partial gastrectomy with anastomosis to the jejunum. *C*, Total gastrectomy with anastomosis of the oesophagus to the jejunum

Nursing care of the person who has undergone gastric surgery is outlined in the following box.

Complications

Several long-term complications may develop following gastrectomy procedures. **Dumping syndrome** is the most common problem. It may follow a partial gastrectomy with duodenal or jejunal anastomosis. When the pylorus has been resected or bypassed, a hypertonic, undigested food bolus may rapidly enter the duodenum or jejunum. Water is pulled into the lumen of the intestine by the hyperosmolar character of the chyme, resulting in decreased blood volume and intestinal dilation. Peristalsis is stimulated and intestinal motility is increased.

Early symptoms of dumping syndrome occur within 5 to 30 minutes after eating. These symptoms result from intestinal dilation, peristaltic stimulation and hypovolaemia caused by undigested food in the proximal small intestine. Manifestations include nausea with possible vomiting, epigastric pain with cramping and borborygmi (loud, hyperactive bowel sounds), and diarrhoea. Systemic symptoms from the hypovolaemia and reflex sympathetic stimulation include tachycardia, orthostatic hypotension, dizziness, flushing and diaphoresis.

The entry of hyperosmolar chyme into the jejunum also causes a rapid rise in the blood glucose. This stimulates the release of an excessive amount of insulin, leading to hypoglycaemic symptoms 2 to 3 hours after the meal. The pathogenesis and clinical manifestations of dumping syndrome are represented in Figure 22.11. Dumping syndrome is typically self-limiting, lasting 6 to 12 months after surgery; however, a small percentage of people continue to experience long-term symptoms.

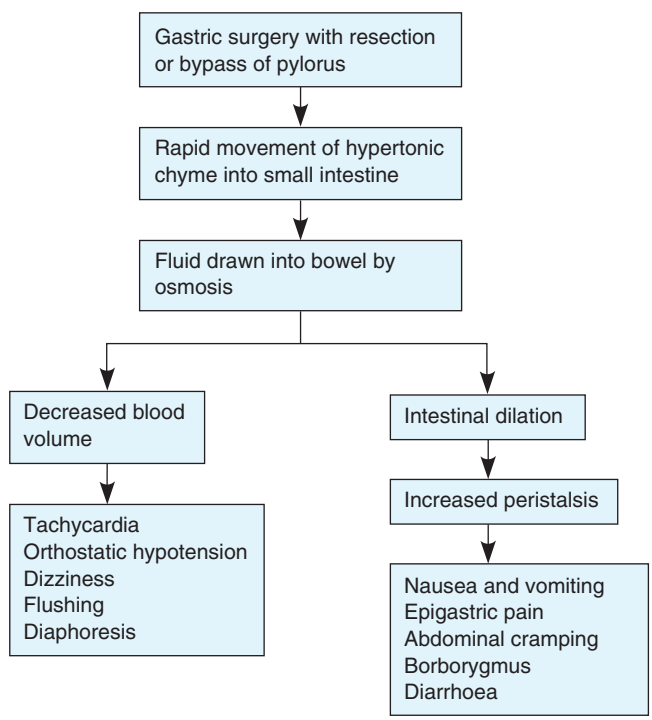


FIGURE 22.11 ■ The pathogenesis and manifestations of dumping syndrome

NURSING CARE OF THE PERSON having gastric surgery

PREOPERATIVE NURSING CARE

- See Chapter 3 for routine preoperative care and teaching.
- Insert a nasogastric tube if ordered preoperatively. *Although it is often inserted in the surgical suite just prior to surgery, the nasogastric tube may be placed preoperatively to remove secretions and empty stomach contents.*

POSTOPERATIVE NURSING CARE

- Provide routine care for the surgical person as outlined in Chapter 3.
- Assess position and patency of nasogastric tube, connecting it to low suction. Gently irrigate with sterile normal saline if tube becomes clogged. The nasogastric tube will be placed in surgery to avoid disruption of the gastric suture lines and should be well secured. If repositioning or tube replacement is needed, notify the surgeon. *Patency must be maintained to keep the stomach decompressed, reducing pressure on sutures.*
- Assess colour, amount and odour of gastric drainage, noting any changes in these parameters or the presence of clots or bright bleeding. Initial drainage is bright red. It becomes dark, then clear or greenish-yellow over the first 2 to 3 days. *A change in the colour, amount or odour may indicate a complication such as haemorrhage, intestinal obstruction or infection.*
- Maintain intravenous fluids while nasogastric suction is in place. *The person on nasogastric suction is not only unable to take oral food and fluids but also is losing electrolyte-rich fluid through the nasogastric tube. If replacement fluid and electrolytes are not maintained, the person is at risk of dehydration, imbalances of electrolytes and metabolic alkalosis.*
- Provide anti-ulcer and antibiotic therapy as ordered. *These medications may be ordered for the postoperative person, depending on the procedure performed. Antibiotic therapy is a common preventive measure for infection that may result from contamination of the abdominal cavity with gastric contents.*
- Monitor bowel sounds and abdominal distension. *Bowel sounds indicate resumption of peristalsis. Increasing distension may indicate third spacing, obstruction or infection.*
- Resume oral food and fluids as ordered. Initial feedings are clear fluids, progressing to free fluids and then frequent small feedings of regular foods. Monitor bowel sounds and for abdominal distension frequently during this period. *Oral feedings are reintroduced slowly to minimise trauma to the suture lines by possible gastric distension.*
- Encourage ambulation. *Ambulation stimulates peristalsis.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Begin discharge planning and teaching. Consult with a dietitian for diet instructions and menu planning; reinforce teaching. Teach the person about potential postoperative complications, such as abdominal abscess, dumping syndrome, postprandial hypoglycaemia or pernicious anaemia. Also, teach the person to recognise signs and symptoms and to take preventive measures. *The person's gastric capacity is reduced after partial gastrectomy, necessitating a corresponding reduction in meal size. Changes in gastric emptying and reduction in gastric secretions may change the person's tolerance for many foods, requiring slow reintroduction of these foods. Dumping syndrome, postprandial hypoglycaemia and pernicious anaemia are possible long-term complications of partial gastrectomy. For most people, dietary modifications can control both dumping syndrome and postprandial hypoglycaemia.*

Dumping syndrome is managed primarily by a dietary pattern that delays gastric emptying and allows smaller boluses of undigested food to enter the intestine. Meals should be small and more frequent. Liquids and solids are taken at separate times instead of together during a meal. The amount of proteins and fats in the diet is increased, because they exit the stomach more slowly than carbohydrates. Carbohydrates, especially simple sugars, are reduced. The person is instructed to rest in a recumbent or semirecumbent position for 30 to 60 minutes after meals. Anticholinergics, sedatives and antispasmodics may be prescribed.

Anaemia may be a chronic problem after a major gastric resection. Iron is absorbed primarily in the duodenum and proximal jejunum; rapid gastric emptying or a gastrojejunostomy may interfere with adequate absorption.

The cells of the stomach produce intrinsic factor, required for the absorption of vitamin B_{12}. Vitamin B_{12} deficiency leads to pernicious anaemia. Because of hepatic stores of vitamin B_{12}, symptoms of anaemia may not be seen for 1 to 2 years after surgery. Vitamin B_{12} levels are routinely monitored following extensive gastric resections.

Other nutritional problems seen following surgery include folic acid deficiency and decreased absorption of calcium and vitamin D. Poor absorption of nutrients, combined with the inability to eat large meals, puts the person at risk of weight loss in addition to the more specific nutrient deficiencies. Nearly 50% of people who have gastric surgery experience significant weight loss, primarily because of insufficient kilojoule intake. Factors contributing to insufficient intake of kilojoules include early satiety (feeling of fullness), decreased stomach size and altered emptying patterns.

Other therapies

Radiation or chemotherapy may be used to eliminate any lymphatic or metastatic spread. For the person with more advanced disease, treatment is palliative and may include surgery and chemotherapy. These people may require a gastrostomy or jejunostomy feeding tube (see Figure 22.12). See the following box for nursing care of the person with a gastrostomy or jejunostomy tube.

Because gastric cancer is generally advanced by the time of diagnosis, the prognosis is poor. The 5-year survival rate of all people treated for gastric carcinoma is 20%.

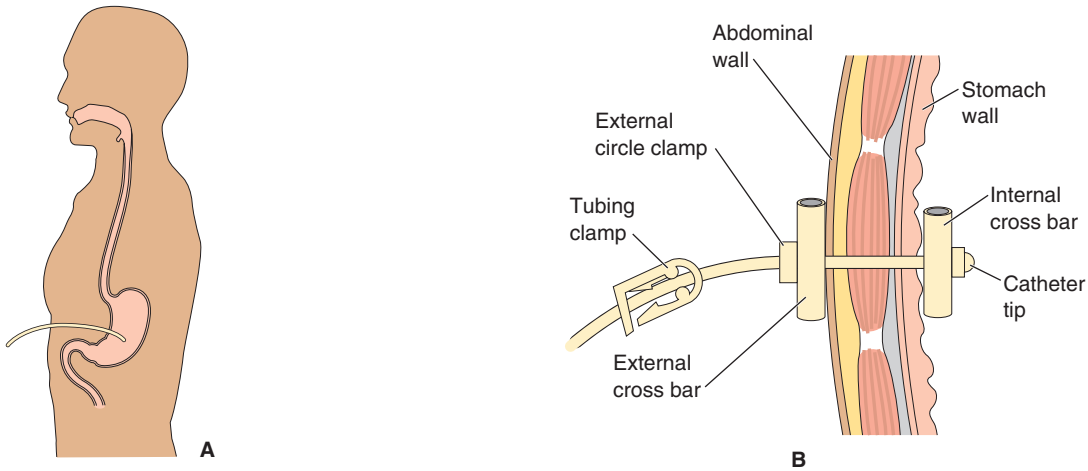


FIGURE 22.12 ■ Gastrostomy. *A*, Gastrostomy tube placement. *B*, The tube is fixed against both the abdominal and the stomach walls by cross bars

NURSING CARE OF THE PERSON with a gastrostomy or jejunostomy tube

People who have had extensive gastric surgery or who require long-term enteral feedings to maintain nutrition may have a gastrostomy or jejunostomy tube inserted.

PROCEDURE

Gastrostomy tubes are surgically placed in the stomach, with the stoma in the epigastric region of the abdomen (see Figure 22.12). Jejunostomy tubes are placed in the proximal jejunum. Immediately following the procedure, the tube may be connected to low suction or plugged. If the person has been receiving tube feedings, these may be reinitiated shortly after tube placement.

NURSING CARE

- Assess tube placement by aspirating stomach contents and checking pH of aspirate to determine gastric or intestinal placement. A pH of 5 or less indicates gastric placement; the pH is generally 7 or higher with intestinal placement. *Recent studies show auscultation to be ineffective in determining feeding tube placement. Measuring the pH of aspirate from the tube is more reliable as a means of determining tube placement.*
- Inspect the skin surrounding the insertion site for healing, redness, swelling and the presence of any drainage. If drainage is present, note the colour, amount, consistency and odour. *Changes in the insertion site, drainage or lack of healing may indicate an infection.*
- Assess the abdomen for distension, bowel sounds and tenderness *to evaluate functioning of the gastrointestinal tract.*
- Until the stoma is well healed, use sterile technique for dressing changes and site care. Clean technique is appropriate for use once healing is complete. *Sterile technique reduces the risk of wound contamination by pathogens that can lead to infection. Once healing has occurred, clean technique is acceptable because the gastrointestinal tract is not a sterile body cavity.*
- Wearing clean gloves, remove old dressing. Cleanse the site with saline or tap water and rinse as appropriate. A well-healed stoma may be cleansed in the shower with the tube clamped or plugged. Pat dry with 4 × 4 gauze pads and allow to air dry. Apply Stomahesive, karaya or other protective agents around tube as needed to protect the skin. *Gastric acid and other wound drainage is irritating to the skin. Meticulous care is important to maintain the integrity of the skin surrounding the stoma.*
- Re-dress the wound using a stoma dressing or folded 4 × 4 gauze pads. Do not cut gauze pads, because threads may enter the wound, causing irritation and increasing the risk of inflammation.
- Irrigate the tube with 30 to 50 mL of water and clean the tube inside and out as indicated or ordered. Soft gastric tubes may require cleaning of the inner lumen to maintain patency. *Tube feeding formulas may coat the inside of the gastrostomy tube and eventually cause it to become occluded. Regular irrigation with water as indicated maintains tube patency.*
- Provide mouth care or remind the person to do so. *When feedings are not being taken orally, the usual stimulus to do mouth care is lost. In addition, salivary fluids may not be as abundant and oral mucous membranes may become dry and cracked.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- If indicated, teach the person and family how to care for the tube and feedings. *Refer to a home health agency or community nurse for support and reinforcement of learning. Gastrostomy tubes are often in place long term. When the person and family are able to assume care, independence and self-image are enhanced.*

Nursing care

Health promotion

Although the exact causes of gastric cancer are unknown, contributing factors such as *H. pylori* infection and consumption of foods preserved with nitrates have been identified. To reduce their risk of developing gastric cancer, encourage people with known *H. pylori* infection to complete the prescribed course of treatment and verify that it has eradicated the infection. With all people, discuss the relationship between gastric cancer and consumption of foods preserved with nitrates (such as bacon and other processed meats) and encourage limited consumption of these products.

Assessment

Assessment data related to gastric cancer include the following:

- *Health history:* manifestations such as anorexia, early satiety, indigestion or vomiting; epigastric pain after meals; recent unintentional weight loss.
- *Physical assessment:* general appearance, weight for height; abdominal distension or a palpable upper abdominal mass; occult blood in stool or vomitus.

Nursing diagnoses and interventions

Priorities of nursing care for the person with gastric cancer focus on the effects of the disease and its treatment on nutritional status and on the effects of a potentially fatal disease on the person and their family. See the accompanying nursing care plan for a person with gastric cancer.

Risk of imbalanced nutrition (less than body requirements) manifested by unintentional weight loss

The person with gastric cancer may be malnourished because of anorexia, early satiety and increased metabolic needs related to the tumour. Extensive gastric resection also makes it difficult to consume an adequate diet. Malnourishment, in turn, impairs healing and the person's ability to tolerate cancer treatment.

- Consult with dietitian for a complete nutrition assessment and diet planning. *The person is at risk of protein energy malnutrition, which impairs the ability to heal and recover from extensive surgery.*
- Weigh daily. Monitor laboratory values such as haemoglobin, haematocrit and serum albumin levels. *Daily weights are a valuable measurement of both fluid and nutritional status. Laboratory values provide further evidence of nutritional status.*
- Provide preferred foods; have family prepare meals when possible. Provide supplemental feedings between meals. *Small, frequent feedings and preferred foods encourage intake of nutrients.*

CONSIDERATION FOR PRACTICE

Assess ability to consume adequate nutrients. Nausea and feelings of early satiety may impair nutrient consumption, indicating a need to institute enteral or parenteral feedings.

- Arrange for visitors to be present during meals. Eating is a social function as well as a physiological one. *Companionship often improves food intake.*
- Administer pain and anti-emetic medications as needed before meals. *Pain and nausea suppress the appetite; relief promotes food intake.*

Anticipatory grieving

- Encourage family members to spend as much time as possible with the person. The family may feel helpless and ineffectual. *Supporting family members' presence can encourage this vital interaction.*
- Do not negate denial if present. *Denial is a coping mechanism that protects the person from hopelessness.*
- Allow the person to talk openly if desired about their condition and the prognosis. *Acceptance of the person's fears helps reduce anxiety and promote coping behaviours.*
- Actively listen to the person's and family's expressions of grieving. Avoid interrupting or offering meaningless words of consolation. *Being present and listening actively are often the most effective interventions for the grieving person.*

Community-based care

Although the person with gastric cancer may be hospitalised for surgery, most care is provided in the home and community-based settings such as hospice care. When preparing the person and family for home care, discuss the following topics:

- care of incision and feeding tube (if present) or central venous line
- maintaining nutrition and preventing complications of surgery such as dumping syndrome
- pain management
- provide referrals to home care agencies, hospice and cancer support groups as appropriate
- provide information about services available through the local branch of Cancer Council Australia.

NURSING CARE PLAN A person with gastric cancer

George Harvey is a 61-year-old lawyer who lives with his wife, Harriet. For the last 3 months, Mr Harvey has had increasing anorexia and difficulty eating. He has lost 4.5 kg. His doctor has diagnosed gastric cancer and Mr Harvey is admitted for a partial gastrectomy and gastrojejunostomy. The oncologist has recommended postoperative chemotherapy and radiation. Mr Harvey reports that the doctor told him 'that will give me the best chance for a cure'.

ASSESSMENT

On admission before surgery, Mr Harvey tells his nurse, Lauren Walsh, that he has eaten very little in the past few weeks. He asks, 'What will happen to my wife if something happens to me? I'm afraid this cancer will get me.' Mr Harvey weighs 67 kg and is 183 cm tall. He is pale and thin; his vital signs are BP 148/86, P 92, R 18 and T 36.5°C PO. A firm mass is palpable in the left epigastric region. The rest of his physical assessment data are within normal limits. Mr Harvey's haemoglobin is 12.8 g/dL, haematocrit is 39% and serum albumin level is 3.2 g/dL, indicating that he is mildly malnourished. All other preoperative laboratory and diagnostic studies are within normal limits.

DIAGNOSES

- *Risk of imbalanced nutrition (less than body requirements)* related to anorexia and difficulty eating.
- *Acute pain* related to surgical incision and manipulation of abdominal organs.
- *Risk of ineffective airway clearance related to upper abdominal surgery.*
- *Anticipatory grieving* related to recent diagnosis of cancer.

PLANNING

- Arrange for dietary education, including strategies to prevent dumping syndrome, prior to surgery.
- Encourage Mrs Harvey to visit at mealtimes to assist with and promote oral intake.
- Discuss the grief process and encourage verbalisation of feelings about diagnosis and perceived losses.
- Establish a schedule to cough, deep breathe and use breathing incentive every 2 to 4 hours and as needed. Demonstrate how to splint abdomen during coughing.
- Ensure buzzer is kept within reach at all times and encourage Mr Harvey to alert nursing staff promptly when he is experiencing pain.

Expected outcomes

- Maintain present weight during hospitalisation.
- Resume a high-kilojoule, high-protein diet by the time of discharge.
- Verbalise effective pain management, maintaining a reported pain level of 3 or less on a scale of 1 to 10.
- Maintain a patent airway and clear breath sounds.
- Verbalise feelings regarding diagnosis and participate in decision making.

IMPLEMENTATION

- Weigh daily.
- Maintain nasogastric tube placement, patency and suction as ordered.
- Maintain intravenous fluids and total parenteral nutrition as ordered until oral food intake is resumed.
- Maintain patient-controlled analgesia until able to take oral analgesics.
- Assess respiratory status, including rate, depth and breath sounds, every hour initially, then every 4 hours.
- Encourage participation in decision making.

EVALUATION

Mr Harvey's weight remained stable through his hospitalisation. On discharge he is taking a high-protein, high-kilojoule diet in six small feedings per day. He and his wife have reviewed his diet with the dietitian and are planning on using some dietary supplements at home to meet protein needs. He verbalises an understanding of measures to prevent dumping syndrome, including separating his intake of solid foods and liquids. Mr Harvey is using oral analgesics in the morning and at bedtime to control his pain. He and his wife have begun to discuss the meaning of his diagnosis. Mrs Harvey tells the discharge nurse, 'We are going to go to a support group called "Coping with cancer" when George is stronger.'

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the rationale for maintaining nasogastric suction after gastrojejunostomy?
2. Develop a preoperative teaching plan for a person undergoing a partial gastrectomy.
3. Design interventions to ensure adequate nutrition for people with advanced gastric cancer.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Mr Harvey calls you just before the initial dose of chemotherapy and says, 'Everyone tells me that chemotherapy will cause vomiting and I don't think I can take being sick again.' Reflect on how you would respond.

CHAPTER HIGHLIGHTS

- Nausea and vomiting, common GI symptoms, may be indicative of disorders affecting many organ systems, including the GI tract, inner ear, CNS or heart. Complications such as dehydration, electrolyte imbalance and aspiration of gastric contents are primary concerns in treating nausea and vomiting.
- Stomatitis and oral mucositis are common disorders of the mouth, potentially having a significant effect on comfort and nutrition.
- Tobacco and alcohol use contribute to a number of upper GI disorders, including GORD, oral and oesophageal cancers, and peptic ulcer disease. Encourage all people to stop smoking or using smokeless tobacco and to reduce alcohol to moderate amounts, if at all, to reduce their risk of developing these disorders.
- Gastro-oesophageal reflux disease (GORD) is common. While it often is considered to be a benign condition, prolonged exposure of the lower oesophagus to gastric juices can lead to oesophagitis, haemorrhage and scarring.
- Both oesophageal and gastric cancer are often diagnosed late in the disease because their symptoms may be vague. Encourage people with complaints of dysphagia, a sensation of gastric fullness or heartburn to seek medical evaluation. Surgical resection of the cancerous portion of the oesophagus or stomach is the treatment of choice when the tumour is diagnosed early.
- Upper gastrointestinal bleeding can lead to significant blood loss and shock. Peptic ulcer disease accounts for the majority of UGI haemorrhage, although erosive gastritis and oesophageal varices also are common causes.
- Acute gastritis, often associated with aspirin or NSAID use, is generally benign and self-limited. Erosive gastritis, a complication of critical conditions such as shock, trauma, a major burn or head injury, can lead to unexpected gastric haemorrhage. Chronic gastritis is an unrelated disorder associated with *H. pylori* infection.
- *H. pylori* infection also is a major risk factor for peptic ulcer disease and gastric cancer. Effectively treating the infection can reduce or eliminate the risk of future exacerbations of PUD.
- An acute change in the nature of abdominal pain in a person with PUD, especially when accompanied by vomiting, guarding of the abdomen or a change in bowel sounds, could indicate an obstruction or perforation and release of gastric contents into the peritoneal cavity.

CONCEPT CHECK

1 The nurse assessing for oral cancer risk factors in a person with a persistent sore on his tongue asks about:
1. consumption of highly spiced food
2. thumb sucking or pacifier use as a child
3. regular use of dental floss
4. tobacco use in any form

2 The nurse teaching a person with gastro-oesophageal reflux disease includes which of the following instructions? (Select all that apply.)
1. This is a benign disease requiring no treatment.
2. Elevate the head of the bed.
3. Stop taking the prescribed proton-pump inhibitor once symptoms are relieved.
4. Peppermint and chocolate lollies can help relieve symptoms.
5. Avoid lying down for several hours after eating.

3 The nurse evaluates his teaching of a person with acute stress gastritis as effective when the person states that she will:
1. avoid using aspirin or NSAIDs for routine pain relief
2. consume only bland foods
3. return for yearly upper endoscopy exams
4. fully cook all meat, poultry and egg products

4 The nurse identifies which of the following nursing diagnoses as highest priority for the person admitted with peptic ulcer disease and possible perforation?
1. *Acute pain*
2. *Ineffective health maintenance*
3. *Nausea*
4. *Impaired gastrointestinal tissue integrity*

5 Following a partial gastrectomy for gastric cancer, the person complains of nausea, abdominal pain and cramping, and diarrhoea after eating. Recognising manifestations of dumping syndrome, the nurse recommends:
1. fasting for a period of 6 to 12 hours before meals
2. decreasing the protein content of meals
3. frequent small meals that contain solid foods or liquids, but not both
4. a diet rich in carbohydrates to maintain blood glucose levels

6 The nurse caring for a person with oesophageal cancer affecting the middle portion of the oesophagus would immediately report which of the following?
1. crackles in the base of the right lung
2. bright bleeding from the mouth
3. weight loss
4. difficulty swallowing solid foods

7 The physician has ordered omeprazole 20 mg twice daily, clarithromycin 500 mg twice daily and amoxicillin 1 g daily for a person with peptic ulcer disease. It is most important for the nurse to instruct the person to:
1. stop the drugs immediately and notify the physician if a rash, hives or itching develop
2. consume yoghurt daily while taking these drugs
3. take the drugs on an empty stomach, 1 hour before breakfast and at least 2 hours after dinner
4. take the drugs with a full glass of water

8 When planning care for a person with stomatitis, the nurse identifies which of the following as a priority intervention?
1. Assist to cleanse mouth with mouthwash following meals.
2. Allow the person to select appealing foods from a menu.
3. Provide viscous lignocaine to relieve mouth pain before meals.
4. Refer the person to a smoking cessation program.

9 The evening following a gastric resection, the nurse notes that there has been no drainage from the nasogastric tube for the past 3 hours. The nurse should:
1 chart the finding
2 reposition the nasogastric tube
3 gently irrigate the tube with normal saline
4 notify the surgeon

10 A person with a history of peptic ulcer disease suddenly begins to complain of severe abdominal pain. The nurse should: (Select all that apply.)
1 administer the prescribed proton-pump inhibitor
2 obtain an order for a narcotic analgesic
3 withhold oral food and fluids
4 place the person in Fowler's position
5 notify the physician

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CHAPTER 23

NURSING CARE OF PEOPLE WITH BOWEL DISORDERS

ELSPETH HILLMAN

LEARNING OUTCOMES

- Explain the pathophysiology, manifestations, interprofessional and nursing care of people with disorders of intestinal motility.
- Explain the pathophysiology, complications and interprofessional care of people with acute inflammatory or infectious bowel disorders.
- Compare and contrast the pathophysiology, manifestations and complications of chronic inflammatory bowel disorders.
- Discuss the risk factors, pathophysiology and interprofessional care associated with neoplastic bowel disorders.
- Discuss the pathophysiology, manifestations and nursing care of people suffering from structural and obstructive bowel disorders.
- Describe the pathophysiology, manifestations and nursing care of a person suffering an anorectal disorder.

CLINICAL COMPETENCIES

- Assess the functional status of a person with bowel disorders and monitor, document and report abnormal manifestations.
- Use evidence-based research to prevent aspiration in critically ill individuals with enteral feedings and to make accurate assessments of faecal incontinence in older adults.
- Determine priority nursing diagnoses based on assessed data and select and implement individualised nursing interventions for a person with bowel disorders.
- Administer medications used in the management of bowel disorders knowledgeably and safely.
- Provide skilled care to a person following the formation of an ileostomy or colostomy, or perianal surgery.
- Integrate interprofessional care into care of a person with bowel disorders.
- Provide appropriate teaching to promote nutrition, prevent infectious and helminth infestations, encourage preventive screening for colon cancer and facilitate community-based care for healthcare needs resulting from bowel disorders.
- Revise plan of care when necessary to provide effective interventions promoting, maintaining or restoring functional health status to a person with a bowel disorder.

KEY TERMS

Disorders of intestinal absorption and bowel elimination do not only affect functional elimination status. Other functional health patterns affected include, but are not limited to, health perception–health management, nutritional–metabolic, activity–exercise, self-perception–self-concept and sexuality–reproductive. Bowel function is affected by inflammations, infections, tumours, obstructions or changes in bowel structure.

A person with an intestinal disorder often faces extensive diagnostic testing, surgery and permanent changes in their physical appearance and lifestyle. Nursing care is directed towards meeting the person's physiological needs, providing emotional support and assisting the person's adaption to lifestyle changes.

DISORDERS OF INTESTINAL MOTILITY

Few body functions respond as readily to internal and external influences as defecation. Factors directly affecting the gastrointestinal (GI) tract include food intake and bacterial population which affect the number and consistency of stools. Indirect factors also affect elimination. Consider the effects psychological stress or voluntary postponement of defecation have on elimination.

'Normal' bowel elimination patterns vary widely. For some people, two to three stools per day is their usual pattern, whereas for other people their usual pattern is three stools per week. It is important to evaluate each person's bowel elimination against their normal pattern.

THE PERSON WITH DIARRHOEA

Diarrhoea is an increase in the frequency, volume and fluid content of the stool. In diarrhoea, the water content of faeces is increased, usually due to either malabsorption or water secretion in the bowel. It is a clinical manifestation, rather than the primary disorder.

Diarrhoea may be acute or chronic. Acute diarrhoea (lasting less than a week) is usually due to an infectious agent. Chronic diarrhoea (persisting longer than 3 to 4 weeks) may be caused by inflammatory bowel disorders, malabsorption or endocrine disorders.

Pathophysiology

Approximately 1500 mL of digested material enters the large intestine daily. Normally, most of the water and some solutes are reabsorbed in the bowel, leaving approximately 200 mL of faeces to be eliminated.

Large-volume diarrhoea is characterised by both increased numbers and volume of stools caused by increased water content of the stool. This increased water content results from either osmotic or secretory processes. Water is pulled into the bowel lumen by osmosis when the faeces contain osmotically active molecules. Some stool softeners and laxatives work on this principle. When lactose in milk is not broken down and absorbed, the lactose molecules exert an osmotic pull, causing diarrhoea. The diarrhoea associated with cholera and *Escherichia coli* infection is caused by increased water secretion in the small and large intestines. Unabsorbed dietary fat, some laxatives and drugs cause secretory diarrhoea.

Small-volume diarrhoea, characterised by frequent small stools, is usually caused by inflammation or disease of the colon. Diseases affecting the intestinal mucosa—for example, inflammatory bowel disease—cause exudative diarrhoea. Mucosal inflammation causes plasma, serum proteins, blood and mucus to accumulate in the bowel, increasing faecal bulk and fluidity. An increased propulsion rate within the bowel decreases the amount of water normally absorbed from chyme, leading to diarrhoea. For this reason, laxatives increasing bowel motility and bowel resection or bypass can lead to diarrhoea.

Antibiotic-associated diarrhoea occurs as a result of disruption of normal intestinal flora by antibiotic therapy. Loss of normal flora affects digestion of food leading to diarrhoea, or allows an overgrowth of pathogens (e.g. *Clostridium difficile* (*C. difficile*)). The accompanying 'Translation to practice' box provides information about other causes of diarrhoea in hospitalised people.

Manifestations

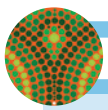
Clinical manifestations of diarrhoea depend on the cause, duration, severity and area of bowel affected, as well as a person's age and general health. Diarrhoea presents as several large, watery stools daily or very frequent small stools containing blood, mucus or exudates.

Complications

Diarrhoea can have devastating effects. Water and electrolytes are lost in diarrhoeal stools, leading to dehydration, particularly in the very young, older adults or debilitated individuals unable to respond to thirst. With severe diarrhoea, vascular collapse and hypovolaemic shock may occur. Potassium and magnesium are lost, potentially leading to hypokalaemia and hypomagnesaemia. The loss of bicarbonate in the stool can lead to metabolic acidosis. See Chapter 9 for further discussion of the effects of fluid and electrolyte imbalances.

INTERPROFESSIONAL CARE

Management of diarrhoea focuses on identifying and treating the underlying cause. Additionally, the diarrhoea itself needs to be treated, comfort promoted and complications prevented. A health history (including the onset and associated circumstances of the diarrhoea) and physical examination often provide enough information to identify its cause. However, precise diagnosis is only achieved with laboratory investigations.



TRANSLATIONTO PRACTICE Evidence-based practice: diarrhoea

People who have been hospitalised often have a number of risk factors for diarrhoea. *Clostridium difficile* is now recognised as the cause of a significant portion of treatment-related diarrhoea; it does not, however, account for all cases. Previous studies have demonstrated a relationship between diarrhoea and enteral tube feedings, medications containing sorbitol, lactose intolerance and other factors. A review by Chang and Huang (2013) looked at the risk of developing diarrhoea during enteral feeding. Findings illustrate the contributory effects of medications, severe illness, infection and and enteral tube feeding.

IMPLICATIONS FOR NURSING

People who are severely ill often require enteral tube feedings for nutritional support. Healing and immune function require adequate nutrition; other studies point to the beneficial effects of enteral nutrition for the majority of people. Discontinuing enteral feedings due to diarrhoea is not a desirable option. In some cases, changing the enteral feeding formula or adding probiotic (cultures of beneficial yeasts or bacteria) supplements may help normalise bowel function. Other options include antidiarrhoeal medications or soluble fibre supplements.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Administering medications containing sorbitol, a sugar that is not absorbed by the gut, is associated with an increased risk of developing diarrhoea among people who have been hospitalised. How does sorbitol increase the risk of diarrhoea? What would you do if you realised that a seriously ill person you are caring for was receiving enteral tube feeding and medications containing sorbitol?
2. The position of the feeding tube tip in the gut is also identified as a risk factor for diarrhoea associated with enteral tube feedings. Thinking about the functions of the stomach, pyloric valve, duodenum and jejunum, which type of tube (gastric, duodenal or jejunal) might carry the highest risk of diarrhoea? The lowest? What other enteral tube feeding risk factors are considered in tube placement?
3. A person is being discharged home with a gastrostomy tube and directions for enteral feedings. What teaching will you provide to the person and their family regarding tube care, feeding administration and bowel management?

Diagnosis

Diagnostic tests ordered to help identify the cause of diarrhoea may include a stool specimen analysis, culture and sensitivity. A sigmoidoscopy to visualise the bowel mucosa may be conducted. (See Chapter 20 for further information on diagnostic tests.) Tissue biopsy to identify chronic inflammatory processes, infection and other causes of diarrhoea may be obtained. Recently developed and improved diagnostic methods include enzyme immunoassays for faecal antigens and reverse transcriptase-polymerase chain reaction (RT-PCR) to identify some viruses (e.g. norovirus), and ELISA and the fluorescein-labelled antibody test to detect antigens or oocysts of some protozoa (e.g. *Giardia* and *Cryptosporidium*) (Lee & Bishop, 2013). Additionally, serum electrolytes, serum osmolality and arterial blood gases (ABGs) are collected to assess for adverse effects of diarrhoea. Increased serum osmolality indicates water loss and dehydration.

Medications

Antidiarrhoeal medications are used sparingly or not at all until the cause of diarrhoea is identified. In diarrhoea associated with botulism or bacillary dysentery, giving an antidiarrhoeal agent worsens or prolongs the infection by slowing toxin elimination from the bowel. Once the underlying cause for diarrhoea is established, specific medications, if appropriate, are ordered to treat the underlying cause. Antibiotics are used cautiously as these alter the bowel's normal bacterial population and may actually increase diarrhoea. A balanced electrolyte solution may be required to replace fluid and electrolyte losses. Intravenous or oral potassium preparations may also be prescribed.

Opium and some of its derivatives, anticholinergics, absorbents and demulcents are commonly used as antidiarrhoeal preparations. Specific preparations, their method of action and nursing implications for these medications are outlined in the following 'Medication administration' box.

Nutrition

Fluid and electrolyte replacement is of primary importance in managing a person with diarrhoea. If the person is tolerating oral fluids (i.e. the person is not experiencing nausea and vomiting), an oral glucose/balanced electrolyte solution provides the best fluid replacement. Several commercial preparations (e.g. Gastrolyte) are available, as are paediatric solutions which can be used for adults as well as children.

During acute diarrhoea, the person's diet should be modified to rest the bowel. During the first 24 hours, solid food should be withheld. After this time, frequent, small amounts of starchy foods can be added. Milk and milk products are added last, as these contain lactose which frequently aggravates the diarrhoea. Raw fruit and vegetables, fried foods, bran, wholegrain cereals, condiments, spices, coffee and alcoholic beverages are avoided during the recovery period as the bowel has difficulty processing these complex materials.

People with chronic diarrhoea may benefit by eliminating specific foods from their diet. Foods and non-food substances aggravating diarrhoea are outlined in Table 23.1. The diet should be high in kilojoules and nutritional value. Vitamin supplements may be necessary, particularly the fat-soluble vitamins (A, D, E, and K). Occasionally, people with severe chronic diarrhoea require parenteral nutrition (see Chapter 21).

TABLE 23.1 Foods aggravating chronic diarrhoea

FOODS	REASON
Milk, ice-cream, yoghurt, soft cheeses, cottage cheese	Contain lactose; not tolerated by people with lactase deficiency unable to digest lactose.
Apple juice, pear juice, grapes, honey, dates, nuts, figs, fruit-flavoured soft drinks	Contain fructose; when consumed in large quantities, fructose may not be totally absorbed, causing an osmotic pull of fluid into the bowel.
Table sugar	Contains sucrose; not tolerated by people with sucrase deficiency.
Sugarless gums and mints	May contain sorbitol or mannitol, sugars that are not absorbed causing an osmotic draw.
Antacids	Magnesium-containing antacids decrease bowel transit time and contain poorly absorbed salts exerting an osmotic draw.
Coffee, tea, cola drinks, over-the-counter analgesics containing codeine	Contain caffeine, decreasing bowel transit time.

MEDICATION ADMINISTRATION Antidiarrhoeal preparations

ABSORBENTS AND PROTECTANTS

Kaolin and pectin (Kaopectate, Donnagel-MB)
Polycarbophil (FigerNorm, Equalactin)

Absorbent preparations act locally in the intestines to bind substances that can cause diarrhoea. Absorbents are safe and are generally available over the counter, although their efficacy has not been proved.

Nursing responsibilities

- Assess for contraindications to antidiarrhoeal therapy, such as some infections or chronic inflammatory bowel disease, including ulcerative colitis.
- If fever is present, check with doctor before giving the medication.
- Administer these medications at least 1 hour before or 2 hours after other oral medications; these may interfere with the absorption of other medications.
- Observe the person's response to the medication. Constipation is a potential problem.

Health education for the person and family

- Take the recommended dosage at the onset of diarrhoea and after each loose stool.
- Do not take any of these preparations for more than 48 hours. If diarrhoea persists, notify the doctor.
- Do not give antidiarrhoeal medications to debilitated older adults without medical supervision.

ANTISECRETORY

Bismuth subsalicylate (Pepto-Bismol)

Bismuth subsalicylate, available without a prescription, has antisecretory, anti-inflammatory and antibacterial effects. It is widely used to control traveller's diarrhoea. Although it is generally safe at recommended doses, bismuth subsalicylate has potential toxic effects and interacts with medications such as aspirin and oral anticoagulants.

Nursing responsibilities

- Administer as ordered.
- Do not administer within 1 hour of other medications, as it may interfere with their absorption.
- Monitor for increased anticoagulant effect when given with warfarin or aspirin.

Health education for the person and family

- Chew bismuth subsalicylate tablets, rather than swallowing these whole, for maximal effectiveness.
- This medication may cause harmless darkening of your tongue and stools.
- If you are allergic to aspirin, use bismuth subsalicylate with caution. Do not use aspirin while you are taking this medication unless directed to do so by your doctor. Contact your doctor if diarrhoea persists for more than 2 days.

OPIUM AND OPIUM DERIVATIVES

Camphorated tincture of opium (Paregoric)
Tincture of opium (laudanum, opium tincture)
Difenoxin (Motofen)
Diphenoxylate (Lomotil, Lotrol, others)
Loperamide hydrochloride (Imodium)

Opium and its derivatives act on the central nervous system (CNS) decreasing the motility of the ileum and colon, slowing transit time and promoting more water absorption. These medications also decrease the sensation of a full rectum and increase anal sphincter tone.

Difenoxin, diphenoxylate and loperamide hydrochloride are synthetic opioids chemically related to pethidine (Bullock & Manias, 2014). However, they have minimal analgesic, euphoric or abuse-promoting effects and are in more common use today.

Nursing responsibilities

- Assess for contraindications to antidiarrhoeal or narcotic medications prior to giving these drugs.
- Administer paregoric undiluted with water.
- Do not administer difenoxin and diphenoxylate to a person receiving monoamine oxidase inhibitors (MAOIs); hypertensive crises may occur.
- Observe closely for increased effects of other CNS depressants, such as alcohol, narcotic analgesics or barbiturate sedatives.
- Observe for abdominal distension; toxic megacolon may occur if these medications are given to a person with ulcerative colitis.

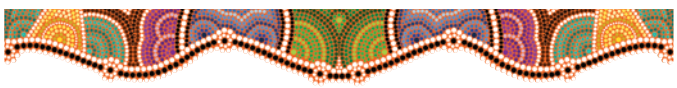
Health education for the person and family

- Take the medication as recommended at the onset of diarrhoea and after each loose stool.
- These medications may be habit forming; use for no more than 48 hours.
- Avoid using alcohol and over-the-counter cold preparations while taking these medications.
- These preparations may cause drowsiness; avoid driving or operating machinery while taking them.

Complementary and alternative therapies

Herbal or homeopathic therapies may be used to help relieve diarrhoea. People with lactose intolerance may use lactase enzymes tablets or drops when consuming milk products. Herbal treatments include a strong tea of black pepper, chamomile, coriander, rosemary, sandalwood or thyme. Ginger tea or capsules are helpful in reducing intestinal inflammation and decreasing the effects of food poisoning.

Probiotics, live microogranisms similar to those normally found in the gut, may be used to prevent or treat antibiotic-associated diarrhoea (Campbell, 2014; Reintam Blaser, Deane & Fruhwald, 2015). Probiotics are available as dietary supplements and food (e.g. yoghurt, yoghurt drinks). The person should consult a qualified medical or homeopathic practitioner when choosing to manage their diarrhoea with complementary and alternative therapies.



Nursing care

Health promotion

Prevention of diarrhoeal diseases essentially involves avoiding infectious agents (Lee & Bishop, 2013). Educating individuals and their families about the importance of handwashing is a primary measure to prevent and reduce the spread of infectious diseases, including those causing diarrhoea. Educating people about safe food handling techniques prevents bacterial contamination. Discuss measures to ensure safe drinking water. For people planning travel to remote areas or outside Australia, discuss the importance of avoiding the consumption of high-risk foods (especially raw foods) and beverages, and purification methods for drinking and cooking water.

Assessment

The nursing assessment helps identify the cause of the person's diarrhoea, as well as early signs of complications. Collect the following assessment data:

- *Health history:* duration and extent of diarrhoea; associated manifestations; dietary intake; recent visits to remote areas or overseas travel or contact with people recently returned from overseas; previous history of diarrhoea; chronic diseases; prescription and non-prescription medications including complementary supplements.
- *Physical examination:* vital signs (including orthostatic blood pressure); peripheral pulses and capillary refill; skin temperature, moisture and turgor; colour and moisture of mucous membranes; abdominal contour and girth; bowel sounds; stool for obvious or occult blood, pus, mucus or steatorrhoea (bulky, foul-smelling stool).

Nursing diagnoses and interventions

Nursing care of people with diarrhoea focuses on identifying the cause, relieving the clinical manifestations and preventing complications and spread of infection to others.

Diarrhoea

Nursing interventions for diarrhoea are provided to assist a person recover their normal elimination pattern without adverse consequences.

- Use standard precautions, including gloves and handwashing. *Standard precautions help prevent the spread of infection to others.*
- Monitor and record the frequency and characteristics of bowel movements *to provide a measure of the effectiveness of treatment.*
- Measure abdominal girth and auscultate bowel sounds every 8 hours as indicated. *Loud, rushing bowel sounds (borborygmi) indicate increased peristalsis and may be heard in a person with acute diarrhoea. Diminished or absent bowel sounds may indicate a complication of treatment, such as constipation or toxic megacolon.*
- Provide ready access to toilet, commode or bedpan. *The person may have little warning of the need to defecate. Easily accessed toileting facilities reduce the risk of soiling or injury.*
- Administer antimuscarinic medications as prescribed *to promote comfort by reducing colicky pain sometimes associated with diarrhoea.*
- Limit food intake if the diarrhoea is acute, reintroducing solid foods slowly, in small amounts, *allowing the bowel to rest and mucosa to heal in acute diarrhoea states.*

Risk of deficient fluid volume

The increased water content of diarrhoeal stool places the person at risk of fluid deficit.

- Record intake and output; weigh daily; assess skin turgor, mucous membranes and urine specific gravity every 8 hours. *These assessments are used to monitor fluid volume status.*

CONSIDERATION FOR PRACTICE

Assess skin turgor over the sternum of older adults. Loss of subcutaneous fat associated with ageing makes assessment of skin turgor on the arms or hands less reliable.

- Monitor vital signs, including orthostatic blood pressures. *A mild fluid volume deficit results in orthostatic hypotension. This is identified by a 10 mmHg or more drop in BP and increase of 10 beats per minute (bpm) in pulse when changing from a lying to a sitting position or from a sitting to a standing position.*

CONSIDERATION FOR PRACTICE

Remember to institute safety precautions when assisting a person with orthostatic hypotension to ambulate. *The decrease in blood pressure with position changes can cause light headedness and syncope.*

- Provide fluid and electrolyte replacement solutions as indicated. Ensure ready access to fluids; assist a debilitated person with fluid intake. Notify the doctor if the person is unable to tolerate oral fluids. *Oral fluids are encouraged as tolerated to prevent dehydration. Intravenous fluids are necessary when oral fluids are not tolerated. An intake of 3000 mL/day or more is often needed to replace fluid losses.*

Risk of impaired skin integrity

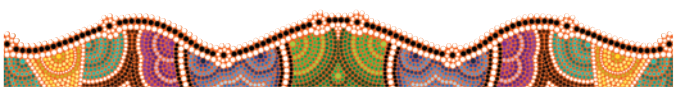
Decreased extracellular fluid volume and the irritating effects of diarrhoeal stool increase the risk of skin breakdown.

- Assist with cleaning the perianal area as needed. Use warm water, a gentle cleanser and soft cloths. *Cleansing removes irritating substances in the stool. Gentle cleansing helps maintain integrity of dehydrated skin.*
- Apply protective ointment to the perianal area. *Moisture-barrier ointments or creams protect the skin from excoriation and help prevent tissue breakdown.*

Community-based care

Acute and chronic diarrhoea generally are managed by the person at home. Teach the individual and their family members about the following topics:

- cause of diarrhoea (as directed by the diagnosis)
- importance of handwashing and hygiene measures
- importance of maintaining adequate fluid intake to replace lost water and electrolytes
- use of a balanced electrolyte solution (e.g. Gastrolyte) or a similar product (purchased) for electrolyte replacement
- recommendations to limit food intake during acute diarrhoea and resume food gradually with small meals of foods with a constipating effect: apple sauce, bananas, water crackers, rice and potatoes
- avoiding foods high in fibre, milk products and caffeine
- ways to maintain nutrition if chronic diarrhoea is a problem: frequent small meals, nutritional supplements, vitamin supplements
- precautions and limitations of antidiarrhoeal preparations
- importance of seeking medical intervention if diarrhoea continues or recurs.



THE PERSON WITH CONSTIPATION

Constipation is defined as the infrequent (less than two bowel movements weekly) or difficult passage of stools. Constipation affects older adults more frequently than younger people. A recent Australian study reported that constipation in community-dwelling older adults had a prevelance between 11% and 55% (Werth, Williams & Pont, 2015). Although faecal transit in the large intestine slows with ageing, the increased incidence of constipation is thought to relate more to impaired general health, increased medication use and decreased physical activity in the older adult than to being part of the ageing process.

Pathophysiology

Constipation may be a primary problem or a manifestation of another disease or condition. Acute constipation, a definite change in a person's bowel elimination pattern, is often caused by an organic process. A change in bowel patterns persisting or becoming more frequent or severe may be due to a tumour or other partial bowel obstruction. However, in chronic constipation functional causes impairing storage, transport and evacuation mechanisms impede the normal passage of stools. Common causes of constipation are listed in Table 23.2.

Psychogenic factors are the most frequent causes of chronic constipation. These factors include postponing defecation when the urge is felt and the perception of satisfaction with defecation. Some people use laxatives and enemas to stimulate a bowel movement when constipation is perceived. Overuse of these leads to intestinal problems, worsening constipation. *Cathartic colon* (impaired colonic motility and changes in bowel structure) mimics ulcerative colitis in that the normal pouchlike or saccular appearance of the colon is lost. *Melanosis coli* is a brownish-black discolouration of the colon mucosa. Both conditions may be caused by long-term laxative use.

Manifestations

Clinical manifestations of constipation include having bowel movements less often than the usual pattern, frequent flatus, abdominal discomfort, anorexia, straining to achieve bowel movement and the passage of hard, dry stools.

Faecal impaction may develop from significant constipation or long-term dependence on laxatives or enemas. Impaction occasionally results after barium administration for radiological exam. The impaction is felt as a rock-hard or putty-like mass of faeces in the rectum. Faecal impaction may

TABLE 23.2 Selected causes of constipation

FACTOR	RELATED CAUSE
Activity	Lack of exercise; bed rest
Dietary	Highly refined, low-fibre foods; inadequate fluid intake
Medications	Antacids containing aluminum or calcium salts; narcotic analgesics; anticholinergics; many antidepressants, tranquillisers and sedatives; antihypertensives, such as ganglionic blockers, calcium channel blockers, beta-adrenergic blockers, diuretics; iron supplements
Large bowel	Diverticular disease, inflammatory disease, tumour, obstruction; changes in rectal or anal structure or function
Psychogenic	Voluntary suppression of urge; perceived need to defecate on schedule; depression
Systemic	Advanced age; pregnancy; neurological conditions (trauma, multiple sclerosis, tumours, cerebrovascular accident, parkinsonism); endocrine and metabolic disorders (hypothyroidism, hypocalcaemia, uraemia, porphyria)
Other	Chronic laxative or enema use

result in abdominal cramping and a full sensation in the rectal area. Additionally, watery mucus or foul-smelling liquid stool may be passed around the impaction, causing the person to report diarrhoea.

INTERPROFESSIONAL CARE

Initial evaluation of constipation is based on the health history and physical examination. The abdomen may appear distended and bowel sounds may be reduced. If faecal impaction is present, digital examination of the rectum reveals a palpable, hard or putty-like faecal mass.

Simple or chronic constipation is treated with education (a daily bowel movement is not necessary for health), modification of diet and increasing fluid intake and exercise routines. If the problem is acute or does not resolve, further diagnostic examination is ordered.

Diagnosis

A barium enema is ordered to identify bowel structure, tumours or diverticula. If the problem is acute, a sigmoidoscopy or colonoscopy may be used for evaluation and biopsy. (See Chapter 20 for nursing implications of these tests.)

Medications

Laxatives—occasionally called aperients or purgative preparations—are used to promote stool evacuation. Laxatives are grouped in categories depending on their mechanism of action. These include osmotic laxatives (non-absorbable inorganic salts, polyethylene glycol, sugars and alcohol), stimulant laxatives, faecal softeners, lubricants and bulk-forming laxatives (Bullock & Manias, 2014). Laxatives are indicated for constipation resulting from poor bowel habits, opioid analgesia, medications with anticholinergic side effects and loss of intestinal muscle tone following surgery or bed rest or due to age. Laxatives are often used to reduce straining, reduce pain associated with anorectal disorders and before surgery or diagnostic procedures.

Stimulant laxatives and enemas interfere with normal bowel reflexes and are not recommended for regular use as these may lead to colon dilation and reduced peristalsis, requiring increased amounts. All laxatives are contraindicated if a person has an intestinal obstruction, undiagnosed abdominal pain, nausea or vomiting, paralytic ileus, suspected appendicitis, undiagnosed rectal bleeding, faecal impaction, rectal fissures, ulcerated haemorrhoids, Crohn's disease, ulcerative colitis or chronic inflammatory bowel disease. When the bowel is obstructed, laxatives may cause serious mechanical damage and perforate the bowel.

If a person is unable to consume enough dietary fibre to prevent constipation, the only appropriate laxatives safe for long-term use are bulking agents, such as psyllium seed (e.g. Metamucil), *Plantago ovata* seeds (ispaghula or psyllium husk—e.g. Fybogel) and sterculia and frangula (e.g. Normacol Plus). These agents act by increasing the bulk of the faeces and drawing water into the bowel, softening it. Some bulking agents (e.g. Fybogel and Normafibre) contain aspartame. Therefore, caution is needed if given to a person with phenylketonuria (Tiziani, 2013). Commonly prescribed laxatives are discussed in the 'Medication administration' box below.

Nutrition

Foods with high fibre content are recommended. Vegetable fibre is largely indigestible and unabsorbable, so it increases stool bulk. Fibre also helps draw water into the faecal mass, softening the stool and making defecation easier. Raw fruits and vegetables are good sources of dietary fibre, as is cereal bran. Use 2 to 3 teaspoons of unprocessed bran with meals (sprinkled on fruit or cereal) or up to ¼ cup daily to supply adequate fibre.

Fluids are also important to maintain bowel motility and soft stools. If not contraindicated a person should drink 6 to 8 glasses (i.e. 250 mL each) of fluid per day. It is important to advise the person to increase fluid intake when dietary fibre is initially increased to decrease flatus and help maintain softer stools.

In older adults, constipation may be due to inadequate food and fluid intake. Carefully evaluate the person's diet history and usual daily fluid intake.

Enemas

Significant or chronic constipation or a faecal impaction may require the administration of an enema. As a general rule, enemas should be used only in acute situations and only on a short-term basis. These may also be ordered to prepare the bowel for diagnostic testing or examination. Enema administration is contraindicated in a person with diarrhoea, cardiac dysrhythmias, following recent myocardical infarction, undiagnosed abdominal pains and following recent surgery to the rectum, bowel or prostate gland (Long, 2015).

The following types of enemas may be prescribed:

- *Isotonic—saline enema* using 500 to 1000 mL of warmed normal saline solution is the least irritating to the bowel. It distends the colon, stimulates peristalsis and softens faeces. The enema solution should be retained for 5 to 10 minutes as tolerated. Evacuation usually occurs within approximately 15 to 20 minutes. Sodium retention is a possible adverse effect of these enemas.
- *Hypotonic—tap-water enemas* use 500 to 1000 mL of water to soften faeces and irritate the bowel mucosa, stimulating peristalsis. The enema solution should be retained for 5 to 10 minutes as tolerated. Evacuation is within approximately 15 to 20 minutes. Adverse effects include fluid and electrolyte imbalance and water intoxication.
- *Soap-suds enemas*—consist of a tap-water solution (500 to 1000 mL) to which soap (3 to 5 mL) is added to distend the colon, stimulate peristalsis and soften faeces. The enema solution should be retained for 5 to 10 minutes as tolerated. Evacuation is usually within 10 to 15 minutes. Adverse effects include irritation and damage to mucosa.
- *Hypertonic—phosphate enemas* (e.g. Fleet) use a hypertonic saline (sodium phosphate) solution (90 to 120 mL) to draw fluid into the bowel and irritate the mucosa, leading to evacuation in approximately 5 to 10 minutes. Adverse effect is possible sodium retention.

MEDICATION ADMINISTRATION Laxatives

BULK-FORMING AGENTS

Bran
Psyllium (Metamucil)
Ispaghula (Fybogel)
Sterculia (Normafibe)

Bulk-forming agents are the only safe laxatives for long-term use. These contain vegetable fibre, which is not digested or absorbed in the gut. This natural fibre creates bulk and draws water into the intestine, softening the stool mass, resulting in increased peristalsis (see Figure 23.1).

Nursing responsibilities

- Mix the agent (e.g. one sachet of Fybogel or 1½ to 3 teaspoons of Metamucil one to three times per day) in a 250 mL glass of cool liquid just prior to administering and followed by an additional glass of water. Sterculia (Normafibe) granules (1 to 2 heaped 5 mL teaspoons of granules one or two times per day) can be placed dry on the tongue and swallowed whole with 250 mL of water.
- Do not administer to a person with possible stool impaction or bowel obstruction, or who is dehydrated or on fluid restrictions—for example, a person with chronic kidney disease.

Health education for the person and family

- Drink at least 6 to 8 full glasses (250 mL) of non-alcoholic fluid per day. Adequate hydration is necessary to produce the medication's laxative effect.
- These agents may be mixed with water, milk or fruit juice. Sterculia (Normafibe) granules can be mixed with jam, honey or ice-cream.
- Take the medication in the morning or with meals. To reduce the risk of impaction, do not take at bedtime.
- Because of the increased risk of impaction, check with the doctor before increasing dietary fibre while you are taking these medications.

FAECAL SOFTENERS

Docusate sodium (Coloxyl)
Paraffin liquid (Agarol)

Faecal softeners reduce stool surface tension and form an emulsion of fat and water, softening the stool. These are used primarily to prevent straining and reduce the discomfort of expelling hard stools after rectal and perianal surgery. These medications have limited use in the management of acute or chronic constipation (Bullock & Manias, 2014).

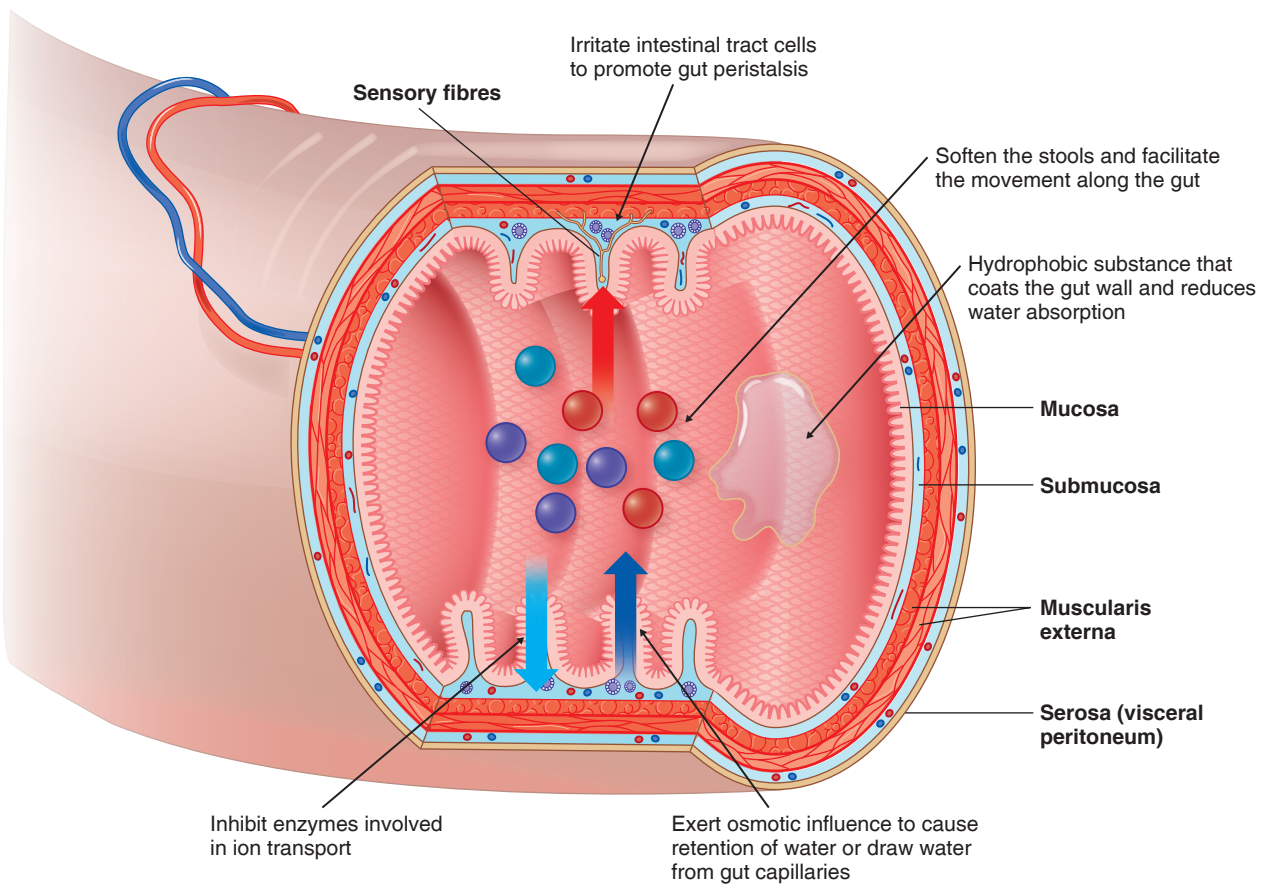


FIGURE 23.1 ■ Mechanisms of laxative action

Source: Fundamentals of Pharmacology (7th ed.) by S. Bullock & E. Manias (2014), p. 715, Figure 57.1. Frenchs Forest, NSW: Pearson Australia.

MEDICATION ADMINISTRATION Laxatives (continued)

Nursing responsibilities

- Administer with ample fluids to promote softening effect.
- Faecal softening agents may alter the absorption of other drugs. Do not administer within 1 to 2 hours of other oral medications.
- Do not attempt to crush or open capsules; a liquid form is available for a person unable to swallow pills or capsules.
- Caution needs to be taken with the administration of docusate sodium (Coloxyl) tablets in a person with hypertension or congestive cardiac failure, due to the sodium content.

Health education for the person and family

- Do not use for more than 1 week unless specifically recommended by the doctor.
- Take the medication in the morning or evening; remember to avoid taking it with other medications.
- Adequate fluid is necessary to obtain the beneficial effect of the medication. Drink 6 to 8 (250 mL) glasses of non-alcoholic fluid per day.

OSMOTIC AND SALINE LAXATIVES/CATHARTICS

Lactulose (Duphalac)
Sorbitol (Sorbilax)
Magnesium salts (Epsom salts)
Sodium phosphate (Fleet phospho-soda buffered saline mixture)
Polyethylene glycol-electrolyte solution (ColonLYTELY)

Laxatives in this group contain poorly absorbed salts or carbohydrates which remain in the bowel, increasing osmotic pressure and drawing water into the intestine. Stool volume increases, consistency decreases and peristalsis is stimulated. Many of these agents also have an irritant effect on the bowel, further stimulating peristalsis. They are used to stimulate rapid or complete bowel evacuation to relieve constipation. Sodium phosphate (Fleet phospho-soda buffered saline mixture) and polyethylene glycol-electrolyte solution (ColonLYTELY) are used to prepare the bowel for diagnostic and surgical procedures. Their use should be limited to acute, short-term use; chronic use may suppress normal bowel reflexes.

Nursing responsibilities

- Assess for possible contraindications to osmotic or saline laxatives, including bowel ulceration or obstruction, dehydration, electrolyte imbalances, heart failure (may be aggravated by the sodium content) or kidney injury.
- Sodium phosphate (Fleet phospho-soda buffered saline mixture) and polyethylene glycol-electrolyte solution (ColonLYTELY) should have no additional flavouring added unless instructed by the doctor. Red, green and orange cordials stain the bowel. Advise the person to slowly drink the solution: the first bowel action usually occurs approximately 1 hour after commencing the preparation. Preparation is complete when the person is passing clear fluid from the bowels.
- Other osmotic laxatives are administered with a full glass of liquid, preferably in the morning to avoid sleep disturbance.
- Monitor fluid and electrolyte status: skin turgor, mucous membranes, intake and output; daily weight and laboratory studies, such as haemoglobin and haematocrit levels, serum osmolality and electrolytes and urine specific gravity.

Health education for the person and family

- Do not use these agents on a routine basis to treat or prevent constipation. Long-term use can produce electrolyte disturbances.
- Chilling the solution increases its palatability.
- Expect some abdominal cramping.
- Use only as directed. Increase fluid intake to at least 6 to 8 (250 mL) glasses of non-alcoholic fluid.
- Notify the doctor if adverse effects occur, including abdominal pain, bloody stool, excessive skin or mucous membrane dryness, rapid weight loss, dizziness or other unusual symptoms.
- These agents work in 3 to 6 hours; take in the morning or early evening to avoid sleep disturbance.

IRRITANT OR STIMULANT LAXATIVES

Bisacodyl (Durolax, Bisolax)
Sennosides (Sennakot, Laxettes)
Castor oil (no longer in common use)

Stimulant laxatives work by stimulating the motility and secretion of intestinal mucosa. Their use results in watery stools, often accompanied by abdominal cramping and pain. These are used to relieve constipation. However, they should not be used as the initial treatment. Long-term use results in the bowel adapting to the strong stimulations of these medications. Rebound constipation can occur when the stimulant laxative is ceased. A normal diet does not provide adequate stimulation as the afferent messages from the intestines to the brain are ignored (Bullock & Manias, 2014). Stimulant laxatives are also used to prepare the bowel for diagnostic testing.

Nursing responsibilities

- Assess for potential contraindications to these laxatives, including abdominal pain and cramping, nausea and vomiting, anal or rectal fissures.
- Administer on an empty stomach to minimise the effects of food on its dissolution and absorption.
- Do not crush enteric-coated bisacodyl tablets or administer with alkaline products. This may hasten their dissolution in the stomach, leading to gastric distress.

Health education for the person and family

- Discourage the use of this type of laxative, even in over-the-counter preparations, for the initial or continuing relief of constipation.
- Do not use the laxative for more than 1 week; chronic use can be habit forming and may suppress normal bowel reflexes.
- These laxatives are excreted in breast milk and should not be used by lactating women.
- Phenolphthalein-containing products may discolour the urine pink or red. Report possible hypersensitivity manifestations, such as difficulty breathing, dizziness or light headedness, or skin rashes, to general practitioner and stop taking the medication.

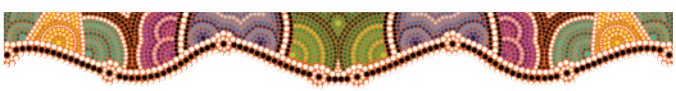
- *Oil retention enemas*—instil 90 to 120 mL mineral or olive or cottonseed oil into the bowel to lubricate the colonic mucosa and the faeces and soften the faecal mass. The instilled oil is retained for as long as possible (30 minutes to 1 hour) before evacuation.

The repeated use of enemas not only leads to impaired bowel function, bowel irritation and loss of muscle tone of the bowel and anal sphincter but also to fluid and electrolyte imbalances (Long, 2015). These imbalances are more likely to be caused by tap water and phosphate enemas. In acute conditions with a risk of bowel obstruction, perforation or ulceration, enemas should not be administered until their safe use is established.

Complementary and alternative therapies

Herbal or homeopathic therapies are used to help relieve constipation. Flaxseed oil lubricates the colon for easier passage of stool. Individuals are instructed to take 1 to 2 tablespoons daily. Flaxseed is a highly concentrated source of fibre; 1 to 2 tablespoons of ground flaxseeds can be sprinkled on cereals or salads daily, followed by 300 mL of water.

Biofeedback can be used to restore normal coordination of the anal sphincter and pelvic floor muscles. However, Chen (2014) suggests that due to the absence of clear research findings, biofeedback and sphincter exercises should be based on clinical judgment. Additionally, Slade (2014) recommends exercise, which stimulates intestinal contractions, encourages normal bowel function and improves appetite.



Nursing care

Health promotion

Education can prevent constipation. Highlight to people the importance of maintaining a diet high in natural fibre. Foods such as fresh fruit, vegetables, wholegrain products and bran provide natural fibre. Encourage reducing consumption of meats and refined foods, which are low in fibre and can be constipating. Emphasise the need to maintain a high fluid intake every day, particularly during hot weather and exercise. Discuss the relationship between exercise and bowel regularity. Encourage the person to engage in daily exercise; for example, walking.

Discuss normal bowel habits and explain that a daily bowel movement is not the norm for all people. Highlight that constipation has more to do with ease of a bowel evacuation, rather than the frequency of bowel movements (Bullock & Manias, 2014). Encourage people to respond to the urge to defecate when it occurs. Suggest establishing a routine by setting aside a time, usually following a meal, for elimination.

Assessment

To assess a person with real or perceived constipation, collect the following data:

- *Health history:* usual and current pattern of defecation, including time of day, amount and stool consistency; usual diet, fluid intake and activity pattern. Assess for possible contributing factors such as narcotic analgesics, activity limitations, painful haemorrhoids, perianal surgery; chronic diseases such as endocrine or neurological disorders; prescribed and non-prescription medications.
- *Physical examination:* abdominal girth and shape, bowel sounds, tenderness and percussion tone; digital exam of the rectum if impaction is suspected. Maintaining a bowel assessment chart (see Figure 23.2), incorporating, for example, the Bristol Stool Scale, provides a better assessment of potential constipation than the absence of faeces. The Bristol Stool Chart or Bristol Stool Scale developed by Heaton and Lewis at the University of Bristol and first published in the *Scandinavian Journal of Gastroenterology* in 1997 is an aid classifying faeces into seven groups. Types 1 and 2 indicate constipation; 3 and 4 are the 'ideal stools' (especially the latter), as these are the easiest to pass; 5 and 6 are more symptomatic of diarrhoea; and type 7 may be a sign of cholera or food poisoning or urgency (see Figure 23.3). Advising individuals or their carers how to use the Bristol Stool Chart enables appropriate intervention to be implemented, resulting in better management of constipation (Slade, 2014).

See 'Nursing care of the older adult' below for a discussion about constipation in older adults.

Nursing diagnoses and interventions

Nursing interventions for a person with constipation focus chiefly on education.

Constipation

Whether real or perceived, constipation is disruptive to a person's activities of daily living (ADLs) and life satisfaction.

- Monitor pattern of defecation and stool consistency. *This information helps to establish the person's usual pattern of defecation and differentiate between actual and perceived constipation.*
- If not contraindicated provide additional fluids to maintain an intake of at least 2500 mL per day. *A generous fluid intake helps to maintain soft stool consistency and promotes intestinal motility.*
- Encourage drinking a glass of warm water before breakfast. Provide time and privacy following breakfast for bowel elimination. *This helps develop a pattern of natural elimination; warm water provides mild stimulation of bowel peristalsis.*
- Consult with the nutritionist to provide a diet high in natural fibre unless contraindicated. Provide foods such as natural bran, prunes or prune juice. *Natural fibre adds bulk to the stool and has a mild stimulant effect.*
- Encourage activities such as ambulation or chair exercises (e.g. range of motion, stretching, wheelchair lifts) as

Bowel assessment chart

Name *Ian Andrews* Month *Jan* Year *2016*

Date	AM	PM	Nocte	Description of bowel action type: see Bristol stool chart	Continence status C = continent I = incontinent	Comments: use of aperients and results, problems concerning mobility, access to toilet, clothing, mood change etc.
1	$\dot{O}$	$\dot{O}$	$\dot{O}$			*Assessed fluids & diet*
2	$\dot{O}$	$\dot{O}$	$\dot{O}$			*Mobility & toilet access assessed*
3	$\dot{I}$	$\dot{O}$	$\dot{O}$	1^{+}	*C*	*Aperient as per chart given*
4	$\dot{O}$	$\dot{I}$	$\dot{I}$	*1 + 2 2*	*C*	*Encouraged fluid & diet & mob.*
5	$\dot{I}$	$\dot{O}$	$\dot{O}$	3^{+}	*C*	
6	$\dot{I}$	$\dot{O}$	$\dot{O}$	3^{++}	*C*	
7	$\dot{O}$	$\dot{O}$	$\dot{O}$			*Drug chart assessed as new* $\mathcal{R}_x$
8	$\dot{I}$	$\dot{O}$	$\dot{O}$	1^{+}	*C*	*Fluids & dietary fibre encouraged*
9	$\dot{I}$	$\dot{O}$	$\dot{O}$	*1 +* 2^{+}	*C*	*Fluids, diet & mobility encouraged*
10	$\dot{I}$	$\dot{O}$	$\dot{O}$	2^{++}	*C*	
11	$\dot{I}$	$\dot{O}$	$\dot{O}$	2^{++}	*C*	
12	$\dot{O}$	$\dot{O}$	$\dot{O}$			*Away for weekend*
13	$\dot{O}$	$\dot{O}$	$\dot{O}$			*Away for weekend*
14	$\dot{O}$	$\dot{O}$	$\dot{O}$			*Resumed ↑ fluid & ↑ fibre*
15	$\dot{I}$	$\dot{O}$	$\dot{O}$	1^{+}	*C*	
16	$\dot{I}$	$\dot{O}$	$\dot{O}$	2^{++}	*C*	
17						
18						
19						
20						
21						
22						
23						
24						
25						
26						
27						
28						
29						
30						
31						

$\dot{O}$ = Bowels not open $\dot{I}$ = the figure below the line indicates the number of times bowels have opened

FIGURE 23.2 ■ Bowel assessment chart

Source: Health and illness in older adults by S. Brown (2007), p. 19. Frenchs Forest, NSW: Pearson Australia.

tolerated. *Activity stimulates peristalsis and strengthens abdominal muscles, facilitating elimination.*

- If indicated, consult with doctor about the use of bulk laxatives, stool softeners or other laxatives as needed. Laxatives may be necessary to relieve acute constipation. *People with long-term activity or nutritional restrictions or impaired abdominal muscle strength may need a bulk-forming laxative to maintain normal elimination patterns and prevent constipation.*

Community-based care

Include the following topics when teaching self-care measures to prevent and manage constipation:

- Increase dietary fibre intake by including fresh fruit and vegetables, whole grains, high-fibre breakfast cereals and unprocessed bran in the diet. (Bran can be sprinkled on cereals, mixed into bread or muffin recipes, or mixed with fruit juice to increase its palatability.)

FIGURE 23.3 ■ Bristol Stool Chart

Source: Reproduced with kind permission of Dr K. W. Heaton, formerly Reader in Medicine at the University of Bristol. © 2000, Norgine group of companies.

- Unless contraindicated maintain fluid intake of 6 to 8 (250 mL) glasses of water per day.
- Remain physically active to promote bowel function and maintain muscle tone.
- Respond to the urge to defecate when perceived.

NURSING CARE OF THE OLDER ADULT

Constipation and older adults

Constipation and perceived constipation are common problems in older adults. Although constipation is not a normal consequence of ageing, factors such as slowed peristalsis, lowered activity levels, reduced food and fluid intake, and decreased sensory perception contribute to the higher incidence of constipation seen in older adults. Chronic diseases such as diabetes mellitus, restricted mobility and medications also increase the risk of constipation in older adults.

Cultural influences and advertising lead many older adults to believe a daily bowel movement is important for health. This belief contributes to an increased incidence of perceived constipation in older adults. Because of this perception, the older adult may come to rely on laxatives, suppositories or enemas to facilitate regular bowel movements. These external aids to defecation further impair the ability to maintain 'normal' bowel habits—a movement of soft stool every 2 to 3 days for older adults. See Box 23.1 for best practice management of constipation in older adults.

BOX 23.1 JBI best practice recommendations for management of constipation in older adults (2014)

- Prevention is better than cure, so it is recommended that older adults receive advice and education about hydration and a good diet with fibre sources such as cereals, nuts and seeds, wholemeal breads, vegetables and raw vegetables, and fruit. However, careful consideration is required as chewing or swallowing can be difficult for some older people. They may also have a poor tolerance to a high-fibre diet.
- Assessment of the older person should include an initial history of diet, medication, activity level and bowel habits. Also, the person should be asked to describe any symptoms such as bloating or pain when passing movements and associated straining. Screen older adults for the following: history of medication use (especially polypharmacy and taking laxatives) and, where feasible, replacement of constipation-causing medications with alternatives.
- For individuals unable to walk or restricted to bed or otherwise incapacitated, exercises such as low trunk rotation, pelvic tilt and single leg lifts are advised.
- Monitor and record bowel movements for frequency, character and pattern, episodes of constipation/faecal spoiling and use of oral or rectal laxative interventions.
- Osmotic laxatives such as polyethylene glycol (PEG), lactulose and bulking agents such as psyllium and bran are beneficial in managing constipation in older adults and should be promoted; it is necessary to determine individual requirements.

Source: Slade, S. (2014). *Constipation management.* Adelaide, Australia: Joanna Briggs Institute.

- Use laxatives appropriately:
  - Do not use laxatives, suppositories or enemas on a regular basis.
  - Bulk-forming agents provide insoluble fibre and are safe for long-term use; it is important to drink at least 6 to 8 (250 mL) glasses of water daily when using these (or any) laxatives.
  - Other laxatives such as docusate sodium (Coloxyl), bisacodyl (Durolax) and sennosides (Senna) should only be used occasionally to relieve constipation.
- Report any change in bowel habits such as new or persistent constipation or diarrhoea, abdominal pain, black or bloody stools, nausea or anorexia, weakness or unexplained weight loss to your general practitioner.

THE PERSON WITH IRRITABLE BOWEL SYNDROME

Irritable bowel syndrome (IBS), also known as spastic bowel or functional colitis, is a motility disorder of the lower GI tract. It is a functional disorder with no identifiable organic cause. IBS is often characterised by abdominal pain with constipation, diarrhoea or both.

Globally IBS is common, affecting 7–21% of the population depending on the diagnostic criteria used and the presence of co-morbidities (Chey, Kurlander & Eswaran, 2015). It usually affects young people, with about 50% of people diagnosed before the age of 35. There is a higher prevalence of IBS in women than in men (Björkman et al., 2014).

Pathophysiology

In IBS, it appears the central nervous system regulation of the motor and sensory functions of the bowel is altered. IBS may develop as a sequela of gastroenteritis, particularly when caused by *Camplylobacter*, *Salmonella* or *Shigella.*

People with IBS often experience increased motor reactivity of the small bowel and colon in response to stimuli such as food intake, hormonal influences and physiological or psychological stressors. Chey et al. (2015) suggest that, although the cause of IBS is ill-defined, many people with IBS attribute diet as a significant trigger of symptoms. IBS is characterised by visceral hypersensitivity and hyperactivity of the GI tract. Hypersecretion of colonic mucus is a common feature of the syndrome.

A lower visceral pain threshold is often found in people with IBS. A person may report pain, bloating and distension even when intestinal gas levels are normal. Serotonin, a neurotransmitter involved in regulating GI motility, and visceral perception may play a role in IBS. Higher than expected postprandial plasma serotonin levels are noted in some individuals with IBS (Chey et al., 2015). Rey de Castro et al. (2015) describe the subcategories of IBS as constipation (IBS-C), diarrhoea (IBS-D), mixed constipation and diarrhoea (IBS-M) or unspecified. Psychological factors such as depression or anxiety have been linked to IBS. However, these have not been identified as causes of IBS. A person may respond to counselling or stress management techniques designed to manage a chronic uncomfortable condition.

Manifestations

Irritable bowel syndrome is characterised by abdominal pain often relieved by defecation and a change in bowel habits (see the 'Manifestations' box below). The pain may be colic-like, occurring in spasms, or dull and continuous. Altered patterns of defecation may include:

- a change in frequency
- abnormal stool form (hard or lumpy, loose or watery)
- altered stool passage (straining, urgency or a sensation of incomplete evacuation)
- the passage of mucus.

A person may also describe abdominal bloating and excess gas. Other manifestations include nausea, vomiting and anorexia, fatigue, headache, depression or anxiety. The abdomen is often tender to palpation, particularly over the sigmoid colon.

MANIFESTATIONS Irritable bowel syndrome

ABDOMINAL PAIN
- May be relieved by defecation
- May be intermittent and colicky or dull and continuous

ALTERED BOWEL ELIMINATION
- Constipation
- Diarrhoea
- Mucus stools
- Abdominal bloating and flatulence
- Abdominal tenderness, especially over sigmoid colon
- Possible nausea or vomiting

INTERPROFESSIONAL CARE

Diagnosis of IBS is based on the presence of abdominal pain or discomfort with two of the following three characteristics: (1) relieved by defecation; (2) associated with a change in frequency of elimination; (3) associated with a change in stool form (Chey et al., 2015). Management is directed towards relieving manifestations and reducing or eliminating precipitating factors. Many people report benefits from psychotherapy and cognitive–behavioural therapy (Sharma, 2014b).

Diagnosis

The primary purpose of diagnostic testing is to rule out other causes of abdominal pain and altered faecal elimination. The stools may be examined for occult blood, ova, cysts and parasites, and white blood cells. A sigmoidoscopy, colonoscopy and/or a small-bowel series (upper GI series with small-bowel follow-through) and barium enema may be performed to visually examine the bowel mucosa, measure intraluminal pressures and biopsy suspicious lesions. Nursing care for these procedures is outlined in Chapter 20. Laboratory tests include a full blood count (FBC) with differential and erythrocyte sedimentation rate (ESR) to evaluate for anaemia from bleeding or a possible tumour. Increased WBCs indicate a bacterial infection.

Medications

Although not curative, medications are prescribed to manage the manifestations of IBS. Bulk-forming laxatives (such as bran or psyllium) may help reduce bowel spasm and normalise the number and form of bowel movements. An anticholinergic drug such as hyoscyamine (Buscopan) may be ordered to inhibit bowel motility by interfering with parasympathetic stimulation of the gastrointestinal tract (Chey et al., 2015). This relieves postprandial abdominal pain when taken 30 to 60 minutes before meals. Because of side effects, such as dry mouth, blurred vision and urinary hesitancy, these medications are used cautiously in older adults. In a person with diarrhoea, loperamide (Imodium) or diphenoxylate (Lomotil) may be used prophylactically to prevent diarrhoea in selected situations.

Antispasmodic medications are often used to control intestinal muscle spasm associated with IBS. Most antispasmodics are antimuscarinic medications blocking muscarinic receptors

on gastrointestinal smooth muscle, inhibiting contractions. One medication used exclusively to manage smooth muscle spasms is mebeverine (Colase). Mebeverine relaxes vascular, cardiac and other smooth muscles. It contains lactose so is contraindicated in a person with lactose intolerance. Additionally, it is used cautiously in a person with cardiac dysrhythmias, angina, ischaemic heart disease and hepatic or renal dysfunction (Tiziani, 2013).

Antidepressant drugs, including tricyclics and selective serotonin reuptake inhibitors (SSRIs), may relieve abdominal pain associated with IBS. Although the anticholinergic side effects of tricyclics (such as amitriptyline (Tryptanol) and imipramine (Tofranil)) may help decrease diarrhoea, these have more adverse effects than SSRIs such as sertraline (Zoloft) and fluoxetine (Prozac). Alosetron (Lotronex) is a serotonin receptor antagonist reducing abdominal pain and diarrhoea in individuals with IBS. Its use is limited, however, by its association with ischaemic colitis.

A systematic review and meta-analysis by Ford et al. (2008) found ispaghula husk, antispasmodics (particularly hyoscine) and peppermint oil to be effective treatments for IBS.

Nutrition

Many people with IBS benefit from additional dietary fibre. Soluble fibres include pectin, beta glucans (oats and barley) and gums in psyllium fibre. Insoluble fibres include cellulose, lignin and hemicelluloses (McKenzie et al., 2012). Adding bran to meals was thought to provide added bulk and water content to the stool, reducing the incidence of both loose diarrhoeal stools and hard, constipated stools. McKenzie et al. (2012) do not recommend increasing intake of insoluble dietary fibre or dietary fibre intake from mixed food sources (cereal and fruit fibre) as these did not improve symptoms associated with IBS. However, inclusion of linseeds for a trial period up to 3 months is recommended by Sharma (2014b) for treatment of constipation associated with IBS. Other dietary changes are specific to individual triggers for IBS manifestations. Some people may benefit from limiting lactose, fructose or sorbitol intake (see Table 23.1). When excess gas and flatulence are problems, reducing the intake of gas-forming foods, such as beans, cabbage, apple and grape juices, nuts and raisins, may be helpful. Caffeinated drinks, such as coffee, tea and soft drinks, act as gastrointestinal stimulants; limiting intake of these fluids may also prove beneficial.

Complementary and alternative therapies

Herbal preparations may provide some benefit for a person with IBS. Herbs with an antispasmodic effect, such as anise, chamomile and sage, may be used to reduce the manifestations of IBS. Peppermint oil is a carminative, causing relaxation of the sphincters; it is considered an effective alternative to antispasmodic medications (Sharma, 2014a). Dill and aniseed are carminatives included in some commercial preparations to relieve wind or colic in babies (Bullock & Manias, 2014). Ginger root when consumed as a tea or capsule assists with reducing gas, bloating and diarrhoea, and improves stomach functioning (Braun & Cohen, 2015). According to Sharma (2014b), probiotic therapies (such as yoghurt with active bacterial cultures, particularly *Bifidobacterium lactis*), improve overall IBS-C symptoms, abdominal pain and urgency. Although it was stressed that probiotic therapies had no effect on bloating, distension, flatulence or stool symptoms. Sharma (2014b) advised informing a person electing to try probiotics to try one preparation at a time for at least 4 weeks and monitor the effects on symptoms of IBS.

Nursing care

People with irritable bowel syndrome rarely require acute care for IBS as a primary problem. However, nurses frequently interact with individuals with IBS in clinics and other community settings.

Assessment

Careful assessment is important to help identify the effects of IBS on the person. Collect the following assessment data:

- *Health history:* current manifestations, their onset and duration; current treatment measures; effect of manifestations on lifestyle; exploration of mental health status.
- *Physical examination:* apparent general state of health; abdominal shape and contour, bowel sounds, tenderness.

Nursing diagnoses and interventions

The primary nursing responsibility is education; providing referrals and counselling are additional nursing responsibilities to a person with IBS. See the previous sections on diarrhoea and constipation for selected nursing interventions.

Community-based care

Include the following topics in teaching for the person with IBS:

- the nature of the disorder and the reality of the person's manifestations
- the relationship between IBS and stress, anxiety and depression
- stress and anxiety reduction techniques, such as meditation, visualisation, exercise, 'time out' and progressive relaxation
- dietary influences that may contribute to IBS and suggested dietary changes; an increase in intake of insoluble dietary fibre is not recommended
- the use and role of prescribed medications, their adverse effects and when to contact the doctor
- stress the importance of routine follow-up appointments and of notifying the primary care provider if manifestations change (such as blood in the stool, significant constipation or diarrhoea, increasing abdominal pain or weight loss).

If needed, refer the person to a counsellor or other mental health professional for assistance in dealing with psychological factors associated with living with a chronic condition.

THE PERSON WITH FAECAL INCONTINENCE

Faecal incontinence, the loss of voluntary control of defecation, occurs less frequently than urinary incontinence; however, it is no less distressing to the person.

Multiple factors contribute to faecal incontinence, including both physiological and psychological conditions (see Box 23.2). Bowel incontinence is usually considered a manifestation of a disorder rather than a disorder in itself. A person often does not reveal faecal incontinence when discussing health concerns. There are approximately 1.3 million Australians (15 years and older) with faecal incontinence (Continence Foundation of Australia (CFA), 2015). The National Continence Management Strategy (NCMS), established in 1998 by the Australian Government Department of Health and Ageing, provides funding to research and service development initiatives aimed at prevention and treatment of this significant problem.

Older adults are more affected by faecal incontinence due to aetiological factors. Le (2015) cites epidemiological data indicating that 1–10% of adults are affected with faecal incontinence and that 0.5–1.0% of adults experience regular faecal incontinence which affects their quality of life. Faecal incontinence is one of the three main causes (along with decreased mobility and dementia) for admittance to a residential aged care facility in Australia (CFA, 2011).

Pathophysiology

To understand the pathophysiology of faecal incontinence, it is necessary to understand normal defecation mechanisms. The rectum is normally empty. When it is distended by faeces entering from the sigmoid colon, the defecation reflex is stimulated. This reflex causes involuntary relaxation of the internal sphincter and stimulates the urge to defecate. When the external sphincter, which is under both somatic (voluntary) and autonomic (involuntary) control, relaxes, defecation occurs. Adults normally can override the defecation reflex by voluntary contraction of the external sphincter and pelvic floor muscles. The wall of the rectum gradually relaxes and the urge to defecate subsides.

The most common causes of faecal incontinence are those interfering with either sensory or motor control of the rectum and anal sphincters. If the external sphincter is paralysed as a result of spinal cord injury or disease, defecation occurs automatically when the internal sphincter relaxes with the defecation reflex. If sphincter muscles are damaged or excessive pelvic floor relaxation occurs, it may not be possible to override the defecation reflex with voluntary control.

Age-related changes in anal sphincter tone and response to rectal distension increase the risk of faecal incontinence in older adults. Resting and maximal anal sphincter pressures are decreased, particularly in older women. Additionally, older females need less rectal distension to produce sustained relaxation of the anal sphincter.

BOX 23.2 Selected causes of faecal incontinence

Neurological causes
- Spinal cord injury or disease
- Head injury, stroke or brain tumour
- Degenerative neurological disease, such as multiple sclerosis, amyotrophic lateral sclerosis (ALS), dementia
- Diabetic neuropathy

Local trauma
- Obstetric tears
- Anorectal injury
- Anorectal surgery with sphincter damage

Inflammatory processes
- Infection
- Radiation

Other causes
- Diarrhoea
- Stool impaction
- Pelvic floor relaxation or loss of sphincter tone
- Tumours

Psychological causes
- Depression
- Confusion and disorientation

INTERPROFESSIONAL CARE

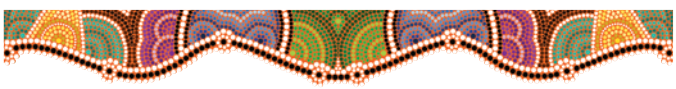
The diagnosis of faecal incontinence based on the person's history is fraught with difficulty as people are often reluctant to reveal faecal incontinence, or their carer may be unaware of previous continence history (Brown, 2007). Physical examination of the pelvic floor and anus evaluates muscle tone and rules out a faecal impaction. Impaired sphincter muscle may be palpable on digital exam. An anorectal manometry or a rectal motility test is used to evaluate the functional ability of the sphincter muscles. In this test, a small, flexible balloon catheter is introduced into the rectum and pressures are measured in the rectum and internal and external sphincters. Normally, rectal dilation causes the internal sphincter to relax and the external sphincter to contract. Sigmoidoscopy may also be used to examine the rectum and anal canal.

Management of faecal incontinence is directed towards the identified cause. D'Arcy (2014) recommends a comprehensive person-centred bowel management program based on formal assessment be developed and then reassessed 6 monthly for a person with faecal incontinence. Medications to relieve diarrhoea or constipation may be prescribed. A high-fibre diet, ample fluids and regular exercise are helpful for many people. Exercises to improve sphincter and pelvic floor muscle tone (Kegel exercises) may be of long-term benefit. See Chapter 26 for more information about Kegel exercises.

A person may also benefit from using loperamide before meals and prophylactically before leaving home. The National Institute for Health and Clinical Excellence (NICE) (2007) advises that antidiarrhoeal medication (e.g. loperamide hydrochloride

(Imodium)) can be used long term in doses from 0.5 mg to 16 mg per day, as required. Codeine phosphate may be offered for people who do not tolerate loperamide. Biofeedback therapy is suggested for mentally alert individuals with intact sphincter muscles but low muscle tone. It is thought that, with motivation and reinforcement, the person can achieve improved sphincter control in response to a stimulus. The goal of biofeedback is to improve sensation, coordination and strength of the sphincter muscle (Joanna Briggs Institute, 2008). However, D'Arcy (2014) suggests that due to the absence of clear research findings, biofeedback and sphincter exercises in adults with faecal incontinence need to be based on clinical judgment.

When damage to the sphincter or rectal prolapse (protrusion of rectal mucous membrane through the anus) is the cause of faecal incontinence, surgical repair is the treatment of choice. Surgery may also be indicated when conservative measures have been ineffective. Permanent colostomy, the creation of an opening from the large bowel on the abdominal wall, is a last-choice option for some people to control faecal output when other measures fail.



Nursing care

Health promotion

A bowel training program to establish a regular pattern of elimination is often effective in relieving faecal incontinence. Encourage the person to establish a regular time of day for elimination, usually 15 to 30 minutes after breakfast. A stimulant, such as a cup of coffee, a rectal suppository or even a phosphate enema, may be given to prompt defecation. A person with neurological incontinence can learn to stimulate the anal canal digitally to initiate defecation.

Dietary changes may be useful in managing faecal incontinence. If incontinence occurs only with mild loose or liquid stools, increasing dietary fibre or using a bulking agent to increase stool bulk and solidity may be effective. The majority of the fibre should come from a fibre-rich diet because fibre supplements provide only a limited amount of additional fibre (D'Arcy, 2014). When incontinence of solid stool occurs, a low-residue diet of easily digested and absorbed foods may be prescribed to reduce the frequency of defecation.

Assessment

- *Health history:* extent, onset and duration of incontinence; identified contributing factors; history of spinal cord or anorectal injury or surgery; chronic diseases such as diabetes mellitus, multiple sclerosis or other neurological disorders.
- *Physical examination:* mental status; general health; examination of perianal tissues; digital rectal examination.

Nursing diagnoses and interventions

Bowel incontinence

Nurses are often responsible for instituting bowel training programs and other measures to manage faecal incontinence.

- Teach caregivers to place the person on a toilet or commode and provide for privacy at a certain time of day. *Placing the person in the normal position to defecate at a consistent time of day stimulates the defecation reflex and helps re-establish a pattern of stool evacuation.*
- If necessary, insert a glycerin or bisacodyl (Durolax) suppository 15 to 20 minutes before positioning the person on the toilet or commode. This helps to stimulate evacuation. *Once a regular elimination pattern is established, it may be possible to discontinue suppository use.*
- Maintain a caring, non-judgmental manner when providing care. *This promotes a feeling of acceptance when the person may feel unacceptable.*

CONSIDERATION FOR PRACTICE

Provide room odour control with deodoriser tablets, sprays or other devices. Controlling odour is important to preserve the person's self-esteem.

Risk of impaired skin integrity

Good skin care is vital for a person with faecal incontinence. Stools contain enzymes and other irritating substances that promote skin breakdown when these are not promptly removed. This can lead to pressure ulcers, particularly when a neurological disorder (such as spinal cord injury, dementia or stroke) impairs mobility.

- Clean the skin thoroughly with mild soap and water after each bowel movement. *Toilet paper may be more irritating to the skin and less effective in removing faecal material.*
- Apply a skin barrier cream or ointment after each bowel movement. *These help to protect the skin from irritating substances in the faeces.*
- If disposable pads or briefs are used, check frequently for soiling and change when faeces are noted. *Although these help to protect bedding and clothing from soiling, these contribute to skin breakdown if they are not checked and changed frequently*.

Community-based care

Managing faecal incontinence is a challenging problem for the person and family caregivers. For the person with intact cognition, it can be psychologically devastating. The person may become socially isolated from fear of odour or soiling clothing. Self-esteem may suffer from a sense of lost control over body functions and the inability to provide self-care. It is important to stress that incontinence is never normal (i.e. ageing alone is not a cause of incontinence) and often is treatable. Encourage the person to seek medical evaluation of the problem.

Topics to include in education for the person and their family education are:

- Recommended dietary measures—for example, consuming a high-fibre diet and ample fluids to maintain soft, formed stools or a low-residue diet to reduce the number of stools.

- Suggestions for regular exercise stimulating bowel peristalsis and regular evacuation.
- Avoid constipation as straining often to achieve bowel movements stretches and weakens the pelvic floor muscles.
- Use of bulk-forming laxatives, such as psyllium seed (Metamucil), to provide stool bulk and reduce the number of small, liquid stools.
- Bowel training program instructions, including correct sitting position on the toilet or commode; techniques for digital anal stimulation, inserting suppositories or administering enemas as recommended. When sitting on the toilet or commode, advise the person or their carer to position with the feet firmly supported. This helps to fully relax the pelvic floor and sphincter muscles. For digital anal simulation, teach to insert a lubricated gloved finger through the anal sphincter into the rectum 2 to 3 cm while seated on the toilet or commode and then to use a circular side-to-side movement to gently stretch the rectal wall until the internal sphincter relaxes.
- Recommend smoking cessation as chronic coughing associated with smoking weakens the pelvic floor muscles, leading to bowel control problems. Encourage discussion with a doctor or pharmacist for information on quitting smoking and managing chronic coughing.
- Prescribed medications (such as loperamide to reduce the number of stools), their appropriate use and management of adverse effects (such as constipation).
- The importance of good skin care, particularly if the person has a neurological impairment.
- The potential benefits and associated risks of biofeedback and surgical treatment, if recommended.
- Provide referrals to appropriate home care or community health services.

ACUTE INFLAMMATORY AND INFECTIOUS BOWEL DISORDERS

The GI tract is particularly vulnerable to inflammation and infection because of its continual exposure to the external environment. Although most pathogens affecting the GI tract are ingested in food or water, infection may also be spread by direct contact, possibly by the respiratory route. Pathogens may also be transmitted sexually through anal intercourse.

Acute disease of the GI tract may be caused by the pathogen itself or by a bacterial or other toxin. Acute inflammatory disorders such as appendicitis and peritonitis result from contamination of damaged or normally sterile tissue by the person's own endogenous or resident bacteria.

THE PERSON WITH APPENDICITIS

Appendicitis, inflammation of the vermiform appendix, is a common cause of acute abdominal pain. It is the most common reason for emergency abdominal surgery, affecting 7% to 14 % of the population (Solomon & Flum, 2015). Appendicitis occurs at any age, but is more common in adolescents and young adults and slightly more common in males than females.

Pathophysiology

The appendix is a tube-like pouch attached to the caecum just below the ileocecal valve. It is usually located in the right iliac region, at an area designated as McBurney's point (see Figure 23.4A). The function of the appendix is not fully understood, although it regularly fills with and empties digested food.

Obstruction of the proximal lumen of the appendix is apparent in most acutely inflamed appendices. The obstruction is often caused by a *faecalith* or hard mass of faeces. Other obstructive causes include a calculus or stone, a foreign body, inflammation, a tumour, parasites (e.g. pinworms) or oedema of

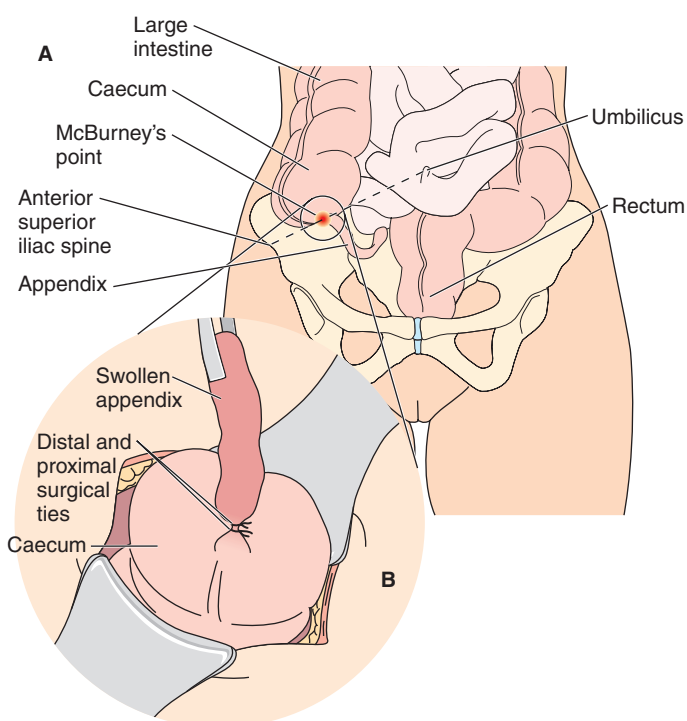


FIGURE 23.4 ■ *A*, McBurney's point, located midway between the umbilicus and the anterior iliac crest in the right lower quadrant. It is the usual site for localised pain and rebound tenderness due to appendicitis. *B*, During an appendectomy, the appendix and caecum are brought through the incision to the surface of the abdomen. The base of the appendix is clamped and ligated; the appendix is then removed

lymphoid tissue. Following obstruction, the appendix distends with fluid secreted by its mucosa. As pressure within the lumen of the appendix increases, blood supply is impaired, leading to inflammation, oedema, ulceration and infection. The formation of purulent exudate further distends the appendix. Within 24 to 36 hours, tissue necrosis and gangrene results, leading to perforation if treatment is not initiated. Perforation results in bacterial peritonitis.

Appendicitis is classified as simple, gangrenous or perforated, depending on the stage of the process. In *simple appendicitis*, the appendix is inflamed but intact. When areas of tissue necrosis and microscopic perforations are present in the appendix, it is classified as *gangrenous appendicitis*. A *perforated appendi*x shows evidence of gross perforation and contamination of the peritoneal cavity.

Manifestations

Continuous mild generalised or upper abdominal pain is the initial characteristic manifestation of acute appendicitis. Over the next 4 hours, the pain intensifies and localises in the right lower quadrant of the abdomen. It is aggravated by moving, walking or coughing. On palpation, localised and rebound tenderness are noted at McBurney's point. Rebound tenderness is demonstrated by relief of pain with direct palpation of McBurney's point followed by pain on release of pressure. Extension or internal rotation of the right hip increases the pain. Pain and local tenderness in the lower right quadrant as a presenting symptom are absent in approximately 45% of older adults compared with less than 5% of younger adults with appendicitis. This delays diagnosis in older adults, contributing to a 15% mortality of perforated appendicitis in older adults (McPhee, Papadakis & Tierney, 2008). Additionally, acute appendicitis in older adults is more virulent with complications developing sooner.

The classic manifestation of appendicitis is abdominal pain developing over 4 to 48 hours accompanied by nausea, vomiting, anorexia and a low-grade temperature. However, clinical manifestations occur in less than 50% of people developing appendicitis. More commonly, people with appendicitis present with a combination of these symptoms.

Pregnant women may develop right lower quadrant, periumbilical or right subcostal (under the rib cage) pain due to possible displacement of the appendix by the distended uterus. Women in their third trimester are at risk of appendicitis. During pregnancy, abdominal pain, nausea and vomiting are more common and many women developing appendicitis during pregnancy do not experience the classic symptoms, making early identification of appendicitis difficult.

Children, like older adults and pregnant women, often have fewer symptoms, making their diagnosis less obvious and the incidence of complications more frequent.

Complications

Perforation, peritonitis and abscess are possible complications of acute appendicitis. Perforation is manifested by increased pain and a high fever. It can lead to a small, localised abscess, local peritonitis or significant generalised peritonitis. The very young, older adults and those with impaired immune systems—for example, those with diabetes mellitus—generally are at increased risk of complications.

A less common disorder is chronic appendicitis, characterised by chronic abdominal pain and recurrent acute attacks at intervals of several months or more. Other conditions, such as inflammatory bowel disease and renal disorders, cause similar manifestations attributed to chronic appendicitis.

INTERPROFESSIONAL CARE

An acutely inflamed appendix can perforate within 24 hours, so rapid diagnosis and treatment is important. Because of this urgency and the low incidence of surgical complications, diagnostic testing and preoperative treatment are limited. The person is admitted to the hospital and intravenous fluids and antibiotics are initiated. Food and oral fluids are withheld until a diagnosis is confirmed. Once the diagnosis is established, an appendectomy is performed.

Diagnosis

Diagnostic and laboratory tests are used to help confirm the diagnosis and rule out other possible causes for the manifestations. Abdominal ultrasound is the most effective test assisting in diagnosing acute appendicitis, reducing the incidence of exploratory surgery, and is particularly useful in a person with atypical symptoms, such as pregnant women, children and older adults (Solomon & Flum, 2015). Other diagnostic tests used to accurately diagnose appendicitis include abdominal x-rays, an intravenous pyelogram, a urinalysis and a pelvic examination. Additionally, a WBC count with differential is obtained. With appendicitis, the total white count is elevated (10 000 to 20 000/mm^3), with an increased number of immature WBCs (bands).

Medications

Prior to surgery, intravenous fluids are given to restore or maintain vascular volume and prevent electrolyte imbalance. Antibiotic therapy with a third-generation cephalosporin effective against many gram-negative bacteria, such as cefotaxime (Cefotaxime Sandoz), ceftazidime (Fortum) or ceftriaxone (Rocephin), is initiated prior to surgery. The antibiotic is repeated during surgery and continued for at least 48 hours postoperatively. (The nursing implications for cephalosporin antibiotics are discussed in Chapter 11.) Analgesic medications are administered as prescribed.

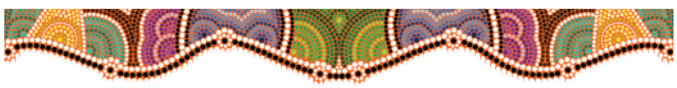
Surgery

The treatment of choice for acute appendicititis is an *appendectomy*, surgical removal of the appendix.

Either a laparoscopic approach (insertion of an endoscope to view abdominal contents) or via a laparotomy (surgical opening of the abdomen) are used for appendectomy. Laparoscopic appendectomy requires a very small incision through which the laparoscope is inserted. This procedure has several advantages: (1) direct visualisation of the appendix allows

definitive diagnosis without laparotomy; (2) postoperative hospitalisation is short; (3) postoperative complications are infrequent; and (4) recovery and resumption of normal activities is rapid.

An open appendectomy is performed by laparotomy. A small transverse incision is made at McBurney's point (see Figure 23.4A); the appendix is isolated and ligated (tied off) to prevent contamination of the site with bowel contents, and then removed (Figure 23.4B). A laparotomy is generally performed when the appendix has ruptured. It allows removal of contaminants from the peritoneal cavity by irrigation with sterile normal saline. Occasionally the wound may be left unsutured for periodic irrigation. Recovery is generally uneventful. Refer to Chapter 3 for specific discussion of preoperative and postoperative nursing care.



Nursing care

A nursing care plan for a person with acute appendicitis is included below.

Assessment

Because appendicitis rapidly progresses from inflammation to perforation, prompt assessment is vital. Obtain the following assessment data:

- *Health history:* current manifestations, including onset, duration, progression and aggravating or relieving factors; most recent food or fluid intake; known medication or other allergies and reaction to allergen; current medications; history of chronic diseases.
- *Physical examination:* vital signs, including temperature; apparent general health; abdominal shape and contour, bowel sounds, tenderness to light palpation.

Nursing diagnoses and interventions

Preoperative nursing care is directed towards preparing the person physically and psychologically for emergency surgery. Generally, limited time is available for preoperative teaching.

CONSIDERATION FOR PRACTICE

Keep the person with suspected appendicitis nil by mouth (NBM). Do not administer laxatives or enemas, which may cause perforation of the appendix. No heat should be applied to the abdomen; this may increase circulation (vasodilation) to the appendix, also causing perforation.

Risk of infection

Preventing complications during the perioperative period is a primary nursing care goal. Perforation and peritonitis are the most likely preoperative complications; postoperative complications include wound infection, abscess and possible peritonitis.

CONSIDERATION FOR PRACTICE

Assess abdominal status frequently, including distension, bowel sounds and tenderness. Increasing generalised pain, a rigid, board-like abdomen and abdominal distension may indicate developing peritonitis.

- Monitor vital signs, including temperature and pain. Perforation is manifested by increased pain and a high fever. *Tachycardia and rapid shallow respirations may indicate perforation of the appendix with resulting peritonitis. The blood pressure may fall if sepsis is present.*
- Maintain intravenous infusion until oral intake is adequate. *Intravenous fluids are given to maintain vascular volume and to provide a route for antibiotic administration.*
- Assess wound, abdominal girth and postoperative pain. *Swelling of the wound, increased abdominal girth or an increase in pain may indicate infection or peritonitis.*

Acute pain

A person with appendicitis experiences pain before and after surgery. Analgesia is limited until the diagnosis is established. Postoperative pain is controlled by narcotic or non-narcotic analgesics.

- Assess pain, including its character, location, severity and duration. Report any unexpected changes in the nature of pain. *Both preoperatively and postoperatively, the person's pain provides important clues about the diagnosis and possible complications such as rupture of the appendix or peritonitis.*

CONSIDERATION FOR PRACTICE

Sudden relief of preoperative pain may signal rupture of a distended and oedematous appendix.

- Administer analgesics as ordered. *Preoperatively, pain medication can be given after a diagnosis is established. Postoperatively, provide analgesics to maintain comfort and enhance mobility.*
- Assess effectiveness of medication 30 minutes after administration. Report increasing or unrelieved pain. *Increasing or pain unrelieved by prescribed analgesic may indicate a complication or the need for further assessment. For example, continued abdominal discomfort and distension may indicate excess intestinal gas that may be better relieved by ambulation.*

Community-based care

Often preoperative teaching is limited by pain and the urgent nature of the surgery. Explain why food and fluids are not permitted during this time. If time allows, teach postoperative turning, coughing, deep breathing and pain management.

Following an uncomplicated appendectomy, the person is often discharged either the day of, or the day following, surgery. Postoperative teaching includes:

- wound or incision care, including hand hygiene and dressing change procedures as indicated

NURSING CARE PLAN A person with acute appendicitis

Lynne James is a 19-year-old university student in her first year of a nursing degree. Ms James arrives at the emergency department (ED) at 0100 hrs. She describes a general lower abdominal pain which started the previous evening. By midnight, the pain was more localised over the right lower quadrant. She is also nauseated and reports episodes of vomiting.

ASSESSMENT

Sue Grady, RN, completes the admission assessment in the ED. Ms James reports nausea and abdominal pain, rating it at 9/10 (0 to 10 pain scale) stating, 'Walking makes my stomach hurt worse.' Physical assessment findings include T 37.8°C, P 84, R 16 and BP 110/70; skin warm to touch; abdomen flat and guarded, with marked tenderness in right lower quadrant. Ms James' FBC shows WBC 14 000/mm^3; neutrophils 81.1%; lymphocytes 12.5%. The diagnosis of acute appendicitis is made. Ms James is kept fasting; prepared for theatre, consented and transferred to operating theatre for a laparoscopic appendectomy.

DIAGNOSES

- *Risk of infection* related to impaired skin integrity secondary to surgical incision.
- *Alteration in comfort* related to acute pain secondary to surgical intervention.
- *Anxiety* related to situational crisis.

PLANNING

When planning nursing care with Ms James, it is vital to consider and ensure her religious and cultural beliefs are incorporated into her plan of care.

Expected outcomes

- Ms James' incision will heal without infection or complications.
- Ms James will verbalise adequate pain relief.
- Ms James will verbalise decreased anxiety.
- Ms James will return to preoperative activities.

IMPLEMENTATION

- Assess Ms James' pain using a pain scale; provide analgesics as needed. Monitor Ms James for effectiveness and adverse reactions.
- Teach Ms James pain management following discharge.
- Teach Ms James abdominal splinting during coughing, turning or ambulating as needed.
- Teach Ms James home care of incision.
- Discuss with Ms James activity limitations as ordered.
- Instruct Ms James to report fever or warmth, redness or drainage from the incision, or increasing abdominal pain.

EVALUATION

On discharge the following evening, Ms James was fully mobile. Her appetite had returned and she was tolerating diet and fluids well. Her temperature was 36.3°C. The RN provided Ms James with written and verbal information on postoperative care following an appendectomy.

CRITICAL THINKING IN THE NURSING PROCESS

1. Outline the physiological basis for Ms James' elevated WBC.
2. Describe differences in Ms James' postoperative care and teaching if she had undergone a laparotomy instead of a laparoscopic appendectomy.
3. Outline the teaching plan you would develop with Ms James for home care following a laproscopic appendectomy.
4. Develop a care plan for Ms James for the nursing diagnosis *Anxiety* related to a situational crisis.

REFLECTION ON THE NURSING PROCESS

1. Reflect on what you learned from completing this section. Outline how you will apply this knowledge to assist you in your clinical reasoning when next on your professional experience placement.
2. Describe nursing interventions you can incorporate into your clinical practice to reduce a person's anxiety levels while they wait for surgery.

- instructions to report fever, increased abdominal pain, swelling, redness, drainage, bleeding or warmth of the operative site to the doctor
- any activity limitations (e.g. lifting, driving)
- when it is appropriate to return to work.

THE PERSON WITH PERITONITIS

Peritonitis, inflammation of the peritoneum, is a serious complication of many acute abdominal disorders. It is usually caused by enteric bacteria entering the peritoneal cavity through a perforated ulcer, ruptured appendix, perforated diverticulum or necrotic bowel, or during abdominal surgery. Pelvic inflammatory disease, gallbladder rupture, abdominal trauma or peritoneal dialysis can also lead to peritonitis.

Pathophysiology

The peritoneum is a double-layered serous membrane lining the walls (parietal peritoneum) and organs (visceral peritoneum) of the abdominal cavity. There is a potential space between the parietal and visceral layers of the peritoneum containing a small amount of serous fluid. This space, the peritoneal cavity, is sterile.

Peritonitis results from contamination of the normally sterile peritoneal cavity by infection or a chemical irritant. Chemical peritonitis often precedes bacterial peritonitis. Perforation of a peptic ulcer or rupture of the gallbladder releases gastric juices (hydrochloric acid and pepsin) or bile into the peritoneal cavity, causing an acute inflammatory response.

Bacterial peritonitis is usually caused by infection by *Escherichia coli*, *Klebsiella*, *Proteus* or *Pseudomonas* bacteria normally inhabiting the bowel. *Neisseria gonorrhoeae* can also cause peritonitis in untreated women with the disease. Inflammatory and

immune defence mechanisms are activated when bacteria enter the peritoneal space. These defences can effectively eliminate small numbers of bacteria. However, they are overwhelmed by a massive or continued contamination. When this occurs, mast cells release histamine and other vasoactive substances, causing local vasodilation and increased capillary permeability. Polymorphonuclear leucocytes (a type of WBC) infiltrate the peritoneum to phagocytise bacteria and foreign matter. A fibrinogen-rich plasma exudate, promoting bacterial destruction and forming fibrin clots, seals off and segregates the bacteria. This process helps limit and localise the infection, allowing host defences to eradicate it. Continued contamination, however, leads to generalised inflammation of the peritoneal cavity. The inflammatory process causes fluid to shift into the peritoneal space (third spacing). Circulating blood volume is depleted, leading to hypovolaemia. *Septicaemia*, a systemic disease caused by pathogens or their toxins in the blood, may develop.

Manifestations

Manifestations of peritonitis depend on the severity and extent of the infection, as well as the age and general health of the person. Both local and systemic manifestations are present (see accompanying box). A person often presents with evidence of an *acute abdomen*, an abrupt onset of diffuse, severe abdominal pain. The pain may localise and intensify near the area of infection. Movement may intensify the pain. The entire abdomen is tender, with guarding or rigidity of abdominal muscles. The acute abdomen is often described as board-like. Rebound tenderness may be present over the area of inflammation. Peritoneal inflammation inhibits peristalsis, resulting in a paralytic ileus. Bowel sounds are markedly diminished or absent. Progressive abdominal distension is noted. Pooling of GI secretions may cause nausea and vomiting. Systemic manifestations of peritonitis include fever, malaise, tachycardia and tachypnoea, restlessness and possible disorientation. The person may be oliguric (having little urine output) with signs of dehydration and shock.

Older adults or chronically debilitated or immunosuppressed individuals may present with few of the classic manifestations of peritonitis. Increased confusion and restlessness, a decreased urinary output and vague abdominal complaints may be the only clinical manifestations. These individuals are at increased risk of delayed diagnosis, contributing to a higher mortality rate.

Complications

Complications of peritonitis may be life threatening. Abscess formation is common. The very defence mechanisms isolating and localising the infection can protect it from immune responses and systemic antibiotics. Fibrous adhesions in the abdominal cavity are a late complication and may lead to subsequent obstruction.

Without prompt and effective treatment, septicaemia and septic shock develop. Fluid loss into the abdominal cavity leads to hypovolaemic shock. These potentially lethal complications require immediate and aggressive intervention to prevent multiple-organ failure and death. Shock and its management are discussed in Chapter 10.

MANIFESTATIONS Peritonitis

ABDOMINAL/GASTROINTESTINAL MANIFESTATIONS
- Diffuse or localised pain
- Tenderness with rebound
- Board-like rigidity of abdomen
- Diminished or absent bowel sounds
- Distension
- Anorexia, nausea, and vomiting

SYSTEMIC MANIFESTATIONS
- Fever
- Malaise
- Tachycardia
- Tachypnoea
- Restlessness
- Confusion or disorientation
- Oliguria

INTERPROFESSIONAL CARE

Care of individuals with peritonitis focuses on establishing the diagnosis and identifying and treating its cause as well as the peritonitis. Preventing complications is a vital aspect of care.

Diagnosis

Diagnostic tests are performed to establish the diagnosis of peritonitis, rule out other disorders and help identify the cause. The tests may include a WBC count (elevated to approximately 20 000/mm^3 in peritonitis), blood cultures, abdominal x-rays, liver and renal function studies, serum electrolytes and an abdominal paracentesis. (In peritonitis, peritoneal fluid contains increased protein and WBCs.) Increased numbers of immature blood cells are present as the bone marrow releases these in response to the infection.

Medications

Until the infecting organism is identified, a broad-spectrum antibiotic effective against organisms commonly implicated in peritonitis is prescribed. Beta-lactum antibiotics such as imipenem (Primaxin) or meropenem (Merrem) are prescribed due to

FAST FACTS

Mortality from peritonitis
- The overall mortality rate associated with peritonitis is about 40%.
- People with other medical conditions, older adults and those with greater bacterial contamination have a higher mortality.
- Young people with perforated ulcers or appendicitis, those with less extensive bacterial contamination and those receiving early surgical intervention have mortality rates of less than 10%.

their broad spectrum of action. Once culture results are obtained, antibiotic therapy is modified to the specific organism(s) responsible.

Other antibiotics ordered may include ampicillin (e.g. Ampicyn), metronidazole (Flagyl), ciprofloxacin (Cipro), clindamycin (Cleocin), a cephalosporin such as ceftriaxone (Rocephin) or an aminoglycoside antibiotic such as gentamicin (Genoptic) or amikacin (Amikin). Nursing implications for antibiotic therapy are discussed in Chapter 11. Analgesics are prescribed to promote comfort.

Surgery

If peritonitis is caused by a perforation, gangrenous bowel or inflamed appendix, a laparotomy is done to close the perforation or remove the damaged and inflamed tissue. If an abscess is present, it may be surgically drained or removed.

Peritoneal lavage, washing of the peritoneal cavity with copious amounts of warm isotonic fluid, may be done during surgery. This procedure dilutes residual bacteria and removes gross contaminants, blood and fibrin clots. In rare instances, peritoneal lavage may be continued for several days following surgery. The solution is infused into the upper portion of the peritoneal cavity and removed via drains in the pelvic cul-de-sac. Careful assessment of fluid and electrolyte status and maintaining aseptic techniques are necessary.

Following a laparotomy for peritonitis, the person often returns from surgery with either Penrose or closed drain systems such as a Jackson–Pratt drain. In some cases, the incision is left unsutured. With severe and long-standing peritonitis, the abdomen may be closed temporarily with polypropylene mesh containing a nylon zipper or Velcro, allowing repeated abdominal exploration and drainage of infectious sites.

Nutrition

Intravenous fluids and electrolyte replacements are administered to maintain vascular volume and fluid and electrolyte balance. Parenteral nutrition is given until adequate oral intake resumes.

Other treatments

The individual is placed on bed rest in Fowler's position; this helps localise the infection and promotes lung ventilation. Oxygen is often ordered, facilitating cellular metabolism and healing.

INTESTINAL DECOMPRESSION The inflammatory process of peritonitis draws large amounts of fluid into the abdominal cavity and the bowel. Additionally, peristaltic activity of the bowel is slowed or halted by the inflammation, causing *paralytic ileus* (or *ileus*), impaired propulsion or forward movement of bowel contents. Intestinal decompression is used to relieve abdominal distension, facilitate closure and minimise postoperative respiratory complications. A nasogastric or long intestinal tube is inserted and connected to continuous drainage (see Figure 23.5). If prolonged intestinal decompression is anticipated, a jejunostomy may be performed for comfort. Suction is maintained until peristalsis resumes, bowel sounds are present and the person is passing flatus. Food and fluids are withheld until intestinal motility has returned and suction is discontinued.

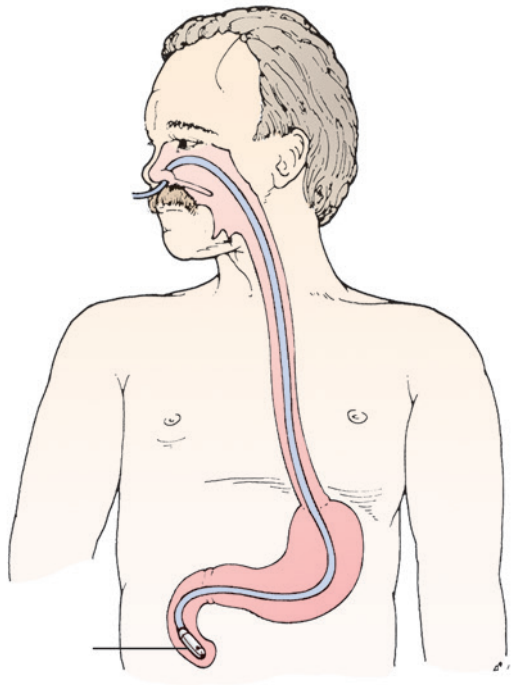


FIGURE 23.5 ■ The weighted tip or inflated balloon at the end of an intestinal tube is drawn into the intestine by gravity and peristalsis

Nursing care

Peritonitis is a serious illness. Early recognition and treatment are important to minimise the risk of complications.

Assessment

- *Health history:* pain, its onset, character, severity, location, aggravating and relieving factors; associated symptoms such as anorexia, nausea, vomiting; current and previous history of peptic ulcer disease, gallbladder disease, chronic diseases; current medications.
- *Physical examination:* vital signs, including temperature; level of consciousness; skin colour, temperature, warmth, capillary refill and turgor; abdominal shape, contour, bowel sounds, tenderness, tympany and guarding.

Nursing diagnoses and interventions

A person with peritonitis requires intensive nursing and medical care to prevent complications and enable a full recovery. Nursing priorities include interventions to relieve pain, restore fluid balance, manage altered protection due to infection and reduce anxiety.

Acute pain

Abdominal distension and acute inflammation contribute to the pain associated with peritonitis. Surgery further disrupts

abdominal muscles and other tissues, and exacerbates pain. See Chapter 8 for specific discussion on acute pain and its management. Effective pain management promotes immune function, healing, mobility and recovery.

- Assess pain at rest and on movement, including its location, severity (using a standard pain scale) and type. Monitor analgesic effectiveness. Report adverse changes to the primary care provider.

CONSIDERATION FOR PRACTICE

Unrelieved pain or a change in the location, severity or type of pain may indicate spread of infection, abscess formation or other complications of peritonitis.

- Place in Fowler's or semi-Fowler's position with the knees and feet elevated. *This position reduces stress on abdominal structures and facilitates respirations, promoting comfort.*
- Administer analgesics as ordered on a routine basis or using patient-controlled analgesia (PCA). *Routine analgesic administration maintains a therapeutic blood level and helps maintain comfort, facilitating healing and movement.*
- Teach and assist with adjunctive pain management techniques such as meditation, visualisation, massage and progressive relaxation. *Adjunctive measures augment analgesics and help promote a sense of control over pain.*

Deficient fluid volume

In peritonitis, significant amounts of fluid are drawn into the abdominal cavity and bowel, reducing vascular volume and cardiac output. Fluid is also lost from the body by intestinal suction or through drains placed in the abdomen during surgery. An unsutured incision causes additional significant fluid loss.

- Maintain accurate intake and output records. Measure urine output every 1 to 2 hours; report outputs less than 0.5 mL/kg/hr. *Measure gastrointestinal output at least every 4 hours. Intake and output records provide valuable assessment of fluid volume status.*

CONSIDERATION FOR PRACTICE

Urine output of less than 0.5 mL/kg/hr may indicate hypovolaemia, decreased cardiac output and impaired tissue perfusion.

- Monitor vital signs, peripheral pulses, capillary refill and haemodynamic parameters (such as central venous pressure, cardiac output and pulmonary artery pressures (if Swan–Ganz catheter in situ)) every hour or as indicated. *These measurements provide important information about fluid and vascular volumes and cardiovascular status.*
- Assess skin turgor, colour, temperature and mucous membranes at least every 8 hours. *Warm, dry skin with poor turgor and dry, shiny mucous membranes indicate dehydration.*
- Measure or estimate fluid losses via abdominal drains and on dressings. *Significant amounts of exudative fluid may be lost.*
- Weigh daily. Weight is an accurate indicator of fluid status. *Rapid weight gains or losses reflect changes in fluid volume.*
- Monitor laboratory values, including haemoglobin and haematocrit, urine specific gravity, serum osmolality and electrolytes and ABGs. Report changes to the doctor. *Laboratory results provide information about fluid and electrolyte status and acid–base balance.*
- Administer intravenous fluids and electrolytes as ordered. Gastrointestinal drainage may be replaced millilitre for millilitre with a balanced electrolyte solution. *Intravenous fluids are necessary to meet daily fluid intake needs, as well as to replace continuing losses of water and electrolytes.*
- Provide meticulous skin care and frequent oral hygiene. *Fluid deficit increases the risk of skin breakdown and mucous membrane ulcerations.*

Delayed surgical recovery

Repeated surgeries, an unsutured incision and the presence of drains and intravenous cannula interrupt skin integrity, the body's first line of defence against microorganisms. Additionally, immune defences are stressed by the infection and potential malnutrition. As a result, the risk of impaired healing and further infection is increased.

- Practise meticulous hand hygiene and use standard precautions at all times. *Hand hygiene reduces transient bacteria on the skin. Hand hygiene remains the most important method of controlling infection. Standard precautions reduce the risk of spreading infection to or from the person.*
- Monitor temperature, pulse rate and pain, and for localised signs of infection, such as redness and swelling around incisions and drain sites, increased or purulent drainage, and cloudy or malodorous urine. *Impaired defences increase the risk of extension of the infection or unrelated infections.*
- Use aseptic non-touch technique for dressing changes, wound care and irrigations. *Disruption of the protective barrier of the skin increases the risk of contamination and further infection.*
- Obtain cultures of purulent drainage from any site. *Early identification of any additional infection allows timely intervention.*
- Monitor WBC and differential, serum protein and albumin. *An increased WBC with a higher percentage of immature cells present in the blood is an indicator of infection and normal immune response. Serum albumin and protein levels are indicators of nutritional status as well as immune function.*
- Maintain fluid balance and nutritional status through enteral or parenteral feedings, as indicated. See the 'Translation to practice' box below for evidence-based recommendations for care of enterally fed people. *Adequate nutrition and fluid balance are necessary for optimal immune system function.*

TRANSLATION TO PRACTICE Evidence-based practice: malnutrition and the critically ill

A vital component of the treatment and care of critically ill people (such as those with peritonitis) is providing nutritional support, primarily through enteral feedings. Malnutrition is common in critically ill people and is associated with poor clinical outcomes as well as increased healthcare spending. Enteral nutrition (EN) is the method of choice for nutrition delivery for many critically ill people. However, EN delivery practices vary widely, and underfeeding is widespread in critical care. Interruptions in enteral nutrition due to performance of procedures, positioning, technical issues with feeding accesses and gastrointestinal intolerance contribute to underfeeding. Strategies such as head-of-bed positioning, use of prokinetic agents, tolerance of higher gastric residual volumes, consideration of postpyloric feeding access and use of a nutrition support protocol may decrease time spent without nutrition.

Stewart (2014) highlighted the consequences of malnutrition for critically ill people and current nursing routines contributing to EN interruptions in critically ill people, and described nursing interventions to improve a critically ill person's nutritional support. Despite strong evidence that early enteral feeding is beneficial to critically ill people, the mean time to enteral feeding remains high at 46.5 hours from admission to initiation of feedings.

Strategies to minimise interruptions include:

- Position the head of the bed at 30 to 45 degrees.
- Assess the gastric residual volume (GRV) every 4 hours. Withhold feedings if GRV is > 500 mL and assess.
- Consider prokinetic agents after two episodes of GRV > 250 mL.
- Consider postpyloric access when gastric feeding intolerance demonstrated.

IMPLICATIONS FOR NURSING

Based on Stewart's (2014) review, further research is warranted into methods to reduce time a critically ill person spends without EN, such as reduced fasting time before procedures or extubations. Alternative continuous enteral feeding methods, incorporating increased hourly rates to compensate for predicted losses in feeding volumes and compensatory increases in hourly rate to make up for experienced losses in feeding volumes, also requires exploration.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Malnutrition results when nutritional intake does not meet a person's metabolic demands. Describe the clinical implications when the nutritional intake of a person who is critically ill does not meet their increased metabolic demands.
2. Discuss how routine nursing interventions interrupt administration of enteral nutrition.
3. Outline nursing responses to improve EN to ensure a person's metabolic demands are maintained.

CONSIDERATION FOR PRACTICE

An acute infection such as peritonitis causes a stress response with excess energy expenditure and loss of body proteins and cell mass. Glycogen stores are rapidly depleted and body proteins are used to meet energy needs. Withholding food further complicates this process, leading to rapid development of protein energy malnutrition (PEM). PEM impairs the immune response and delays healing.

Anxiety

The severity and potential threat to life associated with peritonitis present a situational crisis for the individual and their family. Anxiety is a common response.

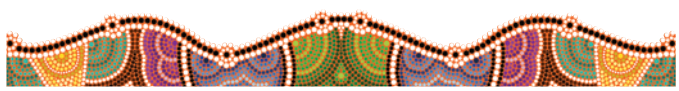
- Present a calm, reassuring manner. Encourage expression of concerns, listen carefully and acknowledge their validity. *This helps establish trust.*
- Assess the individual and their family's anxiety level and present coping skills. *Interventions need to be tailored to the needs and strengths of the individual and their family.*
- Explain all treatments, procedures, tests and examinations. *An increased understanding of what is being done reduces anxiety.*
- Reinforce and clarify information as needed. *This improves understanding and promotes acceptance.*
- Teach and assist with relaxation techniques such as meditation, visualisation and progressive relaxation. *These measures promote positive coping skills and reduce physical manifestations of anxiety.*
- Maintain consistent caregiver assignments. *Consistency of nursing care and care providers helps reduce anxiety. Complex wound care and irrigation procedures are best performed by people familiar with prescribed techniques.*

Community-based care

Education for home care includes the following topics:

- Wound care procedures, including dressing changes or irrigations. Provide verbal and written instructions on how to change dressings or do irrigations, as well as on where to obtain supplies, and provide opportunities for the person to practise and demonstrate the procedure prior to discharge.
- Prescribed medications, including name and purpose of the drug, dosage, how to take the medication correctly (e.g. not chewing or crushing enteric-coated tablets), potential adverse effects and their management.
- Manifestations of further infection (redness, heat, swelling, purulent drainage, chills and fever, and increasing pain) and potential complications to be reported to the care provider.
- Prescribed activity restrictions.
- Instructions for a high-kilojoule, high-protein diet for healing and optimal immune function.

Provide a referral to home health services for assessment, wound care and further assistance, as required.



THE PERSON WITH GASTROENTERITIS

Gastroenteritis, or *enteritis*, is an inflammation of the stomach and small intestine. Enteritis may be caused by bacteria, viruses, parasites or toxins. Gastroenteritis is a major health problem throughout the world, particularly in developing countries. Upper GI manifestations such as anorexia, nausea and vomiting are common. Diarrhoea of varying intensity and abdominal discomfort are nearly universal features of gastroenteritis. In Australia, transmission is mainly via contaminated food or water. However, in many remote Indigenous communities there is a high incidence of gastroenteritis due to poor sanitation facilities and sewage disposal, as is the case in many developing countries where diarrhoeal diseases are major causes of morbidity and mortality.

Globally there are nearly 1.7 billion cases of diarrhoeal diseases each year (World Health Organization (WHO), 2013). Diarrhoeal disease is a leading cause of child mortality and morbidity globally, mostly resulting from contaminated food and water sources. World Health Organization (2013) statistics highlight that worldwide about 1 billion people lack access to improved water and 2.5 billion have no access to basic sanitation. In developing countries, children under 3 years experience on average three episodes of diarrhoea every year. Each episode deprives the child of the nutrition necessary for growth. As a result, diarrhoeal diseases are a major cause of malnutrition, and malnourished children are more likely to fall ill from diarrhoea (WHO, 2013).

The infectious organism is usually acquired by the faecal–oral route, from contaminated water, food or hands. For this reason, gastroenteritis is often called 'food poisoning'. Viruses commonly cause acute diarrhoeal illness. Diarrhoea due to rotaviruses or the noroviruses (formerly known as Norwalk virus) occurs year round (with seasonal peaks in June and December) in both adults and children.

Rotavirus is the most common cause of acute severe gastroenteritis in early childhood and a significant cause of death in young children; more than 500 000 deaths per year (WHO, 2015a). Two rotavirus vaccines were added to the Australian National Immunisation Program in July 2007. Immunisation reduces the risk of developing severe rotavirus gastroenteritis by 85–100% and any rotavirus gastroenteritis by around 70% (Queensland Health, 2016). Prior to vaccine introduction, it is estimated the virus was responsible for up to 50% of diarrhoea hospitalisations in childhood, with approximately 10 000 Australian children hospitalised each year. Australian Aboriginal and Torres Strait Islander children have a higher burden of rotavirus illness—being three to five times more likely to be hospitalised, and for longer periods—than non-Indigenous children (Snelling et al., 2012). Most infections occur in children under 2 years of age and are potentially life threatening due to severe dehydration. Older children and adults generally experience a mild self-limiting illness. However, in older adults or those with impaired immune function, as with children less than 2 years of age, rotavirus can also have severe complications.

Pathophysiology

Bacterial or viral infection of the GI tract produces inflammation, tissue damage and manifestations by two primary mechanisms:

1. *The production of exotoxins.* A number of bacteria produce and excrete an exotoxin that enters the surrounding environment (intestinal lumen), causing damage and inflammation. Exotoxins in the GI tract are often referred to as *enterotoxins*. These impair intestinal absorption and can cause secretion of significant amounts of electrolytes and water into the bowel, resulting in diarrhoea and fluid loss. Common bacterial enterotoxins include those produced by *Staphylococcus*, *Clostridium perfringens*, *Clostridium botulinum*, some strains of *Escherichia coli*, *Vibrio cholera* and *C. difficile*.
2. *Invasion and ulceration of the mucosa.* Other bacteria, including some *Shigella*, *Salmonella* and *E. coli* species, damage tissue more directly. These invade the intestinal mucosa of the small bowel or colon, producing microscopic ulceration, bleeding, fluid exudates and water and electrolyte secretion.

In some cases, the mechanism of injury is unclear. It may be a combination of direct and toxic damage. For example, noroviruses damages the mucosa of the jejunum, with fluid and electrolyte secretion.

Manifestations

Although the manifestations of bacterial and viral enteritis vary according to the organism involved, several features are common (see the 'Manifestations' box below). Anorexia, nausea and vomiting are caused by distension of the upper GI tract from unabsorbed chyme and excess water. Bowel distension, irritation of the bowel mucosa and gas production from fermentation of undigested food lead to abdominal pain and cramping. *Borborygmi*, excessively loud and hyperactive bowel sounds, are another result. The abdomen is often distended and tender.

Diarrhoea is usually predominant with enteritis. Fluid is secreted into the bowel lumen and the unabsorbed chyme and electrolytes create an osmotic pull of fluid into the bowel.

MANIFESTATIONS Gastroenteritis

GASTROINTESTINAL EFFECTS
- Anorexia, nausea and vomiting
- Abdominal pain and cramping
- Borborygmi
- Diarrhoea

GENERAL EFFECTS
- Malaise, weakness and muscle aches
- Headache
- Dry skin and mucous membranes
- Poor skin turgor
- Orthostatic hypotension, tachycardia
- Fever

Motility is stimulated and stools become watery and frequent. Loss of fluids and electrolytes through diarrhoea can lead to serious manifestations of enteritis. Fluid volume is rapidly depleted, leading to dehydration and hypovolaemia. Initally, orthostatic hypotension and fever may be noted. However, if fluid loss continues, hypovolaemic shock develops.

Complications

Electrolyte and acid–base imbalances may result from gastroenteritis. Extensive vomiting leads to metabolic alkalosis due to the loss of hydrochloric acid from the stomach. When diarrhoea predominates, metabolic acidosis is more likely. Potassium is lost in either case, leading to hypokalaemia. Hyponatraemia may develop if fluids are replaced with pure water. Headache, cardiac irregularities, changes in respiratory rate and pattern, malaise and weakness, muscle aching and signs of neuromuscular irritability are the possible manifestations of disturbances in homeostasis.

Specific types of gastrointestinal infections

Several gastrointestinal infections produce specific effects. These are discussed below and summarised in Table 23.3.

TABLE 23.3 Selected bacterial infections of the bowel

DISEASE AND ORGANISM	INCUBATION/ DURATION OF ILLNESS	PATHOGENESIS	MANIFESTATIONS	MANAGEMENT
Traveller's diarrhoea: *Escherichia coli*, *Campylobacter*	24–72 hours/ 5–10 days	Enterotoxin causes hypersecretion of the small intestine.	Abrupt onset of diarrhoea; vomiting rare	Prophylactic bismuth subsalicylate; antidiarrhoeals such as loperamide or diphenoxylate; 3- to 5-day course of norfloxacin, ciprofloxacin or trimethoprim-sulfamethoxazole
Staphylococcal food poisoning	1–8 hours/12–24 hours	Enterotoxin impairs intestinal absorption and affects vomiting centres in the brain.	Severe nausea and vomiting; abdominal cramping and diarrhoea; headache and fever	Fluid and electrolyte replacement as needed
Cholera: *Vibrio cholerae*	1–3 days/5–7 days	Enterotoxin affects entire small intestine, causing secretion of water and electrolytes into bowel lumen.	Severe diarrhoea with 'rice-water stool', grey, cloudy, odourless, with no blood or pus; vomiting; thirst, oliguria, muscle cramps, weakness; dehydration and vascular collapse	Oral or intravenous rehydration; possible antimicrobial therapy with antimicrobial agents sensitive to specific *V. cholerae* strain
Haemorrhagic colitis (*E. coli*)	1–3 days/5–10 days	Enterotoxin causes direct mucosal damage in large intestine; also toxic to vascular endothelial cells.	Severe abdominal cramping, watery diarrhoea becoming grossly bloody; fever; possible complications: haemolytic uraemic syndrome and thrombotic thrombocytopenic purpura	Supportive care with fluid replacement and bland diet; may require dialysis or plasmapheresis for complications
Salmonellosis: *Salmonella*	8–48 hours/2–7 days	Superficial infection of the GI tract without invasion or production of toxins.	Diarrhoea with abdominal cramping, nausea and vomiting; low-grade fever, chills, weakness	Treatment of symptoms; trimethoprim sulfamethoxazole, ampicillin or ciprofloxacin for severe illness
Shigellosis (bacillary dysentery): *Shigella*	1–4 days/1–3 days	Local tissue invasion, primarily involving large intestine and distal ileum; endotoxin causes fluid and electrolyte secretion into bowel lumen.	Watery diarrhoea with severe abdominal cramping and tenesmus; lethargy	Fluid and electrolyte replacement; correction of acidosis; antibiotic therapy with trimethoprim sulfamethoxazole, ciprofloxacin or ampicillin
Clostridium difficile colitis (*C. difficile*)	1 day to 8 weeks following antibiotic exposure	Antibiotic therapy interferes with normal protective bacteria in the colon; *C. difficile* colonises and releases toxins causing mucosal inflammation and damage.	Diarrhoea, abdominal cramps, malaise, fever, anorexia	Cessation of the causative antibiotic; antibiotic therapy with vancomycin or metronidazole (specific for *C. difficile*)

Campylobacter infections

Campylobacter species are a major cause of diarrhoeal illness in humans and are generally regarded as the most common bacterial cause of gastroenteritis worldwide. In Australia, as in other developed and developing countries, they cause more cases of diarrhoea than, for example, food-borne *Salmonella* bacteria. In Australia, 14 000 to 17 000 cases are reported annually (Communicable Diseases Network Australia—National Notifiable Diseases Surveillance System (CDNA–NNDSS), 2015). In developing countries, *Campylobacter* infections in children under the age of 2 years are especially frequent, sometimes resulting in death. A fatal outcome is rare and is usually confined to the very young or older adults, or to those already immunosuppressed from another disease (e.g. HIV/AIDS).

The onset of disease symptoms usually occurs 2 to 5 days after infection, but ranges from 1 to 10 days. Usually, *Campylobacter* infections are acquired from eating contaminated and poorly cooked food, especially poultry, red meats and unpasteurised milk. The most common clinical symptoms of *Campylobacter* infections include diarrhoea (frequently with blood in the faeces), abdominal pain, fever, headache, nausea and/or vomiting. The symptoms last typically for 3 to 6 days.

Complications such as bacteraemia, hepatitis, pancreatitis and abortion have been reported with various degrees of frequency. Post-infection complications may include reactive arthritis (painful inflammation of the joints lasting for several months) and neurological disorders such as Guillain–Barré syndrome, a polio-like form of paralysis resulting in respiratory and severe neurological dysfunction or death in a small, but significant, number of cases (Lee & Bishop, 2013). In Australia, as in almost all developed countries, the incidence of human *Campylobacter* infections has been steadily increasing for several years. The reasons for this are unknown.

Traveller's diarrhoea

People travelling overseas frequently develop diarrhoea within 2 to 10 days, particularly when there is a significant difference in climate, sanitation standards or food and drink. Strains of enterotoxin-producing *E. coli*, *Shigella* species, *Salmonella* and *Campylobacter* are the most frequent causes of traveller's diarrhoea (Lee & Bishop, 2013). Other bacteria and viruses also cause traveller's diarrhoea.

Up to 10 or more loose stools per day and abdominal cramping are common manifestations. Nausea and vomiting are less frequent; fever is rare. Manifestations usually resolve within 2 to 7 days. Complications are rare.

Staphylococcal food poisoning

Certain foods rich in protein, salt and sugar provide an excellent medium for staphylococcal growth when contaminated and left at room temperature. Examples include ham, fish, salads with mayonnaise and bakery products (e.g. cream-filled cakes). Foods usually become contaminated by resident staphylococci in the nose or on the skin of food handlers (Lee & Bishop, 2013). The organism itself does not affect the bowel. However, the enterotoxin produced impairs intestinal absorption and acts on receptors in the gut, stimulating the medullary centre, inducing vomiting.

The onset of staphylococcal food poisoning is abrupt, occurring within 1 to 6 hours after consuming the contaminated food. Nausea and vomiting are severe. Manifestations typically include abdominal cramping, diarrhoea, headache and fever lasting 12 to 24 hours. Complications such as fluid and electrolyte imbalances are rare; however, they may develop in older adults and people with underlying chronic disease processes.

Cholera

Cholera is an acute diarrhoeal illness caused by strains of *Vibrio cholerae*. It is endemic in South-East Asia, the Middle East, parts of Africa and most of Central and South America. In endemic areas, water is the primary vehicle of cholera transmission, although secondary transmission may occur via food. Transmission of cholera in non-endemic areas is more commonly associated with consumption of foods, such as raw or undercooked seafood, imported from cholera-endemic regions (WHO, 2015b). Cholera appears to be increasing worldwide in terms of both the number of cases and their distribution. Epidemics occur periodically, often associated with natural disasters (floods and earthquakes) and situations of social unrest and upheaval. Lee and Bishop (2013) outline an outbreak in Kirkuk, Northern Iraq, where a cholera epidemic in 2007 affected more than 30 000 people. Fortunately, as most people were able to access adequate treatment in time, the fatality rate was low, with only 14 deaths.

In Australia, two to six cases (almost all imported from other countries) are reported annually (CDNA–NNDSS, 2015). *V. cholerae* is established in coastal rivers in some parts of Queensland and New South Wales; however, human disease from these sources is rare (Lee & Bishop, 2013). The importance of not eating raw or undercooked seafood from endemic areas was highlighted by Forssman et al. (2007) in their review of the first reported cluster of cholera in Australia for over 30 years, reported in Sydney in November 2006. Three Italian-born women (aged 71, 72 and 84 years) living within a few kilometres of each other but who did not know each other became unwell with severe watery diarrhoea within a 4-day period. It was an unusual outbreak as the women had no history of recent travel or contact with anyone visiting cholera-endemic areas. However, all three women were undergoing long-term therapy with proton-pump inhibitors, which may have contributed to their susceptibility to the disease by reducing their gastric acidity (Forssman et al., 2007). A food trace-back investigation found the only exposure common to all three cases was consumption of raw whitebait imported from Indonesia. All women reported eating cooked seafood before their illness. Forssman et al. (2007) explained that further investigation revealed the women had independently prepared the same dish—whitebait fritters—over the same weekend. During preparation, the women sampled a spoonful of the uncooked fritter mixture of whitebait, eggs, flour and seasoning, for taste and consistency. The mixture was then cooked. As no other food exposures were common to the women, the raw whitebait mixture was considered to be the source of infection.

V. cholerae causes infections only in humans, with symptomatic and asymptomatic carriers being reservoirs of infection (Lee & Bishop, 2013). Cholera is spread by the faecal–oral

route through contaminated water or food. The organism produces an enterotoxin, enzymes and other substances affecting the entire small intestine. The cholera toxin increases the levels of cyclic adenosine monophospate (AMP) in intestinal epithelial cells, resulting in a massive outflow of water and electrolytes into the bowel lumen (Lee & Bishop, 2013). The enzymes and other substances produced by the bacteria may affect mucosal protection of bowel endothelium.

Cholera ranges in severity from very mild, with few or no manifestations, to acute and fulminant. Its onset is typically abrupt, with severe, frequent, watery diarrhoea. Up to 30 L of stool may be passed in a day, rapidly depleting fluid volume. Stool is often described as 'rice-water stool', characteristically grey and cloudy, with no faecal odour, blood or pus. Vomiting may accompany the diarrhoea. Other manifestations related to the loss of fluid and electrolytes include thirst, oliguria, muscle cramps, weakness and significant signs of dehydration. Metabolic acidosis and hypokalaemia develop. Altered fluid, electrolyte and acid–base balance are discussed in Chapter 9.

Among those infected, about 20% develop acute watery diarrhoea, of which 10–20% develop severe watery diarrhoea with vomiting. The mainstay of treatment is rehydration, with up to 80% of cholera cases treated successfully using only oral rehydration salts (WHO, 2015b). If untreated, circulatory collapse and acute kidney injury or dysfunction occurs. The WHO (2015b) warns without fluid and electrolyte replacement, mortality can be as high as 30–50%, mostly observed in crisis situations with overcrowding, limited access to healthcare and precarious environmental management. However, with prompt and adequate fluid replacement, mortality is less than 1%. Recovery from cholera usually occurs spontaneously within 3 to 6 days.

The WHO (2015b) recommends vaccination when travelling to cholera-endemic areas. It is a live vaccine and therefore is used cautiously in immunocompromised individuals, young children and pregnant women. It contains phenylalanine so caution is needed in those with phenylketonuria. The person should be advised to avoid contact with anyone who is immunosuppressed for at least 8 days after taking the vaccine (Tiziani, 2013). Chloroquine (antimalarial prophylaxis) should not be started within 1 week of taking the vaccine. The vaccine should be separated by at least 8 hours from oral typhoid vaccine (Tiziani, 2013).

Escherichia coli haemorrhagic colitis

Most pathological forms of *E. coli* bacteria cause little more than common traveller's diarrhoea. However, some strains—the most common serotype 0157:H7 and *E. coli* 0111—produce potent enterotoxins in the large intestine after being ingested. These toxins damage bowel mucosa and the endothelial cells of blood vessels in the GI tract. If absorbed, the toxin damages other blood vessels as well, such as those of the kidney.

Cattle provide the reservoir for *E. coli* 0157:H7. It is usually spread through undercooked beef (mince, in particular) and unpasteurised milk or apple juice. It may also be spread by direct contact via the faecal–oral route. The onset of haemorrhagic colitis is abrupt, with severe abdominal cramping and watery diarrhoea that becomes grossly bloody within 24 hours. Fever may be present.

Throughout the world, *E. coli* 0157:H7 is the most common serotype associated with haemolytic uraemic syndrome (HUS) (Lee & Bishop, 2013). HUS is characterised by uraemia, thrombocytopenia and acute kidney injury or dysfunction, occurring within the first few days of the infection. HUS is a severe complication of *E. coli* haemorrhagic colitis, affecting about 5% of people with the disease. HUS can occur in any age group; however, it is most common in children under 4 years. Older adults have the highest risk of developing complications. HUS has been a notifiable disease in Australia since 1998, with approximately 10 to 30 cases notified each year (CDNA–NNDSS, 2015).

Clostridium difficile colitis

Clostridium difficile colitis is associated with antibiotic therapy. While antibiotics are recognised as the main predisposing factor, anything disrupting the intestinal flora of the gut, including another gastrointestinal infection, can lead to *C. difficile* infection. The normal colonic flora of adults and children over the age of 1 year effectively prevent the colonisation by *C. difficile*; however, treatment with antibiotics (especially broad-spectrum antibiotics) interferes with the normal protective bacteria of the colon, allowing colonisation of *C. difficile* by the faecal–oral route.

The subsequent release of two exotoxins, A and B, by the bacteria is cytotoxic to a number of cell types, causing mucosal damage and haemorrhage. This induces a local inflammatory response and destruction of the intestinal mucosa, resulting in a pseudomembrane forming consisting of necrotic tissue, mucus, neutrophils and monocytes (Lee & Bishop, 2013) (see Figure 23.6).

C. difficile infection is primarily a problem in hospitalised individuals, in whom infection occurs after ingestion of spores. The spores enable the organism to survive for long periods in the hospital environment. It has been cultured from floors, toilets, bedding, mops and furniture, and also from the hands of healthcare workers (Lee & Bishop, 2013). Transmission of the organism to a susceptible person can readily occur in hospital. It is also being seen in the community in healthy adults.

A hypervirulent strain called ribotype 027, associated with high morbidity and mortality, has emerged and is responsible for hospital outbreaks in North America and Europe.

An infection with *C. difficile* manifests in various ways, from mild diarrhoea and abdominal cramping to the potentially life-threatening pseudomembranous colitis. Perforation of the bowel, abscess formation and vascular thrombi are late complications. Lee and Bishop (2013) advise that *C. difficile* infection should be considered in any person receiving antimicrobial therapy who develops diarrhoea. It commonly begins within 1 to 2 weeks; however, it can commence from 1 day up to 8 weeks after commencing antibiotic treatment (Lee & Bishop, 2013).

The bacteria can be identified in the stool by anaerobic culture and the detection of toxin B in stool filtrate. An enzyme immunoassay method to detect *C. difficile* toxin in the stool

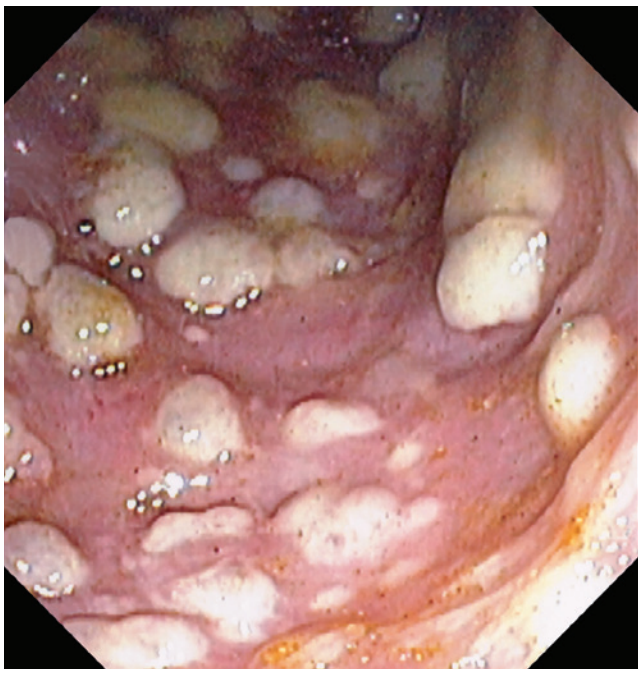


FIGURE 23.6 ■ Pseudomembranous colitis

Source: © SPL/Getty Images.

sample is also commonly used. It is less sensitive than culture, but a much quicker process (Lee & Bishop, 2013).

Salmonellosis

Salmonellosis is food poisoning caused by ingesting raw or improperly cooked meat, poultry, eggs and dairy products contaminated with *Salmonella* bacteria. In developed countries, salmonellae are the second main cause of diarrhoea; 7000–9000 cases are reported annually in Australia (CDNA–NNDSS, 2015). These bacteria do not produce a toxin; they cause superficial infection of the GI tract, rarely invading further.

Manifestations develop from 8 to 48 hours after ingesting the bacteria. Diarrhoea may be violent, with abdominal cramping, nausea and vomiting. A low-grade fever, chills and weakness may accompany GI manifestations. The disease is usually self-limiting, resolving within 3 to 5 days. Many people become asymptomatic carriers for weeks to months, continuing to shed organisms in faeces. Bacteraemia, particularly in the very young, older adults or immunosuppressed individuals, may cause arthritis, osteomyelitis, pneumonia or meningitis when the organism invades the mesenteric lymph nodes (Lee & Bishop, 2013).

Shigellosis (bacillary dysentery)

Shigellosis (or bacillary dysentery) occurs worldwide, accounting for 5–10% of diarrhoeal illness in some regions. It may be endemic or it may occur in epidemics. In Australia, shigellosis is more common in disadvantaged communities and more prevalent among Indigenous children than non-Indigenous children. Approximately 500 to 900 cases are notified annually, with the highest notifications rates in Australian Aboriginal and Torres Strait Islander children less than 4 years of age (CDNA–NNDSS, 2015). Humans are reservoirs for *Shigella* organisms, which spread directly via the faecal–oral route or indirectly through contaminated food, fomites (inanimate objects) and vectors (such as fleas). The incubation period for shigellosis is 1 to 4 days.

Shigella organisms infect the lower intestine and sometimes the distal ilium. They invade the tissue, causing inflammation and producing an enterotoxin. The result is watery diarrhoea containing blood, mucus and inflammatory exudate. The onset of diarrhoea is abrupt, with severe abdominal cramping and *tenesmus*, a sensation of urgent and continuing need to defecate. Lethargy is common; rarely, neurological manifestations occur.

In adults, shigellosis is usually mild and self-limiting. Older adults and immunosuppressed people are at risk of volume depletion and electrolyte imbalances. Secondary infection is another potential complication, as is acute blood loss from mucosal ulcerations.

INTERPROFESSIONAL CARE

The goals of care for gastroenteritis are to manage the manifestations, prevent complications, identify the cause of the infection and prevent its spread. The history and manifestations provide valuable clues about the cause. Diagnostic testing is used to identify the pathogen and evaluate its effects. In most cases, treatment is supportive, directed towards relieving manifestations, restoring fluid and electrolyte balance, and maintaining function.

Diagnosis

If manifestations are severe or do not resolve within about 48 hours, laboratory testing is used to identify the causative organism and to assess fluid, electrolyte and acid–base balance. A stool specimen for culture, ova, cysts and parasites, and faecal leucocytes usually reveals the infective organism. Some bacteria require up to 6 weeks to be identified. In infections such as botulism, the toxin itself may be isolated in the stool. Contamination of the stool by urine or treatment with antibiotics, bismuth subsalicylate or mineral oil interferes with pathogen growth, altering stool culture results. Use a clean bedpan or collection device to obtain the stool specimen and instruct the person to avoid mixing the stool with urine or toilet paper.

A colonoscopy may be done to differentiate inflammatory bowel disease from infectious processes. It does not replace stool cultures, because the lesions associated with some infectious processes are indistinguishable from those of ulcerative colitis. (Nursing care of a person having a colonoscopy is discussed in Chapter 20.)

Serum osmolality and electrolytes and ABGs are done to assess and monitor fluid, electrolyte and acid–base balance. Common imbalances associated with enteritis and diarrhoea are outlined in Table 23.4.

TABLE 23.4 Laboratory values associated with enteritis and diarrhoea

TEST	NORMAL VALUE	CHANGE WITH SIGNIFICANT DIARRHOEA
Serum osmolality	275–295 mOsm/kg	Increased; levels above 320 mOsm/kg indicate significant dehydration.
Serum potassium	3.5–5.0 mmol/L	Decreased due to loss through stool and vomitus; levels below 2.5 mmol/L are critical.
Serum sodium	136–148 mmol/L	Decreased due to loss through stool and vomitus; may be significant when fluid losses are replaced with pure water; levels below 120 mmol/L are critical.
Serum chloride	96–106 mmol/L	Increased when sodium loss is greater than chloride loss; decreased with severe diarrhoea and with vomiting; possible critical values are below 80 mmol/L or above 115 mmol/L.
Blood gases		
• pH	Arterial: 7.35–7.45	Decreased in metabolic acidosis, a possible result of severe diarrhoea; increased in metabolic alkalosis, a possible result of severe vomiting and chloride loss; values below 7.25 or above 7.55 are critical.
• PCO_2	Arterial: 35–45 mmHg	Typically decreased in metabolic acidosis as the body attempts to eliminate excess acid by 'blowing off' CO_2; increased with metabolic alkalosis as the body retains CO_2 in an attempt to normalise pH.
• Bicarbonate	22–26 mmol/L	Decreased in metabolic acidosis; increased in metabolic alkalosis.
Haematocrit	Male: 40–50% Female: 37–47%	Increased with dehydration and hypovolaemia as a result of concentration of blood cells.
Urine specific gravity	1.010–1.025	Increased with dehydration and hypovolaemia as kidneys attempt to conserve fluid.

Medications

Acute enteritis usually resolves spontaneously and no drug treatment is required. If the person is severely ill and manifestations are prolonged, medications may be prescribed.

Antibiotic therapy specific to the organism is used to treat bacterial colitis, cholera, salmonellosis or shigellosis. Trimethoprim-sulfamethoxazole (Septra, Bactrim), ciprofloxacin (Cipro), ampicillin (Ampicin) or another antibiotic may be prescribed. Stool culture is obtained prior to commencing antibiotics. However, treatment may begin before culture results are available. A presumptive diagnosis based on history and presenting manifestations guides the choice of antibiotic. Multidrug-resistant (MDR) strains of *Salmonella* are now encountered frequently and the rate of multidrug resistance has increased considerably since the 1990s (WHO, 2013). Some variants of *Salmonella* have developed multidrug resistance as an integral part of the genetic material of the organism and are likely to retain their drug-resistant genes even when antimicrobial drugs are no longer used, whereas other resistant strains typically lose their resistance. The emergence of MDR *Salmonella* strains with resistance to fluoroquinolones and third-generation cephalosporins is a serious development, severely limiting possibilities for effective treatment of human infections (WHO, 2013).

An antidiarrhoeal drug may be prescribed to promote comfort and reduce fluid loss. Nursing measures related to antidiarrhoeal medications are outlined in the 'Medication administration' box on page 704.

Treatments nutrition and fluids

Replacing lost fluids and electrolytes is vital when vomiting and/or diarrhoea are severe or prolonged. In many cases of enteritis, fluid and electrolyte replacement are all that is required until the infection resolves.

Oral rehydration is preferred for replacing physiological fluids. An oral glucose–electrolyte solution is often well tolerated in sips, even when vomiting is present. Commercial preparations such as Gastrolyte are available.

Intravenous rehydration may be necessary with severe diarrhoea and fluid loss. In some cases, a combination of oral and intravenous fluids is used to replace lost fluids and maintain vascular volume. Balanced electrolyte solutions, such as dextrose in normal saline and Hartmann's solution, are used. Hartmann's solution or another alkalinising solution may be ordered if metabolic acidosis is present.

GASTRIC LAVAGE Gastric lavage and catharsis—in effect, 'washing out' the stomach and intestines—may be performed to remove unabsorbed toxin from the GI tract if botulism is suspected. A person with botulism is closely observed for clinical manifestations of respiratory distress. Respiratory support with endotracheal intubation or tracheostomy and mechanical ventilation may be required (see Chapter 34).

PLASMAPHERESIS Plasmapheresis (plasma exchange therapy) may be performed to remove circulating toxins for haemorrhagic colitis caused by *E. coli*. See Chapter 27 for more information on plasmapheresis. Potential complications include those associated with intravenous catheters, shifts in fluid balance and altered blood clotting.

DIALYSIS Acute tubular necrosis and kidney injury associated with haemorrhagic colitis may necessitate dialysis to remove wastes and prevent severe fluid and electrolyte imbalances, and metabolic acidosis. Although acute kidney injury or

dysfunction often resolves spontaneously and renal function improves, dialysis can be lifesaving. Either haemodialysis or peritoneal dialysis is used, generally as a temporary measure. Nursing care related to acute kidney injury or dysfunction and dialysis is discussed in Chapter 27.

Nursing care

Although *C. difficile* colitis bacterial infections are hospital acquired, few people with acute enteritis require hospitalisation. Most are treated in community settings. Assessment, education and support of self-care measures are the main nursing responsibilities.

Health promotion

Nurses play a significant role in preventing enteritis as educators, community health providers and advocates for environmental safety.

Teach the importance of proper food handling and maintaining appropriate temperatures. Advise those working as food handlers to use disposable paper towels or an air dryer to dry hands rather than cloth towels as cloth towels get dirty quickly and transmit organisms from one person to another.

Adequate cooking of meat products is vital to prevent disorders such as staphylococcal food poisoning, *E. coli* haemorrhagic colitis and salmonellosis. Emphasise the importance of not consuming raw meat products and cooking mince, in particular, to the point where no redness is noted in the meat and the juice from the meat is clear. The highly pathogenic *E. coli* serotype 0157:H7 is present in the gut of infected animals. Meats from the animal may be contaminated with bowel contents. The organism is readily destroyed by heat, so cuts of meat such as steaks or roasts are less likely to cause infection, since the organism is on the outside of the meat. However, the process of mincing meat allows *E. coli* to be mixed throughout the meat. Thorough cooking destroys the organism. This pathogen (and others) may also be spread through unpasteurised milk. Discuss the dangers of consuming unpasteurised milk and encourage people to avoid it. Children should avoid eating unpasteurised and uncooked dairy products, and uncooked meat products such as salami.

Dairy products, eggs and egg products left at room temperature provide a good growth medium for bacteria. Discuss the importance of prompt refrigeration of meats and these products to minimise this risk. Many gastrointestinal infections are spread through contaminated water. Encourage travellers to consume only bottled water and to avoid ice added to drinks unless local water supplies are safe. Water purification tablets are available for bushwalkers and campers and can be used when travelling overseas if local water sources are considered unsafe.

Assessment

- *Health history:* onset, duration and severity of manifestations; recent activities such as attending a picnic or barbecue, camping, international travel or contact with someone recently returned from overseas; other affected members of the household; measures taken to relieve manifestations or replace fluids.
- *Physical examination:* vital signs, including temperature and orthostatic blood pressure; skin colour, temperature, moisture and turgor; peripheral pulses and capillary refill; abdominal shape, contour, bowel sounds, tenderness.

Nursing diagnoses and interventions

Diarrhoea and fluid volume deficit are priority nursing diagnoses. See the earlier section of this chapter on diarrhoea for specific nursing interventions related to these diagnoses. Nausea and vomiting frequently accompany diarrhoea associated with gastroenteritis. Nursing care of a person experiencing nausea and vomiting is discussed in Chapter 22.

Community-based care

Discuss the following topics with the person for self-care:

- the importance of thorough handwashing, with soap and warm running water, for at least 15 seconds, particularly before handling food (preparing food, between handling raw and ready-to-eat foods), before eating, after going to the toilet or changing nappies, and after playing with pets or working in the garden
- the need to wash clothing and linen contaminated with faeces separately in hot water (if available) and detergent
- oral solutions to replace lost fluids and electrolytes
- appropriate use of antidiarrhoeal medications if recommended
- manifestations of complications to report to the healthcare provider.

THE PERSON WITH A PROTOZOAL BOWEL INFECTION

Parasites live within, on or at the expense of other organisms. Parasitic intestinal infections are common in developing countries. These include both protozoal and helminthic (parasitic worm) infections. Parasites infecting the bowel usually enter the GI tract through the mouth by the faecal–oral route; some are spread by direct contact or through sexual activity.

Of the protozoal bowel infections, *giardiasis* is the most common in Australia. Cryptosporidiosis, a form of coccidiosis, is an important worldwide cause of sporadic mild diarrhoea, traveller's diarrhoea and severe diarrhoea in immunocompromised people. Amoebiasis (*Entamoeba histolytica*) is found chiefly in the tropics and where sanitation infrastructure is limited.

Pathophysiology and manifestations

The most common protozoal infections of the bowel are discussed below and summarised in Table 23.5.

TABLE 23.5 Common protozoal infections of the bowel

DISEASE AND ORGANISM	INCUBATION	PATHOGENESIS	MANIFESTATIONS	MANAGEMENT
Giardiasis: *Giardia lamblia*	1–3 weeks or more	Trophozoite attaches to mucosa in duodenum and jejunum, causing superficial invasion, inflammation and tissue destruction.	Diarrhoea, mild or severe, daily or intermittent; anorexia, nausea, vomiting; epigastric pain, cramping, distension; flatulence and belching; may be asymptomatic	Metronidazole quinacrine, furazolidone
Amoebiasis: *Entamoeba histolytica*	2–4 weeks	Organisms may reside in large intestine without causing disease or can invade colon wall, causing ulceration; may be carried via blood to liver to produce abscess.	Usually asymptomatic; diarrhoea may be mild with few semi-formed, mucus-containing stools per day, or severe with 10–20 blood-streaked liquid stools per day; abdominal cramps and flatulence; colic, tenesmus, vomiting, tenderness; fatigue, weight loss; prostration and toxicity	Metronidazole and diloxanide furoate; chloroquine for hepatic abscess
Cryptosporidiosis: *Cryptosporidium*	2–10 days	Organisms attach to epithelial surface of small bowel (jejunum), causing villous atrophy and mild inflammatory changes; may secrete enterotoxin.	In immunocompetent people: asymptomatic to profuse, watery diarrhoea of sudden onset, abdominal cramping; malaise, fever; anorexia, nausea, vomiting. In immunodeficient people: profuse watery diarrhoea with loss of up to 15–20 L/day; severe malabsorption, electrolyte imbalance; weight loss; lymphadenopathy	Self-limiting in immunocompetent people. For immunodeficient people: spiramycin, zidovudine (AZT), paromomycin (Humatin), octreotide, eflornithine; fluid and electrolyte replacement; parenteral nutrition as needed

Giardiasis

Giardiasis is a flagellate protozoal infection of the upper small intestine caused by *Giardia lamblia*, the most common intestinal pathogen worldwide (O'Dempsey, 2014). Humans and other mammals are the reservoir for *Giardia*. Giardiasis occurs where there is overcrowding, or inadequate sanitation or treatment of drinking water. In developed countries, the prevalence rates range between 2% and 7%; the faecal–oral route is the most common form of transmission (O'Dempsey, 2014). It is also spread by direct contact. *Giardia* is readily transmitted in institutions, such as daycare centres, where there is a higher probability of faecal–oral transmission. Other risk factors for infection include travel to high-risk areas, immunosuppression, unprotected anal sexual intercourse and if a person has achlorhydria (Kelly, 2014). In tropical and temperate countries, children are more frequently infected than adults, particularly those who are malnourished (Kelly, 2014).

In Australia, high rates of infection are reported in remote Aboriginal communities. A high prevalence of the parasite has been found in domestic cats and dogs in Australia. However, it is uncertain if they act as a reservoir for human infection (Lee & Bishop, 2013).

When the cyst form of the organism is ingested, trophozoites emerge in the duodenum and jejunum. These attach to the intestinal mucosa, leading to superficial invasion, inflammation and destruction of the mucosa of the small intestine.

Giardiasis is usually asymptomatic or manifests as acute or chronic diarrhoea associated with abdominal cramps, bloating, nausea, vomiting, fever and fatigue, and weight loss. Other manifestations include weight loss and weakness, anorexia, nausea and vomiting, epigastric pain, abdominal distension and cramping, foul flatulence and belching.

Giardiasis is usually self-limiting with symptoms lasting 1 to 2 weeks; however, it may persist for months. Fat malabsorption leads to steatorrhoea (frequent, copious, frothy, malodorous and greasy stools).

In Australia, Aboriginal and Torres Strait Islander children have high rates of chronic giardiasis (Holt, McCarthy & Carapetis, 2010). Chronic giardiasis is associated with fat, d-xylose and vitamin A and B_{12} malabsorption, exacerbating malnutrition which contributes to growth and developmental delays in Aboriginal and Torres Strait Islander children (Holt et al., 2010). Those with chronic infections often manifest with symptomatic episodes alternating with periods when asymptomatic.

Amoebiasis

Amoebiasis (amoebic dysentery) is caused by the protozoan *Entamoeba histolytica*, which is thought to infect as much as

10% of the world's population (Kelly, 2014). Several strains of protozoa have been identified. Tropical strains tend to be more virulent and pathogenic than those found in temperate climates. It is endemic in Mexico, India and Africa, where up to 50% of the population may be infected (Kelly, 2014). It is also endemic in northern parts of Australia, with the highest incidence in the Indigenous population (Lee & Bishop, 2013).

Humans are the host for this parasite. It is usually transmitted through food or water contaminated by faeces, and by person-to-person contact. The parasite enters the intestines, where it lives without causing disease, or it invades the intestinal wall, causing ulceration and inflammation. The caecum, appendix, ascending colon, sigmoid colon and rectum are most often affected. Ulcers may deepen to cause haemorrhage, oedema and mucosal sloughing. The trophozoites of some strains may spread via the blood to the liver, lungs or brain.

Amoebiasis is usually asymptomatic. Mild manifestations include abdominal cramps, flatulence and intermittent diarrhoea containing blood and mucus. Severe manifestations of amoebic dysentery include frequent watery stools containing blood, mucus and necrotic tissue; colic, tenesmus and abdominal tenderness; nausea and vomiting; and fever. The liver may be enlarged and tender to palpation.

Complications are rare, and include appendicitis, bowel perforation with peritonitis and fulminating colitis.

Cryptosporidiosis (coccidiosis)

Cryptosporidiosis causes sporadic mild diarrhoea and traveller's diarrhoea in all age groups. In people with impaired immune function, such as those with human immunodeficiency virus (HIV) disease, it causes severe diarrhoea, malabsorption and significant weight loss.

This organism is transmitted by the faecal–oral route. Contaminated water is a frequent source of infection. *Cryptosporidium* is resistant to the usual levels of chlorine in swimming pools, able to survive for days and may be spread through swallowing contaminated swimming pool water (Lee & Bishop, 2013). High doses of chlorine and the cleaning of filters remove *Cryptosporidium* from a contaminated pool.

The organism attaches to bowel epithelium, causing surface damage and inflammation. It does not invade the tissues but secretes an enterotoxin causing characteristic watery diarrhoea. Watery diarrhoea may be accompanied by low-grade fever, nausea, vomiting, abdominal cramps and general malaise.

The disease is self-limiting in people with competent immune systems. In an immunocompromised person, clinical manifestations include profuse watery diarrhoea with significant fluid and electrolyte losses, and severe malabsorption. Lymphadenopathy (enlarged lymph nodes) may also develop.

INTERPROFESSIONAL CARE

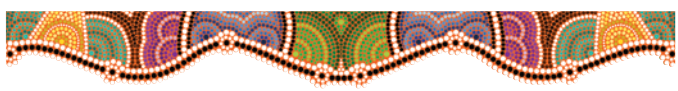
Management of protozoal bowel infections includes identifying the causative organism and administering medications.

Diagnosis

Diagnostic testing includes a stool examination for ova, cysts and parasites and possibly for their antigens. Many protozoa are shed intermittently rather than continuously; stools are collected sequentially (e.g. every other day for a total of three specimens). These organisms are often fragile, requiring a fresh stool specimen. Serology testing for an immune response to the suspected parasite may also be performed. A sigmoidoscopy may be performed to examine the bowel mucosa and collect a stool specimen for examination. (In this case, no bowel prep is done prior to the test.) When giardiasis is suspected, duodenal aspirate may be stained and examined microscopically for the protozoa. Small-bowel biopsy can identify giardiasis or *Cryptosporidium* infection.

Medications

Pharmacological treatment includes local and systemic antiparasitic medications—for example, tinidazole (Fasigyn), cotrimoxazole (trimethoprim with sulfamethoxazole–Bactrim), metronidazole (Flagyl) or albendazole (Eskazole). Treatment is usually provided on an outpatient basis. Severe amoebic dysentery requires hospitalisation for intravenous fluid and electrolyte replacement. Nursing care related to common antiprotozoal drugs is outlined in the 'Medication administration' box below.



Nursing care

Nursing assessment, diagnoses and interventions for the person with a protozoal GI infection are similar to those indicated for people with bacterial or viral infections. *Diarrhoea* and *Risk of deficient fluid volume* are priority nursing diagnoses. See previous sections of this chapter for specific nursing interventions related to these diagnoses.

Nurses need to teach the public how parasitic diseases are transmitted and how to avoid spreading the infection. Prevention of amoebiasis, cryptosporidiosis and giardiasis infections involves the following:

- Maintain good personal hygiene. Everyone should wash their hands thoroughly with soap and warm running water for at least 15 seconds before preparing food and eating, after going to the toilet or after changing nappies, and after cleaning up when someone has vomited. The towels and face washers of someone diagnosed with protozal GI infections should not be used by others.
- Ensure safe food storage, handling and preparation.
- Instruct people living in high-risk areas (e.g. tropical climates, areas with untreated water supplies) to boil, filter or treat water supplies with iodine to eliminate protozoal contamination, and to avoid foods that cannot be peeled or cooked. Teach the manifestations of protozoal infections and where to obtain treatment.
- Emphasise the importance of keeping toilet areas clean, paying particular attention to cleaning surfaces such as

MEDICATION ADMINISTRATION Antiprotozoal agents

LOCAL (GASTROINTESTINAL) AGENTS

Tinidazole (Fasigyn)
Co-trimoxazole (trimethoprim with sulfamethoxazole—Bactrim)
Metronidazole (Flagyl)

These medications exert a local amoebicidal effect in the intestines and are poorly absorbed when administered orally. Local agents have the advantage of provoking fewer side effects than systemically active agents.

Nursing responsibilities

- Assess for potential contraindications or hypersensitivity to the drug or drug class.
- Observe for adverse effects: anorexia, nausea, vomiting, abdominal cramping, diarrhoea or constipation, and increased flatulence; report skin rash, visual disturbances, dizziness, somnolence, vertigo, tremor or changes in blood work (leucopenia, neutropenia) to primary care provider.

Health education for the person and family

- Take as prescribed for the full course of therapy.
- Take with food to reduce gastrointestinal effects.
- Advise against driving or operating machinery if drowsy or dizzy.
- Report any of the following adverse effects to doctor:
  a. any change in vision
  b. numbness, tingling or pain in extremities
  c. chills, fever, skin rash or boils
  d. a change in urination or character of urine
  e. diminished hearing or tinnitus
  f. weight loss, diarrhoea, fatty stools
  g. candidiasis of the mouth or vagina.
- Keep follow-up appointments to evaluate the effects of treatment.

SYSTEMIC AGENTS

Metronidazole (Flagyl)
Pentamidine (Pentamidine Isethionate for injection)
Albendazole (Eskazole)

A person with symptomatic protozoal infections is generally treated with a systemic antiprotozoal agent. Metronidazole is the most widely used of these antiprotozoal agents and is the medication of choice for treating amoebiasis.

Nursing responsibilities

- Assess for possible contraindications to therapy:
  a. hypersensitivity to the prescribed agent or related medications
  b. liver dysfunction or blood dyscrasias
  c. concurrent use of alcohol or a monoamine oxidase inhibitor (MAOI)
  d. pregnancy.
- Administer as ordered.
  a. Metronidazole may be given orally after meals or as a continuous or intermittent intravenous infusion.
  b. Administer albendazole orally with meals to minimise gastric distress.
- Observe for possible adverse effects; notify the doctor if significant. Gastrointestinal effects are common.
  a. Peripheral neuropathy and CNS effects may occur with metronidazole.
  b. Severe hypotension, syncope, cardiac arrhythmias, including ventricular tachycardia and cardiac arrest, may occur with pentamidine.
  c. Blood dyscrasias may develop with albendazole; monitor FBC and report abnormal results.
  d. Pentamidine can cause severe hypoglycaemia; sometimes followed by hyperglycaemia. Carefully monitor blood glucose levels, particularly in people with diabetes mellitus.
  e. Report abnormal liver function test results and increased serum amylase.
- Monitor the character and number of stools; obtain specimens as ordered to evaluate the effectiveness of therapy.

Health education for the person and family

- Take the drug as prescribed for the full duration of the prescription.
- Taking oral preparations after meals helps minimise gastrointestinal side effects. Notify the physician if nausea and vomiting continue.
- Report adverse effects to the doctor, including dizziness and other nervous system changes, sore throats, fatigue, bruising or infection.
- Candidiasis of the mouth or vagina may occur with metronidazole therapy. Report symptoms to doctor.
- A harmless change in urine colour to deep yellow, rust or brown (metronidazole or chloroquine) may occur while taking these drugs.
- If you are a diabetic taking pentamidine, carefully monitor blood glucose levels because hypoglycaemia may develop.

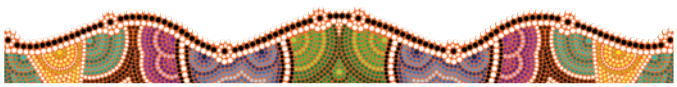
ADVICE REGARDING ALCOHOL

Do not use alcohol while taking these drugs and for 72 hours after stopping tinidazole therapy. A disulfiram–alcohol (Antabuse) type response with severe headache, flushing and vomiting, tachycardia, hypertension initially then hypotension, vertigo, blurred vision, chest pain and, in severe cases, cardiopulmonary arrest may occur. Disulfiram reaction is managed by administering 1 g ascorbic acid intravenously, chlorpromazine 5 to 100 mg intramuscularly and resuscitation measures as required (Tiziani, 2013).

toilet seats and handles, taps and nappy change tables. Ensure all potentially contaminated areas are regularly cleaned and disinfected using a hypochlorite solution of about 1000 ppm (250 mL or 1 cup of household bleach diluted in 10 L or one bucket of water) strength.

- Ensure sandpits do not become contaminated with animal faeces and urine: cover the area when not in use, rake the sand frequently and remove any animal faeces.
- Advise the person to avoid rectal contact during sexual activity. Other household members should also have stool specimens examined for parasites. Following a

cryptosporidiosis infection do not use swimming pools for 2 weeks after diarrhoea stops.
- Provide safe water supplies.
- Dispose of human faeces appropriately.



THE PERSON WITH A HELMINTHIC DISORDER

Helminths are parasitic worms capable of causing infectious diseases in humans. Helminths are subclassified as round worms (nematodes), flukes (trematodes) or tapeworms (cestodes). Parasitic helminths are a major cause of morbidity and sometimes death in humans, particularly in the tropics, subtropics and where sanitation is poor. In Australia, helminth infections occur mostly in the northern tropical parts. However, the most common helminth, the pin (or thread) worm (*Enterobius vermicularis*), is found throughout Australia (Lee & Bishop, 2013). Intestinal nematodes are found in northern Aboriginal communities with poor sanitation and inadequate health facilities. The WHO (2016) warns that infestations compromise nutritional status, affecting cognitive processes, inducing tissue reactions (e.g. granuloma) and provoking intestinal obstruction or rectal prolapse. Control of helminthiasis is based on drug treatment, improved sanitation and health education.

Pathophysiology

Although all helminths can infect humans, the definitive host and intermediate hosts vary with each organism. In nearly all instances of helminthic disorders, the organism enters the body through the GI tract in contaminated and inadequately cooked foods. Some of these organisms remain in the intestinal tract; others migrate to infect the liver, lungs or other structures. Table 23.6 summarises the most common helminths and their effects.

INTERPROFESSIONAL CARE

The management of helminthic disorders includes diagnostic testing and medications.

Diagnosis

The primary means of diagnosing helminthic disorders is examination of the stool for ova and parasites. Enterobiasis is diagnosed by the presence of the parasite's eggs on the perianal skin or on cellulose tape placed over the anus.

A full blood count may also be ordered. Anaemia may be present, particularly with hookworm disease. *Eosinophilia* (an increased percentage of eosinophils in the blood) is common in helminthic disorders. With trichinosis, serum muscle enzymes such as creatinine kinase (CK) and aspartate aminotransferase (AST) are typically elevated. Serological testing for antibodies to the worm may also be performed. Blood, duodenal washings and cerebrospinal fluid (CSF) may be examined for the presence of the trichinosis larvae. Inflamed muscle may be biopsied.

Medications

Helminthic infections are often treated with a single oral dose or a 3-day course of aldendazole (Eskazole) or mebendazole (Vermox). Doses may need to be repeated every 2 weeks for a person with heavy infections. These medications are generally safe, requiring few precautions. Giving the drug after meals minimises GI side effects. Treatment is followed by a stool culture at 2 weeks to evaluate effectiveness. If necessary, an additional course of the medication is prescribed. Other members of the household are generally also treated.

Trichinosis is a serious infection caused by the roundworm *Trichinella spiralis*, acquired by eating raw or undercooked pork contaminated with cysts of the worm. It is not a problem in Australia as the pork sold is currently free of the parasite; however, it is widespread in North America (Lee & Bishop, 2013). Most people with trichinosis recover spontaneously without long-term effects. Hospitalisation may be required during the muscle invasion phase of the disease if the infection is severe. Corticosteroids may be used to reduce the inflammation and manage the clinical manifestations.

Strongyloides stercoralis is a commonly seen threadworm in tropical and subtropical areas (Lee & Bishop, 2013). It is acquired by larval penetration through the skin (see Figure 23.7). If only a few worms are present, the person may be asymptomatic. Heavy infestations cause abdominal pain; rashes and pruritus also occur (Holt et al., 2010). *Strongyloides* infection is often difficult to diagnose and is done by finding larvae in fresh stools. Serological testing is available; however, this does not identify between a past or current acute infection. It is usually treated with ivermectin or thiabedazole. However, worm eradication is difficult, especially in an immunosuppressed person. Immunocompromised or malnourished individuals often have more severe symptoms and repeated regular treatment is required to eradicate the infection (Lee & Bishop, 2013).

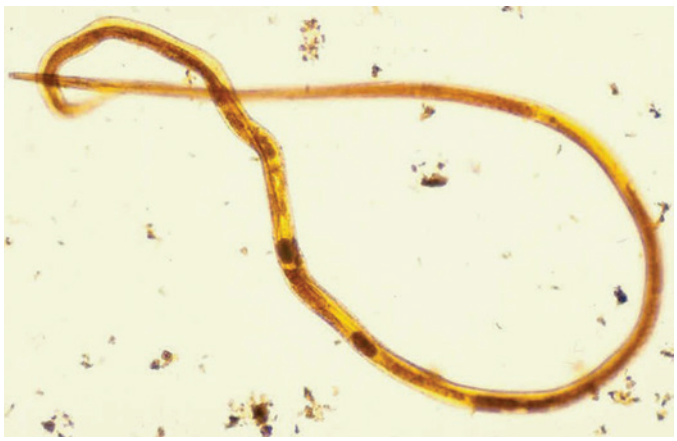
Strongyloides parasites are important causes of morbidity in Indigenous communities in northern Australia and their presence is of particular concern in children, contributing to poor weight gain and failure to thrive (Holt et al., 2010).

Hookworm infections are caused by *Anclostoma duodenale* and *Nector americanus*. In Australia, hookworms are endemic in some remote areas with poor sanitation (Lee & Bishop, 2013). The Aboriginal and Torres Strait Islander population is particularly susceptible due to chronic malnutrition and the practice of walking barefoot, as the larvae burrow through the skin, enter the blood vessels and are carried to the lungs. The larvae are coughed up in sputum. If swallowed, the larvae finally arrive in the intestine, attaching to mucosal cells and establishing an infection (Lee & Bishop, 2013).

Humans are the intermediate host for *Echinococcus granulosis*, causing a condition known as hydatid cysts. Eggs are shed in the faeces and may be transmitted to humans from faeces on the fur or tongue of animals. Eggs hatch in the intestines and migrate to various parts of the body, forming hydatid cysts—large fluid-filled sacs containing thousands of larvae (O'Dempsey & Beeching, 2014). These most commonly form in the liver, lungs and brain, with serious consequences. Young

TABLE 23.6 Selected helminthic diseases

INFECTION	HOST	AREA	PATHOGENESIS	MANIFESTATIONS
Nematode infections				
Ascariasis (roundworm)	Humans	Worldwide, cosmopolitan; warm, moist climates. Common in Australia	Eggs are ingested in faecally contaminated food and drink; motile larvae migrate to lungs and back to small intestine, where they mature to produce more eggs.	Pulmonary: low-grade fever, cough, blood-tinged sputum, wheezing, dyspnoea, substernal chest pain. GI: ulcer-like epigastric pain, vomiting, abdominal distension.
Enterobiasis (pinworm infection)	Humans	Worldwide, cosmopolitan. Common in Australia	Infect caecum; eggs deposit on perianal skin. Organisms may be transmitted to others or reinfect host by oral ingestion.	Nocturnal perianal and perineal pruritus; insomnia, irritability, restlessness.
Hookworm disease	Humans	Tropics and subtropics. Common in remote areas in Australia	Larvae enter through skin or by ingestion and migrate to lungs, up bronchial tree and down oesophagus to mature in upper small bowel, where they attach and suck blood.	Skin: pruritic dermatitis at site of entry. Pulmonary: dry cough, wheezing, blood-tinged sputum. GI: anorexia, diarrhoea, abdominal pain. Systemic: anaemia, pallor, cardiac insufficiency.
Trichinosis	Pigs, dogs, cats, rats, many wild animals	Temperate areas where pork is consumed. Not found in Australian pork	Larvae are ingested in undercooked meat; adult female burrows into mucosa of small intestine to produce larvae that disseminate via blood and lymphatic system to body tissues and become encysted in striated muscle.	GI: diarrhoea, abdominal cramps, malaise. Muscle: fever; muscle pain, tenderness, oedema and spasm. Systemic: periorbital and facial oedema, sweating; photophobia and conjunctivitis; manifestations of inflammation in tissues invaded by larvae.
Cestode infections				
Fasciolopsiasis (intestinal fluke)	Humans; other mammals and fish	Worldwide. Sheep-raising areas in mainland Australia	Organism is ingested by eating uncooked fish or meat containing embryo cysts, by faecal contamination or by swallowing infected intermediate hosts, such as arthropods, fleas or lice. Head (scolex) of adult worm attaches in upper small intestine and eggs form in individual segments.	Large tapeworms: often asymptomatic; infection may cause mild nausea, diarrhoea, abdominal pain, anaemia, thrombocytopenia and mild leucopenia. Small tapeworms: may be asymptomatic; diarrhoea, abdominal pain, anorexia, vomiting, weight loss and irritability.
Tapeworm (beef tapeworm in cattle-raising areas and occasionally dwarf tapeworm found in Northern Australia)	Dogs and cats Humans are intermediate host			
Echinococcus granulosi Hydatid cysts			Eggs of organism transmitted to humans from faeces on fur or tongue of infected animal. Eggs hatch in human intestines, migrating commonly to liver, lungs and brain, forming large fluid-filled sacs—hydatid cysts.	Localised pressure of cyst on the infected organ. Manifestations vary depending on organ involved. Occasional anaphylactic reaction to hydatid antigens when cysts burst.



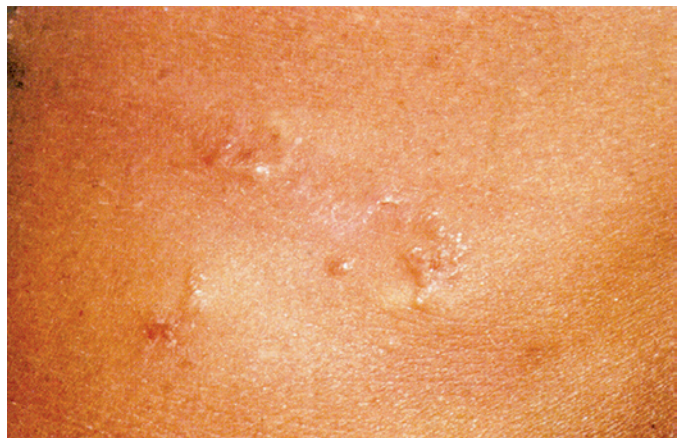


FIGURE 23.7 ■ Strongyloides infection

Source: © Stephen Neville, Department of Microbiology & Infectious Diseases, Sydney South-West Pathology Service, Liverpool Campus.

children in sheep-farming areas are at greatest risk. In Australia, there are approximately 50 notifications of hydatid infection, with an average of three deaths per year (Lee & Bishop, 2013; Queensland Health, 2015).

Nursing care

Because many people with these disorders are asymptomatic, nurses need to be alert for histories indicating risk and subtle manifestations of the disorder. Use standard precautions to minimise the risk of spreading these infections to others. Wear gloves and gowns as necessary to prevent faecal contamination of hands and clothing. On rare occasions, parasites may be present in the sputum or vomitus, so handle these secretions with care. Disinfect toilets, toilet seats and commodes after use. Teach the person the importance of washing hands after using the toilet and before handling food to prevent reinfection.

Discuss measures to prevent spread of the disease in the household. Emphasise the importance of hygiene measures, including changing bedding, daily cleaning of toilets with disinfectant and handwashing.

Many helminthic disorders are acquired by consuming faecally contaminated food or food containing larvae of the organism. Explain the importance of not fertilising food or grain crops with faecal material, particularly human faeces. Teach individuals to cook all meats and fish adequately to destroy possible larvae. In general, pickled or salt-preserved meats and fish are no safer than if raw. Smoking, another means of preserving fish and meat, may not achieve temperatures high enough to destroy the organisms. Vegetables grown in soil that may be contaminated with eggs or larvae should be peeled or cooked prior to eating. Hydatids can be avoided by deworming domestic animals regularly and not feeding them uncooked meat.

Emphasise the importance of safe water supplies. Encourage people travelling to areas where water supplies are questionable to drink only bottled water or carry purification tablets. Work with individuals with private water systems to protect water from faecal contamination by either humans or animals.

A person with a helminthic disorder may feel dirty or be ashamed of the disease. Emphasise the prevalence of these disorders, assuring the person that infections can occur despite good health practices when the eggs or larva of the organism are prevalent.

CHRONIC INFLAMMATORY BOWEL DISORDERS

THE PERSON WITH INFLAMMATORY BOWEL DISEASE

Chronic **inflammatory bowel disease (IBD)** includes two separate but closely related conditions: ulcerative colitis and Crohn's disease. These conditions have a number of similarities. The aetiology of both illnesses is unknown, although current evidence implicates both genetic and environmental factors. The geographical distribution of ulcerative colitis and Crohn's disease is similar worldwide, the highest incidences being in the United States, Canada, the United Kingdom and Scandinavia (Crohn's & Colitis Foundation of America (CCFA), 2009).

IBD affects certain ethnic groups more than others: it is prevalent among Ashkenazi Jews and decreases progressively

in other people of Jewish descent, in non-Jewish Caucasians, Africans, Hispanics and Asians. In Australia, it affects approximately 1 in 250 people aged 5–40. Almost 75 000 Australians have Crohn's disease or ulcerative colitis, with this number projected to increase to 100 000 by 2022 (Crohn's & Colitis Australia (CCA), 2014).

Irritable bowel disease tends to run in families, with 20–25% of people having a close relative with one of the types of IBD (CCA, 2014). Factors such as an abnormal immune response to microogranisms normally found in the gut are thought to play a role in IBD development. IBD has been linked to the recently discovered TH cell (TH 17), certain cytokines and a deficit of antimicrobial substances (lysoenzyme, defensins and other) secreted by the gut mucosa (Marieb & Hoehn, 2013). Autoimmunity is thought to play a role (see Chapter 12) and lifestyle factors (such as smoking) may also affect its development. Smoking certainly worsens Crohn's disease. CCA (2014) stresses that those who stop smoking have a 65% lower risk of flare-up (comparable to the benefit conferred by drugs such as Imuran) than continuing smokers. Children developing Crohn's disease are more likely to have been exposed to passive smoking at home (CCA, 2014).

The peak incidence of IBD is in adolescents and young adults between the ages of 15 and 35 years but it can also affect older adults (Solomon & Flum, 2015). IBD is a chronic and recurrent disease process. Responses to physiological or psychological stresses do not cause IBD, but often play a role in exacerbations of the disease.

Despite the similarities, ulcerative colitis and Crohn's disease have distinct differences. Ulcerative colitis primarily affects the large bowel in a continuous pattern, progressing distally to proximally. In Crohn's disease, a patchy pattern of involvement is seen, affecting primarily the small intestine. Ulcerative colitis shows mainly mucosal involvement; in Crohn's disease, the submucosal layers of the bowel are affected. A comparison of ulcerative colitis and Crohn's disease is found in Table 23.7. The multisystem effects of inflammatory bowel disease are illustrated below.

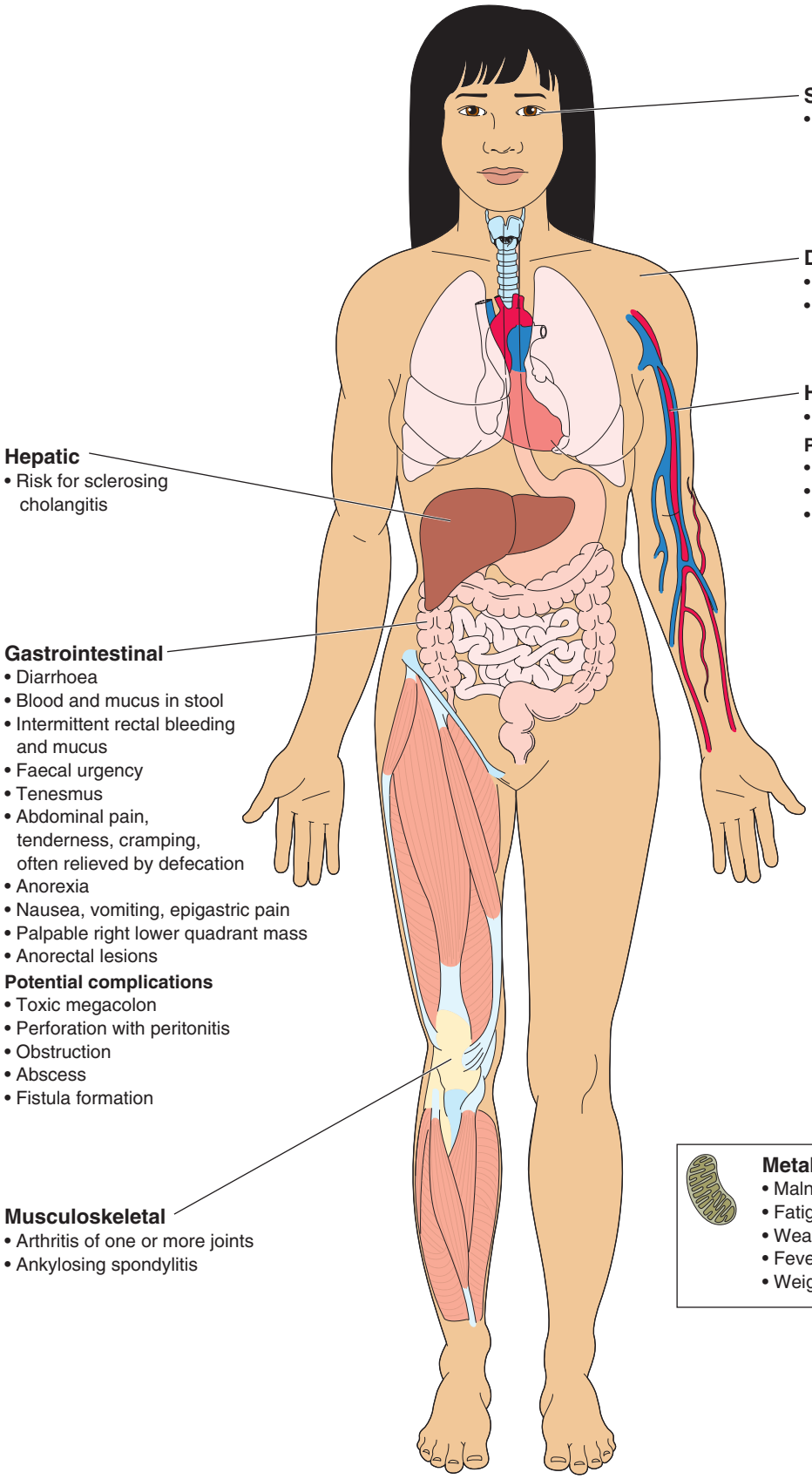
Ulcerative colitis

Ulcerative colitis is a chronic inflammatory bowel disorder affecting the mucosa and submucosa of the colon and rectum. *Chronic intermittent colitis* (recurrent ulcerative colitis) is the most common form of the disease and affects 33 000 people in Australia each year, resulting in steep hospital and drug costs as well as lost work (CCA, 2014). Most people with ulcerative colitis have mild or moderate disease, with six or fewer stools per day. Its onset is insidious, with attacks lasting 1 to 3 months occurring at intervals of months to years. Typically, only the distal colon is affected, with few systemic manifestations of the disease. Approximately 15% of people with ulcerative colitis develop *fulminant colitis*, with the entire colon involved, severe bloody diarrhoea, acute abdominal pain and fever. Those with fulminant disease are at high risk of complications.

TABLE 23.7 Characteristics of ulcerative colitis and Crohn's disease

	CHARACTERISTIC	ULCERATIVE COLITIS	CROHN'S DISEASE
Clinical	Gender	Equal	Equal
	Age at onset	15 to 35 years; secondary peak between 60 and 80 years	15 to 30 years; secondary peak between 60 and 80 years
	Course of disease	Typically chronic and intermittent	Slowly progressive, relapsing
	Diarrhoea	5 to 30 stools per day with blood and mucus	Common, usually less severe than colitis, with no obvious blood or mucus in stool
	Abdominal pain	Cramping in left lower quadrant; relieved by defecation	Cramping or steady right lower quadrant or periumbilical pain; tenderness and mass noted in right lower quadrant
	Nutritional deficit	Common; involves anaemia, hypoalbuminaemia and weight loss	Common and significant: involves anaemia, weight loss and multiple vitamin and mineral deficits
	Constitutional manifestations	Fever rare; may have associated arthritic, skin or other organ involvement, such as erythema nodosum or uveitis	Fever, malaise, fatigue; may have some associated conditions plus urinary complications
Pathological	Depth of involvement	Mucosa and submucosa	Transmural (entire bowel wall)
	Portion of bowel involved	Typically rectum and sigmoid colon; may extend to involve entire large bowel	Any portion of GI tract; terminal ileum and ascending colon involvement predominates
	Distribution	Continuous from rectum	Patchy; skip lesions
	Appearance of mucosa	Granular, dull, hyperaemic, friable; disease uniform in affected bowel; pseudopolyps may be seen	Cobblestone appearance, with areas of normal tissue surrounded by ulceration and fissures
Complications	Acute	Toxic megacolon, perforation, massive haemorrhage	Obstruction, fistulisation, abscess formation, malabsorption
	Long term	Colorectal cancer	Colon cancer

MULTISYSTEM EFFECTS OF INFLAMMATORY BOWEL DISEASE



Sensory
- Uveitis

Dermatological
- Skin lesions
- Mucous membrane lesions

Haematological
- Anaemia

Potential complications
- Thromboemboli
- Haemorrhage
- Hypovolaemia

Hepatic
- Risk for sclerosing cholangitis

Gastrointestinal
- Diarrhoea
- Blood and mucus in stool
- Intermittent rectal bleeding and mucus
- Faecal urgency
- Tenesmus
- Abdominal pain, tenderness, cramping, often relieved by defecation
- Anorexia
- Nausea, vomiting, epigastric pain
- Palpable right lower quadrant mass
- Anorectal lesions

Potential complications
- Toxic megacolon
- Perforation with peritonitis
- Obstruction
- Abscess
- Fistula formation

Musculoskeletal
- Arthritis of one or more joints
- Ankylosing spondylitis

Metabolic processes
- Malnutrition
- Fatigue
- Weakness
- Fever
- Weight loss

Pathophysiology

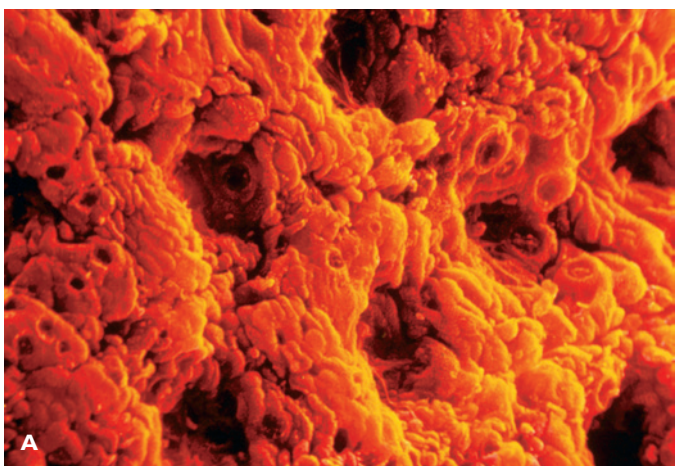
The inflammatory process of ulcerative colitis begins at the rectosigmoid area of the anal canal and progresses proximally. In most individuals, it is confined to the rectum and sigmoid colon. It may progress to involve the entire colon, stopping at the ileocaecal junction.

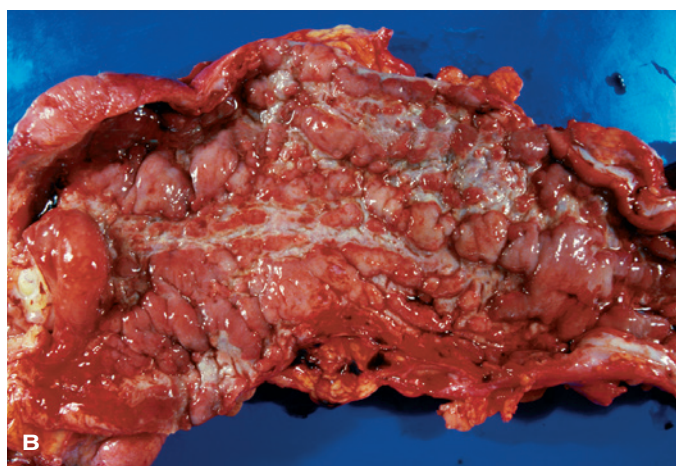
Ulcerative colitis begins with inflammation at the base of the crypts of Lieberkühn in the distal large intestine and rectum. Microscopic, pinpoint mucosal haemorrhages occur and crypt abscesses develop (see Figure 23.8). These abscesses penetrate the superficial submucosa and spread laterally, leading to necrosis and sloughing of bowel mucosa. Further tissue damage is caused by inflammatory exudates and the release of inflammatory mediators, such as prostaglandins and other cytokines (see Chapter 11 for further discussion of the inflammatory process). The mucosa is red and oedematous due to vascular congestion, friable (easily broken) and ulcerated. It bleeds easily and haemorrhage is common. Oedema creates a granular appearance. Pseudopolyps, tongue-like projections of bowel mucosa into the lumen, may develop as the epithelial lining of the bowel regenerates. Chronic inflammation leads to atrophy, narrowing and shortening of the colon, with loss of its normal haustra.

Manifestations

Diarrhoea is the predominant manifestation of ulcerative colitis. Stools contain both blood and mucus. Nocturnal diarrhoea may occur. Mild ulcerative colitis is characterised by fewer than 4 stools per day, intermittent rectal bleeding and mucus, and few systemic manifestations. Severe ulcerative colitis leads to more than 6 to 10 bloody stools per day, extensive colon involvement, anaemia, hypovolaemia and malnutrition. Rectal inflammation causes faecal urgency and tenesmus. Left lower quadrant cramping relieved by defecation is common. Other manifestations include fatigue, anorexia and weakness.

Individuals with severe disease may also have systemic manifestations such as arthritis involving one or several joints, skin and mucous membrane lesions, or *uveitis* (inflammation of the uvea, the vascular layer of the eye, which may also involve the sclera and cornea). Some individuals develop thromboemboli, with blood vessel obstruction due to clots carried from the site of their formation. The liver and biliary system may be affected by the disease as may the kidneys, with an increased risk of gallstones, cirrhosis, renal calculi and ureteral obstruction (Martin, Chan & Hart, 2014).





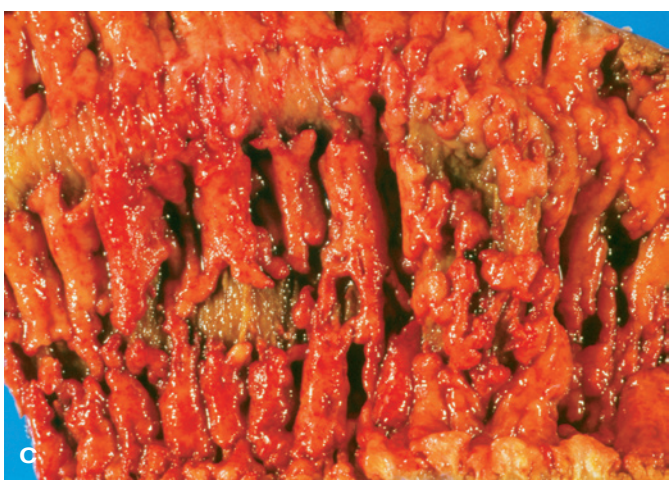


FIGURE 23.8 ■ *A*, Photomicrograph of the mucosa of the large intestine showing the entrances to the crypts of Lieberkühn. The crypts are the focal points for *B*, ulcerative colitis and *C*, Crohn's disease

Sources: A, © CNRI/Science Source; B, © Dr E. Walker/Science Source; C, © J. Watson/Custom Medical Stock Photo.

Complications

Most people with ulcerative colitis (80–90%) respond well to treatment and do not develop complications (CCA, 2014). Acute complications of ulcerative colitis include haemorrhage, toxic megacolon and colon perforation. Massive haemorrhage may occur with severe attacks of the disease. *Toxic megacolon* is a condition characterised by acute motor paralysis and dilation of the colon to greater than 6 cm, affecting part or the entire colon. The transverse segment of the bowel is most commonly affected. Toxic megacolon may be triggered by the use of laxatives, narcotics and anticholinergic drugs, and the presence of hypokalaemia (Devuni, 2014). Manifestations of toxic megacolon include fever, tachycardia, hypotension, dehydration, abdominal tenderness and cramping, and a change in the number of stools per day. Perforation is rare; however, this is a dangerous complication increased with toxic megacolon. Perforation leads to peritonitis.

The risk of colorectal cancer is increased in people with ulcerative colitis. Beginning 8 to 10 years after diagnosis, annual or biennial colonoscopies with biopsy to detect masses or cell dysplasia are recommended for individuals with extensive ulcerative colitis (Devuni, 2014).

Crohn's disease

Like ulcerative colitis, **Crohn's disease**, also known as regional enteritis, is a chronic, relapsing inflammatory disorder affecting the gastrointestinal tract. Crohn's disease can affect any portion of the GI tract from the mouth to the anus, but usually affects the terminal ileum and ascending colon. Only the small bowel is involved in about 30–40% of people with Crohn's disease. The disease is limited to the colon only in 30% of those affected. Both the small and the large intestine are involved in the remaining 30% of individuals (Porth & Matfin, 2013).

Pathophysiology

Crohn's disease typically begins as a small inflammatory *aphthoid lesion* (shallow ulcers with a white base and elevated margin, similar to a canker sore) of the mucosa and submucosa of the bowel. These initial lesions may regress or the inflammatory process can progress to involve all layers of the intestinal wall. Deeper ulcerations, granulomatous lesions and fissures (knife-like clefts extending deeply into the bowel wall) develop. The inflammatory process involves the entire bowel wall (transmural).

The lumen of the affected bowel assumes a 'cobblestone appearance' as fissures and ulcers surround islands of intact mucosa over oedematous submucosa. The inflammatory lesions of Crohn's disease are not continuous; rather, they often occur as 'skip' lesions with intervening areas of normal-appearing bowel. Some evidence suggests that, despite its normal appearance, the entire bowel is affected by this disorder.

As the disease progresses, fibrotic changes in the bowel wall cause thickening and decreased flexibility of the bowel (which takes on an appearance likened to a rubber hose). The inflammation, oedema and fibrosis can lead to local obstruction, abscess development and the formation of fistulas between loops of the bowel or the bowel and other organs (see Figure 23.9). Fistulas between loops of the bowel are called enteroenteric fistulas. Enterovesical fistulas occur between the bowel and bladder, and enterocutaneous fistulas occur between the bowel and skin. Perineal fistulas originating in the ileum are relatively common.

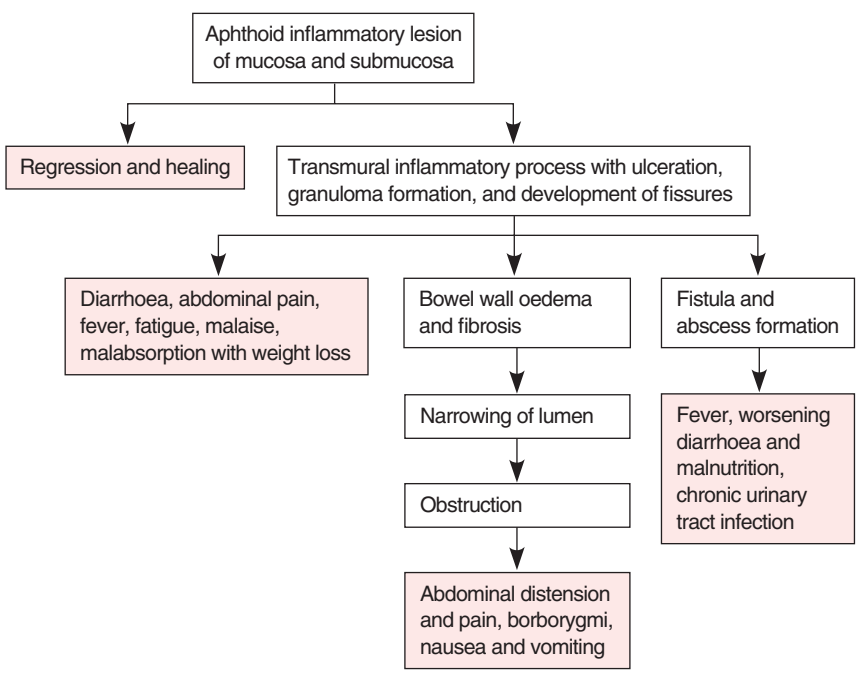


FIGURE 23.9 ■ The progression of Crohn's disease

Depending on the severity and extent of the disease, malabsorption and malnutrition may develop as the ulcers prevent absorption of nutrients. When the jejunum and ileum are affected, the absorption of multiple nutrients may be impaired, including carbohydrates, proteins, fats, vitamins and folate. Disease in the terminal ileum can lead to vitamin B_{12} malabsorption and bile salt reabsorption. The ulcerations also lead to protein loss and chronic, slow blood loss with consequent anaemia.

Manifestations

The diverse nature of GI system involvement in Crohn's disease means that clinical manifestations vary between individuals. The majority of people with Crohn's disease experience persistent diarrhoea. Stools are liquid or semi-formed and typically do not contain blood, although blood may be passed if the colon is involved. Abdominal pain and tenderness are also common. Pain may be located in the right lower quadrant and relieved by defecation. A palpable right lower quadrant mass is often present. Systemic manifestations such as fever, fatigue, malaise, weight loss and anaemia are common. Anorectal lesions such as fissures, ulcers, fistulas and abscesses are also common and may occur years before intestinal disease is apparent. If the stomach and duodenum are involved, nausea, vomiting and epigastric pain occur.

Complications

Certain complications of Crohn's disease (e.g. intestinal obstruction, abscess and fistula) are so common that they are considered part of the disease process. For many people, the disease initially presents with one of these complications. Intestinal obstruction is a common complication caused by repeated inflammation and scarring of the bowel, leading to fibrosis and stricture. Obstruction of the bowel lumen causes abdominal distension, cramping pain and borborygmi. Nausea and vomiting may occur.

Fistulas may be asymptomatic, particularly those occurring between loops of small bowel. When fistulisation causes an abscess, chills and fever, a tender abdominal mass and leucocytosis develop. A fistula between the small bowel and colon may exacerbate diarrhoea, weight loss and malnutrition. When the bladder is involved, recurrent urinary tract infections occur.

Perforation of the bowel is uncommon. However, it can lead to generalised peritonitis. Massive haemorrhage also is an uncommon complication of Crohn's disease. Long-standing Crohn's disease increases the risk of cancer of the small intestine or colon by 5 to 6 times, which is significantly lower than the risk associated with ulcerative colitis.

INTERPROFESSIONAL CARE

Interprofessional care for inflammatory bowel disease begins by establishing the diagnosis and the extent and severity of the disease. Treatment is supportive, including medications and dietary measures to decrease inflammation, promote intestinal rest and healing, and reduce intestinal motility. Many people with IBD require surgery at some point to manage the disease or its complications.

Diagnosis

Diagnostic testing is used to establish the diagnosis of IBD, assess the extent of the disease and evaluate the effects of the disorder. A colonoscopy or barium upper and lower x-ray series is performed to inspect the bowel mucosa for characteristic changes of IBD. (Nursing implications for these diagnostic tests are outlined in Chapter 20.)

Laboratory tests to differentiate IBD and identify effects and complications of the disease include a stool examination for blood and mucus and stool cultures to rule out infectious causes of bowel inflammation and diarrhoea. FBC with haemoglobin and haematocrit shows anaemia from chronic inflammation, blood loss and malnutrition, and leucocytosis due to inflammation and possible abscess formation. ESR and C-reactive protein levels are typically elevated during periods of acute inflammation. Serum albumin may be decreased because of malabsorption, malnutrition, protein loss through intestinal lesions and chronic inflammation. Folic acid and serum levels of most vitamins, including A, B complex, C and the fat-soluble vitamins are often decreased due to malabsorption. Liver function tests may show elevated liver enzymes (such as ALT, ALP, AST, GGTP and LDH) and bilirubin levels if sclerosing cholangitis is present.

Medications

The ultimate goal of care is to terminate acute attacks as quickly as possible and reduce the incidence of relapse. Drug therapy plays a key role in achieving this goal. Locally acting and systemic anti-inflammatory drugs are the primary medications used to manage mild to moderate IBD. Medications suppressing the immune response may be used to treat people with severe disease.

Sulfasalazine (Azulfidine) is a sulfonamide antibiotic poorly absorbed from the gastrointestinal tract and acts topically on the colonic mucosa to inhibit the inflammatory process. The active anti-inflammatory ingredient in sulfasalazine, 5-aminosalicylic acid (5-ASA), is also available in sulfa-free preparations, such as olsalazine and mesalazine. These have the advantage of causing fewer adverse effects than sulfasalazine. Azo compounds, such as balsalazide and olsalazine, are 5-aminosalicylic acid compounds released in the colon and are especially useful to treat ulcerative colitis. Mesalamine is an orally or rectally administered 5-ASA compound providing topical anti-inflammatory action in the colon of individuals with ulcerative colitis. Specific preparations, their method of action and nursing implications for these medications are outlined in the 'Medication administration' box below.

For acute exacerbations of IBD, corticosteroids are given to reduce inflammation and induce remission. For ulcerative colitis, the drug may be administered rectally for its local effect and to minimise systemic effects. Hydrocortisone can be administered rectally. Intravenous corticosteroids may be required to treat severe disease; oral preparations are used for less severe manifestations and long-term therapy. Corticosteroids are tapered off once remission is achieved. However,

MEDICATION ADMINISTRATION Inflammatory bowel disease

SULFASALAZINE (SALAZOPYRIN)

Sulfasalazine is an anti-inflammatory drug used for its local effect on the intestinal mucosa in inflammatory bowel disease. The active part of the drug, 5-aminosalicylic acid, inhibits prostaglandin production in the bowel. Prostaglandin is an important mediator of the inflammatory process; blocking its production reduces inflammation.

Nursing responsibilities

- Assess for contraindications, including pregnancy or a history of hypersensitivity to sulfonamides or salicylates.
- Assess baseline values for renal function tests (serum creatinine, BUN, urinalysis), liver function tests and FBC. Monitor these routinely during therapy.
- Administer as ordered. Suppositories or retention enemas may be administered at bedtime. Administer oral forms with a full glass of water.
- Have resuscitation equipment available, as anaphylactic responses may occur.
- Evaluate for therapeutic response, including reduced number of stools, reduced mucus and blood, and improved stool consistency. Onset of action may take 6 to 12 weeks.
- Monitor for possible adverse responses:
  a. skin rash, dermatitis, urticaria or pruritus
  b. evidence of blood dyscrasias, such as bleeding, easy bruising or fever
  c. leucopenia, thrombocytopenia, haemolytic anaemia or agranulocytosis
  d. changes in urinary output or renal function analysis (including urinalysis)
  e. evidence of hepatitis or myocarditis.

Health education for the person and family

- Take oral preparations after meals to decrease gastric distress.
- Drink at least 2–3 L of fluid per day to reduce the risk of kidney damage.
- Use sunscreen to prevent burns; this drug increases sensitivity to sun.
- Do not take aspirin, vitamin C or any other over-the-counter medications containing aspirin or vitamin C without consulting your doctor.
- This medication may interfere with the effectiveness of oral contraceptives; use alternative methods of contraception.
- Notify your doctor if you develop skin rash or hives, a sore throat or mouth, bleeding gums or joint pain, or bruise easily or develop fever.

MESALAZINE (MESASAL) AND OLSALAZINE (PREDSOL)

Mesalamine and olsalazine contain the same active ingredient, 5-aminosalicylic acid, as sulfasalazine; however, they cause fewer adverse effects. Their mechanism of action is the same as sulfasalazine. These drugs are available as suppositories, suspension for enema or oral tablets.

Nursing responsibilities

- Assess for possible contraindications such as pregnancy, lactation or hypersensitivity to these drugs or aspirin.
- Administer as ordered. If more than one dose per day is ordered; space doses evenly over the 24-hour period.
- Evaluate for desired effects (as for sulfasalazine) and potential adverse effects:
  a. nausea, diarrhoea, abdominal cramps or flatulence
  b. CNS effects, including headache, dizziness, insomnia, weakness or fatigue
  c. rash or itching
  d. flu-like symptoms or general malaise.

Health education for the person and family

- Teach the recommended method of administration, including how to insert rectal suppositories or administer a retention enema.
- Shake suspension forms well prior to using.
- Diarrhoea is the most common side effect of these drugs. Notify your doctor if adverse effects occur.

CORTICOSTEROIDS

Methylprednisolone (Medrol, Solu-Medrol)
Prednisolone (Delta-Cortel) Prednisone

Glucocorticoids are hormones produced by the adrenal cortex. See Chapter 18 for discussion on hormones and stress response. These hormones are necessary for the stress response. Cortisol, the main glucocorticoid, has potent anti-inflammatory effects. Corticosteroids are used to treat acute episodes of IBD. Because of their multiple and significant side effects, they are not used to maintain remission.

Nursing responsibilities

- Assess for conditions adversely affected by corticosteroid medications: peptic ulcer disease, glaucoma or cataracts, diabetes mellitus or psychiatric disorders.
- Obtain baseline vital signs and weight; monitor both routinely during therapy. Hypertension and weight gain may result from sodium and water retention.
- Monitor for oedema.
- Administer as ordered. For daily or alternate-day dosing, administer in the morning, when physiological glucocorticoid levels are highest, to reduce adrenal cortisone suppression.
- Administering oral preparations with food decreases gastrointestinal side effects. Antacids or histamine H_2-receptor blocking agents may be prescribed during corticosteroid therapy.
- Monitor for desired effects: reduced diarrhoea, less blood and mucus in the stool and less abdominal cramping.
- Monitor for adverse effects:
  a. increased susceptibility to infection and masking of early signs of infection
  b. hyperglycaemia
  c. hypokalaemia, as manifested by muscle weakness, nausea, vomiting and cardiac rhythm disturbances
  d. oedema, hypertension and signs of heart failure
  e. peptic ulcer formation and possible gastrointestinal haemorrhage (abdominal pain, black or tarry stools, and signs of bleeding)

(continued)

MEDICATION ADMINISTRATION Inflammatory bowel disease (continued)

f. changes in mental status, including depression, euphoria, aggression and behavioural changes
g. with long-term use, Cushingoid effects, such as abnormal fat deposits in the face (moon facies) and trunk (buffalo hump), muscle wasting and thin extremities, thinning of the skin and osteoporosis. See Chapter 18 for discussion of nursing care of a person with hypercortisolism (Cushing's syndrome).

Health education for the person and family

- Take as prescribed; do not change the dose or time of day. Do not stop the medication abruptly. The dose will be tapered down gradually when the medication is discontinued.
- Notify the doctor if adverse or Cushingoid effects occur.
- Take with food or at mealtimes to decrease the gastrointestinal effects.
- Monitor weight. If more than 2.5 kg gain is noted, notify the doctor.
- Moderate salt intake and avoid foods and snacks high in sodium, such as processed meats and potato chips. Increase intake of foods high in potassium, such as fruits, vegetables and lean meats.
- Carry a card or wear a MedicAlert® bracelet or tag at all times identifying corticosteroid use.

many individuals are unable to withdraw from steroid therapy without experiencing relapse and require long-term, low-dose therapy.

Mercaptopurine (6-MP, Purinethol) and other immunosuppressive agents such as azathioprine (Imuran) and cyclosporin (Sandimmun) are used to treat individuals unresponsive to other treatments or who require long-term steroid therapy. These drugs may allow withdrawal of corticosteroids, maintain remission and facilitate healing. Long-term therapy may be required to produce a beneficial effect. For more information about immunosuppressive medications, see Chapter 12.

Newer treatments for IBD employ other immune response modifiers, such as the monoclonal antibody infliximab (Remicade) to suppress tumour necrosis factor (TNF, an inflammatory mediator substance) in those unresponsive to standard therapies. Mesalazine (Mesal) is an orally or rectally administered anti-inflammatory medication providing topical anti-inflammatory action in the colon of individuals with ulcerative colitis.

Although antibiotic therapy generally is not indicated in IBD, metronidazole (Flagyl) has active anti-inflammatory effects. It may be prescribed to help prevent remission after ileal resection in Crohn's disease. Ciprofloxacin (Cipro) is an alternative to metronidazole.

Antidiarrhoeal agents, such as loperamide and diphenoxylate, may be given to slow gastrointestinal motility and reduce diarrhoea. These drugs are safe for individuals with mild, chronic manifestations. However, these are not given during acute attacks because they may precipitate toxic dilation of the colon.

Nutrition

Antigens in the diet may stimulate the immune response in the bowel, exacerbating IBD. As a result, dietary management for inflammatory bowel disease is individualised. Some people benefit from eliminating all milk and milk products from the diet. Increased dietary fibre may help reduce diarrhoea and relieve rectal manifestations. However, this is contraindicated for individuals with intestinal strictures caused by repeated inflammation and scarring.

All food may be withheld to promote bowel rest during an acute exacerbation of Crohn's disease. Nutritional status is maintained using enteral or total parenteral nutrition (TPN). See Chapter 21 for more information about enteral feedings and TPN. TPN carries a higher risk of complications than does enteral nutrition.

Surgery

Surgical interventions for IBD differ, depending on the primary disease process and the portion of bowel affected. Generally, surgery is performed only when necessitated by complications of the disease or failure of conservative treatment measures.

Bowel obstruction is the leading indication for surgery in Crohn's disease. Other complications requiring surgical intervention include perforation, internal or external fistula, abscess and perianal complications. Resection of the affected portion of bowel with an end-to-end anastomosis to preserve as much bowel as possible is the usual treatment. The disease process tends to recur in other areas following removal of affected bowel segments. There is an increased risk of fistula formation following surgery. Bowel strictures may be treated with a stricturoplasty in which longitudinal incisions are made in the narrowed segment, relieving the stricture while preserving bowel.

COLECTOMY Individuals with extensive chronic ulcerative colitis may require a total **colectomy** (surgical resection and removal of the colon) to treat the disease itself; for complications such as toxic megacolon, perforation or haemorrhage; or as a prophylactic measure due to the high colon cancer risk associated with extensive ulcerative colitis.

The surgical procedure of choice for extensive ulcerative colitis is a total colectomy with an *ileal pouch–anal anastomosis (IPAA)*. In this procedure, the entire colon and rectum are removed; a pouch is then formed from the terminal ileum and is brought into the pelvis and anastomosed to the anal canal (see Figure 23.10). A temporary or loop ileostomy (described in the next section) is generally performed at the same time and maintained for 2 to 3 months, allowing the anal anastomosis to heal. When the healing is complete, the ileostomy is closed. The person then has six to eight

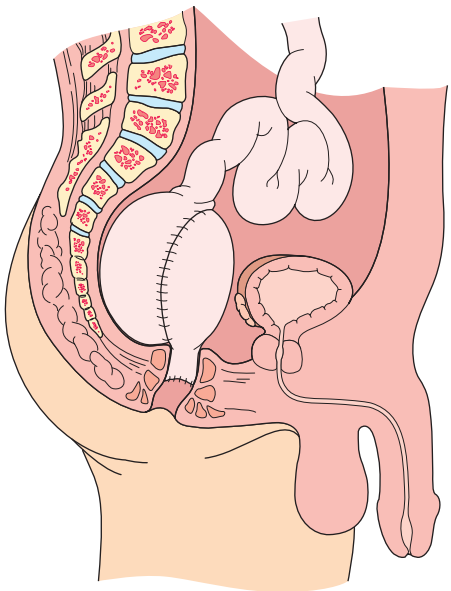


FIGURE 23.10 ■ Ileal pouch–anal anastomosis (IPAA)

daily bowel movements through the anus. Advanced age, obesity or other factors may preclude an IPAA. For these individuals, a permanent ileostomy or continent ileostomy may be created.

OSTOMY An intestinal ostomy is a surgically created opening between the intestine and the abdominal wall that allows the passage of faecal material. The surface opening is called a **stoma** (see Figure 23.11). The precise name of the ostomy depends on the location of the stoma. An **ileostomy** is an ostomy made in the ileum of the small intestine. In an ileostomy, the colon, rectum and anus are usually completely removed (*total proctocolectomy with permanent ileostomy*). The anal canal is closed and the end of the terminal ileum is brought to the body surface through the right abdominal wall to form the stoma. A *temporary* or *loop ileostomy* may be formed to eliminate faeces and allow tissue healing for 2 to 3 months

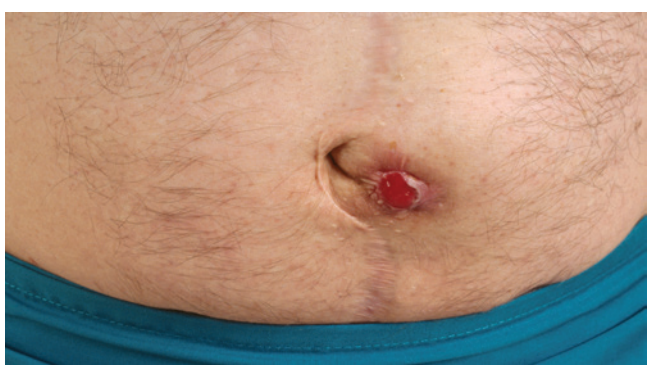


FIGURE 23.11 ■ A healthy-appearing stoma

Source: CMSP/Custom Medical Stock Photo.

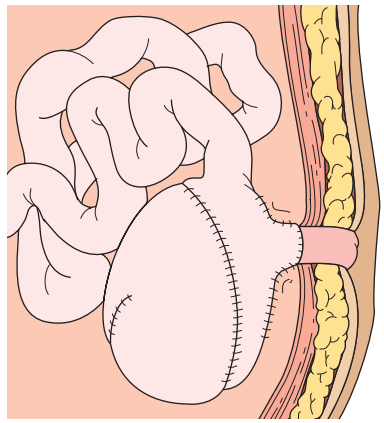


FIGURE 23.12 ■ Continent (Kock's) ileostomy

following an IPAA. A loop of ileum is brought to the body surface to form a stoma, allowing stool drainage into an external pouch. When the ileostomy is no longer necessary, surgery is performed to close the stoma and repair the bowel, restoring faecal elimination through the anus.

In a *continent ileostomy* (see Figure 23.12), an intra-abdominal reservoir is constructed and a nipple valve formed (the ileum folded back on itself) from the terminal ileum, before it is brought to the surface of the abdominal wall. Stool collects in the internal pouch. The nipple valve prevents leaking through the stoma. A catheter is inserted into the pouch to drain the stool.

Nursing care of a person with an ileostomy is outlined in the box below. When devising nursing care plans for a person requiring stoma care it is important to incorporate the person's cultural, religious and personal viewpoints into their care plan. Procedure 23.1 describes how to apply one- and two-piece drainable ostomy pouches.

Complementary and alternative therapies

The chronic nature of inflammatory bowel disease the adverse effects of many of the prescribed treatments leads up to 50% of people with IBD to seek or use complementary and alternative therapies. The most common complementary therapies used by people with IBD include herbal therapies, nutritional supplements, probiotics and fish oil (Solomon & Flum, 2015). Herbal remedies such as aloe vera gel and curcumin appear to have beneficial effects, as do Chinese traditional medicine including Jian Pi Ling tablets and Yukui tang tablets. Probiotics, bacteria similar to those normally found in the gut, appear to have beneficial anti-inflammatory effects in the bowel (Sharma, 2014a). Probiotics are available in foods such as yoghurt, miso and soy beverages, as well as in dietary supplements. Results are mixed from studies of the beneficial effects of fish oil or omega-3 fatty acid supplements for IBD. Peppermint tea is an excellent tonic for reducing nausea, relieving abdominal pain and providing a calming effect. Chamomile tea helps to reduce intestinal inflammation (Bullock & Manias, 2014).

NURSING CARE OF THE PERSON having an ileostomy

PREOPERATIVE CARE

- Provide routine pre-operative care and teaching as outlined in Chapter 3.
- Refer to a stomal therapist for marking and teaching about the stoma location, ostomy care and options for ostomy appliances. *It is important to begin teaching prior to surgery to facilitate learning and acceptance of the ostomy postoperatively.*
- Discuss the availability of a local Stoma Association and provide a referral as necessary or desired. *Local branches often have members with ostomies willing to provide both preoperative and postoperative teaching, listening and support.*
- Provide preoperative bowel preparation as ordered. *Laxatives, enemas and preoperative antibiotics are often ordered to reduce the risk of abdominal contamination and infection after surgery*.

POSTOPERATIVE CARE

- Provide routine postoperative care and teaching as outlined in Chapter 3.
- Apply an ostomy pouch over the stoma (see Procedure 23.1). *Stool from an ileostomy is expressed continuously or irregularly and is liquid in nature; continuous use of a pouch to collect the drainage is necessary.*
- Assess frequently for bleeding, stoma viability and function. In the early postoperative period, small amounts of blood in the pouch are expected. *A healthy stoma is pink or red and moist as a result of mucus production (see Figure 23.11) and protrudes approximately 2 cm from the abdominal wall. Frequent assessment is important in the initial postoperative period to ensure stoma health and to monitor for possible complications. A dusky, brown, black or white stoma indicates circulatory compromise. Other possible stoma complications include retraction (indentation or loss of the external portion of the stoma) or prolapse (outward telescoping of the stoma—that is, an abnormally long stoma).*
- As the stoma starts functioning, empty the pouch, explaining the procedure to the person. Initial drainage is dark green, viscid and usually odourless. Drainage gradually thickens and becomes yellow-brown. Empty the pouch when it is one-third full. Measure drainage and include it as output on fluid balance records. Rinse the pouch and reapply the clamp. *Emptying the pouch when it is no more than one-third full helps prevent the skin seal from breaking as a result of the weight of the pouch. Because of the potential for excess fluid loss through ileostomy drainage, it is important to include it as fluid output.*
- Assess the peristomal skin. Skin around the stoma should remain clean and pink and free of irritation, rashes, inflammation or excoriation. *Skin complications may arise from appliance irritation or hypersensitivity, excoriation from a leaking appliance or* Candida albicans *(a yeast infection).*
- Protect peristomal skin from enzymes and bile salts in the ileostomy effluent. Using a skin barrier on the pouch is essential. Change the pouch if leakage occurs or if the person reports burning or itching skin. *Enzymes and bile salts normally reabsorbed in the large intestine are irritating to the skin. Excoriation of skin surrounding the stoma impairs the first line of defence against microorganisms and can interfere with the ability to achieve a tight skin seal and prevent pouch leakage.*
- Report the following abnormal assessment findings to the surgeon:
  a. Allergic or contact dermatitis. *A rash may result from contact with faecal drainage or indicate sensitivity to pouch, paste, tape or sealant.*
  b. Purulent ulcerated areas surrounding the stoma. *Disruption of the protective barrier of the skin allows bacterial entry.*
  c. A red, bumpy, itchy rash or white-coated area. *This is a manifestation of* Candida albicans, *a yeast infection.*
  d. Bulging around the stoma. *This finding may indicate herniation, caused by loops of intestine protruding through the abdominal wall.*
- Apply protective ointments to the perirectal area of people with newly functioning ileoanal reservoirs and anastomoses. This helps protect the skin from the initial stools. *As stools thicken and become fewer per day, the person experiences less perirectal irritation.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- While caring for the stoma, explain procedures to the person. *Teaching is immediate and ongoing to assist in acceptance of the stoma and facilitate self-care.*
- Teach to manage the pouch clamp, to empty, rinse and perform pouch changes. *Self-care is vital to independence and self-esteem.*
- Instruct now to use an electric razor to shave the peristomal hair if necessary. *An electric razor prevents accidental cutting of the stoma with a razor blade.*
- Teach to check the stoma and peristomal skin with each pouch change. Ongoing assessment is important for optimal health and function of the stoma and surrounding skin. Stripping of tape or excessively frequent pouch removal may cause mechanical trauma to peristomal skin. *Chronic skin irritation by ileostomy effluent may lead to pseudoverrucous lesions or wartlike nodules.*
- Instruct to report abnormal appearance of the stoma or surrounding skin (as noted previously and below) to the doctor:
  a. Narrowing of the stoma lumen. *This indicates stenosis and may interfere with faecal elimination.*
  b. Lacerations or cuts in the stoma. *The stoma contains no nerves, so trauma may occur without pain.*
  c. Separation of the stoma from the abdominal surface. *This potential complication may require surgical repair.*
- Emphasise the importance of adequate fluid and salt intake; the risk of dehydration and hyponatraemia is increased particularly during hot weather, when fluid is lost through perspiration as well as ileostomy drainage. Water intake should be sufficient to maintain pale urine and an output of at least 2.5 L per day. When exercising in

NURSING CARE OF THE PERSON having an ileostomy (continued)

hot weather, the person needs to consume extra water and salt. High-potassium foods, such as bananas and oranges, may also be recommended. *Loss of the re-absorptive surface of the large bowel increases the amount of water and sodium loss in the stool. If the ileostomy is high (more proximal in the ileum), additional potassium losses may also occur.*

- Discuss manifestations of fluid and electrolyte imbalances:
  a. Extreme thirst
  b. Dry skin and oral mucous membrane
  c. Decreased urine output
  d. Weakness, fatigue
  e. Muscle cramps
  f. Abdominal cramps, nausea, vomiting
  g. Shortness of breath
  h. Orthostatic hypotension (feeling faint when suddenly changing positions).
- Discuss dietary concerns. A low-residue diet is recommended initially (see Table 23.8). Foods causing excessive odour or gas are typically avoided as well. Because food blockage is a potential problem, high-fibre foods are limited and foods that may cause blockage, such as popcorn, corn, nuts, cucumbers, celery, fresh tomatoes, figs, strawberries, blackberries and caraway seeds, are avoided. *Symptoms of food blockage include abdominal cramping, swelling of the stoma and absence of ileostomy output for more than 4 to 6 hours.*
- Teach self-care measures to relieve food blockage:
  a. Take a warm shower or bath. *This can help relax the abdominal muscles.*
  b. Assume a knee–chest position. *The knee–chest position reduces intra-abdominal pressure.*
  c. Drink warm fluids or grape juice if not vomiting. *This provides a mild cathartic effect.*
  d. Massage peristomal area. *Massage may stimulate peristalsis and faecal elimination.*
  e. Remove pouch if the stoma is swollen and apply a pouch with a larger opening. *If the stoma swells, the pouch may create a mechanical obstruction to output.*
- Notify the doctor or stomal therapy nurse if:
  a. The above measures fail to relieve the obstruction.
  b. Signs of a partial obstruction persist, including high-volume odorous fluid output, abdominal cramps, nausea and vomiting.
  c. There is no ileostomy output for 4 to 6 hours.
  d. Clinical manifestations of fluid and electrolyte imbalance occur, such as weakness, dizziness, light-headedness or headache.

Should self-care measures not succeed in breaking up a blockage, ileostomy lavage, as described in Procedure 23.2, may be required.

TABLE 23.8 Low-residue diet

FOOD GROUP	ALLOWED	AVOID
Beverages	Coffee, teas, juices, carbonated beverages; milk limited to 300 mL per day	Alcohol, prune juice
Breads and cereals	Products made from refined flours (white bread, water crackers) or finely milled grains (e.g. cornflakes, rice bubbles, puffed wheat)	Wholegrain breads, rolls or cereal; breads or rolls with seeds, nuts or bran
Desserts	Gelatins, tapioca, plain custards or puddings; instant puddings; sponge cake; ice-cream or frozen desserts without fruit or nuts	Any desserts containing dried fruits, nuts, seeds or coconut; rich pastries, pies
Fruits	Fruit juices and strained fruits; cooked or canned apples, apricots, cherries, peaches, pears; bananas	All other raw or cooked fruits
Meats and other protein sources	Roasted, baked or grilled tender or minced beef, veal, pork, lamb, poultry or fish; smooth peanut butter; cottage, creamed cheese or mild cheddar cheeses in small amounts	Tough or spiced meats and those prepared by frying; highly flavoured cheeses; nuts
Potatoes, rice and pasta	Peeled potatoes; white rice; most pasta products	Potato skins, potato chips or fried potatoes; brown rice; wholegrain pasta products
Sweets	Sugar, honey, jelly, hard-boiled lollies and gumdrops, plain chocolates	Jam, marmalade; lollies made with seeds, nuts, coconut
Vegetables	Vegetable juices and strained vegetables; cooked or canned vegetables	Raw or whole cooked vegetables
Other	Salt, ground seasonings; cream sauce and plain gravy	Chilli sauce, horseradish; popcorn, seeds of any kind; whole spices, olives, vinegar

PROCEDURE 23.1 Changing a one- or two-piece drainable ostomy pouch

GATHER SUPPLIES

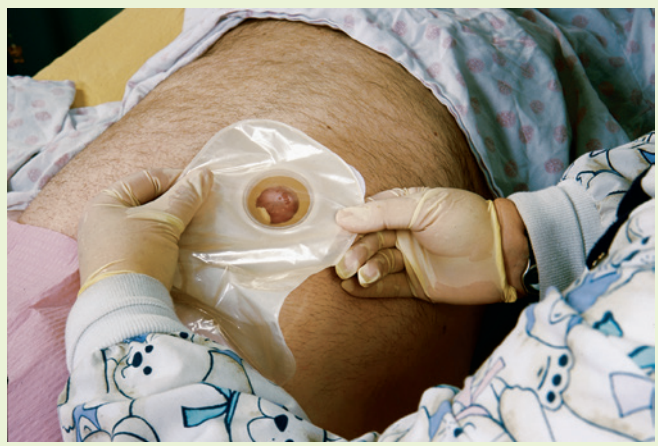
- Disposable gloves and personal protective equipment (PPE)
- One- or two-piece pouch
- Skin barrier paste
- Skin prep
- Clamp
- Pouch deodorant
- Measuring guide
- Adhesive remover
- Skin cleanser
- Wash cloths
- Plastic bag
- Bed protector
- Air freshener

BEFORE THE PROCEDURE

- Explain the procedure and provide for privacy.
- Position the person (assist as required) in a lying position making removing and applying the pouch easier.
- Use air freshener as required and preferred by the person.
- Follow standard precautions. Don PPE and gloves.

PROCEDURE

1. Remove soiled pouch (and the flange if a two-piece pouch) by gently pulling on the pouch or flange and pushing on skin. Use adhesive remover to remove skin barrier paste.
2. Empty pouch, discarding it and the flange (if applicable) in a plastic bag. Save the tail closure clamp. The pouch from a two-piece system may be cleaned out and reused.
3. Cleanse skin and stoma with warm water and skin cleanser or mild soap. Rinse skin and stoma and pat dry.
4. Note stoma colour and peristomal skin condition.
5. If necessary, clip or shave peristomal hair.
6. Use measuring guide or previous pattern to check size of stoma.
   a. Presized pouch: check to verify size is correct.
   b. Cut-to-fit pouch or flange: trace the correct size of the stoma onto the back of the flange and cut the opening to match the pattern. The opening should be no more than 3 to 4 mm larger than the stoma.
7. Apply skin prep to skin covered by a wafer, pouch or tape. Allow to dry.
8. Remove backings from pouch or flange.
9. Apply a bead of skin barrier paste around the stoma base or around the opening of the pouch or flange. Allow the paste to air dry for 1 to 2 minutes.
10. Centre the pouch or flange over the stoma and press to adhere.
11. For a two-piece pouch, snap the pouch onto the skin barrier flange.
12. Place deodorising powder or a few drops of liquid pouch deodoriser in the pouch. Apply the clamp.
13. 'Picture frame' the pouch with tape to provide extra security.



AFTER THE PROCEDURE

1. Leave the person comfortable.
2. Dispose of equipment correctly.
3. Document assessment of stoma and surrounding skin and the person's response to the procedure.

Anecdotal evidence supports the use of homeopathy to promote comfort in individuals with IBD. Acupuncture improves perceived wellbeing in people with IBD; however, it does not appear to effect remission (Lizarondo, 2015). Herbal remedies, such as slippery elm, fenugreek, devil's claw, Mexican yam, termentil and wei tong ning, have been found to have an antioxidant effects and may provide an effect similar to 5-aminosalicylic acid preparations (Lizarondo, 2015). Many complementary and alternative therapies for IBD may interact with prescribed medications; advise the person to discuss all potential therapies with the primary care provider. Acupressure, body massage, reflexology, aromatherapy and stress reduction therapies can also aid in reducing manifestations of IBD.

Nursing care

Health promotion

Although inflammatory bowel disease cannot, at this time, be predicted or prevented, effective management may help the person avoid complications of the disease. Stress the importance of following the prescribed treatment regimen and promptly reporting manifestations of exacerbations to the doctor.

PROCEDURE 23.2 Ileostomy lavage

GATHER SUPPLIES

- Disposable gloves and PPE
- Disposable irrigation sleeve
- 60 mL catheter-tipped syringe
- #14 Fr. catheter
- Water-soluble lubricant
- Normal saline for irrigation
- Bedpan
- Clean ostomy pouch
- Bed protector
- Air freshener

BEFORE THE PROCEDURE

- Explain the procedure and provide for privacy.
- Follow standard precautions.
- Don PPE and gloves.

PROCEDURE

1. Remove the pouch. Apply disposable irrigation sleeve.
2. Clamp the bottom of the sleeve or place it in the bedpan.
3. Gently examine stoma digitally to break up any faecal mass proximal to stoma and determine direction of the bowel.
4. Lubricate catheter and insert into stoma until blockage is reached. If the catheter does not reach the blockage after 8 to 10 cm, notify the doctor. This may indicate a more proximal obstruction.
5. Instill 30 to 50 mL normal saline.
6. Remove catheter. Allow stoma to drain.
7. Repeat the procedure until the mass is removed.
8. When the blockage is removed, remove the irrigation sleeve.
9. Clean peristomal skin.
10. Apply pouch and clamp.

AFTER THE PROCEDURE

1. Leave the person dry and comfortable.
2. Dispose of equipment correctly.
3. Document the procedure, amount of solution used, consistency of results and the person's response to the procedure.
4. Discuss dietary intake to help determine cause of blockage.

Assessment

Assessment data related to inflammatory bowel disease include the following subjective and objective data:

- *Health history:* current manifestations, including onset, duration, severity (number of stools per day, presence of blood or mucus in stool, abdominal pain or cramping, tenesmus); usual diet, ability to maintain weight and nutrition, smoking habits, food intolerances; associated manifestations such as arthralgias, fatigue, malaise; current medications; previous treatment and diagnostic tests.
- *Physical examination:* general appearance; weight; vital signs, including orthostatic blood pressure, pulse and temperature; abdominal assessment, including shape, contour, bowel sounds, palpation for tenderness and masses, presence of stoma or scars.

Nursing diagnoses and interventions

When planning nursing care with the person with inflammatory bowel disease, it is vital to consider the chronic, recurrent nature of the disorder. Religious and cultural beliefs must also be incorporated when planning care for a person with IDB. Teaching is a major aspect of care. Diarrhoea and disturbed body image are significant nursing care problems for a person with IBD. With severe disease, impaired nutrition must also be considered a priority problem. See accompanying nursing care plan for a person with ulcerative colitis.

Diarrhoea

During an acute exacerbation of IBD, diarrhoea can be frequent and painful. The frequency of defecation and associated abdominal pain and cramping may interfere with ADLs and increase the risk of fluid volume deficit and impaired skin integrity.

- Record the frequency, amount and colour of stools using a stool chart. Measure and record liquid stool as output. *The severity of diarrhoea is an indicator of the severity of the disease and helps determine the need for fluid replacement.*
- Monitor vital signs every 4 hours. *Tachycardia, tachypnoea and fever may be indicators of fluid volume deficit.*
- Weigh daily and record. *Rapid weight loss (over days to a week) usually indicates fluid loss, whereas weight loss over weeks to months may indicate malnutrition.*
- Assess for other indications of fluid deficit: warm, dry skin, poor skin turgor, dry shiny mucous membranes, weakness, lethargy, complaints of thirst. *The extent of fluid loss may not be readily evident with diarrhoea, particularly if the person uses the bathroom without assistance. Systemic manifestations of fluid volume deficit may be the first indicators of the problem.*
- Maintain bowel rest by keeping NBM or limiting oral intake to elemental feedings as indicated. *Bowel rest during an acute exacerbation of IBD promotes healing and reduces diarrhoea and other manifestations.*

CONSIDERATION FOR PRACTICE

Observe stools for obvious blood and test for occult blood as indicated. Report grossly bloody stools (haematochezia), which may indicate haemorrhage and necessitate emergency surgery.

NURSING CARE PLAN A person with ulcerative colitis

Estelle Lewis is a 42-year-old real estate agent and mother of three school-age children. Mrs Lewis has had ulcerative colitis for 18 years and has been treated with prednisone and sulfasalazine. Over the past 4 months she has been having abdominal pain and cramping and frequent bloody diarrhoeal stools. During the same period, she lost 9 kg and has had difficulty maintaining her career. She recently developed several lesions of the lower leg identified as erythema nodosum. A recent colonoscopy revealed extensive involvement of the entire colon. On admission, Mrs Lewis states, 'I'm tired of fighting this disease. I am a prisoner in my home because of the diarrhoea.' She is admitted for a total proctocolectomy and ileal pouch–anal anastomosis.

ASSESSMENT

Janet Wheeler, RN, completes the admission assessment. Mrs Lewis weighs 52.2 kg. She complains of abdominal cramping, pain and frequent bloody diarrhoeal stools. Several reddened lesions are noted on her lower legs. Physical assessment findings include T 36.6°C, P 72, R 20 and BP 104/72. Skin, peripheries cool and pale. Abnormal laboratory findings include haemoglobin 73 g/L (normal 117 to 157 g/L); haematocrit 23.3% (normal 35–47%); WBC 15 580/mm^3 (normal 3500 to 11 000/mm^3); platelet count 995 000/mm^3 (normal 150 000 to 450 000/mm^3); serum protein 46 g/L (normal 60 to 80 g/L); serum albumin 24 g/L (normal 35 to 50 g/L). Preparation for surgery is begun.

DIAGNOSES

- *Imbalanced nutrition: less than body requirements* related to impaired absorption.
- *Diarrhoea* related to inflammation of bowel.
- *Risk of deficient fluid volume* related to abnormal fluid loss.
- *Risk of impaired tissue integrity* related to drainage from temporary ileostomy.
- *Alteration in comfort* related to acute pain secondary to surgical intervention.
- *Risk of sexual dysfunction* related to temporary ileostomy.

PLANNING

When planning nursing care with Mrs Lewis, it is vital to consider her preferences. Religious and cultural beliefs are incorporated into her plan of care.

Expected outcomes

- Mrs Lewis will resume prescribed diet within 5 days after surgery.
- Mrs Lewis will demonstrate normal faecal elimination through the temporary ileostomy.
- Mrs Lewis will maintain adequate fluid balance.
- Mrs Lewis will demonstrate appropriate stoma care prior to discharge.
- Mrs Lewis will report a tolerable level of discomfort at rest and during activity.
- Mrs Lewis will verbalise feelings about sexuality and acknowledge importance of discussing sexual issues with partner.

IMPLEMENTATION

- Discuss with Mrs Lewis dietary modifications related to her nutritional status and presence of the ileostomy. Provide Mrs Lewis with a referral to a dietitian for diet planning and teaching.
- Explain to Mrs Lewis the importance of maintaining a high fluid intake and manifestations of dehydration.
- Teach Mrs Lewis to empty and change ileostomy pouch of choice.
- Teach Mrs Lewis stoma and peristomal skin assessment with each pouch change.
- Teach Mrs Lewis food blockage management.
- Provide Mrs Lewis with information about the Australian Stoma Appliance Scheme. It is subsidised by the Australian government and provides stoma products (medicine and appliances) for people with a permanent or temporary stoma free of charge to members of one of the 22 approved volunteer stoma associations.
- Refer Mrs Lewis to local Stoma Association.

EVALUATION

On discharge, Mrs Lewis is caring for her ileostomy by demonstrating her ability to empty, rinse and change the pouch. The stomal therapy nurse provided written and verbal instructions on ileostomy care. Mrs Lewis verbalises her understanding of the recommended diet and the need to limit high-fibre food intake and avoid enteric-coated and timed-release medications. The stomal therapy nurse discusses sexual aspects of having an ileostomy and has given Mrs Lewis a booklet, *A beginning not an end*, available through the Australian Council of Stoma Associations. Mrs Lewis is looking forward to the planned surgery to close the temporary ileostomy.

CRITICAL THINKING IN THE NURSING PROCESS

1. Why is a person with an ileostomy at risk of dehydration? How can Mrs Lewis monitor her fluid status at home?
2. Why were Mrs Lewis's haemoglobin and haematocrit low on admission? If her haemoglobin had been low but her haematocrit normal on admission, what might be the explanation?
3. Outline a teaching plan Mrs Lewis could be given for home care of her ileostomy.
4. Develop a care plan for Mrs Lewis for the nursing diagnosis *Risk of impaired skin integrity*.

REFLECTION ON THE NURSING PROCESS

1. What are the most important things you feel you have learned from the care planning for Mrs Lewis?
2. If you were caring for a person following the formation of an ileostomy, what additional knowledge and clinical skills do you require to care for a person postoperatively?

- Administer prescribed anti-inflammatory and antidiarrhoeal medications as indicated. *Anti-inflammatory medications reduce the extent of bowel inflammation and diarrhoea. Unless contraindicated, antidiarrhoeal medications help reduce fluid loss and increase comfort.*

CONSIDERATION FOR PRACTICE

When antidiarrhoeal medications are administered to a person with ulcerative colitis, closely observe for manifestations of toxic megacolon: fever, tachycardia, hypotension, dehydration, abdominal pain and cramping, and an abrupt relief of diarrhoea.

- Maintain fluid intake by mouth or intravenously as indicated. *The person with IBD requires fluid to replace ongoing losses, as well as fluid to meet the usual daily needs of the body. If an elemental diet or total parenteral nutrition is prescribed, additional fluids may be required to meet fluid intake needs.*
- Provide good skin care. *Fluid deficit and tissue dehydration increase the risk of skin excoriations or breakdown.*
- Assess perianal area for irritation or denuded skin from the diarrhoea. Use gentle cleansing agents, such as Peri-Wash, nappy wipes or cotton lint-free gauze squares saturated with witch hazel. Apply a protective cream, such as zinc-oxide-based preparations, to protect skin from the irritating effects of diarrhoeal stool. *Digestive enzymes in the stool are very corrosive, increasing the risk of skin breakdown where exposed to diarrhoeal stool.*

Disturbed body image

A person with IBD may experience frustration at not being able to control, or even predict, faecal elimination, particularly when the disease is severe. Diarrhoea interferes with the ability to complete tasks, maintain employment, engage in social activities and even to meet basic needs such as eating, sleeping and sexual activity. Body image can suffer as a result. Treatment of IBD, be it total colectomy with ileal pouch–anal anastomosis, ileostomy or chronic corticosteroid therapy, can affect the view of self.

- Provide care in an accepting, non-judgmental manner. *Acceptance of the person despite potential embarrassment about odours or diarrhoea enhances self-esteem.*
- Accept feelings and perception of self. *Negating or denying the reality of the person's perception impairs trust.*
- Encourage discussion of physical changes and their consequences as they relate to self-concept. *This demonstrates acceptance and provides an opportunity to express the impact of the disease and its treatment on the person's life.*
- Encourage discussion about concerns regarding the effect of the disease or treatment on close personal relationships. *This demonstrates understanding and provides an opportunity for the person to express feelings about the impact of the disease on relationships and significant others.*
- Encourage the person to make choices and decisions regarding care. *This increases the person's sense of control over the disease and their future.*
- Discuss possible treatment options and their effects openly and honestly. *Open discussion allows more informed decisions.*
- Involve the person in their care, teaching and demonstrating as needed. *This encourages and facilitates independence and decision making.*
- Arrange for interaction with other individuals or groups of people with IBD or ostomies. *The person may feel that someone who has not experienced a similar problem cannot understand their feelings.*
- Teach coping strategies (e.g. odour control, dietary modifications) and support their use. *This facilitates healthy adaptation to the disease.*

Imbalanced nutrition: less than body requirements

Crohn's disease can significantly alter the bowel's ability to absorb nutrients. In both forms of IBD, blood and protein-rich fluid may be lost in diarrhoeal stools. With malabsorption and continuing nutrient losses, multiple nutrient deficits can develop, affecting growth and development, healing, muscle mass, bone density and electrolyte balances.

- Monitor laboratory results, including haemoglobin and haematocrit, serum electrolytes, and total serum protein and albumin levels. *These studies provide an indicator of nutritional status.*
- Provide the prescribed diet: high-kilojoule, high-protein, low-fat diet with restricted milk and milk products if lactose intolerance is present. *Kilojoules and protein are important to replace lost nutrients. Fat restriction helps reduce diarrhoea and nutrient loss, particularly when significant portions of the terminal ileum have been resected.*
- Provide parenteral nutrition as necessary if the person is unable to absorb enteral nutrients. *Parenteral nutrition helps reverse nutritional deficits and promotes weight gain and healing in the individual with acute manifestations.*
- Arrange for dietary consultation. Consider food preferences as allowed. *Providing preferred foods in the prescribed diet increases intake and supports nutritional status.*
- Provide or administer elemental enteral nutrition and supplements as ordered. *Elemental enteral nutritional supplements support healing while providing for bowel rest. These replace losses and improve nutritional status more rapidly than diet alone.*
- Include family members—the primary food preparer, especially—in teaching and dietary discussions. *Families can reinforce teaching and help the person maintain required restrictions or kilojoule intake.*

Community-based care

Inflammatory bowel disease is a chronic condition for which the person provides daily self-management. For this reason, teaching is a vital component of care. Teach the person and their family about the following topics:

- the type of IBD affecting the person, including the disease process, short- and long-term effects, the relationship of stress to disease exacerbations and the manifestations of complications
- prescribed medications, including drug names, desired effects, schedules for tapering the doses if ordered (as with corticosteroids), and possible side effects or adverse reactions and their management
- the recommended diet and the rationale for any specific restrictions

- use of nutritional supplements such as Ensure to maintain weight and nutritional status
- indicators of malabsorption and impaired nutrition; recommendations for self-care and when to seek medical intervention
- if discharged with a central venous catheter and home parenteral nutrition, written and verbal instructions on catheter care, troubleshooting and TPN administration (i.e. have the person and a family member demonstrate catheter care and TPN maintenance)
- the importance of maintaining a fluid intake of at least 2 to 3 L per day, and of increasing fluid intake during warm weather, exercise or strenuous work, and when fever is present
- the increased risk of colorectal cancer and the importance of regular bowel exams
- risks and benefits of various treatment options.

If surgery is planned or has been done, include the following topics in home care instructions:

- ileal pouch–anal anastomosis or ileostomy care as indicated
- where to obtain ostomy supplies
- use of non-prescription drugs, such as enteric-coated and timed-release capsules that may not be adequately absorbed before elimination through the ileostomy
- community and national ostomy support groups (see below).

Provide referrals to a nutritionist, a community healthcare agency, home care services and home intravenous care services as indicated. In addition, suggest the following resources:

- Crohn's & Colitis Australia
- Australian Council of Stoma Associations.

MALABSORPTION SYNDROMES

Malabsorption is a condition in which the intestinal mucosa ineffectively absorbs nutrients—including carbohydrates, proteins, fats, water, electrolytes, minerals and vitamins—resulting in their excretion in the stool. Multiple bowel disorders can lead to malabsorption.

Diseases of the small intestine often cause malabsorption. Other medical and/or surgical conditions can result in malabsorption if these affect digestion or the intestinal mucosa. Primary diseases of the small-bowel mucosa, such as coeliac disease (also known as tropical and non-tropical sprue), Crohn's disease and acute gastrointestional tract infections, can lead to malabsorption. It can also result from maldigestion (inadequate preparation of chyme for absorption). For example, major gastric resections, pancreatic disorders with impaired pancreatic enzyme secretion and biliary disorders affecting bile secretion impair digestion and absorption of chyme. Selected causes of impaired absorption and digestion are listed in Table 23.9.

TABLE 23.9 Selected causes of malabsorption

CAUSE	RELATED FACTORS OR CONDITIONS
Impaired absorption	Tropical sprue Short bowel syndrome Acute enteritis and other bowel infections or infestations AIDS-related opportunistic infections and Kaposi's sarcoma Coeliac disease (non-tropical sprue) Crohn's disease Intestinal ischaemia or infarction Scleroderma
Impaired digestion	Lactose intolerance Gastrectomy Chronic pancreatitis, cancer of the pancreas Cystic fibrosis Biliary obstruction Cirrhosis, hepatitis or liver failure Zollinger–Ellison syndrome

Regardless of the cause, malabsorption causes common manifestations resulting from impaired absorption of chyme and the nutrients it contains (see Table 23.10). Predominant GI manifestations include anorexia; abdominal bloating; diarrhoea with loose, bulky, foul-smelling stools; and steatorrhoea (fatty stools). Weight loss, weakness, general malaise, muscle cramps, bone pain, abnormal bleeding and anaemia are common systemic manifestations of malabsorption. These manifestations result from malnutrition and fluid loss due to poor absorption.

Three common malabsorption disorders in adults are coeliac disease (non-tropical sprue), lactose intolerance and short bowel syndrome.

THE PERSON WITH SPRUE

Sprue is a chronic primary disorder of the small intestine in which the absorption of nutrients, particularly fats, is impaired. The severity of the disease depends on the extent of mucosal involvement in the intestine and the duration of the disease. Two major forms of sprue are coeliac disease (coeliac or non-tropical sprue) and tropical sprue.

Pathophysiology

Most absorption of nutrients occurs in the small intestine. The mucosa of the small intestine is arranged in microscopic folds, containing even smaller finger-like projections called *villi*. The cells of the villi are covered with microscopic hairs, *microvilli*, projecting from the cell membrane. The folds, villi and microvilli of the intestinal mucosa provide a huge surface area for nutrient absorption. Cells of the intestines are specialised to absorb different nutrients. Readily digested nutrients are absorbed in the proximal intestine; others are absorbed more distally in the intestines. Nutrients are absorbed by the processes of simple diffusion (water and small lipids), facilitated diffusion (water-soluble vitamins) and active transport (glucose and amino acids). Once

TABLE 23.10 Local and systemic manifestations of malabsorption

CATEGORY	MANIFESTATION	CAUSE
Local (GI)	Diarrhoea	Disruption of bowel mucosa impairing absorption of fluid and electrolytes, leading to excess water in the stool
	Abdominal distension	Gas formation from fermentation of undigested carbohydrates
	Steatorrhoea	Impaired fat absorption, leading to excess fat in faeces
Systemic	Weight loss	Carbohydrate, protein and fat deficit
	Weakness and malaise	Kilojoule deficit, anaemia, fluid and electrolyte losses
	Anaemia	Vitamin B_{12}, folic acid and iron deficits
	Bone pain	Calcium and vitamin D deficits
	Muscle cramps, paresthesias	Protein wasting, vitamin B_{12} and electrolyte deficits
	Easy bruising and bleeding	Vitamin K deficit
	Glossitis, cheilosis	Iron, folic acid and vitamin B_{12} deficits

absorbed into the cells of the villi, nutrients enter the blood or lymph for systemic distribution.

Sprue is characterised by flattening of the intestinal mucosa with a loss of villi and microvilli. With the loss of villi, intestinal absorptive surface is lost and digestive enzyme production, including disaccharidase and particularly lactase, is reduced.

Coeliac disease

Coeliac disease, also known as coeliac sprue or *non-tropical sprue* and gluten-sensitive enteropathy, is a chronic immune-mediated malabsorption disorder characterised by sensitivity to the gliadin fraction of gluten, a cereal protein. Gluten is found in wheat, rye, barley and oats. It is also used as filler in many prepared foods and in medications. The cause of coeliac disease is unknown; however, genetic, environmental and immune factors are known to play a role in its development. The development of coeliac disease involves a particular genetic make-up (HLA type), with the genes HLA-DQ2 and HLA-DQ8 being identified as the 'coeliac genes' (Porth & Matfin, 2013). Coeliac disease more commonly affects Caucasians and West Asians. It is uncommon in the East Asian and Australian Aboriginal and Torres Strait Islander populations (Gastroenterological Society of Australia (GSA), 2007). Environmental factors also play a role. Coeliac disease was considered a rare malabsorption syndrome manifesting during early childhood; however, it is one of the most common genetic diseases, with a mean prevalence of 1% in susceptible populations (Porth & Matfin, 2013).

In coeliac disease, it appears that the intestinal mucosa is damaged by an immunological response. Gliadin acts as an antigen (a substance inducing the formation of antibodies interacting specifically with it), prompting an inappropriate T-cell-mediated immune response. People with coeliac disease have increased antibodies to other antigens as well. The immune response prompts an inflammatory response in the small bowel, resulting in loss of intestinal folds and absorptive surface. Digestive enzyme production, including disaccharidase and particularly lactase, is reduced. The proximal small bowel is affected to the greatest extent, likely due to its greater exposure to dietary gluten.

Manifestations

Manifestations of coeliac sprue may develop at any age. Local manifestations include abdominal bloating and cramps, diarrhoea and steatorrhoea. Systemic manifestations result from the effects of malabsorption and resulting deficiencies. Anaemia is common. People with coeliac disease are often small in stature and may have delayed maturity. Other signs of nutrient deficiencies include tetany, vitamin deficiencies, muscle wasting and osteomalacia (impaired bone development). When gluten is removed from the diet, these manifestations resolve.

Gastrointestinal malignancies and intestinal lymphoma are potential complications of coeliac disease. Other complications include intestinal ulceration and development of refractory sprue or disease no longer responding to a gluten-free diet.

Tropical sprue

Tropical sprue is a chronic disease of unknown cause, although bacterial infection or toxins are thought to contribute. It seems likely that tropical sprue is not a single disease, but rather a pathophysiological process with manifestations resulting from a combination of the initial infection and the person's diet, living standards and genetic predisposition. A milder form of this may lead to asymptomatic abnormalities referred to as tropical enteropathy, while more severe expression leads to debilitating tropical sprue syndrome. The nature and the extent of the nutritional deficiencies are likely to be related to the duration of the disease and the extent of bowel involved (Beeching & Beadsworth, 2014). Tropical sprue occurs chiefly in the Caribbean, south India and South-East Asia. Hanson (2005) described three cases of tropical sprue in Aboriginal people living in remote Aboriginal or Torres Strait Islander communities in far north Queensland. A number of studies of chronic diarrhoea in Australian Aboriginal and Torres Strait Islander children have documented partial villous atrophy on small bowel biopsy. Additionally, other studies identified higher colony counts and more frequent isolation of gram-negative organisms in duodenal aspirates from Australian Aboriginal and Torres Strait Islander children with chronic diarrhoea. Some of these studies, Hanson (2005) noted, had similarity with contemporary Indian studies of tropical sprue, suggesting that tropical sprue should be considered in people from remote Australian Aboriginal or

Torres Strait Islander communities presenting with weight loss, diarrhoea and nutritional deficiency. The pathophysiological changes in bowel mucosa closely resemble those of coeliac sprue, although gluten intake has no effect on this condition. Its onset may be abrupt or insidious.

Manifestations

Clinical manifestations of tropical sprue include sore tongue, diarrhoea and weight loss. Initially, diarrhoea may be explosive and watery; as the disease progresses, stools become fewer in number and more solid with obvious steatorrhoea. Folic acid deficiency is common. Vitamin B_{12} and iron deficiencies may occur, resulting in glossitis; stomatitis; dry, rough skin; and anaemia.

INTERPROFESSIONAL CARE

With any malabsorptive disorder, the initial focus of management is to identify the cause. Once this has been determined, specific therapy can be prescribed.

Diagnosis

Laboratory and diagnostic testing are used to make the differential diagnosis for various causes of malabsorption syndromes and determine the severity of nutrient deficiencies.

An endoscopy permits direct examination of intestinal mucosa and collection of a tissue specimen for biopsy. Upper GI series with small-bowel follow-through may be done to evaluate the structures of the upper GI tract. With sprue, the typical 'feathery' pattern of barium in the small bowel is lost and the barium may precipitate and clump. Nursing implications of diagnostic tests are included in Chapter 20.

Laboratory tests are used to identify pathophysiological effects and monitor concordance with the prescribed diet. Faecal fat is measured to document the presence of steatorrhoea. The expected result is 2 to 7 g per 24 hours for adults (Kee, 2013). The fat content of stool is increased in many malabsorptive disorders, including coeliac and tropical sprue. Serological testing for IgA endomysial antibodies and IgG and IgA antigliadin antibodies is used to diagnose coeliac disease and evaluate concordance with the prescribed gluten-free diet. Serum levels of protein, albumin, cholesterol, electrolytes and iron may be ordered to evaluate for nutrient deficiencies. The haemoglobin, haematocrit and RBC indices are used to evaluate anaemia. Prothrombin time is increased in vitamin K deficiency.

Medications

Individuals with severe nutritional deficits require vitamin and mineral supplements, as well as iron and folic acid to correct anaemia. Vitamin K may be administered parenterally if the prothrombin time is prolonged. In those whose disease fails to respond to dietary management, corticosteroids may be ordered to suppress the inflammatory response.

Tropical sprue is treated with a combination of folic acid and tetracyclines. This regimen is continued for 3 to 6 months.

Nutrition

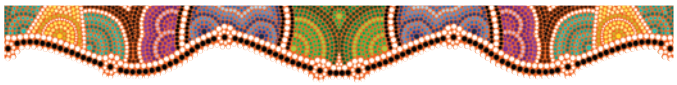
An individual with coeliac disease is placed on a gluten-free diet. The GSA (2007) stresses that all people with coeliac disease require a strict gluten-free diet irrespective of clinical manifestations of the disease. This treatment is generally successful, as long as the individual avoids gluten totally. However, gluten is so widely used in prepared foods that this is no easy task. In Australia, food labelled gluten-free contains no detectable gluten, oats or malt, and most packaged food containing grain (wheat, rye, oats or barley (GSA, 2007)) must list gluten. Individuals need to be aware of hidden sources of gluten and to analyse dietary labels. The GSA (2007) warns that alchoholic beverage labels are not required to list ingredients derived from gluten-containing grain, and highlights that all regular beers contain gluten. Common sources of gluten and foods to be avoided are indicated in Table 23.11.

Consultation with a nutritionist to obtain detailed dietary instructions is necessary.

TABLE 23.11 Dietary sources of gluten

FOOD GROUP	CONTAINS GLUTEN	MAY CONTAIN GLUTEN
Cereals, grains and grain products	Bread, biscuits, cereal and pasta containing wheat, rye or barley grain or flour	Seasoned rice and potato mixes
Beverages	Malt, Milo, Ovaltine, beers and ales	Commercial chocolate milk, cocoa and other beverage mixes, such as instant tea mix, dietary supplements
Desserts	Cakes, biscuits and pastries made with wheat, rye or barley flour	Commercial ice-cream and gelato
Meats and other protein sources		Meat loaf, manufactured and prepared meats, crumbed meats and fish; cheese products; soy protein meat substitutes; commercial egg products
Fruits and vegetables		Commercial seasoned vegetable mixes or vegetables with sauce; canned baked beans; commercial pie fillings
Miscellaneous		Commercial salad dressings and mayonnaise; tomato sauce and prepared mustard; gravy, white sauce; non-dairy creamer; syrups; commercial pickles

The prescribed diet is high in kilojoules and protein to correct nutrient deficits. Fat content is restricted to minimise steatorrhoea. Initially, the diet is usually restricted in lactose as well to compensate for the loss of lactase-containing microvilli. Foods containing lactose may be reintroduced once remission has occurred (Marieb & Hoehn, 2013).



Nursing care

Nursing care of a person with sprue (tropical and non-tropical) focuses on the effects of the disorder on health and nutrition, as well as the person's ability to manage their disease.

Assessment

- *Health history:* onset, duration and severity of manifestations; number and character of stools; history of travel to the Caribbean, southern India or South-East Asia, or living in remote Indigenous communities in northern Queensland; previous teaching related to the disorder; current treatment and diet.
- *Physical examination:* vital signs; abdominal shape, contour, bowel sounds; manifestations of malnutrition (e.g. anaemia, small stature, muscle wasting, signs of other nutrient deficiencies).

Nursing diagnoses and interventions

Diarrhoea and malnutrition are significant problems for a person with sprue and the priority foci for nursing intervention.

Diarrhoea

Steatorrhoea and diarrhoea typically occur with sprue because fat, water and other nutrients are poorly absorbed, remaining in the bowel to be eliminated in the stool. Diarrhoea interferes with lifestyle, ADLs, skin integrity, and fluid and electrolyte balance.

- Assess and document the frequency and nature of stools. *Bowel elimination reflects the severity of the disease and efficacy of treatment. With effective treatment, stools become less frequent and more normal in colour and appearance.*
- Weigh daily, monitor intake and output, and assess skin turgor and mucous membranes for indications of fluid balance. *Diarrhoea increases the risk of hypovolaemia and dehydration resulting from excess fluid loss in the stool.*
- Assess and document perianal skin condition. *Frequent defecation irritates skin and mucous membranes, increasing the risk of breakdown.*
- Encourage a liberal fluid intake. *Oral fluids help replace fluid lost through diarrhoeal stool.*

Imbalanced nutrition: less than body requirements

Coeliac disease is a chronic condition. With continuing malabsorption, multiple nutrient deficits may occur, resulting in impaired growth and development, impaired healing, muscle wasting, bone disease and electrolyte imbalances.

- Maintain accurate dietary intake records. *Assessment of dietary intake provides information about compliance with the prescribed diet as well as the adequacy of nutrient intake.*
- Monitor laboratory results, including haemoglobin and haematocrit, serum electrolytes, total serum protein and albumin levels. *These studies provide information about nutritional status.*
- Arrange for nutritionist consultation. Provide for food preferences as allowed. *An individualised diet developed to address the person's food preferences as well as nutrient needs will promote appetite and food intake.*
- Provide the prescribed high-kilojoule, high-protein, low-fat, gluten-free diet for the person with coeliac disease. Restrict lactose (dairy product) intake as indicated. *Kilojoules and protein are important to replace lost nutrients. Fat restriction helps reduce diarrhoea and nutrient loss. Lactose may be restricted during initial treatment and then slowly reintroduced into the diet as the gut heals and its normal structure is restored.*
- Provide parenteral nutrition as ordered if the person is unable to absorb enteral nutrients. *Parenteral nutrition can help reverse nutritional deficits and promote weight gain when manifestations are acute.*
- Encourage nutritional supplements. *Nutritional supplements often are necessary to replace losses and restore nutrient levels to normal more rapidly than diet alone can achieve.*

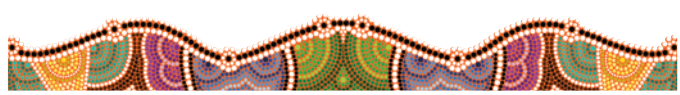
Include family members—the primary food preparer, in particular—in teaching and dietary discussions. *Families can reinforce teaching and help the person maintain required restrictions or kilojoule intake.*

Community-based care

Although tropical sprue is treated with antibiotic and folic acid therapy, *a person with coeliac sprue has a chronic condition requiring lifelong dietary management.* Encourage the person to join the Coeliac Society of Australia (CSofA). The CSofA provides information, including the availability of gluten-free foods, recipes and support for individuals with coeliac disease and their families (CSofA, 2015).

Provide a detailed list of foods containing gluten which need to be eliminated from the person's diet, as well as foods that are allowed. Teach the person and their family how to identify gluten-containing commercial products by reading labels and lists of ingredients. Encourage the purchase and use of a gluten-free cookbook.

If corticosteroids are prescribed, stress the importance of taking the medication as ordered. Emphasise the need to avoid stopping the medication abruptly and to notify all caregivers that a corticosteroid is part of the individual's medication regimen. Instruct to monitor weight frequently. A weight gain of 2.3 kg or more in less than a week usually reflects fluid gain, a possible adverse effect of corticosteroids. Other potential effects include decreased resistance to infection, an impaired inflammatory response and changes in carbohydrates, protein and fat metabolism.



THE PERSON WITH LACTASE DEFICIENCY

For carbohydrates to be absorbed from the small intestine, they first must be broken down into simple sugars (monosaccharides). Lactose is the primary carbohydrate in milk and milk products. It is a disaccharide, requiring the enzyme lactase for digestion and absorption. Lactase deficiency leads to lactose intolerance and manifestations of malabsorption. Lactase deficiency is usually genetic in origin, but also occurs secondarily to coeliac disease, Crohn's disease and other disorders affecting the mucosa of the small intestine. There is a racial/ethnic component to the disorder; it is rare for Caucasians to develop lactose intolerance. However, it is common among people from Asia, Africa, the Middle East and some Mediterranean countries, as well as among Australian Aboriginal and Torres Strait Islander peoples (Genauer & Hammer, 2010).

Manifestations

Many people with lactase deficiency are asymptomatic. Small to moderate amounts of milk (250 mL) may be well tolerated. Manifestations of lactose intolerance include lower abdominal cramping, pain and diarrhoea following milk ingestion. Undigested lactose ferments in the intestine, forming gases and contributing to bloating and flatus. Lactic and fatty acids produced by this fermentation irritate the bowel, leading to increased motility and abdominal cramping. The undigested lactose draws water into the intestine, leading to increased motility and diarrhoea. The diarrhoea associated with lactose intolerance may be explosive.

INTERPROFESSIONAL CARE

The diagnosis of lactose intolerance is usually based on a history of intolerance to milk and milk products and a trial of a lactose-free diet. If manifestations resolve when lactose intake is eliminated, the diagnosis of lactose intolerance is confirmed.

Diagnosis

The lactose breath test is a non-invasive test used to diagnose lactose intolerance. Expired hydrogen gas (H_2) is measured following oral administration of 50 g of lactose. If lactose is digested and absorbed normally, then little change occurs in the amount of exhaled H_2 from fasting to post lactose administration. With lactose intolerance, exhaled H_2 increases following lactose administration as the sugar ferments in the bowel.

For the *lactose tolerance test*, 100 g of lactose solution is orally administered, followed by measurement of blood glucose levels at intervals of 30, 60 and 120 minutes. If lactose is digested and absorbed normally, the blood glucose rises more than 20 mg/dL. The expected blood glucose elevation does not occur in lactose intolerance.

Nutrition

A lactose-free or reduced lactose diet relieves the manifestations of the disorder. Some people require total elimination of milk and milk products from the diet. Many can tolerate limited amounts of lactose. Avoid low-fat or non-fat milk as this travels quickly through the gut and tends to cause symptoms in those who are lactose intolerant. Also, many low-fat milk products contain skim-milk powder, providing a higher lactose dose. The fat content in full-cream milk slows the absorption of the milk through the intestines, allowing the lactase enzymes more time to break down the sugars. Milk pre-treated with lactase is available. Non-prescription lactase enzyme preparations are available to improve milk tolerance. Yoghurt containing bacterial lactases may be well tolerated. Soy foods are lactose free and provide a good substitute source of calcium. Calcium supplements are often recommended, particularly for women on a reduced-lactose or lactose-free diet.

Nursing care

Nursing care for a person with lactose intolerance focuses on providing education and support. Discuss sources of lactose: milk, ice-cream and cottage cheese are high in lactose; aged cheese and yoghurt contain much smaller amounts. Potential hidden sources of lactose include desserts made from milk and milk chocolate, sauces and gravies, and cream soups. Suggest a trial of lactase-treated milk or lactase enzyme supplements. Emphasise the importance of obtaining nutrients contained in dairy products from other sources. Proteins may be obtained from meats, eggs, legumes and grains. Other sources of calcium include soy products, sardines, oysters and salmon, as well as plant sources such as beans, cauliflower, rhubarb and green leafy vegetables.

THE PERSON WITH SHORT BOWEL SYNDROME

The small bowel may be resected due to tumours, infarction of bowel mucosa, incarcerated hernias, Crohn's disease, trauma and enteropathy resulting from radiation therapy. Resection of significant portions of the small intestine may result in a condition known as *short bowel syndrome*. The severity of the disorder depends on the total amount of bowel resected, as well as the portions of bowel removed. Removal of the proximal portions, including the duodenum, jejunum and proximal ileum, and of the distal portion of the ileum is associated with more severe malabsorption and manifestations than is resection of mid portions of the ileum.

Resection of the small intestine affects the absorption of water, nutrients, vitamins and minerals. Transit time of ingested foods and fluids is reduced and digestive processes are impaired. The bowel undergoes an adaptive process in which the remaining villi enlarge and lengthen to increase the absorptive surface following resection. For many people, absorption and bowel function return to preoperative or near-normal levels. Others continue to have

significant impairment of digestion and absorption, leading to nutrient deficiencies, weight loss and diarrhoea.

INTERPROFESSIONAL CARE

Management of short bowel syndrome focuses on alleviating manifestations. The person often requires frequent, small, high-kilojoule and high-protein meals.

Diagnosis

Laboratory and diagnostic studies are used to evaluate nutrient deficiencies. Total serum proteins and albumin are reduced, as are serum levels of folate, iron, vitamins, minerals and electrolytes. Anaemia and a prolonged prothrombin time (indicative of vitamin K deficiency) may develop.

Medications

Multivitamin and mineral supplementation are also frequently necessary. Antidiarrhoeal medications are used to reduce bowel motility, allowing a greater amount of time for nutrient absorption. Some people are affected by gastric hypersecretion following bowel resection. For these individuals, a proton-pump inhibitor such as omeprazole (Losec) may be ordered. Individuals with severe manifestations of short bowel syndrome may require TPN.

Nursing care

Nursing care for a person with short bowel syndrome focuses on the problems of potential fluid volume deficit, malnutrition and diarrhoea.

Fluid losses are generally greatest in the initial periods following surgery, warranting the closest attention during that time. Close monitoring of vital signs, intake and output, daily weights, skin turgor and condition of mucous membranes is vital. It is important to remember the risk is also high when other abnormal fluid losses occur through, for example, fever, wound drainage or excessive diaphoresis.

Documentation of nutritional status includes weight, anthropometric measurements, laboratory values and kilojoule intake. Provide nutritional supplementation with enteral feedings as needed. Maintain central lines and TPN, using aseptic technique.

For diarrhoea, document the number, volume and character of stools. Administer antidiarrhoeal medications as ordered. If the person is lactose intolerant, limit intake of milk and milk products. Provide good skin care for the perianal region to prevent breakdown from frequent bowel movements. Refer to the discussion of nursing care for a person with coeliac disease for other measures for altered nutrition and diarrhoea.

A person affected by this condition and their family require extensive education. Because there is no way to cure or replace the lost bowel, the individual must manage the disorder on a daily basis. Provide instructions about the recommended diet and medication regimen. Emphasise the importance of maintaining an adequate fluid intake, particularly in hot weather or during strenuous exercise. Teach the person to monitor their weight frequently and report changes. Include teaching about possible manifestations of dehydration and nutrient deficiencies to be reported to the doctor. Referring the person to a nutritionist or counsellor helps the person manage and cope with what will be a lifelong condition.

NEOPLASTIC DISORDERS

Cancer is the fourth leading cause of death in Australia, preceded by heart disease, stroke, and dementia and Alzheimer's disease (Australian Bureau of Statistics (ABS), 2015). Although cancer may affect any portion of the digestive tract, the large intestine and rectum are common sites. Colorectal cancer (CRC) comprises cancers of the colon and rectum, the two main sections of the large bowel. It is the most commonly diagnosed cancer in Australia after prostate cancer and is a major contributor to mortality, making it a significant healthcare concern (ABS, 2015).

THE PERSON WITH POLYPS

A *polyp* is a mass of tissue arising from the bowel wall and protruding into the lumen. Polyps may develop in any portion of the bowel, occurring most often in the sigmoid colon and rectum. They vary considerably in size and may be single or multiple. It is estimated that approximately 30% of people over the age of 50 have polyps. Although most polyps are benign, some have the potential to become malignant. Familial adenomatous polyposis (FAP) is a syndrome with a dominant inheritance pattern leading to hundreds to thousands of adenomatous polyps developing. Some of these polyps inevitably become malignant (Fauci et al., 2008).

Pathophysiology

Polyps are identified by their structure and tissue type. Most polyps are adenomas, benign epithelial tumours considered premalignant lesions. More than 95% of adenocarcinomas arise from adenomas. However, less than 1% of polyps become malignant (Shussman & Wexner, 2014). Of polyps removed during colonoscopy, more than 70% are adenomatous (Shussman & Wexner, 2014).

Adenomatous polyps represent disruption of the normal process of cell proliferation to replace epithelial cells lining the intestine. Cells are constantly being reproduced

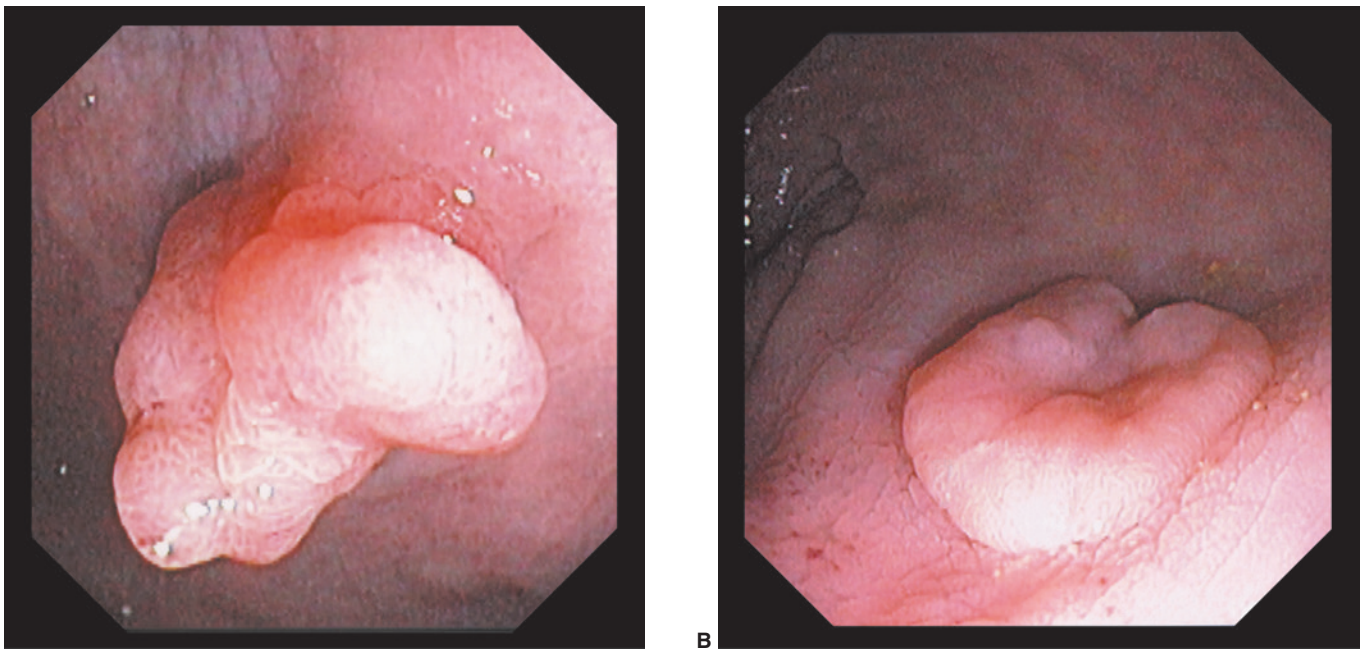


FIGURE 23.13 ■ *A*, Tubular (or pedunculated) polyps; *B*, villous (or sessile) polyps

Source: © David M. Martin MD/Science Source.

to replace those shed as faeces move through the colon. Disruption of the normal process of cell division and maturation lead to formation of a polyp composed of tightly packed epithelial cells. The cells may appear grossly normal or show signs of dysplasia. Polyps may develop as tubular, villous or tubulovillous adenomas. Polyps are named by the way they are attached to the bowel wall as either sessile (raised nodules) or pedunculated (attached by a stalk) (see Figure 23.13).

Tubular adenomas (also called pedunculated polyps) are more common than sessile polyps and account for about 65% of benign polyps of the large intestine (Marieb & Hoehn, 2013). A tubular adenoma is a globe-like structure attached to the intestinal wall by a thin, stalk-like stem. The incidence of this type of polyp increases with age, although it occurs in all age groups and in both genders. Most are small, 1 cm or less in diameter, although they may be as large as 4 to 5 cm. The malignant potential of these polyps seems to be related to their size. Adenomas smaller than 1 cm have a low risk of being malignant and adenomas larger than 1 cm have a much higher risk of harbouring malignancy or a high-grade dysplasia. Adenomatous polyps are present in 35% of adults over 50 years of age (Shussman & Wexner, 2014).

Villous adenomas (also called sessile polyps) are broad based with an elevated, cauliflower-like surface (see Figure 23.13B). These typically develop in the rectosigmoid colon. This type of polyp is often larger than tubular adenomas; usually more than 5 cm. Villous adenomas are not common, accounting for about 10% of colon polyps. These have a higher malignant potential than tubular adenomas. Some adenomatous polyps contain both tubular epithelium and villi and are known as *tubulovillous adenomas*.

Manifestations

Most polyps are asymptomatic, found coincidentally during routine examination or diagnostic testing. Intermittent painless bright or dark red rectal bleeding is the most common presenting complaint. A large polyp may cause abdominal cramping, pain or manifestations of obstruction. Diarrhoea and mucus discharge are associated with a large villous adenoma.

INTERPROFESSIONAL CARE

The diagnosis of intestinal polyps is generally based on diagnostic studies such as sigmoidoscopy or colonoscopy. A rectal polyp may be palpable on digital examination. However, further studies are necessary to determine its size and type and the extent of colon involvement and to assess for malignancy.

Once identified, polyps are removed because of the risk of malignancy. Pedunculated polyps and small villous lesions may be removed during colonoscopy using an electrocautery snare or hot biopsy forceps passed through the scope. This relatively safe procedure has less than a 2% risk of complications such as perforation or haemorrhage. Large villous adenomas are completely excised and examined histologically for evidence of malignancy. In some cases, the colon segment containing the polyp is resected. A person with FAP usually undergoes a total colectomy with ileorectal anastomosis before age 20 years to significantly reduce their risk of developing colon cancer.

Treatment following polypectomy depends on histological examination of the excised tissue. Because polyps tend to

recur, follow-up colonoscopy is recommended in 3 years and then every 5 years if no further polyps are detected. When the polyp is found to be malignant, follow-up care is determined by the tissue type and degree of invasion.

Nursing care

Health promotion

The incidence of intestinal polyps increases with age. They affect men and women equally. It is believed an adenomatous polyp requires more than 5 years of growth to become significant in size and malignant potential. Advise the individual to have a screening for colorectal cancer (with a colonoscopy being the 'gold standard' for diagnosis) at age 50 and as recommended thereafter for early detection of polyps (Australian Institute of Health and Welfare (AIHW), 2015).

Assessment

Polyps are a 'silent' disease, with few or no manifestations.

- *Health history:* rectal bleeding; personal or family history of intestinal polyps or colorectal cancer.

Nursing diagnoses and interventions

Nursing care for a person with polyps focuses on informing and assisting the person through diagnostic testing and polyp removal. Before and after the colonoscopy and polypectomy, provide direct care and teaching about the procedure, expected sensations during the procedure and anticipated postoperative care. Cathartics are prescribed prior to colonoscopy; cleansing enemas may also be ordered. Observe for evidence of fluid and electrolyte imbalance during preoperative preparation. If enemas are ordered, use normal saline (not tap water) to reduce the risk of electrolyte imbalances. Following polypectomy, observe closely for possible complications such as haemorrhage.

Community-based care

Include the following topics when teaching for home care:

- the significance of polyps and their relationship to colorectal cancer
- the importance of keeping follow-up appointments and undergoing repeat colonoscopy as recommended: at 3 years following polypectomy, then every 5 to 10 years unless additional polyps are found
- manifestations to be reported to the doctor, such as diarrhoea, pain, rectal bleeding, light headedness or other indications of possible blood loss.

THE PERSON WITH COLORECTAL CANCER

Colorectal cancer (cancer of the colon or rectum) is the second most common cancer diagnosed in Australia (ABS, 2015). In 2012, it was estimated that 15 840 (8760 males and 7080 females) were diagnosed with colorectal cancer (AIHW, 2015). In Australia in 2011, the risk of being diagnosed with colorectal cancer by the age of 85 years was 1 in 10 for males and 1 in 15 for females; however, from the age of 45 years the risk increases sharply (AIHW, 2015). Colorectal cancer occurs most frequently after age 50. The incidence continues to rise with increasing age.

Bowel cancer accounts for 12.7% of all deaths from invasive cancers, with 3092 deaths in 2010, making bowel cancer the second most common cause of cancer-related death after lung cancer (AIHW, 2015). Unfortunately, there is no national data on the incidence of colorectal cancer in Australian Aboriginal and Torres Strait Islander peoples as not all states and territories record the incidence of cancer in Indigenous Australians. However, where the incidence of cancer is recorded specifically for Aboriginal and Torres Strait Islander peoples, colorectal cancer is a commonly occurring cancer in Indigenous men. Cancer has a greater impact on Aboriginal and Torres Strait Islander peoples, who are less likely to have an early diagnosis and receive adequate treatment such as preventive, curative and palliative services (AIHW, 2015).

Earlier diagnosis and improved treatment have improved the survival rate for colorectal cancer. With early diagnosis and treatment, the 5-year survival rate for colorectal cancer is around 90%; however, only 39% of colorectal cancers are diagnosed at this early stage (AIHW, 2015). Survival varies according to the extent of the cancer development at diagnosis: at stage I colorectal cancer, 90% of individuals are still alive at 5 years; this falls to 87% with stage II, 57% with stage III and 10% for widespread disease (AIHW, 2015).

Although the specific cause of colorectal cancer is unknown, a number of risk factors have been identified (see Box 23.3). Genetic factors are strongly linked to the risk of colorectal cancer. Up to 25% of people developing colorectal cancer have a family history of the disease (Shussman & Wexner, 2014). People with familial adenomatous polyposis inevitably develop colon cancer unless the colon is removed. Hereditary nonpolyposis colorectal cancer (also known as Lynch syndrome) is an autosomal dominant disorder that significantly increases the risk of developing colorectal and other cancers. Tumours associated with Lynch syndrome often affect the ascending colon

BOX 23.3 Risk factors for colorectal cancer

- Age over 50 years
- Polyps of the colon and/or rectum
- Family history of colorectal cancer
- Inflammatory bowel disease
- Exposure to radiation
- Diet: high animal fat and kilojoule intake

and occur at an earlier age. Inflammatory bowel diseases also increase the risk of colorectal cancer.

Diet plays a role in the development of colorectal cancer. The disease is prevalent in economically prosperous countries where people consume diets high in kilojoules, meat proteins and fats. This dietary pattern, common in Australia, is thought to increase the population of anaerobic bacteria in the gut. These anaerobes convert bile acids into carcinogens. Diets high in fruits and vegetables, folic acid and calcium appear to reduce the risk of colorectal cancer. Cereal fibre, once thought to reduce colorectal cancer risk, does not appear to play a role either way in its development. Other factors that may reduce the risk of colorectal cancer include regular exercise, taking a daily multivitamin and the use of aspirin and other NSAIDs.

Pathophysiology

Nearly all colorectal cancers are adenocarcinomas beginning as adenomatous polyps. Most tumours develop in the rectum and sigmoid colon, although any portion of the colon may be affected (see Figure 23.14). The tumour typically grows undetected, producing few manifestations. By the time manifestations occur, the disease may have spread into deeper layers of the bowel tissue and adjacent organs. Colorectal cancer spreads by direct extension to involve the entire bowel circumference, the submucosa and the outer bowel wall layers.

Neighbouring structures such as the liver, greater curvature of the stomach, duodenum, small intestine, pancreas, spleen, genitourinary tract and abdominal wall may also be involved by direct extension. Metastasis to regional lymph nodes is the most common form of tumour spread. This is not always an orderly process; distal nodes may contain cancer cells while regional nodes remain normal. Cancerous cells from the primary tumour may also spread via the lymphatic system or circulatory system to secondary sites such as the liver, lungs, brain, bones and kidneys. 'Seeding' of the tumour to other areas of the peritoneal cavity can occur when the tumour extends through the serosa or during surgical resection.

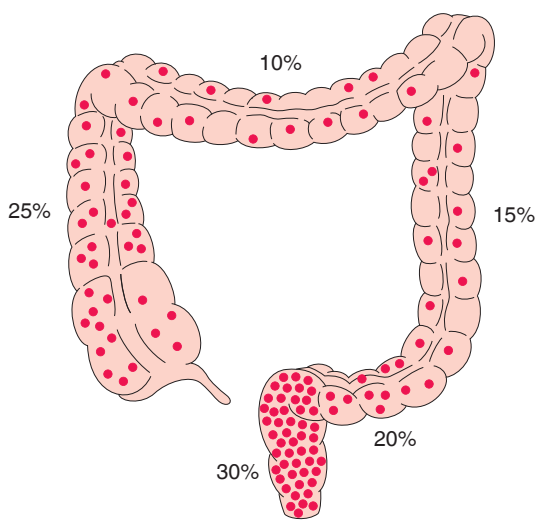


FIGURE 23.14 ■ The distribution and frequency of cancer of the colon and rectum

Manifestations

Bowel cancer often produces no manifestations until it is advanced. Because it grows slowly, 5 to 15 years of growth may occur before manifestations develop. The manifestations depend on its location, type and extent, and complications. Rectal bleeding is often the initial manifestation prompting a person to seek medical care. Other common early manifestations include a change in bowel habits, either diarrhoea or constipation. Pain, anorexia and weight loss are characteristic in advanced disease. A palpable abdominal or rectal mass may be present. Occasionally a person presents with anaemia from occult bleeding.

Complications

The primary complications associated with colorectal cancer are (1) bowel obstruction due to narrowing of the bowel lumen by the lesion; (2) perforation of the bowel wall by the tumour, allowing contamination of the peritoneal cavity by bowel contents; and (3) direct extension of the tumour to involve adjacent organs.

Most recurrences of colorectal cancer after tumour removal occur within the first 4 years. The size of the primary tumour does not necessarily relate to long-term survival. The numbers of lymph nodes involved, penetration of the tumour through the bowel wall and tumour adherence to adjacent organs are better predictors of the prognosis for the disease.

INTERPROFESSIONAL CARE

The focus of interprofessional care for colorectal cancer is prevention, early detection and intervention. Colorectal cancer is always treated by surgical resection, with chemotherapy and radiation therapy used as adjuncts.

Prevention

Measures to prevent colon cancer considered effective and safe include diets high in fruit and vegetables and low in saturated fat and red meat, regular exercise, avoiding obesity and quitting smoking. Consuming fibre supplements, minerals such as calcium, vitamins and non-steriodal anti-inflammatory medications may help prevent colorectal cancer; however, their effectiveness is not yet proven (Principi & DeCensi, 2015). Although considered safe, these measures are the subject of further research to demonstrate conclusive proof of effectiveness (Principi & DeCensi, 2015).

Screening

Cancer Council Australia (2015) recommends one of the following testing schedules for the early detection of colorectal cancer, beginning at age 50. These options are acceptable choices for average-risk adults. Screening for blood in the faeces (faecal occult blood test (FOBT)) is available through the National Bowel Cancer Screening Program (NBCSP) to Australians turning 50. From 2015 NBCSP is being expanded

to implement a biennial screening interval for those aged 50–74 by 2020 (AIHW, 2015). Abnormal tests are followed up with a colonoscopy.

- Yearly FOBT or faecal immunochemical test (FIT). (For FOBT, the take-home multiple sample method should be used.)
- Flexible sigmoidoscopy every 5 years.
- Yearly FOBT or FIT plus flexible sigmoidoscopy every 5 years.
- Double-contrast barium enema every 5 years.
- Colonoscopy every 10 years.

Diagnosis

Diagnostic and laboratory tests are used for screening, diagnosis and monitoring purposes. Diagnostic tests include a sigmoidoscopy or colonoscopy as the primary diagnostic test used to detect and visualise tumours. While flexible sigmoidoscopy detects 50% to 65% of colorectal cancers, many clinicians recommend colonoscopy. Tissue for biopsy is obtained at the time of endoscopy to confirm cancerous tissue and evaluate cell differentiation (see Chapter 13).

Laboratory tests used are an FOBT (by guaiac or haemoccult testing) to detect blood in the faeces, an FBC to identify anaemia resulting from chronic blood loss and tumour growth, and a carcinoembryonic antigen (CEA) level, a tumour marker detected in the blood of individuals with colorectal cancer. CEA levels are used to estimate prognosis, monitor treatment and detect cancer recurrence. Newer screening methods for diagnosis of colorectal cancer in the very early stage of development include faecal DNA, immunochemical faecal occult blood tests (iFOBTs) and computed tomographic colonography (CTC) (Leggett & Hewitt, 2015).

Current staging methods primarily use the TNM (tumour, node, metastasis) system, as outlined in Table 23.12. Radiological examinations may include a chest x-ray to detect tumour metastasis to the lung, and computed tomography (CT) scan, magnetic resonance imaging (MRI) or ultrasonic examination may be used to assess tumour depth and involvement of other organs by direct extension or metastasis.

Laser photocoagulation

Laser photocoagulation uses a very small, intense beam of light to generate heat in tissues towards which it is directed. The heat generated by the laser beam can be used to destroy small tumours. It is also used for palliative surgery of advanced tumours to remove obstruction. Laser photocoagulation can be performed endoscopically and is useful for people unable to tolerate major surgery.

Surgery

Stage I and II disease can be treated with surgery to remove the bowel and surrounding lymph nodes. Stage III disease requires surgery and additional chemotherapy to try to prevent recurrence. Widespread disease is treated with chemotherapy. More recently, targeted therapies are being trialled in addition to chemotherapy. Surgical resection of the tumour, adjacent colon and regional lymph nodes is the treatment of choice for colorectal cancer. Options for surgical treatment vary from destruction of the tumour by laser photocoagulation performed during endoscopy to abdominoperineal resection with permanent colostomy. When possible, the anal sphincter is preserved and colostomy avoided.

Other surgical treatment options for small, localised tumours include local excision and fulguration. These procedures may also be performed during endoscopy, eliminating the need for

TABLE 23.12 The TNM classification for colorectal cancer

STAGE	PRIMARY TUMOUR (T)	REGIONAL LYMPH NODES (N)	DISTANT METASTASIS (M)
	TX—Primary tumour cannot be assessed T0—No evidence of primary tumour	NX—Regional lymph node cannot be assessed N0—No regional lymph node metastasis	MX—Presence of distant metastasis cannot be assessed M0—No distant metastasis
Stage 0	Tis—Carcinoma in situ		
Stage I	T1—Tumour invades submucosa		
	T2—Tumour invades muscularis propria		
Stage II	T3—Tumour invades through muscularis propria into subserosa or into non-peritonealised pericolic or perirectal tissues		
	T4—Tumour perforates visceral peritoneum or directly invades other organs or structures		
Stage III	Any T	N1—Metastasis in 1 to 3 pericolic or perirectal lymph nodes	
		N2—Metastasis in 4 or more pericolic or perirectal lymph nodes	
		N3—Metastasis in any lymph node along course of a major named vascular trunk	
Stage IV	Any T	Any N	M1—Distant metastasis

abdominal surgery. Local excision may be used to remove a disk of rectum containing a tumour in a person with a small, well-differentiated, mobile polypoid lesion. *Fulguration* or electrocoagulation is used to reduce the size of some large tumours in a person who is a poor surgical risk. This procedure requires general anaesthesia and may need to be repeated at intervals.

Most people with colorectal cancer undergo surgical resection of the colon with anastomosis of remaining bowel as a curative procedure. The distribution of regional lymph nodes determines the extent of resection because these may contain metastatic lesions. Most tumours of the ascending, transverse, descending and sigmoid colon can be resected.

Tumours of the rectum usually are treated with an abdominoperineal (AP) resection in which the sigmoid colon, rectum and anus are removed through both abdominal and perineal incisions. A permanent sigmoid colostomy is performed to provide for elimination of faeces. Nursing care for a person having bowel surgery is outlined below.

COLOSTOMY

Surgical resection of the bowel may be accompanied by a colostomy for diversion of faecal contents. A **colostomy** is an ostomy made in the colon. It may be created if the bowel is obstructed by the tumour, as a temporary measure to promote healing of anastomoses or as a permanent means of faecal evacuation when the distal colon and rectum are removed. Colostomies take the name of the portion of the colon from which they are formed: ascending colostomy, transverse colostomy, descending colostomy and sigmoid colostomy (see Figure 23.15).

A *sigmoid colostomy* is the most common permanent colostomy performed, particularly for cancer of the rectum. It is usually created during an AP resection. This procedure involves the removal of the sigmoid colon, rectum and anus through abdominal and perineal incisions. The anal canal is closed and a stoma formed from the proximal sigmoid colon. The stoma is usually located on the lower left quadrant of the abdomen.

When a *double-barrel colostomy* is performed, two separate stomas are created (see Figure 23.16). The distal colon is not

NURSING CARE OF THE PERSON requiring bowel surgery

PREOPERATIVE NURSING CARE

- Provide routine preoperative care for a person undergoing surgery as outlined in Chapter 3.
- Arrange for consultation with stomal therapy (ST) specialist if appropriate. *The ST nurse is trained to identify and mark an appropriate stoma location, taking into consideration the level of stoma, skinfolds and the person's clothing preferences. Initial stomal care teaching also is provided by the ST nurse during the preoperative visit.*
- Insert a nasogastric tube if ordered. *Although it is often inserted in the operating theatre just prior to surgery, the nasogastric tube may be placed preoperatively to remove secretions and empty stomach contents.*
- Perform bowel preparation procedures as ordered. *Oral and parenteral antibiotics as well as laxatives and enemas may be prescribed preoperatively to clean the bowel and reduce the risk of peritoneal contamination by bowel contents during surgery.*

POSTOPERATIVE NURSING CARE

- Provide routine care for the person postoperatively (see Chapter 3).
- Monitor bowel sounds and degree of abdominal distension. *Surgical manipulation of the bowel disrupts peristalsis, resulting in an initial ileus. Bowel sounds and the passage of flatus indicate a return of peristalsis.*
- Assess the position and patency of the nasogastric tube, connecting it to low suction. If the tube becomes clogged, gently irrigate with sterile normal saline. *A nasogastric or gastrostomy tube is used postoperatively to provide gastrointestinal decompression and facilitate healing of the anastomosis. Ensuring its patency is important for comfort and healing.*
- Assess colour amount and odour of drainage from surgical drains and the colostomy (if present), noting any changes or the presence of clots or bright bleeding. Initial drainage may be bright red and then become dark and finally clear or greenish yellow over the first 2 to 3 days. *A change in the colour, amount or odour of the drainage may indicate a complication such as haemorrhage, intestinal obstruction or infection.*
- Alert all personnel caring for the person with an AP resection to avoid rectal temperatures, suppositories or other rectal procedures. *These procedures could disrupt the anal suture line, causing bleeding, infection or impaired healing.*
- Maintain intravenous fluids while nasogastric suction is in place. *A person on nasogastric suction is unable to take oral food and fluids, and is losing electrolyte-rich fluid via the nasogastric tube. If replacement fluid and electrolytes are not maintained, the person is at risk of dehydration; sodium, potassium and chloride imbalance; and metabolic alkalosis.*
- Provide antacids, histamine$_2$ receptor antagonists and antibiotic therapy as ordered. *The above medications may be ordered for the person postoperatively depending on the procedure performed. Antibiotic therapy is a common measure to prevent infection resulting from contamination of the abdominal cavity with gastric contents.*
- Resume oral food and fluids as ordered. Initial feedings may be clear fluids, progressing to free fluids and then frequent small feedings of regular foods. Monitor bowel sounds and for abdominal distension frequently during this period. *Oral feedings are reintroduced slowly, minimising abdominal distension and trauma to the suture lines.*
- Begin discharge planning and teaching. Consult with a nutritionist for instructions and menu planning; reinforce teaching. *Advise about potential postoperative complications such as abdominal abscess or bowel obstruction, their preventive measures and clinical manifestations.*

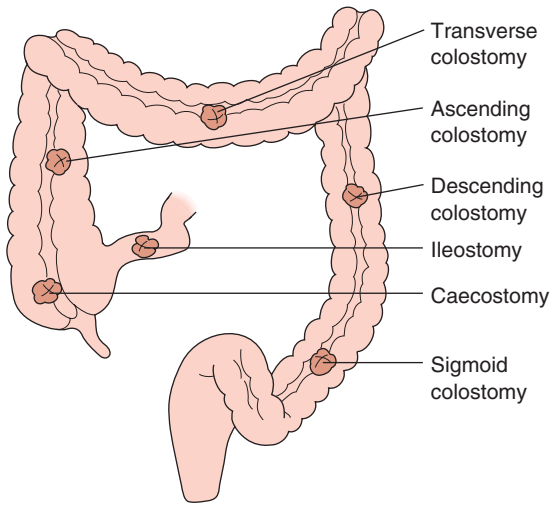


FIGURE 23.15 ■ Various ostomy levels and sites

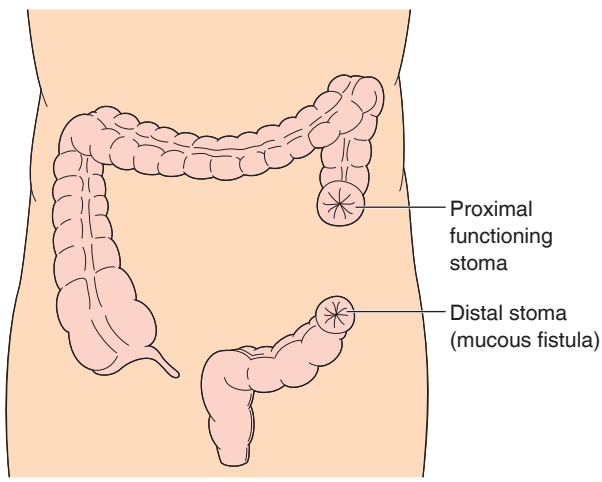


FIGURE 23.16 ■ A double-barrel colostomy. The proximal stoma is the functioning stoma; the distal stoma expels mucus from the distal colon

removed, but bypassed. The proximal stoma, which is functional, diverts faeces to the abdominal wall. The distal stoma, also called the mucous fistula, expels mucus from the distal colon. It may be pouched or dressed with a 4 × 4 lint-free gauze dressing. A double-barrel colostomy may be created for cases of trauma, tumour or inflammation, and may be temporary or permanent.

An emergency procedure used to relieve an intestinal obstruction or perforation is called a *transverse loop colostomy*. During this procedure, a loop of the transverse colon is brought out from the abdominal wall and suspended over a plastic rod or bridge, preventing the loop from slipping back into the abdominal cavity. The loop stoma may be opened at the time of surgery or a few days later at the person's bedside. The bridge may be removed in 1 to 2 weeks. Transverse loop colostomies are generally temporary.

In a *Hartmann procedure*, a common temporary colostomy procedure, the distal portion of the colon is left in place and is over-sewn for closure. A temporary colostomy may be done to allow bowel rest or healing, such as following tumour resection or inflammation of the bowel. It may also be created following traumatic injury to the colon, such as a gunshot wound. Anastomosis of the severed portions of the colon is delayed because bacterial colonisation of the colon prevents proper healing of the anastomosis. About 3 to 6 months after a temporary colostomy, the colostomy is closed and the colon is reconnected. A person with a temporary colostomy requires the same care as an individual with permanent colostomy. See the 'Nursing care of a person with a colostomy' box below.

Radiation therapy

Although radiation therapy is not used as a primary treatment for colon cancer, it is used with surgical resection for treating rectal tumours. Small rectal cancers may be treated with intracavitary, external or implantation radiation. Rectal cancer has a high rate of regional recurrence following complete surgical resection, particularly when the tumour has invaded tissues outside the bowel wall or regional lymph nodes. Pre- or postoperative radiation therapy reduces the recurrence of pelvic tumours, although the effect of radiation therapy on long-term survival is less clear. Radiation therapy is also used preoperatively to shrink large rectal tumours enough to permit their surgical removal. Further discussion about radiation therapy and nursing implications is included in Chapter 13.

Chemotherapy

Chemotherapeutical agents, such as intravenous fluorouracil (5-FU) and folinic acid (leucovorin), are also used postoperatively as adjunctive therapy for colorectal cancer. When combined with radiation therapy, chemotherapy reduces the rate of tumour recurrence and prolongs survival in people with stage II and stage III rectal tumours. The benefit for colon cancers is less clear, but chemotherapy may be used to reduce its spread to the liver and prevent recurrence. Irinotecan (CPT-11) or oxaliplatin may also be used in chemotherapy regimens for colorectal cancer. Further discussion about chemotherapy and nursing implications is included in Chapter 13.

Nursing care

Health promotion

Primary prevention of colorectal cancer is a significant nursing care issue. Discuss the dietary recommendations provided by the Cancer Council Australia (2015) for the prevention of colorectal cancer. These recommendations include decreasing the amount of fat, refined sugar and red meats in the diet while increasing intake of dietary fibre. Foods containing high amounts of fibre include raw fruit and vegetables, legumes and wholegrain products.

NURSING CARE OF THE PERSON with a colostomy

- Assess the location of the stoma and the type of colostomy performed. *Stoma location is an indicator of the section of bowel in which it is located and a predictor of the type of faecal drainage to expect.*
- Assess stoma appearance and surrounding skin condition frequently (see the box 'Nursing care of a person having an ileostomy' on page 746). *Assessment of stoma and skin condition is particularly important in the early postoperative period, when complications are most likely to occur and are most treatable.*
- Position a collection bag or drainable pouch over the stoma. Initial drainage may contain more mucus and serosanguineous fluid than faecal material. As the bowel resumes function, drainage becomes faecal in nature. *The consistency of drainage depends on the stoma location in the bowel.*
- If ordered, irrigate the colostomy, instilling water into the colon similar to an enema procedure. *The water stimulates the colon to empty.*
- When colostomy irrigation is ordered for a person with a double-barrel or loop colostomy, irrigate the proximal stoma. Digital assessment of the bowel direction from the stoma can assist in determining the proximal stoma. *The distal bowel carries no faecal contents and does not need irrigation. It may be irrigated for cleansing just prior to reanastomosis.*
- Empty a drainable pouch or replace the colostomy bag as needed or when it is no more than one-third full. *If the pouch is allowed to overfill, its weight may impair the seal, causing leakage.*
- Provide stomal and skin care for a person with a colostomy as for a person with an ileostomy (see the box 'Nursing care of a person having an ileostomy' on page 746). *Good skin and stoma care is important to maintain skin integrity and function as the first line of defence against infection.*
- Use caulking agents, such as Stomahesive or karaya paste and a skin barrier wafer, as needed to maintain a secure ostomy pouch. *This is particularly important for a person with a loop colostomy. The main challenge for a person with a transverse loop colostomy is to maintain a secure ostomy pouch over the plastic bridge.*
- If the pouch does not incorporate an air vent, a small needle hole high on the colostomy pouch allows flatus to escape. This hole may be closed with a Band-Aid, and opened when the person is in the bathroom, for odour control. *Ostomy bags may 'balloon' out, disrupting the skin seal, if excess gas collects.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Prior to discharge, provide written, verbal and psychomotor instruction on colostomy care, pouch management, skin care and irrigation to the person. Whether the colostomy is temporary or permanent, the person will be responsible for its management. *Good understanding of procedures and care enhances the ability to provide self-care, as well as self-esteem and control.*
- Allow ample time for the person (and their family, if necessary) to practise changing the pouch, either on the person or a model. *Practice of psychomotor skills improves learning and confidence.*
- If an abdominoperineal resection has been performed, emphasise the importance of using no rectal suppositories, rectal temperatures or enemas. Suggest the person carry medical identification or a MedicAlert® tag or bracelet. *These measures are important to prevent tissue trauma when the rectum has been removed.*
- The diet for a person with a colostomy is individualised and may require no alteration from that consumed preoperatively. Dietary teaching should, however, include information on foods causing stool odour and gas, and foods that thicken or loosen stools. *Foods causing these effects on ostomy output are listed below.*

Foods increasing stool odour

- Asparagus
- Beans
- Cabbage
- Eggs
- Fish
- Garlic
- Onions
- Some spices

Foods increasing intestinal gas

- Beer
- Broccoli
- Brussels sprouts
- Cabbage
- Carbonated drinks
- Cauliflower
- Corn
- Cucumbers
- Dairy products
- Dried beans
- Peas
- Radishes
- Spinach

Foods thickening stools

- Apple sauce
- Bananas
- Bread
- Cheese
- Pasta
- Pretzels
- Rice
- Smooth peanut paste
- Tapioca
- Yoghurt

Foods loosening stools

- Chocolate
- Dried beans
- Fried foods
- Greasy foods
- Highly spiced foods
- Leafy green vegetables
- Raw fruits and juices
- Raw vegetables

Foods colouring stools

- Beetroot
- Red and green jelly

Stress the importance of regular health examinations, including digital rectal exams. Discuss recommendations for regular haemoccult testing of stool after age 50. Include the importance of seeking medical treatment if blood is noted in or on the stool. Teach the warning signs for cancer, including those specific to bowel cancer, such as a change in bowel habits.

Assessment

- *Health history:* usual bowel patterns and any recent changes; weight loss, fatigue, decreased activity tolerance; presence of blood in the stool; pain with defecation, abdominal discomfort, perineal pain; usual diet; family history of colon cancer; other specific risk factors such as inflammatory bowel disease or colon polyps.
- *Physical examination:* general appearance; weight; abdominal shape, contour; bowel sounds, abdominal tenderness; stool haemoccult or guaiac.

Nursing diagnoses and interventions

In planning and implementing care, consider both physical care needs and the person's psychological response to the diagnosis. The psychological recovery of a person who is diagnosed with colorectal cancer may be influenced by the possible permanent changes to their body image and lifestyle. When planning care, open and genuine communication enables nurses to understand the person's anxieties, personal, family and social needs (Kunde, 2012). As colorectal cancer is often advanced at the time of diagnosis, the prognosis, even with treatment, may be poor. Denial and anger are common. Extensive abdominal surgery and potentially a colostomy may be necessary and the effects of chemotherapy and radiation therapy can leave the person fatigued and discouraged. A nursing care plan for a person with colorectal cancer follows.

Nursing care includes providing emotional support, teaching and direct care before and after diagnostic procedures and surgery and during adjunctive treatments. When developing nursing care plans it is imperative to incorporate the person's cultural, religious and personal viewpoints into the plan of care. Priority nursing diagnoses include *Acute pain*, *Imbalanced nutrition* and *Anticipatory grieving*. *Risk of sexual dysfunction* should be considered as a priority diagnosis if a colostomy was created.

CONSIDERATION FOR PRACTICE

If an adominoperineal resection (AP) resection was performed, alert all care personnel to avoid rectal temperatures, suppository use or other procedures that could damage sutures.

Acute pain

A person with colorectal cancer may experience pain related to preparatory procedures, diagnostic examinations and surgery. Following an AP, 'phantom' rectal pain related to the severing of nerves during the wide excision of the rectum may develop. Finally, the primary tumour itself and, potentially, metastatic tumours may impinge on nerves and other organs, causing pain. In the early postoperative period, an epidural infusion or PCA is often used to manage pain. PCA, routine administration of ordered analgesics or a continuous analgesia delivery (CAD) system may also be used for pain management when the tumour is far enough advanced to preclude surgical resection. See Chapter 8 for more information on caring for a person with pain, Chapter 13 for discussion of pain associated with cancer and Chapter 4 for discussion of end-of-life care.

- Assess and monitor for adequate pain relief. Use subjective and objective information, including the location, intensity and character of the pain, as well as non-verbal signs such as grimacing, muscle tension, apparent dozing, changes in pulse or blood pressure, and rapid, shallow respirations. Response to pain varies between individuals and is influenced by culture, beliefs, pathophysiological factors and psychological factors such as previous pain events (McArthur, 2011). A person may assume that pain is to be expected or tolerated or may fear becoming addicted to analgesic medications. *Careful questioning and a holistic assessment can provide accurate information about pain status, allowing better control of discomfort.*
- Ask the person to rate pain using a 0 to 10 pain scale. Document the level of pain. Pain is a subjective experience. *People perceive and respond to pain differently. Religion and ethnic background may affect the response to pain.*
- Monitor analgesic effectiveness 30 minutes after administration. Monitor for pain relief and adverse effects. *The method of delivery, dosage or medication itself may need to be adjusted to provide adequate pain relief.*
- Assess the incision for inflammation or swelling; assess drainage catheters and tubes for patency. *Poorly controlled pain, or pain that changes, may be related to organ distension from an obstructed nasogastric tube, urinary catheter or wound drain, or may indicate an infection.*
- Assess the abdomen for distension, tenderness and bowel sounds. *Intra-abdominal bleeding, peritonitis or paralytic ileus can cause pain that may be confused with incisional pain.*
- Administer analgesia prior to an activity or procedure. *Adequate pain relief reduces muscle tension, allowing for more comfortable participation in activities.*
- Assist with adjunctive comfort measures, such as positioning, diversional activities, management of environmental stimuli, guided imagery and teaching relaxation techniques. *These measures enhance the effects of analgesia by reducing muscle tension.*
- Splint incision with a pillow and teach the person how to self-splint when coughing and deep breathing *to prevent respiratory complications related to fear of pain.*

Imbalanced nutrition: less than body requirements

Bowel preparation for diagnostic procedures, surgery, radiation therapy and chemotherapy place the person with colorectal cancer at risk of nutritional deficiencies. Fluid and electrolyte replacement is provided following surgery, along with possible TPN (see Chapter 21). Adequate kilojoule and nutrient intake is necessary for healing after surgery. Additionally, if the

NURSING CARE PLAN A person with colorectal cancer

Bill Cunningham is a 65-year-old retired railway employee, husband and father of three grown children. For the past 3 months, Mr Cunningham has noticed small amounts of blood and occasional mucus in his stools. He has a sensation of pressure in the rectum and noticed his stools are smaller in diameter, about the size of a pencil. After palpating a mass on digital examination of the rectum, the doctor orders a colonoscopy. A large sessile lesion is found in the rectum and biopsied. The pathology report shows the lesion to be adenocarcinoma. Mr Cunningham is scheduled for an abdominoperineal resection and sigmoid colostomy.

ASSESSMENT

Madonna Hart, RN, completes the admission assessment. Mr Cunningham states his bowel habits have recently changed, but denies pain or other symptoms. Physical assessment findings include T 36.9°C, P 82, R 18 and BP 118/78. He is 178 cm tall and weighs 84 kg. Laboratory findings are normal except for the previous pathology report of adenocarcinoma of rectal lesion.

Mr Cunningham states, 'I really don't want a colostomy, but if that is what it takes to get rid of this, I'm ready to get it over with.'

DIAGNOSES

- *Altered comfort* related to acute pain secondary to surgical intervention.
- *Risk of impaired skin integrity (peristomal)* related to faecal drainage and pouch adhesive.
- *Risk of constipation/diarrhoea* related to effects of surgery on bowel function.
- *Disturbed body image* related to colostomy.
- *Risk of sexual dysfunction* related to wide rectal incision, radiation therapy and colostomy.

PLANNING

When planning nursing care with Mr Cunningham, it is vital to consider his preferences, religious and cultural beliefs for incorporation into his plan of care.

Expected outcomes

- Mr Cunningham will report pain within an acceptable range that allows ease of movement and ambulation.
- Mr Cunningham will perform colostomy care using correct technique.
- Mr Cunningham will demonstrate willingness to discuss changes in sexual function.
- Mr Cunningham will wear clothing to enhance physical and emotional self-esteem.

IMPLEMENTATION

- Provide Mr Cunningham with analgesia as ordered, evaluating its effectiveness and monitoring for adverse reactions.
- Provide for privacy when teaching and discussing concerns about ostomy.
- Discuss with Mr Cunningham foods that cause odour and gas.
- Teach Mr Cunningham how to care for his colostomy.
- Maintain consistent nursing personnel assignment to facilitate trust.
- Provide Mr Cunningham information about the Australian Stoma Appliance Scheme. It is subsidised by the Australian government and provides stoma products (medicine and appliances) for people with a permanent or temporary stoma free of charge to members of one of the 22 approved volunteer stoma associations.
- Refer Mr Cunningham to the local Stoma Association.

EVALUATION

At discharge, Mr Cunningham is able to empty and rinse out his colostomy pouch. He is changing the pouch and caring for surrounding skin appropriately. RN Hart gave him verbal and written instructions on colostomy care. He verbalised understanding of phantom rectal pain and the importance of avoiding rectal suppositories. He expresses an understanding of the need to avoid heavy lifting and the importance of follow-up care. RN Hart refers Mr Cunningham to a home healthcare agency in his community if he has further questions and for his follow-up care.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the cause of phantom rectal pain.
2. Outline why it is important to discuss dietary concerns with a person with a colostomy, especially odour- and gas-forming foods.
3. Outline a plan to teach Mr Cunningham how to irrigate a colostomy.
4. Develop a care plan for Mr Cunningham for the nursing diagnosis *Disturbed body image*.

REFLECTION ON THE NURSING PROCESS

1. Reflect on what you learned from completing this section. Outline how you will apply this knowledge to assist you in your clinical reasoning when next on clinical placement.
2. Describe nursing interventions you can incorporate into your clinical practice to assist a person to be independent in caring for their colostomy.

tumour is advanced, metabolic needs may be increased and the appetite decreased.

- Assess nutritional status using data such as height and weight, skinfold measurements, body mass index (BMI) calculation (see Chapter 20) and laboratory data including serum albumin level. Refer to nutritionist for dietary management. *A person malnourished before beginning aggressive cancer treatment requires more vigorous nutrition management to promote healing.*
- Assess readiness for resumption of oral intake after surgery or procedures using data such as statements of hunger, presence of bowel sounds, passage of flatus and minimal abdominal distension. *Manipulation of the bowel interrupts peristalsis of the GI tract. It is important to ensure that peristalsis has resumed prior to resumption of oral intake.*
- Monitor and document food and fluid intake. *Documentation helps identify the adequacy of kilojoule and other nutrient intake.*

- Weigh daily. *Weight fluctuation may indicate adequate or inadequate dietary intake.*
- Maintain TPN and central intravenous lines as ordered. *Parenteral nutrition prevents tissue catabolism and promotes healing when food intake is disrupted for more than 2 to 3 days.*
- When oral intake resumes, help the person develop a meal plan incorporating their food preferences and that considers their schedule and environment. *Consideration of likes, dislikes and circumstances in meal planning promotes adequate intake.*

Anticipatory grieving

When a bowel resection is performed for colorectal cancer, the person needs to adjust to the loss of a major body part as well as to the diagnosis of cancer. Even when the prognosis for recovery is good, many people perceive cancer as fatal. Supporting the person and their family during the initial stages of grieving can improve physical recovery as well as psychological coping and eventual adaptation. See Chapter 4 for discussion of nursing care of people experiencing loss and grief.

- Work to develop a trusting relationship with the person and their family. *This increases the nurse's effectiveness in helping them work through the grieving process.*
- Listen actively, encouraging the person and their family to express their fears and concerns. Assist to identify strengths, experiences and support systems.
- Demonstrate respect for cultural, spiritual and religious values and beliefs; encourage use of these resources to cope with losses.
- Encourage discussion of the potential impact of loss on individual family members, family structure and family function. Assist family members to share concerns with one another.
- Refer to cancer support groups, social services or counselling as appropriate. *These resources can be used throughout the grieving process.*

Risk of sexual dysfunction

Colorectal cancer and ostomy surgery increase the risk of sexual dysfunction, defined as a change in sexual function so that it becomes unsatisfying, unrewarding or inadequate (NANDA-I, 2011). Physical factors leading to sexual dysfunction include disruption of nerves and blood vessels supplying the genitals, radiation therapy, chemotherapy and other medications prescribed after surgery.

Psychologically, an *ostomate* (person with an ostomy) experiences an altered body image and may develop low self-esteem. The person may feel undesirable and fear rejection. They may be concerned about odours or pouch leakage during sexual activity. This emotional stress can also contribute to sexual dysfunction.

- Provide opportunities for the person and their family to express feelings about the cancer diagnosis, stoma and effects of other treatments. *Encouraging verbalisation of feelings about the diagnosis, stoma and treatments provides an opportunity to validate that feelings of anger and depression are normal responses to the diagnosis and change in body function.*
- Provide consistent care. *An accepting attitude and consistent care by nurses, as well as securing the appliance and controlling odour and leakage, can increase the person's confidence and independence in caring for the appliance.*
- Encourage expression of sexual concerns. Provide privacy for discussions with a caregiver who has an established trust with the person and their partner. Sexuality is a private concern to most people. *The person and their partner are not likely to express their concerns openly unless trust has been established.*
- Reassure the person and their partner that the effect of physical illness and prescribed interventions on sexuality is usually temporary. *The person and their partner may misinterpret an initial decrease in libido as evidence sexual activity will not be possible or resume following recovery.*
- Refer the person and their partner to social services or a family counsellor for further interventions. A person is often discharged from acute care settings well before concerns about sexual activity surface. *Ongoing counselling provides a continuing resource.*
- Arrange for a visit from a member of the Stoma Association. *People living and coping with an ostomy can provide information and support, helping the new ostomate overcome feelings of isolation and rejection.*

Community-based care

During the diagnostic and preoperative periods, provide instruction about the following topics:

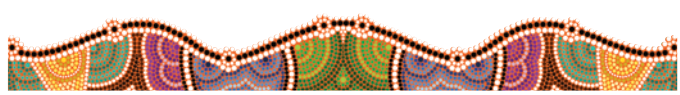
- tests to be performed and preparatory procedures, including dietary restrictions, laxatives, enemas and food and fluid restrictions just prior to the procedure
- recommended postprocedure care and potential adverse effects to report
- preoperative care, such as intestinal preparation and food and fluid restrictions.

If a colostomy is planned, refer to a stomal therapist for stoma placement and initial teaching.

Once treatment has been initiated, include the following topics (as appropriate) in teaching for home care:

- pain management
- skin care and management of potential adverse effects of radiation therapy and/or chemotherapy (Refer to Chapter 13 for further discussion of teaching needs related to these therapies.)
- incision and stoma care
- recommended diet
- follow-up appointments and care.

If the tumour is inoperable or a cure is not anticipated, provide information about pain and symptom management. Discuss the hospice philosophy and available services. Provide a referral to a local hospice or home health department. See Chapter 4 for discussion of end-of-life care and Chapter 13 for nursing care of a person with cancer.



STRUCTURAL AND OBSTRUCTIVE BOWEL DISORDERS

Any portion of the intestines may be affected by a structural or obstructive disorder. When the structural defect is in the bowel wall, the intestine may be directly affected, as is the case with diverticula. Defects in the abdominal wall may allow intra-abdominal contents (such as loops of bowel) to protrude, indirectly affecting bowel function. Likewise, obstructions may result from disease of the bowel itself or from obstruction of the bowel lumen by an external force.

THE PERSON WITH A HERNIA

A hernia is a defect in the abdominal wall allowing abdominal contents to protrude out of the abdominal cavity. Trauma, surgery and increased intra-abdominal pressure caused by such conditions as pregnancy, obesity, weight lifting or tumours are risk factors for hernia formation.

Pathophysiology

Hernias are classified by location (see Figure 23.17) and are congenital or acquired. Most hernias occur in the groin (inguinal or femoral hernias). Inguinal hernias are often congenital, caused by improper closure of the tract developing as the testes descend into the scrotum during foetal development. Inguinal hernias may be acquired, resulting from weakness of fascia in a region called Hesselbach's area or from dilation of the femoral ring (e.g. during pregnancy and childbirth). Ventral or incisional hernias of the abdominal wall generally are acquired, caused by weakening of normal abdominal wall musculature. Umbilical hernias also are congenital and usually are detected in infancy. Hiatal hernias develop in the diaphragm (see Chapter 22).

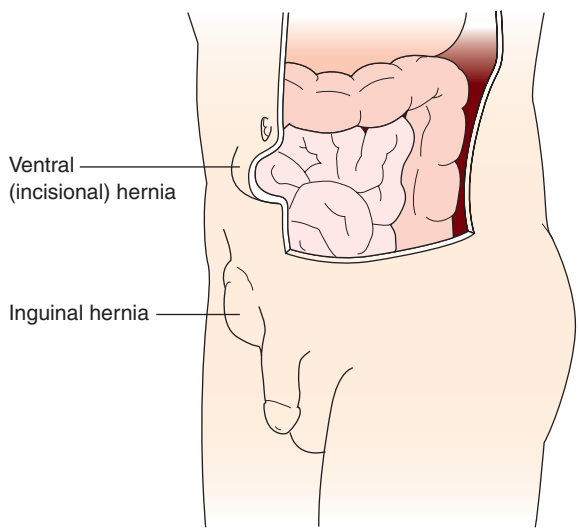


FIGURE 23.17 ■ An abdominal wall (ventral or incisional) hernia and an inguinal hernia

Inguinal hernia

Inguinal hernias usually affect males and are classified as indirect or direct inguinal hernias. *Indirect inguinal hernias* are caused by improper closure of the tract developing as the testes descend into the scrotum before birth. A sac comprising abdominal contents protrudes through the internal inguinal ring into the inguinal canal. It often descends into the scrotum. Although indirect inguinal hernias are congenital defects, these often are not evident until adulthood, when increased intra-abdominal pressure and dilation of the inguinal ring allow abdominal contents to enter the channel.

Direct inguinal hernias are acquired defects resulting from weakness of the posterior inguinal wall. Direct inguinal hernias usually affect older adults. *Femoral hernias* are also acquired defects in which a peritoneal sac protrudes through the femoral ring. These hernias usually affect obese or pregnant women.

Inguinal hernias may produce no manifestations and are discovered during routine physical examination. These may cause a lump, swelling or bulge in the groin, particularly during lifting or straining. An inguinal hernia may cause sharp pain or a dull ache radiating into the scrotum. A palpable mass may be present in the groin, although it may be felt only with increased intra-abdominal pressure (as occurs during coughing) and invagination of the scrotum towards the inguinal ring.

Umbilical hernia

Pregnancy and obesity contribute to the development of umbilical hernias in adults. *Umbilical hernias* may be congenital and evident during infancy, or acquired as the tissue closing the umbilical ring weakens, allowing protrusion of abdominal contents. These hernias are more common in women. Other predisposing factors include multiple pregnancies with prolonged labour, ascites and large intra-abdominal tumours.

Umbilical hernias tend to enlarge steadily and contain omentum, although these may also contain small or large bowel. These hernias may cause sharp pain on coughing or straining, or a dull, aching sensation. Strangulation is a common complication of umbilical hernias.

Incisional or ventral hernia

Incisional or *ventral hernias* occur at a previous surgical incision or following abdominal muscle tears. Inadequate healing of the incision or tear leads to hernia development. Contributing factors include poor wound closure, postoperative infection, age or debility, obesity, inadequate nutrition and excess incisional stress caused by vigorous coughing.

Ventral hernias are characterised by a bulge at the incisional site, often noted when the person pulls to a sitting position from a lying position. Ventral hernias often are asymptomatic and the risk of incarceration is low because of the size of the defect.

Manifestations

Abdominal contents (peritoneum, bowel and other abdominal organs) can protrude through the abdominal wall forming a sac covered by skin and subcutaneous tissues. In most cases,

abdominal contents move into the sac when intra-abdominal pressure increases, then return to the abdominal cavity when pressure returns to normal or when manual pressure is placed on the bulging sac. This is known as a *reducible hernia*.

Complications

The risk of complications is low with a reducible hernia. If the contents of a hernia cannot be returned to the abdominal cavity, it is said to be *incarcerated*. Contents of an incarcerated hernia are trapped, usually by a narrow neck or opening to the hernia. Incarceration increases the risk of complications, including obstruction and strangulation. Obstruction occurs when the lumen of the bowel contained within the hernia becomes occluded, much like crimping of a hose. A *strangulated hernia* develops when blood supply to bowel and other tissues in the hernia sac is compromised, leading to necrosis. The affected bowel infarcts, leading to perforation and contamination of the peritoneal cavity. Clinical manifestations of a strangulated hernia include severe abdominal pain and distension, nausea, vomiting, tachycardia and fever.

INTERPROFESSIONAL CARE

The diagnosis of a hernia is made by a physical examination. The person is examined in a supine or standing position. A bulge may be seen or felt when the person coughs or bears down. No laboratory or diagnostic testing is usually required, unless bowel obstruction or strangulation is suspected.

Surgical repair, or *herniorrhaphy*, is the usual treatment of hernia. Surgery is generally well tolerated by people of all ages and carries a much lower risk than the complications of incarceration, obstruction and strangulation. Emergency surgery is indicated for hernias that are incarcerated, painful or tender. In a herniorrhaphy, the abdominal wall defect is closed by suturing or with wire or mesh over the defect. If incarceration occurs or strangulation is suspected, the abdominal cavity is explored during surgery and any infarcted bowel resected. Heavy lifting and heavy manual labour are restricted for approximately 3 weeks after surgery.

When surgery is contraindicated, a person can be taught to reduce their hernia by lying down and gently pushing against the mass. However, the person needs to be instructed if suspected hernia incarceration is suspected, not to attempt to reduce their hernia and to seek medical assistance. A binder or truss may be worn to prevent or control the protrusion.

Nursing care

Assessment

- *Health history:* manifestations of hernia, such as bulging in the groin or of the abdominal wall when coughing, straining or moving from lying to standing; pain (abdominal, groin or scrotal); history of hernia or abdominal surgery.
- *Physical examination:* observe for bulging of the abdominal wall or around the umbilicus when raising head and shoulders from supine position; wearing gloves, palpate inguinal region for bulges when the person coughs or bears down (Valsalva manoeuvre) while standing.

Nursing diagnoses and interventions

Herniorrhaphy is generally an uncomplicated procedure, usually performed as day surgery. Preoperative assessment and teaching and immediate postoperative care are the primary nursing care needs. Care is similar to that provided for a person following an appendectomy.

Risk of decreased gastrointestinal tissue perfusion

When providing care for a person with a known hernia, the possibility of obstruction and strangulation must be considered throughout nursing assessments. Although nursing interventions may not be able to prevent these complications, rapid identification of the problem allows timely surgical treatment. Prompt treatment may prevent major complications related to infection and peritoneal contamination by bowel contents.

- Assess bowel sounds and abdominal distension at least every 8 hours. A change in bowel sounds—either cessation of sounds or an onset of hyperactive, high-pitched sounds—may indicate obstruction. *With obstruction, abdominal girth may increase.*

CONSIDERATION FOR PRACTICE

Promptly report any acute increase in abdominal, groin, perineal or scrotal pain. An abrupt increase in the intensity of pain may indicate bowel ischaemia due to strangulation.

- Notify medical staff if the hernia becomes painful or tender. *Pain and tenderness indicates incarceration and increased risk of strangulation.*
- If signs of possible obstruction or strangulation occur, notify the doctor. Place the person in a supine position with the hips elevated and knees slightly bent. Withhold all food and fluids (NBM) and begin preparations for surgery. *This position helps relax abdominal muscles and may facilitate reduction of the hernia. Strangulation or obstruction requires immediate surgical intervention.*

Community-based care

Include the following topics when teaching a person about hernias and home care:

- rationale for examining the groin and abdomen for bulges
- the nature of hernia, risk factors and manifestations
- surgical intervention for hernia
- how to reduce a hernia if necessary
- the importance of seeking immediate medical intervention for signs of strangulation or obstruction
- the need to notify the doctor if upper respiratory infection and cough develop preoperatively (forceful coughing is not recommended postoperatively)
- postoperative pain management and activity restrictions.

THE PERSON WITH INTESTINAL OBSTRUCTION

Intestinal obstruction is failure of intestinal contents to move through the bowel lumen. Intestinal obstructions affect either the large or small bowel. The small intestine is more commonly affected; however, bowel obstructions may also occur in the large intestine. Obstruction is the most common reason for small-bowel surgery.

Pathophysiology

Intestinal obstructions are either mechanical or functional in nature. *Mechanical* obstructions may be caused by: (1) problems outside the intestine, such as bands of scar tissue or hernias; (2) problems within the intestine—for example, tumours or inflammatory bowel disease; or (3) obstruction of the intestinal lumen. Intestinal obstructions are partial or complete. *Functional* obstruction occurs when peristalsis fails to propel intestinal contents although there is no mechanical obstruction. *Adynamic ileus* (also known as **paralytic ileus** or simply *ileus*) is the most common functional obstruction after abdominal surgery and probably accounts for most intestinal obstructions (Porth & Matfin, 2013). Obstructions are further classified by the portion of intestine affected.

When the intestine is obstructed, gas and fluid accumulate proximal to and within the obstructed segment, distending the bowel. Swallowed air accounts for most of the gas. Ingested fluid, saliva, gastric juice and pancreatic secretions contribute to accumulated fluid. Water and sodium are drawn into the bowel lumen, contributing to fluid accumulation, distension and vascular fluid losses. Distension of the bowel lumen interferes with peristaltic movement, leading to atony and further distension. Significant distension of the bowel lumen comprises blood flow to mucosa, and eventually leads to necrosis. Gangrenous bowel may perforate resulting in peritonitis. Rapid bacterial growth in the obstructed bowel leads to sepsis and often death.

Significant bowel distension, vomiting and third spacing of fluids in the bowel and peritoneal cavity leads to massive loss of fluids and electrolytes with resulting hypovolaemia, hypokalaemia, renal insufficiency and shock.

Small-bowel obstruction

Adhesions or bands of scar tissue and hernias account for most mechanical small-bowel obstructions. In adults, adhesions develop following abdominal surgery or inflammatory processes. Adhesions usually produce a *simple obstruction* or single blockage in one portion of the intestine (see Figure 23.18A). The obstruction produced by an incarcerated hernia is a *closed-loop obstruction*, with two different portions of the bowel lumen obstructed (see Figure 23.18B).

Tumours, either intrinsic (of the bowel itself) or extrinsic (of another organ but affecting the bowel because of their size), can progressively occlude the bowel lumen and eventually obstruct it (see Figure 23.18C). Other, less common causes of bowel obstruction include intussusception (rare in adults) (see Figure 23.18D); volvulus, the rotation of loops of bowel about a fixed point (see Figure 23.18E); foreign bodies; stricture; and inflammatory bowel disease.

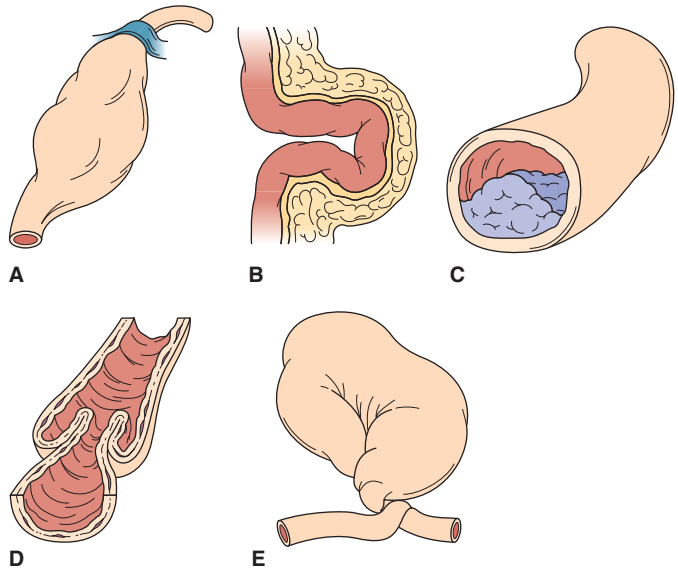


FIGURE 23.18 ■ Selected causes of mechanical obstruction. *A*, Adhesions; *B*, incarcerated hernia; *C*, tumour; *D*, intussusception; *E*, volvulus

Both volvulus and an incarcerated hernia can cause a *strangulated obstruction*. In a strangulated obstruction, not only is the lumen of the bowel obstructed, but the blood supply to the affected portion is also compromised.

In a functional obstruction or adynamic ileus, peristalsis stops due to either neurogenic or muscular impairment. The bowel lumen remains patent; however, the contents are not propelled forward. Temporary ileus commonly follows gastrointestinal surgery. It may also result from tissue anoxia or peritoneal irritation due to haemorrhage, peritonitis or perforation of an organ. Other conditions precipitating paralytic ileus include renal colic, spinal cord injuries, uraemia and electrolyte imbalances, particularly hypokalaemia. Additionally, the effects of some narcotics, anticholinergic drugs and antidiarrhoeal medications such as diphenoxylate can produce a functional obstruction.

MANIFESTATIONS The manifestations of a small-bowel obstruction vary, depending on the level of obstruction and how rapidly it develops. Cramping or colic-like abdominal pain may be intermittent or increasing in intensity. Vomiting is common, particularly in high or proximal obstructions, because lumen distension stimulates the vomiting centre. As bacterial fermentation occurs, vomitus often contains faecal matter, particularly with a low or distal obstruction. Flatus and faeces already present in the lower bowel may be expelled early in the obstructive process. However, this expulsion ceases as the obstruction continues.

Early in the course of a mechanical obstruction, borborygmi and high-pitched tinkling bowel sounds are present, as

the intestine attempts to propel contents past the obstruction. Visible peristaltic waves may be noted in the distended loops of bowel in thin people. In the later stages, the bowel becomes silent. With a paralytic ileus, bowel sounds are greatly diminished or absent throughout the process. Abdominal distension is minimal with proximal obstructions, but may be pronounced with distal obstruction and paralytic ileus. The abdomen may be tender to palpation as well.

In addition to abdominal and gastrointestinal manifestations, signs of fluid and electrolyte imbalance develop. Hypovolaemia can develop rapidly as extracellular fluid is sequestered in the bowel and vomiting occurs. Although early vital signs may be normal, changes are noted as dehydration and hypovolaemia develop. The person becomes tachycardic and tachypnoeic and blood pressure falls. Temperature may be elevated. Urine output decreases and clinical manifestations of hypovolaemic shock may be seen. The clinical manifestations of mechanical small-bowel obstruction with their accompanying pathophysiological process are outlined in Table 23.13.

COMPLICATIONS Hypovolaemia and hypovolaemic shock with multiple-organ dysfunction is a significant complication of bowel obstruction and can lead to death. Renal insufficiency from hypovolaemia leads to acute kidney injury or dysfunction. Pulmonary ventilation may be impaired because abdominal distension elevates the diaphragm, impeding respiratory processes.

Strangulation associated with incarcerated hernia or volvulus impairs the blood supply to the bowel. Gangrene may rapidly result, causing bleeding into the bowel lumen and peritoneal cavity and eventual perforation. With perforation, bacteria and toxins from the strangulated intestine enter the peritoneum and, potentially, the circulation, resulting in peritonitis and possible septic shock. Strangulation greatly increases the risk of mortality.

Large-bowel obstruction

Obstruction of the large intestine occurs much less frequently than small-bowel obstruction. Although any portion of the colon may be affected, obstruction usually occurs in the sigmoid segment. Bowel cancer is the most common cause; other causes include volvulus, diverticular disease, inflammatory disorders and faecal impaction.

MANIFESTATIONS Constipation and colic-like abdominal pain are usual manifestations of large-bowel obstruction. The pain is often deep and cramping; severe continuous pain signals bowel ischaemia and possible perforation. Vomiting is a late sign, if it occurs at all. The abdomen is distended, with high-pitched, tinkling bowel sounds with rushes and gurgles. On palpation, localised tenderness or a mass may be noted.

COMPLICATIONS If the ileocaecal valve between the small and large intestines is competent, distension proximal to the obstruction is limited to the colon itself. This is known as a *closed-loop obstruction*. It leads to massive colon dilation as the ileum continues to empty gas and fluid into the colon. Increasing pressure within the obstructed colon impairs circulation to the bowel wall. Gangrene and perforation are potential complications.

INTERPROFESSIONAL CARE

The management of a bowel obstruction focuses on relieving the pressure and obstruction and providing supportive care. The intestine is decompressed and fluid and electrolyte balance is restored. Surgery may be necessary to relieve a mechanical obstruction or if strangulation is suspected.

TABLE 23.13 Clinical manifestations and pathophysiological processes of mechanical small-bowel obstruction

MANIFESTATION	PATHOPHYSIOLOGY
Abdominal pain: intermittent mid-abdominal, colicky; intensity may initially decrease, then become severe and steady	Peristaltic waves attempt to propel bowel contents past the obstruction. As the bowel becomes increasingly distended, peristalsis is inhibited and pain may decrease in intensity. If unrelieved, distension of bowel lumen impairs mucosal blood supply, leading to ischaemia and necrosis. Bowel infarction or perforation may occur, leading to chemical and bacterial peritonitis.
Bowel sounds: initially loud, possibly high pitched; may correspond with waves of abdominal pain; later infrequent or absent	Initial distension of the bowel proximal to the obstruction stimulates peristalsis as the bowel attempts to propel contents past the obstruction. With further distension and resulting electrolyte imbalances, peristalsis is inhibited and bowel sounds become less frequent to inaudible.
Vomiting	Distension of the bowel stimulates the vomiting centre of the brain, which, in turn, stimulates the vomiting reflex.
Abdominal distension	Fluid (saliva, gastric juice, bile, pancreatic secretions) and air are trapped in the bowel proximal to the obstruction.
Hypovolaemia, electrolyte imbalance	Normal movement of water and sodium from the bowel lumen to the interstitial and intravascular spaces is initially inhibited. Fluids and electrolytes are lost through vomiting. With continued obstruction and bowel distension, sodium and water move from the vascular system into the bowel lumen, further distending it. Intestinal venous return is inhibited, leading to tissue oedema and accumulation of fluid and electrolytes within the peritoneal cavity.

Diagnosis

Radiological studies (x-rays and CT scan) are used to confirm the diagnosis of bowel obstruction. Laboratory testing is used to evaluate for the presence of infection and fluid and electrolyte imbalances.

An abdominal x-ray often shows distended loops of intestine with fluid and gas in a small-bowel obstruction. Free air under the diaphragm indicates a perforation. X-ray or CT scan with contrast media may be required to confirm a mechanical obstruction and assess the completeness of the obstruction. Meglumine diatrizoate (Gastrografin) is often used to provide contrast, rather than barium, when a bowel obstruction is suspected. Barium enema may be used to confirm the diagnosis of large-bowel obstruction and determine its location, unless perforation is suspected.

Laboratory tests include WBC, serum amylase, serum osmolality, electrolytes and arterial blood gases. These tests will show the following results with a bowel obstruction:

- *WBC* often shows mild leucocytosis due to an inflammatory response to changes within the obstructed bowel lumen. With strangulation, leucocytosis is marked.
- *Serum amylase levels* may be elevated, particularly when strangulation is present.
- *Serum osmolality* and *electrolyte levels* are affected by fluid and electrolyte losses from vomiting and fluid sequestering in the bowel lumen. With hypovolaemia, the serum osmolality and urine specific gravity increase. Potassium and chloride are lost through vomiting, leading to hypokalaemia and hypochloraemia.
- *ABGs* may reveal metabolic alkalosis (pH > 7.45, bicarbonate > 26 mmol/L, PCO_2 > 45 mmHg) with small-bowel obstruction due to loss of hydrochloric acid from the stomach.

Gastrointestinal decompression

Most partial small-bowel obstructions are successfully treated with gastrointestinal decompression using a nasogastric or long intestinal tube. Functional obstructions respond to treatment with bowel rest and intestinal decompression as well. Intestinal tubes (see Figure 23.5) may be inserted through the nares or via gastrostomy. A balloon or weighted tip draws the tube from the stomach into the intestine and to the area of obstruction. Collected fluid and gas are removed using low suction until peristalsis resumes or the obstruction is relieved. Sayakkara (2012) outlined the current clinical guidelines for low-pressure gastric suctioning, including regularly checking suction reaches the specified pressure level—gastric contents move back down the tube towards the person when the suction is off and rise towards the container when the suction is on. If the fluid fails to move, the Salem sump may be blocked or the suction container is positioned too low or high relative to the height of the person's stomach. In a few hours a person can lose more than 1500 mL containing electrolytes and hydrogen ions when low-pressure suction is used (Sayakkara, 2012). It is important to closely assess the person's fluid, electrolyte and acid–base balance.

Surgery

Surgical intervention is required for complete mechanical obstructions as well as for strangulated or incarcerated obstructions of the small intestine. A person with incomplete mechanical obstruction may also require surgery if the obstruction persists.

Prior to surgery, a nasogastric tube is inserted to relieve vomiting and abdominal distension and to prevent aspiration of intestinal contents. Fluid and electrolyte balance must be restored before surgery. Isotonic intravenous fluids, such as normal (physiological) saline, Hartmann's solution or other balanced electrolyte solutions are used. Additional electrolytes may be added to the solution to correct low levels. It is particularly important to correct hypokalaemia prior to surgery. Acid–base imbalances are also addressed, often using intravenous acidifiers or alkalinising agents. If strangulation has occurred, the person may require plasma or blood replacement. Intravenous broad-spectrum antibiotics are administered prophylactically (see the section on peritonitis).

A laparotomy usually is performed to allow inspection of the small intestine and removal of infarcted or gangrenous tissue. If the obstruction was caused by adhesions, these are removed or lysed. Obstructing tumours are resected and foreign bodies are removed. Any bowel appearing gangrenous is resected, usually followed by an end-to-end anastomosis of remaining intestine. If a large tumour mass or dense adhesions are found, the area of obstruction may be bypassed by anastomosis of proximal small bowel to small or large intestine distal to the obstruction. See the box 'Nursing care of the person requiring bowel surgery'.

Obstructions of the large intestine usually necessitate surgery. The primary goal is to relieve colonic distension and prevent perforation. The secondary goal is to remove the obstructing lesion. In some cases, colonoscopy may be used to relieve the distension. If the person's condition prohibits major surgery or the obstructing tumour is advanced, laser photocoagulation may be used to enlarge the bowel lumen. Removal of the obstructing lesion is the preferred treatment. The proximal and distal bowel segments may be anastomosed or a permanent colostomy or ileostomy may be required.

Nursing care

Health promotion

Advise the person, particularly older adults, regarding health promotion activities, such as increasing dietary fibre intake, maintaining a generous fluid intake and exercising daily to help prevent constipation and possible large-bowel obstruction. Stress the importance of maintaining dietary restrictions (such as avoiding popcorn) for a person experiencing repeated small-bowel obstructions.

Assessment

Nurses may be instrumental in the early identification of intestinal obstructions in older adults, the homebound or the institutionalised individual. Early identification and intervention significantly reduce morbidity from bowel obstruction.

- *Health history:* complaints of abdominal pain and bloating, constipation; previous history of bowel obstruction or risk factors such as hernia, inflammatory bowel disease, diverticulosis or previous abdominal surgery; current medications.
- *Physical examination:* vital signs including orthostatic blood pressure, temperature; skin colour, temperature, texture and turgor; colour and moisture of mucous membranes; abdominal shape, contour, bowel sounds, presence of tenderness or masses on palpation.

Nursing diagnoses and interventions

In a person with suspected or confirmed bowel obstruction, frequent assessment for complications such as fluid and electrolyte imbalance, acid–base imbalances, hypovolaemic shock, perforation and peritonitis is necessary.

Deficient fluid volume

Because of the large collection of fluid in the bowel proximal to an obstruction, and the accompanying vomiting and nasogastric suction, a person with an intestinal obstruction often has a fluid volume deficit. If not corrected promptly, hypovolaemic shock, acute kidney injury and multiple-organ system dysfunction from poor tissue perfusion may result.

- Monitor vital signs, peripheral perfusion (skin temperature, peripheral pulses and capillary refill); pulmonary artery pressures, cardiac output (CO) (if Swan–Ganz catheter in situ) and central venous pressure (CVP) hourly (if able). *A decrease in blood pressure, tachycardia, tachypnoea and a decrease in peripheral perfusion may indicate hypovolaemia. Although invasive, haemodynamic parameters such as pulmonary artery pressures, CO and CVP allow accurate assessment of fluid volume status.*
- Measure urinary output hourly and nasogastric drainage every 2 to 4 hours. *A urinary output of 0.5 mL/kg/hr or more usually indicates an adequate glomerular filtration rate (GFR). Nasogastric output provides a tool for evaluating fluid replacement needs.*

CONSIDERATION FOR PRACTICE

Report promptly urine output of less than 0.5 mL/kg/hr for 2 consecutive hours. This often indicates hypovolaemia and an increased risk of shock and acute kidney injury or dysfunction.

- Maintain intravenous fluids and blood volume expanders as ordered. *The amount of fluid administered is calculated to meet ongoing fluid needs and replace previous and current losses. Restoration and maintenance of blood volume are necessary to maintain cardiac output and tissue and organ perfusion.*
- Measure abdominal girth every 4 to 8 hours. Mark the level of measurement on the abdomen. *A reference mark allows consistent, accurate measurements. An increase in abdominal girth indicates increasing intestinal distension.*
- Notify the doctor of changes in status. *Changes in vital signs, pain and signs of increasing distension can indicate the need for immediate surgical intervention.*

Ineffective tissue perfusion: gastrointestinal

Perfusion of the intestinal wall and mucosa may be impaired by the obstructive process itself (e.g. strangulation or volvulus) or by significant intestinal distension. The goal is to maintain tissue perfusion and promote normal peristalsis and bowel elimination.

- Monitor vital signs hourly. Assess peripheral pulses, skin colour, temperature and capillary refill. *Cardiovascular assessment is vital to detect early signs of hypovolaemic shock resulting from sequestering large volumes of fluid in the intestines. Hypovolaemia and shock can convert mild bowel ischaemia to infarction as the blood supply to the tissue falls.*
- Monitor urine output hourly. Report output of less than 0.5 mL/kg/hr. *Urine output is a good indicator of the GFR and tissue perfusion. The urine output often falls before vital sign changes are apparent in hypovolaemia.*
- Monitor temperature at least every 4 hours. *An elevated temperature may be an early indication of sepsis from bowel perforation as a result of gangrene.*
- Frequently assess pain. *A change in the character of pain or a rapid increase in its intensity may signal bowel infarction or perforation.*
- Maintain NBM status until peristalsis resumes. *Enteral food or fluids may increase distension and bowel ischaemia. These also are restricted until the possibility of perforation is eliminated.*

Ineffective breathing pattern

Significant abdominal distension from a bowel obstruction can cause the diaphragm to flatten, impairing pulmonary ventilation. Following surgery, splinting of abdominal muscles to avoid pain can lead to shallow respirations. These factors, plus the risk of aspiration of gastrointestinal contents during vomiting, place a person at high risk of respiratory complications, particularly with a small-bowel obstruction.

- Assess respiratory rate, pattern, pulse oximetry and lung sounds at least every 2 to 4 hours. *Tachypnoea, shortness of breath or apparent dyspnoea are early signs of respiratory compromise. Diminished breath sounds, particularly in the bases of the lungs, or crackles indicate poor lung expansion and possible impaired ventilation.*
- Monitor ABG results for possible effects of altered respiratory status. *Tachypnoea may lead to respiratory alkalosis as excess carbon dioxide is eliminated. Conversely, impaired chest expansion can lead to respiratory acidosis because of alveolar hypoventilation.*
- Elevate the head of the bed. *Elevating the head of the bed reduces the work of breathing and improves alveolar ventilation by reducing the pressure of abdominal distension on the diaphragm.*
- Provide a pillow or folded towel to use in splinting the abdomen while coughing postoperatively. *Splinting abdominal muscles and incisions improves the ease and effectiveness of coughing postoperatively.*
- Maintain nasogastric or intestinal tube patency. *Maintaining gastrointestinal suction helps reduce*

abdominal distension and prevent aspiration associated with vomiting.

- Encourage use of incentive spirometer or other assistive device hourly. *These devices encourage deep breathing, opening distal airways and preventing atelectasis.*
- Contact physiotherapist as indicated. *The physiotherapist may suggest or perform additional measures to maintain effective pulmonary ventilation.*
- Provide good oral care 2 to 4 hourly. *Dehydration and nasogastric suction dry the mucous membranes of the mouth and throat, increasing the risk of bacterial growth. Many respiratory infections result from aspirated organisms.*

Community-based care

Include the following topics when teaching a person with intestinal obstruction in preparation for home care:

- wound care
- activity level, return to work and any other recommended restrictions
- recommended follow-up care
- care of temporary colostomy (if appropriate) and planned reanastomosis
- recurrent obstructions, their cause, early identification of manifestations and possible preventive measures.

THE PERSON WITH DIVERTICULAR DISEASE

Diverticula are small (0.5 to 1.0 cm) outpouchings of the colon occurring in rows (see Figure 23.19). Diverticula may occur anywhere in the intestinal tract, excluding the rectum. The vast majority, however, affect the large intestine, with 85–95% occurring in the sigmoid colon (Fauci et al., 2008; McPhee et al., 2008).

FAST FACTS

Diverticular disease

- People in Australia, the United Kingdom, France and the United States have high and increasing incidence rates of diverticular disease. The incidence of diverticula increases with age, with 5–10% of the population older than 45 years of age and almost 80% of those older than 85 years of age experiencing it.
- Most people diagnosed with diverticular disease remain asymptomatic.
- Men and women are equally affected.

Cultural factors—diet, in particular—are thought to play an important role in the development of diverticula. A diet consisting of highly refined and fibre-deficient foods is believed to be a major contributor to the disease. Decreased activity levels and delaying defecation have also been suggested as contributing factors. The increasing incidence of diverticula with ageing suggests dietary factors (lack of fibre), a decrease in physical activity, poor bowel habits (neglecting the urge to defecate) and the effects of ageing contribute to development of the disease (Marieb & Hoehn, 2013).

Pathophysiology

Diverticula form when increased pressure within the bowel lumen causes bowel mucosa to herniate through defects in the colon wall. The circular and longitudinal muscles often thicken or hypertrophy in the area affected by diverticula. This narrows the bowel lumen, increasing intraluminal pressure. Deficient dietary fibre and a lack of faecal bulk contribute to muscle hypertrophy and narrowing of the bowel. Contraction of the muscles in response to normal stimuli such as meals may occlude the narrowed lumen, further increasing intraluminal pressure. The high pressure causes mucosa to herniate through the muscle wall, forming a diverticulum. Areas where

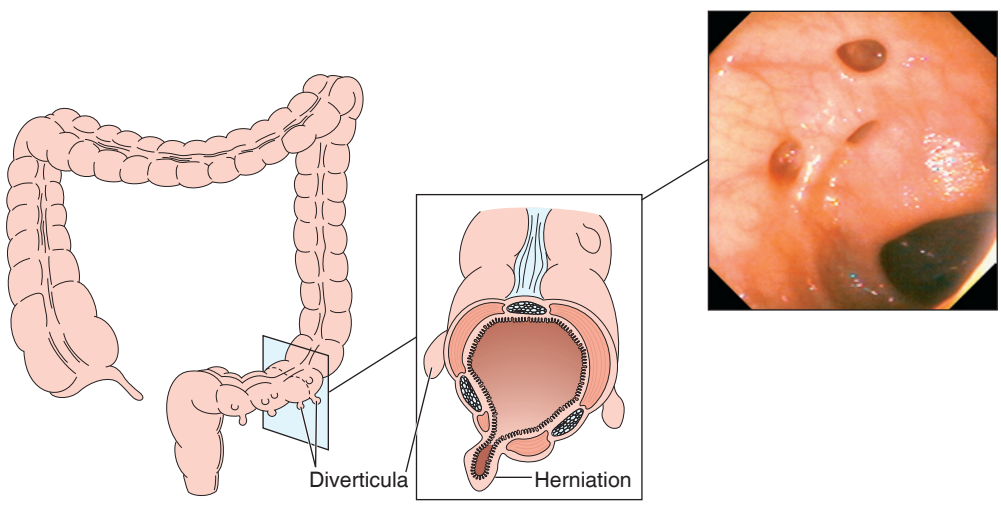


FIGURE 23.19 ■ Diverticula of the colon

Source: Image © David M. Martin MD/Science Source.

nutrient blood vessels penetrate the circular muscle layer are the most common sites for diverticula formation.

Diverticulosis

Diverticulosis indicates the presence of diverticula. More than two-thirds of people with diverticulosis are asymptomatic. When manifestations such as episodic pain (usually left-sided), constipation and diarrhoea occur, these often are attributed to irritable bowel syndrome, commonly accompanying diverticular disease. As the disease progresses, abdominal cramping, narrow stools (decrease in calibre), increased constipation, bleeding in the stools, weakness and fatigue may develop.

Complications of diverticulosis include haemorrhage and diverticulitis. A diverticulum may bleed, whether it is inflamed or not, possibly due to erosion of an adjacent blood vessel by a faecalith (hard mass) in the diverticulum.

Diverticulitis

Diverticulitis is inflammation in and around the diverticular sac. It typically affects only one diverticulum, usually in the sigmoid colon. Undigested food and bacteria collect in the diverticula, forming a hard mass impairing the mucosal blood supply, allowing bacterial invasion. Mucosal ischaemia leads to perforation. With microscopic perforation, inflammation is localised. Gross perforation of a diverticulum results in more extensive bacterial contamination, leading to abscess formation or peritonitis.

MANIFESTATIONS Pain is a common clinical manifestation of diverticulitis. It is usually left-sided and may be mild to severe and either steady or cramping. The person may also experience either constipation or increased frequency of defecation. Depending on the location and severity of the inflammation, nausea, vomiting and a low-grade fever may occur. On examination, the abdomen may be distended, with tenderness and a palpable mass in the left lower quadrant resulting from the inflammatory response.

Older adults may have less specific manifestations, complaining of vague abdominal pain. A palpable mass and signs of a large-bowel obstruction may be present.

COMPLICATIONS Complications associated with diverticulitis (in addition to peritonitis and abscess formation) include bowel obstruction, fistula formation and haemorrhage. Severe or repeated episodes of diverticulitis may lead to scarring and fibrosis of the bowel wall, further narrowing the bowel lumen. This increases the risk of obstruction of the large bowel. Acutely inflamed tissue adheres to the small bowel, increasing the potential for small-bowel obstruction as well. Fistulas may form, usually between the sigmoid colon and the bladder. Urinary tract infection is the usual sign of a colovesical fistula. Fistulas may also perforate into the small intestine, ureter, vagina, perineum or abdominal wall. Bleeding from perforation of a vessel wall can occur with diverticulitis. Although it may be significant, bleeding usually stops spontaneously.

INTERPROFESSIONAL CARE

Management of diverticular disease varies from no prescribed treatment to surgical resection of affected colon, depending on the severity of the disease and its complications.

Diagnosis

Diagnostic testing is used to identify diverticular disease when the disease is symptomatic or complications develop. In addition to illustrating diverticula, a barium enema and x-rays can reveal segmental spasm and muscular thickening with a narrowed bowel lumen. Flexible sigmoidoscopy or colonoscopy may be done to detect diverticulosis, assess for strictures or bleeding, and rule out tumour as the cause of the person's manifestations. Abdominal x-ray films may show free abdominal air associated with diverticulitis and perforation. CT scan may be done with or without contrast media to assess inflammation and detect an abscess or fistula.

Laboratory tests include haemoccult or guaiac testing of stool to identify the presence of occult blood, and a WBC count may show leucocytosis with a left shift (an increased number of immature WBCs) due to inflammation in diverticulitis.

Medications

Systemic broad-spectrum antibiotics effective against usual bowel flora are prescribed to treat acute diverticulitis. Oral antibiotics such as metronidazole (Flagyl) and ciprofloxacin (Cipro) or trimethoprim-sulfamethoxazole (Septra, Bactrim) may be prescribed if manifestations are mild. Rifixamin (Xifaxan) is a poorly absorbed antibiotic that may be used together with fibre to treat unmanaged diverticular disease. Severe, acute attacks often require hospitalisation and treatment with intravenous fluids and antibiotics effective against anaerobic and gram-negative bacteria. Therapy may include a second-generation cephalosporin such as cefoxitin (Mefoxin) or another antibiotic such as piperacillin-tazobactam (Tazocin) or ticarcillin-clavulanate (Timentin). Antibiotics and their nursing implications are discussed in Chapter 11.

Although a stool softener such as docusate sodium (Coloxyl) may be prescribed, it is important to note that laxatives (which can further increase intraluminal pressure in the colon) are avoided for a person with diverticular disease.

Nutrition

Dietary modification is central to the management of diverticular disease. Dietary changes appear to reduce the risk of complications of diverticulosis. A high-fibre diet is recommended; this increases stool bulk, decreases intraluminal pressures and may reduce spasm (see Table 23.14). Bran is a low-cost fibre supplement that can be added to cereal, soups, salads or other foods. Commercial bulk-forming products—for example, psyllium seed (Metamucil)—may be recommended. These products are discussed in the 'Medication administration' box—'Laxatives'—on pages 708–709. A person should be advised to avoid foods with small seeds (such as caraway seeds, figs or berries), which could obstruct diverticula.

Bowel rest is prescribed during an acute episode of diverticulitis. Initially the person may be NBM with intravenous fluids

TABLE 23.14 Foods recommended in a high-fibre, high-residue diet

FOOD GROUP	RECOMMENDED FOODS
Cereals and grains	Wheat or oat bran; cooked cereals, such as oatmeal; dry cereals, such as bran buds or flakes, cornflakes, shredded wheat; wholegrain breads or biscuits; brown rice; popcorn
Fruits	Unpeeled raw apples, peaches and pears; blackberries, raspberries, strawberries; oranges
Vegetables	Dried beans (navy, kidney, pinto), lima beans; broccoli; peas; corn; squash; raw vegetables, such as carrots, celery and tomatoes; potatoes (with skins)

and occasionally TPN. Feeding is resumed gradually. Initially, a clear liquid diet is prescribed, with gradual advancement to a soft, low-roughage diet (i.e. a diet low in insoluble fibre) with daily added psyllium seed to soften stool and increase its bulk. Among foods a person should avoid are wheat and corn bran, vegetable and fruit skins, nuts and dry beans. The high-fibre diet is resumed following full recovery.

Surgery

A person with acute diverticulitis may require surgery, usually to treat generalised peritonitis or an abscess failing to respond to medical treatment. Haemorrhage that recurs or cannot be controlled may also necessitate surgery. Elective surgery may be performed for recurrent episodes of diverticulitis or persistent diverticulitis with continuing pain, tenderness and a palpable mass.

The affected bowel segment is resected and if possible an anastomosis of the proximal and distal portions is performed. When an acute infection and diverticulitis are present, a two-stage Hartmann procedure is required. A temporary colostomy is created and anastomosis delayed until the inflammation has subsided. A second surgery is performed 2 to 3 months later to reconnect the bowel and close the temporary colostomy.

Nursing care

Health promotion

Advising a person about the benefits of a high-fibre diet is important primary prevention for diverticular disease. Nurses working with groups and individuals in the community should emphasise the importance of a high-fibre diet and its benefits in preventing diverticular disease and other disorders. In facilities such as residential settings, nurses can work with dietary staff and care providers to increase the amount of fibre in residents' diets, unless this is contraindicated by a pre-existing condition.

Assessment

Because most people with diverticular disease have few or no manifestations, nursing assessment focuses on manifestations of complications.

- *Health history:* abdominal pain or cramping, chronic constipation or irregular bowel habits; nausea and vomiting; history of diverticular disease or irritable bowel syndrome.
- *Physical examination:* bowel sounds, presence of abdominal tenderness of masses and location; stool for occult blood.

Nursing diagnoses and interventions

A person with acute diverticulitis is acutely ill and has multiple nursing care needs. Priority nursing diagnoses include *Potential complication: perforation*, *Acute pain* and *Anxiety* related to the possibility of a significant complication or possible surgery.

Potential complication: perforation

During an acute attack of diverticulitis, inflammation and mucosal ischaemia the person is at risk of perforation and peritonitis. In addition to maintaining bowel rest to reduce the risk of perforation, nurses monitor for manifestations of perforation and possible sepsis.

- Monitor vital signs including temperature at least every 4 hours. *Tachycardia and tachypnoea may be early indications of increased inflammation and resulting fluid shift. Fever greater than 38.3°C may indicate increased inflammation or spread of inflammation. Note, however, that little temperature elevation may occur in older adults. A change in behaviour or increasing lethargy may be subtle indications of infection in the older adult.*
- Assess abdomen every 4 to 8 hours or more often as indicated, including measuring abdominal girth, auscultating bowel sounds and palpating for tenderness. Report promptly significant changes to the doctor. *Increasing abdominal distension, a decrease or change in the quality of bowel sounds, and/or increasing tenderness or guarding may indicate spread of the infectious process or peritonitis.*
- Assess for evidence of lower intestinal bleeding by visual examination and guaiac testing of stools for occult blood. *Perforation of a diverticulum may produce either intestinal or intra-abdominal bleeding and require immediate treatment such as surgery.*
- Maintain intravenous fluids, TPN and accurate intake and output records. *During acute diverticulitis, oral intake is usually prohibited or restricted. Intravenous fluids are given to maintain fluid and electrolyte balance; TPN is used to maintain nutritional status, facilitating healing and recovery.*

Acute pain

Pain is a common clinical manifestation of acute diverticulitis. It results from inflammation of the bowel and oedema of affected tissues. If surgery is required, postoperative pain is managed with narcotic analgesics.

- Ask the person to rate pain using a 0 to 10 pain scale. Document the level of pain and note any changes in location or character of pain. *The perception and response to pain is individual and is affected by past experiences, culture, ethnic background and other factors. A change in the character or intensity of the pain indicates a complication such as perforation or abscess formation.*
- Administer prescribed analgesic or maintain PCA as ordered. Assess analgesic effectiveness. Avoid administering morphine. Provide adjunctive medications as ordered and encourage use of adjunctive techniques, such as relaxation, positioning and distraction. Notify the doctor if pain management is inadequate. *If a person has not obtained adequate pain relief, further assessment and intervention are required.*
- Maintain bowel rest and total body rest (bed rest with limited activity). *Rest helps reduce inflammation and promotes healing, increasing comfort.*
- Reintroduce oral foods and fluids slowly, providing a soft, low-fibre diet with bulk-forming agents. *This allows continued healing of the affected bowel while promoting soft, easily expelled stools.*

Anxiety

A person with acute diverticulitis faces not only hospitalisation but also potential serious complications such as peritonitis and haemorrhage. Surgery and formation of a temporary colostomy may be necessary. Furthermore, episodes of acute diverticulitis are often recurrent and the person may fear future problems.

- Demonstrate empathy and awareness of the perceived health threat. *It is important to recognise and respect the person's feelings and perceptions as reality.*
- Assess level of understanding about disease and condition. *This allows misperceptions contributing to anxiety to be corrected.*
- Assess and document level of anxiety. *Severe anxiety or panic states interfere with the ability to respond to instructions and assist with care. Low to moderate anxiety levels enhance learning and compliance with prescribed interventions.*
- Assist the person to identify and use appropriate coping mechanisms. *Coping mechanisms provide immediate relief of anxiety while the person adapts to the situation.*
- Attend to physical care needs. *This provides reassurance these needs will be met and relieves concerns about them.*
- Spend as much time as possible with the person. *Presence of caring nurses helps relieve fears of abandonment or that help will not be available if needed. It also enhances trust and provides opportunity for expression of fears or concerns.*
- Encourage family and friends to remain with the person as much as possible. *This provides a supportive environment for the person and also distracts from physical concerns.*
- Involve the person and their family (as appropriate) in care decisions. *This increases a person's sense of control over the situation.*

Community-based care

A person with diverticular disease is responsible for self-care. Discuss the following topics for home care:

- prescribed high-fibre diet and the need to maintain the diet for life to reduce the incidence of complications, including ways to increase dietary fibre
- complications and their manifestations of diverticular disease
- provide a referral to a nutritionist for teaching as indicated.

Prior to discharge of a person with acute diverticulitis, discuss the following:

- food and fluid limitations, including recommendations for a low-residue diet during the initial period of healing
- colostomy management (if a temporary colostomy has been created), including where to obtain supplies and dietary management
- planned procedure to reanastomose the colon and revise the colostomy. Refer to community healthcare organisations as indicated.

ANORECTAL DISORDERS

Anorectal lesions include haemorrhoids, anal fissure, anorectal abscess, anorectal fistula and pilonidal disease.

THE PERSON WITH HAEMORRHOIDS

The anus and anal canal contain two superficial venous plexuses with the haemorrhoidal veins. When pressure on these veins is increased or venous return impeded, they can develop varices or varicosities, becoming weak and distended. This condition is commonly known as **haemorrhoids** or 'piles'. When asymptomatic, haemorrhoids are considered to be a normal condition found in all adults.

Pathophysiology and manifestations

Haemorrhoids develop when venous return from the anal canal is impaired. Straining to defecate increases venous pressure and is the most common cause of distended haemorrhoids. Pregnancy increases intra-abdominal pressure, raising venous pressure, and is another cause of haemorrhoids. Other factors that may contribute to symptomatic haemorrhoids include prolonged sitting, obesity, chronic constipation and a low-fibre diet.

Haemorrhoids are classed as either internal or external. *Internal haemorrhoids* affect the venous plexus above the

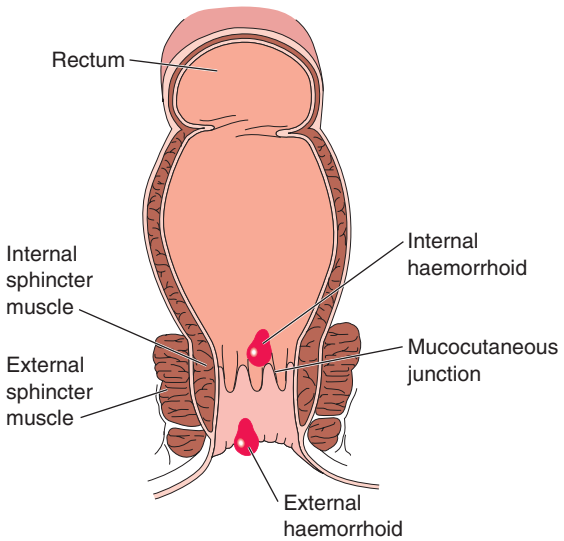


FIGURE 23.20 ■ The location of internal and external haemorrhoids

mucocutaneous junction of the anus (see Figure 23.20). Internal haemorrhoids rarely cause pain, usually presenting with bleeding. Bleeding from internal haemorrhoids is bright red and unmixed with the stool. It varies in quantity from streaks on toilet tissue to enough to colour the water in the toilet. Recurrent bleeding of internal haemorrhoids may be sufficient to cause anaemia. Mucus discharge and a feeling of incomplete evacuation of stool may also be manifestations of internal haemorrhoids.

External haemorrhoids affect the inferior haemorrhoidal plexus below the mucocutaneous junction. Bleeding is rare with external haemorrhoids. Anal irritation, a feeling of pressure and difficulty cleaning the anal region may be manifestations of external haemorrhoids.

As these enlarge, haemorrhoids may prolapse or protrude through the anus. Initially, prolapse occurs only with defecation and the haemorrhoids spontaneously regress back into the anal canal. Eventually, a person may need to manually replace internal haemorrhoids after defecation or these may become permanently prolapsed, in which case replacement is not possible. Manifestations of permanently prolapsed haemorrhoids include mucus discharge and clothing soilage.

'Normal' haemorrhoids are not painful. Prolapsed haemorrhoids may become strangulated as a result of congestion and oedema, leading to thrombosis. Haemorrhoidal thrombosis causes extreme pain and may lead to infarction of skin and mucosa overlying the haemorrhoid. Internal haemorrhoids associated with portal hypertension in liver disease may bleed profusely if ruptured. (See Chapter 24 for further discussion of portal hypertension.)

A *thrombosed external haemorrhoid* is a thrombosis of the subcutaneous external haemorrhoidal veins of the anal canal, rather than a true haemorrhoid. It appears as a painful bluish haematoma beneath the skin and typically occurs following a sudden increase in venous pressure—for example, heavy lifting, coughing or straining. Pain is significant at onset; however, it gradually subsides. Spontaneous rupture with bleeding may occur. Thrombosed external haemorrhoids resolve without intervention.

INTERPROFESSIONAL CARE

Because haemorrhoids are a normal condition, management is conservative unless complications such as permanent prolapse or thrombosis occur.

Diagnosis

Haemorrhoids are diagnosed by the person's history and by examination of the anorectal area. External haemorrhoids can be seen on visual inspection, especially if thrombosed. The person is asked to strain (Valsalva's manoeuvre) during the examination to detect prolapse. Internal haemorrhoids are usually not palpable or tender on digital examination of the rectum. *Anoscopic* examination is used to detect and evaluate internal haemorrhoids. For this exam, a speculum or endoscope is introduced into the anus to provide visual inspection of the tissues. Additional diagnostic examinations include testing of stool for occult blood and sigmoidoscopy, performed to rule out colon or rectal cancer, which may aggravate haemorrhoidal manifestations or produce similar manifestations. If liver disease with portal hypertension is suspected, liver function studies are ordered.

Medications

Bulk-forming laxatives such as psyllium seed (Metamucil) or stool softeners such as docusate sodium (Coloxyl) may be prescribed to relieve constipation as well as reducing discomfort. Suppositories and local ointments such as Preparation H or Nupercaine have an anaesthetic and astringent effect, reducing discomfort and irritation of surrounding tissues. These medications have little or no effect on the haemorrhoid itself. Warm sitz baths, bed rest and local astringent compresses may be recommended to reduce the swelling of oedematous prolapsed haemorrhoids after digital reduction.

Nutrition

Haemorrhoids that are not permanently prolapsed or acutely thrombosed generally are treated conservatively. A high-fibre diet and increased water intake to increase stool bulk, improve its softness and reduce straining are effective for most people with internal or external haemorrhoids.

Sclerotherapy

Haemorrhoids that are permanently prolapsed, are thrombosed or produce significant manifestations are treated more aggressively. *Sclerotherapy* involves injecting a chemical irritant into tissues surrounding the haemorrhoid to induce inflammation and eventual fibrosis and scarring. It is used to treat recurrent bleeding and early prolapse of internal haemorrhoids. The treatment produces minimal pain. Enlarged or prolapsing haemorrhoids may also be treated with rubber band ligation. A rubber band is placed snugly around the haemorrhoidal plexus

and surrounding mucosa, causing the tissue to necrose and slough within 7 to 10 days. Treatment is limited to one haemorrhoidal complex at a time, so repeat treatments may be necessary. Pain should be minimal if the band is placed appropriately; persistent pain following band ligation may signal an infection. Bleeding can occur as the haemorrhoid sloughs. Other procedures used to treat haemorrhoids include cryosurgery (in which haemorrhoids are necrosed by freezing with a cryoprobe), infrared photocoagulation or electrocoagulation.

Haemorrhoidectomy

A person with chronic manifestations, permanent prolapse, chronic bleeding with associated anaemia or painful thrombosed haemorrhoids may be treated surgically with a *haemorrhoidectomy*. In this procedure, haemorrhoids are surgically excised, leaving normal skin and surrounding tissues. This procedure may use conventional techniques or a laser to remove both internal and external haemorrhoids. Few complications are associated with haemorrhoidectomy.

Nursing care

Primary prevention of symptomatic haemorrhoids involves education of people of all ages. Stress the importance of maintaining an adequate intake of dietary fibre, a liberal fluid intake and regular exercise to maintain stool bulk, softness and regularity. Discuss the need to respond to the urge to defecate rather than postponing defecation. Teach appropriate constipation management, including the use of bulk-forming laxatives.

Most people with haemorrhoids are treated in community settings where the primary nursing focus is educational. Discuss the appropriate use of over-the-counter preparations and sitz baths for the relief of minor haemorrhoidal manifestations. If necessary, teach the person how to reduce prolapsed haemorrhoids digitally.

Advise the person about possible haemorrhoidal complications, such as chronic bleeding, prolapse and thrombosis. Stress the need to seek medical evaluation if manifestations persist. Discuss the link between manifestations of haemorrhoids and colorectal cancer and urge the person to seek medical intervention for persistent, unresolved or progressive manifestations.

When a haemorrhoidectomy is performed, a person requires more direct nursing intervention. Postoperative care of a person with perianal surgery is outlined in Box 23.4. Anal packing may be in place for the first 24 hours following the procedure. When removed, observe the person closely for bleeding. Pain is a common postoperative problem. Although the operative procedure is minor, postoperative discomfort can be significant because the anal region is richly innervated and muscle spasms may occur. In addition to systemic analgesics, sitz baths usually are ordered. These not only help promote relaxation and reduce discomfort but also clean the anal area. Use of a rubber ring or doughnut device minimises pressure on the surgical site while the person sits in the bath.

BOX 23.4 Perianal postoperative care

Assessment
- Monitor vital signs every 4 hours for 24 hours.
- Inspect rectal dressing every 2 to 3 hours for 24 hours.
- Monitor fluid balance—urinary output.

Pain control
- Assist to position of comfort, usually side lying.
- Provide analgesics as prescribed.
- Keep fresh ice packs over the rectal dressing as ordered.
- Assist with sitz bath water sprays three to four times per day.
- Provide a flotation pad for use when sitting.

Elimination
- Give stool softeners as prescribed.
- Give an analgesic before the first postoperative bowel movement if possible.
- When tolerated, encourage fluid intake of at least 2000 mL per day.

Individual and family teaching
- Take sitz bath or water spray after each bowel movement for 1 to 2 weeks after surgery.
- Drink at least 2000 mL of fluid per day.
- Eat adequate dietary fibre and exercise moderately.
- Take stool softeners as prescribead.
- Report to the doctor the following symptoms: rectal bleeding, continued pain on defecation, fever greater than 38.3°C, purulent rectal drainage.

A person may remain hospitalised until after the first postoperative bowel movement. Stool softeners, adequate fluids and analgesia before defecation aid in reducing anxiety and discomfort. Adequate cleaning following defecation, usually with a sitz bath, is vital.

Whether caring for a person with haemorrhoids or following a haemorrhoidectomy, consider the following nursing diagnoses:
- *Altered comfort* related to acute or chronic pain secondary to inflamed anal tissues.
- *Constipation* related to dietary habits and/or delay of defecation.
- *Risk of infection* related to disruption of anal tissue.

THE PERSON WITH AN ANORECTAL LESION

Unlike the rectum, which is relatively insensitive to pain, the anal canal is richly supplied with sensory nerves and is highly sensitive to painful stimuli. Lesions of the anorectal area may cause significant pain, particularly with defecation. Infection is a potential complication of anorectal lesions because of contamination by

faecal bacteria. The superior boundary of the anal canal (the anorectal juncture or pectinate line) contains 8 to 12 anal crypts where anorectal abscesses or fistulas can form. Lesions of the anorectal area include fissures, abscesses, fistulas and pilonidal disease.

Anal fissure

Anal fissures or ulcers occur when the epithelium of the anal canal over the internal sphincter becomes denuded or abraded. Irritating diarrhoeal stools and tightening of the anal canal with increased sphincter tension are frequent causes of anal fissures. Other factors contributing to their development include childbirth trauma, habitual purgative use, laceration by a foreign body and anal intercourse. Chronic inflammation and infection of surrounding tissues accompanies an anal fissure.

A person with an anal fissure typically has periods of exacerbation and remission. Because these occur below the mucocutaneous line, anal fissures are painful. Pain occurring with defecation is described as tearing, burning or cutting. Bright red bleeding is noted with a bowel movement. Bleeding is typically minor and noted on toilet paper. Because of fear of defecation, the person may develop constipation, further disrupting normal bowel habits and aggravating manifestations.

The diagnosis of anal fissure is made by gentle digital examination of the anal canal and anoscopy using a small anoscope. Treatment is usually conservative, involving dietary changes to increase fibre intake and stool bulk, increased fluid intake and use of bulk-forming laxatives. A topical agent such as hydrocortisone cream may be prescribed. Surgical intervention with an internal sphincterotomy, an incision into the internal sphincter to increase its diameter, is considered when the fissure does not heal with medical intervention.

Anorectal abscess

Invasion of the pararectal spaces by pathogenic bacteria can lead to an *anorectal abscess*. Commonly caused by infection extending from the anal crypt into a pararectal space, the abscess may appear small but often contains a large amount of pus. Multiple pathogens may be present, including *Escherichia coli*, *Proteus*, streptococci and staphylococci. Other factors contributing to the development of an anorectal abscess include infection of a hair follicle, sebaceous gland or sweat gland, and abrasions, fissures or anal trauma. The incidence of anorectal abscess is higher in men.

Pain is the primary manifestation of an anorectal abscess. Sitting or walking may aggravate the pain, but it is unrelated to defecation. External swelling, redness, heat and tenderness are apparent on examination. With a deeper abscess, swelling may not be visible, but the abscess is palpable on digital examination.

If the abscess either does not drain spontaneously or is not drained surgically, adjacent anatomical spaces are affected. Systemic sepsis is also a potential complication.

Incision and drainage is the treatment of choice for an anorectal abscess because it rarely resolves with antibiotic therapy alone. This treatment often leads to a persistent fistula, which is surgically closed after the infection has cleared.

Anorectal fistula

A fistula is a tunnel or tube-like tract with openings at each end. *Anorectal fistulas* have one opening in the anal canal, with the other usually found in perianal skin. Most occur spontaneously or as a result of anorectal abscess drainage. Crohn's disease is also a predisposing factor to fistula development.

The primary manifestation of an anorectal fistula is intermittent or constant drainage or purulent discharge. This may be accompanied by local itching, tenderness and pain associated with defecation.

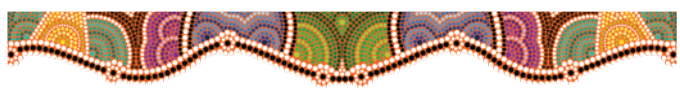
Anoscopic and digital examination with gentle probing of the fistula tract are used to establish the diagnosis. Although some fistulas may heal spontaneously, the treatment of choice is a fistulotomy. The primary opening of the fistula is removed and the tract is opened to allow it to heal by secondary intention, from the inside outward. If the sphincter is involved, a two-stage operation may be done to preserve the muscle and prevent faecal incontinence.

Pilonidal disease

A person with *pilonidal disease* has an acute abscess or chronic draining sinus in the sacrococcygeal area. Underlying the abscess or sinus is a cyst with granulation tissue, fibrosis and, often, hair tufts. This disease usually affects young hirsute (hairy) males and is probably due to hair entrapment in deep tissues of the sacrococcygeal area. Some researchers, however, believe it is a congenital disorder.

The lesion of pilonidal disease is generally asymptomatic unless it becomes acutely infected. Manifestations of acute inflammation accompany infection, including pain, tenderness, redness, heat and swelling of the affected area. Purulent discharge may be noted from one or more sinuses or openings in the midline.

The preferred treatment option for pilonidal disease is incision and drainage. The sinus tract and underlying cyst are excised and closed by either primary- or secondary-intention healing. The person may be instructed to remove hair from the area routinely by shaving or using a depilatory to prevent further hair entrapment and recurrence of the problem.



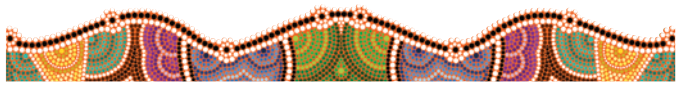
Nursing care

People with anorectal disorders are often treated in the community and the primary nursing responsibility is education. Highlight the importance of maintaining a high-fibre diet and liberal fluid intake to increase stool bulk and softness, thereby decreasing discomfort with defecation. Stress the importance of responding to the urge to defecate to prevent constipation.

Following surgical treatment of any of these disorders, inform the person to keep the perianal region clean and dry. If a dressing is in place, instruct to avoid soiling it with urine or faeces during elimination. Following removal of the dressing, teach to clean the area gently with soap and water following a bowel movement. Discuss the use of sitz baths for cleaning and comfort. Sitz baths are often recommended conservative management pre- and postoperatively for anorectal disorders. A systematic review by Slade (2014) found no strong evidence to support the use of the sitz bath for pain relief or to accelerate fissure or wound healing among adult people with anorectal disorders. However, people

with anorectal disorders were more satisfied when using interventions such as sitz baths and water sprays. Slade (2014) highlighted that a person may find the use of the water spray method more convenient and an appropriate alternative to the sitz bath. The use of sitz baths is a personal choice.

Suggest taking an analgesic if necessary prior to defecation. However, caution that some analgesics promote constipation. Teach clinical manifestations of infection or other possible complications to report to the doctor. If an antibiotic has been prescribed, provide written and verbal instructions about its use, its desired and possible adverse effects, and their management.



CHAPTER HIGHLIGHTS

- Disorders of intestinal motility include diarrhoea, constipation, irritable bowel syndrome and faecal incontinence. Diarrhoea is a manifestation of many other bowel disorders, including lactose intolerance, infections with bacteria, and inflammatory diseases of the bowel. Constipation may be a primary problem (especially for the older adult) or a manifestation of another disorder. Irritable bowel syndrome (IBS) is a functional disorder without any identifiable organic cause. Faecal incontinence is usually considered to be the manifestation of a disorder, rather than a disorder itself.
- Appendicitis is an acute inflammation of the vermiform appendix, manifested by abdominal pain that localises in the right lower quadrant of the abdomen. On palpation, localised and rebound tenderness is present at McBurney's point. It is treated most often with an appendectomy.
- Peritonitis (inflammation of the peritoneum from infection or chemical irritant) is a serious complication of a wide variety of acute abdominal disorders, including perforated ulcer, ruptured appendix, abdominal trauma or surgery, or necrotic bowel. Complications may be life threatening; without prompt and effective treatment, septicaemia and septic shock may occur.
- Gastroenteritis may result from bacterial or viral infections, parasites or toxins, and is often the result of consuming contaminated water or food. Manifestations include nausea and vomiting, diarrhoea and abdominal discomfort.
- Nurses provide education to help prevent protozoal infections (e.g. giardiasis, amoebiasis) and helminthic infestations (by roundworms or tapeworms). Both are treated with medications.
- Chronic inflammatory bowel disease (IBD) includes two separate but closely related conditions: ulcerative colitis and Crohn's disease. Ulcerative colitis affects the mucosa and submucosa of the colon and rectum. Crohn's disease can affect any part of the GI tract, but usually involves the terminal ilium and ascending colon. Diarrhoea is common to both disorders. A colectomy (removal of the large colon) may be performed to treat ulcerative colitis; an ileostomy (artificial opening from the abdomen to the ileum) may be performed to treat Crohn's disease.
- Malabsorption syndromes, in which the intestinal mucosa ineffectively absorbs nutrients, are caused by a wide variety of diseases. However, three common malabsorption disorders in adults are coeliac disease (non-tropical and tropical), lactose intolerance of milk and milk products (resulting from a lactase deficiency) and short-bowel syndrome (a condition developing following resection of the small bowel).
- Malignant tumours of the lower bowel are the third leading cause of death from cancer in Australia. The risk of colon cancer may be reduced through health-related screenings and a diet high in fruits, vegetables, folic acid and calcium. Rectal bleeding is the most common initial manifestation but may not occur until the cancer is well advanced. Surgical treatment is through surgical resection of the bowel, accompanied by a colostomy for diversion of faecal contents.
- A hernia is a defect in the abdominal wall allowing intra-abdominal contents to protrude out of the abdominal cavity. Hernias may follow trauma, surgery and increased intra-abdominal pressure (as from pregnancy or obesity). Hernias may be congenital or acquired and may be inguinal, umbilical, incisional or ventral.
- Intestinal obstructions occur when intestinal contents cannot move through the lumen of the bowel. These may occur in either the large or small intestine, may be partial or complete, and are caused by many factors, ranging from surgical ileus following abdominal surgery to adhesions or tumours.
- Diverticula are sac-like projections of mucosa through the muscular layer of the colon. When these sacs become inflamed, the condition is labelled diverticulitis. A diet high in fibre is recommended for self-care.
- Anorectal disorders include haemorrhoids, anorectal lesions (fissures, abscess and fistula) and pilonidal disease. These disorders are painful and pose a risk of bleeding and infection.

CONCEPT CHECK

1 Mr William Brown presents at the urgent care clinic with complaints of diarrhoea for the past week. The RN should first:

1. advise Mr Brown to abstain from all food intake until the diarrhoea subsides
2. ask Mr Brown to describe the number and character of daily stools
3. question Mr Brown about possible exposure to an enterotoxin or protozoal infection
4. recommend an over-the-counter antidiarrhoeal preparation such as Kaomagma with Pectin Suspension

2 RN Sandra Smith, who is caring for Ms Wong admitted with possible appendicitis, appropriately plans which of the following?

1. Initiate bowel preparation for a barium enema.
2. Restrict intake to clear liquids.
3. Prepare for possible immediate appendectomy.
4. Insert saline lock for intravenous antibiotic therapy.

3 RN David Smith is teaching Mrs Doris Bishop with inflammatory bowel disease about prescribed sulfasalazine. RN Smith instructs Mrs Bishop to:

1. use a sunscreen while taking the drug
2. take the drug on an empty stomach
3. limit fluid intake to 1500 mL per day or less
4. take vitamin C while on this drug

4 Mr Adam Jones reports frequent large, fatty, foul-smelling stools. RN Brown recognises this as:
1 haematochezia, a manifestation of GI bleeding
2 characteristic of inflammatory bowel disease
3 a common early manifestation of colorectal cancer
4 steatorrhoea, a manifestation of malabsorption

5 Mr Stanley Green tells RN Davis that his father and grandfather died of colon cancer and he is worried that he is going to die from 'the same horrible disease'. Which of the following does RN Davis include in his recommendations?
1 There is no genetic link seen in colon cancer, so his risk is equal to that of people with no family history of the disease.
2 He should plan for annual digital rectal exams and periodic colonoscopy for early identification of possible tumours.
3 He should have annual CEA levels drawn to screen for early tumour development.
4 It is imperative that he change his diet immediately, significantly increasing his intake of dietary fibre.

6 Mrs Phyllis Jones is a 75 year old with a nasogastric tube in place to maintain gastric decompression. Which nursing actions are important in monitoring responses to *Deficient fluid volume* when caring for Mrs Jones? (Select all that apply.)
1 Low suction is used to decompress the stomach.
2 Give her as much water as she wants to drink.
3 Listen to bowel sounds prior to checking the placement of the NG tube.
4 Document the amount and colour of NG tube drainage every shift.
5 Keep an accurate record of intake and output every 2 to 4 hours.
6 Listen to bowel sounds after palpating the stomach for tenderness.
7 Measure abdominal girth every 4 to 8 hours.

7 Ms Stella Black developed a paralytic ileus following a recent abdominal surgery. What is the most important nursing consideration when caring for Ms Black?
1 Ensure Ms Black is able to eat a clear liquid diet.
2 Maintain Ms Black on strict bed rest.
3 Monitor Ms Black's bowel sounds every hour.
4 Ensure Ms Black's nasogastric tube is functioning.

8 Mrs Jones has a history of diverticulosis and has been having abdominal pain recently. When advising Mrs Jones about her diet prior to discharge from the hospital, what type of foods should Mrs Jones exclude from her diet?
1 wholemeal bread
2 popcorn
3 soup
4 apples

9 Mr Allen, an 85 year old, was admitted with a diagnosis of constipation. Which of the following is important for you to incorporate in Mr Allen's discharge education? (Select all that apply.)
1 Eat plenty of fresh fruits and vegetables daily.
2 Take bisacodyl (Durolax) daily.
3 Drink 6 to 8 glasses of non-alcoholic fluid daily.
4 Take docusate (Coloxyl) at night time only.
5 Eat wholemeal bread instead of white bread.
6 Eat a bran cereal for breakfast.

10 A small-bowel obstruction can occur due to:
1 eating extra fibre in the diet
2 abdominal adhesions
3 drinking too much water
4 a nasogastric tube

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CHAPTER 24

NURSING CARE OF PEOPLE WITH GALLBLADDER, LIVER AND PANCREATIC DISORDERS

NICOLE KNOX

KEY TERMS

LEARNING OUTCOMES

- Discuss the pathophysiology, manifestations and associated nursing care of the person suffering from a disorder of the gallbladder.
- Compare and contrast the pathophysiology, manifestations and complications of hepatitis, cirrhosis, trauma and cancer of the liver.
- Discuss pathophysiology, manifestations and interprofessional care of a person with pancreatitis or cancer of the pancreas.

CLINICAL COMPETENCIES

- Assess functional health status of people with gallbladder, liver or pancreatic disease.
- Monitor for, document and report expected and unexpected manifestations in people with gallbladder, liver or pancreatic disease.
- Prepare people for, and understand the purpose and significance of, diagnostic tests for gallbladder, liver and pancreatic disorders.
- Integrate appropriate dietary, pharmacological and other interprofessional measures into nursing care and teaching of the person with a gallbladder, liver or pancreatic disorder.
- Provide appropriate nursing care for the person who has surgery of the gallbladder, liver or pancreas.
- Integrate psychosocial, cultural and spiritual considerations into the plan of care for a person with a gallbladder, liver or pancreatic disorder.
- Use evidence-based practice to develop, implement, evaluate and, as needed, revise the plan of care for people with disorders of the gallbladder, liver or pancreas.
- Provide appropriate person and family health education to promote, maintain and restore functional health status for people with gallbladder, liver and pancreatic disorders.

Gallbladder, liver and exocrine pancreatic disorders may occur as primary disorders or develop secondarily to other disease processes. One organ's functioning frequently affects that of another. Duct inflammation or obstruction and changes in the multiple functions of these organs can cause significant health effects.

People with a gallbladder, liver or pancreatic disorder may experience pain, multiple metabolic and nutritional disturbances, and altered body image. Nursing care addresses the physiological and psychosocial needs of the person and family.

GALLBLADDER DISORDERS

Altered bile flow through the hepatic, cystic or common bile duct is a common problem. It often leads to inflammation and other complications. Gallstones are the most common cause of obstructed flow. Tumours and abscesses also can obstruct bile flow.

THE PERSON WITH GALLSTONES

Cholelithiasis is the formation of stones (*calculi* or *gallstones*) within the gallbladder or biliary duct system. Cholelithiasis is a common problem worldwide with an estimated prevalence of 10–15% in white adults (Stinton, Myers & Schaffer, 2010; Venneman & van Erpecum, 2010). Box 24.1 lists risk factors for gallstones. The incidence of gallstones varies among people of different ethnic backgrounds.

Physiology review

Normally bile is formed by the liver and stored in the gallbladder. Bile contains bile salts, bilirubin, water, electrolytes, cholesterol, fatty acids and lecithin. In the gallbladder, some of the water and electrolytes are absorbed, further concentrating the bile. Food entering the intestine stimulates the gallbladder to contract and release bile through the common bile duct and sphincter of Oddi into the intestine. The bile salts in bile increase the solubility and absorption of dietary fats.

Pathophysiology and manifestations

Cholelithiasis

Gallstones form when several factors interact: abnormal bile composition, biliary stasis and inflammation of the gallbladder. Most gallstones (80%) consist primarily of cholesterol; the rest contain a mixture of bile components. Excess cholesterol in bile is associated with obesity; a high-kilojoule, high-cholesterol diet; and drugs that lower serum cholesterol levels. When bile is supersaturated with cholesterol, it can precipitate out to form stones. Biliary stasis, or slowed emptying of the gallbladder, contributes to cholelithiasis. Stones do not form when the gallbladder empties completely in response to hormonal stimulation. Slowed or incomplete emptying allows cholesterol to concentrate and increases the risk of stone formation. Finally, inflammation of the gallbladder allows excess water and bile salt reabsorption, increasing the risk of lithiasis.

CONSIDERATION FOR PRACTICE

Certain very-low-kilojoule diets are associated with a high risk of cholelithiasis. Increased cholesterol concentration in the bile and decreased gallbladder contractions associated with fasting increase the risk of gallstone formation.

Most gallstones are formed in the gallbladder. They then may migrate into the ducts (see Figure 24.1), leading to *cholangitis* (duct inflammation). Although some people with cholelithiasis are asymptomatic, many develop manifestations. Early manifestations of gallstones may be vague: epigastric fullness or mild gastric distress after eating a large or fatty meal. Stones that obstruct the cystic duct or common bile duct lead to distension and increased pressure behind the stone. This causes **biliary colic**, a severe, steady pain in the epigastric region or right upper quadrant of the abdomen.

BOX 24.1 Risk factors for gallstones

- Age
- Family history of gallstones
- Race or ethnicity
- Obesity, hyperlipidaemia
- Rapid weight loss
- Female gender; use of oral contraceptives
- Biliary stasis: pregnancy, fasting, prolonged parenteral nutrition
- Diseases or conditions: cirrhosis; ileal disease or resection; sickle cell anaemia; glucose intolerance

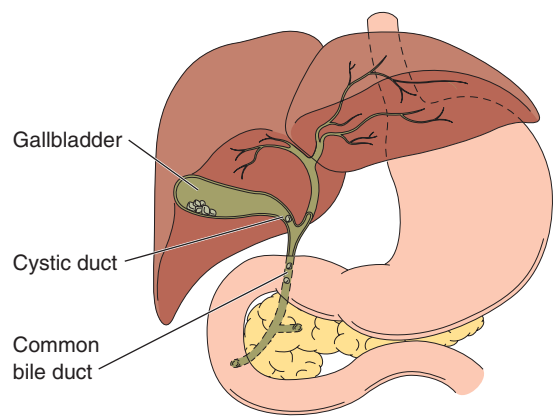


FIGURE 24.1 ■ Common locations of gallstones

TABLE 24.1 Manifestations and complications of cholelithiasis and cholecystitis

MANIFESTATIONS	CHOLELITHIASIS	CHOLECYSTITIS
Pain	• Abrupt onset • Severe, steady • Localised to epigastrium and RUQ of abdomen • May radiate to back, right scapula and shoulder • Lasts 30 minutes to 5 hours	• Abrupt onset • Severe, steady • Generalised in RUQ of abdomen • May radiate to back, right scapula and shoulder • Lasts 12 to 18 hours • Aggravated by movement, breathing
Associated symptoms	• Nausea, vomiting	• Anorexia, nausea, vomiting • RUQ tenderness and guarding • Rigors and fever
Complications	• Cholecystitis • Common bile duct obstruction with possible jaundice and liver damage • Common duct obstruction with pancreatitis	• Gangrene and perforation with peritonitis • Chronic cholecystitis • Empyema • Fistula formation • Gallstone ileus

The pain may radiate to the back, right scapula or shoulder. The pain often begins suddenly following a meal and may last as long as 5 hours. It often is accompanied by nausea and vomiting.

Obstruction of the common bile duct may cause bile reflux into the liver, leading to jaundice, pain and possible liver damage. If the common duct is obstructed, pancreatic enzymes will be unable to enter the small intestine and pancreatitis (discussed later in this chapter) becomes a potential complication.

Cholecystitis

Cholecystitis is inflammation of the gallbladder. *Acute cholecystitis* usually follows obstruction of the cystic duct by a stone. The obstruction increases pressure within the gallbladder, leading to ischaemia of the gallbladder wall and mucosa. Chemical and bacterial inflammation often follows. The ischaemia can lead to necrosis and perforation of the gall bladder wall.

Acute cholecystitis usually begins with an attack of biliary colic. The pain involves the entire right upper quadrant (RUQ) and may radiate to the back, right scapula or shoulder. Movement or deep breathing may aggravate the pain. The pain usually lasts longer than biliary colic, continuing for 12 to 18 hours. Anorexia, nausea and vomiting are common. Fever often is present and may be accompanied by rigors. The RUQ is tender to palpation.

Chronic cholecystitis may result from repeated bouts of acute cholecystitis or from persistent irritation of the gallbladder wall by stones. Bacteria may be present in the bile as well. Chronic cholecystitis is often asymptomatic.

Complications of cholecystitis include *empyema*, a collection of infected fluid within the gallbladder; gangrene and perforation with resulting peritonitis or abscess formation; formation of a fistula into an adjacent organ (such as the duodenum, colon or stomach); or obstruction of the small intestine by a large gallstone (*gallstone ileus*). Table 24.1 compares the manifestations and complications of acute cholelithiasis with those of cholecystitis.

INTERPROFESSIONAL CARE

Treatment of the person with cholelithiasis or cholecystitis depends on the acuity of the condition and the person's overall health status. When gallstones are present but asymptomatic and the person has a low risk of complications, conservative treatment is indicated. However, when the person experiences frequent symptoms, has acute cholecystitis or has very large stones, the gallbladder and stones are usually surgically removed.

Diagnosis

Diagnostic tests are ordered to identify the presence and location of stones, identify possible complications and help differentiate gallbladder disease from other disorders.

- *Serum bilirubin* is measured. Elevated direct (conjugated) bilirubin may indicate obstructed bile flow in the biliary duct system (see Box 24.2).
- *Full blood count (FBC)* may indicate infection and inflammation if the white blood cell (WBC) count is elevated.
- *Serum amylase* and *lipase* are measured to identify possible pancreatitis related to common duct obstruction.
- *Abdominal x-ray* may show gallstones that have high calcium content.
- *Ultrasonography of the gallbladder* is a non-invasive exam that can accurately diagnose cholelithiasis. It also can be used to assess emptying of the gallbladder.
- *Oral cholecystogram* is performed using a dye administered orally to assess the gallbladder's ability to concentrate and excrete bile.
- *Gallbladder scans* use an intravenous radioactive solution that is rapidly extracted from the blood and excreted into the biliary tree to diagnose cystic duct obstruction and acute or chronic cholecystitis.

See Chapter 20 for more information about these diagnostic tests and their nursing implications.

The primary disadvantages of pharmacological treatment for gallstones include its cost, long duration (2 years or more) and the high incidence of recurrent stone formation when

BOX 24.2 Total, direct and indirect bilirubin levels

When serum bilirubin levels are measured, the results usually are reported as the total bilirubin, direct bilirubin and indirect bilirubin levels. Most bilirubin is formed from haemoglobin, as ageing or abnormal red blood cells (RBCs) are removed from circulation and destroyed. It is then bound to protein and transported to the liver. This protein-bound bilirubin is called *indirect* or *unconjugated* bilirubin. Once in the liver, bilirubin is separated from the protein and converted to a soluble form, *direct* or *conjugated* bilirubin. Conjugated bilirubin is then excreted in the bile.

- *Total* (serum) *bilirubin*, the total bilirubin in the blood, includes both indirect and direct forms. In adults, the normal total bilirubin is 0.3 to 1.2 mg/dL. Total bilirubin levels increase when more is being produced (e.g. RBC haemolysis) or when its metabolism or excretion is impaired (e.g. liver disease or biliary obstruction).
- *Direct* (conjugated) *bilirubin* levels, normally 0 to 0.2 mg/dL in adults, rise when its excretion is impaired by obstruction within the liver (e.g. in cirrhosis, hepatitis, exposure to hepatotoxins) or in the biliary system.
- *Indirect* (unconjugated) *bilirubin* levels, normally < 1.1 mg/dL in adults, rise in RBC haemolysis (e.g. sickle cell disease or transfusion reaction).

treatment is discontinued. If infection is suspected, antibiotics may be ordered to cure the infection and reduce associated inflammation and oedema. People with pruritus (itching) due to severe obstructive jaundice and an accumulation of bile salts on the skin may be given cholestyramine (Questran Lite). This drug binds with bile salts to promote their excretion in the faeces. A narcotic analgesic such as morphine may be required for pain relief during an acute attack of cholecystitis.

Treatments

SURGERY **Laparoscopic cholecystectomy** (removal of the gallbladder) is the treatment of choice for symptomatic cholelithiasis or cholecystitis. This minimally invasive procedure has a low risk of complications and generally requires a hospital stay of less than 24 hours. Not all people are candidates for laparoscopic cholecystectomy and there is a risk that a laparoscopic cholecystectomy may be converted to a *laparotomy* (surgical opening into the abdomen) during the procedure. See below for nursing care for a person having a laparoscopic cholecystectomy. The 'Translation to practice' box below discusses evidence-based practice for managing pain in people undergoing laparoscopic cholecystectomy.

When stones are lodged within the ducts, a cholecystectomy with common bile duct exploration may be done. A T-tube (see Figure 24.2) is inserted to maintain patency of the duct and promote bile passage while the oedema decreases. Excess bile is collected in a drainage bag secured below the surgical site. If it is suspected that a stone has been retained following surgery, a postoperative cholangiogram via the T-tube or direct visualisation of the duct with an endoscope may be performed. See the box below for nursing care for a person with a T-tube.

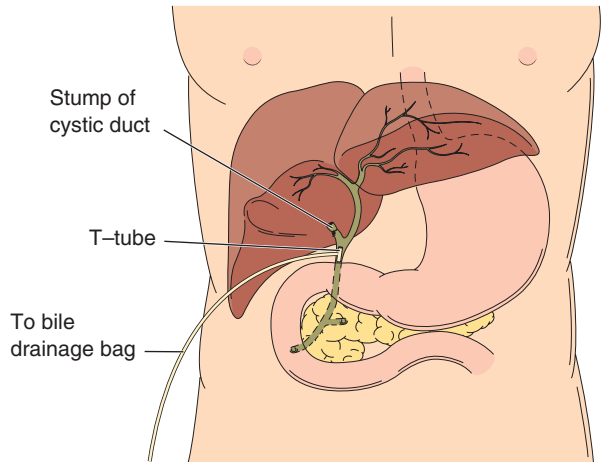


FIGURE 24.2 ■ T-tube placement in the common bile duct. Bile fluid flows with gravity into a drainage collection device below the level of the common bile duct

Some people who are poor surgical risks and for whom laparoscopic cholecystectomy is inappropriate may have either a *cholecystostomy* to drain the gallbladder or a *choledochostomy* to remove stones and position a T-tube in the common bile duct.

NUTRITION Food intake may be eliminated during an acute attack of cholecystitis and a nasogastric tube inserted to relieve nausea and vomiting. Dietary fat intake may be limited, especially if the person is obese. If bile flow is obstructed, fat-soluble vitamins (A, D, E and K) and bile salts may need to be administered.

OTHER THERAPIES In some cases, shock wave lithotripsy may be used with drug therapy to dissolve large gallstones. In extracorporeal shock wave lithotripsy, ultrasound is used to align the stones with the source of shock waves and the computerised lithotripter. Positioning is of prime importance throughout the procedure, which usually takes an hour. Mild sedation may be given during the procedure. Nursing care after the procedure includes monitoring for biliary colic, which can result from the gallbladder contracting to remove stone fragments; nausea; and transient haematuria. *Percutaneous cholecystostomy*, ultrasound-guided drainage of the gallbladder, may be done in high-risk people to postpone or even eliminate the need for surgery.

Nursing care

In addition to the nursing care discussed in this section, a nursing care plan for a person with cholelithiasis is found below.

Health promotion

Although most risk factors for cholelithiasis cannot be controlled or modified, several can. Modifiable risk factors include obesity, hyperlipidaemia, extremely low-kilojoule diets and diets high in cholesterol. Encourage people who are obese to increase their activity level and follow a low carbohydrate, low-fat, low-cholesterol diet to promote weight loss and reduce their

risk of developing gallstones. Discuss the dangers of 'yo-yo' dieting, with cycles of weight loss followed by weight gain, and of extremely low-kilojoule diets. Encourage people with high serum cholesterol levels to discuss using cholesterol-lowering drugs with their primary care provider.

Assessment

Assessment data related to cholelithiasis and cholecystitis include the following:

- *Health history:* current manifestations, including RUQ pain, its character and relationship to meals, duration and radiation, nausea and vomiting or other symptoms; duration of symptoms; risk factors or previous history of symptoms; chronic diseases such as diabetes, cirrhosis or inflammatory bowel disease; current diet; use of oral contraceptives or possibility of pregnancy.
- *Physical assessment:* current weight; colour of skin and sclera; abdominal assessment including light palpation for tenderness; colour of urine and stool.
- *Diagnostic tests:* monitor results of WBC count, serum bilirubin, liver enzymes and pancreatic enzymes (amylase and lipase).

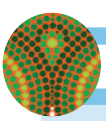
NURSING CARE OF THE PERSON having a laparoscopic cholecystectomy

PREOPERATIVE CARE

- Provide routine preoperative care as ordered (see Chapter 3).
- Reinforce teaching about the procedure and postoperative expectations, including pain management, deep breathing and mobilisation. *Preoperative teaching reduces anxiety and promotes rapid postoperative recovery.*

POSTOPERATIVE CARE

- Provide routine postoperative recovery care as outlined in Chapter 3.
- Assist to chair at bedside as allowed. *Early mobilisation promotes lung ventilation and circulation, reducing the potential for postoperative complications.*
- Advance oral intake from ice chips to regular diet as tolerated. *Oral intake can be rapidly resumed due to minimal disruption of the gastrointestinal tract during surgery.*
- Provide and reinforce teaching: pain management, incision care, activity level and postoperative follow-up appointments. *With early discharge, the person and family assume responsibility for the majority of postoperative care. A clear understanding of this care and expected needs reduces anxiety and the risk of postoperative complications.*
- Initiate follow-up contact 24 to 48 hours after discharge to evaluate adequacy of pain control, incision management and discharge understanding. *Contact following discharge provides an opportunity to evaluate care and reinforce teaching.*



TRANSLATION TO PRACTICE Evidence-based practice: a person undergoing laparoscopic cholecystectomy

The purpose of pre-emptive analgesia administration is to reduce the central sensitisation from a variety of harmful stimuli. Its proposed benefits include better postoperative pain management, therefore reducing the use of analgesia and improving patient outcomes (Singh et al., 2013). In a study of pre-emptive ketamine analgesia administration among laparoscopic cholecystectomy patients, Singh et al. (2013) studied pain scores in the first 24 hours post surgery. This is an important time frame considering the majority of laparoscopic cholecystectomy patients are discharged within the first 24 hours post surgery. Patients were randomised into four groups, the first three groups (A, B and C) receiving 1 mg/kg, 0.75 mg/kg and 0.5 mg/kg of intravenous ketamine respectively, 30 minutes prior to surgery. The final group (group D) received a placebo of isotonic saline. The study found that all groups that received pre-emptive ketamine had a reduced pain score postoperatively compared to the group that did not receive the drug.

IMPLICATIONS FOR NURSING

Effective pain relief is known to promote healing and immune function following surgery. This study indicates a benefit from the use of pre-emptive analgesia administration in people undergoing ambulatory surgery, including laparoscopic cholecystectomy, as a pain management strategy. Effective postoperative pain management requires a combination of good preoperative education, discharge planning related to the person's expectations of pain and postoperative pain management.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Two people in group A of this study (those that received 1 mg/kg of ketamine) reported hallucinations. How can the nurse ensure appropriate patient safety while promoting effective postoperative pain management? What teaching could you provide to help people manage adverse effects of the prescribed drug?
2. The incidence of nausea and vomiting reported was similar in all four study groups. How will the incidence of nausea and vomiting impact on the pain management of a person undergoing laparoscopic cholecystectomy?

Source: Preemptive analgesia with ketamine for laprascopic cholecystectomy by Singh, H. et al. (2013). *Journal of Anaesthesiology, Clinical Pharmacology, 29*(4), 478–484.

NURSING CARE OF THE PERSON with a T-tube

- Ensure that the T-tube is properly connected to a sterile container; keep the tube below the level of the surgical wound. *This position promotes the flow of bile and prevents backflow or seepage of caustic bile on to the skin. The tube itself decreases biliary tree pressure.*
- Monitor drainage from the T-tube for colour and consistency; record as output. Normally, the tube may drain up to 500 mL in the first 24 hours after surgery; drainage decreases to less than 200 mL in 2 to 3 days and is minimal thereafter. Drainage may be blood tinged initially, changing to green–brown. Report excessive drainage immediately. (After 48 hours, drainage greater than 500 mL is considered excessive.) *Stones or oedema and inflammation can obstruct ducts below the tube, requiring treatment.*
- Place in Fowler's position. *This promotes gravity drainage of bile.*
- Assess skin for bile leakage during dressing changes. *Bile irritates the skin; it may be necessary to apply skin protection with a barrier product.*
- Teach the person how to manage the tube when turning, ambulating and performing activities of daily living. *Direct pulling or traction on the tube must be avoided.*
- If indicated, teach care of the T-tube, how to clamp it, signs of infection and when to report these symptoms to clinicians. People may be discharged home with the tube in place. *Reporting early signs of infection facilitates prompt treatment.*

NURSING CARE PLAN A person with cholelithiasis

Joyce Wing is a 44-year-old married mother of three children. She works full time as a cook at a local restaurant. Recently Mrs Wing has noticed a dull pain in her upper abdomen that gets worse after eating fatty foods; nausea and sometimes vomiting accompany the pain. She had a similar pain after the birth of her last child. She is diagnosed with cholelithiasis and is admitted for a laparoscopic cholecystectomy.

ASSESSMENT

David Corbin, RN, takes Mrs Wing's admission history. It includes intolerance to fatty foods and intermittent 'stabbing' abdominal pain that radiates to her back. Her usual diet includes regular takeaway meals due to her busy lifestyle. She reports 'not wanting to eat much of anything lately'. She states she has never had surgery before and hopes 'everything goes well'. Physical assessment includes T 37.7°C, P 88, R 20 and BP 130/84. She has had a recent 3 kg weight loss, currently weighing 59 kg. She is 160 cm tall. Abdominal examination elicits tenderness in the right upper abdominal quadrant. She has no jaundice, rigors or evidence of complications.

DIAGNOSES

- *Risk of imbalanced nutrition: less than body requirements* related to anorexia and manifested by recent weight loss.
- *Risk of pain* related to inflamed gallbladder and surgical incisions manifested by increased heart rate and blood pressure and decreased inspiratory volume.
- *Risk of infection* related to potential bacterial contamination of abdominal cavity, manifested by increased white cell count, temperature, heart and respiratory rate and decreased blood pressure.
- *Risk of anxiety* related to lack of information about perioperative experience and manifested by restlessness, increased heart rate and blood pressure.

PLANNING

- Teach about the gallbladder and the function of bile.
- Discuss pre- and postoperative care, including self-care following discharge.
- Teach home care of incisions and recognition of signs of infection.

Expected outcomes

- Maintain present weight within 2 kg over the next 3 weeks.
- Resume regular diet, decreasing intake of foods high in fat.
- Verbalise adequate pain control after surgery and with activity resumption.
- Remain free of infection.
- Verbalise a decrease in anxiety before surgery.

IMPLEMENTATION

- Promote mobility as soon as allowed after surgery.
- Review specific high-fat foods to avoid and ways to maintain her weight. Consultation with a dietitian if available would be beneficial.
- Provide analgesia as needed postoperatively. Teach appropriate analgesic use after discharge.

EVALUATION

Mrs Wing is discharged the morning after her surgery. She is afebrile, has no signs of infection and is able to appropriately care for her incisions. She identifies signs of infection and talks about ways to reduce her fat intake while keeping her weight stable. She verbalises understanding of initial activity restrictions and resumption of normal activities. Mrs Wing states, 'It wasn't as bad as I thought it would be at first.' She has an appointment to see her surgeon in 1 week.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the rationale for a low-fat diet with cholelithiasis? Discuss nutritional practices as they relate to the medical problem and Mrs Wing's lifestyle.
2. How would your discharge teaching for Mrs Wing differ if she had had an open cholecystectomy instead of a laparoscopic cholecystectomy?

REFLECTION ON THE NURSING PROCESS

1. Develop a care plan for Mrs Wing for the nursing diagnosis *Risk of infection.*
2. Discuss strategies you can use to encourage postoperative patients to mobilise.

Nursing diagnoses and interventions

Priority nursing diagnoses for the person with cholelithiasis or cholecystitis often include pain related to biliary colic or surgery, imbalanced nutrition related to the effects of altered bile flow and to nausea and anorexia, and risk of infection related to potential rupture of an acutely inflamed gallbladder. Nursing interventions for the person who has undergone a laparoscopic or open cholecystectomy are similar to those for other people having abdominal surgery. (See Chapter 3.)

Pain

The pain associated with cholelithiasis can be severe. Sometimes a combination of interventions is indicated.

- Discuss the relationship between fat intake and the pain. Teach ways to reduce fat intake. *Fat entering the duodenum initiates gallbladder contractions, causing pain when gallstones are present in the ducts.*
- Withhold oral food and fluids during episodes of acute pain. Insert nasogastric tube and connect to low suction if ordered. *Emptying the stomach reduces both the amount of chyme entering the duodenum and the stimulus for gallbladder contractions, thus reducing pain.*
- For severe pain, administer morphine or other narcotic analgesia as ordered. *Recent research indicates that morphine is not likely to cause spasms of the sphincter of Oddi.*
- Place in Fowler's position. *Fowler's position decreases pressure on the inflamed gallbladder.*
- Monitor vital signs, including temperature, at least every 4 hours. *Bacterial infection often is present in acute cholecystitis and may cause an elevated temperature and respiratory rate.*

Imbalanced nutrition: less than body requirements

The person with severe gallbladder disease may develop nutritional imbalances related to anorexia, pain and nausea following meals and impaired bile flow that alters absorption of fat and fat-soluble vitamins (A, D, E and K) from the gut.

- Assess nutritional status, including diet history, height and weight, and skinfold measurements (see Chapters 20 and 21). *Although often obese, people with gallbladder disease may have an imbalanced diet or may have specific vitamin deficiencies, particularly of the fat-soluble vitamins.*
- Evaluate laboratory results, including serum bilirubin, albumin, glucose and cholesterol levels. Report abnormal results to the doctor. *Elevated serum bilirubin may indicate impaired bilirubin excretion due to obstructed bile flow. A low serum albumin may indicate poor nutritional status. Glucose intolerance and hypercholesterolaemia are risk factors for cholelithiasis.*
- Refer to a dietitian or nutritionist for diet counselling to promote healthy weight loss and reduce pain episodes. *A low-carbohydrate, low-fat, higher-protein diet reduces symptoms of cholecystitis. While fasting and very low kilojoule diets are contraindicated, a moderate reduction in kilojoule intake and increased activity levels promote weight loss.*
- Administer vitamin supplements as ordered. *People who do not absorb fat well due to obstructed bile flow may require supplements of the fat-soluble vitamins.*

Risk of infection

An acutely inflamed gallbladder may become necrotic and rupture, releasing its contents into the abdominal cavity. While the resulting infection often remains localised, peritonitis can result from chemical irritation and bacterial contamination of the peritoneal cavity.

CONSIDERATION FOR PRACTICE

Rupture of an acutely inflamed gallbladder may be heralded by abrupt but transient pain relief as contents are released from the distended gallbladder into the abdomen. Promptly report this change to the doctor.

Following open cholecystectomy (*laparotomy*), the risk of pulmonary infection is significant due to the high abdominal incision.

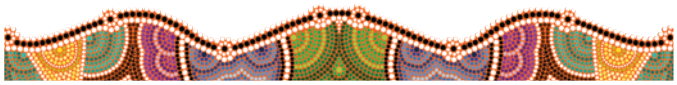
- Monitor vital signs, including temperature, every 4 hours. Promptly report vital sign changes or temperature elevation. *Tachycardia, increased respiratory rate or an elevated temperature may indicate an infectious process.*
- Assess abdomen every 4 hours and as indicated (e.g. when pain level changes abruptly). *Increasing abdominal tenderness or a rigid, board-like abdomen may indicate rupture of the gallbladder with peritonitis.*
- Assist to cough and deep breathe or use an incentive spirometer every 1 to 2 hours while awake. Splint abdominal incision with a blanket or pillow during coughing. *The high abdominal incision of an open cholecystectomy interferes with effective coughing and deep breathing, increasing the risk of atelectasis and respiratory infections such as pneumonia.*
- Place in Fowler's position and encourage ambulation as allowed. *Fowler's position and ambulation promote lung expansion and airway clearance, reducing the risk of respiratory infections.*
- Administer antibiotics as ordered. *Antibiotics may be given preoperatively to reduce the risk of infection from infected gallbladder contents and may be continued postoperatively to prevent infection.*

Community-based care

Teaching varies, depending on the choice of treatment options for cholelithiasis and cholecystitis. If surgery is not an option, teach about maintaining a low-fat, low-carbohydrate diet if indicated. Include an explanation about the role of bile and the function of the gallbladder in terms that the person and family can understand.

Provide appropriate preoperative teaching for the planned procedure. Discuss the possibility of open cholecystectomy even when a laparoscopic procedure is planned. Teach postoperative self-care measures to manage pain and prevent complications. If the person will be discharged with a T-tube, provide instructions about its care (see the 'Nursing care' box above). Discuss manifestations of complications to report to the doctor. Stress the importance of follow-up appointments.

Following cholecystectomy, a low-fat diet may be initially recommended. Refer the person and food preparer to a dietitian to review low-fat foods. (See Box 24.3 for examples of high-fat foods to avoid.) Higher-fat foods may be gradually added to the diet as tolerated.



THE PERSON WITH CANCER OF THE GALLBLADDER

Gallbladder cancer is rare, primarily affecting women over age 65. Manifestations of gallbladder cancer include intense pain and a palpable mass in the RUQ of the abdomen. Jaundice and weight loss are common. Gallbladder cancers spread by direct extension to the liver and metastasise via the blood and lymph system.

At the time of diagnosis, the cancer usually is too advanced to treat surgically. Most people present with advanced, incurable disease and are not candidates for surgical resection. The mean survival for people with inoperable tumours is 2–4 months. Even with surgical intervention, the 5-year survival rate is around 35% (Jayaraman & Jarnagin, 2010).

Nursing care is palliative, focusing on maintaining comfort and independence to the extent possible.

BOX 24.3 Examples of high-fat foods

- Whole-milk products (e.g. cream, ice-cream, cheese)
- Doughnuts, deep-fried
- Sausage, bacon, hot dogs
- Gravies with fat, cream
- Most nuts (e.g. pecans, cashews)
- Corn chips and potato chips
- Butter and cooking oils
- Fried foods (e.g. hamburgers, hot chips)
- Peanut butter
- Chocolate

LIVER DISORDERS

The liver is a complex organ with multiple metabolic and regulatory functions. Optimal liver function is essential to health. Because of the significant amount of blood in the liver at all times, it is exposed to the effects of pathogens, drugs, toxins and possibly malignant cells. As a result, liver cells may become inflamed or damaged, or cancerous tumours may develop.

Physiology review

The essential functions of the liver include the metabolism of proteins, carbohydrates and fats. It also is responsible for the metabolism of steroid hormones and most drugs. It synthesises essential blood proteins, including albumin and clotting factors in particular. The liver detoxifies alcohol and other toxic substances. Ammonia, a toxic by-product of protein metabolism, is converted to urea in the liver for elimination by the kidneys. The liver produces bile, an essential substance for absorbing fats and eliminating bilirubin from the body. Minerals and fat-soluble vitamins are stored in the liver, as is glycogen (stored carbohydrate for energy reserves). The Kupffer cells that line the sinusoids phagocytise foreign cells and damaged blood cells. See Chapter 20 for more information about the liver.

Common manifestations of liver disorders

Although many different disorders can disrupt liver function, their manifestations relate to three primary effects: disrupted liver cell function, impaired bilirubin conversion and excretion leading to jaundice and disrupted blood flow through the liver, with resulting portal hypertension.

Hepatocellular failure

The liver is vital to the digestion and metabolism of nutrients; the production of plasma proteins, including those involved in clotting; and the metabolism and excretion of compounds such as bilirubin, steroid hormones and ammonia, as well as toxins (such as alcohol) and drugs. Impaired function of liver cells has multiple effects, including:

- Impaired protein metabolism with decreased production of albumin and clotting factors. Low albumin levels contribute to oedema in peripheral tissues and **ascites** (accumulation of fluid in the abdomen) as plasma oncotic pressure is reduced. Impaired clotting factor production increases the risk of bleeding.
- Disrupted glucose metabolism and storage with resulting alterations in blood glucose levels (either hyperglycaemia or hypoglycaemia).
- Reduced bile production that impairs the absorption of lipids and fat-soluble vitamins. Inadequate vitamin K, a fat-soluble vitamin, affects the production of clotting factors, leading to a bleeding tendency.
- Impaired metabolism of steroid hormones (including oestrogen and testosterone) leads to feminisation in men and irregular menses in women.

Jaundice

Disrupted metabolism and excretion of bilirubin allows it to accumulate in tissues, leading to **jaundice** (yellow staining of tissues). Jaundice (also called *icterus*) often is first noticeable in the sclera of the eyes, then the skin.

When RBCs are destroyed (due to cell ageing or disease), haemoglobin is released. The haemoglobin molecule breaks up into globin, a protein, and haem, the iron-containing portion of

the molecule. In this process, biliverdin, later converted to fat-soluble bilirubin (*unconjugated bilirubin*), is released. The bilirubin binds with albumin to be transported to the liver. In the liver, it is converted to a water-soluble form (*conjugated bilirubin*) to be excreted in the bile. See Box 24.2 for more information about bilirubin metabolism.

Jaundice can result from disruptions at any point in the production and metabolism of bilirubin:

- *Haemolytic jaundice* develops when excess RBC destruction (haemolysis) releases more bilirubin into circulation than the liver is able to process. High blood levels of unconjugated bilirubin are seen.
- *Hepatic jaundice* occurs when impaired liver cell (*hepatocyte*) function disrupts the conversion and excretion of bilirubin. Blood levels of both conjugated and unconjugated bilirubin may be elevated. Stools may appear normal or clay coloured and urine is dark because the conjugated bilirubin is excreted by the kidneys.
- Obstruction of bile flow within the biliary system (the gallbladder and bile ducts) impairs bilirubin excretion, leading to *obstructive jaundice*. Levels of conjugated bilirubin are elevated. Stools are light or clay coloured due to lack of bile pigment, and urine is dark because the kidneys excrete bilirubin.

Portal hypertension

Impaired blood flow through the liver increases pressure in the portal venous system that drains the gastrointestinal tract, the spleen and surface veins of the abdomen. **Portal hypertension**, increased pressure in the portal system, has several effects when it is prolonged:

- Dilation of veins in the gastrointestinal tract and the abdominal wall. This congestion tends to suppress the appetite and lead to the formation of collateral vessels in the distal oesophagus, stomach and rectum. The dilated, congested vessels in the oesophagus are known as *oesophageal varices*; in the rectum, they lead to the development of haemorrhoids. In advanced liver failure, superficial varices may develop around the umbilicus, a feature known as *caput medusae*.
- Splenomegaly or enlargement of the spleen.
- Ascites (accumulation of fluid in the peritoneal cavity). Increased hydrostatic pressure in abdominal vessels forces fluid out of the vessels and into the peritoneal cavity. Low serum albumin levels (*hypoalbuminemia*) contribute to fluid accumulation by reducing the osmotic draw of fluid back into vessels.
- **Portal systemic encephalopathy** (or hepatic encephalopathy), impaired consciousness and mental status due to the accumulation of toxic waste products in the blood (ammonia, in particular) as blood bypasses the congested liver. It appears that factors other than elevated ammonia levels contribute, including toxic fatty acids, altered neurotransmitters and an imbalance of plasma amino acid ratios. Cerebral oedema develops late in the course of liver failure, resulting from both the accumulation of toxins and vascular mechanisms. As cerebral oedema progresses, intracranial pressure increases, cerebral perfusion decreases and brain cells become hypoxic.
- **Hepatorenal syndrome** is acute renal failure due to disrupted blood flow to the kidneys. See Chapter 27 for more information about renal failure.

See the section of this chapter on cirrhosis for more information about the effects and complications associated with portal hypertension.

THE PERSON WITH HEPATITIS

Hepatitis is inflammation of the liver. It is usually caused by a virus, although it may result from exposure to alcohol, drugs and toxins, or other pathogens. Hepatitis may be acute or chronic in nature. Cirrhosis, discussed in the next section, is a potential consequence of severe hepatocellular damage. Chronic hepatitis also increases the risk of developing liver cancer.

Pathophysiology and manifestations

The inflammatory process of hepatitis, whether caused by a virus, a toxin or another mechanism, damages hepatic cells and disrupts liver function. Cell-mediated immune responses damage hepatocytes and Kupffer cells, leading to hyperplasia, necrosis and cellular regeneration. The flow of bile through bile canaliculi and into the biliary system can be impaired by the inflammatory process, leading to jaundice. When the inflammatory process is mild (e.g. hepatitis A), the liver parenchyma is not significantly damaged. The inflammatory processes associated with hepatitis B and hepatitis C, however, can lead to severe liver damage. The metabolism of nutrients, drugs, alcohol and toxins and the process of bile elimination are disrupted by the inflammation of hepatitis. See Chapter 20 for more information about the liver and the preceding section for more information about the effects of disrupted liver function.

Viral hepatitis

At least five viruses are known to cause hepatitis: hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), the hepatitis B-associated delta virus (HDV) and hepatitis E virus (HEV). With the exception of HBV, all of the hepatitis viruses are RNA viruses; HBV is a DNA virus. The viruses differ from one another in mode of transmission, incubation period, the severity and type of liver damage they cause, and their ability to become chronic or develop a carrier (asymptomatic) state. The illnesses they cause, however, are clinically very similar. Table 24.2 identifies unique features of the primary hepatitis viruses.

Hepatitis viruses replicate in the liver, damaging liver cells (hepatocytes). The viruses provoke an immune response that causes inflammation and necrosis of hepatocytes as well. Although the extent of damage and the immune response vary among the different hepatitis viruses, the disease itself usually follows a predictable pattern.

No manifestations are present during the incubation period after exposure to the virus. The *prodromal* or *preicteric* (before jaundice) *phase* may begin abruptly or insidiously, with general malaise, anorexia, fatigue and muscle and body aches.

TABLE 24.2 Comparison of types of viral hepatitis

VIRUS	HEPATITIS A (HAV)	HEPATITIS B (HBV)	HEPATITIS C (HCV)	HEPATITIS D (HDV)	HEPATITIS E (HEV)
Mode of transmission	Faecal–oral	Blood and body fluids; perinatal	Blood and body fluids	Blood and body fluids; perinatal	Faecal–oral
Incubation (in weeks)	2–6	6–24	5–12	3–13	3–6
Onset	Abrupt	Slow	Slow	Abrupt	Abrupt
Carrier state	No	Yes	Yes	Yes	Yes
Possible complications	Rare	Chronic hepatitis Cirrhosis Liver cancer	Chronic hepatitis Cirrhosis Liver cancer	Chronic hepatitis Cirrhosis Fulminant hepatitis	May be severe in pregnant women
Laboratory findings	Anti-HAV antibodies present	Positive HBsAg (HBV surface antigen); anti-HBV antibodies present	Anti-HCV antibodies present	Positive HDVAg (delta antigen) early; anti-HDV antibodies later	Anti-HEV antibodies present

These manifestations often are mistaken for the flu. Nausea, vomiting, diarrhoea or constipation may develop, as well as mild RUQ abdominal pain. Rigors and fever may be present.

The *icteric* (jaundiced) *phase* usually begins 5 to 10 days after the onset of symptoms. It is heralded by jaundice of the sclera, skin and mucous membranes. Inflammation of the liver and bile ducts prevents bilirubin from being excreted into the small intestine. As a result, the serum bilirubin levels are elevated, causing yellowing of the skin and mucous membranes. Pruritus may develop due to deposition of bile salts on the skin. The stools are light brown or clay coloured because bile pigment is not excreted through the normal faecal pathway. Instead, the pigment is excreted by the kidneys, causing the urine to turn brown. Whereas people with acute hepatitis A or B are likely to develop jaundice, many people with hepatitis C do not develop jaundice. As a result, the infection may go undiagnosed for an extended period of time.

During the icteric phase, the initial prodromal manifestations usually diminish even though the serum bilirubin increases. The appetite increases and the temperature returns to normal. When uncomplicated, spontaneous recovery usually begins within 2 weeks of the onset of jaundice.

The *convalescent phase* follows jaundice and lasts several weeks. During this time, manifestations gradually improve: serum enzymes decrease, liver pain decreases and gastrointestinal symptoms and weakness subside. See the box below for the manifestations of each phase of hepatitis.

HEPATITIS A Hepatitis A, or *infectious hepatitis*, often occurs in either sporadic attacks or mild epidemics. It is transmitted by the faecal–oral route via contaminated food, water, shellfish and direct contact with an infected person. The virus has an incubation period of up to 4 weeks. Once jaundice develops, the amount of virus in the stool and the risk of spreading the disease decrease rapidly (Longo et al., 2012). Although hepatitis A usually has an abrupt onset, it is typically a benign and self-limiting disease with few long-term consequences. Symptoms last up to 2 months.

Focus on cultural diversity Hepatitis

Hepatitis is a significant issue for Indigenous Australians, particularly those in rural and remote communities. The infection rate for HAV, HBV and HCV is considerably higher in Indigenous people than in non-Indigenous people. The infection rate for HAV is exacerbated by poor sanitation, inadequate water supply and overcrowding, all of which are issues for many Indigenous communities (Thomson et al., 2008).

The Australian government has implemented vaccination programs in many states for HAV and HBV, with some success. However, there is a need for more culturally appropriate initiatives to decrease injecting drug rates among Indigenous people and to improve the quality of living conditions (Thomson et al., 2008).

FAST FACTS

- In Australia, hepatitis B and hepatitis C predominate.
- Notification rates of HBV and HCV have remained stable in recent years at just over 1 and just over 2 per 100 000 population, respectively.
- There has been a decline in new HBV infection rates in the 15- to 29-years age group in recent years, possibly due to vaccine programs.
- Injecting drug use accounted for 50% of new cases of HBV infection and heterosexual sex accounted for another 18%.
- Between 2004 and 2008 there was a decline in new HCV infection rates in the 15- to 39-years age group, possibly due to the decrease in intravenous drug use, one of the main causes of HCV transmission (Australian Institute of Health and Welfare (AIHW), 2010).

MANIFESTATIONS Acute hepatitis

PREICTERIC PHASE
- 'Flu-like' symptoms: malaise, fatigue, fever
- Gastrointestinal: anorexia, nausea, vomiting, diarrhoea, constipation
- Muscle aches, polyarthritis
- Mild right upper abdominal pain and tenderness

ICTERIC PHASE
- Jaundice
- Pruritus
- Clay-coloured stools
- Brown urine
- Decrease in preicteric phase symptoms (e.g. appetite improves; no fever)

POSTICTERIC/CONVALESCENT PHASE
- Serum bilirubin and enzymes return to normal levels
- Energy level increases
- Pain subsides
- Appetite returns

HEPATITIS B It is estimated that there are approximately 350 million chronic carriers of HBV worldwide (Hughes, Wedemeyer & Harrison, 2011). Hepatitis B can cause acute hepatitis, chronic hepatitis, *fulminant* (rapidly progressive) hepatitis or a carrier state. In a *carrier state*, the person harbours the active virus and is capable of spreading it to others, even though there are no discernible manifestations of the disease. This virus is spread through contact with infected blood and body fluids. Healthcare workers are at risk through exposure to blood and needle-stick injuries. Other high-risk groups for hepatitis B include intravenous drug users, people with multiple sex partners, men who have sex with men, and people frequently exposed to blood products (such as people on haemodialysis). Hepatitis B is a major risk factor for primary liver cancer.

The exact mechanism of liver injury by HBV is unclear: however, it is known that liver cells are damaged by the immune response to this antigen. The liver shows evidence of injury and scarring, regeneration and proliferation of inflammatory cells. Damage may affect only portions of the liver or the majority of the liver. During the prodromal period, people with HBV may experience nausea, vomiting, fatigue, malaise, muscle and joint pains, headache, photophobia and flu-like symptoms 1 to 2 weeks prior to the onset of jaundice (Longo et al., 2012).

HEPATITIS C It is estimated that up to 170 million people worldwide are infected with HCV (Casey & Lee, 2012; Longo et al., 2012). HCV is a blood-borne infection, transmitted through blood transfusion, sexual intercourse or intravenous drug use. Other at-risk populations include people with haemophilia treated with clotting factors prior to 1987, haemodialysis patients, children born to women with HCV and health workers following a needle-stick or splash injury (Longo et al., 2012).

Acute hepatitis C usually is asymptomatic; if symptoms do develop, they often are mild and non-specific. The disease often is recognised long after exposure occurred, when secondary effects of the disease develop. Out of every 100 people who contract HCV, 75–80% will develop chronic infection, 60–70% will develop chronic liver disease, 5–20% will develop cirrhosis and 1–5% will die from complications of the infection, including liver carcinoma. Worldwide, HCV is the leading cause of liver transplantation (Casey & Lee, 2012).

HEPATITIS DELTA It is estimated that more than 15 million people worldwide are infected with HDV. HDV can only occur in a person who also has the HBV infection; either by simultaneous transmission of the two viruses or via superinfection of an established HBV carrier (Hughes et al., 2011). Like HBV, HDV is transmitted through exposure to infected blood or body fluids. At-risk populations include intravenous drug users and people with high-risk sexual activity (Hughes et al., 2011). People with HBV and HDV co-infection are at greater risk of severe liver disease that progresses swiftly to liver cirrhosis, hepatic failure and death (Hughes et al., 2011).

HEPATITIS E Hepatitis E is rare in Australia. It is transmitted by faecal contamination of water supplies in developing areas such as South-East Asia, parts of Africa and Central America. It primarily affects young adults. It can cause fulminant, fatal hepatitis in pregnant women.

Chronic hepatitis

Chronic hepatitis is chronic infection of the liver. Although it may cause few symptoms, it is the primary cause of liver damage leading to cirrhosis, liver cancer and liver transplantation. Three of the known hepatitis viruses cause chronic hepatitis: HBV, HCV and HDV. Manifestations of chronic hepatitis include malaise, fatigue and hepatomegaly. Occasional icteric (jaundiced) periods may occur. Liver enzymes, particularly serum aminotransferase levels, typically are elevated.

In *chronic active hepatitis*, inflammation extends to involve entire hepatic lobules. Chronic active hepatitis usually leads to cirrhosis and end-stage liver failure.

Fulminant hepatitis

Fulminant hepatitis is a rapidly progressive disease, with liver failure developing within 2 to 3 weeks after the onset of symptoms. Although uncommon, it is usually related to HBV with concurrent HDV infection.

Toxic hepatitis

Many substances, including alcohol, certain drugs and other toxins, can directly damage liver cells. Alcoholic hepatitis can result from chronic alcohol abuse or from an acute toxic reaction to alcohol. Alcoholic hepatitis causes necrosis of hepatocytes and inflammation of the liver parenchyma (functional tissue). Unless alcohol intake is avoided, progression to cirrhosis is common.

Other potential hepatotoxins include paracetamol, benzene, carbon tetrachloride, halothane, chloroform and poisonous mushrooms. These substances directly damage liver cells, leading to necrosis. The degree of damage often depends on age and the extent of exposure to (dose of) the hepatotoxin. Paracetamol overdose is a common cause of hepatocellular damage.

Hepatobiliary hepatitis

Hepatobiliary hepatitis is due to cholestasis, the interruption of the normal flow of bile. Cholestasis may result from obstruction of the hepatic duct with stones or inflammation secondary to cholelithiasis. Other agents, such as oral contraceptives and allopurinol (a drug used to lower uric acid levels), also can cause cholestasis. When bile flow is disrupted, the liver parenchyma may become inflamed. Re-establishing bile flow by removing the stone or other causative agent is the treatment for hepatobiliary hepatitis.

INTERPROFESSIONAL CARE

Management of hepatitis focuses on determining its cause, providing appropriate treatment and support, and teaching strategies to prevent further liver damage. Effective management begins with thorough assessment of diagnostic and laboratory data.

Diagnosis

Liver function tests, such as blood levels of bilirubin and enzymes commonly released when liver cells are damaged, are obtained. These include the following:

- *Alanine aminotransferase (ALT)* is an enzyme contained within each liver cell. When liver cells are damaged, ALT is released into the blood. Levels may exceed 1000 U/L or more in acute hepatitis.
- *Aspartate aminotransferase (AST)* is an enzyme found predominantly in heart and liver cells. AST levels rise when liver cells are damaged; with severe damage, blood levels may be 20 to 100 times normal values.
- *Alkaline phosphatase (ALP)* is an enzyme present in liver cells and bone. Serum ALP levels often are elevated in hepatitis.
- *Gamma-glutamyltransferase (GGT)* is an enzyme present in cell membranes. Its blood levels rise in hepatitis and obstructive biliary disease and remain elevated until function is restored.
- *Lactic dehydrogenase (LDH)*, an enzyme present in many body tissues, is a non-specific indicator of tissue damage. Its isoenzyme, LDH5, is a specific indicator of liver damage.
- *Serum bilirubin* levels, including *conjugated* and *unconjugated*, are elevated in viral hepatitis due to impaired bilirubin metabolism and obstruction of the hepatobiliary ducts by inflammation and oedema. The bilirubin level decreases as inflammation and oedema subside.
- Laboratory tests for viral antigens and their specific antibodies may be done to identify the infecting virus and its state of activity.
- A *liver biopsy* may be done to detect and evaluate chronic hepatitis. (Nursing implications for this test are outlined in the 'Diagnostic tests' box in Chapter 20.)

Medications

PREVENTION Hepatitis A and hepatitis B are preventable diseases. Vaccines are available, as are preparations to prevent the disease following known or suspected exposure.

Vaccines Hepatitis A vaccine provides long-term protection against HAV infection. It is an inactivated whole-virus vaccine available in paediatric and adult formulations. Although more than 95% of adults achieve immunity after one dose of the vaccine, two doses are recommended for full protection. (See Table 24.3.)

Three doses of hepatitis B vaccine provide immunity to HBV infection in 90% of healthy adults. Because the hepatitis delta virus requires the presence of HBV, hepatitis B vaccine also protects against HDV. Hepatitis B vaccine is a recombinant vaccine. Vaccines produced by different manufacturers may be used interchangeably, although their dosages differ. Older adults are less likely to achieve immunity than younger adults. People on haemodialysis and people who are immunocompromised may need larger or more doses of the vaccine to achieve adequate protection. Serological testing for immunity is recommended on completion of the series for people in these high-risk groups.

A combined hepatitis A and hepatitis B vaccine is available for use. It is recommended for the same high-risk populations as the single vaccines. Three doses are given: the initial dose, followed by doses no sooner than 4 weeks and 6 months later.

Post-exposure prophylaxis Post-exposure prophylaxis may be recommended for household or sexual contacts of people with HAV or HBV and other people who are known to have been exposed to these viruses. It may also be necessary for health professionals who are not immunised against HBV post needle-stick injury. It is not necessary if the exposed person has been vaccinated and is known to be immune.

Hepatitis A prophylaxis is provided by a single dose of immune globulin (IG) given within 2 weeks after exposure. IG is recommended for all people with household or sexual contact with a person known to be infected with hepatitis A. See Table 24.3 for further recommendations.

Hepatitis B post-exposure prophylaxis is indicated for people exposed to the hepatitis B virus. Hepatitis B immune globulin (HBIG) is given to provide short-term immunity. HBV vaccine may be given concurrently. Candidates for post-exposure prophylaxis include those with known or suspected percutaneous or permucosal contact with infected blood, sexual partners of people with acute HBV or who are HBV carriers, and household contacts of people with acute HBV infection (Department of Health and Ageing, 2012).

Treatments

In most cases of acute viral hepatitis, pharmacological treatment of the infection is not indicated. Acute hepatitis C generally is treated with interferon alpha, an antiviral agent, to reduce the risk of chronic hepatitis C. While treatment with interferon alpha alone is common, it may be combined with the antiviral drug ribavirin (Virazide).

Interferon alpha is used to treat both chronic hepatitis B and chronic hepatitis C. Interferon alpha interferes with viral replication, reducing the viral load. It is given by intramuscular or subcutaneous injection. Virtually all people treated with interferon alpha develop a flu-like syndrome with fever, fatigue, muscle aches, headache and chills. Paracetamol helps alleviate

TABLE 24.3 Recommendations for hepatitis prevention in adults

DISEASE/ STRATEGY	IMMUNISATION	ADVERSE REACTIONS	POPULATION RECOMMENDATIONS
Hepatitis A			
Prevention	Hepatitis A vaccine (Havrix; VAQTA Adult), 2 doses (initial dose with booster in 6–12 months) given IM into deltoid muscle Combined hepatitis A and hepatitis B vaccine (Twinrix), 3 doses (initial dose followed by doses 1 and 6 months later) given IM into deltoid muscle	Pain at injection site	• All travellers to, and all expatriates living in, moderately to highly endemic areas • Indigenous Australian children residing in the Northern Territory, Queensland, South Australia and Western Australia • Those whose lifestyle may put them at risk of acquiring hepatitis A • People with occupational risk
Post-exposure prophylaxis	Standard immune globulin IM into large muscle mass within 2 weeks of exposure	Rare; risk of anaphylaxis in people with IgA deficiency	• Close contacts of people with known hepatitis A • People potentially exposed to hepatitis A at childcare centre or restaurant with infected food handler
Hepatitis B			
Prevention	Recombinant hepatitis B vaccine (Engerix-B Adult), 3 doses (initial dose followed by doses at 1 and 6 months later) given IM into deltoid muscle Combined hepatitis A and hepatitis B vaccine (Twinrix), 3 doses (initial dose followed by doses 4 weeks and 6 months later) given IM into deltoid muscle	Pain at injection site; fever, nausea, dizziness, malaise, myalgia and arthralgia	• Infants and adolescents • Household contacts of acute and chronic hepatitis B carriers • Men who have sex with men • Haemodialysis patients, HIV-positive individuals and other adults with impaired immunity • Prostitutes; heterosexuals with multiple sexual partners • People with an STD • Intravenous drug users • Long-term male prisoners • Healthcare workers
Post-exposure prophylaxis	Hepatitis B immune globulin (HBIG) given IM into large muscle mass within 72 hours of exposure.	Infrequent; muscle stiffness, pain, fever, nausea, dizziness, malaise, myalgia and arthralgia	• Infants born to women with HBV infection • Percutaneous or permucosal exposure to HBV when unvaccinated or antibody response is negative or unknown

Source: Department of Health and Ageing, Office of Health Protection (2013; updated 2014, 2015). *Australian Immunisation Handbook* (10th ed.). Used by permission of the Australian Government.

some of these adverse effects. Depression also is a common adverse effect of this drug.

An alternative drug for treating chronic hepatitis B is lamivudine (3TC, Zeffix), an antiviral drug that can reduce liver inflammation and fibrosis. Although it has minimal side effects, people may become resistant to the beneficial effects of lamivudine.

The treatment of choice for chronic hepatitis C is combination therapy of interferon alpha with ribavirin, an oral antiviral drug. This combination therapy improves the response rate over either drug used alone. Ribavirin has two major adverse effects: haemolytic anaemia and birth defects. Blood counts are obtained before and during treatment to detect early signs of haemolytic anaemia. Because of the risk of birth defects, this drug is contraindicated for use during pregnancy and two reliable methods of contraception must be used by women taking the drug and female sexual partners of men taking the drug.

Treatment of acute hepatitis also includes as-needed bed rest, adequate nutrition as tolerated and avoidance of strenuous activity, alcohol and agents that are toxic to the liver. In most cases, clinical recovery takes 3 to 16 weeks.

Nursing care

Health promotion

Nurses play an instrumental role in preventing the spread of hepatitis. Stress the importance of hygiene measures such as handwashing after toileting and before all food handling. Discuss the dangers of intravenous drug use and, with drug users, of sharing needles or other equipment. Encourage all sexually active people to use safer sexual practices such as mutual monogamy and barrier protection (such as male or female condoms).

Discuss recommendations for hepatitis A and hepatitis B vaccine with people in high- or moderate-risk groups for these infections. Ensure that nurses and other healthcare workers at risk of exposure to blood and body fluids are effectively vaccinated against hepatitis A and B. Encourage all people with known or probable exposure to HAV or HBV to obtain post-exposure prophylaxis.

Assessment

Collect assessment data related to hepatitis, such as the following:

- *Health history:* current manifestations, including anorexia, nausea, vomiting, abdominal discomfort, changes in bowel elimination or colour of stools; muscle or joint pain, fatigue; changes in colour of skin or sclera; duration of symptoms; known exposure to hepatitis; high-risk behaviours such as intravenous drug use or multiple sexual partners; previous history of liver disorders; current medications, prescription and over the counter.
- *Physical assessment:* vital signs including temperature; colour of sclera and mucous membranes; skin colour and condition; abdominal contour and tenderness; colour of stool and urine.
- *Diagnostic tests:* serum bilirubin, liver function tests, serological antibody–antigen levels.

Nursing diagnoses and interventions

People with acute or chronic hepatitis usually are treated in community settings; rarely is hospitalisation required. Nursing care focuses on preventing spread of the infection to others and promoting the person's comfort and ability to provide self-care.

Risk of infection (transmission)

An important goal when caring for people with acute viral hepatitis is preventing spread of the infection.

- Use standard precautions. Practise meticulous handwashing. The hepatitis viruses are spread by direct contact with faeces or blood and body fluids. *Standard precautions and good handwashing protect both healthcare workers and other people from exposure to the virus.*
- For people with HAV or HEV, use standard precautions and contact isolation if faecal incontinence is present. *The faecal–oral route is the primary mode of transmission of these viruses. Other hepatitis viruses are transmitted through blood and other body fluids.*
- Encourage prophylactic treatment of all members of household and intimate sexual contacts. *Prophylactic treatment of others in close contact with the person decreases their risk of contracting the disease or, if already infected, the severity of the disease.*

CONSIDERATION FOR PRACTICE

If the person diagnosed with hepatitis A is employed as a food handler or childcare worker, contact the local health department to report possible exposure of patrons. Maintain confidentiality. Prophylactic treatment of people who have possibly been exposed to the virus can prevent a local epidemic of the disease.

Fatigue

Fatigue and possible weakness are common in acute hepatitis. Although bed rest is rarely indicated, adequate rest periods and limitation of activities may be necessary. Many people with acute hepatitis may be unable to resume normal activity levels for 4 or more weeks.

- Encourage planned rest periods throughout the day. *Adequate rest is necessary for optimal immune function.*
- Assist to identify essential activities and those that can be deferred or delegated to others. *Identifying essential and non-essential activities promotes the person's sense of control.*
- Suggest using level of fatigue to determine activity level, with gradual resumption of activities as fatigue and sense of wellbeing improves. *Fatigue associated with activity is an indicator of appropriate and inappropriate activity levels. As recovery progresses, increasing activity levels are tolerated with less fatigue.*

Imbalanced nutrition: less than body requirements

Adequate nutrition is important for immune function and healing in people with acute or chronic hepatitis.

- Help plan a diet of appealing foods that provides a high-kilojoule intake of approximately 65 carbohydrate kilojoules per kilogram of ideal body weight. *Sufficient energy is required for healing; adequate carbohydrate intake can spare protein.*
- Encourage planning food intake according to symptoms of the disease. Discuss eating smaller meals and using between-meal snacks to maintain nutrient and kilojoule intake. *People with acute hepatitis often are more anorexic and nauseated in the afternoon and evening; planning the majority of kilojoule intake for in the morning helps maintain adequate intake. Limiting fat intake and the size of meals may reduce the incidence of nausea.*
- Instruct to avoid alcohol intake and diet drinks. *Alcohol avoidance is vital to prevent further liver damage and promote healing. Diet drinks (e.g. diet soft drinks or juice drinks) provide few kilojoules when an increased kilojoule intake is needed for healing.*
- Encourage use of nutritional supplements such as Ensure or instant breakfast drinks to maintain kilojoule and nutrient intake. *Nutritional supplement drinks are an additional source of concentrated kilojoules and nutrients.*

Disturbed body image

Jaundice and associated rashes and itching can affect the person's body image. Nursing measures to prevent skin breakdown and address body image are discussed in the following section on cirrhosis.

Community-based care

Provide discharge teaching to people and their families for home care. Include the following topics:

- recommended prophylactic treatment
- infection control measures such as frequent handwashing, not sharing eating utensils, avoiding food handling or preparation activities by the person with hepatitis A; abstaining from sexual relations during acute infection and using barrier protection if a carrier or for chronic infection
- managing fatigue and limited activity
- managing pruritus and maintaining skin integrity: use warm, not hot, water when bathing, use mild or no soap, limit duration of baths and showers; pat dry, do not rub, apply an alcohol-free lotion soon after bathing to retain skin moisture; wear loose cotton garments that allow moisture to evaporate from skin; reduce room temperature, especially at night, to prevent overheating; keep fingernails short and wear cotton mittens or gloves as needed to prevent scratching during sleep
- promoting nutrient intake
- avoiding hepatic toxins such as alcohol, paracetamol and selected other drugs; encourage to alert all care providers to presence of infection
- recommended follow up.

If chronic hepatitis B or C is being treated with medications, teach how to administer the drug, its dosing schedule, precautions and management of adverse effects. Stress the importance of keeping follow-up appointments, including recommended laboratory testing.

THE PERSON WITH CIRRHOSIS

Cirrhosis is the end-stage of chronic liver disease. It is a progressive, irreversible disorder, eventually leading to liver failure.

Alcoholic or Laënnec's cirrhosis is the most frequent cause of death related to long-term, high-risk alcohol consumption. Excessive alcohol consumption is one of the leading causes of morbidity and mortality in Australia (Liang et al., 2011). Cirrhosis also may result from chronic hepatitis B or C; prolonged obstruction of the biliary (bile drainage) system; long-term, severe right heart failure and other uncommon liver disorders. The incidence of and mortality attributable to cirrhosis and chronic liver disease vary significantly among populations.

Pathophysiology

In cirrhosis, functional liver tissue is gradually destroyed and replaced by fibrous scar tissue. As hepatocytes and liver lobules are destroyed, the metabolic functions of the liver are lost. Structurally abnormal nodules encircled by connective tissue form. This fibrous connective tissue forms constrictive bands that disrupt blood and bile flow within liver lobules. Blood no longer flows freely through the liver to the inferior vena cava. This restricted blood flow leads to portal hypertension (increased pressure in the portal venous system).

FAST FACTS

- Alcoholic liver disease was ranked 20th as a leading cause of death in Australia in 2013 (down from 17th in 2004) (Australian Bureau of Statistics, 2013).
- The mortality from alcoholic liver disease has significantly decreased in Australia from 1993 to 2005, possibly due to a significant increase in hospital admissions for alcoholic liver failure during the same period. Mortality rates for males have decreased by 21.7%, and for females by 8.3% (Liang et al., 2011).

Alcoholic cirrhosis

Alcoholic or Laënnec's cirrhosis is the end result of alcoholic liver disease. Its development is directly related to alcohol consumption: total amount of alcohol consumed, number of years of excessive alcohol consumption and blood alcohol levels. Cirrhosis mortality is less prevalent in women than men due to men generally consuming more alcohol than women and the percentage of men consuming large amounts of alcohol (Addolorato et al., 2009).

Alcohol causes metabolic changes in the liver: triglyceride and fatty acid synthesis increases and the formation and release of lipoproteins decrease, leading to fatty infiltration of hepatocytes (fatty liver). At this stage, abstinence from alcohol can allow the liver to heal. With continued alcohol abuse, the disease progresses. Inflammatory cells infiltrate the liver (alcoholic hepatitis), causing necrosis, fibrosis and destruction of functional liver tissue. In the final stage of alcoholic cirrhosis, regenerative nodules form and the liver shrinks and develops a nodular appearance. Malnutrition commonly accompanies alcoholic cirrhosis. See 'Pathophysiology illustrated: cirrhosis and portal hypertension' on the following pages.

Biliary cirrhosis

When bile flow is obstructed within the liver or in the biliary system, retained bile damages and destroys liver cells close to the interlobular bile ducts. This leads to inflammation, fibrosis and formation of regenerative nodules.

Post-hepatic cirrhosis

Advanced progressive liver disease resulting from chronic hepatitis B or C or from an unknown cause is known as post-hepatic or post-necrotic cirrhosis. The liver is shrunken and nodular, with extensive liver cell loss and fibrosis.

Manifestations and complications

Early in the course of cirrhosis, few manifestations may be present. The liver usually is enlarged and may be tender. Symptoms may be vague such as a dull, aching pain in the RUQ along with fever, nausea, vomiting, diarrhoea, anorexia and malaise (Bacon, 2012).

As the disease progresses, manifestations related to complications of liver cell failure and portal hypertension develop including

MANIFESTATIONS Cirrhosis with underlying cause

MANIFESTATION	UNDERLYING PATHOPHYSIOLOGY
Oedema, ascites	Impaired plasma protein synthesis (hypoalbuminaemia) Disrupted hormone balance and fluid retention Increased pressure in portal venous system
Bleeding, bruising	Decreased clotting factor synthesis Increased platelet destruction by enlarged spleen Impaired vitamin K absorption and storage
Oesophageal varices, haemorrhoids	Increased pressure in portal venous system with collateral vessel development
Gastritis, anorexia, diarrhoea	Engorged veins in gastrointestinal system Alcohol ingestion Impaired bile synthesis and fat absorption
Abdominal wall vein distension (caput medusae)	Portal hypertension
Jaundice	Impaired bilirubin metabolism and excretion
Malnutrition, muscle wasting	Impaired nutrient metabolism Impaired fat absorption Impaired hormone metabolism
Anaemia, leucopenia, increased risk of infection	Bleeding Increased blood cell destruction by spleen
Asterixis, encephalopathy	Accumulated metabolic toxins Impaired ammonia metabolism and excretion
Gynaecomastia, infertility, impotence	Altered sex hormone metabolism

ascites, oedema, gastrointestinal haemorrhage, jaundice or encephalopathy. Men may have decreased body hair, gynaecomastia and testicular atrophy; women may have dysmenorrhoea or amenorrhoea. Laboratory tests may show anaemia from chronic gastrointestinal bleeding, hypersplenism or nutritional deficiencies, total serum bilirubin may be elevated with advanced cirrhosis, clotting factors are prolonged and liver enzymes elevated (Bacon, 2012). See 'Multisystem effects of cirrhosis', page 803.

See the box above for selected manifestations of cirrhosis and their underlying pathophysiology.

PORTAL HYPERTENSION Portal hypertension causes blood to be rerouted to adjoining lower-pressure vessels. This *shunting* of blood involves collateral vessels. Affected veins, which become engorged and congested, are located in the oesophagus, rectum and abdomen. Portal hypertension increases the hydrostatic pressure in vessels of the portal system. Increased hydrostatic pressure in the capillaries pushes fluid out, contributing to ascites formation.

SPLENOMEGALY The spleen enlarges (splenomegaly) because portal hypertension causes blood to be shunted into the splenic vein. Splenomegaly increases the rate at which red and white blood cells and platelets are removed from circulation and destroyed. This increased blood cell destruction leads to anaemia, leucopenia and thrombocytopenia (Porth & Matfin, 2009).

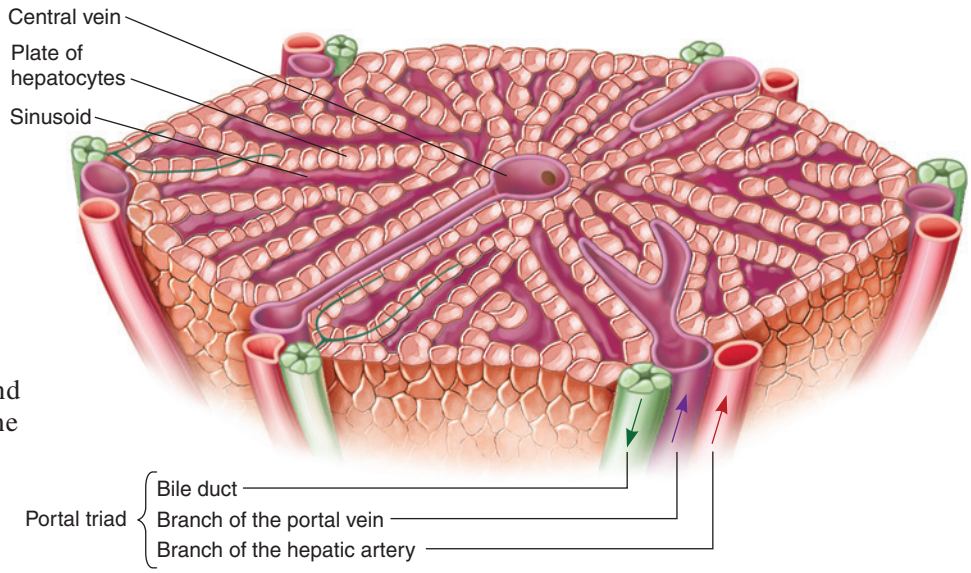
ASCITES Ascites is the accumulation of plasma-rich fluid in the abdominal cavity. Although portal hypertension is the primary cause of ascites, decreased serum proteins and increased aldosterone also contribute to the fluid accumulation. *Hypoalbuminaemia*, low serum albumin, decreases the colloidal osmotic pressure of plasma. This pressure normally holds fluid in the intravascular compartment; when plasma colloidal osmotic pressure decreases, fluid escapes into extravascular compartments. *Hyperaldosteronism*, an increase in aldosterone, causes sodium and water retention, contributing to ascites and generalised oedema.

OESOPHAGEAL VARICES **Oesophageal varices** are enlarged, thin-walled veins that form in the submucosa of the oesophagus. These collateral vessels form when blood is shunted from the portal system due to portal hypertension. The thin-walled varices may rupture, causing massive haemorrhage; even eating high-roughage foods can precipitate bleeding. Thrombocytopenia, platelet deficiency and impaired production of clotting factors by the liver contribute to the risk of haemorrhage.

Cirrhosis and portal hypertension

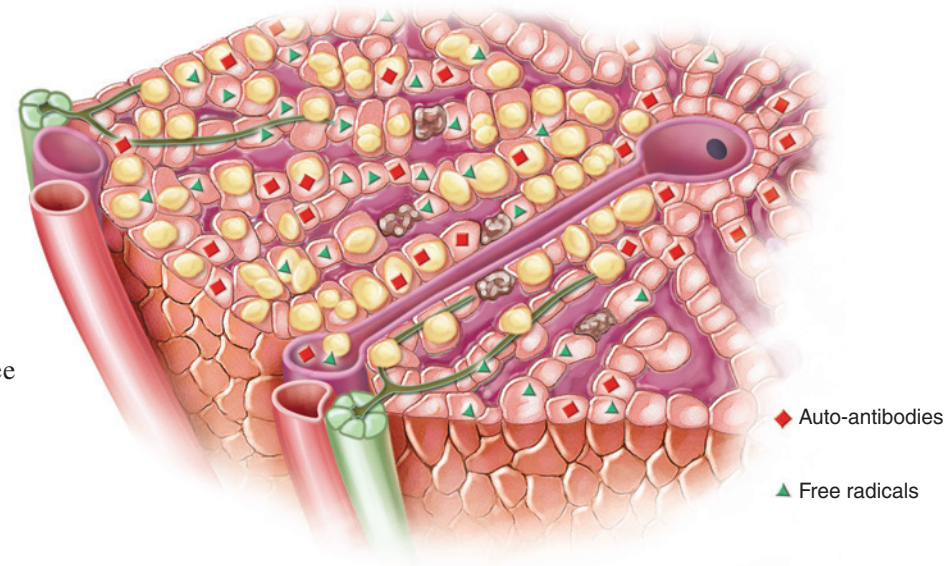
Normal liver

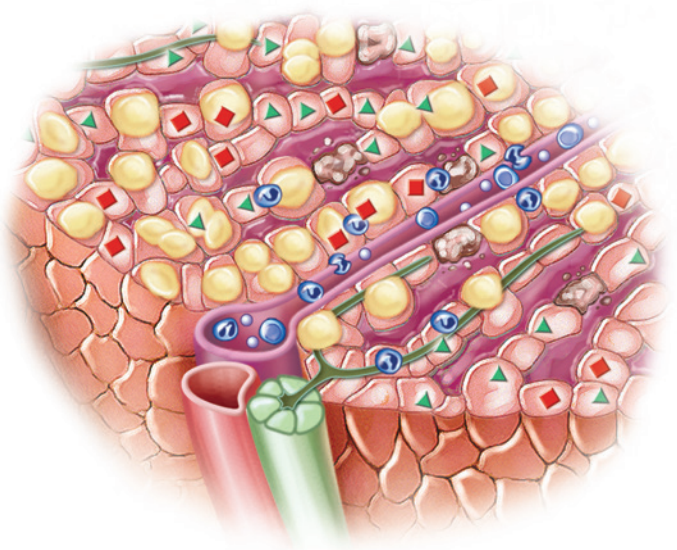
The liver contains multiple lobules made up of plates of hepatocytes, the functional cells of the liver, surrounded by small capillaries called sinusoids. These sinusoids receive a mixture of venous and arterial blood from branches of the portal vein and hepatic artery. Blood from the sinusoids drains into the central vein of the lobule. Hepatocytes produce bile, which drains outward to bile ducts.



Fatty liver

Ingested alcohol is primarily metabolised in the liver. Acetaldehyde, formed when alcohol is metabolised, damages hepatocytes and impairs the oxidation of fatty acids. As a result, fat accumulates within hepatocytes and liver lobules. Other alcohol metabolism by-products, including oxygen free radicals, promote inflammation and may stimulate autoantibody production.



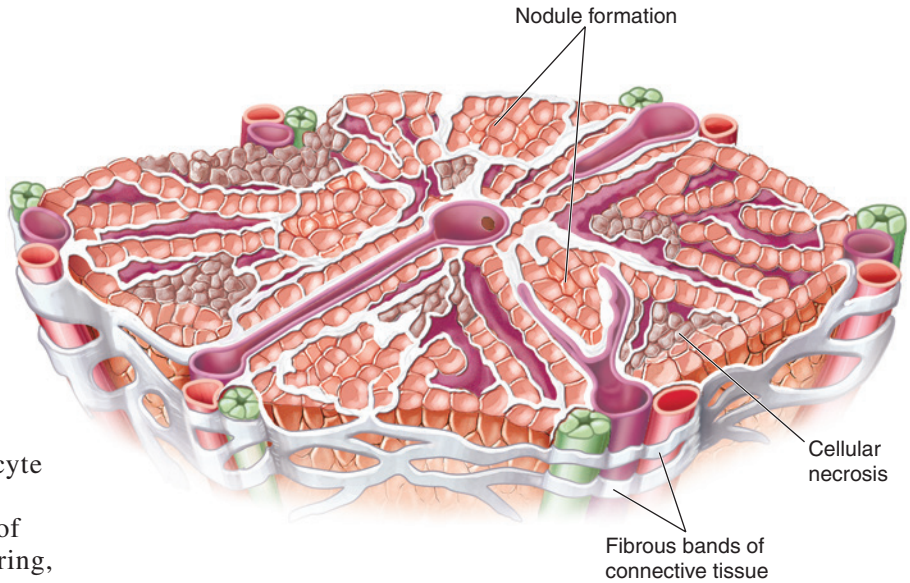


Alcoholic hepatitis

With continued alcohol intake, liver cells degenerate and spotty cellular necrosis occurs. Inflammatory cells such as polymorphonuclear leucocytes and lymphocytes infiltrate the lobule.

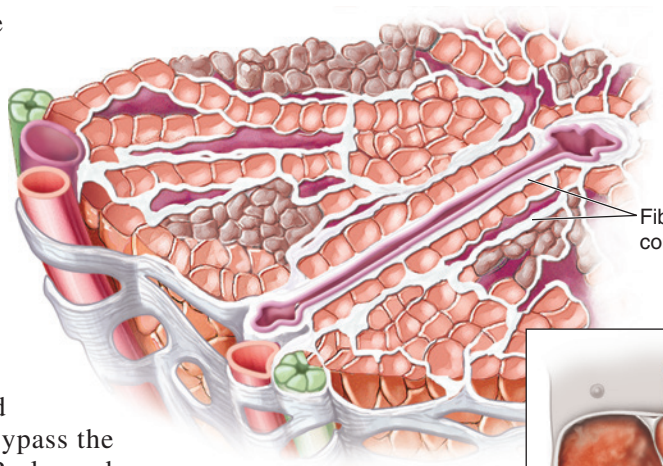
Alcoholic cirrhosis

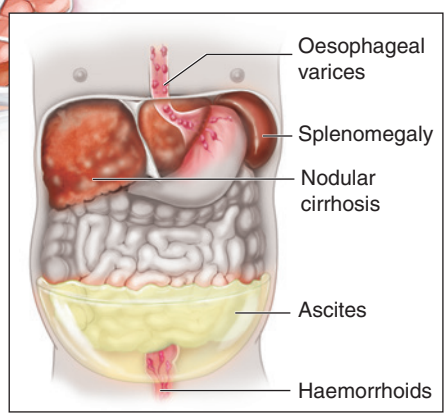
Cellular necrosis and inflammation transform some liver cells into fibroblasts that produce and deposit collagen. Web-like bands of connective tissue develop around the portal triads and central vein, eventually connecting with one another. Small islands of liver cells continue to regenerate, forming nodules. Hepatocyte destruction outpaces regeneration. As a result of cell loss, fibrosis and scarring, the liver shrinks and becomes hard and nodular.



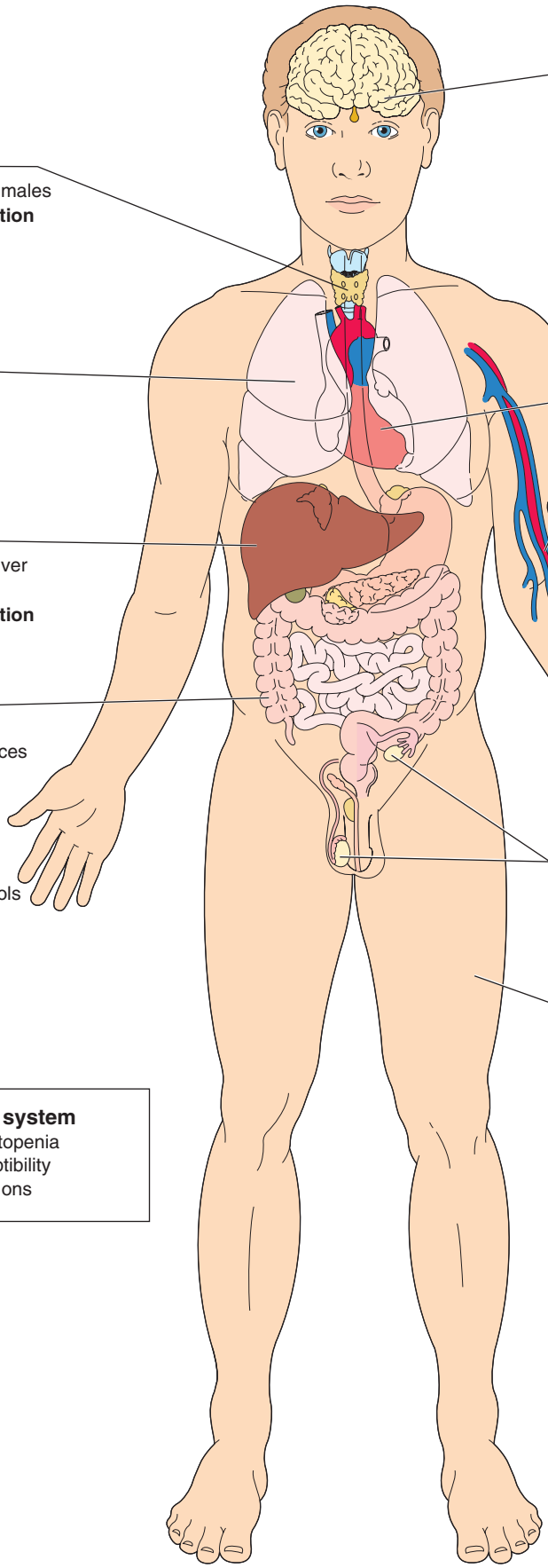
Portal hypertension

Bands of fibrotic scar tissue obstruct the sinusoids and blood flow from the portal vein to the hepatic vein. Pressure in the portal venous system, which drains the gastrointestinal tract, pancreas and spleen, increases. This increased pressure opens collateral vessels in the oesophagus, anterior abdominal wall and rectum, allowing blood to bypass the obstructed portal vessels. Prolonged portal hypertension leads to the development of (1) varices (fragile, distended veins) in the lower oesophagus, stomach and rectum; (2) splenomegaly (an enlarged spleen); (3) ascites (accumulation of fluid in the abdomen); and (4) hepatic encephalopathy (disrupted CNS function with altered consciousness).





MULTISYSTEM EFFECTS OF CIRRHOSIS



Neurological
- Portal systemic encephalopathy (agitation → lethargy → stupor → coma)
- Paraesthesias
- Sensory disturbances
- Asterixis ('liver flap')

Endocrine
- Gynaecomastia in males

Potential complication
- Diabetes mellitus

Respiratory
- Dyspnoea

Cardiovascular
- Bounding pulse
- Pulmonary hypertension
- Portal hypertension
- Arrhythmias

Hepatic
- Atrophic, nodular liver
- Splenomegaly

Potential complication
- Liver cancer

Haematological
- ↓ clotting factors
- Thrombocytopenia
- Anaemia

Potential complication
- Disseminated intravascular coagulation

Gastrointestinal

Oesophageal:
- Oesophageal varices

Stomach/intestines:
- Abdominal pain
- Anorexia
- Ascites
- Nausea
- Clay-coloured stools
- Peptic ulcers
- GI bleeding
- Haemorrhoids

Reproductive
- Oligomenorrhoea (female)
- Testicular atrophy (male)

Integumentary
- Jaundice (skin, sclera of eyes)
- Erythema of palms
- Spider angioma
- Decreased body hair
- Pruritis
- Ecchymoses
- Caput medusae (dilated veins around the umbilicus)

Immune system
- Leucocytopenia
- ↑ susceptibility to infections

Metabolic processes
- Fluid and electrolyte imbalances
  - Hypoalbuminaemia
  - Hypokalaemia
  - Hypocalcaemia
- Malnutrition
- Muscle wasting

PORTAL SYSTEMIC ENCEPHALOPATHY Portal systemic encephalopathy (*hepatic encephalopathy*) results from accumulation of neurotoxins in the blood and cerebral oedema. Ammonia, a by-product of protein metabolism, contributes to hepatic encephalopathy. Ammonium ion is produced as proteins and amino acids are broken down by bacteria in the intestinal tract. Normally, the ammonia produced is then converted by the liver to urea before entering the general circulation. As functional liver tissue is destroyed, ammonia can no longer be converted to urea and it accumulates in the blood. Other nervous system depressants, such as narcotics and tranquillisers, also can contribute to hepatic encephalopathy. Box 24.4 lists selected precipitating factors for hepatic encephalopathy. Accumulation of other metabolic toxins is thought to contribute as well.

Asterixis (liver flap), a muscle tremor that interferes with the ability to maintain a fixed position of the extremities and causes involuntary jerking movements, is an early sign of portal systemic encephalopathy. Asterixis primarily affects the upper extremities, but also may affect the tongue and feet. Asterixis is elicited by instructing the person to extend the arms and dorsiflex the wrists. If present, asterixis causes a downward flapping of the hands (see Figure 24.3). Changes in personality and mentation develop; agitation, restlessness, impaired judgment and slurred speech also are early manifestations of hepatic encephalopathy. As it progresses, confusion, disorientation and incoherence develop. Cerebral oedema that leads to increased intracranial pressure and cerebral hypoxia is the leading cause of death in people with portal systemic encephalopathy and liver failure.

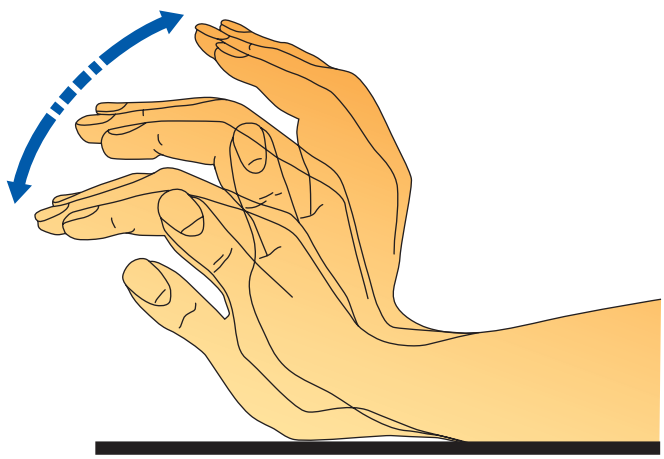


FIGURE 24.3 ■ Asterixis. Note the downward tremor of the hand on dorsiflexion of the wrist

BOX 24.4 Precipitating factors for hepatic encephalopathy

- High serum ammonia level
- Constipation
- Blood transfusions
- Gastrointestinal bleeding
- Medications: sedatives, tranquillisers, narcotic analgesics, anaesthetics
- Hypoxia
- High-protein diet
- Severe infection
- Surgery

HEPATORENAL SYNDROME Although the cause is unclear, renal failure with azotaemia (excess nitrogenous waste products in the blood), sodium retention, oliguria and hypotension may develop in people with advanced cirrhosis and ascites. Hepatorenal syndrome appears to be the result of imbalanced blood flow, leading to constriction of vessels leading to and within the kidneys. The syndrome may be precipitated by gastrointestinal bleeding, aggressive diuretic therapy or by an unknown cause.

SPONTANEOUS BACTERIAL PERITONITIS People with cirrhosis and ascites may develop bacterial peritonitis, even in the absence of known contamination of the peritoneal cavity or other specific risk factors (e.g. paracentesis). The inflammatory response to peritonitis worsens ascites by increasing the permeability of capillaries in the mesentery. The manifestations of spontaneous bacterial peritonitis may be subtle, with increased abdominal discomfort or pain, fever, increasing ascites, worsening encephalopathy and an overall decline in condition.

INTERPROFESSIONAL CARE

Care for the person with cirrhosis is holistic, addressing physiological, psychosocial and spiritual needs. The importance of including the family in the plan of care cannot be overemphasised, particularly if alcohol abuse is identified as the cause. Abstinence from alcohol is the cornerstone of treatment for alcoholic liver cirhossis (Bacon, 2012). Other treatment includes medications to help regulate protein metabolism, maintenance of fluid and electrolyte balance, and supportive therapies, including treatment of underlying problems such as malnutrition, anaemia, bleeding, encephalopathy, renal failure and infections.

Diagnosis

Studies to confirm the diagnosis of cirrhosis and identify its cause and effects are performed. Diagnostic tests may include the following:

- *Liver function studies* include *ALT*, *AST*, *ALP* and *GGT*. All may be elevated in cirrhosis, but usually not as severely as in acute hepatitis. Elevations in these enzymes may not correlate well with the extent of liver damage in cirrhosis.
- *FBC with platelets* is done. A low RBC count, haemoglobin and haematocrit demonstrate anaemia related to bone marrow suppression, increased RBC destruction, bleeding and deficiencies of folic acid and vitamin B_{12}. Platelets are low, related to increased destruction by the spleen. Leucopenia (low WBC count) also relates to splenomegaly.
- *Coagulation studies* show a prolonged prothrombin time due to impaired production of coagulation proteins and lack of vitamin K.

- *Serum electrolytes* are measured. Hyponatraemia is common, due to haemodilution. Hypokalaemia, hypophosphataemia and hypomagnesaemia also are frequently seen, related to malnutrition and altered renal excretion of these electrolytes.
- *Bilirubin* levels are usually elevated in severe cirrhosis, including both direct (conjugated) and indirect (unconjugated) bilirubin.
- *Serum albumin* levels show hypoalbuminaemia due to impaired liver production.
- *Serum ammonia* levels are elevated, because the liver fails to effectively convert ammonia to urea for renal excretion.
- *Serum glucose* and *cholesterol* levels frequently are abnormal in people with cirrhosis.
- *Abdominal ultrasound* is performed to evaluate liver size, detect ascites and identify liver nodules. Liver biopsy under ultrasound is useful to aid diagnosis; however, this is contraindicated in people who are still drinking alcohol (Bacon, 2012). Biopsy may be deferred if the bleeding time is prolonged (such as a prothrombin time (PT) greater than 3 seconds over the control). See the 'Diagnostic tests' box in Chapter 20 for nursing implications for a person having a liver biopsy. Figure 20.8 shows the site and position for liver biopsy.
- Endoscopy may be done to determine the presence of oesophageal varices.

More information about the above diagnostic tests and their nursing implications can be found in Chapter 20.

Medications

Medications are used to treat the complications and effects of cirrhosis; they do not reverse or slow the process of cirrhosis itself. Known hepatotoxic drugs and alcohol are avoided, as are drugs metabolised by the liver (e.g. barbiturates, sedatives, hypnotics and paracetamol). Several groups of drugs are commonly prescribed. See the 'Medication administration' box below for nursing responsibilities and teaching for commonly used drugs for people with cirrhosis.

- Diuretics reduce fluid retention and ascites. Spironolactone (Aldactone) is frequently the drug of first choice because it addresses one of the causes of ascites—increased aldosterone levels. If additional diuresis is necessary, a loop diuretic such as frusemide (Lasix) may be added to the regimen.
- Medications to reduce the nitrogenous load and lower serum ammonia levels are added when manifestations of hepatic encephalopathy develop. Two commonly administered medications are lactulose and neomycin. Both exert their effects locally, in the bowel. Lactulose reduces the number of ammonia-forming organisms in the bowel and increases the acidity of colon contents, converting ammonia into ammonium ion. Ammonium ion is not absorbable and is excreted in the faeces. Neomycin sulfate is a locally acting antibiotic that also reduces the number of ammonia-forming bacteria in the bowel.
- The beta-blocker nadolol (Corgard) may be given together with isosorbide mononitrate (Imdur, Monodur) to prevent rebleeding of oesophageal varices. This drug combination also lowers hepatic venous pressure.
- Ferrous sulfate and folic acid are given as indicated to treat anaemia. Vitamin K may be ordered to reduce the risk of bleeding. When bleeding is acute, packed RBCs, fresh frozen plasma or platelets may be administered to restore blood components and promote haemostasis.
- Antacids are prescribed as indicated. A drug regimen to treat *H. pylori* infection may also be effective (see Chapter 22).
- Oxazepam (Serepax), a benzodiazepine anti-anxiety/sedative drug, is not metabolised by the liver and may be used to treat acute agitation.

Treatments

Treatment of cirrhosis is supportive, directed at slowing the progression to liver failure and reducing complications.

NUTRITION Dietary support is an essential part of care for the person with cirrhosis. Dietary needs change as hepatic function fluctuates.

- Sodium intake is restricted to under 2 g/day and fluids are restricted as necessary to reduce ascites and generalised oedema. Fluids are often limited to 1500 mL/day. Fluid needs are calculated based on response to diuretic therapy, urine output and serum electrolyte values.
- Unless serum ammonia levels are high, a palatable diet with adequate kilojoules and protein is recommended. Previously protein was discouraged in people with encephalopathy; however, the negative impact of that restriction on overall nutrition is thought to outweigh the benefit (Bacon, 2012). The diet is high in kilojoules and includes moderate fat intake to promote healing. Parenteral nutrition is used as needed to maintain nutritional status when food intake is limited.
- Vitamin and mineral supplements are ordered based on laboratory values. Deficiencies in the B-complex vitamins, particularly thiamine, folate and B_{12}, and the fat-soluble vitamins A, D and E are common. These vitamins may need to be administered in a water-soluble form. People with alcohol-induced cirrhosis are at high risk of magnesium deficiency, which needs to be replaced.

COMPLICATION MANAGEMENT **Paracentesis**, aspiration of fluid from the peritoneal cavity, may be a diagnostic or a therapeutic procedure. It may be done therapeutically to relieve severe ascites that does not respond to diuretic therapy. The goal of paracentesis is to relieve respiratory distress caused by excess fluid in the abdomen. Ascites fluid may be withdrawn in moderate amounts of 500 mL to 1 L daily to reduce the risk of fluid and electrolyte imbalances. Large-volume paracentesis, withdrawal of 4 to 6 L of fluid at one time, may be used. Albumin is often administered intravenously during large-volume paracentesis to maintain intravascular volume as the pressure of the ascites fluid in the abdomen is relieved. Nursing implications for the person undergoing paracentesis are listed in Box 24.5. Figure 24.4 shows insertion sites and positioning during paracentesis.

Bleeding oesophageal varices are life threatening and require critical care management. Restoration of haemodynamic stability is the first priority. A central line is inserted and central venous and pulmonary artery pressures are monitored (see Chapter 30). Blood is given to restore blood volume, and fresh frozen plasma may be administered to restore clotting factors. Octreotide, a drug that constricts blood vessels in the

MEDICATION ADMINISTRATION The person with cirrhosis

DIURETICS

Spironolactone (Aldactone)
Frusemide (Lasix)

Spironolactone is a potassium-sparing diuretic that competes with aldosterone. It reduces ascites by increasing renal excretion of fluid and decreasing aldosterone levels. Frusemide is a loop diuretic that promotes the excretion of potassium. Drugs may be given in combination if serum potassium level permits.

Nursing responsibilities

- Monitor ECG, serum potassium, BUN, creatinine levels and hydration status.
- Weigh daily.
- Carefully monitor intake and output.
- Monitor for signs of hyperkalaemia if taking spironolactone alone: bradycardia; widening QRS, spiking T waves or ST segment depression on ECG; diarrhoea; and muscle twitching.
- Assess for hyponatraemia: confusion, lethargy, apprehension.

Health education for the person and family

- Maintain diet and fluid restrictions as prescribed.
- Report increases in weight or oedema.
- Immediately report signs of hyponatraemia, hyperkalaemia or hypokalaemia (see Chapter 9).
- Expect increased urinary output; take medications in morning hours to avoid nocturia.

LAXATIVES

Lactulose (Duphalac, Actilax)

Lactulose is a disaccharide laxative that is not absorbed by the gastrointestinal tract. It reduces the number of ammonia-producing bacteria and lowers the pH in the colon. The lower pH (increased acidity) converts ammonia to ammonium ion, a non-absorbable form that is excreted in the faeces. Lactulose also pulls water into the bowel lumen, increasing the number of daily stools.

Nursing responsibilities

- Assess bowel sounds and abdominal girth.
- Maintain accurate stool chart.
- Adjust dose to achieve two to four soft stools per day.
- Monitor electrolytes and hydration.

Health education for the person and family

- Drink adequate fluids.
- Report diarrhoea; if present, decrease dose. You should have an average of two to four stools per day.
- This drug may cause nausea. Continue taking the drug; taking it with crackers or a soft drink may reduce nausea.

ANTI-INFECTIVE AGENTS

Neomycin sulfate (Neosulf)

Neomycin sulfate is a non-systemic aminoglycoside antibiotic that reduces intestinal bacteria and decreases ammonia production in the bowel lumen. The drug may be administered as an oral or rectal preparation.

Nursing responsibilities

- Monitor hearing, renal and neurological functions. The drug is ototoxic, nephrotoxic and neurotoxic.
- Prior to administration, check for previous hypersensitivity reaction.
- Monitor intake and output.
- Monitor BUN and creatinine levels.
- If the person is taking digitalis, monitor levels; oral neomycin interferes with its absorption.

Health education for the person and family

- Report dizziness, tinnitus (ringing in ears), hearing loss, headaches, tremors or vision changes immediately.
- Keep follow-up appointments.
- Maintain fluids, avoid dehydration. (Teach signs of dehydration.)

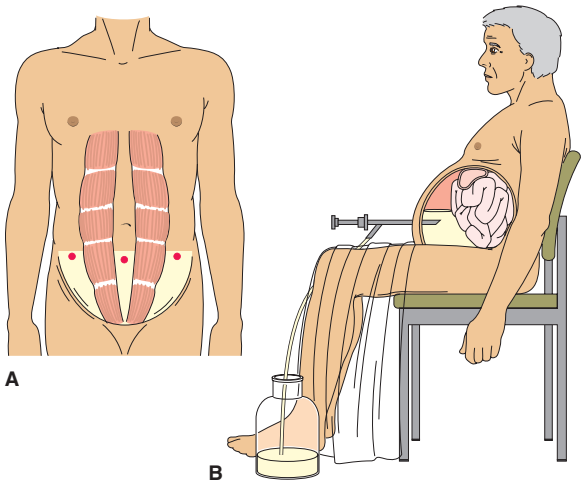


FIGURE 24.4 ■ Sites and position for paracentesis. *A*, Potential sites of needle or trocar insertion to avoid abdominal organ damage. *B*, The person sits comfortably; in this position, the intestines float back and away from the insertion site

gut, is given intravenously to reduce blood flow in the portal venous system. Vasopressin, a drug that produces generalised vasoconstriction, may also be used.

When the blood pressure and cardiac output have stabilised, upper endoscopy is performed to evaluate and treat the varices. During endoscopy, the varices may be banded or sclerosed to reduce the risk of recurrent bleeding. In *banding* (*variceal ligation*), small rubber bands are placed on varices to occlude blood flow. *Endoscopic sclerosis* involves injecting a sclerosing agent directly into the varices to induce inflammation and clotting. See Chapter 20 for the nursing implications of endoscopic investigation.

Balloon tamponade of bleeding varices may be used if bleeding cannot be controlled through vasoconstriction or if endoscopy is unavailable. A multiple-lumen nasogastric (NG) tube (such as a Sengstaken–Blakemore tube or a Minnesota tube) is inserted and the gastric and oesophageal balloons are inflated to apply direct pressure on the bleeding varices (see Figure 24.5). Tension is applied to the tube to further compress the varices. Balloon tamponade carries a number of risks,

BOX 24.5 Nursing implications for abdominal paracentesis

Preparation of the person

- Verify presence of an informed consent.
- Weigh prior to paracentesis.
- Assess vital signs for baseline.
- Have person void immediately prior to the test to avoid bladder puncture.
- Position seated, either on the side of the bed or in a chair, with feet supported.

Health education for the person and family

- Describe what to expect during and following paracentesis. Blood pressure is monitored during the procedure.
- The doctor will clean abdomen with Betadine and insert local anaesthesia. A small incision may be made and a needle or catheter is inserted to withdraw fluid. The needle is connected to tubing and a collection bottle; specimens may be sent to laboratory.
- A small dressing is placed over the puncture site after the needle is withdrawn. There may be some fluid leakage from the site.
- Salt-poor albumin may be given after the procedure to replace lost protein.

including aspiration, airway obstruction and tissue ischaemia and necrosis. An endotracheal tube is inserted prior to nasogastric intubation to support the airway and reduce the risk of aspiration. This short-term measure is used only until more definitive treatment can be done.

Transjugular intrahepatic portosystemic shunt (TIPS) is used to relieve portal hypertension and its complications of oesophageal varices and ascites. A channel is created through the liver tissue using a needle inserted transcutaneously (see Figure 24.6). An expandable metal stent is inserted into this channel to allow blood to flow directly from the portal vein into the hepatic vein, bypassing the cirrhotic liver. The shunt relieves pressure in oesophageal varices and allows better control of fluid retention with diuretic therapy. Stenosis and occlusion of the shunt are frequent complications. TIPS also increases the risk of developing hepatic encephalopathy (due to decreased perfusion of the liver and impaired ammonia metabolism) and may reduce long-term survival. It generally is used as a short-term measure until a liver transplant is performed.

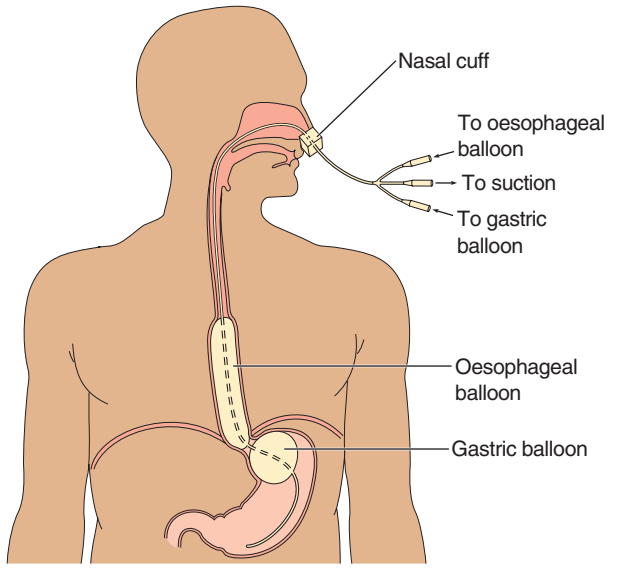


FIGURE 24.5 ■ Triple-lumen nasogastric tube (Sengstaken–Blakemore) used to control bleeding oesophageal varices

SURGERY **Liver transplantation** is indicated for some people with irreversible, progressive cirrhosis. A decline in functional status, increasing bilirubin levels, falling albumin levels and increasing problems with complications that respond poorly to treatment are indications for liver transplantation. Malignancy, active alcohol or drug abuse, and poor surgical risk are contraindications for the surgery. See the box below for nursing care of the person undergoing a liver transplant.

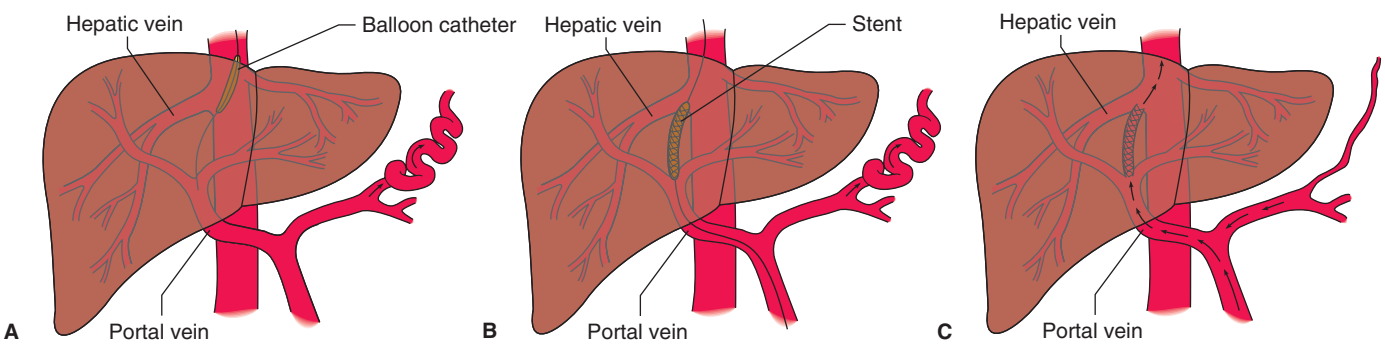
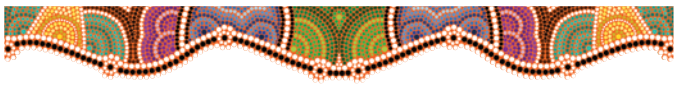


FIGURE 24.6 ■ Transjugular intrahepatic portosystemic shunt (TIPS). *A*, Guided by angiography, a balloon catheter inserted via the jugular vein is advanced to the hepatic veins and through the substance of the liver to create a portacaval (portal vein to vena cava) channel. *B*, A metal stent is positioned into the channel and expanded by inflating the balloon. *C*, The stent remains in place after the catheter is removed, creating a shunt for blood to flow directly from the portal vein into the hepatic vein



Nursing care

In addition to the nursing care discussed in this section, a nursing care plan for a person with alcoholic cirrhosis follows.

Health promotion

For most people, high-risk behaviours are the risk factors for cirrhosis. With all people (including children and young adults), stress the relationship between alcohol and drug abuse and liver disorders. While many people tolerate alcohol use in moderation with no adverse effects on the liver, excess alcohol use is the leading cause of cirrhosis. Injection drug use also is a significant risk factor, increasing the risk of contracting blood-borne hepatitis (B, C or D). These types of viral hepatitis can lead to chronic hepatitis and, ultimately, to cirrhosis. Discuss abstinence or safer sex practices as another measure to prevent viral hepatitis and potential liver damage.

Assessment

Assessment data related to cirrhosis include the following:

- *Health history:* current manifestations, including abdominal pain or discomfort, recent weight loss, weakness and anorexia; altered bowel elimination; excess bleeding or bruising; abdominal distension; jaundice, pruritus; altered libido or impotence; duration of symptoms; history of liver or gallbladder disease; pattern and extent of alcohol or injection drug use; use of other prescription and non-prescription drugs.
- *Physical assessment:* vital signs; mental status; colour and condition of skin and mucous membranes; peripheral pulses and presence of peripheral oedema; abdominal assessment including appearance, shape and contour, bowel sounds, abdominal girth, percussion for liver borders and palpation for tenderness and liver size.

Nursing diagnoses and interventions

Nursing care of the person with cirrhosis presents many challenges because liver function affects all body systems. The nurse is responsible for coordinating care among care providers. Many nursing diagnoses may apply. The diagnoses discussed in this section focus on problems with fluid and electrolyte balance, disturbed thought processes, risk of bleeding, skin integrity and nutrition.

Risk of excess fluid volume related to portal hypertension

Cirrhosis affects water and salt regulation due to portal hypertension, hypoalbuminaemia and hyperaldosteronism. Signs of fluid volume overload and portal hypertension may develop: ascites, peripheral oedema, internal haemorrhoids and varices, and prominent abdominal wall veins. Careful monitoring is necessary, because treatment measures can lead to further fluid and electrolyte imbalances.

- Weigh daily. Assess for jugular vein distension, measure abdominal girth daily and check for peripheral oedema. Monitor intake and output. *Careful assessment is important to detect fluid shifts.*
- Assess urine specific gravity. *Specific gravity measures the concentration of urine, an indicator of hydration.*
- Provide a low-sodium diet (500 to 2000 mg/day) and restrict fluids as ordered. *Excess sodium leads to water retention and can increase fluid volume, ascites and portal hypertension.*

CONSIDERATION FOR PRACTICE

Monitor the person with cirrhosis for signs of impaired renal function, such as oliguria, a fixed urine specific gravity of about 1.012, central oedema (around the eyes and of the face) and increasing serum creatinine and BUN levels. Such signs may indicate hepatorenal syndrome or acute renal failure from another cause.

Risk of disturbed thought processes due to hepatic encephalopathy

Accumulated nitrogenous waste products and other metabolites affect mental status and thought processes. Effects of hepatic encephalopathy can range from mild confusion to agitation to coma.

- Assess neurological status, including level of consciousness and mental status. Observe for signs of early encephalopathy: changes in handwriting, speech and asterixis. *Early identification of evidence of encephalopathy allows prompt intervention—subtle changes in neurological functioning are important!*
- Avoid factors that may precipitate hepatic encephalopathy. Avoid hepatotoxic medications and CNS-depressant drugs. *Cautious use of medications and close monitoring can eliminate iatrogenic causes of encephalopathy.*
- If possible, plan for consistent nursing care assignments. *Consistent care providers facilitate early identification of subtle neurological changes indicative of hepatic encephalopathy.*
- Administer medications or enemas as ordered to reduce nitrogenous products. Monitor bowel function and provide measures to promote regular elimination and prevent constipation. *Oral or rectally administered medications are ordered to reduce intestinal bacteria and the ammonia they produce. Regular bowel elimination promotes protein and ammonia elimination in the faeces.*
- Orientate to surroundings, person and place; provide simple explanations and reassurance. *Modification of verbal interactions to level of understanding and mental status may reduce anxiety and agitation.*

CONSIDERATION FOR PRACTICE

Closely monitor people who have experienced gastrointestinal bleeding for signs of hepatic encephalopathy. Blood in the intestinal tract is digested as a protein, increasing serum ammonia levels and the risk of hepatic encephalopathy.

NURSING CARE OF THE PERSON undergoing liver transplantation

PREOPERATIVE CARE

- Obtain a complete nursing history and physical examination. *A complete preoperative nursing assessment provides baseline data for comparison after surgery.*
- Provide routine preoperative care as ordered (see Chapter 3). *Preoperative care is similar to that provided for other people undergoing major surgery.*
- Discuss preoperative and postoperative expectations with the person and family. Introduce to the intensive care unit and discuss anticipated drainage tubes and supportive measures in the immediate postoperative period. Provide information about visiting policies and family accommodation (if available). *Preoperative teaching helps relieve anxiety in the person and family members. After a transplant people return from surgery to an intensive care or specialised care unit. Restrictions on the number of visitors and the time they may spend with the person are common.*
- Once a donor liver is located, check for evidence of infection; if no infection is present, begin preoperative antibiotics as ordered. *An acute or chronic infection may contraindicate liver transplantation as drugs given postoperatively to suppress rejection of the transplanted organ also impair the ability to fight infection.*

POSTOPERATIVE CARE

- Maintain airway and ventilatory support until awake and alert. *Until the new liver clears the anaesthesia, the person requires measures to support respirations and ventilation.*
- Monitor temperature and implement rewarming measures (such as warming blankets, heating lamps and head covers) as indicated. *The person often is hypothermic after liver transplant, necessitating careful rewarming while maintaining haemodynamic stability.*
- Frequently monitor haemodynamic pressures, including arterial blood pressure, central venous pressure and pulmonary artery pressures. *Postoperative fluid volume status may be difficult to determine without careful pressure measurements. The rate and type of fluids administered are determined by haemodynamic status.*
- Monitor urine output hourly; maintain careful intake and output records. Weigh daily. *Urine output and weight provide additional information about fluid volume status. In addition, renal function may be altered after liver transplant; acute renal failure is a significant risk. See Chapter 27 for more information about acute renal failure.*
- Monitor for signs of active bleeding, including excess drainage, increasing abdominal girth, bloody nasogastric drainage, black tarry stools, tachypnoea, tachycardia, diminished peripheral pulses or pallor. Report immediately. *Altered coagulation in the early postoperative period increases the risk of bleeding. Blood products to replace volume and clotting factors may be necessary.*
- Monitor serum electrolytes and laboratory values related to blood coagulation, liver function and renal function. Report abnormal results or significant changes immediately. *Electrolyte imbalances are common postoperatively. Altered liver or renal function tests may indicate rejection of the transplanted liver or acute renal failure. Other early signs of transplant rejection include fever, a drop in bile output or a change in bile colour and viscosity (Urden, Stacy & Lough, 2013).*
- Monitor neurological status. *With good function of the transplanted organ, mental status should clear within days of the transplant.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Teach how to reduce risk of infection and signs of infection to report.
- Instruct to recognise and report signs of organ rejection.
- Discuss all medications, including their purpose, schedule, adverse effects and potential long-term effects. Stress the importance of complying with all prescribed medications and postoperative precautions for the remainder of the person's life.
- Discuss possible changes in body image and psychological responses to receiving a transplanted organ. Refer to a social worker or support group as indicated.
- Refer for home health services for continued assessment and teaching.
- Stress importance of continued follow-up with transplant team and general practitioner.

Ineffective protection

Impaired coagulation, oesophageal varices and possible acute gastritis place the person with cirrhosis at significant risk of haemorrhage. Clotting is altered by vitamin K deficiency; impaired manufacture of coagulation factors II, VII, IX and X; and increased platelet destruction due to splenomegaly.

- Monitor vital signs; report tachycardia or hypotension. *Increased pulse and decreasing blood pressure may indicate hypovolaemia due to haemorrhage.*
- Institute bleeding precautions (see Box 24.6). *Preventive measures can decrease the risk of active bleeding.*
- Monitor coagulation studies and platelet count. *Report abnormal results. Coagulation studies help determine the risk of bleeding and the need for treatment.*

BOX 24.6 Bleeding precautions

- Prevent constipation.
- Avoid rectal temperatures or enemas.
- Avoid injections; if needed, use small-gauge needle and apply gentle pressure.
- Monitor platelet count, PT and APTT.
- Assess for bruised areas and areas of purpura.
- Apply pressure to bleeding sites. After venepuncture, apply direct pressure for at least 5 minutes.
- Use only a soft toothbrush.
- Avoid blowing nose.
- Assess oral cavity for bleeding gums.

NURSING CARE PLAN A person with alcoholic cirrhosis

Richard Wright is a 48-year-old divorced father of two teenagers. Mr Wright has been admitted to the community hospital with ascites and malnutrition. He has had three previous hospital stays for cirrhosis, the most recent 6 months ago.

ASSESSMENT

Mr Wright is lethargic but responds appropriately to verbal stimuli. He complains of 'spitting up blood the past week or so' and says, 'I'm just not hungry'. He has lost 9 kg since his previous admission. He is jaundiced and has petechiae and bruising on his arms and legs. Liz Mowdi, Mr Wright's nurse, notes pitting pre-tibial oedema. Abdominal assessment reveals a tight, protuberant abdomen with caput medusae. The liver margin is not palpable; the spleen is enlarged. Vital signs are T 37.7°C, P 110, R 24 and BP 110/70.

Abnormal laboratory results include WBC 3700/mm^3 (normal 4300 to 10 800/mm^3); RBC 4.0 million/mm^3 (normal 4.6 to 5.9 million/mm^3); platelets 75 000/mm^3 (normal 150 000 to 350 000/mm^3); serum ammonia 105 µm/dL (normal 35 to 65 µm/dL); total bilirubin 4.9 mcg/dL (normal 0.1 to 1.0 mcg/dL); and serum sodium 150 mEq/L (normal 135 to 145 mEq/L). Potassium, haemoglobin, haematocrit, total protein and albumin levels are markedly decreased. Liver enzymes are elevated. Blood urea nitrogen and creatinine levels are marginally elevated. Oxygen saturation (O_2 sat) is 88% (normal range: 96% to 100%) per pulse oximetry.

Endoscopy shows bleeding from gastric ulcer and the diagnosis of alcoholic cirrhosis with gastritis is made. Mr Wright is started on spironolactone, 25 mg PO q8h; lactulose, 30 mL prn until onset of diarrhoea, then 15 mL tds; and low-sodium diet; fluid restriction of 1500 mL/day.

DIAGNOSES

- *Risk of impaired gas exchange* related to pressure of ascites fluid on the diaphragm as manifested by tachypnoea and decreased oxygen saturation.
- *Risk of excess fluid volume* related to electrolyte imbalance and hypoalbuminaemia as manifested by ascites and peripheral oedema.
- *Risk of imbalanced nutrition: less than body requirements* related to anorexia and possible alcohol abuse as manifested by weight loss and low serum protein levels.
- *Risk of disturbed thought processes* related to effects of high ammonia levels as manifested by lethargy.
- *Risk of ineffective protection* related to impaired platelet formation and malnutrition.

PLANNING

- Teach the importance of activity, turning, coughing and deep breathing prior to the procedure.
- Encourage Mr Wright's family to visit at mealtimes to encourage oral intake.
- Ensure buzzer is kept by bedside at all times and encourage Mr Wright to alert nursing staff immediately if he experiences any signs of bleeding such as haematemesis or dizziness.
- Arrange for social work review for referral to community support services.

Expected outcomes

- Respiratory rate and SpO_2 will be within normal limits.
- Abdominal girth will decrease by 1 to 2 cm per day; peripheral oedema will decrease.
- Will gain 0.5 kg per week without evidence of increased fluid retention. Serum albumin levels will return to normal range.
- Will be alert and oriented; serum ammonia levels are within normal range.
- Will demonstrate no further evidence of active bleeding.
- Will verbalise willingness to join a community support group.

IMPLEMENTATION

- Weigh daily.
- Provide high-kilojoule, low-salt, low-protein diet with between-meal snacks.
- Maintain stool chart.
- Assign same nurses to care as much as possible to facilitate evaluation of mental status. Promptly report changes in status or laboratory values.
- Measure abdominal girth every 8 hours, marking level of measurement.
- Institute bleeding precautions.
- Elevate head of bed; assist to chair with legs elevated as tolerated. When resting in bed encourage to turn, cough and deep breathe every 2 to 4 hours.
- Include significant others in care and teaching.

EVALUATION

A week after admission, Mr Wright's ascites has decreased and no further active bleeding is noted. His serum protein levels have increased and his laboratory values are improving. No further bruising is noted during hospitalisation. Although he shows a 2 kg weight loss as excess water is eliminated, he is consuming 100% of his diet. His serum ammonia levels have returned to normal. On discharge, SpO_2 is 96%; respirations are 18. Lactulose will be continued on discharge.

Ms Mowdi provides both written and verbal information about the medication and cirrhosis, including measures to prevent complications. Mr Wright and his children express interest in Alcoholics Anonymous and are referred to this agency. Prior to discharge, follow-up appointments are made with a mental health team, social worker and a general practitioner.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the relationship between portal hypertension, liver dysfunction and ascites.
2. What is the pathophysiological basis for hepatic encephalopathy? What are the nursing responsibilities related to lactulose and neomycin?
3. Design a nursing care plan for Mr Wright for the diagnosis *Ineffective coping*.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Mr Wright confides to you that he is worried about whether he will be able to completely abstain from alcohol. Reflect on how you would respond.

- Carefully monitor the person who has had bleeding oesophageal varices for evidence of rebleeding: haematemesis, haematochezia (bright blood in the stool) or tarry stools, signs of hypovolaemia or shock. *Rebleeding is common following variceal haemorrhage, especially within the first week.*

Impaired skin integrity

Severe jaundice with bile salt deposits on the skin may cause pruritus. Scratching related to the pruritus damages the skin and impairs its integrity. Malnutrition, particularly protein deficiency, and oedema also increase the risk of tissue breakdown and impaired skin integrity.

- Use warm water rather than hot water when bathing. *Hot water increases pruritus.*
- Use measures to prevent dry skin: apply an emollient or lubricant as needed to keep skin moist, avoid soap or preparations with alcohol, and do not rub the skin. *Dry skin contributes to pruritus.*
- If indicated, apply gloves to prevent scratching. *People with encephalopathy may not understand the need to refrain from scratching.*
- Institute measures to prevent skin and tissue breakdown: turn at least every 2 hours, use an alternating-pressure mattress and frequently assess skin condition. *Frequent position changes relieve pressure and promote circulation and tissue oxygenation.*
- Administer prescribed antihistamine (to relieve pruritus) cautiously. *Decreased liver function increases the risk of altered drug responses.*

Imbalanced nutrition: less than body requirements

The person with cirrhosis is at risk of malnutrition for a number of reasons: possible chronic alcohol use, anorexia, impaired vitamin and mineral absorption, and impaired protein metabolism. In addition, salt restrictions may make the diet less palatable and appealing to the person.

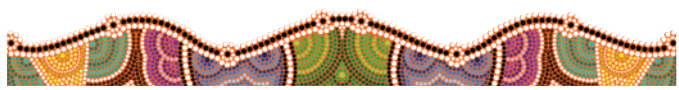
- Weigh daily. Instruct to weigh at least weekly at home. *Weight is a good indicator of both nutritional status and fluid balance. Short-term weight fluctuations tend to reflect fluid balance, while longer-term changes in weight are more reflective of nutritional status.*
- Provide small meals with between-meal snacks. *A small meal is more appealing for an anorexic person. Between-meal snacks help to maintain adequate kilojoule and nutrient intake.*
- Unless protein is restricted due to impending hepatic encephalopathy, promote protein and nutrient intake by providing nutritional supplements such as Ensure or instant breakfasts. *The sodium and protein content of all meals and snacks must be calculated when maintaining restrictions of these nutrients.*
- Arrange for consultation with a dietitian for diet planning while hospitalised and at home. *The dietitian can provide detailed instructions, sample menus and suggestions for improving the palatability of the diet and promoting intake.*

Community-based care

Cirrhosis is a chronic, progressive disease. As such, the person and family assume major roles in managing the disease and its manifestations, and in preventing complications. Teaching topics for home care include:

- The absolute necessity of avoiding alcohol and other hepatotoxic drugs. Suggest inpatient or community-based alcohol treatment programs and Alcoholics Anonymous as indicated.
- Diet and fluid intake restrictions and recommendations. Include suggestions to promote nutritional intake and increase the flavour of food when sodium is restricted.
- Prescribed medications; their timing, intended and adverse effects; and manifestations to report to the primary care provider.
- Bleeding precautions (see Box 24.6).
- Manifestations of potential complications to be reported to the primary care provider. Stress the importance of promptly reporting evidence of gastrointestinal bleeding for prompt intervention for potential haemorrhage.
- Skin care techniques to reduce pruritus and the risk of damage.
- Ways to manage fatigue and conserve energy.

Provide referrals for home health services, dietary consultation, social services and counselling as needed by the person and family. Suggest local support groups where available. If appropriate, suggest hospice services for the person with end-stage liver disease.



THE PERSON WITH CANCER OF THE LIVER

Primary liver cancer is a common malignancy worldwide although it only accounts for 1.2% of all cancers in Australia (AIHW, 2011; Carr, 2012). Hepatocellular carcinoma is common in parts of Asia and Africa which are associated with high endemic hepatitis B carrier rates (Carr, 2012). The incidence of primary liver cancer is three times higher in Australian men than it is in Australian women (AIHW, 2011). The prognosis for primary liver cancer is poor, in part because the disease is often advanced at the time of diagnosis. Metastasis to the liver from primary tumours of the lung, breast and gastrointestinal tract is relatively common.

Pathophysiology

About 80–90% of primary hepatic cancers arise from the liver's parenchymal cells (hepatocellular carcinoma); the remainder form in the bile ducts (cholangiocarcinoma). Regardless of the origin, the progress of the disease is similar. Several aetiological factors have been identified (see Box 24.7). Most primary liver cancer in the Australia is related to alcoholic cirrhosis, HBV or HCV.

BOX 24.7 Suspected causes of primary liver cancer

- Chronic hepatitis C infection
- Chronic hepatitis B infection
- Cirrhosis, regardless of type
- Chronic aflatoxin (a toxin produced by *Aspergillus* moulds) exposure
- Arsenic-contaminated water
- Carcinogens in food
- Possible hormonal factors (e.g. long-term use of androgens)

The underlying pathophysiology of primary liver cancer is damage to hepatocellular DNA. This damage may be caused by integration of HBV or HCV into the DNA or by repeated cycles of cell necrosis and regeneration that facilitate DNA mutations. HBV and aflatoxins damage a specific tumour suppressor gene, p53. Tumours may be limited to one specific area, may occur as nodules throughout the liver or may develop as surface infiltrates. The tumour interferes with normal hepatic function, leading to biliary obstruction and jaundice, portal hypertension and metabolic disruptions (hypoalbuminaemia, hypoglycaemia and bleeding disorders). It also may secrete bile products and produce hormones (paraneoplastic syndrome) that may lead to polycythaemia, hypoglycaemia and hypercalcaemia. Tumours usually grow rapidly and metastasise early.

Manifestations

Initial manifestations of liver cancer develop insidiously and often are masked by the presence of cirrhosis or chronic hepatitis. Weakness, anorexia, weight loss, fatigue and malaise are common early manifestations. Abdominal pain and a palpable mass in the right upper quadrant are common presenting symptoms. See the box below for manifestations of primary liver cancer. Ascites and jaundice may be present at diagnosis. Signs of liver failure with portal hypertension, splenomegaly and altered metabolism develop as the tumour progresses. People with advanced tumours have a median survival of 4 months, with or without treatment (Carr, 2012).

MANIFESTATIONS Primary liver cancer

- Malaise
- Anorexia
- Lethargy
- Weight loss
- Fever of unknown origin
- Jaundice
- Feeling of abdominal fullness
- Painful right upper quadrant mass
- Manifestations of liver failure

INTERPROFESSIONAL CARE

Liver tumours are identified by CT scans and MRI. A liver biopsy may be done to confirm the diagnosis and identify the tumour type or origin. See the 'Diagnostic tests' box in Chapter 20 for the nursing implications of liver biopsy. Serum alpha-fetoprotein (AFP) levels, normally low in non-pregnant adults, rise in most people with hepatocellular cancer.

Small, localised tumours may be surgically resected, offering the only viable chance for cure. Most tumours, however, have spread extensively or have distant metastasis at the time of diagnosis, so this is frequently not an option. Liver transplantation may be done; however, the tumour may recur in the transplanted organ.

Radiation therapy is used to shrink the tumour, decreasing pressure on surrounding organs and reducing pain. Chemotherapy may be used as primary treatment or adjunctive therapy. Direct continuous hepatic arterial infusion with an implanted pump has shown promise in prolonging survival rates. See Chapter 13 for nursing care for people receiving radiation therapy or chemotherapy.

Nursing care

Encourage people with risk factors for primary liver cancer to avoid alcohol and other substances that may further damage the liver. Urge them to discuss regular screening for liver tumours (such as serum AFP levels) with their general practitioner.

Both the person and the family need extensive nursing support. Controlling pain is a priority. Because of the poor prognosis, early referral for hospice services may be appropriate.

Nursing diagnoses, interventions and teaching for the person with liver cancer are similar to those for people with cirrhosis; see above.

THE PERSON WITH LIVER TRAUMA

Blunt or penetrating trauma to the abdomen can damage the liver. Liver trauma is frequently seen in combination with injuries to other abdominal organs. Motor vehicle crashes, stab or gunshot wounds, and iatrogenic sources such as liver biopsy are among the causes of these injuries.

Pathophysiology and manifestations

Liver trauma generally causes bleeding due to the vascularity of the organ. Liver injury may cause a surface haematoma, a haematoma within the liver parenchyma, laceration of liver

tissue or disruption of vessels leading to or from the liver. Severe bleeding can rapidly disrupt haemodynamic stability and lead to shock.

CONSIDERATION FOR PRACTICE

Bleeding due to liver trauma may not be immediately apparent. Instruct the person with apparent or potential liver trauma to immediately report light headedness, rapid heart rate, shortness of breath, thirst or increasing abdominal pain.

INTERPROFESSIONAL CARE

Diagnostic peritoneal lavage (DPL) may be used along with CT scan to diagnose liver trauma. The procedure is performed by making a small abdominal incision into the peritoneum (after the bladder has been emptied) and inserting a small catheter into the peritoneal cavity. If blood is immediately detected, the person is taken directly to surgery for abdominal exploration or transferred to a trauma facility where this surgery can occur. If frank bleeding is not apparent, a litre of isotonic fluid is instilled into the abdomen, then drained and sent for laboratory analysis.

A FAST (focused abdominal sonography in trauma) scan is more frequently used to identify free intraperitoneal fluid associated with liver injuries from blunt trauma. Unlike DPL, FAST scans are non-invasive, quick, and pose no risk to the person. DPL carries a 1–9.5% risk to the person, including perforation of a viscus, vascular laceration and wound complications (Christie-Large, Michaelides & James, 2008).

Intravenous fluids, fresh frozen plasma, platelets and other clotting factors are administered to restore blood volume and promote haemostasis. Haemodynamic status is closely monitored; continued instability may indicate a need for surgical intervention to control haemorrhage. Postoperative nursing care focuses on preventing pulmonary complications, such as atelectasis, and detecting and preventing infection.

Nursing care

Nursing care of the person with liver trauma focuses on fluid management and other supportive care related to shock. Keeping family members informed is an important aspect of care, especially during the period of haemodynamic instability. Diagnoses include the following:

- *Deficient fluid volume* related to haemorrhage.
- *Risk of infection* related to wound or abdominal contamination.
- *Ineffective protection* related to impaired coagulation.

THE PERSON WITH LIVER ABSCESS

Liver abscesses usually are bacterial or amoebic (protozoal) in origin. Bacterial abscesses may follow trauma or surgical procedures, including biopsy. Multiple or single abscesses occur most commonly in the right lobe. Amoebic abscesses most frequently occur following infestation of the liver by *Entamoeba histolytica*. Amoebic infestation is associated with poor hygiene, unsafe sexual practices or travel in areas where drinking water is contaminated.

Pathophysiology and manifestations

Following bacterial or amoebic invasion of the liver, healthy tissue is destroyed, leaving an area of necrosis, inflammatory exudate and blood. This damaged region becomes walled off from the healthy liver tissue. Pyogenic (bacterial) liver abscess may be caused by cholangitis or distant or intra-abdominal infections, such as peritonitis or diverticulitis. *Escherichia coli* is the most frequently identified causative organism. The onset of pyogenic abscess is usually sudden, causing acute symptoms such as fever, malaise, vomiting, hyperbilirubinaemia and pain in the right upper abdomen.

The infection pathway for amoebic hepatic abscesses usually is the portal venous circulation from the right colon. Generally, the onset of amoebic abscess is insidious.

INTERPROFESSIONAL CARE

Hepatic abscess is diagnosed through biopsy, hepatic aspirate, blood and faecal cultures, and CT scan and ultrasound studies. Therapy is based on identifying the causative organism through laboratory cultures. Pyogenic abscesses are treated with antibiotics to which the causative organism is sensitive.

Pharmacological agents used for amoebic hepatic abscess are the same as those used for intestinal amoebic infestation (see Chapter 23); combination therapy is commonly used. Two commonly used drugs for treating amoebic liver abscesses are metronidazole (Flagyl) and iodoquinol (Diquinol). Both medications can cause gastrointestinal symptoms. Bone marrow suppression is a risk with metronidazole.

If the abscess does not respond to antibiotic therapy, percutaneous aspiration or surgical drainage may be done. In these procedures, a *percutaneous closed-catheter drain* is placed in the abscess to promote drainage of purulent material.

Nursing care

A major aspect of nursing care is prevention; teaching people to avoid contaminated water and foods is especially important. Nursing interventions include teaching hikers to treat water and food handlers to wash hands thoroughly.

People who have a liver abscess require supportive care to prevent dehydration from the accompanying fever, nausea, vomiting and anorexia. Careful monitoring of fluid and electrolyte status is indicated, as are comfort measures for abdominal pain. Possible nursing diagnoses include the following:

- *Risk of deficient fluid volume* related to effects of prolonged fever and vomiting.
- *Deficient knowledge* related to transmission of amoebic abscess.
- *Activity intolerance* related to pain and weakness.

EXOCRINE PANCREAS DISORDERS

The pancreas is both an exocrine and an endocrine gland. It is made up of two basic cell types, each having different functions. The exocrine cells produce enzymes that empty through ducts into the small intestine, whereas the endocrine cells produce hormones that enter the bloodstream directly. Disorders of the exocrine pancreas affect the secretion and glandular control of digestive enzymes, whereas disorders of the endocrine pancreas affect the production of hormones necessary for normal carbohydrate, protein and fat metabolism. Disorders of the exocrine pancreas are discussed in this section of the chapter; diabetes mellitus, a disorder of the endocrine pancreas, is discussed in Chapter 19.

THE PERSON WITH PANCREATITIS

Pancreatitis, or inflammation of the pancreas, is characterised by release of pancreatic enzymes into the tissue of the pancreas itself, leading to haemorrhage and necrosis. Pancreatitis may be either acute or chronic.

Gallstones and chronic alcohol abuse account for 80% of acute pancreatitis causes (Nesvaderani, Eslick & Cox, 2015). The incidence of acute pancreatitis varies in different countries depending on the cause. In Australia, the estimated incidence is around 70 cases per 100 000 people.

The incidence of chronic pancreatitis is less clear, because many people with chronic pancreatitis do not have classic manifestations of the disease. People with pancreatitis may have long-term effects of the disease, with chronic changes in enzyme and hormone production.

Physiology review

Knowledge of the normal structure and functions of the exocrine pancreas is important to understand how inflammation affects it and the person. The exocrine pancreas consists of lobules of acinar cells. The acinar cells secrete digestive enzymes and fluids (pancreatic juices) into ducts that empty into the main pancreatic duct (the duct of Wirsung). The pancreatic duct joins the common bile duct and empties into the duodenum through the ampulla of Vater. (In some people the main pancreatic duct empties directly into the duodenum.) The epithelial lining of the pancreatic ducts secretes water and bicarbonate to modify the composition of the pancreatic secretions. Pancreatic enzymes are secreted primarily in an inactive form and are activated in the intestine, a modification that prevents digestion of pancreatic tissue by its own enzymes (Porth & Matfin, 2009). The pancreatic enzymes, with related functions, are as follows:

- proteolytic enzymes, including trypsin, chymotrypsin, carboxypolypeptidase, ribonuclease and deoxyribonuclease, which break down dietary proteins
- pancreatic amylase, which breaks down starch
- lipase, which breaks down fats into glycerol and fatty acids.

Pathophysiology

Acute pancreatitis

Acute pancreatitis is an inflammatory disorder that involves self-destruction of the pancreas by its own enzymes through autodigestion. The milder form of acute pancreatitis, *interstitial oedematous pancreatitis*, leads to inflammation and oedema of pancreatic tissue. It often is self-limiting. The more severe form, *necrotising pancreatitis*, is characterised by inflammation, haemorrhage and, ultimately, necrosis of pancreatic tissue.

Acute pancreatitis is more common in middle adults; its incidence is higher in men than in women. Acute pancreatitis is usually associated with gallstones in women and with alcoholism in men. Some people recover completely, others experience recurring attacks and still others develop chronic pancreatitis. The mortality and symptoms depend on the severity and type of pancreatitis: with mild pancreatic oedema, mortality is low; with severe necrotic pancreatitis, the mortality rate is high (Porth & Matfin, 2009).

Although the exact cause of pancreatitis is not known, the following factors may activate pancreatic enzymes within the pancreas, leading to autodigestion, inflammation, oedema and/or necrosis.

- Gallstones may obstruct the pancreatic duct or cause bile reflux, activating pancreatic enzymes in the pancreatic duct system.
- Alcohol causes duodenal oedema and may increase pressure and spasm in the sphincter of Oddi, obstructing pancreatic outflow. It also stimulates pancreatic enzyme production, thus raising pressure within the pancreas.

Other factors associated with acute pancreatitis include tissue ischaemia or anoxia, trauma or surgery, pancreatic tumours, third-trimester pregnancy, infectious agents (viral, bacterial or parasitic), elevated calcium levels and hyperlipidaemia. Some

medications have been linked with this disorder, including thiazide diuretics, oestrogen, steroids, salicylates and NSAIDs.

Regardless of the precipitating factor, the pathophysiological process begins with the release of activated pancreatic enzymes into pancreatic tissue. Activated proteolytic enzymes—trypsin, in particular—digest pancreatic tissue and activate other enzymes such as phospholipase A, which digests cell membrane phospholipids, and elastase, which digests the elastic tissue of blood vessel walls. This leads to proteolysis, oedema, vascular damage and haemorrhage, and necrosis of parenchymal cells. Cellular damage and necrosis release activated enzymes and vasoactive substances that produce vasodilation, increase vascular permeability and cause oedema. A large volume of fluid may shift from circulating blood into the retroperitoneal space, the peripancreatic spaces and the abdominal cavity.

MANIFESTATIONS Acute pancreatitis develops suddenly, with an abrupt onset of continuous severe epigastric and abdominal pain. This pain commonly radiates to the back and is relieved somewhat by sitting up and leaning forward. The pain often is initiated by a fatty meal or excessive alcohol intake.

Other manifestations include nausea and vomiting, abdominal distension and rigidity, decreased bowel sounds, tachycardia, hypotension, elevated temperature, and cold, clammy skin. Within 24 hours, mild jaundice may appear. Retroperitoneal bleeding may occur 3 to 6 days after the onset of acute pancreatitis; signs of bleeding include bruising in the flanks (Turner's sign) or around the umbilicus (Cullen's sign). See the 'Manifestations' box below.

COMPLICATIONS Systemic complications of acute pancreatitis include intravascular volume depletion with acute tubular necrosis and renal failure (see Chapter 27 for more information about acute renal failure) and acute respiratory distress syndrome (ARDS). Acute renal failure usually develops within 24 hours after the onset of acute pancreatitis. Manifestations of ARDS may be seen 3 to 7 days after its onset, particularly in people who have experienced severe volume depletion. See Chapter 36 for more information about ARDS.

Localised complications include pancreatic necrosis, abscess, pseudocysts and pancreatic ascites. Pancreatic necrosis causes an inflammatory mass that may be infected. It may lead to shock and multiple organ failure. A pancreatic abscess may form late in the course of the disease (6 or more weeks after its onset), causing an epigastric mass and tenderness (McPhee, Papadakis & Tierney, 2008). Pancreatic pseudocysts, encapsulated collections of fluid, may develop both within the pancreas itself and in the abdominal cavity. They may impinge on other structures or may rupture, causing generalised peritonitis. Rupture of a pseudocyst or of the pancreatic duct can lead to pancreatic ascites. Pancreatic ascites is recognised by gradually increasing abdominal girth and persistent elevation of the serum amylase level without abdominal pain.

MANIFESTATIONS Acute and chronic pancreatitis

ACUTE PANCREATITIS
- Abrupt onset of severe epigastric and left upper quadrant pain; may radiate to back
- Nausea, vomiting; fever
- Decreased bowel sounds; abdominal distension and rigidity
- Tachycardia, hypotension; cold, clammy skin
- Possible jaundice
- Positive Turner's sign (flank bruising) or Cullen's sign (periumbilical bruising)

CHRONIC PANCREATITIS
- Recurrent epigastric and LUQ pain; radiates to back
- Anorexia, nausea and vomiting, weight loss
- Flatulence, constipation
- Steatorrhoea

Chronic pancreatitis

Chronic pancreatitis is characterised by gradual destruction of functional pancreatic tissue. In contrast to acute pancreatitis, which may completely resolve with no long-term effects, chronic pancreatitis is an irreversible process that eventually leads to pancreatic insufficiency. Alcoholism is the primary risk factor for chronic pancreatitis in the Australia. Malnutrition is a major worldwide risk factor. About 10–20% of chronic pancreatitis is idiopathic, with no identified cause. A genetic mutation on a gene associated with cystic fibrosis may play a role in these cases. Children or young adults with cystic fibrosis may develop chronic pancreatitis as well.

In chronic pancreatitis related to alcoholism, pancreatic secretions have an increased concentration of insoluble proteins. These proteins calcify, forming plugs that block pancreatic ducts and the flow of pancreatic juices. This blockage leads to inflammation and fibrosis of pancreatic tissue. In other cases, a stricture or stone may block pancreatic outflow, causing chronic obstructive pancreatitis. In chronic pancreatitis, recurrent episodes of inflammation eventually lead to fibrotic changes in the parenchyma of the pancreas, with loss of exocrine function. This leads to malabsorption from pancreatic insufficiency. If endocrine function is disrupted as well, clinical diabetes mellitus may develop.

MANIFESTATIONS Chronic pancreatitis typically causes recurrent episodes of epigastric and left upper abdominal pain that radiates to the back. This pain may last for days to weeks. As the disease progresses, the interval between episodes of pain becomes shorter. Other manifestations include anorexia, nausea and vomiting, weight loss, flatulence, constipation and steatorrhoea (fatty, frothy, foul-smelling stools caused by a decrease in pancreatic enzyme secretion).

COMPLICATIONS Complications of chronic pancreatitis include malabsorption, malnutrition and possible peptic ulcer disease. Pancreatic pseudocysts or abscesses may form or stricture of the common bile duct may develop. Diabetes mellitus may develop and there is an increased risk of pancreatic cancer. Narcotic addiction related to frequent, severe pain episodes is common.

INTERPROFESSIONAL CARE

Acute pancreatitis often is a mild, self-limiting disease. Treatment focuses on reducing pancreatic secretions and providing supportive care. Treatment to eliminate the causative factor is begun after the acute inflammatory process resolves. Severe necrotising pancreatitis may require intensive care management. Treatment for chronic pancreatitis often focuses on managing pain and treating malabsorption and malnutrition.

Diagnosis

The laboratory tests that may be ordered when pancreatitis is suspected are summarised in Table 24.4. Diagnostic studies include the following:

- *Ultrasonography* can identify gallstones, a pancreatic mass or pseudocyst.
- *CT scan* may be ordered to identify pancreatic enlargement, fluid collections in or around the pancreas, and perfusion deficits in areas of necrosis.
- *Endoscopic retrograde cholangiopancreatography (ERCP)* may be performed to diagnose chronic pancreatitis and to differentiate inflammation and fibrosis from carcinoma.
- *Magnetic endoscopic retrograde cholangiopancreatography (MRCP)* is a non-invasive procedure that does not use contrast media and is safer for people with known contrast adverse reactions.
- *Endoscopic ultrasonography* can detect changes indicative of chronic pancreatitis in the pancreatic duct and parenchyma.
- *Percutaneous fine-needle aspiration biopsy* may be performed to differentiate chronic pancreatitis from cancer of the pancreas; the cells that are aspirated are examined for malignancy.

More information about these tests and their nursing implications can be found in Chapter 20.

Medications

The treatment of acute pancreatitis is largely supportive. Early, aggressive fluid replacement with intravenous crystalloid fluids is recommended for mild to severe pancreatitis diagnoses. In additional the person should be kept nil by mouth until nausea, vomiting and pain dissipates. Narcotic analgesics such as morphine sulfate are used to control pain. Antibiotics may be prescribed to treat infection.

People with chronic pancreatitis also require analgesics, but are closely monitored to prevent drug dependence. Narcotics are avoided when possible. Pancreatic enzyme supplements are given to reduce steatorrhoea (see the 'Medication administration' box below). People with chronic pancreatitis may need to remain on pancreatic enzyme supplements for life. H_2 blockers such as cimetidine (Tagamet) and ranitidine (Zantac) and proton pump inhibitors such as omeprazole (Losec) may be given to neutralise or decrease gastric secretions. Octreotide (Sandostatin), a synthetic hormone, suppresses pancreatic enzyme secretion and may be used to relieve pain in chronic pancreatitis.

Treatments

NUTRITION Oral food and fluids are withheld during acute episodes of pancreatitis to reduce pancreatic secretions and promote rest of the organ. A nasogastric tube may be inserted and connected to suction. Intravenous fluids are administered for 48–72 hours to maintain vascular volume. If there is no improvement enteral feeding will be commenced via either a nasogastric or a nasojejunal tube. Enteral feeding is preferable to total parenteral nutrition (TPN) as it maintains gut barrier integrity, which reduces bacteria translocation risk, and is more physiological than TPN (Brenner & Krenzer, 2010; Greenberger, Conwell & Banks, 2012). Oral food and fluids are begun once the serum amylase

TABLE 24.4 Laboratory tests in exocrine pancreatic disorders

TEST	NORMAL VALUE	SIGNIFICANCE
Serum amylase	0–130 U/L (U = units)	Rises within 2 to 12 hours of onset of acute pancreatitis to three times normal. Returns to normal in 3 to 4 days.
Serum lipase	0–160 U/L	Levels rise to three times normal in acute pancreatitis; remain elevated for 7 to 14 days.
Serum trypsinogen	< 80 mcg/L	Elevated in acute pancreatitis; may be decreased in chronic pancreatitis.
Urine amylase	4–37 U/L/2 h	Urine amylase levels rise in acute pancreatitis.
Serum glucose	70–110 mg/dL	May be transient elevation in acute pancreatitis.
Serum bilirubin	0.1–1.0 mg/dL	Compression of the common duct may increase bilirubin levels in acute pancreatitis.
Serum alkaline phosphatase	30–95 U/L	Compression of the common duct may increase levels in acute pancreatitis.
Serum calcium	8.9–10.3 mg/dL or 4.5–5.5 mEq/L	Hypocalcaemia develops in up to 25% of people with acute pancreatitis.
White blood cells	4500–10 000/mm^3	Leucocytosis indicates inflammation and is usually present in acute pancreatitis.

MEDICATION ADMINISTRATION The person with chronic pancreatitis

PANCREATIC ENZYME REPLACEMENT

Pancrelipase

Pancrelipase enhances the digestion of starches and fats in the gastrointestinal tract by supplying an exogenous source of the enzymes protease, amylase and lipase. The drug promotes nutrition and decreases the number of bowel movements.

Nursing responsibilities

- Assess for allergy to pork protein.
- Monitor frequency and consistency of stools.
- Weigh every other day and record.
- Give with meals; if not enteric coated, H_2 antagonists or antacids may be given concurrently to prevent destruction of the enzymes by hydrochloric acid.
- Monitor for side effects: rash, hives, respiratory difficulty, haematuria, hyperuricaemia or joint pain.

Health education for the person and family

- Take with meals or snacks.
- If medicine is enteric coated, do not crush, chew or mix with alkaline foods (e.g. milk, ice-cream).
- Be sure to follow prescribed diet.
- Continue taking this drug until or unless advised by physician that it is no longer necessary.

levels have returned to normal, bowel sounds are present and pain disappears. A low-fat diet is ordered and alcohol intake is strictly prohibited.

SURGERY If the pancreatitis is the result of a gallstone lodged in the sphincter of Oddi, an *endoscopic transduodenal sphincterotomy* may be performed to remove the stone. When cholelithiasis is identified as a causative factor, a cholecystectomy is performed once the acute pancreatitis has resolved. Surgical procedures to promote drainage of pancreatic enzymes into the duodenum or resection of all or part of the pancreas may be done to provide pain relief in people with chronic pancreatitis. Large pancreatic pseudocysts may be drained endoscopically or surgically.

Nursing care

In addition to the nursing care discussed in this section, a nursing care plan for a person with acute pancreatitis is found below.

Health promotion

Teach people who abuse alcohol about the risk of developing pancreatitis. Advise abstinence to reduce this risk and refer to an alcohol treatment program or Alcoholics Anonymous.

Assessment

Assessment data related to acute or chronic pancreatitis include the following:

- *Health history*: current manifestations; abdominal pain (location, nature, onset and duration, identified precipitating factors); anorexia, nausea or vomiting; flatulence, diarrhoea, constipation or stool changes; recent weight loss; history of previous episodes or gallstones; alcohol use (extent and duration); current medications.
- *Physical assessment*: vital signs including orthostatic vitals and peripheral pulses; temperature; skin temperature and colour, presence of any flank or periumbilical ecchymoses; abdominal assessment including bowel sounds, presence of distension, tenderness or guarding.

Nursing diagnoses and interventions

Nursing care for the person with acute pancreatitis focuses on managing pain, nutrition and maintaining fluid balance.

Pain

Obstruction of pancreatic ducts and inflammation, oedema and swelling of the pancreas caused by pancreatic autodigestion cause severe epigastric, left upper abdominal or mid-scapular back pain. The pain often is accompanied by nausea and vomiting, abdominal tenderness and muscle guarding.

- Using a standard pain scale (see Chapter 8), assess pain, including location, radiation, duration and character. Note non-verbal cues of pain: restlessness or remaining rigidly still; tense facial features; clenched fists; rapid, shallow respirations; tachycardia; and diaphoresis. Administer analgesics on a regular schedule. *Pain assessment before and after analgesic administration measures its effectiveness. Administering analgesics on a regular schedule prevents pain from becoming established, severe and difficult to control. Unrelieved pain has negative consequences; for example, pain, anxiety and restlessness may increase pancreatic enzyme secretion.*
- Maintain NBM status and nasogastric tube patency as ordered. *Gastric secretions stimulate hormones that stimulate pancreatic secretion, aggravating pain. Eliminating oral intake and maintaining gastric suction reduce gastric secretions. Nasogastric suction also decreases nausea, vomiting and intestinal distension.*
- Maintain bed rest in a calm, quiet environment. Encourage use of non-pharmacological pain management techniques such as meditation and guided imagery. *Decreasing physical movement and mental stimulation decreases metabolic rate, gastrointestinal secretion, pancreatic secretions and resulting pain. Adjunctive pain relief measures enhance the effectiveness of analgesics* (see Chapter 8).

- Assist to a comfortable position, such as a side-lying position with knees flexed and head elevated 45 degrees. *Sitting up, leaning forward or lying in a foetal position tends to decrease pain caused by stretching of the peritoneum by oedema and swelling.*
- Remind family and visitors to avoid bringing food into the person's room. *The sight or smell of food may stimulate secretory activity of the pancreas through the cephalic phase of digestion.*

CONSIDERATION FOR PRACTICE

Regularly assess respiratory status (at least every 4 to 8 hours), including respiratory rate, depth and pattern; breath sounds; oxygen saturation and arterial blood gas results. Report tachypnoea, adventitious or absent breath sounds, oxygen saturation levels below 92%, $PaO_2 < 70$ mmHg or $PaCO_2 > 45$ mmHg. Severe abdominal pain causes shallow respirations and hypoventilation, and suppresses cough effectiveness, which can lead to pooling of secretions, atelectasis and pneumonia.

Imbalanced nutrition: less than body requirements

The effects of pancreatitis and its treatment may result in malnutrition. Inflammation increases metabolic demand and frequently causes nausea, vomiting and diarrhoea. At a time of increased metabolic demand, NBM status and gastric suction further decrease available nutrients. In the person with chronic pancreatitis, loss of digestive enzymes affects the digestion and use of nutrients.

- Monitor laboratory values: serum albumin, serum transferrin, haemoglobin and haematocrit. *Serum albumin, serum transferrin (which transports iron in the blood), haemoglobin and haematocrit levels are decreased in malnutrition. Decreased pancreatic enzymes affect protein catabolism and absorption; decreased transferrin affects iron absorption and transport, thereby decreasing haematocrit and haemoglobin levels.*
- Weigh daily or every other day. *Short-term weight changes (over hours to days) accurately reflect fluid balance, whereas weight changes over days to weeks reflect nutritional status.*
- Maintain stool chart; note frequency, colour, odour and consistency of stools. *Protein and fat metabolism are impaired in pancreatitis; undigested fats are excreted in the stool. Steatorrhoea indicates impaired digestion and, possibly, an increase in the severity of pancreatitis.*
- Monitor bowel sounds. *The return of bowel sounds indicates return of peristalsis; nasogastric suction usually is discontinued within 24 to 48 hours thereafter.*
- Administer prescribed intravenous fluids and/or enteral feeds. *Intravenous fluids are given to maintain hydration. Enteral feeds are used to provide fluids, electrolytes and kilojoules when fasting is prolonged (more than 2 to 3 days).*
- Provide oral and nasal care every 1 to 2 hours. *Fasting and nasogastric suction increase the risk of mucous membrane irritation and breakdown.*
- When oral intake resumes, offer small, frequent feedings. Provide oral hygiene before and after meals. *Oral hygiene decreases oral microorganisms that can cause foul odour and taste, decreasing appetite. Small, frequent feedings reduce pancreatic enzyme secretion and are more easily digested and absorbed.*

Risk of deficient fluid volume

Acute pancreatitis can lead to a fluid shift from the intravascular space into the abdominal cavity (third spacing). Third spacing of fluid may cause hypovolaemic shock, affecting cardiovascular function, respiratory function, renal function and mental status.

- Assess cardiovascular status every 4 hours or as indicated, including vital signs, cardiac rhythm, haemodynamic parameters (central venous and pulmonary artery pressures); peripheral pulses and capillary refill; skin colour, temperature, moisture and turgor. *These measurements are indicative of fluid volume status and are used to monitor response to treatment. Stable values are as follows: heart rate less than 100; blood pressure within 10 mmHg of baseline; central venous pressure 0–8 mmHg; pulmonary wedge pressure 8–12 mmHg; cardiac output approximately 5 L/min; and skin warm, dry, with good turgor and colour.* (See Chapter 10 for a full discussion of hypovolaemic shock.)
- Monitor renal function. Obtain hourly urine output; report if less than 30 mL per hour. Weigh daily. *Urine output of less than 30 mL per hour indicates decreased renal perfusion or acute renal failure, a major complication of acute pancreatitis. Weight changes are an effective indicator of fluid volume status.*
- Monitor neurological function, including mental status, level of consciousness and behaviour. *Hypotension and hypoxaemia may decrease cerebral perfusion, causing changes in mental status, decreased level of consciousness and changes in behaviour. In addition, alcohol withdrawal is a risk in the person with acute pancreatitis.*

Community-based care

The person with pancreatitis is often acutely ill and, along with family members, needs information about both hospital procedures and self-care at home following discharge. During the acute stage, keep explanations brief and simple.

Prior to discharge, teach the person and family about the disease and how to prevent further attacks of inflammation. Include the following topics as appropriate:

- Alcohol can cause stones to form, blocking pancreatic ducts and the outflow of pancreatic juice. Continued alcohol intake is likely to cause further inflammation and destruction of the pancreas. Avoid alcohol entirely.
- Smoking and stress stimulate the pancreas and should be avoided.
- If pancreatic function has been severely impaired, discuss appropriate use of pancreatic enzymes, including timing, dose, potential side effects and monitoring of effectiveness.
- A low-fat diet is recommended. Provide a list of high-fat foods to avoid. Crash dieting and binge eating also should

NURSING CARE PLAN A person with acute pancreatitis

Rose Schliefer is a 59-year-old wife, mother of three and grandmother of four. She has been hospitalised for the past 6 weeks for acute haemorrhagic pancreatitis and pseudocyst. The pancreatitis was caused by gallstones. Mrs Schliefer spent 3 weeks in intensive care and then underwent surgery to remove the gallstones and to insert drains into the pseudocyst. Prior to discharge, she had progressed to a soft, high-carbohydrate, low-fat diet; had all drains removed; and was able to walk in the hall. Mrs Schliefer was referred to the community nurses in her home town for continued follow-up.

ASSESSMENT

Lee Quinn, the community health nurse, assesses Mrs Schliefer at home after discharge. Mrs Schliefer is thin and appears anxious and tired. She states that she lost 13 kg in the hospital and now weighs only 46 kg. She is 168 cm tall. Her vital signs are within normal limits. Mrs Schliefer has a well-healed upper abdominal scar and two small wounds (from drains) on each side of her abdomen. The wounds are closed but still have scabs. Her skin is cool and dry and turgor is poor. She is alert and oriented and responds appropriately to questions. Blood glucose levels are normal. Mrs Schliefer states that her main problems are lack of energy and lack of appetite for the low-fat diet that has been ordered. Mrs Schliefer's husband and daughters express concern about their ability to provide care. Although they have been taught all about the disease and how to provide care, they still are not sure they know exactly what should be done now that Mrs Schliefer is at home.

DIAGNOSES

- *Fatigue* related to decreased metabolic energy production.
- *Imbalanced nutrition: less than body requirements* related to prolonged hospitalisation, dietary restrictions and impaired digestion.
- *Bathing/hygiene self-care deficit* (Level II: requires help of another person, supervision and teaching) related to decreased strength and endurance.
- *Risk of caregiver role strain* related to inexperience with care-giving tasks.

PLANNING

- Develop activity goals, incorporating small, incremental steps towards achieving goals. Instruct Mrs Schliefer to:
  - a. Rest in bed each day from 1 pm to 3 pm.
  - b. Eat six small meals a day with family members or friends.
  - c. Sit and rest quietly for 15 minutes before eating.
- Arrange for regular family discussions of concerns about future and goal setting and acknowledge family strengths.

Expected outcomes

- Set priorities for daily and weekly activities and incorporate a rest period into daily activity.
- Gain 0.5–1 kg per week.
- Bathe and maintain personal hygiene without assistance.
- Family members will verbalise comfort with providing necessary care.

IMPLEMENTATION

- Explain causes of fatigue. Review effects of pancreatitis, surgery and acute illness on energy levels.
- Mrs Schliefer indicates that she wants to cook a meal for the whole family. To reach this goal, she will:
  - a. Schedule the meal when her energy level is highest.
  - b. List actions necessary to prepare the meal and delegate difficult tasks to family members.
  - c. Ask her daughters to reorganise the kitchen to avoid unnecessary steps.
  - d. Plan the meal no sooner than the third week after being home.
- Discuss dietary restrictions and how to adapt them to usual diet.
- Advise to use shower chair and develop self-care goals for bathing and hygiene in small steps. Add self-care tasks gradually as tolerated.
- Discuss division of responsibilities for physical care, home maintenance and medical care with family members.

EVALUATION

One month after discharge, Mrs Schliefer and her family have established new routines based on her energy levels. Mrs Schliefer now fixes lunch because she feels best around midday. She and her husband share this time together without interruption. Mrs Schliefer still rests during the day but can now provide self-care. She has gained only 1 kg, but states that she is getting used to the new diet and that 'things are even starting to taste good without butter'. She also says that sitting quietly before meals is helpful and that she prefers eating six small meals a day. Mr and Mrs Schliefer and their daughters agree that their initial worries about Mrs Schliefer's care have been resolved now they all know what they must do and the future looks much brighter.

CRITICAL THINKING IN THE NURSING PROCESS

1. You are caring for a person who has acute pancreatitis and is also an alcoholic. Describe assessments that indicate the beginnings of withdrawal.
2. Discuss the pathophysiological basis of hypovolaemic shock in acute necrotic pancreatitis.
3. Outline a teaching plan that includes specific foods to omit and to include in a high-carbohydrate, low-protein, low-fat diet.
4. Develop a plan of care for the nursing diagnosis of *Impaired home maintenance*.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Reflect on Mrs Schliefer's discharge process from hospital. What could have been done differently to avoid her loss of appetite and fatigue at home?

be avoided as they may sometimes precipitate attacks. Spicy foods, coffee, tea or colas, and gas-forming foods stimulate gastric and pancreatic secretions and may precipitate pain. Avoid them if this occurs.

- Report symptoms of infection (fever of 38.5°C or more, pain, rapid pulse, malaise) because a pancreatic abscess can develop after initial recovery.

Refer to a dietitian or nutritionist for diet teaching as needed. If appropriate, refer to community agencies, such as Alcoholics Anonymous, or to an alcohol treatment program. Provide referrals to community or home health agencies, as needed, for continued monitoring and teaching at home.

THE PERSON WITH PANCREATIC CANCER

Pancreatic cancer accounts for about one-sixth of all deaths associated with cancers in Australia. It has a 5-year survival rate of less than 6%. Complete resection is the best hope for cure if the cancer is in the head of the pancreas. However, by the time of initial presentation most people already have advanced disease. Only about 30% of these people can be offered curative resection. If the cancer is in the body or tail of the pancreas, only about 10% of these people can be offered curative resection (Cancer Australia, 2015; Samra, Gananadha & Hugh, 2008).

In contrast to acute and chronic pancreatitis, alcohol abuse and gallstones are not identified risk factors for pancreatic cancer.

FAST FACTS

Identified risk factors for pancreatic cancer include:

- Cigarette smoking—the incidence is twice as high in smokers as in non-smokers
- Exposure to industrial chemicals or environmental toxins
- Chronic pancreatitis
- Diabetes mellitus
- Obesity; high-fat diet.

Pathophysiology and manifestations

Most cancers of the pancreas occur in the exocrine pancreas, are adenocarcinomas and cause death within 1 to 3 years after diagnosis.

Cancer of the pancreas has a slow onset, with manifestations of anorexia, nausea, weight loss, flatulence and dull epigastric pain. The pain increases in severity as the tumour grows. Other manifestations depend on the location of the tumour. Cancer of the head of the pancreas, which is the most common site, often obstructs bile flow through the common bile duct and the ampulla of Vater, resulting in jaundice, clay-coloured stools, dark urine and pruritus. Cancer of the body of the pancreas presses on the coeliac ganglion, causing pain that increases when the person eats or lies supine. Cancer of the tail of the pancreas often causes no symptoms until it has metastasised. Other late manifestations include a palpable abdominal mass and ascites. Because the manifestations are non-specific, up to 85% of people with cancer of the pancreas do not seek healthcare until the cancer becomes too far advanced for a cure.

INTERPROFESSIONAL CARE

Early cancers of the head of the pancreas may be resectable. A pancreatoduodenectomy (commonly called Whipple's procedure) is performed to remove the head of the pancreas, the entire duodenum, the distal third of the stomach, a portion of the jejunum and the lower half of the common bile duct. The common bile duct is then sutured to the end of the jejunum and the remaining pancreas and stomach are sutured to the side of the jejunum (see Figure 24.7). Radiation and chemotherapy are often used in addition to surgery.

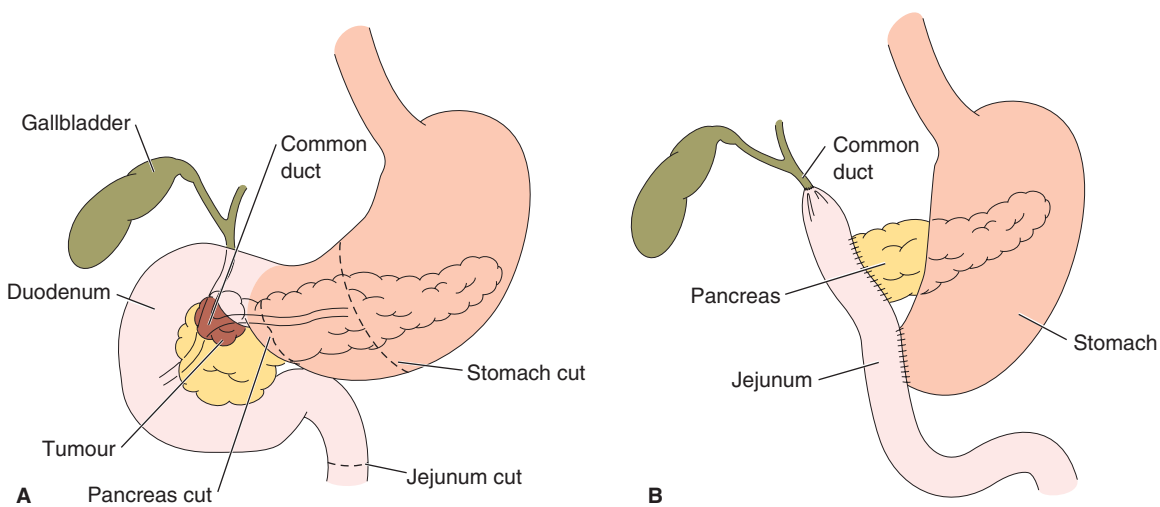


FIGURE 24.7 ■ Pancreatoduodenectomy (Whipple's procedure). *A*, areas of resection; *B*, appearance following resection

NURSING CARE OF THE PERSON undergoing Whipple's procedure

PREOPERATIVE CARE

- Provide routine preoperative nursing care as outlined in Chapter 3.
- Clarify teaching and learning as needed. Provide psychological support for person and family. *The person and family faced with a diagnosis of pancreatic cancer may require reinforcement of teaching as anxiety, fear and possible denial can interfere with learning.*

POSTOPERATIVE CARE

- Provide postoperative care as outlined in Chapter 3.
- Maintain in semi-Fowler's position. *Semi-Fowler's position facilitates lung expansion and reduces stress on the anastomosis and suture line.*
- Maintain low gastrointestinal suction. If drainage is not adequate, obtain an order to irrigate, using minimal pressure. Do not reposition nasogastric tube. *Pressure within the operative area from retained secretions increases intraluminal pressure and places stress on the suture line. Forceful irrigations and repositioning of the nasogastric tube may disrupt the suture line.*
- Maintain pain control using analgesics as prescribed (PCA, infusion or given on a regular basis). Assess effectiveness of pain management. *Doses higher than normal may be required if narcotic analgesics have been used prior to surgery to manage pain.*
- *Increased pain may indicate complications such as disruption of suture line, leakage from anastomosis or peritonitis. Adequate pain management increases resistance to stress, facilitates healing and increases the ability to cough, deep breathe and change position.*
- Assist with coughing, deep breathing and changing position every 1 to 2 hours. Splint incision during coughing and deep breathing. *The location of the incision makes coughing and deep breathing more painful. The prolonged surgical procedure, anaesthesia, location of incision and immobility increase the risk of retained secretions, atelectasis and pneumonia. Changing position facilitates drainage of secretions; effective coughing and deep breathing remove secretions and open distal alveoli.*
- Monitor for complications:
  a. Take vital signs every 2 to 4 hours or as indicated; immediately report changes (such as elevated temperature; hypotension; weak, thready pulse; increased or difficult respirations).
  b. Assess skin colour, temperature, moisture and turgor.
  c. Measure urinary output, gastrointestinal output and drainage from any other tubes; monitor amount and type of wound drainage.
  e. Assess level of consciousness.
  f. Monitor results of laboratory tests, especially arterial blood gases, haemoglobin and haematocrit.

The main potential complications following Whipple's procedure are haemorrhage, hypovolaemic shock and hepatorenal failure. *The assessments listed provide information about the person's status and alert the nurse to abnormal findings that signal the onset of these complications.*

Postoperative nursing care of the person undergoing Whipple's procedure is outlined above. Immediate postoperative care is often provided in the intensive care unit.

The person with pancreatic cancer has multiple problems requiring nursing care. Chapter 13 provides a discussion of care of the person with cancer. The nursing diagnoses and interventions discussed for the person with pancreatitis are also appropriate for the person with pancreatic cancer.

CHAPTER HIGHLIGHTS

- Gallstones (cholelithiasis) are common and often unrecognised until the person develops manifestations of biliary colic or acute cholecystitis. Laparoscopic cholecystectomy is the treatment of choice for symptomatic gallbladder disease.
- Hepatitis, inflammation of functional liver tissue, is usually a viral disease and therefore cannot be cured at this time. Preventing the spread of hepatitis through use of standard and body substance precautions is an important nursing responsibility.
- Hepatitis A, commonly transmitted via the faecal–oral route, is generally a self-limiting disease with few long-term sequelae. Some types of viral hepatitis, most notably hepatitis B and C, can become chronic and ultimately lead to liver failure and an increased risk of liver cancer. Hepatitis B and C can result in a carrier state in which the infected person has no symptoms of the disease, but can spread it to others.
- Alcohol abuse is a significant risk factor for liver and pancreatic disorders. Prevention, early identification and treatment of alcohol abuse reduce the risk of these disorders. Absolute abstinence from alcohol is an important part of the treatment plan for people with liver and pancreatic disorders.
- Cirrhosis leads to portal hypertension and liver failure, which, in turn, account for most of the manifestations and complications of the disorder. Complications, such as ascites, splenomegaly, oesophageal varices and hepatic encephalopathy, affect multiple body systems and significantly contribute to mortality and morbidity associated with cirrhosis.
- Bleeding from oesophageal varices may be massive, resulting in a medical emergency and requiring prompt control to maintain cardiac output.
- Acute pancreatitis often develops as a complication of gallstones. Acute pancreatitis often resolves with no long-term consequences. Chronic pancreatitis is more frequently related to alcohol abuse and can lead to continuing pain and digestive disruptions.

CONCEPT CHECK

1 When assessing the person admitted for a laparoscopic cholecystectomy, the nurse would expect to find:
1 a history of intermittent episodes of right upper quadrant pain
2 significant jaundice of the sclera and skin
3 complaints of recurrent heartburn and acid reflux
4 ascites and peripheral oedema

2 Which of the following does the nurse include in her teaching for a person with acute cholecystitis? (Select all that apply.)
1 Avoid consumption of foods high in fat, such as gravies and peanut butter.
2 Limit your intake to dry cracker biscuits and clear liquids during episodes of acute pain.
3 A low-carbohydrate diet such as the Atkins diet is recommended for weight loss.
4 Call your doctor if you develop severe abdominal pain and a temperature.
5 Surgery for gallstones is optional; they pose little risk when fat intake is minimal.

3 During an outbreak of hepatitis A traced to a food handler at a local restaurant, the nurse teaches staff at the restaurant that the most cost-effective means of protecting customers from further outbreaks is to:
1 insist that all food handlers be immunised against hepatitis A
2 test all new employees for hepatitis A antigen
3 wash hands thoroughly before handling food and after using the bathroom
4 use gloves for handling food if any cuts or scrapes are on hands

4 The nurse would evaluate teaching as effective when a person with chronic hepatitis C states which of the following?
1 'I will reduce my alcohol intake and use only paracetamol for pain relief.'
2 'I understand that I must return to the doctor every year for a follow-up liver biopsy.'
3 'Even though no treatment is available for this disease, I plan to live a long life.'
4 'I will avoid donating blood and will use barrier protection during sex.'

5 When evaluating for people possibly exposed to hepatitis A by a recently diagnosed person, the nurse inquires about:
1 sexual partners within the past 6 months
2 close household contacts within the past 4 weeks
3 food preparation activities since the development of jaundice
4 immunisation status of the person

6 A person hospitalised with cirrhosis, ascites and mild hepatic encephalopathy suddenly vomits 200 mL of bright red blood. Which of the following should the nurse do first?
1 Insert a nasogastric tube.
2 Place in Fowler's position.
3 Contact the physician.
4 Check stool for occult blood.

7 The nurse caring for a person scheduled for an abdominal paracentesis instructs the person to:
1 avoid eating or drinking fluid for 6 hours prior to the procedure
2 scrub the abdomen with antiseptic soap before the procedure
3 empty the bladder before the procedure
4 report excess flatus following the procedure to the physician

8 A person hospitalised with severe ascites due to cirrhosis develops a fever and confusion. The nurse should:
1 auscultate bowel sounds and palpate for abdominal tenderness
2 enquire about headache and check for nuchal rigidity
3 observe for neck vein distension and auscultate lung sounds
4 measure abdominal girth and percuss for shifting dullness

9 A 54-year-old woman admitted with acute pancreatitis says, 'I don't understand how I got this disease. I thought alcoholics got pancreatitis—I never drink.' Which of the following is the most appropriate response by the nurse?
1 'Was there a time in your life that you did drink heavily?'
2 'It also is prevalent in smokers; do you smoke cigarettes?'
3 'Gallstones also are a risk factor. We'll evaluate for them.'
4 'Intravenous drug use is a risk factor. Do you use drugs by injection?'

10 The nurse caring for a person returning to the unit following Whipple's procedure identifies which of the following as of highest priority in the plan of care?
1 referral to a smoking cessation program
2 frequent turning, coughing and deep breathing exercises
3 early mobilisation including ambulation as tolerated
4 maintaining patency of the nasogastric tube

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UNIT 6 BUILDING CLINICAL COMPETENCE

Responses to altered gastrointestinal function

CLINICAL SCENARIO

You have been assigned to work with the following four people for the 0700 shift. Significant data obtained during report are as follows:

- Thomas Jones, aged 56, was transferred to your unit yesterday after treatment in the critical care unit for oesophageal varices. Significant history includes alcohol consumption (6 to 12 beers daily for several years) and smoking (2 packets per day for the past 30 years). Current vital signs are T 37.7°C, P 96, R 28, BP 150/90. He complains of abdominal tenderness and dyspnoea. He appears anxious and irritable.
- Ruth Green, aged 35, was admitted with right upper quadrant pain radiating to the left shoulder and a feeling of abdominal fullness. She has a history of cholelithiasis and cholecystitis. Her assessment reveals T 37.2°C, P 90, R 24, BP 140/84, with pallor, diaphoresis and complaints of nausea. She is scheduled for a cholecystectomy at 9 am.
- Tanya Cooper, aged 21, was admitted with dehydration, weakness and fainting. Her weight is 40.9 kg and height is 165 cm. Her vital signs are T 36.1°C, P 70, R 26, BP 90/56 mmHg with orthostatic BP 70/48 mmHg. She has a 3-year history of anorexia nervosa and laxative abuse. She has an IV of 0.9% NaCl with 20 mmol KCl infusing. She is to be monitored for food intake and watched for 1 hour after meals. She is ringing her call light to get up to the bathroom.
- Grace Freeman is a 36 year old who had a temporary colostomy formed 5 days ago following an abdominal injury from a motor vehicle crash. Vital signs at 0400 were T 36.8°C, P 78, R 14, BP 112/78. She buzzed for assistance because her colostomy bag is full and she needs help emptying it.

Critical thinking questions

1 In what order would you visit these people after report?

1. _____
2. _____
3. _____
4. _____

2 What top two priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Thomas Jones		
Ruth Green		
Tanya Cooper		
Grace Freeman		

3 You need to complete preoperative preparation on Mrs Green. Which of the following do you need to do?

1. Complete preoperative checklist, witness signed consent and administer preoperative medication when requested.
2. Explain the procedure, obtain informed consent and complete the preoperative checklist.
3. Sign the operative consent, explain complications of the procedure and take vital signs on call.
4. Obtain signed consent, discuss with the family the surgical procedure and have the person void prior to going to the OR.

4 Mrs Green understands the postoperative teaching done by the nurse when she states:

1. 'I will be on bed rest for two days after surgery.'
2. 'I will need to cough and deep breathe while splinting my incision.'
3. 'I will be able to begin eating when I return from surgery.'
4. 'I will be medicated for pain without having to request it.'

5 The nurse explains a diet of low-fat foods to Mrs Green. She understands this diet when she picks which meal plan?

1. eggs, sausage and toast
2. chicken, mashed potatoes and gravy and corn
3. grilled fish, tossed salad, peaches
4. hamburger with lettuce and tomato, chips

6 To prepare Mr Jones for an oesophagoscopy, the nurse institutes the following interventions:

1. Explain that it is not a painful procedure but he will be medicated for pain.
2. Keep Mr Jones NBM for 12 hours prior to the procedure.
3. Remove dentures and provide mouth care.
4. Place in a supine position with the head slightly hyperextended.

7 Which discharge instructions will the RN advise Mrs Freeman about regarding how to take care of the colostomy?

1. 'The types of foods you eat will not affect the colostomy output.'
2. 'Empty the colostomy pouch or replace the bag when it is half full.'
3. 'Irrigate the colostomy with water to stimulate the colon to empty.'
4. 'Cleanse the area around the stoma with deodorant soap to decrease odour.'

8 Which of the following is the most common initial manifestation of malignant tumours of the lower bowel?

1. rectal bleeding
2. diarrhoea
3. rectal pain
4. constipation

9 A person with severe diarrhoea may develop metabolic acidosis. Which arterial blood gases indicates metabolic acidosis?

1. pH 7.45, $PaCO_2$ 40 mmHg, bicarbonate 25 mEq/L
2. pH 7.28, $PaCO_2$ 30 mmHg, bicarbonate 19 mEq/L
3. pH 7.55, $PaCO_2$ 50 mmHg, bicarbonate 30 mEq/L
4. pH 7.33, $PaCO_2$ 36 mmHg, bicarbonate 24 mEq/L

10 Teaching appropriate constipation management includes which actions? (Select all that apply.)

1. Decrease dietary fibre.
2. Increase fluid intake.
3. Increase exercise activity.
4. Use bulk-forming laxatives.
5. Use enemas daily.

11 With Ms Cooper's history of anorexia for 3 years, which is a priority nursing intervention in the plan of care for her?

1. Monitor for cardiac arrhythmias due to electrolyte imbalances.
2. Monitor weight for loss or gain to determine effectiveness of nursing care.
3. Maintain close observation for at least 1 hour after meals.
4. Serve small, frequent meals, increasing serving size gradually.

12 In planning discharge for Ms Cooper, the family and person participate in teaching and diet counselling sessions. Which is the priority item for the family and Ms Cooper to follow after discharge?

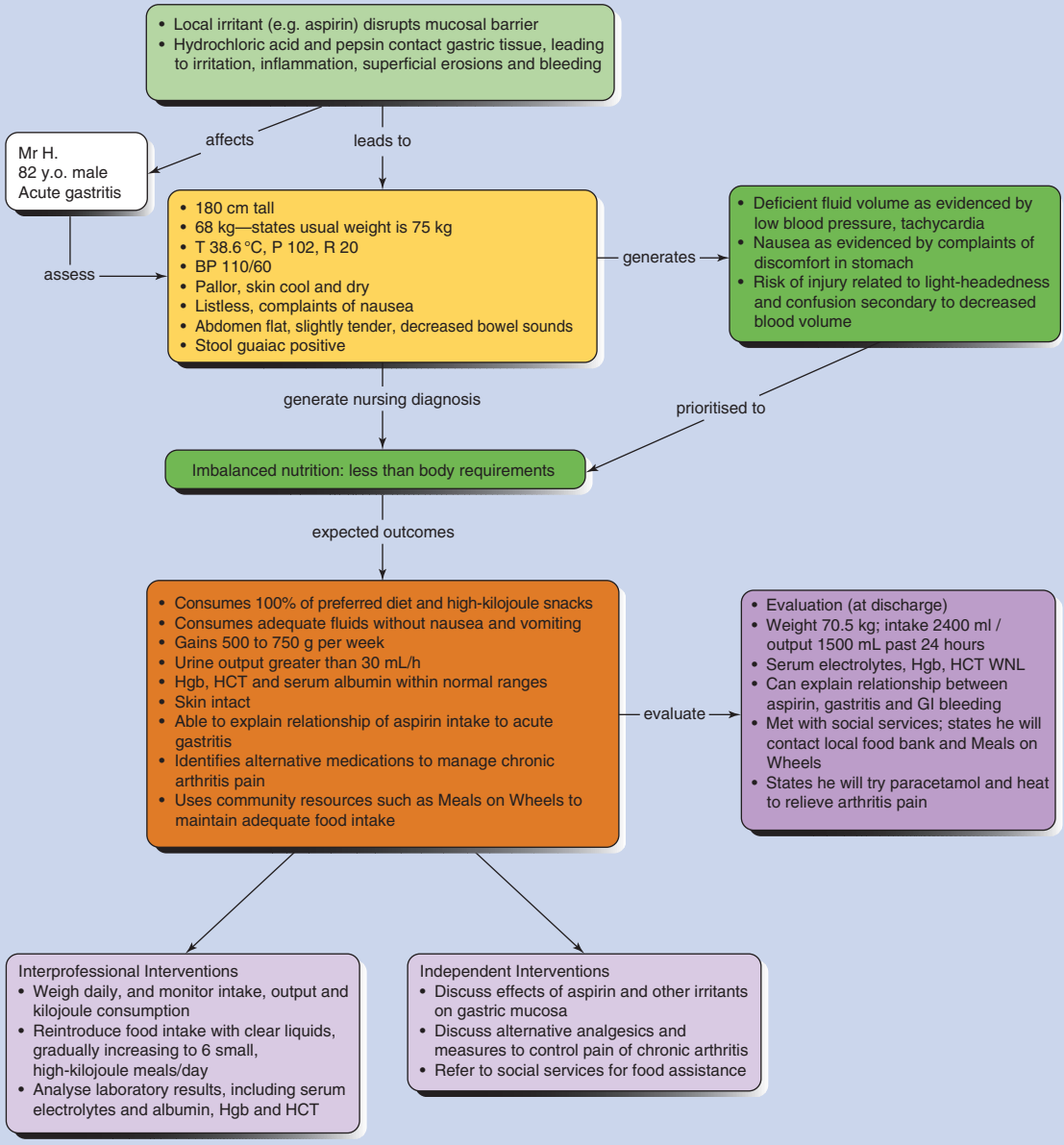
1. Monitor weight regularly to determine further weight loss.
2. Use rewards for food and kilojoule intake rather than weight gain.
3. Gradually increase the amount of food taken at meals.
4. Attend support groups for people with eating disorders.

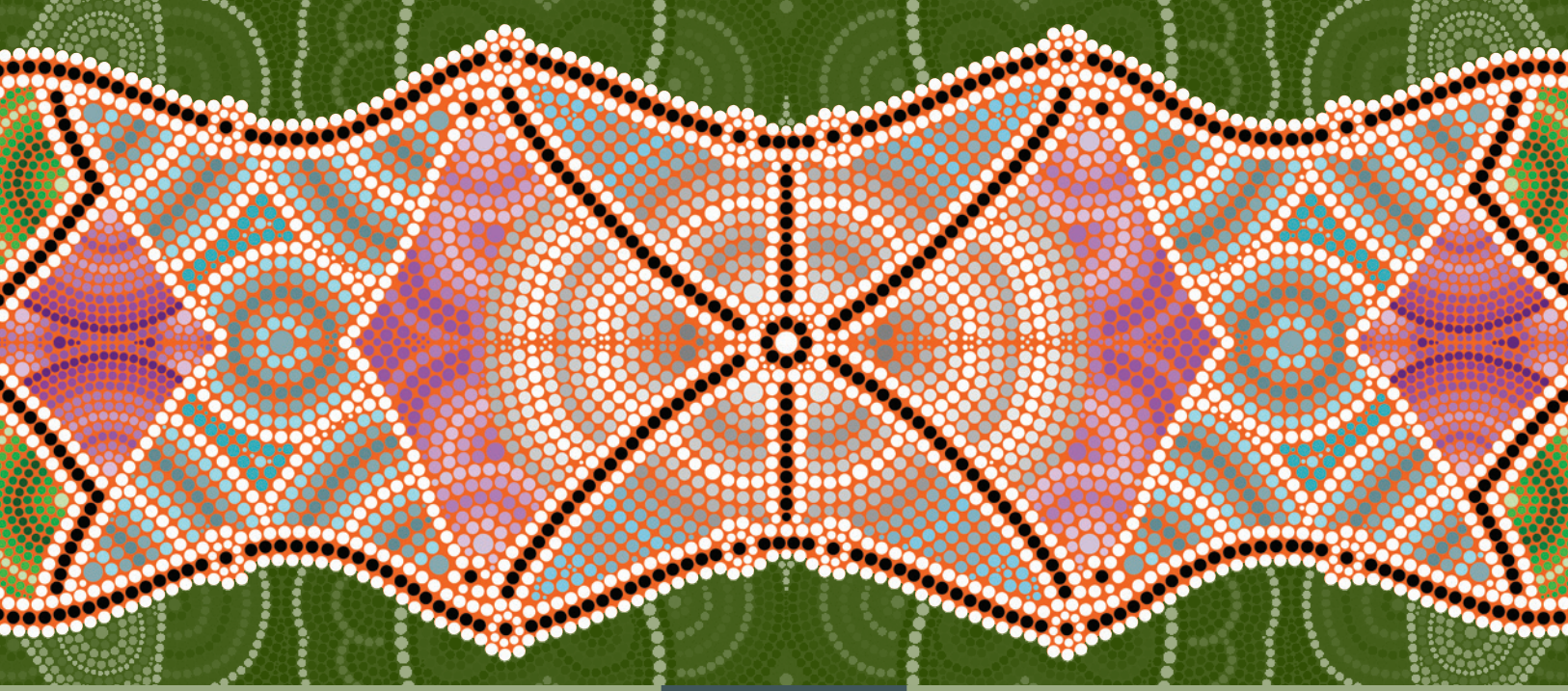
CASE STUDY

Lewis Haches, an 82-year-old white male, has a history of hypertension, degenerative arthritis and angina pectoris for which he takes frusemide, atenolol, extra-strength aspirin and glyceryl trinitrate. He states that his blood pressure is controlled when he takes his medications as prescribed. Lately, he has been having some financial difficulties and does not take his medications as often as he should. He lives with his 80-year-old wife, who also has health problems. He does the cooking and grocery shopping. He has a son who lives nearby who looks in on them every couple of days.

Mr Haches feels that he cooked some outdated food that made him ill. He states he has been trying to stretch their grocery budget by reducing their serving sizes and shopping less frequently. He says he has had nausea and loose, dark stools for the past 2 days. This morning, he is weak and dizzy and states he nearly fainted in the shower. He also states he has not been able to take his medications for the past 2 days and feels that his heart is beating too fast. His son brought him to the hospital because he was weak, confused and very pale when he checked on him prior to going to work in the morning.

Based on Mr Haches's medical diagnosis and treatment plan, *Imbalanced nutrition: less than body requirements* is identified as the priority nursing diagnosis at this time.





UNIT 7

RESPONSES TO ALTERED URINARY ELIMINATION

CHAPTER 25

A PERSON-CENTRED APPROACH TO ASSESSING THE RENAL SYSTEM

TRUDY DWYER, JENNIFER BORG, DIANE GOLDSWORTHY

KEY TERMS

albuminuria 838
calculi 835
chronic kidney disease (CKD) 831
creatinine 832
dysuria 834
glomerular filtration rate (GFR) 829
haematuria 834
micturition 833
nocturia 834
oliguria 834
polyuria 834
pyuria 834
urea 831

LEARNING OUTCOMES

- Describe the anatomy, physiology and functions of the renal system.
- Examine investigations, techniques and observations important for assessing a person's renal system function.
- Demonstrate accurate interpretation of normal and aberrant data obtained from assessment of a person's renal system.

CLINICAL COMPETENCIES

- Conduct and document a health history for people who have or are at risk of alterations in urinary elimination.
- Conduct and document a physical assessment of the renal system.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Urine specimen cup
- Disposable gloves

The functions of the renal system (also called the urinary system) are to regulate and maintain body fluids and electrolyte balance, to filter metabolic wastes from the bloodstream, to reabsorb needed substances and water into the bloodstream, and to eliminate metabolic wastes and water as urine. The renal system also indirectly maintains the body's the blood pressure, acid–base balance and an endocrine function. Any alteration in the structure or function of the renal system affects the whole body. In turn, healthy renal system function depends on the health of other body systems, especially the circulatory, endocrine and nervous systems.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE RENAL SYSTEM

The organs of the renal system are the paired kidneys (produce urine), the paired ureters (transport urine to the bladder), the urinary bladder (collects the urine) and the urethra (transports urine to outside the body) (see Figure 25.1). There are three major functions of the renal system:

1. *excretion*, the removal of wastes from body fluids
2. *elimination*, the elimination of these wastes from the body
3. *homeostatic regulation* of the volume and solute concentration of the plasma in the blood (Martini, Nath & Bartholomew, 2012).

Each structure is essential to the total functioning of the renal system.

THE KIDNEYS

The two kidneys are located outside the peritoneal cavity and on either side of the vertebral column at the levels of T_{12} to L_3. The left kidney is slightly superior to the right kidney. These highly vascular, bean-shaped organs are approximately 11.5 cm long, 5–7.5 cm wide, 2.5 cm thick and weigh about 150 g. The lateral surface of the kidney is convex; the medial

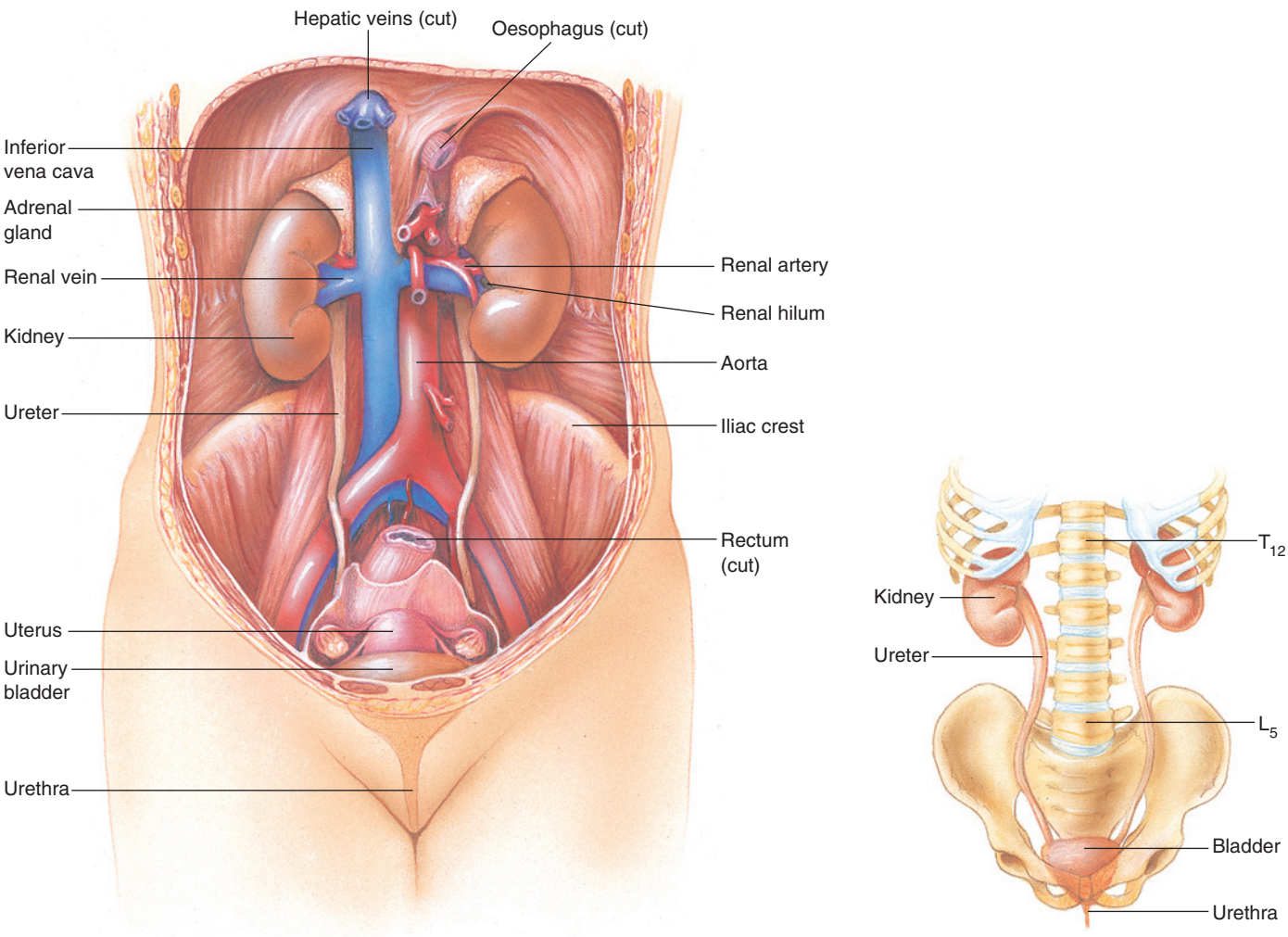


FIGURE 25.1 ■ The renal system. *A*, Anterior view of the renal system in a female. *B*, The kidneys are shown in relation to the vertebrae and ribs

surface is concave and forms a vertical cleft, the hilum. The ureter, renal artery, renal vein, lymphatic vessels and nerves enter or exit the kidney at the level of the hilum.

The kidney is supported by three layers of connective tissue: the outer renal fascia, the middle adipose capsule and the inner renal capsule. The renal fascia, made up of dense connective tissue, surrounds the kidney (and the adrenal gland, a discrete organ that sits on top of each kidney) and anchors it to surrounding structures. The middle adipose capsule is a fatty mass that holds the kidney in place and also cushions it against trauma. The inner renal capsule provides a barrier against infection and helps protect the kidney from trauma.

Internally, each kidney has three distinct regions: the cortex, medulla and pelvis. The outer region, or renal cortex, is light in colour and has a granular appearance (see Figure 25.2). This region of the kidney contains the glomeruli, small clusters of capillaries. The glomeruli bring blood to and carry waste products from the nephrons, the functional units of the kidney.

The renal medulla, just below the cortex, contains cone-shaped tissue masses called renal pyramids, formed almost entirely of bundles of collecting tubules. Areas of lighter-coloured tissue called renal columns are extensions of the cortex and serve to separate the pyramids. The collecting tubules that make up the pyramids channel urine into the innermost region, the renal pelvis.

The renal pelvis is continuous with the ureter as it leaves the hilum. Branches of the pelvis known as the major and minor calyces extend towards the medulla and serve to collect urine and empty it into the pelvis. From the pelvis, urine is channelled through the ureter and into the bladder for storage. The walls of the calyces, the renal pelvis and the ureter contain smooth muscle that moves urine along by peristalsis.

Each kidney contains approximately 1 million nephrons, which process the blood to make urine (see Figure 25.3). Each nephron consists of a renal corpuscle and a renal tubule (Martini et al., 2012). The renal corpuscle contains a tuft of capillaries called the glomerulus, which is completely

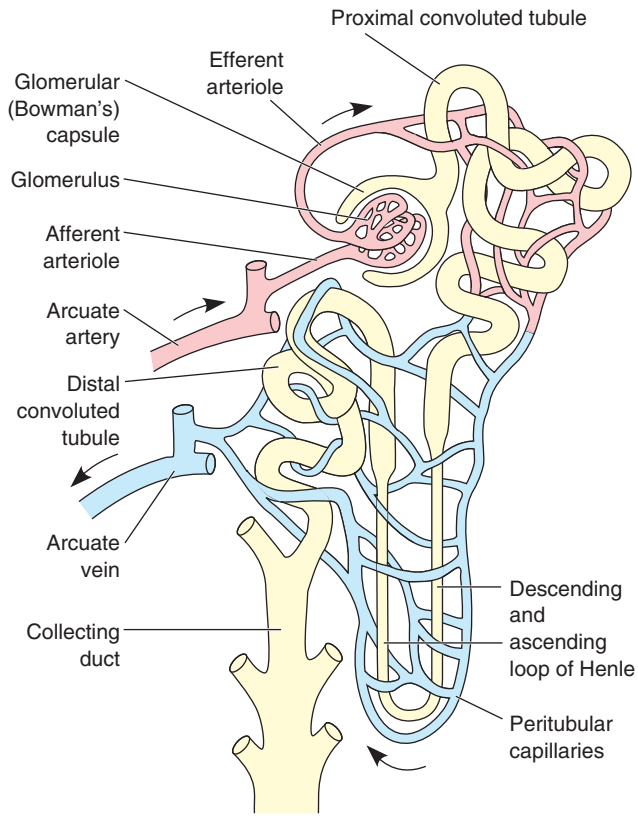


FIGURE 25.3 ■ The structure of a nephron, showing the glomerulus within the glomerular capsule

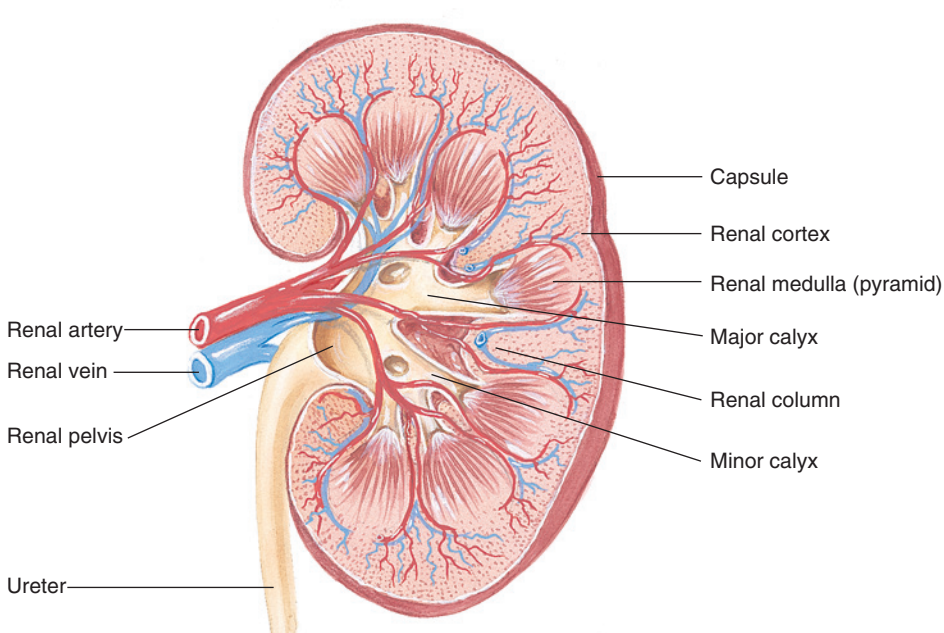


FIGURE 25.2 ■ Internal anatomy of the kidney

surrounded by the glomerular capsule (or Bowman's space). The renal tubule is a tubular passageway that may be up to 50 mm in length (Martini et al., 2012).

The endothelium of the glomerulus allows capillaries to be extremely porous. Thus, large amounts of solute-rich fluid pass from the capillaries into the capsule. This fluid, called the filtrate, is the raw material of urine. Filtrate leaves the capsule and is channelled into the renal tubule which consists of two convoluted (twisted or coiled) segments: the proximal convoluted tubule (PCT) and the distal convoluted tubule (DCT) (Martini et al., 2012). In the PCT of the nephron, microvilli on the tubular cells increase the surface area for reabsorption of substances from the filtrate into plasma in the peritubular capillaries. Substances moved by active transport include glucose, sodium, potassium, amino acids, proteins and vitamins. About 70% of the water in the filtrate, as well as chloride and bicarbonate, is reabsorbed by passive transport.

The filtrate then moves into the U-shaped loop of Henle where it is concentrated. The descending limb of the U is relatively thin and freely permeable to water, whereas the ascending segment is thick and thereby less permeable. The DCT receives filtrate from the loop of Henle. Although this segment is structurally similar to the PCT, it lacks microvilli and is more involved with secreting solutes into the filtrate than in reabsorbing substances from it. The collecting duct receives the newly formed urine from many nephrons and channels urine through the minor and major calyces of the renal pelvis and into the ureter.

The functions of the kidney are to:

- form urine
- balance solute and water transport
- excrete metabolic waste products
- conserve nutrients
- regulate acid–base balance
- secrete hormones to help regulate blood pressure, erythrocyte production and calcium metabolism.

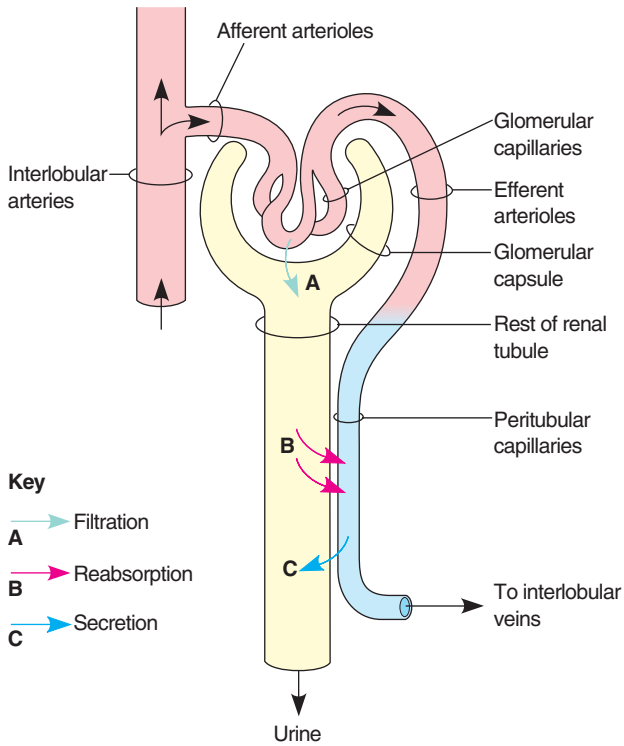


FIGURE 25.4 ■ Schematic view of the three major mechanisms by which the kidneys adjust to the composition of plasma: *A*, glomerular filtration; *B*, tubular reabsorption; and *C*, tubular secretion

Formation of urine

The complex structures of the kidneys process about 180 L of blood-derived fluid each day. Of this amount, only 1% is excreted as urine; the rest is returned to the circulation. (Normal and abnormal findings of urine on laboratory analysis are listed in Table 25.1.) Urine formation is accomplished entirely by the nephron through three processes: glomerular filtration, tubular reabsorption and tubular secretion (see Figure 25.4).

Glomerular filtration

Glomerular filtration is a passive, non-selective process in which hydrostatic pressure forces fluid and solutes through a membrane. The amount of fluid filtered from the blood into the capsule per minute is called the **glomerular filtration rate (GFR)**. Three factors influence this rate: the total surface area available for filtration, the permeability of the filtration membrane and the net filtration pressure.

The glomerulus is a far more efficient filter than most capillary beds, because the filtration membrane of the glomerulus is much more permeable to water and solutes than are other capillary membranes. In addition, the glomerular blood pressure is much higher, resulting in higher net filtration pressure.

Net filtration pressure is responsible for the formation of filtrate and is determined by two forces: hydrostatic pressure ('push') and osmotic pressure ('pull'). The glomerular hydrostatic pressure pushes water and solutes across the membrane. This pressure is opposed by the osmotic pressure in the glomerulus (primarily the colloid osmotic pressure of plasma proteins in the glomerular blood) and the capsular hydrostatic pressure exerted by fluids within the glomerular capsule. The difference between these forces determines the net filtration pressure, which is directly proportional to the GFR.

The normal GFR in both kidneys is 120 to 125 mL/min in adults or about 10% of the blood delivered to the glomeruli (Martini et al., 2012). This rate is held constant under normal conditions by intrinsic controls (or renal autoregulation). The myogenic mechanism, which responds to pressure changes in the renal blood vessels, controls the diameter of the afferent arterioles, thereby achieving autoregulation. An increase in systemic blood pressure causes the renal vessels to constrict, whereas a decline in blood pressure causes the afferent arterioles to dilate. These changes adjust the glomerular hydrostatic pressure and, indirectly, maintain the glomerular filtration rate. The GFR is difficult to measure so an estimated GFR (eGFR) is calculated from the serum creatinine (Kidney Health Australia (KHA), 2012). The eGFR is discussed in greater depth in Chapter 27.

TABLE 25.1 Normal and abnormal findings: urinalysis

CHARACTERISTIC OR COMPONENT	NORMAL RESULTS	ABNORMAL FINDING WITH POSSIBLE CAUSE
Colour	Light straw to amber-yellow	• Red, dark, smoky colour may be the result of blood in the urine (haematuria or menstrual blood). • Cloudy urine occurs from infection. • Colourless urine indicates very dilute urine, such as in overhydration, kidney disease, alcohol ingestion or diabetes insipidus. • Very dark yellow urine indicates dehydration and/or fever. • Red or red brown urine may be caused by medications such as phenytoin (Dilantin), chlorpromazine (Largactil) and phenolphthalein (Probanthine), and by beetroot, carrots, rhubarb or food colouring. • Purple urine may occur in people with permanent bladder catheters who have *Escherichia coli* infections. Sometimes called 'purple bag syndrome'. • Orange urine may be caused by fever, urobilin, nitrofurantoin (Macrodantin), sulfonamides and foods such as carrots and beetroot, or food colouring. • Blue or green urine is caused by *Pseudomonas* urinary tract infection, ingested substances such as amitriptyline (Endep), methylene blue, cimetidine (Tagamet), propofol (Diprivan) infusion and yeast concentrate. • Brown or black urine is caused by Lysol poisoning, melanin, bilirubin, methaemoglobin, herbal medicines such as cascara and injectable iron.
Appearance	Clear	• Hazy or cloudy urine indicates bacteria, pus, RBCs, WBCs, phosphates, prostatic fluid spermatozoa or urates. • Milky urine is the result of fats or pyuria. • Yellow foam results from bilirubin, bile or severe cirrhosis of the liver. • A dark yellow to brownish colour is seen with deficient fluid volume.
Odour	Aromatic	• Ammonia smell increases as urine stands outside the body. • Urinary tract infection (UTI) causes a foul or unpleasant odour, depending on the causative organism. • Asparagus causes a distinctive odour. • Mousy odours result from phenylketonuria. • Sweet or fruity odours occur in starvation and diabetic ketoacidosis.
pH	4.5–8.0	• < 4.5: metabolic acidosis, respiratory acidosis, diet high in protein or cranberries, ammonium chloride and mandelic acid. • > 8.0: bacteriuria, UTI, antibiotics (neomycin, kanamycin), sulfonamides, sodium bicarbonate, acetazolamide (Diamox), potassium citrate, vegetarian diet, low-carbohydrate diet and ingestion of citrus fruits.
Specific gravity	1.005–1.030	• < 1.005: diabetes insipidus, overhydration, renal disease, severe potassium deficit. • > 1.030: dehydration, fever, diabetes mellitus, vomiting, diarrhoea, contrast media.
Protein	2–8 mg/dL	• > 8 mg/dL: proteinuria, exercise, fever, stress, acute infection, kidney disease, lupus erythematosus, leukaemia, multiple myeloma, cardiac disease, toxaemia of pregnancy, sexual intercourse (in men), septicaemia, lead, mercury, neomycin, barbiturates, sulfonamides.
Glucose	Negative	• > 15 mg/dL or +4: diabetes mellitus, stroke, Cushing's syndrome, anaesthesia, glucose infusions, severe stress, infections, ascorbic acid, aspirin, cephalosporins and adrenaline.
Ketones	Negative	• +1 to +3: ketoacidosis, starvation, high-protein diet, severe exercise, exposure to cold, loss of carbohydrates.
RBCs	Rare	• > 2 per low-power field: kidney trauma, kidney diseases, renal calculi, cystitis, excess aspirin, anticoagulants, sulfonamides, menstrual contamination. *Note:* high false-positive rate of haematuria with 'dipstick testing'.
WBCs	3–4	• > 4 per low-power field: UTI, fever, strenuous exercise, kidney diseases.
Bilirubin	Negative	• Liver disease, biliary obstruction.
Urobilinogen	Low concentrations	• Liver disease such as hepatitis or cirrhosis or haemolytic conditions.
Casts	Occasional hyaline	• Fever, kidney diseases, heart failure.

The normal eGFR in the healthy adult is equal to or greater than 90 mL/min/1.73m^2 (KHA, 2012). Two aspects of the kidney are used to determine kidney function: eGFR and the presence of albuminuria (Department of Health, Victoria (DHV), 2012). **Chronic kidney disease (CKD)** is a general term used to refer to all conditions of the kidney (CKD stages 1–5) that last longer than 3 months (Australian Institute of Health and Welfare (AHIW), 2009).

Another intrinsic control of the GFR results from the renin–angiotensin mechanism at work in the kidneys. Special cells known as the juxtaglomerular apparatus are located in the distal tubules and respond to slow filtrate flow by releasing chemicals that cause intense vasodilation of the afferent arterioles. Conversely, an increase in the flow of filtrate promotes vasoconstriction, decreasing the GFR. A drop in systemic blood pressure often triggers the juxtaglomerular cells to release renin. Renin acts on a plasma globulin, angiotensinogen, to release angiotensin I, which is in turn converted to angiotensin II. As a vasoconstrictor, angiotensin II activates vascular smooth muscle throughout the body, causing systemic blood pressure to rise. Thus, the renin–angiotensin mechanism is a factor in renal autoregulation, even though its main purpose is the control of systemic blood pressure.

Glomerular filtration is also under an extrinsic control mechanism through the sympathetic nervous system. During periods of extreme stress or emergency, sympathetic nervous system stimulation causes strong constriction of the afferent arterioles and inhibits filtrate formation. The sympathetic nervous system also stimulates the juxtaglomerular cells to release renin, increasing systemic blood pressure.

Tubular reabsorption

Tubular reabsorption is a transepithelial process that begins as the filtrate enters the proximal tubules. In healthy kidneys, virtually all organic nutrients such as glucose and amino acids are reabsorbed. However, the tubules constantly regulate and adjust the rate and degree of water and ion reabsorption in response to hormonal signals. Reabsorption may be active or passive. Substances reclaimed through active tubular reabsorption are usually moving against electrical and/or chemical gradients. These substances, including glucose, amino acids, lactate, vitamins and most ions, require an ATP-dependent carrier to be transported into the interstitial space. In passive tubular reabsorption, which includes diffusion and osmosis, substances move along their gradient without expenditure of energy.

Tubular secretion

The final process in urine formation is tubular secretion, which is essentially reabsorption in reverse. Substances such as hydrogen and potassium ions, creatinine, ammonia and organic acids move from the blood of the peritubular capillaries into the tubules themselves as filtrate. Thus, urine consists of both filtered and secreted substances. Tubular secretion is important for disposing of substances not already in the filtrate, such as medications. This process eliminates undesirable substances that have been reabsorbed by passive processes and rids the body of excessive potassium ions. It is also a vital force in the regulation of blood pH.

Maintaining normal composition and volume of urine

Maintaining the normal composition and volume of urine involves a countercurrent exchange system. In this system, fluid flows in opposite directions through the parallel tubes of the loop of Henle and the vasa recta, tiny capillaries that run along the loop of Henle. Fluid is exchanged across these parallel membranes in response to a concentration gradient (see Figure 25.5). When the filtrate enters the proximal convoluted tubule, its osmolality (300 mOsm/kg) is the same as plasma and the interstitial fluid of the renal cortex. Note the following steps in the process:

1. The descending loop of Henle is highly permeable to water and allows chloride and sodium to enter the loop through diffusion. The hyperosmotic interstitium causes water to move out of the descending loop, so remaining filtrate becomes increasingly concentrated.
2. The lumen of the ascending loop of Henle is impermeable to water but allows chloride and sodium to move out into the interstitium of the medulla. As a result, the filtrate in the ascending loop becomes hypoosmotic and the medullary interstitium becomes hyperosmotic.
3. As the filtrate progresses through the ascending limb of the loop of Henle and enters the distal convoluted tubule, sodium and chloride are removed and water is retained. Thus, the filtrate becomes more dilute.
4. As the filtrate passes through the deep medullary regions, urea (an end product of protein metabolism and, along with water, the main constituent of urine) begins to diffuse out from the collecting tubules into the interstitial space and establishes a concentration gradient to facilitate water movement.
5. Some urea enters the ascending loop of Henle. Urea entering the vasa recta typically diffuses out again.

The dilution or concentration of urine is largely determined by the action of antidiuretic hormone (ADH), which is secreted by the posterior pituitary gland. ADH causes the pores of the collecting tubules to enlarge, so that increased amounts of water move into the interstitial space. As the end result, water is reabsorbed and urine is more highly concentrated. When ADH is not secreted, the filtrate passes through the system without further water reabsorption, so that the urine is more dilute.

Urine is composed, by volume, of about 95% water and 5% solutes. The largest component of urine by weight is urea. Other solutes normally excreted in the urine include sodium, potassium, phosphate, sulfate, creatinine, uric acid, calcium, magnesium and bicarbonate.

Clearing waste products

The kidneys excrete water-soluble waste products and other chemicals or substances from the body. This process is called renal plasma clearance, which refers to the ability of the kidneys to clear (cleanse) a given amount of plasma of a particular substance in a given time (usually 1 minute). The kidneys clear 25 to 30 g of **urea** (a nitrogenous waste product

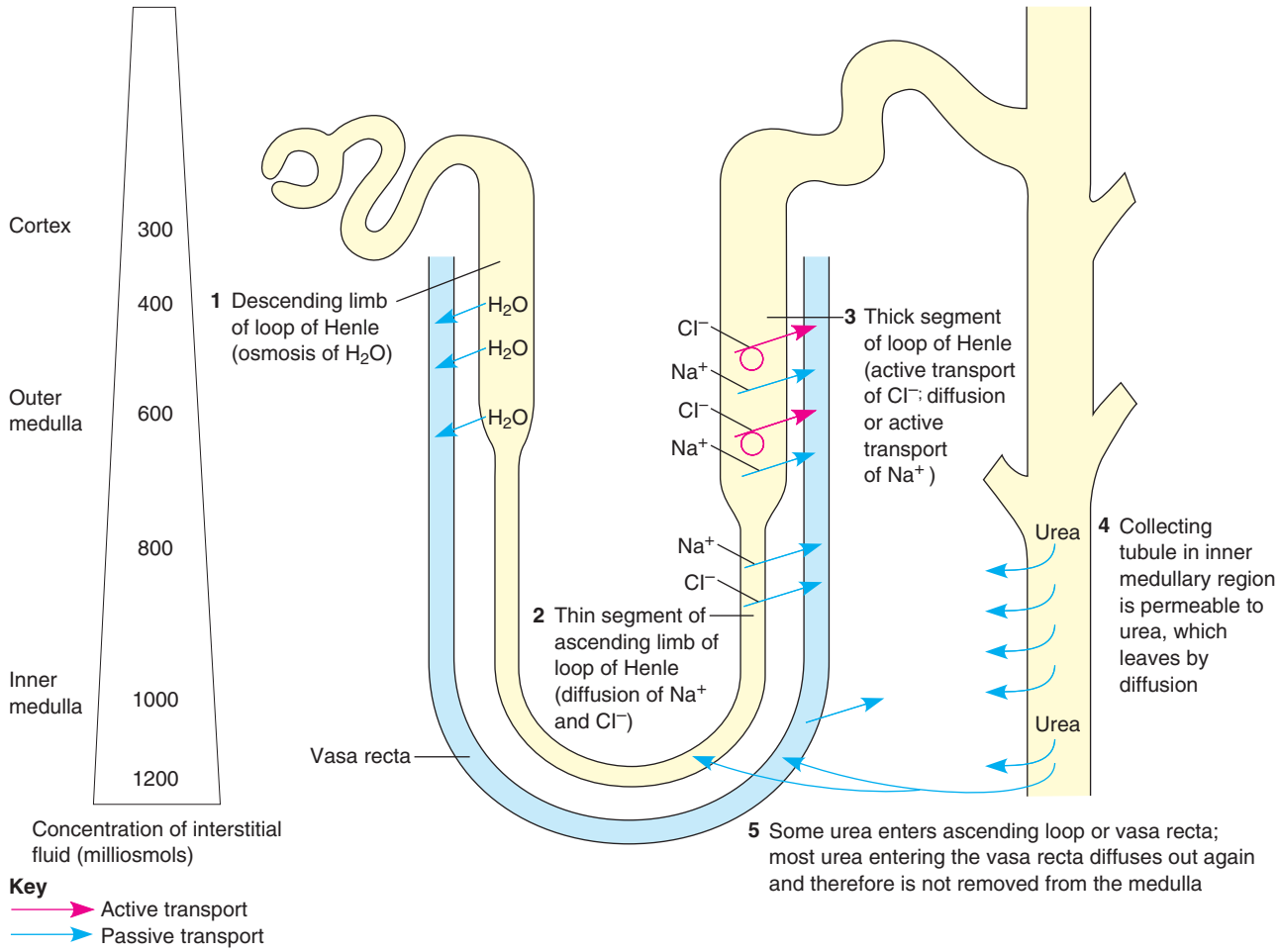


FIGURE 25.5 ■ The countercurrent exchange system is responsible for establishing and maintaining an osmotic gradient necessary to the composition, volume and pH of urine

formed in the liver from the breakdown of amino acids) each day. They also clear **creatinine** (an end product of creatine phosphate, found in skeletal muscle), uric acid (a metabolite of nucleic acid metabolism) and ammonia, as well as bacterial toxins and water-soluble drugs. Tests of renal clearance are often used to determine the GFR and glomerular damage.

Renal hormones

Hormones either activated or synthesised by the kidneys include the active form of vitamin D, erythropoietin and natriuretic hormone.

Vitamin D is necessary for the absorption of calcium and phosphate by the small intestine. In an inactive form, vitamin D enters the body either by dietary intake or through the action of ultraviolet rays on cholesterol in the skin. Activation occurs in two steps, the first in the liver and the second in the kidneys. The renal step is stimulated by parathyroid hormone, which in turn responds to a decreased plasma calcium level. High calcium and phosphate levels may lead to calcium deposits in the small capillaries in the eyes, lungs and heart. Calcium deposits increase the risk of cardiovascular disease over time (KHA, 2012).

Erythropoietin (EPO) stimulates the bone marrow to produce red blood cells in response to tissue hypoxia. The stimulus for the production of erythropoietin by the kidneys is decreased oxygen delivery to kidney cells.

The right atria of the heart releases natriuretic hormone in response to increased volume and stretch, as occurs in increased extracellular volume. This hormone inhibits ADH secretion, so that the collecting tubules are less porous and a large amount of dilute urine is produced.

THE URETERS

The ureters are bilateral tubes approximately 25 to 30 cm long. They transport urine from the kidney to the bladder through peristaltic waves originating in the renal pelvis. The wall of the ureter has three layers: an inner epithelial mucosa, a middle layer of smooth muscle and an outer layer of fibrous connective tissue.

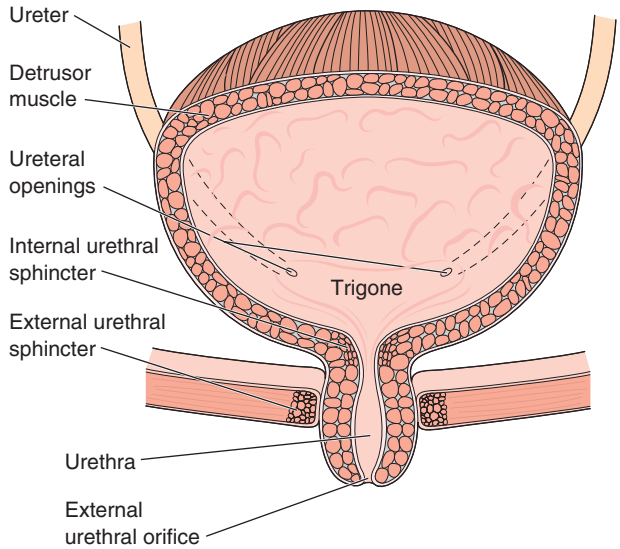


FIGURE 25.6 ■ Internal view of the urinary bladder and trigone

THE URINARY BLADDER

The urinary bladder is posterior to the pubic symphysis and serves as a storage site for urine. In males, the bladder lies immediately in front of the rectum; in females, the bladder lies in front of the vagina and the uterus. Openings for the ureters and the urethra are inside the bladder: the trigone is the smooth triangular portion of the base of the bladder outlined by these three openings (see Figure 25.6).

The layers of the bladder wall (from internal to external) are the epithelial mucosa lining the inside, the connective tissue submucosa, the smooth muscle layer and the fibrous outer layer. The muscle layer, called the detrusor muscle, consists of fibres arranged in inner and outer longitudinal layers and in a middle circular layer. This arrangement allows the bladder to expand or contract according to the amount of urine it holds.

The size of the bladder varies with the amount of urine it contains. In healthy adults, the bladder holds about 300 to 500 mL of urine before internal pressure rises and signals the need to empty the bladder through **micturition** (also called urination or voiding). However, the bladder can hold more than twice that amount if necessary. The bladder has an internal urethral sphincter that relaxes in response to a full bladder and signals the need to urinate. A second external urethral sphincter is formed by skeletal muscle and is under voluntary control.

THE URETHRA

The urethra is a thin-walled muscular tube that channels urine to the outside of the body. It extends from the base of the bladder to the external urinary meatus. In females, the urethra is approximately 3 to 5 cm long and the urinary meatus is anterior to the vaginal orifice. In males, the urethra is approximately 20 cm long and serves as a channel for semen as well as urine. The prostate gland encircles the urethra at the base of the bladder in males. The male urinary meatus is located at the end of the glans penis.

HEALTH ASSESSMENT, DIAGNOSTICS AND DOCUMENTATION

Renal system function is assessed by findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests. Sample documentation of an assessment of renal system function is included in the box below.

Health assessment interview

A health assessment interview to determine problems with urinary structure and function may be conducted during a health screening, may focus on a chief complaint (such as burning on urination or difficulty starting the stream when urinating) or may be part of a total health assessment. As with alterations in bowel function, people with problems with renal system function may be embarrassed to talk about urinary elimination patterns. It is often helpful to discuss less personal information first.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 9:
Recognising and Responding to Clinical Deterioration in Acute Health Care.

Essential element 4: Clinical communication

'Patient, family and carer concerns about possible deterioration are valued and acted on by the clinicians.' (Australian Commission on Safety and Quality in Health Care (ACSQHC), 2011)

People, their families and carers are well placed to identify the subtle signs of changes in a person's health. Identify opportunities for open communication such as conducting assessments in a private environment, discussing less personal information first, regular communication checks during healthcare team rounds and establishing an agreed communication process (written and verbal).

Source: © Australian Commission on Safety and Quality in Health Care.

SAMPLE DOCUMENTATION

Assessment of renal system function

10/7/2014 NURS 1030 hrs — Home visit made to 66-year-old woman with end-stage chronic kidney disease. Skin pale and oral mucous membranes dry. 4+ oedema in ankles and feet. Eyelids swollen. Skin tight and shiny over abdomen and bilateral lower extremities. Abdomen distended and tender on light palpation; further palpation deferred. Urinary bladder not palpable. Urine output for past 24 hours is 15 mL.———— RN A Lam (A LAM RN)

Current urinary status should include the following data:

- colour, odour and amount of urine
- difficulty initiating a stream of urine
- frequency of urination
- painful urination (**dysuria**)
- excessive urination at night (**nocturia**)
- blood in the urine (**haematuria**)
- voiding scant amounts of urine (**oliguria**)
- voiding excessive amounts of urine (**polyuria**)
- discharge
- flank pain.

If you identify a problem with urinary elimination, analyse its onset, characteristics and course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, you may ask the following questions:

- Have you noticed any burning when you pass urine?
- Do you have difficulty starting to urinate?
- When did you first notice that you were unable to control the loss of urine from your bladder?

Further explore any abnormalities in the person's current renal status. Focus questions on changes in patterns of urination, changes in the urine and pain.

Assess changes in patterns of urination by asking the person: How many times a day do you urinate? Do you feel that you empty your bladder each time? How many times do you get up at night to urinate? Do you experience a very strong desire to urinate and feel that you just cannot wait? Have you noticed that you urinate small amounts of dark, strong-smelling urine?

Changes in the urine that should be explored include the presence of blood or a cloudy appearance of the urine. If the person has noticed blood, explore the use of medications (such as anticoagulants or dye-containing drugs) and other bleeding problems. Women may not understand that blood in the toilet or on toilet tissue after urination is normal during menstruation. Cloudy, foul-smelling urine often indicates infection (**pyuria**); ask the person about temperature elevations, chills and general malaise. Cloudy urine in men may result from retrograde ejaculation (when semen is discharged into the bladder instead of from the penis) during intercourse.

If the person reports pain, explore its location, duration and intensity. Kidney pain is experienced in the back and the costovertebral angle (the angle between the lower ribs and adjacent vertebrae) and may spread towards the umbilicus. Renal colic (pain in response to renal calculi moving through the ureter) is severe, sharp, stabbing and excruciating; often it is felt in the flank, bladder, urethra, testes or ovaries. Bladder and urethral pain is usually dull and continuous but may be experienced as spasms. The person with a distended bladder experiences constant pain increased by any pressure over the bladder.

Information about surgeries or other treatment of previous urinary problems is essential to the health history, as is a family history of altered structure or function. A family history of renal problems may be the first clue to abnormalities in the person's renal function. Explore information regarding family occurrence of end-stage renal disease, renal calculi and frequent infections, as well as related problems such as hypertension and diabetes mellitus.

Questions about lifestyle, diet and work history should explore cigarette smoking and/or exposure to toxic chemicals (to identify risks for cancer), usual fluid intake, type of fluid intake and self-care measures to replace fluids lost during work or physical activity in hot temperatures.

Interview questions categorised by functional health patterns are listed in the box below.

Physical assessment

The structure and function of the renal system is assessed by examination of the skin, abdomen, kidneys, bladder and urinary meatus. Guidelines for abdominal assessment are outlined in Chapter 20. Normal age-related findings for the older adult are summarised in Table 25.2. More information on the age-related changes in kidney function is presented in Table 27.2.

Physical assessment of the renal system may be performed as part of a total health assessment, as part of an abdominal assessment or as part of a back examination (for the kidneys). The techniques of inspection, auscultation, palpation and percussion are used.

Before beginning the assessment, ask the person to provide a clean urine specimen. Assess the specimen for colour, odour and clarity before sending to the laboratory.

Prior to the examination, collect all necessary equipment and explain the techniques to the person to decrease anxiety. Because the examination involves exposure of the genital area, give the person a gown and drape them appropriately to minimise exposure.

Guidelines for percussion and palpation of the kidneys are outlined in Box 25.1.

CONSIDERATION FOR PRACTICE

Auscultate immediately after inspection, because percussion or palpation may increase bowel motility and interfere with sound transmission during auscultation.

TABLE 25.2 Age-related renal system changes

AGE-RELATED CHANGE	SIGNIFICANCE
Kidneys: ↓ loss of renal mass, size of renal cortex and number of nephrons, ↓ growth of renal tissue, ↑ risk of atherosclerosis, all of which may result in atrophy of the kidneys (Abdel-Kader & Palevsky, 2009).	• Decreased renal blood flow. • Decreased GFR by about 50% between ages 20 and 90. eGFR of < 60 mL/min/1.73 m^2 is very common in older people (KHA, 2012).
Renal tubules: ↓ function, with less effective exchange of substances, water and sodium conservation, and ↓ plasma renin and aldosterone levels; ↑ vasoconstrictive response to stimuli (e.g. volume depletion) (Abdel-Kader & Palevsky, 2009).	• Risk of dehydration, hyponatraemia, hyperkalaemia, and nocturia. • Effects of medications may be altered (with decreased filtration). • Decreased reabsorption of glucose may result in proteinuria and glycosuria, which are not of major clinical significance.
Bladder: • Muscles weaken and bladder capacity decreases. • More difficult to empty bladder. • Delayed micturition reflex. Prostatic gland enlargement compressing the urethra (Martini et al., 2012).	• Urinary frequency, urgency and nocturia are more common with ageing. • Larger amounts of residual urine present after voiding. • Some stress incontinence may occur, especially in women who have had several children. • Urinary incontinence is not a normal outcome of ageing. • Urinary retention is more common.

Diagnostic tests

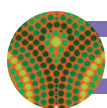
The results of diagnostic tests of renal system function are used to support the diagnosis of a specific disease, to provide information to identify or modify the appropriate medication or therapy, and to monitor the person's responses to treatment and nursing care interventions. Diagnostic tests used to assess the structures and functions of the renal system are described in the table below and summarised in the bulleted list that follows. More information is included in the discussion of specific disorders in Chapters 26 and 27.

- Urine may be tested through routine analysis, a urine culture, a post-voiding residual urine and a 24-hour collection for creatinine. Results of these tests include findings to serve as baseline data, to support diagnosis of various health problems, to evaluate the ability to empty the bladder of urine and to evaluate renal function.
- The ability to empty the bladder of urine may be evaluated by a portable bladder scan to evaluate for residual urine, uroflowmetry to measure the volume of urine voided per second and a cystometrogram (CMG) to evaluate bladder capacity, neuromuscular functions of the bladder, urethral pressures and causes of bladder dysfunction.
- Radiological examinations include an intravenous pyelogram, a retrograde pyelogram and a renal arteriogram or angiogram. These examinations are useful in visualising (via x-ray film) the urinary tract to identify abnormal size, shape and function of the kidneys, the kidney pelvis and ureters; and to detect renal **calculi** (stones), tumours or cysts.
- A cystoscopy allows direct visualisation of the bladder wall and urethra. During the procedure small stones can be removed, a sample of tissue may be taken for biopsy and a retrograde pyelogram may be done at the same time. If a contrast dye is instilled in the bladder, fistulas, tumours or ruptures can be identified.
- Non-invasive tests include a renal ultrasound, computed tomography (CT) scan, magnetic resonance imaging (MRI) and renal scan. These tests are used to identify and evaluate kidney size and structure, as well as renal or perirenal masses and obstructions. In addition, a renal scan may be used to evaluate kidney blood flow, perfusion and urine production.
- A kidney biopsy is done to obtain tissue to diagnose or monitor kidney disease.

The nurse should explain the procedure and required preparation, assess medication use that may affect the outcome of the tests, support the person during the examination as necessary, and document and monitor the results of the tests.

Genetic considerations

When conducting a health assessment interview and physical assessment consider the genetic influences on adult health (see 'Genetic considerations' below). Ask if family members have health problems affecting kidney function such as polycystic disease or diabetes mellitus. If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 8 provides further information about genetics in medical–surgical nursing.



GENETIC CONSIDERATIONS Renal system

- Adult polycystic kidney disease (APKD) is characterised by large cysts in one or both kidneys and a gradual loss of kidney tissue with resultant chronic kidney disease. There are three recognised forms: ~85% are called APKD1 and are linked to a familial chromosome 16 disorder; APKD2 (~15%) have an abnormality on chromosome 4; and the gene locus of APKD3 (rare) is yet to be identified.
- Chronic kidney disease (CKD) may be a complication of type 1 and type 2 diabetes mellitus (DM), but is seen more often in people with type 1 DM. Type 1 and type 2 DM are classified as multifactorial inheritance disorders because both genetic and environmental factors are necessary for onset of the disorder.

FUNCTIONAL HEALTH PATTERN INTERVIEW Renal system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Have you ever had a bladder or kidney disease, injury or surgery? Describe. ■ If so, how was the problem treated? ■ Describe your usual intake of fluids for a 24-hour period. What type of fluids do you drink? ■ Have you ever smoked? If so, how many cigarettes per day? ■ Describe the problem you are having with your kidneys or bladder. ■ Are you taking medications for this or any other health problem? If so, what do you take and how often? ■ *For women*: Describe how you care for yourself when you urinate (e.g. direction of wiping with tissue). ■ If you have a surgical diversion of urine, describe how you care for yourself. (What skin and appliance care do you use? How often do you empty the bag?) ■ Do you wear or have you ever worn an external catheter, indwelling catheter or incontinence briefs? Explain. ■ Have you ever done self-catheterisation? If so, why and how often?
Nutritional–Metabolic	■ How much coffee, tea or alcohol do you drink in a 24-hour period? ■ Have you ever limited your fluid intake? Explain. ■ Do you limit the amount of salt you eat? Explain. ■ Do you have swelling in your ankles? If so, what do you do?
Elimination	■ How many times a day do you urinate? Do you have to get up at night to urinate? Has there been a change in your usual pattern of urination? ■ Do you experience a sudden urge to urinate? ■ Has there been a change in your urine, such as a change in amount, colour or odour? Have you ever noticed blood in your urine or on the tissue after you wipe? ■ Is it difficult for you to begin or end your flow of urine? ■ Have you ever had problems controlling your urine when you laugh, sneeze or cough? ■ Do you have any discharge from your urethra? Explain.
Activity–Exercise	■ Do your urinary problems interfere with your activities of daily living? Explain. ■ Describe your usual energy level. Has there been a change? Explain. ■ Have you ever been taught to do Kegel exercises to help you control your urination? If so, how often do you practise these?
Sleep–Rest	■ Does a problem with urination interfere with your ability to sleep and rest? Explain. ■ Has there been a change in the number of times you wake up at night to urinate? Explain.
Cognitive–Perceptual	■ Do you have any pain or burning when you urinate? ■ Have you experienced any tenderness or pain over the lower sides of your back or severe pain that spreads over your lower abdomen? If so, describe its location, intensity, aggravating factors and duration.
Self-perception–Self-concept	■ How does having this condition make you feel about yourself?
Role–Relationships	■ How does having this condition affect your relationships with others?
Sexuality–Reproductive	■ Has this condition interfered with your usual sexual activity?
Coping–Stress–Tolerance	■ Has having this condition created stress for you? ■ Have you experienced any kind of stress that makes this condition worse? Explain. ■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments that you would not use to treat this condition?

BOX 25.1 Guidelines for physical assessment of the kidneys

Percussion of the kidneys

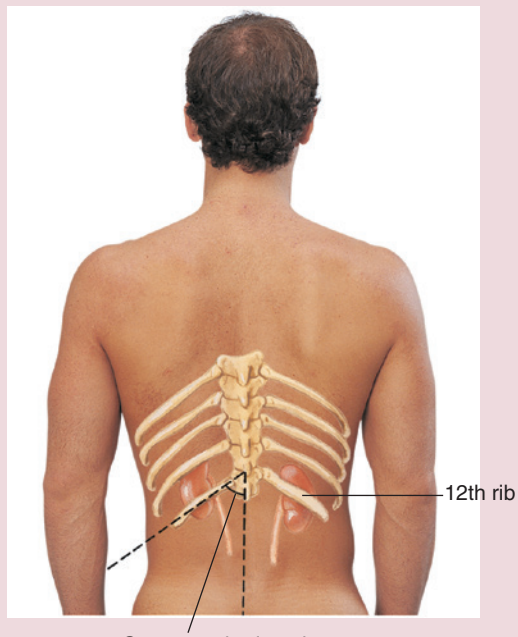
Percussion of the kidneys helps assess pain or tenderness. Assist the person to a sitting position and stand behind them. For indirect percussion, place the palm of your non-dominant hand over the costovertebral angle (see Figure A). Strike this area with the ulnar surface of your dominant hand, curled into a fist (see Figure B). For direct percussion, also strike the area over the costovertebral angle with the ulnar surface of your dominant hand, curled into a fist. Repeat the technique for the other kidney.

You should do percussion of the kidneys with only enough force so the person feels a gentle thud. Percussion is usually done at the end of the assessment.

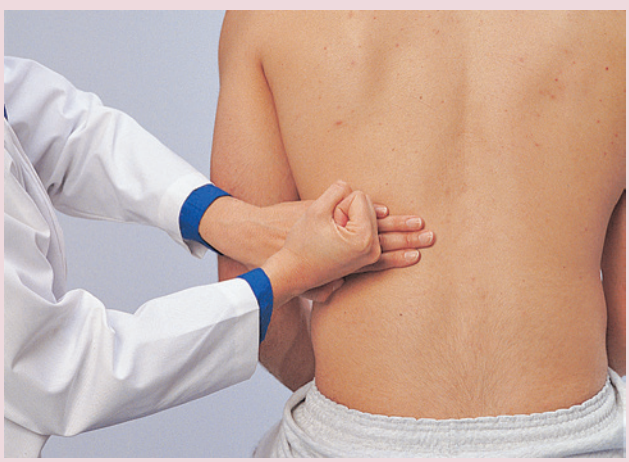
Palpation of the kidneys

Although the technique of palpation of the kidneys is outlined here, this technique is best performed by an advanced practitioner, because it involves deep palpation. In addition, the kidneys are difficult to palpate.

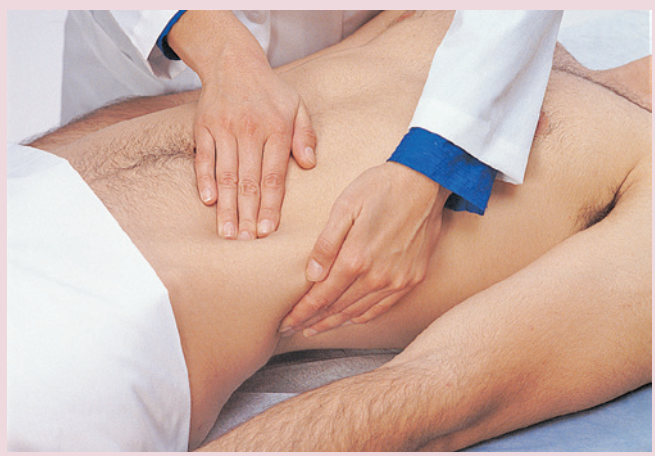
Assist the person to the supine position and stand at their right side. To palpate the left kidney, reach across the person and place your left hand under their left flank with your palm upward. Elevate the left flank with your fingers, displacing the kidney upward. Ask the person to take a deep breath and use the palmar surface of your right hand to palpate the kidney (see Figure C). Repeat the technique for the right kidney.



A Location of the kidneys and the costovertebral angle



B Percussing the kidney



C Palpating the left kidney

DIAGNOSTIC TESTS Renal system disorders

NAME OF TEST Serum urea

PURPOSE AND DESCRIPTION This blood test measures urea, the end product of protein metabolism. Increased levels may result from dehydration, vomiting, diarrhoea, GI bleeding, renal impairment, excessive protein catabolism or congestive cardiac failure. It may be decreased with protein deficiency in diet, dieresis and pregnancy.

Normal values: 3.0–8.0 mmol/L

RELATED NURSING CARE No special preparation is needed. If values are increased in a person who is dehydrated, they should return to normal with hydration. If not, this is an indicator of renal impairment.

(continued)

DIAGNOSTIC TESTS Renal system disorders (continued)

NAME OF TEST Serum creatinine

PURPOSE AND DESCRIPTION This blood test is used to diagnose renal impairment. Creatinine is a by-product of the breakdown of muscle and is excreted by the kidneys. Serum creatinine levels may rise when 50% or more nephrons are destroyed or there is acute muscle wasting. Levels may fall with chronic muscle wastage (such as muscular dystrophy, muscle sclerosis).

Normal values: Serum (adult female): 0.05–0.11. Serum (adult male): 0.06–0.12 mmol/L.

(Creatinine varies with size and muscle mass. Older adults may have decreased values due to decreased muscle mass.)

RELATED NURSING CARE No special preparation is needed. Values may be increased by antibiotics, ascorbic acid, L-dopa, methyldopa and lithium carbonate. Values are not affected by hydration status. Three to four hours after eating meat the plasma creatinine can double, so fasting prior to measurements may be recommended.

NAME OF TEST Estimated glomerular filtration rate (eGFR)

PURPOSE AND DESCRIPTION GFR is the best measurement of kidney function; the eGFR can estimate the GFR.

Normal values: GFR < 60 mL/min/1.73m^2 for 3 months. Normal findings and abnormal findings with causes are outlined in Table 25.1.

RELATED NURSING CARE eGFR may be unreliable if diet is vegetarian, high in protein or includes creatine supplements; in people with large muscle mass and amputees; in children under 18 years; and in those with severe liver disease.

NAME OF TEST Routine urinalysis (UA) (dipstick or ward urinalysis)

PURPOSE AND DESCRIPTION An examination of the constituents of a sample of urine to establish a baseline, to provide data for diagnosis or to monitor results of treatment. Normal findings and abnormal findings with causes are outlined in Table 25.1.

RELATED NURSING CARE Provide a clean specimen cup for a sample of urine. Note if the person is menstruating. Assess medications, fluid status and foods that might affect urinalysis results.

NAME OF TEST Urine culture (microscopy urine (MSU), clean-catch)

PURPOSE AND DESCRIPTION A culture of a urine sample to identify the causative organism of a urinary tract infection (UTI).

Normal value: < 10 000 organisms/mL (urine is sterile but urethra contains bacteria and a few WBCs).

Values of > 100 000 organisms/mL indicate UTI.

RELATED NURSING CARE Provide the person with sterile container. Ask women to separate labia with one hand and clean labia with other hand, using sterile cotton sponges saturated with a cleansing solution (may be 0.9% sodium chloride) and wiping three times front to back. Ask men to retract the foreskin and cleanse glans with three cotton sponges saturated with a cleansing solution, using a circular motion. After cleaning, ask the person to begin voiding and then collect specimen in the container (initial voiding will contain urethral contaminants). If the person is unable to void, it may be necessary to obtain a specimen with a urinary catheterisation.

NAME OF TEST Urinary albumin:creatinine ratio (ACR)

PURPOSE AND DESCRIPTION Used to detect albumin (a protein) in the urine. A marker of kidney damage is excessive amounts of albumin in the urine (**albuminuria**). The presence of albuminuria is determined by the albumin-to-creatinine ratio (ACR). Low levels of albumin in the urine are normal; the prevalence increases sharply from 65 years of age onwards (DHV, 2012). Other causes of transient elevations in urinary ACR include UTIs, diurnal variations in urinary excretion, non-steroidal anti-inflammatory drugs (NSAIDs), fluid overload and acute febrile illness (Phoon, 2012). Elevated ACR may occur when kidney damage is present.

Normal value: < 3.5 mg/mmol (Salem & Harvie, n.d.).

Albuminuria—Male: > 2.5 mg/mmol; Female: > 3.5mg/mmol

Microalbuminuria—Male: 2.5–25 mg/mmol; Female: 3.5–35 mg/mmol

Macroalbuminuria (moderate to severe CKD)—Male: > 25 mg/mmol; Female: > 35 mg/mmol (Salem & Harvie, n.d.; KHA 2015).

RELATED NURSING CARE This should be a first-void spot specimen. Albuminuria is said to be present if two out of three ACR results are positive (KHA, 2012). A routine 'dipstick' for protein in the urine is not recommended to investigate for CKD.

DIAGNOSTICTESTS Renal system disorders (continued)

NAME OF TEST Portable ultrasonic bladder scan

PURPOSE AND DESCRIPTION Used to obtain information about bladder volume or residual urine left in the bladder after voiding. Where possible, place the person in a supine position. Warm ultrasound gel and apply over the lower abdomen. The ultrasound probe is placed just above the pubic bone. Select 'male' on the machine if the person has had a hysterectomy. The scanner shows an outline of the bladder and displays the amount of urine in the bladder in millilitres. Obtain several readings and use the largest (the most accurate). Print the information, place it on the person's chart and document the residual urine amount.

RELATED NURSING CARE No special preparation is needed, but the test is usually not used for pregnant women. Remember the machine will measure any fluid collection in the suprapubic region such as ascites, haematoma or lymphocele. Report and monitor a residual of more than 100 mL or if the residual is greater than the amount voided. A urinary catheter may have to be inserted if the volume is greater than 600 mL, or if the person has not voided for 5 to 6 hours or is uncomfortable.

NAME OF TEST Creatinine clearance

PURPOSE AND DESCRIPTION A 24-hour urine test and a blood sample (collected at either the end or beginning of the 24-hour urine collection) are used to identify renal dysfunction and to monitor renal function.

Normal value: 90–125 mL/min

Calculation of estimated glomerular function rate (eGFR), using a single blood sample, is another straightforward method of estimating kidney function.

RELATED NURSING CARE Assess medications: drugs that may decrease the creatinine clearance measurement are steroids, aminoglycosides, thiazides, cimetidine, cisplatin and cephalosporins. Ascorbic acid, steroids, methyldopa (Aldomet), cefoxitin and diuretics can increase the result. Levels of creatinine are elevated in hypothyroidism, hypertension, pregnancy and exercise.

Obtain appropriate specimen container. Ask person to void and discard first voiding. Instruct the person, family and staff to then save all urine for a clearly designated 24-hour period, keeping the specimen in the fridge. If the person misses a sample, the test is invalid.

NAME OF TEST Uroflowmetry

PURPOSE AND DESCRIPTION This test measures the volume passed per second, maximum and average flow rate, and voiding time.

RELATED NURSING CARE Ask the person to increase fluid intake and refrain from voiding for several hours before the test to ensure a full bladder and a strong urge to void during testing. Tell the person they will be asked to urinate into a funnel. Residual urine may be measured following the test.

NAME OF TEST Micturating cysto-urethrogram (cystometrogram (CMG))

PURPOSE AND DESCRIPTION Conducted to evaluate bladder capacity and neuromuscular functions of the bladder, urethral pressures and causes of bladder dysfunction. A measured quantity of fluid (contrast media) is instilled into the bladder (via urethral catheter) and the filling capacity and voiding pressures are measured. The catheter is removed and a series of x-ray pictures are taken as the person micturates. A catheter may also be placed in the rectum during the test.

RELATED NURSING CARE Tell the person that the bladder will be filled and during filling they will be asked to describe the first urge to void and the sensation of being unable to delay urination any longer.

NAME OF TEST Intravenous pyelogram (IVP)

PURPOSE AND DESCRIPTION This radiological examination is done to visualise the entire urinary tract to identify abnormal size, shape and function of the kidneys, or to detect renal calculi (stones), tumours or cysts. A urinary catheter is placed into the bladder and a radiopaque substance is injected intravenously and a series of x-rays taken.

PERSON PREPARATION

- Assess knowledge and understanding of procedure, clarifying information as needed. The person may have to fast (from food); clear fluids allowed up to 2 hours prior to the procedure.
- Schedule IVP prior to any ordered barium test or gallbladder studies using contrast material.
- Assess if the person has any form of reaction to x-ray contrast dye, or an iodine or seafood allergy, or if they are asthmatic.
- Verify the presence of a signed consent for the procedure.
- Assess renal and fluid status, including serum osmolality, creatinine and urea results. Notify the doctor of any abnormal values.
- Instruct the person to follow preparation protocol. This may include pretest bowel preparation and fasting for 3 hours prior to the procedure.
- Obtain baseline vital signs and record.

(continued)

DIAGNOSTIC TESTS Renal system disorders (continued)

AFTER THE TEST

- Monitor vital signs and urine output.
- Report manifestations of delayed reaction to the contrast media such as dyspnoea, tachycardia, itching, hives or flushing.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- X-rays and a dye that is rapidly excreted in the urine are used to show the structures of the kidney, ureters and bladder. The test takes about 30 minutes.
- As the dye is injected, you may feel a transient warm flushing sensation along with possible nausea and a metallic taste.
- A wide band may be placed around your waist at some stage and tightened for a few minutes to stop the contrast from draining to the bladder through the ureters (the tubes joining the kidneys to the bladder).
- Notify your doctor immediately if you develop a rash, difficulty breathing, rapid heart rate or hives during or after the test.
- Increase fluid intake after the test is completed.

NAME OF TEST Retrograde pyelogram

PURPOSE AND DESCRIPTION This radiological test is done to evaluate the structures of the ureters and kidney pelvis. It may be performed alone or in conjunction with a cystoscopy. A contrast dye is injected through a catheter into the ureters and kidney pelvis and x-rays are taken.

RELATED NURSING CARE Nursing care for the person having a retrograde pyelogram is the same as that for people having an IVP.

NAME OF TEST Renal arteriogram or angiogram

PURPOSE AND DESCRIPTION This radiological test is done to visualise renal blood vessels in order to detect renal artery stenosis, renal thrombosis or embolism, tumours, cysts or aneurysm; to determine the causative factor for hypertension; and to evaluate renal circulation. A contrast medium is injected into the femoral artery.

RELATED NURSING CARE Assess for allergy to iodine, seafood or other contrast dye from other x-ray procedures. A laxative or cleansing enema may be required the night before and the person should be fasted for 8–12 hours prior to the test. Results may be affected by faeces, gas and barium sulfate. Anticoagulants should be discontinued.

After the test, pressure will be applied to the insertion site for up to 20 minutes to prevent bleeding. Continue to monitor for bleeding from the femoral artery, restrict activity for a day, assess peripheral pulses and monitor urine output.

NAME OF TEST Cystoscopy (cystogram), cystography

PURPOSE AND DESCRIPTION Direct visualisation of the bladder wall and urethra is accomplished by using a cystoscope. During the procedure small renal calculi can be removed from the ureter, bladder or urethra and tissue biopsy can be done. It also permits determination of the cause of haematuria or UTI. A stent may be inserted during the procedure to facilitate urinary drainage past an obstruction. A retrograde pyelogram may be done during the cystoscopy. By instilling a contrast dye into the bladder (*cystography*), neurogenic bladder, fistulas, tumours or ruptures can be identified.

PERSON PREPARATION

- Assess knowledge and understanding of the procedure, clarifying information as needed.
- Verify the presence of a signed consent for the procedure.
- Instruct in pre-test preparation as ordered, including prescribed laxatives the evening prior to the test and any ordered food or fluid restrictions.
- Administer sedation and other medications as ordered prior to the test.
- Immediately notify the doctor if your urine remains bloody for more than three voidings after the procedure or if you develop bright bleeding, low urine output, abdominal or flank pain, chills or fever.
- Warm baths, analgesic agents and antispasmodic medications may relieve discomfort after the procedure.
- Increase fluid intake to decrease pain and difficulty voiding and reduce the risk of infection.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Cystoscopy is performed using either a local or general anaesthetic. You may feel some pressure or a need to urinate as the scope is inserted through the urethra into the bladder. The procedure takes approximately 15 to 30 minutes.
- Do not attempt to stand without assistance immediately after the procedure because you may feel dizzy or faint.
- Burning on urination for a day or two after the procedure is to be expected.

NAME OF TEST Renal ultrasound

PURPOSE AND DESCRIPTION This non-invasive test is conducted to detect renal or perirenal masses, identify obstructions and diagnose renal cysts and solid masses. It is done by applying a conductive gel to the skin and placing a small external ultrasound probe on the person's skin. Sound waves are recorded on a computer as they are reflected off tissues.

RELATED NURSING CARE No special preparation is indicated.

DIAGNOSTICTESTS Renal system disorders (continued)

NAME OF TEST CT scan of kidneys

PURPOSE AND DESCRIPTION The CT scan allows evaluation of kidney size, tumours, abscesses, suprarenal masses and obstructions. A contrast dye is injected intravenously, allowing increased visualisation of the density of renal tissue and masses in comparison to an ultrasound.

RELATED NURSING CARE Assess the person for allergies to iodine, x-ray contrast dye and seafood. Tell the person to fast for 4 hours prior to the test and that laxatives or enemas may be ordered to remove gas or faecal material from the bowel.

NAME OF TEST MRI of the kidneys

PURPOSE AND DESCRIPTION An MRI is used to visualise the kidneys by assessing computer-generated films of radiofrequency waves and changes in magnetic fields.

RELATED NURSING CARE Ask the person to remove all metal objects. Assess for metal implants. (Test may not be conducted if present.)

NAME OF TEST Renal scan

PURPOSE AND DESCRIPTION This test is done to evaluate kidney blood flow, location, size and shape; and to assess kidney perfusion and urine production. Radioactive isotopes are injected intravenously and radiation detector probes are placed over the kidneys to monitor activity in the kidneys. Radioisotope distribution in the kidneys is scanned and graphed. Non-functioning tissue, such as in tumours and cysts, appears as cold spots.

RELATED NURSING CARE Ask the person to drink several glasses of water prior to the test. Obtain weight and have the person void. After the procedure, increase fluid intake.

NAME OF TEST Renal biopsy

PURPOSE AND DESCRIPTION A renal biopsy is done to obtain tissue to diagnose or monitor kidney disease. The test is usually done by inserting a needle through the skin into the lower lobe of the kidney. It can also be done with CT, fluroscopy or ultrasound guidance.

PERSON PREPARATION

- Informed consent is required for a kidney biopsy. Answer questions and provide additional information as needed.
- Fast for 4 to 6 hours prior to the procedure.
- Continue to take all medications EXCEPT anticoagulants.
- Note haemoglobin, haematocrit and coagulation profile prior to the procedure.
- If the procedure is to be performed at the bedside, obtain biopsy tray and other necessary supplies.
- Following the procedure, apply a pressure dressing and position supine, for up to 4 hours, to help maintain pressure on the biopsy site.
- Monitor closely for bleeding during the first 24 hours after the procedure:
  a. Check vital signs frequently. Notify the doctor of tachycardia, hypotension or other signs of bleeding or shock.
  b. Monitor biopsy site for bleeding.
  c. Check haemoglobin and haematocrit, comparing with preprocedure values.
  d. Observe for and report complaints of flank or back pain, shoulder pain (caused by diaphragmatic irritation if haemorrhage occurs), pallor, light headedness.
  e. Monitor urine output for quantity and haematuria. Initial haematuria should clear within 24 hours.
- Monitor for other potential complications such as inadvertent penetration of the liver or bowel. Report abdominal pain, guarding and decreased bowel sounds.
- Encourage fluids during the initial post-procedure period.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Local anaesthesia is used at the injection site. The procedure may be uncomfortable but should not be painful.
- When the needle is inserted, you will be instructed not to breathe to prevent kidney motion.
- The entire procedure takes approximately 10 minutes.
- Avoid coughing during the first 24 hours after the procedure. Strenuous activity such as heavy lifting may be prohibited for approximately 2 weeks after the procedure.
- Report any manifestations of complications, such as haemorrhage or urinary tract infection, to the doctor.

RENAL ASSESSMENTS

Technique/normal findings	Abnormal findings
Skin assessment Inspect the skin and mucous membranes, noting colour, turgor and excretions. *The colour of skin and mucous membranes should be even and appropriate to the person's age and race; skin should be dry with no visible excretions.*	■ Pallor of the skin and mucous membranes may indicate kidney disease with resultant anaemia. ■ Decreased turgor of the skin may indicate dehydration. ■ Oedema (generalised or in the lower extremities) may indicate fluid volume excess. (Changes in skin turgor may indicate renal insufficiency with either excess fluid loss or retention.) ■ An accumulation of uric acid crystals, called uraemic frost, may be seen on the skin of the person with late-stage kidney disease.
Abdominal assessment Inspect the abdomen, noting size, symmetry, masses or lumps, swelling, distension, glistening or skin tightness. *The abdomen should be slightly concave, symmetric, without distension or masses.*	■ Enlargements or asymmetry may indicate a hernia or superficial mass. ■ If the urinary bladder is distended, it rises above the pubic symphysis as a rounded mass. ■ Distension, glistening or skin tightness may be associated with fluid retention. ■ Ascites is an accumulation of fluid in the peritoneal cavity.
Urinary meatus assessment This technique is not part of a routine assessment, but it is an important component in caring for people with health problems of the renal system. Further discussion is included in Chapter 46. *For the male:* With the person in a sitting or standing position, compress the tip of the glans penis with your gloved hand to open the urinary meatus (see Figure 25.7). *For the female:* With the person in the dorsal lithotomy position, spread the labia with your gloved hand to expose the urinary meatus. *The urinary meatus should be midline and free of redness, lesions or discharge.*	■ Increased redness, swelling or discharge from the urinary meatus may indicate infection or sexually transmitted infection. ■ Ulceration of the urinary meatus may indicate a sexually transmitted infection. ■ Hypospadias is displacement of the urinary meatus to the ventral surface of the penis. ■ Epispadias is displacement of the urinary meatus to the dorsal surface of the penis.
Kidney assessment See Box 25.1 for assessment guidelines for percussion and palpation of the kidneys. Auscultate the renal arteries by placing the bell of the stethoscope lightly in the areas of the renal arteries, located in the left and right upper abdominal quadrants. *Bruits are not normally heard over the renal arteries.*	■ Systolic bruits ('whooshing' sounds) may indicate renal artery stenosis.
Percuss the kidneys for tenderness or pain. *No tenderness or pain should be elicited.*	■ Tenderness and pain on percussion of the costovertebral angle suggest glomerulonephritis or glomerulonephrosis.
Palpate the kidneys. The lower pole of the right kidney may be palpable with deep palpation; the remaining right kidney and the left kidney are normally not palpable. *If palpable, they should be non-tender, bilaterally of appropriate size and density, without palpable masses.*	■ A mass or lump may indicate a tumour or cyst. ■ Tenderness or pain on palpation may suggest an inflammatory process. ■ A soft kidney that feels spongy may indicate chronic renal disease. ■ Bilaterally enlarged kidneys may suggest polycystic kidney disease. ■ Unequal kidney size may indicate hydronephrosis.

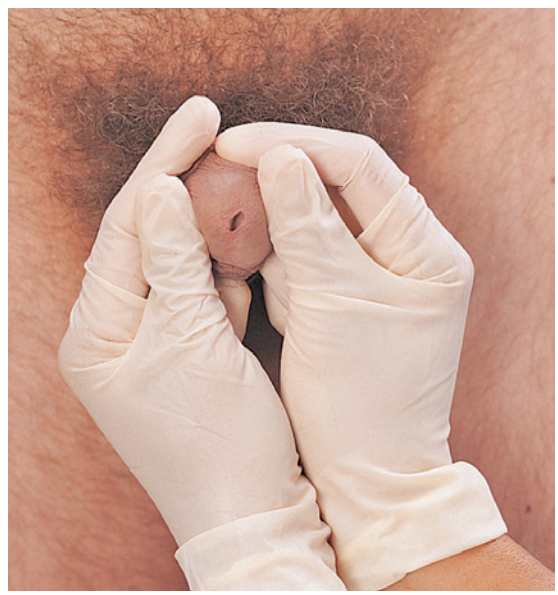


FIGURE 25.7 ■ Inspecting the urinary meatus of the male

RENAL ASSESSMENTS (continued)

Technique/normal findings	Abnormal findings
Bladder assessment	
Percuss the bladder for tone and position. *The bladder should be midline without dullness.*	■ Dull percussion tone over the bladder of a person who has just urinated may indicate urinary retention.
Palpate the bladder (over the pubic symphysis and abdomen) for distension. *The bladder is normally not palpable.*	■ Distended bladder may be palpated at any point from the pubic symphysis to the umbilicus and is felt as a firm, rounded organ. It indicates urinary retention.

CONCEPT CHECK

1 What part of the kidney processes the blood to make urine?
1 ureter
2 medulla
3 pyramids
4 nephrons

2 A person has been vomiting for 4 hours. What hormone is increased as a result?
1 thyroxine
2 renin
3 aldosterone
4 ADH

3 What diagnostic test can be used to determine GFR as well as glomerular damage?
1 routine urinalysis
2 renal scan
3 creatinine clearance
4 renal biopsy

4 What gland encircles the male urethra at the base of the bladder?
1 spleen
2 pancreas
3 prostate
4 adrenal

5 The person tells you of having to get up to void several times a night. You record this finding as:
1 polyuria
2 nocturia
3 dysuria
4 haematuria

6 Which question would you ask a person prior to an IVP?
1 'Are you allergic to shellfish?'
2 'Do you have burning on urination?'
3 'Have you ever had kidney stones?'
4 'Why are you having this test?'

7 Before beginning the physical assessment of the renal system, you should ask the person to:
1 empty the bladder
2 take several deep breaths
3 provide a urine specimen
4 drink several glasses of water

8 Following surgery, an older adult has not voided for 12 hours. What assessment should you make?
1 Palpate for bladder distension.
2 Auscultate for bowel sounds.
3 Inspect for oedema of the urethra.
4 Percuss for gastric tympany.

9 Of the following health problems an older woman may have, which is not normally a part of ageing of the renal system?
1 increased risk of haematuria
2 decreased risk of infection
3 urine that is darker in colour
4 urinary incontinence

10 What assessment would you use to assess hydration status of a person?
1 auscultation of renal arteries
2 palpation for skin turgor
3 percussion for dullness over bladder
4 palpation of both kidneys

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CHAPTER 26

NURSING CARE OF PEOPLE WITH URINARY TRACT DISORDERS

JENNIFER BORG, TRUDY DWYER, DIANE GOLDSWORTHY

LEARNING OUTCOMES

- Relate the interprofessional care and health management implications for a person with urinary tract infection to the pathophysiology of the condition.
- Explain the risk factors, course, pathophysiology and interventions required to competently care for a person with an obstruction of the urinary tract.
- Describe the interprofessional care and health management when caring for individuals experiencing various urinary flow disorders.

CLINICAL COMPETENCIES

- Assess the functional health status of people with urinary tract disorders, using data to determine priority nursing diagnoses and select individualised nursing interventions.
- Identify, report and document abnormal or unexpected assessments and their effect on personal status.
- Use evidence-based research to plan and implement nursing care for people with urinary tract disorders.
- Integrate the interprofessional plan of care into care for people with urinary tract disorders.
- Knowledgeably and safely administer prescribed medications and treatments for people with urinary tract disorders.
- Provide effective nursing care for people undergoing surgery of the urinary tract.
- Plan and provide appropriate teaching for prevention and self-care of urinary tract disorders.
- Evaluate personal responses, revising plan of care as needed to promote, maintain or restore functional health of individuals with urinary tract disorders.

KEY TERMS

The urinary system includes the kidneys, ureters, urinary bladder and urethra. This organ system can be affected by a variety of disorders, including congenital malformations, infections, obstructions, trauma, tumours and neurological conditions. Any portion of the system—from the kidney through to the urethra—can be affected, with serious or even life-threatening consequences unless the problem is appropriately diagnosed and treated. Kidney disorders that can affect urine production and waste elimination directly are discussed in the next chapter. Disorders of the **urinary drainage system** may obstruct urine flow or may affect the kidneys and urine production and elimination. The anatomy, physiology and nursing assessment related to the urinary tract are presented in Chapter 25.

When caring for people with urinary tract disorders, it is important to consider their modesty with respect to voiding, possible difficulty in discussing the genitals, embarrassment over exposure during examination and testing, and fear of changes in body image or function. These psychosocial issues may interfere with willingness to seek help, discuss treatment and learn about preventive measures.

Nursing interventions for people with urinary tract disorders are directed towards primary prevention, early detection and management of disorders through nursing care and health education.

THE PERSON WITH URINARY TRACT INFECTION

There were 2987 deaths in Australia in 2013 from diseases of the kidney and urinary tract with these being the tenth leading cause of death (Australian Bureau of Statistics, 2013). Untreated or poorly managed urinary tract infections (UTIs) may ultimately result in kidney damage, kidney failure and potentially, death (Kidney Health Australia (KHA), 2015). People who are diabetic or have nephritis or hypertensive vascular disease have an increased risk of developing end-stage or chronic kidney disease (CKD). Indigenous Australians have an even greater chance than non-Indigenous Australians with an almost 40% higher death rate from the disease (KHA, 2015).

Bacterial infections of the urinary tract are a frequent reason for seeking health services and are a contributing factor to CKD. In Australia, 1.6 in every 10 doctor consultations (1.1% of the population) was for urinary tract infection (Australian Institute of Health and Welfare (AIHW), 2008). Community-acquired UTIs are more common in women than males, are unusual in men under the age of 50 and may be symptomatic or asymptomatic.

Most community-acquired UTIs (80–90%) are caused by *Escherichia coli* (KHA, 2015), a common Gram-negative enteral bacteria. About 5–10% of symptomatic UTIs are caused by *Staphylococcus saprophyticus*, a Gram-positive organism. Catheter-associated UTIs often involve other Gram-negative bacteria such as *Proteus*, *Klebsiella*, *Serratia* and *Pseudomonas*.

Risk factors

A variety of factors can predispose to UTI (see Box 26.1). Some factors cannot be changed (e.g. ageing and the short urethra of the female); others may be a result of individual behaviour—for example, poor fluid intake or delaying going to the toilet, especially in the elderly.

BOX 26.1 Risk factors for UTI

Female
- Short, straight urethra
- Proximity of urinary meatus to vagina and anus
- Sexual intercourse
- Use of diaphragm and spermicidal compounds for birth control
- Pregnancy

Male
- Uncircumcised
- Prostatic hypertrophy
- Anal intercourse

Both
- Ageing
- Urinary tract obstruction
- Neurogenic bladder dysfunction
- Vesicoureteral reflux
- Genetic factors
- Catheterisation

In women, poor perineal hygiene or cleansing techniques after micturition or defecation may result in bacterial deposition close to the external urethral orifice. Sexual activity also increases the risk of UTI, because bacteria can be introduced into the bladder via the urethra during sexual intercourse. Use of spermicidal compounds with a diaphragm, cervical cap or condom alters the normal bacterial flora of the vagina and perineal tissues and further increases the risk of UTI. Some females lack a normally protective mucosal enzyme and have decreased levels of cervicovaginal antibodies to enterobacteria, further increasing their risk. Prostatic hypertrophy and bacterial prostatitis are risk factors among males, as is anal intercourse, although circumcision appears to have a protective effect. Congenital or acquired factors contributing to the risk of infection include urinary tract obstruction by tumours or calculi, structural abnormalities such as strictures, impaired bladder innervation, bowel incontinence and chronic diseases such as diabetes mellitus.

Instrumentation of the urinary tract (e.g. catheterisation or cystoscopy) is a major risk factor for UTI. The placement of the catheter prevents the flushing action of voiding and bacteria may ascend to the bladder either through the catheter lumen or via exudate between the urethral mucosa and the catheter. Even when performed under strict aseptic conditions, catheterisation can result in bladder infection. Research indicates that the risk of catheter-associated UTI is reduced when anaesthetic lubricating gels are inserted into the urethra prior to catheter insertion (Bardsley, 2005). Prophylactic antibiotics are not recommended at insertion or replacement of an indwelling urinary catheter unless otherwise recommended. Use of a local anaesthetic minimises the discomfort experienced by the patient (Australian & New Zealand Urological Nurses Society (ANZUNS), 2013).

Older individuals have an increased incidence of UTI. The greatest degree of increase is seen in men, as the ratio of female-to-male UTI in older adults changes from 50:1 to less than 5:1. An increased risk of urinary stasis, chronic disease states (such as diabetes mellitus) and an impaired immune response contribute to the higher incidence of UTI in the older adult. In men, the prostate typically hypertrophies with ageing, potentially resulting in urinary retention as the urethra narrows. Prostatic secretions are lessened, diminishing their protective, antibacterial effect. In older women, loss of tissue elasticity and weakening of perineal muscles often contribute to the development of a cystocele or rectocele. Resulting changes in bladder and urethral position increase the risk of incomplete bladder emptying.

FAST FACTS

- For females there is a 1 in 3 lifetime incidence of UTI (Jarvis, Chan & Gottlieb, 2014).
- In healthy adult men, however, UTIs are unusual and may prompt additional diagnostic testing.

Physiology review

The urinary tract is normally sterile above the urethra. Adequate urine volume, a free flow from the kidneys through the urinary meatus and complete bladder emptying are the most important mechanisms in maintaining sterility. Any pathogens that enter and contaminate the distal urethra are then washed out during voiding. Other defences for maintaining sterile urine include its normal acidity and the bacteriostatic properties of the bladder and urethral cells. The peristaltic activity of the ureters and a competent vesicoureteral junction help maintain sterility of the upper urinary tract. As the ureter enters the bladder, its distal portion tunnels between the mucosa and muscle layers of the bladder wall (see Figure 26.1). During voiding, increased *intravesicular* (within the bladder) pressure compresses the ureter, preventing **reflux** or backflow of urine towards the kidneys. In males, a long urethra and the antibacterial effect of zinc in prostatic fluid also help prevent contamination of this normally sterile environment.

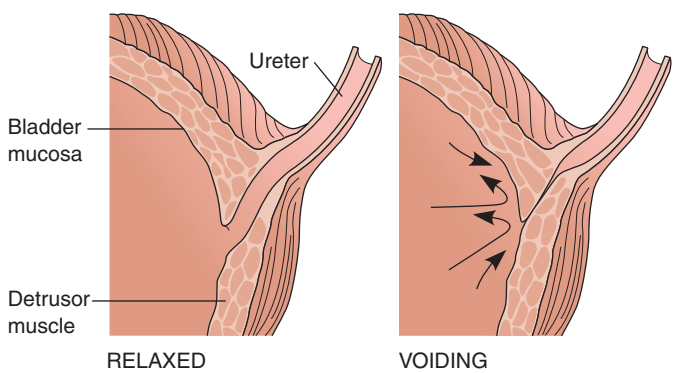


FIGURE 26.1 ■ A competent vesicoureteral junction. Note how increased intravesicular pressure during voiding occludes the distal portion of the ureter, preventing reflux

Pathophysiology and manifestations

Pathogens usually enter the urinary tract by ascending from the mucous membranes of the perineal area into the lower urinary tract. Bacteria that have colonised the urethra, vagina or perineal tissues are the usual source of infection (Porth & Matfin, 2009). From the bladder, bacteria may continue to ascend the urinary tract, eventually infecting the *parenchyma* (functional tissue) of the kidneys (Fauci et al., 2008). Haematogenous spread of infection to the urinary tract is rare. Infections introduced in this manner are usually associated with previous damage or scarring of the urinary tract. Bacteria introduced into the urinary tract may cause asymptomatic bacteriuria or an inflammatory response with manifestations of UTI.

Urinary tract infections can be categorised in several ways. Anatomically, UTIs may affect the lower or the upper urinary tract. Lower urinary tract infections include *urethritis*, inflammation of the urethra; *prostatitis*, inflammation of the prostate gland (discussed in Chapter 47); and **cystitis**, inflammation of the urinary bladder. The most common upper urinary tract infection is **pyelonephritis**, inflammation of the kidney and renal pelvis. The infection may involve superficial tissues such as the bladder mucosa or may invade other tissues such as prostate or renal tissues. Epidemiologically, UTIs are identified as community-acquired infections or **healthcare-associated infections** associated with catheterisation.

Cystitis

Cystitis, inflammation of the urinary bladder, is the most common UTI. The infection tends to remain superficial, involving the bladder mucosa. The mucosa becomes hyperaemic (red) and may haemorrhage (see Figure 26.2). The inflammatory response results in pus formation, causing the classic manifestations associated with cystitis. Typical presenting symptoms of cystitis include **dysuria** (painful or difficult urination), urinary frequency and **urgency** (a sudden, compelling need to

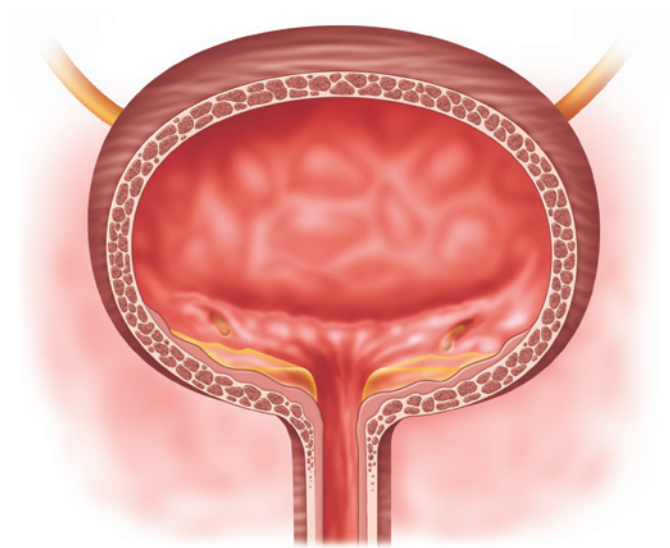


FIGURE 26.2 ■ Appearance of the bladder wall affected by cystitis

urinate), and **nocturia** (voiding two or more times at night). In addition, the urine may be foul smelling and cloudy (*pyuria*) or bloody (**haematuria**) because of mucus, excess white cells in the urine and bleeding of the inflamed bladder wall. Suprapubic pain and tenderness also may be present. See the box below for manifestations of cystitis.

Cystitis occurs most frequently in adult females, usually because of colonisation of the bladder by bacteria normally found in the lower gastrointestinal tract. These bacteria gain entry by ascending the short, straight female urethra. In addition to the risk factors listed above, personal hygiene practices and voluntary urinary retention can contribute to the risk of UTI in women.

Older people may not experience the classic symptoms of cystitis. Instead, they often present with non-specific manifestations such as nocturia, incontinence, confusion, behaviour change, lethargy, anorexia or 'just not feeling right'. Fever and hypothermia may also be present.

Although the bacteriostatic effect of prostatic fluid and a longer urethra provide an effective barrier to bladder infection for adult males, the prostatic hypertrophy commonly associated with ageing increases the risk of cystitis in elderly males. An enlarged prostate can impede urine flow, leading to incomplete bladder emptying and urinary stasis. Bacteria are not completely flushed with voiding, allowing colonisation of the bladder.

Cystitis is usually uncomplicated and readily responds to treatment. Severe or prolonged infection may lead to sloughing of bladder mucosa and ulcer formation; when left untreated, the infection can ascend to involve the kidneys. Chronic cystitis can also lead to bladder stones (discussed later in this chapter).

Catheter-associated UTI

At least 20% (Wynne et al., 2014) of hospitalised people with indwelling urinary catheters develop bacteriuria. The longer the catheter remains in place, the greater the risk of infection. Bacteria, including *E. coli*, *Proteus*, *Pseudomonas* and *Klebsiella*, reach the bladder either by migrating through the column of urine within the catheter or by moving up the mucous sheath of the urethra outside the catheter (Fauci et al., 2008). Bacteria enter the catheter system at the connection between the catheter and drainage system or through the emptying tube of the drainage bag. Colonisation of perineal skin by bowel flora is a common source of infection in catheterised women.

Catheter-associated UTIs are often asymptomatic. Gram-negative bacteraemia is the most significant complication associated with these. Most catheter-associated UTIs resolve quickly when the catheter is removed and a short course of antibiotics is given. Intermittent catheterisation carries a lower risk of infection than does an indwelling catheter and is preferred for those who are unable to empty their bladder by normal voiding.

Pyelonephritis

Pyelonephritis is inflammation of the renal pelvis and parenchyma, the functional kidney tissue. *Acute pyelonephritis* is a bacterial infection of the kidney; *chronic pyelonephritis* is associated with repeated non-bacterial infections and inflammatory processes that may be metabolic, chemical or immunological in origin.

ACUTE PYELONEPHRITIS Acute pyelonephritis usually results from an infection that ascends to the kidney from the lower urinary tract. Asymptomatic bacteriuria or cystitis can lead to acute pyelonephritis. Risk factors include pregnancy (because of slowed ureteral peristalsis), urinary tract obstruction and congenital malformation. Urinary tract trauma, scarring, calculi (stones), kidney disorders such as polycystic or hypertensive kidney disease, and chronic diseases such as diabetes may also contribute to pyelonephritis. *Vesicoureteral reflux*, a condition in which urine moves from the bladder back towards the kidney, is a common risk factor in children who develop pyelonephritis and is also seen in adults when bladder outflow is obstructed.

The infection spreads from the renal pelvis to the renal cortex. The pelvis, calyces and medulla of the kidney are primarily affected, with white blood cell (WBC) infiltration and inflammation. The kidney becomes grossly oedematous. Localised abscesses may develop on the cortical surface of the kidney. As with cystitis, *E. coli* is the organism responsible for 80–90% (KHA, 2015) of the cases of acute pyelonephritis. Other organisms commonly found include *Proteus* and *Klebsiella*, bacteria that normally inhabit the intestinal tract.

The onset of acute pyelonephritis is typically rapid, with chills and fever, malaise, vomiting, flank pain, costovertebral tenderness, urinary frequency and dysuria (see the 'Manifestations' box below). Symptoms of cystitis also may be present. The older adult may present with a change in behaviour, acute confusion, incontinence or a general deterioration in condition.

CHRONIC PYELONEPHRITIS Chronic pyelonephritis involves chronic inflammation and scarring of the tubules and interstitial tissues of the kidney and is a common cause of chronic renal failure. It may develop as a result of ascending

MANIFESTATIONS Cystitis

- Dysuria
- Pyuria
- Frequency
- Haematuria
- Urgency
- Suprapubic discomfort
- Nocturia

MANIFESTATIONS Acute pyelonephritis

URINARY
- Urinary frequency
- Dysuria
- Pyuria
- Haematuria
- Flank pain
- Costovertebral tenderness

SYSTEMIC
- Vomiting
- Diarrhoea
- Acute fever
- Shaking chills
- Malaise

UTIs or other conditions that damage the kidneys, such as hypertension, vascular conditions, severe vesicoureteral reflux or obstruction of the urinary tract.

The person with chronic pyelonephritis may be asymptomatic or have mild manifestations such as urinary frequency, dysuria and flank pain. Hypertension can develop as kidney tissue is destroyed.

FAST FACTS

- The most common route of entry for a urinary tract infection is ascending, from colonisation of the perineal tissues by faecal bacteria (usually *E. coli*), through the urethra, into the bladder (cystitis) and possibly kidney tissue (pyelonephritis).

INTERPROFESSIONAL CARE

Treatment of UTI focuses on eliminating the causative organism, preventing relapse or reinfection, and identifying and correcting any contributing factors. Drug treatment with antibiotics and urinary anti-infectives is commonly used. In some cases, surgery may be indicated to correct contributing factors.

Diagnosis

Laboratory testing for UTI includes:

- *Urinalysis*, to assess for pyuria, bacteria and blood cells in the urine. A bacteria count greater than 10×10^6/L suggests infection. Rapid tests for bacteria in the urine include using a *nitrite dipstick* (which turns pink in the presence of bacteria) and the *leucocyte esterase test*, which is positive in the presence of WBCs (neutrophils), indicating infection or inflammation.

  Urine should be a midstream clean-catch specimen in a sterile container. If necessary, catheterisation or 'mini-cath', with aseptic technique, may be used. However, catheterisation should be avoided if possible to reduce the risk of further infection. (A 'Diagnostic tests' box for nursing care related to collecting a urinalysis specimen is included in Chapter 25.)
- *Gram stain of the urine* may be done to identify the infecting organism by shape and characteristic (Gram-positive or Gram-negative).
- *Urine culture and sensitivity* tests may be ordered to identify the infecting organism and the most effective antibiotic. Culture requires 24 to 72 hours, so treatment to eliminate the most common organisms may be initiated without culture followed by antibiotic therapy according to sensitivity results.
- *WBC with differential* may be done to detect typical changes associated with infection, such as *leucocytosis* (elevated WBC) and increased numbers of neutrophils.

In men and in adult women with recurrent infections or persistent bacteriuria, additional diagnostic testing may be ordered to evaluate for structural abnormalities and other contributing factors:

- *Intravenous pyelography (IVP)* is used to evaluate the structure and excretory function of the kidneys, ureters and bladder. As the kidneys clear an intravenously injected contrast medium from the blood, the size and shape of the kidneys, their calyces and pelvises, the ureters and the bladder can be identified and structural or functional abnormalities, such as vesicoureteral reflux, may be detected.
- *Antegrade pyelogram* is used when an IVP fails to identify an obstruction in the ureters or bladder. Contrast medium is injected into the collecting system of the kidney and renal flow is monitored by radiology.
- *Computed tomography (CT scan)* may be used in preference to IVP for identifying renal calculi. It uses a radiation beam to build a three-dimensional picture of the renal system.
- *Ultrasound* is a non-invasive technique that utilises sound waves to visualise soft tissue masses, obstructions, some renal calculi and renal blood flow.
- *Micturating cystogram (MCU)* is a procedure conducted on infants or children, and involves instilling contrast medium into the bladder, then using x-rays to assess the bladder and urethra when filled and during voiding. This study can detect structural or functional abnormalities of the bladder and urethral strictures. This test has a lower risk of allergic response to the contrast dye than IVP.
- *Cystoscopy*, direct visualisation of the urethra and bladder through a cystoscope, may be used to diagnose conditions such as prostatic hypertrophy, urethral strictures, bladder calculi, tumours, polyps or diverticula, and congenital abnormalities. A tissue biopsy may be obtained during the procedure and other interventions performed (e.g. stone removal or stricture dilation).
- *Manual pelvic* or prostate examinations are performed to assess for structural changes of the genitourinary tract, such as prostatic enlargement, cystocele or rectocele.

Nursing implications for these diagnostic procedures are presented in Chapter 25.

Medications

Most uncomplicated infections of the lower urinary tract can be treated with a short course of antibiotic therapy. Upper urinary tract infections, in contrast, usually require longer treatment (2 or more weeks) to eradicate the infecting organism.

Short-course therapy (either a single antibiotic dose or a 3-day course of treatment) reduces treatment cost, increases compliance and has a lower rate of side effects. Single-dose therapy is associated with a higher rate of recurrent infection and continued vaginal colonisation with *E. coli*, making a 3-day course of treatment the preferred option for uncomplicated cystitis. Oral trimethoprim (TMP) or cephalexin as a first choice may be ordered (Jarvis et al., 2014). Treatment for acute uncomplicated cystitis should be individualised when choosing an antibiotic, as no single agent is considered best (Gilbert & Weiner, 2013).

Men and women with pyelonephritis, urinary tract abnormalities, stones or a history of previous infections with antibiotic-resistant infections may be prescribed a variety of drugs including cephalexin, amoxicillin/clavulanic acid, trimethoprim or ciprofloxacin as a 7- to 10-day course. Those with severe illness may need hospitalisation for initial parenteral treatment, changing to oral therapy when there is clinical improvement. Drugs used for severe illness or sepsis associated with UTI

include ciprofloxacin, gentamicin or ceftriaxone (Rocephin), or amoxicillin/ampicillin. (See Chapter 11 for the nursing implications for antibiotic therapy.)

The outcome of treatment for UTI is determined by follow-up urinalysis and culture. *Cure*, as evidenced by no pathogens present in the urine, is the desired outcome. When therapy fails to eradicate bacteria in the urine, it is known as unresolved bacteriuria. *Persistent bacteriuria* or *relapse* occurs when a persistent source of infection causes repeated infection after initial cure. *Reinfection* is the development of a new infection with a different pathogen following previous successful UTI treatment (McPhee, Papadakis & Rabow, 2012).

CONSIDERATION FOR PRACTICE

Follow-up urine culture should be performed 10 days to 2 weeks following completion of antibiotic therapy for UTI to ensure bacterial eradication from the urinary tract.

Individuals experiencing frequent symptomatic UTIs may be treated with prophylactic antibiotic therapy with a drug such as TMP or nitrofurantoin (Furadantin, Macrodantin). TMP and nitrofurantoin do not achieve effective plasma concentrations at recommended doses, but do reach effective concentrations in the urine. Nitrofurantoin also may be used to treat UTI in pregnant women. Nursing implications for these urinary anti-infectives are outlined in the 'Medication administration' box below.

Antibiotics and urinary anti-infectives are not generally recommended to treat asymptomatic bacteriuria in catheterised people. The recommended treatment for catheter-associated UTI is removal of the indwelling catheter followed by a 10- to 14-day course of antibiotic therapy to eliminate the infection.

Surgery

Surgery may be indicated for recurrent UTI if diagnostic testing indicates calculi, structural anomalies or strictures that contribute to the risk of infection. Table 26.1 lists major causes of urinary tract obstruction that may contribute to UTI.

Stones or *calculi* in the renal pelvis or in the bladder are an irritant and provide a matrix for bacterial colonisation. Treatment may include surgical removal of a large calculus from the renal pelvis or cystoscopic removal of bladder calculi. *Percutaneous*

MEDICATION ADMINISTRATION Urinary anti-infectives

URINARY ANTI-INFECTIVES

Nitrofurantoin (Furadantin, Macrodantin)
Trimethoprim (Bactrim, Triprim)

Urinary anti-infectives are usually used prophylactically to prevent recurrence of UTI in those with frequent symptomatic infections. Nitrofurantoin may be used to treat UTI in pregnant women but only if medically essential.

Nursing responsibilities

- Ensure adequate fluid intake (1500 to 2000 mL/day) to maintain a urine output of at least 1500 mL of urine per 24 hours. Do not overhydrate.
- Administer with meals to minimise GI side effects, such as nausea, gastric upset and abdominal cramping.
- Trimethoprim is contraindicated for use in people with renal or hepatic impairment; nitrofurantoin is contraindicated for those with impaired renal function. Report abnormal laboratory values such as elevated creatinine or BUN, decreased eGFR, bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and lactic dehydrogenase (LDH).
- Monitor for skin irritations, rash, nausea, vomiting and fever. If bruising or unusual bleeding occurs, discontinue the drug and notify the doctor.
- Use with caution in older or chronically ill people. Monitor closely for adverse effects.
- Do not administer trimethoprim to pregnant women unless essential because of possible adverse effects on the foetus.
- Monitor the person taking nitrofurantoin for an acute or chronic pulmonary reaction with manifestations of dyspnoea, cough, chills, fever and chest pain. Discontinue the drug and notify the doctor.
- Nitrofurantoin may cause peripheral neuropathy, especially in older individuals and adult diabetics. Notify the doctor if symptoms develop.
- Nitrofurantoin oral suspension may stain the teeth; therefore, instruct the person to rinse the mouth thoroughly after administering.
- Monitor for signs of phenytoin toxicity (sedation, ataxia and increased blood levels) if trimethoprim is given concurrently. Phenytoin doses may need to be reduced.

Health education for the person and family

- These drugs are used with good hygiene practices to prevent UTI recurrence. Take as directed even when no symptoms are present, until the course is completed.
- Drink six to eight glasses of water a day (2500–3500 mL) while taking these drugs.
- Take the drug with meals or food to reduce gastric effects; however, avoid milk products because they may interfere with absorption.
- Trimethoprim should not be taken during pregnancy. Contact your doctor before attempting to become pregnant.
- Contact your doctor if you develop any of the following: chest pain, difficulty breathing, cough, chills and fever; numbness and tingling or weakness of the extremities; rash or pruritus (itching), bruising or bleeding.
- If you are taking an oral suspension of nitrofurantoin, rinse your mouth thoroughly after each dose to avoid staining the teeth.
- Nitrofurantoin turns the urine brown. This is not harmful and disappears when the drug is discontinued.
- If you are taking trimethoprim along with phenytoin (Dilantin) or a related anticonvulsant, contact your doctor if you become sedated or begin to stagger.

TABLE 26.1 Major causes of urinary tract obstruction by location

LOCATION	OBSTRUCTIVE PROCESS
Kidney pelvis	Calculi (stones) Polycystic kidney disease Infection and scarring
Ureters	Calculi Scarring and stricture Congenital defects or strictures External processes such as pregnancy, tumours, lymph node enlargement
Bladder	Neurogenic bladder Tumours Calculi and other foreign bodies
Urethra	Benign prostatic hypertrophy Tumours Scarring and stricture Trauma

ultrasonic pyelolithotomy or *extracorporeal shock wave lithotripsy* (described in the next section of this chapter) may be used instead of surgery to crush and remove stones. (See the 'Diagnostic tests' box in Chapter 25 for nursing care related to cystoscopy.)

Ureteroplasty, surgical repair of a ureter, may be indicated for structural abnormality or stricture of a ureter. This may be combined with a ureteral reimplantation if vesicoureteral reflux is present. The person returns from these surgeries with an indwelling urinary catheter (Foley or suprapubic) and a **ureteral stent** (a thin catheter inserted into the ureter to provide for urine flow and ureteral support) which remains in place for 3 to 5 days. Care of those with a ureteral stent is outlined in the box below.

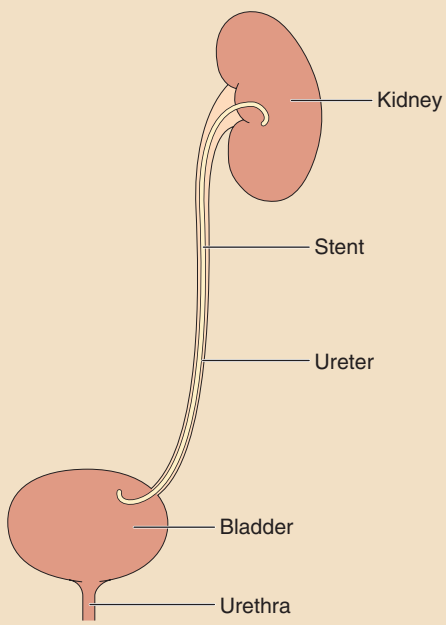
Complementary therapies

Complementary therapies such as aromatherapy or herbal preparations, particularly cranberry juice, have been advocated for use in conjunction with antibiotics to treat UTI. However, despite being recommended for prevention and treatment of UTI, cranberry juice and capsule studies have been inconclusive as to their effectiveness. While some studies have shown there may be benefits from cranberry juice, in controlled studies cranberry juice does not prevent UTIs (KHA, 2015). There are also risks of interaction between cranberry juice and other drugs—for example, warfarin. People who wish to take herbal remedies should consult their doctor because of drug interaction risks.

NURSING CARE OF THE PERSON with a ureteral stent

Ureteral stents are used to maintain patency and promote healing of the ureters (see figure below). A stent may be temporary, used during and after a surgical procedure, or it may be used for longer periods in individuals with ureteral obstruction due to tumours, strictures or other causes.

Stents may be positioned during surgery or cystoscopy. They are made of a non-toxic material such as silicone or polyurethane, with side drainage holes placed along the length of the stent. Stents are radiopaque for easy radiographic identification and one or both ends of the stent may be pigtail or J shaped to prevent migration or dislodgement.



- Label all drainage tubes, including stents, for easy identification. Attach each catheter and stent to a separate closed drainage system. *Careful labelling allows close monitoring of output from all sources and reservoirs. Separate drainage systems minimise the risk of infection.*
- If the stent has been brought to the surface, secure it and maintain its position. The stent is usually placed in the renal pelvis. *It is important to secure it well to prevent trauma to the kidney, inadvertent removal of the stent and ureter obstruction.*
- Monitor urine output, including colour, consistency and odour. Monitor for signs of infection, bleeding, fever, tachycardia, pain, haematuria and cloudy or foul-smelling urine. *The stent facilitates urine flow but may become obstructed because of bleeding, calculi or sediment. Obstruction may result in hydronephrosis and kidney damage. The stent itself is a foreign body in the urinary tract and can increase the risk of UTI.*
- Maintain fluid intake, encouraging the use of fluids that acidify urine, such as low-sugar apple, cranberry and blueberry juice. *The stent can precipitate calculus formation as well as UTI. Increasing fluid intake and acidifying the urine help to prevent these complications.*
- For an indwelling stent, stress the need for regular follow-up to monitor for and prevent complications such as UTI and calculi. *The person with an indwelling stent may tend to forget that the stent is in place and become lax in compliance with follow-up and preventive measures.*

Nursing care

Health promotion

Teach measures to prevent UTI to all sexually active women, particularly young ones. Encourage the maintenance of a generous fluid intake of 2000–2500 mL per day, increasing intake during hot weather or strenuous activity. Discuss the need to avoid voluntary urinary retention, emptying the bladder every 3 to 4 hours. Instruct women to cleanse the perineal area from front to back after voiding and defecating. Teach to void before and after sexual intercourse to flush out bacteria introduced into the urethra and bladder. Teach measures to maintain the integrity of perineal tissues: avoid bubble baths, feminine hygiene sprays and vaginal douches; wear cotton briefs, avoid synthetic materials as they may irritate the perineal and vaginal mucosa. Postmenopausal women may be advised to use hormone replacement therapy or oestrogen cream. Avoid excess intake of milk and milk products, non-acidic fruit juices and sodium bicarbonate (baking soda), which is an alkaline substance.

Assessment

Focused assessment data for the person with a UTI include the following:

- *Health history:* current symptoms, including frequency, urgency, burning on urination, voidings per night; colour, clarity and odour of urine; other manifestations such as lower abdominal, back or flank pain, nausea or vomiting, fever; duration of symptoms and any treatment attempted; history of previous UTIs and their frequency; possibility of pregnancy and type of birth control used; chronic diseases such as diabetes; current medications and any known allergies.
- *Physical examination:* general health; vital signs including temperature; abdominal shape, contour, tenderness to palpation (especially suprapubic); percuss for costovertebral tenderness (see Box 25.1).

See Chapter 25 for complete nursing assessment of the urinary system.

Nursing diagnoses and interventions

General health, abilities for self-care and risk factors that may contribute to UTI are considered when planning and implementing nursing care for the person with a UTI. Priority nursing diagnoses focus on comfort, urinary elimination and teaching/learning needs. A nursing care plan for the person with cystitis can be found below.

Pain

Pain is a common manifestation of both lower and upper UTI. Urinary tract pain is caused primarily by distension and increased pressure within the tract. The severity of the pain is related to the rate at which inflammation and distension develop.

In cystitis, inflammation causes a sensation of fullness; dull, constant suprapubic pain; and possibly lower back pain. The inflamed bladder wall and urethra cause dysuria, pain and burning on urination. Bladder spasms may develop, causing periodic severe, stabbing discomfort. Pain associated with pyelonephritis is often steady and dull, localised to the outer abdomen or flank region. Urological disorders rarely cause central abdominal pain.

- Assess pain: timing, quality, intensity, location, duration and aggravating and alleviating factors. *A change in the nature, location or intensity of the pain could indicate an extension of the infection or a related but separate problem.*
- Teach or provide comfort measures such as warm baths, warm packs or heating pads, and balanced rest and activity. Systemic analgesics, urinary analgesics or antispasmodic medication may be used as ordered. *Warmth relaxes muscles, relieves spasms and increases local blood supply. Because pain can stimulate a stress response and delay healing, it should be relieved when possible.*
- Increase fluid intake unless contraindicated. *Increased fluid dilutes urine, reducing irritation of the inflamed bladder and urethral mucosa.*
- Instruct to notify primary care provider if pain and discomfort continue or intensify after therapy is initiated. *Pain and discomfort in voiding typically are relieved within 24 hours of the initiation of antibiotic therapy. Continued discomfort may indicate a complicated UTI or other urinary tract disorder.*

CONSIDERATION FOR PRACTICE

The older adult with a UTI may not complain of dysuria with a UTI. Be alert for other manifestations of UTI such as incontinence or cloudy or foul-smelling urine. Inflammatory and immune responses tend to diminish with ageing, reducing the irritative effects of UTI symptoms.

Impaired urinary elimination

Inflammation of the bladder and urethral mucosa affects the normal process and patterns of voiding, causing frequency, urgency and burning on urination, as well as nocturia. Urine may be tinged with blood, cloudy and foul smelling. The person with short- or long-term urinary retention requires additional measures in assessing for and preventing UTI. (See the section on urinary retention later in this chapter.)

- Monitor (or give instructions to monitor) colour, clarity and odour of urine. *Urine should return to clear yellow within 48 hours, unless drug therapy causes a change in the colour of urine. If clarity does not return, further investigation may be necessary.*

CONSIDERATION FOR PRACTICE

Provide for close, easy access to a bedpan, urinal, commode or bathroom. Make sure that lighting is adequate and that pathways are free of obstacles. Frequency, urgency and nocturia increase the risk of urinary incontinence and, importantly, of injury due to falls, particularly in the older or debilitated individual.

NURSING CARE PLAN A person with cystitis

Marja Waisanen is a 25-year-old second-year university student. She was recently married and lives in an apartment with her husband near the university she attends. Mrs Waisanen has never been pregnant and uses a diaphragm for birth control. She presents at the local family planning clinic complaining of lower back pain, frequency, urgency and burning on urination that began the day before.

ASSESSMENT

Patricia Purcel, RN, admits Mrs Waisanen to the clinic and completes an assessment. Mrs Waisanen denies having had similar symptoms in the past or ever having been diagnosed with a urinary tract infection. The data collected show a constant dull ache that does not change with movement. Mrs Waisanen feels the need to urinate almost constantly, but experiences difficulty in starting her stream, with a burning pain and cramping when voiding. She reports getting up four times the night before to urinate. She denies painful intercourse and states that her last menstrual period began 2 weeks previously. A physical examination reveals: BP 112/68; P 90 and regular, T afebrile. Suprapubic tenderness was noted but no flank or costovertebral angle tenderness. A clean-catch urine specimen shows haematuria, multiple WBCs and a bacteria count greater than 10^6/L.

After assessing for allergies and diabetes, Mrs Waisanen was prescribed trimethoprim (TMP) 160/800 mg PO bd for 3 days and paracetamol 1 g PO every 4 hours as needed for pain; with a maximum of 4 g per day. She was also advised to increase her fluid intake to at least 3000 mL every day to flush out the urinary system.

Mrs Waisanen is instructed to return to the clinic in 7 days for a follow-up urine culture or sooner if her symptoms do not improve.

DIAGNOSES

- *Pain* related to infection and inflammatory process in the urinary tract.
- *Impaired urinary elimination* related to inflammation as evidenced by frequency, urgency, nocturia and dysuria.
- *Deficient knowledge* related to lack of information about risk factors for UTI.

PLANNING

- Plan for effective pain management.
- Plan for elimination of burning on urination.
- Plan for effective psychological care.
- Plan to monitor for fluid balance.
- Plan for incorporating health prevention activities into daily routine.

Expected outcomes

- Relief of lower back pain and burning on urination.
- Regains a normal voiding pattern without frequency, urgency, nocturia and abnormal urine characteristics.
- Verbalises understanding of the disease process, related risk factors, follow-up instructions and symptoms of any recurrence indicating the need for medical attention.

IMPLEMENTATION

- Teach comfort measures: warm baths, a heating pad on low heat applied to her lower back or abdomen, rest, increased fluid intake, avoiding caffeinated beverages and taking paracetamol as ordered.
- Advise to avoid the use of antacids when taking trimethoprim-sulfamethoxazole and to take the medication on an empty stomach.
- Advise to stop taking the medication and return to doctor if rash and/or skin irritation occurs which may signify allergic response to medication.
- Advise to refrain from sexual intercourse until infection and inflammation have cleared to avoid further irritation of inflamed tissues.
- Discuss the possible relationship between using a diaphragm for birth control and UTI in women.
- Discuss dietary and hygiene practices to prevent UTI, symptoms indicating the need for further intervention and the risks of under treatment.
- Suggest how strategies designed to prevent further attacks of UTI can be incorporated into daily activities.
- Ensure details of Mrs Waisanen's history, diagnosis, treatment and response are recorded on the appropriate documents.

EVALUATION

On her follow-up visit, Mrs Waisanen reports that her symptoms and urine cleared within about a day after starting the antibiotic and she has had no further problems. She has seen her healthcare practitioner to change her birth control to oral contraceptives, increased her intake of fluid and vitamin C, and no longer puts off urinating until she 'has time to go'.

CRITICAL THINKING IN THE NURSING PROCESS

1. Which physiological and psychosocial factors put Mrs Waisanen at risk of developing a UTI?
2. Why is it necessary to ascertain whether or not Mrs Waisanen had allergies or diabetes? What would be the implications of these for the prescribed treatment?
3. Why was it appropriate to use short-course therapy for Mrs Waisanen? What was the importance of advising her to return if the symptoms did not clear?

REFLECTION ON THE NURSING PROCESS

1. Compare and contrast the benefits and disadvantages to short-course therapy versus conventional therapy for UTI in this case.
2. What are the potential dangers of repeated attacks of UTIs and why is it important to prevent these?
3. What lessons from this scenario could be used in health education programs to promote awareness of UTI and its treatment?

- Instruct to avoid caffeinated drinks, including coffee, tea and cola; citrus juices; drinks containing artificial sweeteners; and alcoholic beverages. *Caffeine, citrus juices and artificial sweeteners irritate bladder mucosa and the detrusor muscle, and can increase urgency and bladder spasms.*

- Use strict aseptic technique and a closed urinary drainage system when inserting a straight or indwelling urinary catheter. Insert indwelling catheters to the full recommended length (5–6 cm in women and 15–25 cm in men) until urine flows before inflating the balloon. Never inflate the balloon until urine flows freely and stop if pain is felt. *Bacteria colonising the perineal tissues or on the nurse's hands can be introduced into the bladder during catheterisation. Aseptic technique reduces this risk. Inflation of the balloon while in the urethra damages urethral tissues and can cause significant discomfort. See the 'Translation to practice' box below for evidence-based practice for catheterisation.*

CONSIDERATION FOR PRACTICE

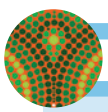
In males, fully insert the catheter to the 'Y' connection; in females, 2.5 cm beyond the point of urine flow (SUNA, 2014).

- When possible, use intermittent straight catheterisation to relieve urinary retention. Remove indwelling urinary catheters as soon as possible. *Using intermittent straight catheterisation allows the bladder to fill and completely empty in a more normal manner, maintaining physiological function. The risk of infection associated with an indwelling catheter is about 3–5% per day of catheterisation* (Fauci et al., 2008; Gilbert & Weiner, 2013).

Promoting health maintenance

The person with a urinary tract infection is at an increased risk of future UTI and needs to understand the disease process, risk factors, measures needed to prevent recurrent infection, diagnostic procedures required and appropriate home care. In addition, once the symptoms of UTI are relieved, individual motivation to continue the treatment plan declines. Failure to complete the full course of therapy and recommended follow-up can lead to continued bacteriuria and recurrent infections.

- Teach how to obtain a midstream clean-catch urine specimen. *Ninety per cent of urethral bacteria are cleared in the first 10 mL of voided urine; a midstream specimen is representative of urine in the bladder. Cleansing of the urinary meatus and perineal area reduces contamination of the specimen by external cells and bacteria.*
- Assess knowledge about the disease process, risk factors and preventive measures. *The individual may have little understanding of UTI, its causes and contributing factors.*
- Discuss the prescribed treatment plan and the importance of taking all prescribed antibiotics.
- Help the person develop a plan for taking medications, such as taking them with meals (unless contraindicated) or setting out all doses for the day in the morning. *Missed doses of antibiotic can result in lowered therapeutic blood levels and reduced effectiveness. Taking medication in association with meals helps with remembering doses.*



TRANSLATION TO PRACTICE Evidence-based practice: catheterisation

Insertion of an indwelling (retention) catheter is performed in hospitals and long-term care facilities. The location of the female urethral meatus presents a challenge to maintaining catheter sterility during insertion, whereas the anatomy of the male urethra presents a different set of challenges. Best practice guidelines for inserting the catheter in females is to insert the catheter until urine flows, then advance the catheter a further 2–4 cm to ensure the balloon is clear of the urethra; for males insert the catheter until resistance is felt at the first sphincter, then continue to the Y bifurcation. Ensure the retention balloon is well within the urinary bladder prior to its inflation (ANZUNS, 2013). Insertion to any lesser distance is inadequate to ensure safe balloon inflation without potential damage to the urethra.

IMPLICATIONS FOR NURSING

Nursing fundamentals and skills texts recommend inserting the catheter from 15.25 cm into the male urethra until urine flows. To ensure safe practice and reduce the risk of injury and discomfort, insert a retention catheter to the bifurcation before inflating the balloon.

SAFETY ALERT

Maintain the closed urinary drainage system and use aseptic technique when emptying the catheter drainage bag. Maintain gravity flow, preventing reflux of urine into the bladder from the drainage system. Bacteria can enter the drainage system when its integrity is interrupted as, for example, when disconnecting the catheter from the drainage system or during emptying of the drainage bag. Bacteria can ascend the column of urine to the bladder, causing UTI.

- Provide perineal care on a regular basis and following defecation. Use antiseptic preparations only as ordered. Regular cleansing of perineal tissues reduces the risk of colonisation by bacteria from the bowel or other sources. *While antiseptic solutions may be ordered for catheter care, they can dry perineal tissues and reduce normal flora, increasing the risk of colonisation by pathogens and therefore should not be routinely used.*

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Why is insertion of a urinary catheter frequently a more uncomfortable and difficult procedure for a male than a female? What nursing measures or techniques can be used to reduce this discomfort?
2. Sterile technique is generally used when catheterising people in acute care settings. However, those who require intermittent catheterisation to empty their bladder typically use clean technique. Would clean technique be appropriate in an acute or long-term care setting? Why or why not?

Source: ANZUNS (2013).

CONSIDERATION FOR PRACTICE

Symptoms are largely relieved within 24 to 48 hours of starting antibiotic therapy; however, bacteria may remain in the urinary tract. Completing the prescribed regimen is important to prevent recurrent infections and resistant bacteria.

- Instruct to keep appointments for follow-up and urine culture. *Follow-up urine culture, often scheduled 7 to 14 days after completion of antibiotic therapy, is vital to ensure complete eradication of bacteria and prevention of relapse or recurrence.*
- Teach measures to prevent future UTI (see the preceding 'Health promotion' section). *Keep urine dilute and acidic and void regularly to flush bacteria out of the bladder and urethra. The proximity of the female urethral meatus to the vagina and anus increases the risk of bacterial contamination, especially during intercourse. Bubble baths, feminine hygiene sprays, synthetic fibres and douches may dry and irritate perineal tissues, promoting bacterial growth. Maintain adequate fluid intake to ensure renal function and production of urine.*

Community-based care

Because both upper and lower urinary tract infections are usually managed in the community, health education is the most important nursing intervention. Provide instruction on the following topics:

- Risk factors for UTI and how to minimise or eliminate these factors through increased fluid intake, regular elimination and personal hygiene measures.
- Early manifestations of UTI and the importance of seeking medical intervention promptly.
- Maintaining optimal immune system function by attending to contributory physical and psychosocial stressors, such as lack of adequate rest, poor nutrition and high levels of emotional stress.
- The importance of completing the prescribed treatment and keeping follow-up appointments.
- Minimising the risk of UTI when an indwelling urinary catheter is necessary:
  a. Use alternatives to an indwelling catheter when possible. For urinary incontinence, try scheduled toileting, incontinence pads or external catheters if possible. For urinary retention, teach the person or a family member to perform straight catheterisation every 3 to 4 hours using clean technique.
  b. Teach care measures such as perineal care, managing and emptying the collection chamber, maintaining a closed system and bladder irrigation or flushing if ordered when an indwelling catheter is necessary.

THE PERSON WITH URINARY CALCULI

Urinary calculi, stones in the urinary tract, are the most common cause of upper urinary tract obstruction (Porth & Matfin, 2009). The term **lithiasis** means 'stone formation'; when the stones form in the kidney, it is known as *nephrolithiasis*; when they form elsewhere in the urinary tract (e.g. the bladder), it is called *urolithiasis*. Stones may form and obstruct the urinary tract at any point (see Figure 26.3). In the Australia, the United States and other industrialised countries, renal or kidney stones are the most common.

Incidence and risk factors

Urolithiasis is the third most common urological condition. It affects more males (1:10) than females (1:35) and in Australia the Indigenous population is at greater risk than non-Indigenous Australians. The incidence may vary according to geographical areas. Most people affected are in young or middle adulthood.

Although the majority of stones are idiopathic (having no demonstrable cause), a number of risk factors have been identified. The greatest risk factor for stone formation is a prior personal or family history of urinary calculi. A genetic predisposition towards the accumulation of certain mineral substances in the urine or a congenital lack of protective factors may explain the familial link. Other identified risk factors include dehydration with resultant increased urine concentration, immobility and excess dietary intake of calcium, oxalate or proteins. Gout, hyperparathyroidism and urinary stasis or repeated infections also contribute to calculus formation.

Physiology review

Normally, a balance exists in the kidneys between the need to conserve water and to eliminate poorly soluble materials such as calcium salts. This balance is affected by factors such as diet, environmental temperature and activity. Protective inorganic and organic substances in the urine, such as pyrophosphate, citrate and glycoproteins, normally inhibit stone formation.

Pathophysiology

Three factors contribute to urolithiasis: supersaturation, nucleation and lack of inhibitory substances in the urine.

When the concentration of an insoluble salt in the urine is very high (supersaturated), crystals may form. Usually, these crystals disperse and are eliminated because the chemical bonds holding them together are weak. However, a nucleus of crystals may develop stable bonds to form a stone with crystals forming around an organic matrix or mucoprotein nucleus. The stimulus required to initiate crystallisation in supersaturated urine may be minimal. Ingesting a meal high in insoluble salt, or decreased fluid intake as occurs during sleep, allows the concentration to increase to the point where salt precipitation occurs, and stones are formed and grow. When fluid intake is adequate, no stone growth occurs. The acidity or alkalinity of the urine and the presence or absence of calculus-inhibiting compounds also affect lithiasis.

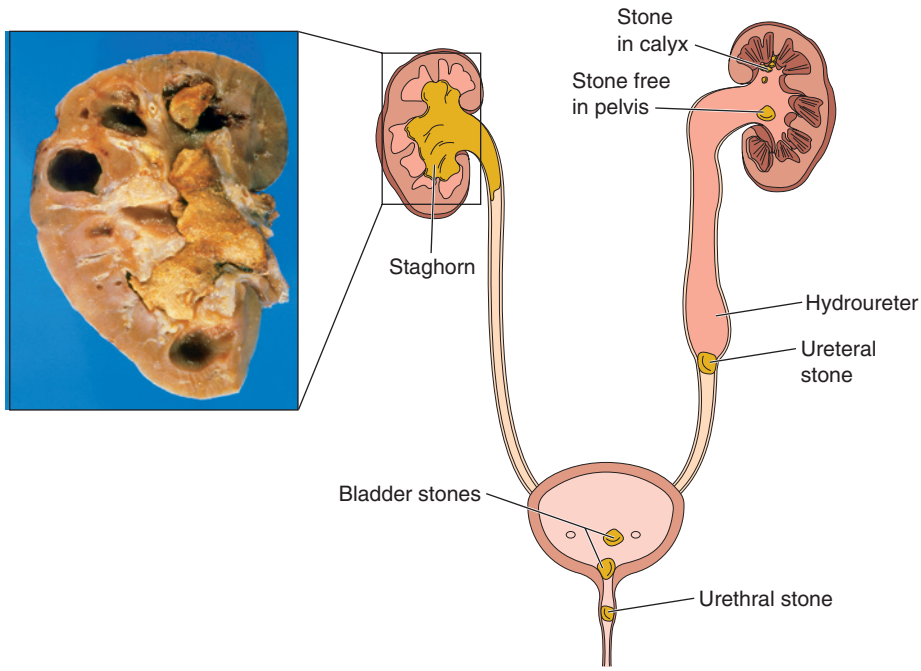


FIGURE 26.3 ■ Development and location of calculi within the urinary tract

Source: Image © Dr E. Walker/Science Source.

Most (75–80%) kidney stones are *calcium stones*, composed of calcium oxalate and/or calcium phosphate. These stones are generally associated with high concentrations of calcium in the blood or urine. *Uric acid stones* develop when the urine concentration of uric acid is high. They are more common in men and may be associated with gout. Genetic factors contribute to the development of uric acid stones and calcium stones. *Struvite stones* are associated with UTI caused by urease-producing bacteria such as *Proteus*. These stones can become very large, filling the renal pelvis and calyces, and are often called *staghorn stones* because of their shape. *Cystine stones* are rare and are associated with a genetic amino acid metabolic defect. The types of renal calculi, contributing factors and recommended dietary modifications are listed in Table 26.2.

Manifestations

The symptoms caused by urinary calculi vary with their size and location (see the 'Manifestations' box below). Manifestations

TABLE 26.2 Risk factors and interventions for renal calculi

STONE TYPE AND INCIDENCE	RISK FACTORS	MANAGEMENT
Calcium phosphate and/or oxalate 75–80%	Hypercalciuria and hypercalcaemia: hyperparathyroidism, immobility, bone disease, vitamin D intoxication, multiple myeloma, renal tubular acidosis, prolonged steroid intake, alkaline urine, dehydration, inflammatory bowel disease	Pharmacology: thiazide diuretics, phosphates, calcium-binding agents Dietary: limit foods high in calcium and oxalate, increase foods that acidify urine Other: increase hydration, exercise
Struvite 15–20%	UTIs, especially *Proteus* infections	Pharmacology: antibiotic therapy for UTI Other: surgical intervention or lithotripsy to remove stone
Uric acid 5–10%	Gout, increased purine intake, acid urine	Pharmacology: potassium citrate, allopurinol Dietary: low-purine diet Other: increase hydration
Cystine (uncommon)	Genetic defect, acid urine	Pharmacology: penicillamine, sodium bicarbonate Dietary: sodium restriction Other: increase hydration

develop as a result of obstructed urine flow with resulting distension, and tissue trauma caused by passage of the rough-edged, crystalline stone.

FAST FACTS

- Most urinary stones form in the renal pelvis and are composed primarily of calcium salts.
- Loss of calcium from the bones (e.g. due to immobility, osteoporosis) and dehydration are major risk factors for urinary stones.

Calculi affecting the kidney calyces and pelvis may cause few symptoms. If the stone has gradually or partially obstructed urinary flow, dull, aching flank pain may be present, but renal calculi may be silent, without symptoms. Bladder calculi may cause few symptoms other than dull suprapubic pain on exercise or after voiding.

Renal colic—acute, severe flank pain on the affected side—develops when a stone obstructs the ureter, causing ureteral spasm. The pain of renal colic may radiate to the suprapubic region, groin and external genitals (the scrotum or labia). The severity of the pain often causes a sympathetic response with associated nausea, vomiting, pallor and cool, clammy skin.

Manifestations of UTI, including chills and fever, frequency, urgency and dysuria, may accompany urinary calculi at any level. Trauma to the urinary tract by the calculi may cause gross or microscopic haematuria. Gross haematuria may also be the only sign of bladder stones.

Complications

Urinary stones may obstruct urine flow at any point of the urinary tract, leading to complications such as hydronephrosis and urinary stasis with subsequent infection.

MANIFESTATIONS Urinary calculi

KIDNEY STONES
- Often asymptomatic
- Dull, aching flank pain
- Microscopic haematuria
- Manifestations of UTI

URETERAL STONES
- Renal colic
- Acute, severe flank pain on affected side
- Often radiates to suprapubic region, groin and external genitals
- Nausea, vomiting, pallor and cool, clammy skin

BLADDER STONES
- May be asymptomatic
- Dull suprapubic pain, possibly associated with exercise or voiding
- Gross or microscopic haematuria
- Manifestations of UTI

Obstruction

Stones can obstruct the urinary tract at any point from the calyces of the kidney to the distal urethra, impeding the outflow of urine. If the obstruction develops slowly, there may be few or no symptoms, whereas sudden obstruction (e.g. blockage of a ureter by a passing stone) may cause severe manifestations. Urinary tract obstruction can ultimately lead to renal failure. The degree of obstruction, its location and the duration of impaired urine flow determine the effect on renal function.

HYDRONEPHROSIS The kidneys continue to produce urine, causing increased pressure and distension of the urinary tract behind the obstruction. **Hydronephrosis**, distension of the renal pelvis and calyces, and *hydroureter*, distension of the ureter, are possible results. If the pressure is unrelieved, the collecting tubules, proximal tubules and glomeruli of the kidney are damaged, causing a gradual loss of renal function.

Acute hydronephrosis typically causes colicky pain on the affected side. The pain may radiate into the groin. Chronic hydronephrosis develops slowly and may have few manifestations other than dull, aching back or flank pain. When hydronephrosis is significant, a palpable mass may be felt in the flank region. Haematuria and signs of UTI such as pyuria, fever and discomfort may occur. Gastrointestinal symptoms such as nausea, vomiting and abdominal pain may accompany hydronephrosis (see the 'Manifestations' box). In severe cases, shock caused by pain may develop and urgent treatment may be necessary.

INFECTION The urinary stasis associated with partial or complete obstruction increases the risk of urinary infection in the upper or lower urinary tract.

MANIFESTATIONS Acute and chronic hydronephrosis

ACUTE
- Acute, colicky pain; may radiate into groin
- Haematuria, pyuria
- Fever
- Nausea, vomiting, abdominal pain

CHRONIC
- Dull, aching flank pain
- Haematuria, pyuria
- Fever
- Palpable flank mass

INTERPROFESSIONAL CARE

Management of urinary calculi focuses on relieving acute symptoms, destroying or removing stones, and preventing further stone formation. Asymptomatic stones (those not causing pain, infection or obstruction) are treated conservatively.

Diagnosis

Laboratory and diagnostic tests that may be ordered when urinary calculi are suspected include the following:

- *Urinalysis* to assess for haematuria and the possible presence of WBCs and crystal fragments. The urine pH is helpful in identifying the type of stone.
- *Chemical analysis* of any stones passed in the urine determines the type of stone and suggests measures to prevent further stone formation. Retrieving stones or teaching the person to do so is a nursing responsibility. All urine is strained and may be saved. Any visible stones or sediment is sent for analysis.
- *Urine calcium*, *uric acid* and *oxalate* levels measure the amount of these substances excreted over a 24-hour period and may be assessed to help identify possible causes of lithiasis. Elevated calcium levels occur in hyperparathyroidism, Cushing's syndrome and osteoporosis, all of which may contribute to lithiasis. Uric acid levels may be elevated in people with gout and those at risk of forming uric acid calculi. Urine oxalate excretion may help to differentiate calcium oxalate from calcium phosphate stones.
- *Serum calcium*, *phosphorus* and *uric acid* levels may be obtained to help identify factors contributing to calculus formation.
- *X-ray* (kidneys, ureters and bladder) of the lower abdomen that requires no special preparation. Calculi may be identified as opacities in the kidneys, ureters and bladder.
- *Renal ultrasound* is a non-invasive test that uses reflected sound waves to detect stones and evaluate the kidneys for possible hydronephrosis.
- *Computed tomography (CT) scan* of the kidney, with or without contrast medium, directed at the kidney from many angles to provide a computer-generated photograph that shows calculi, ureteral obstruction and other renal disorders.
- *IVP* may be done to visualise the kidneys, ureters and bladder after injection of a contrast medium. IVP may be done when x-rays, renal ultrasonography and CT scan fail to demonstrate clear evidence of urinary calculi. (See 'Diagnostic tests, intravenous pyelogram (IVP)' in Chapter 25.)
- *Cystoscopy* is used to visualise and possibly remove calculi from the urinary bladder and distal ureters.

Nursing implications and care for people undergoing these tests and procedures are outlined in Chapter 25.

Medications

An acute episode of renal colic is treated with analgesia and hydration. A narcotic analgesic such as morphine sulfate is given, often intravenously, to relieve pain and reduce ureteral spasm. Indomethacin, a non-steroidal anti-inflammatory drug (NSAID), given as a suppository, may reduce the amount of narcotic analgesia required for acute renal colic. Oral or intravenous fluids reduce the risk of further stone formation and promote urine output.

After analysis of the calculus, various medications may be ordered to inhibit or prevent further lithiasis. A thiazide diuretic, frequently prescribed for calcium calculi, acts to reduce urinary calcium excretion and is very effective in preventing further stones. Potassium citrate alkalinises urine by raising the pH and is often prescribed to prevent stones that tend to form in acidic urine (uric acid, cystine and some forms of calcium stones). See Table 26.2 for other preparations related to types of stones. Nursing responsibilities focus on teaching the person about the prescribed medication, its importance in preventing further stone formation and potential adverse effects.

Nutrition and fluid management

Diet modifications are often prescribed to change the character of the urine and prevent further lithiasis.

Increased fluid intake of 2500 to 3000 mL per day is recommended, regardless of stone composition. A fluid intake to ensure the production of approximately 2000 to 2500 mL of urine a day prevents the stone-forming salts from becoming concentrated enough to precipitate. Fluid intake should be spaced throughout the day and evening. Some authorities recommend that drinking one to two glasses of water at night helps to prevent concentration of urine during sleep.

Recommended dietary changes may include reduced intake of the primary substance forming the calculi. For calcium stones, dietary calcium and vitamin-D-enriched foods are limited. Limiting vitamin D inhibits the absorption of calcium from the GI tract. Calcium stones may be a calcium phosphate salt, calcium oxalate or a combination of both; therefore, phosphorus and/or oxalate may also be limited in the diet.

The person with uric acid stones requires a diet low in purines. Organ meats, sardines and other high-purine foods may be limited.

In addition to limiting certain foods, the diet may be modified to maintain a urinary pH that does not promote lithiasis. Uric acid and cystine stones tend to form in acid urine. Foods that tend to alkalinise the urine may be recommended. Because alkaline urine promotes formation of calcium stones and urinary tract infections, the diet may be modified to lower the pH of the urine. Foods that affect urinary pH and foods high in various stone components are summarised in Table 26.3.

TABLE 26.3 Teaching people with urolithiasis: possible food and fluid modifications

Foods high in calcium	Beans and lentils, chocolate and cocoa, dried fruits, canned or smoked fish except tuna, flour, milk and milk products
Foods high in oxalate	Asparagus, beer and colas, beetroot, cabbage, celery, chocolate and cocoa, fruits, green beans, nuts, tea, tomatoes
Purine-rich foods	Goose, organ meats, sardines and herring, venison; moderate in beef, chicken, crab, pork, salmon, veal
Acidifying foods	Cheese, cranberries, eggs, grapes, meat and poultry, plums and prunes, tomatoes, whole grains
Alkalinising foods	Green vegetables, fruit (except as noted above), legumes, milk and milk products, rhubarb

Surgery

Treatment of existing calculi depends on the location of the stones, the extent of obstruction, renal function, the presence or absence of UTI, and the individual's general state of health. In general, a stone is removed if it is causing severe obstruction, infection, unrelieved pain or serious bleeding (Fauci et al., 2008).

Lithotripsy, using sound or shock waves to crush a stone, is the preferred treatment for urinary calculi. Several techniques are available. **Extracorporeal shock wave lithotripsy (ESWL)** is a non-invasive technique for fragmenting kidney stones using shock waves generated outside the body. Acoustic shock waves are aimed under fluoroscopic guidance at the stone (see Figure 26.4). These shock waves travel through soft tissue without causing damage, but shatter the stone as its greater density stops their progress. Repeated shock waves pulverise the stone into fragments small enough to be eliminated in the urine. The procedure may require 30 minutes to 2 hours to complete. Intravenous sedation generally is adequate to maintain comfort during the procedure (Doherty, 2009). See the box below for nursing care of the person undergoing a lithotripsy procedure.

Lithotripsy also may be performed using a percutaneous ultrasonic or laser technique. *Percutaneous ultrasonic lithotripsy* uses a nephroscope inserted into the kidney pelvis through a small flank incision (see Figure 26.5). The stone is fragmented using a small ultrasonic transducer and the fragments are removed through the nephroscope. *Laser lithotripsy* is an alternative to ultrasonic lithotripsy. Laser beams are used to disintegrate the stone, without damaging soft tissue. A nephroscope or a ureteroscope (passed up the ureter from the bladder during cystoscopy) is used to guide the laser probe into direct contact with the stone.

A double J stent may be inserted into the affected ureter to maintain its patency following ESWL or other lithotripsy procedures. (See the box below for 'Nursing care of the person having lithotripsy'.)

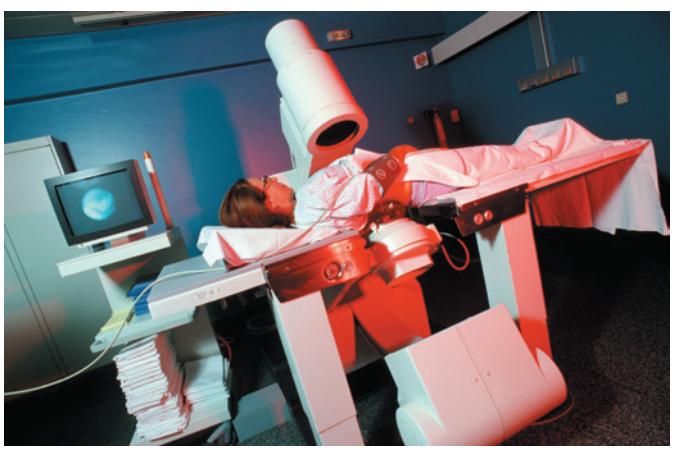


FIGURE 26.4 ■ Extracorporeal shock wave lithotripsy. Acoustic shock waves generated by the shock wave generator travel through soft tissue to shatter the urinary stone into fragments, which are then eliminated in the urine

Source: PHANIE/Science Source.

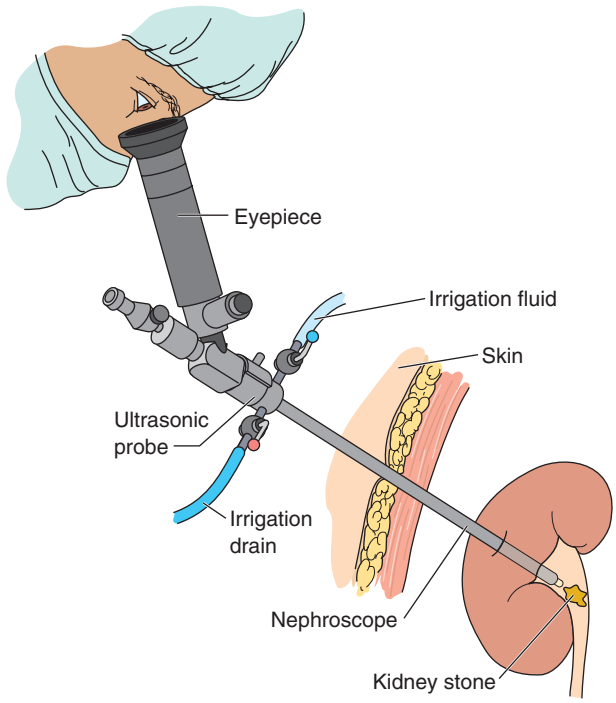


FIGURE 26.5 ■ Percutaneous ultrasonic lithotripsy. A nephroscope is inserted into the renal pelvis and ultrasonic waves are used to fragment the stone. The fragments are then removed through the nephroscope

On rare occasions, surgical intervention is necessary to remove a calculus in the renal pelvis or ureter. *Ureterolithotomy* is incision in the affected ureter to remove a calculus. *Pyelolithotomy* is incision into and removal of a stone from the kidney pelvis. A staghorn calculus that invades the calyces and renal parenchyma may require a *nephrolithotomy* for removal. See Chapter 3 for care of the person having surgery.

Bladder stones may be removed using an instrument passed through a cystoscope to crush the stones. The remaining stone fragments are then irrigated out of the bladder using an acid solution to counteract the alkalinity that precipitated stone formation.

Nursing care

Nursing care for the person with urolithiasis is directed at providing for comfort during acute renal colic, assisting with diagnostic procedures, ensuring adequate urinary output and teaching information necessary to prevent future stone formation.

Health promotion

Discuss the importance of maintaining an adequate fluid intake with all people. Stress the need to increase fluid intake during warm weather and strenuous exercise or physical labour. Discuss the relationship between weight-bearing activity and retention of calcium in the bones. Encourage the maintenance

NURSING CARE OF THE PERSON having lithotripsy

PREOPERATIVE CARE

- Assess knowledge and understanding of the procedure, providing information as needed. *Anxiety is reduced and recovery is enhanced and hastened when the individual is fully prepared for surgery*.
- Follow directions from the radiology department, doctor or anaesthetist for withholding food and fluids and for bowel preparation prior to surgery. Conscious sedation, general anaesthesia or spinal anaesthesia may be required, depending on the procedure. *Faecal material in the bowel may impede fluoroscopic visualisation of the kidney and stone.*

POSTOPERATIVE CARE

- In the initial period, monitor vital signs frequently. *The kidney is highly vascular; therefore, haemorrhage and resulting shock are potential complications of lithotripsy. Bleeding may be internal or retroperitoneal and difficult to detect.*
- Monitor amount, colour and clarity of urine output. *Urine is often bright red initially, but bleeding should diminish within 48 to 72 hours. Cloudy urine may indicate the presence of an infection.*
- Maintain placement and patency of urinary catheters. Anchor ureteral catheters or nephrostomy tubes securely. Irrigate gently if ordered. *A kinked or plugged catheter may result in hydroureter, hydronephrosis and kidney damage. Decreased urinary output and flank pain are possible symptoms of obstructed urine flow. Excessive force in irrigation may cause trauma and bleeding.*
- Ensure fluid intake of at least 3 L per day to promote urine production and to flush the renal system.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Prepare for discharge by teaching care of the indwelling catheter, urine-collection device and incision site (if present). Teach signs and symptoms to report: urine leakage from incision for more than 4 days, symptoms of infection, pain, bright haematuria. *If discharged with dressings and catheters in place, the individual and family need necessary information to provide self-care.*
- Teach measures to reduce the risk of further lithiasis. Prevention of stone formation is important to preserve renal function, as many people *have repeated episodes of lithiasis and renal colic. Dietary advice may be required.*

of physical activity as much as possible to prevent bone resorption (loss) and possible hypercalciuria.

Instruct people with known gout to maintain a generous fluid intake so as to produce at least 2000 mL of urine every day. Discuss the risk of lithiasis with individuals who have frequent UTIs and teach measures to reduce the incidence of UTI and the risk of lithiasis.

Assessment

Obtain subjective and objective assessment data specific to urolithiasis:

- *Health history:* complaints of flank, back or abdominal pain, radiation, characteristics and timing, aggravating or relieving factors; other symptoms such as nausea and vomiting; possible contributing factors such as dehydration; previous or family history of kidney stones; current or previous treatment measures.
- *Physical examination:* general appearance including position, vital signs; skin colour, temperature, moisture, turgor; abdominal, flank or costovertebral tenderness; amount, colour and characteristics of urine (presence of haematuria, bacteria, pyuria, pH).

Nursing diagnoses and interventions

See the nursing care plan that follows this section for additional nursing diagnoses and interventions.

Acute pain

Pain is the primary outward manifestation of urolithiasis, particularly when a stone lodges within a ureter causing acute obstruction and distension. Invasive and non-invasive procedures to remove or crush stones also may be painful. People undergoing surgery also experience incisional pain.

CONSIDERATION FOR PRACTICE

The intensity of renal colic pain can cause a vasovagal response with resulting hypotension and syncope. Always provide for the person's personal safety and ensure they are not at risk from shock in this situation.

- Assess pain using a standard pain scale and its characteristics. Administer analgesia as ordered and monitor its effectiveness. *The intensity, type of pain and its responsiveness to analgesia provide valuable clues as to its cause. Regular administration of prescribed analgesics controls pain more effectively than waiting until pain becomes intolerable. Administering an ordered NSAID on a routine schedule may significantly reduce the need for narcotic analgesia for those with renal colic.*
- Unless contraindicated, encourage fluid intake and ambulation for people with renal colic. *Increased fluids and ambulation encourage urinary output, thereby facilitating movement of the calculus through the ureter and decreasing pain.*
- Use non-pharmacological measures such as positioning, moist heat, relaxation techniques, guided imagery and diversion as adjunctive therapy for pain relief. *Adjunctive pain relief measures can enhance the effectiveness of analgesics and other prescribed treatment.*
- If surgery has been performed, monitor urinary output, catheters, incision and wound drainage. *Pain may be a symptom of proximal distension due to a blocked catheter. Infection or haematoma at the surgical site can significantly increase perceived pain.*

Impaired urinary elimination

Obstruction of the urinary tract is the primary problem associated with urolithiasis. Obstruction can ultimately lead to stasis, infection or irreversible renal damage.

- Monitor amount and character of urine output. If catheterised, measure output hourly. Document any haematuria, dysuria, frequency, urgency and pyuria. Strain all urine for stones, saving any recovered stones for laboratory analysis. *The amount of urine output helps determine possible urinary tract obstruction and adequacy of hydration. Gross or microscopic haematuria is often associated with calculi and with procedures used to remove stones, such as cystoscopy or lithotripsy. A change in the amount of haematuria may indicate stone passage or a complication. Dysuria, frequency, urgency and cloudy urine are symptoms of UTI, often associated with urolithiasis. Antibiotic therapy may be required. Analysis of stones recovered from the urine can direct measures to prevent further lithiasis.*

CONSIDERATION FOR PRACTICE

A stone that completely obstructs the ureter can lead to hydronephrosis and kidney damage on the affected side. Report symptoms of hydronephrosis such as dull flank pain or aching and changes in renal function studies (BUN and serum creatinine). Because the other kidney continues to function, urine output may not fall significantly with obstruction of one ureter. A rising BUN and serum creatinine may be early signs of renal failure.

- Maintain patency and integrity of all catheter systems. Secure catheters well, label as indicated and use sterile technique for all ordered irrigations or other procedures. *A kinked or plugged catheter, particularly a ureteral catheter or nephrostomy tube, may damage the urinary system. Labelling catheters can prevent mistakes such as inappropriate irrigation or clamping. Any catheter increases the risk of infection; aseptic technique in all procedures reduces this risk.*

Deficient knowledge

The person with urolithiasis has multiple learning needs. These include information about the disease and its possible consequences, any diagnostic or therapeutic procedures performed and strategies to prevent future lithiasis.

- Assess understanding and previous learning. *Relating information to previously learned material enhances retention and understanding.*
- Present all information in a manner appropriate to knowledge base, developmental and educational level, and current needs. *Learning is an active process that requires the individual's participation. Tailoring teaching to the individual increases involvement.*
- Educate about all diagnostic and treatment procedures. *Knowing what to expect reduces anxiety, enhances compliance and hastens recovery.*
- If the person will be managed in the community, educate to:
  a. Collect and strain all urine, saving any stones.
  b. Report stone passage to the doctor and deliver the stone for analysis.
  c. Report any changes in the amount or character of urine output to doctor.

  When pain can be managed with oral analgesics, urinary stones are managed in the community. The individual needs to know how and why to collect the calculus and indicators of complications, such as reduced urine output and cloudy or bloody urine.
- Teach measures to prevent further urolithiasis:
  a. Increase fluid intake to 2500 to 3500 mL per day.
  b. Follow recommended dietary guidelines.
  c. Maintain activity level to prevent urinary stasis and bone resorption.
  d. Take medications as prescribed.
  e. Obtain prompt treatment for any further urinary infections.
  f. Avoid too much tea or coffee, and reduce salt in the diet. Restrict carbonated drinks such as cola and beer to 1000 mL per week as these contain phosphoric acid, a precursor to calculi formation.

  The risk of recurrent lithiasis is approximately 50%; however, this risk can be reduced by measures to prevent conditions favouring stone formation.
- Teach about the relationship between urinary calculi and UTI, emphasising preventive measures and the importance of prompt treatment. *Urinary tract infection promotes urolithiasis and thus requires prompt treatment to reduce this risk.*

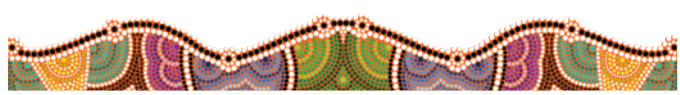
Community-based care

The person with urinary calculi needs to know how to manage existing stones and what to do to reduce the risk of future stone formation. Discuss the following topics to prepare the person and family for home care:

- importance of maintaining a fluid intake adequate to produce 2000 to 2500 mL of urine per day
- prescribed medications, their management and potential adverse effects
- dietary recommendations
- prevention, recognition and management of UTI
- any further diagnostic or treatment measures planned.

If the person is to be discharged with dressings, a nephrostomy tube or a catheter, educate the individual and family about the following:

- how to change dressings, maintaining aseptic technique
- assessment of the wound and skin for healing and possible complications such as infection or skin breakdown
- how to manage drainage systems and maintain their patency
- emptying drainage bags and assessing urine output
- recommendations for follow-up care and when to contact the doctor.



NURSING CARE PLAN A person with urinary calculi

Richard Leton, aged 44, owns a small business. He is admitted to the medical unit from the emergency department after awakening at 4 am with severe right-sided pain. His FBC is normal and urinalysis reveals microscopic haematuria, but no protein or bacteria. A renal ultrasound shows a 4–5 mm stone partially obstructing the right ureter.

The admitting nurse notes that Mr Leton is pale, diaphoretic and very anxious. He complains of nausea and asks for an emesis basin. Mr Leton received 4 mg of intravenous morphine sulfate approximately 2.5 hours ago. He denies pain at this time but says, 'I'm scared to death that it'll come back. I couldn't even move, it hurt so bad.'

ASSESSMENT

Mr Leton's history reveals no previous episodes of renal calculi. He felt well until the pain awakened him during the night. He admits that he has been working under a deadline to complete a construction project and that he probably has not been drinking enough fluids, 'considering how hot it's been'. Physical assessment findings include T 38.0°C, P 98, R 24 and BP 160/86. Colour is pale to ashen, skin cool and moist. His abdomen is firm with moderate tenderness in the right upper outer quadrant. The doctor orders an IV of 5% dextrose in normal saline at 200 mL/h until nausea is relieved, then PO fluids of at least 3000 mL/24 h; morphine sulfate 2 to 10 mg IV prn for pain; indomethacin (Indocid) 50 mg per rectal suppository q8h; promethazine (Phenergan) 25 mg PO or per suppository q6h prn for nausea; activity to tolerance; and strain all urine, sending recovered stones for analysis.

DIAGNOSES

- *Anxiety* related to anticipation of recurrent severe pain.
- *Risk of imbalanced nutrition: less than body requirements* related to nausea.
- *Acute pain* related to partial obstruction of right ureter by calculus.
- *Impaired urinary elimination* related to partial obstruction of ureter by calculus.
- *Deficient knowledge* related to lack of information about disease process, contributing factors and management.

PLANNING

- Plan interventions to reduce pain and anxiety.
- Plan interventions to reduce nausea and facilitate adherence to increased fluid intake.
- Develop collaboratively a nutrition and fluid education program.

Expected outcomes

- Demonstrate reduced anxiety by relaxed facial expression, vital signs within his normal range and ability to rest when not disturbed.
- Consume at least 50% of diet and 100% of ordered fluids without nausea or vomiting.
- Request analgesia as needed at onset of pain; report effective pain relief.
- Maintain urine output of 2500 mL/24 h with no signs of infection or obstruction (such as increased pain, dysuria, pyuria or haematuria).
- Demonstrate an understanding of the process of urolithiasis and contributing factors.
- Verbalise knowledge of dietary, fluid intake and other measures required to reduce risk of future stone formation.

IMPLEMENTATION

- Reassure that measures to prevent further episodes of renal colic are being implemented and that medication is available to relieve pain promptly.
- Assess the effectiveness of analgesia and its adverse effects, especially nausea.
- Maintain IV as ordered until oral fluid intake exceeds 200 mL of fluid per hour while awake.
- Measure and strain all urine. Assess urine for colour, clarity and odour.
- Teach about urolithiasis and its risk factors, especially as they relate to Mr Leton.
- Teach the importance of maintaining a high fluid intake, especially when working outdoors in hot weather; recommended dietary modifications and their rationale; ordered medications and their effects; how to identify and prevent UTI; and symptoms that should be reported to the doctor.

EVALUATION

Mr Leton passed the obstructing stone the evening after admission and is discharged the following day. On discharge, he states he has no pain or nausea, his urine is clear and pale yellow, and urinalysis is normal. Laboratory analysis shows that the calculus was calcium. Mr Leton is able to affirm the importance of continuing a high fluid intake. He confirms that he will reduce his intake of calcium-rich foods, such as milk and milk products, and that he will increase his intake of foods to acidify his urine. He is able to list foods to include in his diet and comments: 'You can be sure I'll follow my diet, drink my water and make sure I don't get an infection. I hope to never feel pain like that again!'

CRITICAL THINKING IN THE NURSING PROCESS

1. What factors contributed to the onset and timing of Mr Leton's ureteral colic?
2. What is the rationale for administering indomethacin, an NSAID, to a person with ureteral colic?
3. Why did the nurse include a nursing intervention to assess for a relationship between Mr Leton's nausea, his pain and the ordered analgesic agent?

REFLECTION ON THE NURSING PROCESS

1. Reflect on the educational strategies offered to Mr Leton and consider how you could incorporate these into your work day to prevent a reoccurrence for people at risk.
2. Considering the case study above, identify what new knowledge you have gained that will enhance how you deliver care to a person with ureteral colic.

THE PERSON WITH A URINARY TRACT TUMOUR

A malignancy can develop in any part of the urinary tract; however, 90% develop in the bladder, about 8% develop in the renal pelvis and only 2% develop in the ureter or urethra (Fauci et al., 2008). When diagnosed early, the 5-year survival rate for bladder cancer is 94% (American Cancer Society (ACS), 2009).

Incidence and risk factors

The incidence of bladder cancer is higher in people who smoke or are over the age of 65, and is three times higher in men than in women. Women experience poorer outcomes and survival (Patel et al., 2015).

Two main factors are implicated in the development of bladder cancer: the presence of carcinogens in the urine, and chronic inflammation or infection of bladder mucosa. Cigarette smoking is the primary risk factor for bladder cancer. The risk in smokers is twice that of non-smokers (ACS, 2009). The chemicals and dyes used in the plastics, rubber and cable industries; substances in the work environment of textile workers, leather finishers, spray painters and petroleum workers; and the chronic use of phenacetin-containing analgesic agents also are associated with a higher risk. Additional risk factors for bladder cancer include residence in an urban area, chronic UTIs and bladder calculi. The parasite *Schistosoma haematobium*, endemic to Egypt and the Sudan, also increases the risk of bladder cancer (Porth & Matfin, 2009). The risk of bladder cancer appears to be reduced by increasing the intake of fluids and vegetables (ACS, 2009).

FAST FACTS

The main risk factors for bladder cancer are:

- Male gender, age > 55, residence in urban area
- Cigarette smoking
- Occupational exposure to dyes or solvents
- Chronic UTI or bladder calculi
- Long-term use of cyclophosphamide

Pathophysiology

Most urinary tract malignancies arise from epithelial tissue. Transitional epithelium lines the entire tract from the renal pelvis through to the urethra. Carcinogenic products of nicotine metabolism and cigarette smoke are stored in the bladder then excreted in urine. It is possible the presence of carcinogens in bladder urine may cause abnormal bladder cell development. Squamous cell carcinoma of the urinary tract occurs less frequently than transitional epithelial cell tumours.

Urinary tract tumours begin as non-specific cellular alterations that develop into either flat or papillary lesions. These lesions may be either superficial or invasive. About 75% of bladder tumours are papillary lesions (*papillomas*), a polyp-like structure attached by a stalk to the bladder mucosa (see Figure 26.6). Papillomas are generally superficial, non-invasive tumours that bleed easily and frequently recur (Fauci et al., 2008). They rarely progress to become invasive and the prognosis for recovery is good.

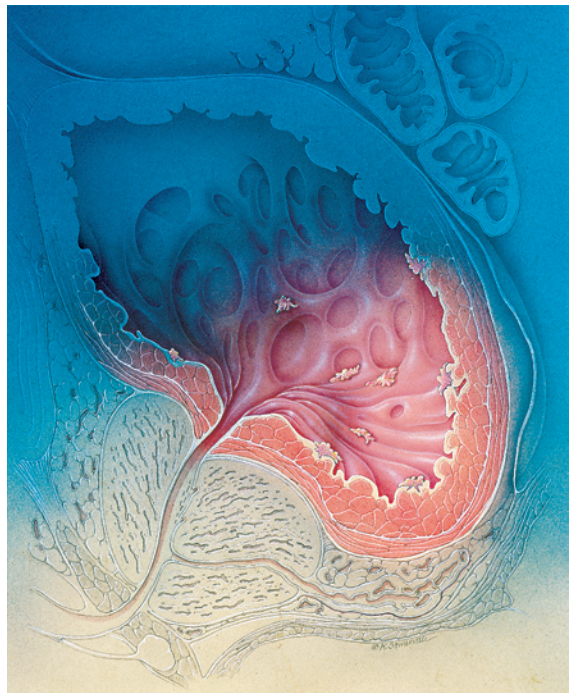


FIGURE 26.6 ■ Papillary transitional cell carcinoma of the urinary bladder

Source: Custom Medical Stock Photo, Inc.

Carcinoma in situ (CIS), which occurs less frequently, is a poorly differentiated flat tumour that invades directly and is associated with a poorer prognosis. Bladder tumours are rated by their cell type and grade. Grade I tumours are highly differentiated and rarely progress to become invasive, whereas grade III tumours are poorly differentiated and usually progress (Fauci et al., 2008). The staging of bladder tumours is outlined in Table 26.4. See Chapter 13 for more information about tumour grading and staging. When metastasis occurs, the pelvic lymph nodes, lungs, bones and liver are most commonly involved.

Manifestations

Painless haematuria is the presenting sign in 75% of urinary tract tumours. Haematuria may be gross or microscopic and is often intermittent, causing delay in seeking treatment (Porth & Matfin, 2009). Inflammation surrounding the tumour occasionally causes manifestations of a urinary tract infection, including frequency, urgency and dysuria. Ureteral tumours may cause colicky pain from obstruction. Tumours of the urinary tract typically cause few outward signs and may not be discovered until obstructed urine flow causes flank pain or renal failure.

CONSIDERATION FOR PRACTICE

Intermittent painless haematuria is the most common presenting symptom of bladder cancer. Instruct all people with painless haematuria to contact their doctor for follow-up testing.

TABLE 26.4 Bladder tumour staging

DEPTH OF INVOLVEMENT	TNM (TUMOUR, NODE, METASTASIS) STAGE	TUMOUR INVOLVEMENT
Superficial	T_a	Limited to the bladder mucosa
	T_1	Involvement of the bladder mucosa and submucosal layers
Invasive	T_2	Invasion of superficial muscle of bladder wall
	T_{3a}	Deep muscle invasion
	T_{3b}	Involvement of perivesicular fat
	$T_{3–4}N_+$	Regional (pelvic) lymph node involvement
	$T_{3–4}M_1$	Metastasis to distant lymph nodes or organs

INTERPROFESSIONAL CARE

Treatment of the person with a tumour of the urinary tract focuses on removing or destroying the cancerous tissue, preventing further invasion or metastasis and maintaining renal and urinary function.

Diagnosis

When a urinary tract tumour is suspected, the following diagnostic tests may be ordered:

- *Urinalysis* is done to evaluate for haematuria. Gross or microscopic haematuria is often the first indicator of a neoplasm in the urinary tract.
- *Urine cytology*, microscopic examination of cells in the urine, is performed to identify abnormal cells (tumour or pretumour cells). Periodic urine cytology is recommended for those at high risk of bladder cancer or its recurrence due to carcinogen exposure.
- *Ultrasound of the bladder* is a non-invasive test to detect bladder tumours. No dye is required and there is no exposure to radiation.
- *Intravenous pyelography* is used to evaluate the structure and function of the kidneys, ureters and bladder. IVP may reveal a rigid deformity of the bladder wall, obstruction of urine flow at the point of the tumour, or bladder filling or emptying defects.
- *Cystoscopy* and *ureteroscopy* allow direct visualisation, assessment and biopsy of lesions of the urethra, bladder or ureters using a lighted scope inserted through the urethra. Cystoscopy or ureteroscopy with biopsy allows definitive diagnosis of urinary tract tumours.
- *CT scan* or *MRI* is primarily used to evaluate tumour invasion or metastasis if suspected.
- See Chapter 25 for nursing care related to these diagnostic tests.

Medications

Immunological or chemotherapeutic agents administered by intravesical instillation (into the bladder) may be used either as the primary treatment for bladder cancer when multiple early lesions are present or to prevent recurrence following endoscopic tumour removal. Bacillus Calmette-Guérin (BCG; BCGLive, TheraCys) is a suspension of attenuated *Mycobacterium bovis* used to treat carcinoma in situ (CIS) and recurrent bladder tumours. Instillation into the bladder causes a local inflammatory reaction that eliminates or reduces superficial tumours. Systemic mycobacterial infection is a rare complication of intravesical BCG therapy that may require antituberculin treatment (McPhee et al., 2012). Other chemotherapeutic agents also may be administered intravesically, including doxorubicin, mitomycin C and interferon. Bladder irritation, frequency, dysuria and contact dermatitis are possible adverse reactions to intravesical chemotherapy. Suppression of bone marrow function also can occur as a result of intravesical treatment.

Radiation therapy

Radiation is an adjunctive therapy used in the treatment of urinary tumours. Although radiation alone is not curative, it can reduce tumour size prior to surgery and is used as palliative treatment for inoperable tumours and those who cannot tolerate surgery. Radiation therapy also is used in combination with systemic chemotherapy to improve local and distant relapse rates (McPhee et al., 2012) (see Chapter 13).

Surgery

A number of surgical procedures, ranging from simple resection of non-invasive tumours to removal of the bladder and surrounding structures, are used to treat urinary tract tumours. Indications for each procedure and specific nursing implications are outlined in Table 26.5.

Transurethral tumour resection may be performed by excision, *fulguration* (destruction of tissue using electric sparks generated by high-frequency current) or *laser photocoagulation* (use of light energy to destroy abnormal tissue). Laser surgery carries the lowest risk of bleeding and perforation of the bladder wall. Following cystoscopic tumour resection, individuals are followed at 3-month intervals for tumour recurrence. Recurrences may develop anywhere in the urinary tract, including the renal pelvis, ureter or urethra (Fauci et al., 2008).

Cystectomy, surgical removal of the bladder, is necessary to treat invasive cancers. Partial cystectomy may be done to remove a solitary lesion; however, radical cystectomy is the standard treatment for invasive tumours. The bladder and adjacent muscles and tissues are removed. In men, the prostate and seminal vessels are also removed, resulting in impotence. In women, a total hysterectomy and bilateral salpingo-oophorectomy

TABLE 26.5 Surgical procedures to treat bladder tumours

PROCEDURE	INDICATIONS	NURSING IMPLICATIONS
Transurethral resection of bladder tumour	Diagnose and treat superficial bladder tumours having low rate of recurrence; control bleeding	Maintain continuous bladder irrigation postoperatively; monitor for excessive bleeding; ensure catheter patency. Increase fluids to 2500–3000 mL/day. Give stool softeners to prevent straining.
Partial cystectomy	Resect solitary, isolated tumour at stage T_2 or T_3 not involving trigone	Maintain patency of urethral and/or suprapubic catheter to make sure suture lines are free of pressure; monitor for excess bleeding.
Complete or radical cystectomy	Remove large, invasive tumours; involvement of trigone	Permanent urinary diversion is required. Maintain patency and position of stents; urethral catheter may be in place to drain pelvic cavity.

(removal of the uterus, fallopian tubes and ovaries) accompanies the procedure, causing sterility. At the time of surgery, a **urinary diversion** is created to provide for urine collection and drainage. Either an *ileal conduit* (see Figure 26.7A) or a *continent urinary diversion* (see Figure 26.7B) is created to collect and drain urine. Table 26.6 describes the most frequently used urinary diversion techniques.

Surgical procedures to remove tumours involving other portions of the urinary tract vary according to the site and stage of the tumour. When the distal ureter is involved, the tumour may be resected and the ureter implanted into the opposite ureter to provide for drainage. A proximal ureteral tumour necessitates removal of the ureter and kidney on the affected side.

See the box below for nursing care of the person undergoing tumour resection and a urinary diversion.

Nursing care

The person who undergoes treatment for a tumour of the urinary tract has many nursing care needs because of alterations in the functional health patterns of elimination, health perception–health management, cognitive–perceptual, self-perception–self-concept, role–relationships and coping–stress–tolerance.

Health promotion

Encourage all individuals not to smoke. Provide referral to smoking cessation programs or clinics for those who wish to give up smoking. Encourage people at high risk of developing bladder cancer (see above) to have periodic examinations, including urinalysis and urine cytology.

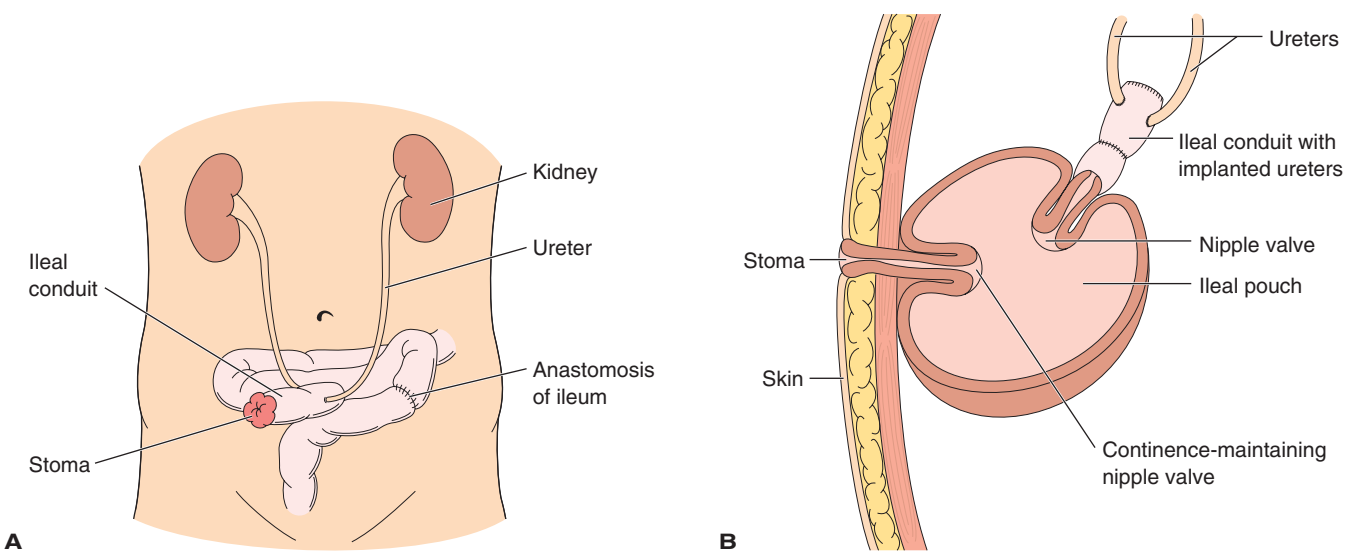


FIGURE 26.7 ■ Common urinary diversion procedures. *A*, Ileal conduit. A segment of ileum is separated from the small intestine and formed into a tubular pouch with the open end brought to the skin surface to form a stoma. The ureters are connected to the pouch. *B*, A continent urinary diversion. A segment of ileum is separated from the small intestine and formed into a pouch. Nipple valves are formed at each end of the pouch by intussuscepting tissue backwards into the reservoir to prevent leakage

TABLE 26.6 Urinary diversion procedures

PROCEDURE	DESCRIPTION	NURSING CONSIDERATIONS
Ileal conduit	Portion of ileum is isolated from small intestine, leaving vascular, lymphatic and neural connections intact; ileum is formed into pouch with open end brought to surface to form a stoma; ureters are inserted into pouch.	Most common urinary diversion. Continuous urine drainage necessitates appliance. Postoperative oedema may interfere with urine output. Risk of infection is less than for cutaneous ureterostomy, but potential for reflux is high. Good skin care vital because of constant contact with urine.
Continent internal ileal reservoir or continent ileal bladder conduit (Kock pouch)	Pouch is created as for ileal conduit but nipple valves are formed by intussuscepting tissue backwards into a reservoir to connect pouch to the skin and ureters to the pouch; filling pressure closes valves, preventing leakage and reflux.	Drainage collection device not necessary. The person must be willing and able to perform clean intermittent self-catheterisation every 2 to 4 hours. Continence valve mechanism may fail, requiring surgery for revision.
Indiana pouch (continent diversion)	A portion of the terminal ileum, ascending colon and caecum is isolated from the bowel with vascular and neural connections intact. Reservoir is formed from colon and caecum; portion of the ileum is brought to the surface to form nipple valve and stoma or is attached to urethral stump.	As for Kock pouch. The person must be able and motivated to manage self-catheterisation. Reservoir may absorb urea and electrolytes, resulting in imbalances. Significant portion of bowel is required to form pouch and stoma.
Ileocystoplasty	Section of the ileum is isolated and formed into U shape. Ureters are implanted in upper portion of the U. Urethra is anastomosed to central section.	Appropriate for men only because urethra is removed with cystectomy in women. Allows the person to void by relaxing pelvic muscles and using Valsalva manoeuvre.

Assessment

Nursing assessment related to urinary tract cancer includes both subjective and objective information:

- *Health history:* risk factors; history of haematuria or manifestations of UTI (dysuria, frequency, urgency, pyuria); lower abdominal discomfort or flank pain.
- *Physical examination:* general health; abdominal tenderness; urine for analysis.

Nursing diagnoses and interventions

Maintaining urinary output is the priority nursing care focus for the person with a bladder tumour. For additional potential nursing diagnoses and interventions for those with a bladder tumour, see the nursing care plan that follows.

Impaired urinary elimination

Whether the person has undergone transurethral resection of a bladder tumour or radical cystectomy with urinary diversion, urinary elimination is altered at least temporarily.

- Monitor urine output from all catheters, stents and tubes for amount, colour and clarity hourly for the first 24 hours postoperatively, then every 4 to 8 hours. *Decreased urine output may indicate impaired catheter or drainage system patency. Prompt intervention is necessary to prevent hydronephrosis. A change in colour or clarity may indicate a complication such as haemorrhage or infection.*
- Label all catheters, stents and their drainage containers. Maintain separate closed gravity drainage systems for each. *Clear identification of each tube can prevent errors in irrigating and calculating outputs. Separate closed systems minimise the risk and extent of potential bacterial contamination and resultant infection.*

CONSIDERATION FOR PRACTICE

Promptly report urine output of less than 30 mL per hour. This may indicate low vascular volume or renal insufficiency. Prompt intervention is vital to restore cardiac output and prevent acute kidney injury (previously known as acute renal failure).

- Secure ureteral catheters and stents with tape; prevent kinking or occlusion; and maintain gravity flow by keeping drainage bag below the level of the kidneys. *Impaired urine flow can lead to urinary retention and distension of the bladder, a newly created reservoir or the renal pelvis (hydronephrosis).*

CONSIDERATION FOR PRACTICE

Using aseptic techniques, strictly follow guidelines for irrigating catheters. Catheters placed in the kidney pelvis are irrigated using gentle pressure and small amounts of fluid (10 to 15 mL) to avoid damaging renal tissues.

- Encourage fluid intake of 3000 mL per day. Increased fluid intake maintains a high urinary output, reducing the risk of infection. *Dilute urine is less irritating to the skin surrounding the stoma site. Electrolyte reabsorption from reservoirs may increase risk of calculi; high fluid intake and urine output reduce this risk.*
- Encourage activity to tolerance. *Ambulation promotes drainage of urine from reservoirs and helps prevent calcium loss from bones, which could precipitate calculus formation.*

CONSIDERATION FOR PRACTICE

Monitor urine output closely for first 24 hours after stents or ureteral catheters are removed. Oedema or stricture of ureters may impede output, leading to hydronephrosis and kidney damage.

Risk of impaired skin integrity

The skin surrounding the stoma site of an ileal conduit is at risk of irritation and breakdown. Because urine is acidic and contains high concentrations of electrolytes, it has a corrosive effect on skin. In addition, adhesives and sealants used to prevent pouch leakage may irritate the skin.

- Assess peristomal skin for redness, excoriation or signs of breakdown. Assess for urine leakage from catheters, stents or drains. Keep the skin clean and dry. Change wet dressings. *Intact skin is the first line of defence against infection. Impaired skin integrity may lead to local or systemic infection and impaired healing.*
- Ensure gravity drainage of urine collection device or empty bag every 2 hours. *Overfilling of the collection bag may damage the seal, allowing leakage and contact of urine with skin.*
- Change urine collection appliance as needed, removing any mucus from stoma. See Box 26.2 for care of a urinary stoma. *Meticulous care and protection of skin surrounding stoma can maintain integrity and prevent breakdown.*

Disturbed body image

A radical cystectomy and urinary diversion affect the person's body image. In most cases, an abdominal stoma is created, requiring either a drainage appliance or regular catheterisation of the stoma to drain urine. Removal of the prostate and seminal vesicles or the uterus and ovaries leaves the person sterile. If radiation or chemotherapy is planned as adjunctive therapy, the individual may experience hair loss, stomatitis, nausea and vomiting, or other disturbing side effects of therapy.

- Use therapeutic communication techniques to help the person deal with the situation by actively listening and responding to the individual's and the family's concerns. *People must know that their feelings and concerns are respected and valued. Denial, anger, guilt, bargaining or depression are common during the grieving process for the person's disordered body and normal for those undergoing a significant change in body image.*
- Recognise and accept behaviours that indicate use of coping mechanisms, encouraging adaptive mechanisms. *The person may initially use defensive coping mechanisms such as denial, minimisation and dissociation from the immediate situation to reduce anxiety and maintain psychological integrity. Adaptive mechanisms include learning as much as possible about the surgery and its effects, practising procedures, setting realistic goals and rehearsing various alternative outcomes.*
- Encourage looking at, touching and caring for the stoma and appliance as soon as possible. Allow the person to proceed gradually, providing support and encouragement. *Accepting the stoma as part of the self is vital to adapting to the changed body image and is indicated by a willingness to begin self-care.*
- Discuss concerns about returning to usual activities, perceived relationship changes and resumption of sexual relations. Provide referral to the support group or provide for contact with someone who has successfully adjusted to a urinary diversion. *Individuals and families may be reluctant to discuss topics of concern. An atmosphere of openness and acceptance facilitates expression of concerns and anxieties related to the changed body image.*

Risk of infection

Diagnostic instrumentation procedures, surgical manipulation and disruption of normal urinary tract defence mechanisms increase the risk of ascending urinary tract infection. When an ileal conduit or artificial bladder is created using bowel tissue, the normal bacteriostatic activity of bladder mucosa is lost. In addition,

BOX 26.2 Urinary stoma care

- Gather all supplies: a clean, disposable pouch; liquid skin barrier or barrier ring; 4 × 4 gauze squares; stoma guide; adhesive solvent; clean gloves; and a clean washcloth.
- Assess knowledge, learning needs and ability and willingness to assist with procedure. Explain the procedure as needed.
- Use standard precautions.
- Remove old pouch, pulling gently away from skin. Warm water or adhesive solvent may be used to loosen the seal if necessary.
- Assess stoma. Normally the stoma is bright red and appears moist. Report a dark purple, black or very pale stoma to the doctor. Slight bleeding with cleansing is normal, especially in the immediate postoperative period.
- Prevent urine flow during cleaning by placing a rolled gauze square or tampon over the stoma opening.
- Cleanse skin around the stoma with soap and water; rinse and pat or air dry.
- Use the stoma guide to determine correct size for the bag opening and/or protective ring seal. Trim the bag or seal as needed.
- Apply skin barrier; allow to dry.
- Apply the bag with an opening no more than 1 to 2 mm wider than outside of stoma. Allow no wrinkles or creases where the bag contacts the skin. Smooth side edges and lower edges first to ensure smooth fit.
- Connect bag to the urine-collection device. Dispose of old pouch, used supplies and gloves appropriately. Wash hands.
- Chart procedure, including stoma appearance and response of the person.

NURSING CARE OF THE PERSON having a cystectomy and urinary diversion

PREOPERATIVE CARE

- Provide routine preoperative care as outlined in Chapter 3.
- Assess knowledge of the proposed surgery and its long-term implications, clarifying misunderstandings and discussing concerns. *Those having surgery for cancer of the urinary tract are trying to cope with a diagnosis of cancer and may not fully understand the surgery and its potential effects. Open discussion can reduce the need for postoperative analgesia, facilitate postoperative recovery and adjustment.*
- Begin teaching about postoperative tubes and drains, self-care of stoma and control of drainage and odour. *Postoperative physiological and psychological stressors may interfere with learning. A basic understanding of what to expect in the way of tubes, drains and procedures reduces stress in the immediate postoperative period. Preoperative teaching can enhance recall and postoperative learning.*
- Refer to the Continence Nurse Advisor to help prepare the person physically and psychologically for the outcomes of surgery and changed body image.
- Assist in identifying a stoma site, avoiding folds of skin, bones, scar tissue and the waistline or belt area. The site should be visible and accessible for manipulation. Be sure to consider the person's occupation and style of clothing. *Stoma placement is a vital component of adjustment and self-care. Care is taken to place the stoma away from areas of constant irritation by clothing or movement. It should be located so that the person can cover and disguise the collecting device, maintain the seal to prevent leakage and effectively cleanse and maintain the site.*
- Perform bowel-preparation activities as ordered. *Bowel preparation is done to prevent faecal contamination of the peritoneal cavity and to decompress the bowel during surgery.*

POSTOPERATIVE CARE

- Provide routine postoperative care (see Chapter 3).
- Monitor intake and output carefully, assessing urine output every hour for the first 24 hours, then every 4 hours or as ordered. Call the doctor if urine output is less than 30 mL per hour. *Tissue oedema and bleeding may interfere with urinary output from stoma, catheters or drains. Maintenance of urine outflow is vital to prevent hydronephrosis and possible renal damage. A urine output of at least 30 mL per hour is necessary for effective renal function.*
- Assess colour and consistency of urine. Expect pink or bright red urine fading to pink and then clearing by the third postoperative day. Urine may be cloudy due to mucus production by bowel mucosa. *Bright red blood in the urine from a urinary diversion may indicate haemorrhage, necessitating further surgery. Excessive cloudiness or foul-smelling urine may indicate infection.*
- Assess size, colour and condition of the stoma and surrounding skin every 2 hours for the first 24 hours, then every 4 hours for 48 to 72 hours. Expect the stoma to initially appear bright red and slightly oedematous. Slight bleeding during cleansing is normal. *Compromised circulation causes the stoma to appear pale, grey or cyanotic, or to blanch when touched. Other complications, such as infection or impaired healing, may be evidenced by a change in the appearance of the stoma or incision.*
- Irrigate the ileal diversion catheter with 30 to 60 mL of normal saline every 4 hours or as ordered. *Mucus produced by the bowel wall may accumulate in the newly devised reservoir or obstruct catheters.*
- Monitor serum electrolyte values, acid–base balance and renal function tests such as BUN and serum creatinine. *Reabsorption of electrolytes from reservoirs created by portions of bowel may result in electrolyte imbalance and metabolic acidosis. Optimal renal function is necessary to maintain a normal state of homeostasis.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Teach the person and family about stoma and urinary diversion care, including odour management, skin care, increased fluid intake, pouch application and leakage prevention, self-catheterisation for those with continent reservoirs and signs of infection and other complications. *The ability to provide self-care is a significant factor in the adjustment to a changed body image. Teaching family members facilitates acceptance and adjustment. The family also needs this knowledge in case illness or disability interferes with the self-care capacity.*
- Ensure the Continence Nurse Advisor is available for counselling, guidance and support for the person and family in the postoperative period.

NURSING CARE PLAN A person with a bladder tumour

Ben Hussain is a 61-year-old car salesman. He is married and has five children, all of whom are grown and living away from home. A week ago Mr Hussain became alarmed when his urine was bright red. He called his doctor, who ordered urinalysis and urine cytology which revealed gross haematuria and poorly differentiated abnormal cells. Cystoscopy and tissue biopsy confirmed a tumour involving the bladder trigone area. Mr Hussain was admitted for a radical cystectomy and continent urinary diversion.

ASSESSMENT

Mr Hussain's admission history, obtained by the Registered Nurse, indicated that he had lost 4–7 kg during the last few months. He smoked two to three packets of cigarettes a day for 40 years, but cut back to a packet a day about a year ago, saying he could not quit smoking entirely. He drinks five to six cups of coffee daily and consumes a moderate amount of alcohol, averaging three to four drinks a day. Mr Hussain says that he is 'a little nervous about surgery and what

NURSING CARE PLAN A person with a bladder tumour (continued)

they're going to find'. The nurse notes that he fidgets and talks rapidly throughout their interview. Mr Hussain also expresses concern about how he will handle the pain after surgery, because he had never been hospitalised before his cystoscopy. Physical assessment findings include T 36.7°C, P 84, R 18 and BP 154/86. Examinations of the skin, neuromuscular and cardiac systems show no abnormalities. Auscultation of lung fields showed scattered expiratory crackles. Bowel sounds were very active; Mr Hussain explained that he began taking his bowel-preparation laxative the day before admission. Slight tenderness was noted in the suprapubic region and Mr Hussain's urine was clear and bright pink. CBC and chemistry screening results were within normal limits. Surgery was planned for 9 am the following day.

DIAGNOSES

- *Anxiety* related to undetermined extent of disease and fear of pain.
- *Deficient knowledge* related to care and management of continent urinary diversion.
- *Impaired urinary elimination* related to cystectomy and urinary diversion.
- *Risk of impaired gas exchange* related to smoking history and effects of anaesthesia.

PLANNING

- Plan education to reduce anxiety and concerns around surgery.
- Plan to include family in education sessions.
- Plan adequate pain reduction interventions.
- Plan a collaborative quit smoking and alcohol-reduction intervention programs.

Expected outcomes

- Verbalise decreased feelings of anxiety.
- Demonstrate appropriate postoperative pain relief through subjective reports of pain severity and objective findings.
- Be able to care for urinary diversion and surrounding skin prior to discharge.
- Demonstrate self-catheterisation of stoma using appropriate technique prior to discharge.
- Maintain normal urine output with acceptable colour and clarity and no signs of infection.
- Maintain adequate gas exchange as evidenced by good skin colour, O_2 saturation greater than 95% and clear lung sounds on auscultation.

IMPLEMENTATION

- Spend as much time as possible with Mr Hussain and his family preoperatively, answering questions fully and encouraging expression of fears and anxieties.
- Reinforce teaching given by the Continence Nurse Advisor and ensure that Mr Hussain understands his condition, the surgery and his postoperative plan of care.
- Provide verbal explanations supported by printed material and application examples when appropriate.
- Administer postoperative analgesia on a regular basis for the first 48 to 72 hours. Monitor for objective signs of unrelieved pain.
- Explain all procedures related to stoma and diversion care as they are being performed.
- Encourage Mr Hussain to look at the stoma and touch it when ready.
- Teach stoma and skin care, as well as self-catheterisation, emphasising measures to prevent skin irritation and urinary tract infection.
- Monitor urine output, colour, clarity and consistency every hour for first 24 hours, then every 4 hours for 24 hours, then every 8 hours. Report output of less than 30 mL per hour, bright bleeding, excessively cloudy or foul-smelling urine.
- Assist with use of incentive spirometer to promote respiratory function every hour while awake. Ambulate as soon as possible. Assess lung sounds every 4 hours, reporting increased crackles or diminished breath sounds.
- Refer Mr and Mrs Hussain to a local stoma group on discharge.

EVALUATION

On discharge, Mr Hussain has performed self-catheterisation and stoma and skin care several times. His wife also is able to catheterise the stoma and demonstrate skin care. His urine is pale yellow and slightly cloudy. Mr Hussain is ambulating independently and using oxycodone (Percocet) twice a day for pain relief. His lungs are clear and he is very proud of having 'survived' 7 days without a cigarette. He says, 'Now I'm going to try for 7 weeks, then 7 months, then 7 years without a cigarette!' A community care referral is made to continue supporting Mr Hussain in caring for his diversion and appliance at home.

CRITICAL THINKING IN THE NURSING PROCESS

1. How does cigarette smoking contribute to the increased risk of urinary tract tumours?
2. Suppose Mr Hussain had become confused, disoriented and tremorous, and had begun to experience visual hallucinations 2 to 3 days postoperatively. What would you suspect the cause to be? What would be the appropriate response?
3. Develop a plan of care for Mr Hussain for the nursing diagnosis *Risk of sexual dysfunction*.

REFLECTION ON THE NURSING PROCESS

1. Reflect on how Mr Hussain's attitudes towards smoking, alcohol and caffeine intake may have influenced his adherence and contribution to planned education programs.
2. How has involvement of the family in education programs influenced Mr Hussain's adherence to the programs?

the peristaltic action of the ureters may be disrupted and the vesicoureteral junction no longer prevents urine reflux. Adjunctive chemotherapy or radiation treatments may impair normal immune function and further increase the risk of infection.

- Maintain separate closed drainage systems, keeping drainage bags lower than the kidney, and prevent loops or kinks in drainage tubing, which impede urine flow. *Although urine is sterile when it leaves the kidney, bacteria grow rapidly in it. Therefore, prevention of urine reflux is essential to preventing UTI.*
- Monitor for signs of infection: elevated temperature, cloudy or foul-smelling urine, haematuria, general malaise, back or abdominal pain, and nausea and vomiting. *Infection undermines the healing process. Early detection and treatment help prevent long-term consequences such as chronic pyelonephritis.*

CONSIDERATION FOR PRACTICE

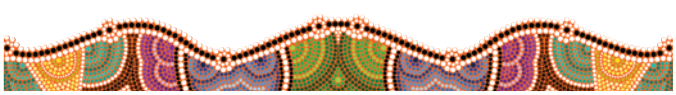
Impaired immune capability (due to ageing or the effects of chemotherapy) and urine cloudiness (related to the effects of urine on ileal mucosa) can mask signs of UTI such as fever and altered urine clarity. Be alert for more generalised manifestations, such as increased fatigue and malaise.

- Teach signs and symptoms of infection and self-care measures to prevent UTI. The person with a cystectomy and ileal diversion, urostomy or continent reservoir is at risk of UTI for life because of impaired urinary defence mechanisms. *Using clean or aseptic technique in providing care, increasing fluid intake and using measures to acidify urine minimise this risk to a certain degree but do not eliminate it.*

Community-based care

The need for individual and family teaching for the person who has had surgery to treat a urinary tract tumour is significant. For many, surgery means a lifelong change in urinary elimination. Even the individual who has undergone transurethral excision of bladder tumours requires follow-up cystoscopy on a regular basis and needs to be alert for signs of tumour recurrence.

The person who has had a urinary diversion needs teaching about care of the stoma and surrounding skin, prevention of urine reflux and infection, signs and symptoms of UTI and renal calculi, and, in some cases, self-catheterisation using clean technique. Referral to community health services will be an important aid to the individual and family in adapting to their changed situation and the impact on their lifestyles.



THE PERSON WITH URINARY RETENTION

Urinary retention, incomplete emptying of the bladder, can lead to overdistension of the bladder, poor detrusor muscle contractility and inability to urinate. If the problem persists, hydroureter and hydronephrosis can result.

Physiology review

Normally, bladder emptying is controlled by the interaction of muscle tone and the autonomic nervous system. The sympathetic nervous system (SNS) relaxes the detrusor muscle, allowing the bladder to fill with urine. The internal sphincter, a continuation of the detrusor muscle, remains closed during filling. Pressures within the bladder remain low during filling, in contrast to high sphincter and urethral pressures. Voluntary muscles of the external sphincter and pelvic floor help maintain these high pressures. When the bladder contains 150 to 300 mL of urine, signals from stretch receptors in the bladder wall are transmitted to the spinal cord and cerebral cortex. Reflexive bladder emptying can be consciously inhibited. During *micturition* (bladder emptying), parasympathetic stimulation causes the detrusor muscle of the bladder fundus to contract, opening the internal sphincter. The external sphincter then relaxes, allowing urine to flow out.

Pathophysiology

Either mechanical obstruction of the bladder outlet or a functional problem can cause urinary retention. Benign prostatic hypertrophy (BPH) is a common cause; difficulty initiating and maintaining urine flow is often the presenting complaint in men with BPH. Acute inflammation associated with infection or trauma of the bladder, urethra or perineal tissues may also interfere with micturition. Scarring due to repeated urinary tract infection can lead to urethral stricture and a mechanical obstruction. Bladder calculi may also obstruct the urethral opening from the bladder.

Surgery, particularly abdominal or pelvic surgery, may disrupt detrusor muscle function, leading to urine retention. Drugs also may interfere with its function. Anticholinergic medications such as atropine, glycopyrrolate (Robinul), propantheline bromide (Pro-Banthine), hyoscine (Buscopan) and others can lead to acute urinary retention and bladder distension. Other drugs with anticholinergic side effects may also cause urinary retention. Among these are anti-anxiety agents such as diazepam (Valium), antidepressant and tricyclic drugs such as imipramine (Tofranil), antiparkinsonian drugs, antipsychotic agents and some sedative/hypnotic drugs. In addition, antihistamines common in over-the-counter cough, cold, allergy and sleep-promoting drugs have anticholinergic effects which may interfere with bladder emptying. Diphenhydramine (Benadryl) is an example of a non-prescription antihistamine.

Voluntary urinary retention (particularly common among nurses!) may lead to overfilling of the bladder and a loss of detrusor muscle tone.

Manifestations

The person with urinary retention is unable to empty the bladder completely. Overflow voiding or incontinence may occur, with 25 to 50 mL of urine eliminated at frequent intervals. Assessment reveals a firm, distended bladder that may be displaced to one side of midline. Percussion of the lower abdomen reveals a dull tone, reflective of fluid in the bladder.

Severe urinary retention with resulting bladder distension impairs the ability of the vesicoureteral junction to prevent

backflow of urine into the ureters (see Figure 26.1 earlier in the chapter). Reflux of urine from the distended bladder distends the ureters (hydroureter) and kidneys (hydronephrosis). Hydronephrosis impairs renal function and acute kidney injury can result. See Chapter 27 for more information about acute kidney injury (previously known as acute renal failure).

INTERPROFESSIONAL CARE

Urinary retention is confirmed using a bladder scan or by inserting a urinary catheter (if possible) and measuring the urine output. Use of a bladder scan is preferred to reduce the risk of UTI (Teng et al., 2005).

An indwelling urinary catheter or intermittent straight catheterisation can prevent urinary retention and overdistension of the bladder. Cholinergic medications such as bethanechol chloride (Urocarb), which promote detrusor muscle contraction and bladder emptying, may be used. A medication with no anticholinergic side effects may be substituted when urinary retention is related to drug therapy.

Mechanical obstructions are treated by removing or repairing the obstruction when possible. Resection of the prostate gland may be done for urinary retention related to BPH. Bladder calculi are removed and measures to prevent their formation are instituted.

Nursing care

Health promotion measures to prevent urinary retention include monitoring urine output in at-risk individuals and evaluating drug regimens for medications known to interfere with detrusor muscle function. Pay particular attention to elimination when these drugs are ordered for (or used by) those with BPH or if there is known mechanical obstruction of urine flow.

Nursing diagnoses and interventions

Impaired urinary elimination

Nursing measures to promote urination include placing the individual in normal voiding position and providing for privacy. Additional measures include running water, placing the person's hands in warm water, pouring warm water over the perineum or taking a warm sitz bath.

In acute urinary retention, catheterisation may be necessary to relieve bladder distension and prevent hydronephrosis. Use a relatively small catheter (14 Fr. for a man, 12 Fr. for a woman). A coudé-tipped catheter is passed more easily in the older man with an enlarged prostate. Using 2% lignocaine gel (10 mL injected into the male urethra or 6 mL injected into the female urethra) reduces discomfort during catheterisation and the risk of catheter-associated infection and promotes pelvic muscle relaxation (Bardsley, 2005). Carefully observe the person as the distended bladder drains for signs of discomfort or shock.

Home care for the person with urinary retention varies, depending on the cause. Some people may be taught intermittent self-catheterisation. Instruct all those who have experienced urinary retention to avoid over-the-counter drugs that affect micturition, especially those with an anticholinergic effect (allergy and cold medications, and many non-prescription sleep aids). Other home care measures include double-voiding (urinate, remain on the toilet for 2 to 5 minutes, then urinate again), scheduled voiding, or, when other measures fail, an indwelling catheter. When an indwelling catheter is necessary, teach the person and family to use clean technique when changing from overnight bag to leg bag and to promptly report signs of UTI to the primary care provider.

CONSIDERATION FOR PRACTICE

Some people may experience a vasovagal response, becoming pale, sweaty and hypotensive if the bladder is rapidly drained (decompression). Draining urine in 500 mL increments and clamping the catheter for 5 to 10 minutes between increments may prevent this response. Haematuria also may occur with rapid bladder decompression. Promptly notify the doctor if haematuria develops.

THE PERSON WITH A NEUROGENIC BLADDER

The neurological connections influencing bladder filling, the perception of fullness and the need to void, and bladder emptying are complex. Disruption of the central or peripheral nervous systems may interfere with normal mechanisms, causing **neurogenic bladder**.

Pathophysiology

As noted in the physiology section on urinary retention, bladder filling and emptying are controlled by the central nervous system (CNS). This neurological control can be disrupted at any level: the cerebral cortex (voluntary impulses), the micturition centre of the midbrain, the spinal cord tracts or the peripheral nerves of the bladder itself.

Spastic bladder dysfunction

A simple reflex arc exists between the bladder and the spinal cord at levels S_2 to S_4. The stimulus of more than 400 mL of urine in the bladder causes reflex contraction of the detrusor muscle and bladder emptying unless voluntary control (cerebral input) is used to suppress it. Disruption of CNS transmission above the sacral spinal cord segment typically leads to *spastic neurogenic bladder*. Both sensory and voluntary control of urination is interrupted partially or totally, while the sacral reflex arc remains intact. The stimuli generated by bladder filling causes frequent spontaneous detrusor muscle contraction and involuntary bladder emptying. Spinal cord injury above the sacral segment is the most

common cause of a spastic bladder. Other causes include stroke, multiple sclerosis and other CNS lesions (Porth & Matfin, 2009).

Flaccid bladder dysfunction

Damage to the sacral spinal cord at the level of the reflex arc, the cauda equina or the sacral nerve roots causes loss of detrusor muscle tone and a *flaccid neurogenic bladder*. The perception of bladder fullness is lost and the bladder becomes overdistended, with weak and ineffective detrusor muscle contractions. Flaccid neurogenic bladder is seen with myelomeningocele and during the spinal shock phase of a spinal cord injury above the sacral region. During the spinal shock phase, all reflex activity below the level of spinal cord injury is suppressed.

Peripheral neuropathies may also cause bladder atony and overfilling. Either sensory or motor pathways (or both) may be disrupted, leading to incomplete bladder emptying and large residual volumes after voiding (Porth & Matfin, 2009). Diabetes mellitus is the most common cause of peripheral bladder neuropathy. Other causes include multiple sclerosis, chronic alcoholism and prolonged overdistension of the bladder.

INTERPROFESSIONAL CARE

Management of a neurogenic bladder focuses on maintaining continence and avoiding complications associated with overfilling or incomplete emptying of the bladder. Because self-care is the goal, teaching is a primary intervention for the healthcare team.

Diagnosis

The following diagnostic tests may be ordered for the person with a neurogenic bladder:

- *Urine culture* to detect possible urinary tract infection related to impaired bladder function.
- *Urinalysis* and *serum creatinine* and *BUN* to evaluate renal function. See the diagnostic tests table in Chapter 25 for normal BUN and creatinine levels. Ascending infection or hydronephrosis resulting from bladder overfilling and vesicoureteral reflux can damage the kidneys. Impaired renal function may lead to blood cells or protein in the urine and elevated BUN and creatinine levels.
- *Post-void catheterisation* to measure residual urine. Amounts greater than 50 mL may indicate ineffective detrusor muscle contractions, common in a neurogenic bladder.
- *Cystometrography* to evaluate bladder filling and the detrusor muscle tone and function. See Chapter 25 for nursing care of the person undergoing cystometrography.

Medications

Medications may be prescribed to increase or decrease the contractility of the detrusor muscle, to increase or decrease the tone of the internal sphincter, or to relax the external urethral sphincter.

Bethanechol (Urocarb), a cholinergic drug, stimulates detrusor muscle contraction in a flaccid neurogenic bladder. It is generally used to manage short-term urinary retention (e.g. following surgery or childbirth). It may be used in combination with bladder-training techniques to promote complete emptying of a neurogenic bladder. Anticholinesterase drugs such as neostigmine also may be used to increase detrusor muscle tone.

Anticholinergic drugs (parasympathetic blockers) relax the detrusor muscle and contract the internal sphincter, increasing bladder capacity in people with spastic bladder dysfunction. Oxybutynin (Ditropan) and tolterodine (Detrol) inhibit the muscarinic effects of acetylcholine on smooth muscle, reducing detrusor muscle spasticity and promoting bladder filling. Other anticholinergic drugs also may be used, including propantheline (Pro-Banthine) or flavoxate (Urispas). Dry mouth, blurred vision and constipation are potential adverse effects of anticholinergic medications. See the 'Medication administration' box below for drugs used to modify detrusor muscle activity.

Nutrition

Dietary measures to reduce the risk of UTI and urinary calculi may be suggested for the person with a neurogenic bladder. A moderate to high fluid intake and a diet that acidifies the urine are helpful. Cranberry juice is recommended to maintain urine acidity. See Table 26.3 for additional foods to include or avoid in the diet to help prevent UTI and urolithiasis. The timing of fluid intake may be regulated to promote continence.

Bladder retraining

Individuals with a spastic neurogenic bladder may use measures to stimulate reflex voiding, allowing scheduled toileting. Techniques include using trigger points—for example, stroking or pinching the abdomen, inner thigh or glans penis. Pulling pubic hairs, tapping the suprapubic region or inserting a gloved finger into the rectum and gently stretching the anal sphincter can also stimulate urination.

Credé's method (applying pressure to the suprapubic region with the fingers of one or both hands), manual pressure on the abdomen and the Valsalva manoeuvre (bearing down while holding the breath) promote bladder emptying for the person with a spastic or flaccid bladder.

CONSIDERATION FOR PRACTICE

Increasing lower abdominal and bladder pressure with Credé's method can stimulate autonomic dysreflexia in some people with spinal cord injuries. Autonomic dysreflexia is a medical emergency in which the blood pressure rises rapidly due to SNS stimulation.

See Chapter 42 for a discussion of autonomic dysreflexia.

The person with a flaccid bladder may require catheterisation to completely empty the bladder. An indwelling catheter may be used initially, but intermittent catheterisation is preferred. Clean intermittent self-catheterisation is performed every 3 to 4 hours to prevent overdistension of the bladder (see Procedure 42.1).

Surgery

Surgery may be required when urination cannot be effectively managed using more conservative measures. *Rhizotomy*, or destruction of the nerve supply to the detrusor muscle or the external sphincter, may be used for those with hyperreflexia or spasticity. Urinary diversion is another surgical technique used when conservative management fails. Implantation of an artificial sphincter may be useful for some people with neurogenic bladder. See Table 26.6 for urinary diversion techniques and above for nursing care of those undergoing a urinary diversion.

MEDICATION ADMINISTRATION The person with a neurogenic bladder

ANTICHOLINERGIC DRUGS TO TREAT SPASTIC BLADDER

Oxybutynin (Ditropan, Ditropan XL)
Tolterodine (Detrol, Detrol LA)
Propantheline bromide (Pro-Banthine)
Flavoxate hydrochloride (Urispas)

Anticholinergic drugs inhibit the response to acetylcholine, relaxing the detrusor muscle and increasing internal sphincter tone. The combination of detrusor relaxation and internal sphincter contraction increases the bladder capacity of those with spastic or hyperreflexive neurogenic bladder. Of these medications, tolterodine has the most specific effects on the detrusor muscle with fewer anticholinergic side effects.

Nursing responsibilities

- Assess for contraindications, such as glaucoma, gastrointestinal or urinary tract obstruction, severe ulcerative colitis or toxic megacolon, unstable cardiovascular status or myasthenia gravis.
- Observe for the desired effect of increased bladder capacity with decreased incontinence and spasm.
- Monitor for possible interaction with other drugs such as narcotic analgesics, antiarrhythmic medications, antihistamines, antidepressants or psychoactive drugs.
- Monitor heart rate and blood pressure, especially when given to people with known cardiovascular disease.
- Assess for adverse effects such as urinary hesitancy or retention, arrhythmias, mental status changes and gastrointestinal disturbances.

Health education for the person and family

- Promptly report eye pain, rapid heartbeat, difficulty breathing, rash or hives, or changes in mental function to your primary care provider.
- These drugs may cause drowsiness or blurred vision. Use caution when driving, operating machinery or performing other tasks requiring mental acuity.
- Boiled sweets help relieve dry mouth associated with these drugs.
- Do not use alcohol or non-prescription antihistamines while taking these drugs.

CHOLINERGIC DRUGS TO STIMULATE MICTURITION

Bethanechol chloride (Urocarb)

- Bethanechol stimulates the parasympathetic nervous system, increasing detrusor muscle tone and producing a contraction strong enough to initiate micturition. It is used primarily to treat acute postoperative and postpartum urinary retention.

Nursing responsibilities

- Assess for contraindications, including hypersensitivity, hyperthyroidism, peptic ulcer disease, asthma, significant bradycardia or hypotension, coronary heart disease, epilepsy and parkinsonism.
- Do not give to individuals who have had recent gastrointestinal or bladder surgery, or those with possible gastrointestinal or urinary tract obstruction.
- Give oral forms on an empty stomach to reduce the risk of nausea and vomiting.
- Administer parenteral bethanechol (Urocarb) subcutaneously. Keep atropine, the antidote for bethanechol overdose or toxicity, available.
- Observe for desired effect within 30 to 60 minutes after oral administration, 5 to 15 minutes after injection.
- Assess for adverse effects such as malaise, headache, abdominal cramping, nausea, hypotension with reflex tachycardia, wheezing and dyspnoea.

Health education for the person and family

- Take the medication 1 hour before or 2 hours after meals.
- Use caution when rising from a recumbent or sitting position; you may feel dizzy or light headed.

Nursing care

Nursing care of the person with a neurogenic bladder is directed towards promoting urinary drainage and continence, preventing complications, and teaching the person and family self-care techniques.

Assessment

Nursing assessment for neurogenic bladder includes obtaining a complete nursing history, focusing on information related to CNS or spinal cord injury or disease, as well as disorders that affect the peripheral nervous system (e.g. diabetes). Ask about measures used to stimulate or control urination. Inspect and palpate the lower abdomen and suprapubic region for tenderness or bladder distension. Percuss the suprapubic region for a dull percussion tone indicative of a full bladder. Dullness up to the level of the umbilicus indicates at least 500 mL of urine in the bladder (Gray, 2000). Assess urine for colour, clarity and odour. Collect a specimen for analysis as indicated.

Nursing diagnoses and interventions

Although each person has individual nursing care needs, examples of nursing diagnoses appropriate for those with a neurogenic bladder include the following:

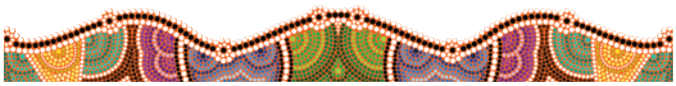
- *Impaired urinary elimination* related to impaired bladder innervation.
- *Self-care deficit: toileting* related to neurological injury.
- *Risk of impaired skin integrity* related to urinary incontinence.
- *Risk of infection* related to impaired urination reflex.

Community-based care

Include the following in teaching for the person with a neurogenic bladder and family members:

- measures to stimulate reflex voiding and promote bladder emptying

- use of prescribed medications, including desired and adverse effects and interactions with other drugs
- manifestations of UTI or urolithiasis and measures to reduce the risk of these complications.



THE PERSON WITH URINARY INCONTINENCE

The most common manifestation of impaired bladder control is **urinary incontinence (UI)**, or involuntary urination. UI can have a significant impact on individuals, leading to physical problems such as skin breakdown, maceration, infection and rashes. Psychosocial consequences include embarrassment, isolation and withdrawal, feelings of worthlessness and helplessness, and depression.

Incidence and prevalence

According to the Continence Foundation of Australia (CFA) (2012), approximately 37% of Australian women and 13% of Australian men are affected by urinary incontinence. An estimated 65% of older women visiting their general practitioner reported UI and in nursing homes the incidence is about 77% (CFA, 2012). The actual prevalence of urinary incontinence is nearly impossible to determine. Embarrassment and the availability of products to protect clothing and prevent detection contribute to people not seeking evaluation of and treatment for incontinence.

Pathophysiology

Urinary continence requires a bladder able to expand and contract, and sphincters that can maintain a urethral pressure higher than that in the bladder. Incontinence results when the pressure within the urinary bladder exceeds urethral resistance, allowing urine to escape. Any condition causing higher than normal bladder pressures or reduced urethral resistance can potentially result in incontinence. Relaxation of the pelvic musculature, disruption of cerebral and nervous system control, and disturbances of the bladder and its musculature are common contributing factors.

Incontinence may be an acute, self-limited disorder or it may be chronic. The causes may be congenital or acquired, reversible or irreversible. Congenital disorders associated with incontinence include *epispadias* (absence of the upper wall of the urethra) and *meningomyelocele* (a neural tube defect in which a portion of the spinal cord and its surrounding meninges protrude through the vertebral column). CNS or spinal cord trauma, stroke and chronic neurological disorders such as multiple sclerosis and Parkinson's disease are examples of acquired, irreversible causes of incontinence. Reversible causes include acute confusion, medications such as diuretics or sedatives, prostatic enlargement, vaginal and urethral atrophy, UTI and faecal impaction.

FAST FACTS

- Urinary incontinence is especially common among older individuals. Although the prevalence of UI increases in older adults, it is *not* a normal consequence of ageing and it can be treated.
- Over 70% of women suffer from UI as a result of childbirth and the menopause.
- The incidence of UI in males increases with age, especially in the 80+ age group.

Incontinence is commonly categorised as stress incontinence, urge incontinence (also known as overactive bladder), overflow incontinence and functional incontinence. Table 26.7 summarises each type with its physiological cause and associated factors. *Mixed incontinence*, with elements of both stress

NURSING CARE OF THE OLDER ADULT Minimising the risk of UTI and UI

Older adults have a higher incidence of two common urinary tract disorders: urinary tract infection and urinary incontinence.

URINARY TRACT INFECTION

Ageing affects the normal protective mechanisms which prevent UTI. The pH of urine increases with ageing, allowing bacteria to grow and multiply more readily. Glucosuria, more common in older adults due to the higher incidence of diabetes, facilitates bacterial growth. Incomplete bladder emptying and urinary retention are more common due to problems such as prostatic hypertrophy in men, bladder prolapse in women and neurogenic bladder in both sexes. Changes in vaginal pH in women post menopause and decreased prostatic secretions in men may also contribute to an increased incidence of UTI.

While many UTIs in older adults are asymptomatic and self-limited, infections can lead to bacteraemia, sepsis and shock. Manifestations of UTI in the elderly include dysuria, urgency, frequency, incontinence, occasional haematuria and confusion. Symptoms such as fever, chills and flank pain and tenderness may be absent. Dementia may make diagnosis more difficult.

URINARY INCONTINENCE

Urinary incontinence, the involuntary loss of urine, is a common problem in older adults. While incontinence should never be considered a *normal* consequence of ageing, age-related changes contribute to its development. Bladder capacity tends to decline with age and involuntary bladder muscle contractions are more common. In women, decreased oestrogen levels and pelvic muscle relaxation decrease bladder outlet and urethral resistance pressures. Decreased oestrogen also causes atrophic vaginitis and urethritis, with manifestations of dysuria and urgency. Other risk factors for UI in older adults include impaired mobility and chronic degenerative diseases, impaired cognition, medications, low fluid intake, diabetes and stroke.

ASSESSING FOR HOME CARE

Assessment for urinary problems in the older adult focuses on risk factors, the extent and manifestations of the disorder, and contributing factors. Using clear language, ask about problems with urine loss, its frequency and any contributing factors. Enquire about frequency, urgency and burning on urination. Identify current medications and the

NURSING CARE OF THE OLDER ADULT **Minimising the risk of UTI and UI (continued)**

time of day each is taken. Assess patterns of fluid intake and output. Assess the abdomen for evidence of bladder distension or tenderness. Perform a mental status examination if indicated.

Assess the home environment (whether in the community or a residential living facility) for possible barriers to urinary elimination:

- inadequate lighting, particularly at night
- narrow doorways that may interfere with access to the toilet
- inadequate toilet facilities
- physical placement of toilet facilities resulting in long walks for disabled or incapacitated individuals
- the need for mobility aids such as safety bars, a raised toilet seat or a bedside commode.

TEACHING FOR HOME CARE

Discuss the following points to help prevent UTI and UI in the older adult:

- Maintain a generous fluid intake. Reduce or eliminate fluid intake after 7 pm in the evening to reduce nocturia.
- Wear comfortable clothing that is easy to remove for toileting.
- Maintain good hygiene, but do not bathe more often than necessary. Frequent bathing and feminine hygiene sprays or douches may dry perineal tissues, increasing the risk of UTI or UI.
- Perform pelvic muscle exercises (Kegel exercises) several times a day to increase perineal muscle tone.
- Reduce consumption of caffeine-containing beverages (coffee, tea, colas), citrus juices and artificially sweetened beverages containing aspartame.
- Use behavioural techniques such as scheduled toileting, habit training and bladder training to reduce the frequency of incontinence. *Scheduled toileting* is toileting at regular intervals (e.g. every 2 to 4 hours). *Habit training* is toileting the person on a schedule that corresponds with the normal pattern. *Bladder training* gradually increases the bladder capacity by increasing the intervals between voidings and resisting the urge to void.
- See your primary care provider regularly for a pelvic or prostate examination to assess for physical abnormalities.
- For women, discuss possible benefits and risks of hormone replacement therapy, physical therapy, incontinence aids or surgery to treat incontinence.
- Report a change in urine colour, odour or clarity, or symptoms such as burning, frequency or urgency, to your primary care provider.

RESOURCES

Continence Foundation of Australia (National Office)
AMA House, 293 Royal Parade
Parkville Vic 3052
Telephone: 03 9347 2522
Fax: 03 9347 2533
E-mail: info@incontinence.or.au
National Continence Helpline: 1800 330 066
Website: www.continence.org.au
(State offices' contact details can be obtained from the national office.)
The organisation provides a wide range of services, from professional advice to referrals to continence clinics and education resources.

Community health services

Blue Care Services
Telephone: 07 3377 3377
Website: www.bluecare.org.au
Provides care to all members of the community.

Continence Advisory Service
Provides confidential and professional advice on bladder and bowel problems.
Telephone: 08 9386 97777
E-mail: info@continencewa.org.au

Continence Management Advice Services
Telephone: 1300 787 055
Website: www.silverchain.org.au
Gives professional advice and may be able to provide access to products at a reduced cost.

and urge incontinence, is common. *Total incontinence* is loss of all voluntary control over urination, with urine loss occurring without stimulus and in all positions.

Incontinence is associated with an increased risk of falls, fractures, pressure ulcers, urinary tract infection and depression. It contributes to the stress of caregivers and may be a factor in institutionalising the affected person.

INTERPROFESSIONAL CARE

Urinary incontinence management is directed at identifying and correcting the cause if possible. If the underlying disorder cannot be corrected, techniques to manage urine output can often be taught.

Evaluation for incontinence begins with a complete history, including the duration, frequency, volume and associated circumstances of urine loss. A voiding diary (see Figure 26.8) is often used to collect detailed information. The history also includes information about chronic or acute illnesses, previous surgeries and current medication use, both prescription and over the counter.

Physical assessment includes abdominal, rectal and pelvic assessment, as well as evaluation of mental and neurological status, mobility and dexterity. Findings often associated with incontinence in women include weak abdominal and pelvic muscle tone, cystocele or urethrocele, and atrophic vaginitis. In men, an enlarged prostate gland is the physical finding most commonly associated with incontinence.

See the 'Translation to practice' feature below on evidence-based practice for diagnosing urge incontinence using specific personal assessment data.

Diagnosis

- *Urinalysis* and *urine culture* using a clean-catch specimen are done to rule out infection and other acute causes of incontinence.

TABLE 26.7 Types of urinary incontinence

	DESCRIPTION	PATHOPHYSIOLOGY	CONTRIBUTING FACTORS
Stress	Loss of urine associated with increased intra-abdominal pressure during sneezing, coughing, lifting. Quantity of urine lost is usually small	Relaxation of pelvic musculature and weakness of urethra and surrounding muscles and tissues leads to decreased urethral resistance	• Multiple pregnancies • Decreased oestrogen levels • Short urethra, change in angle between bladder and urethra • Abdominal wall weakness • Prostate surgery • Increased intra-abdominal pressure due to tumour, ascites, obesity
Urge	Involuntary loss of urine associated with a strong urge to void	Hypertonic or overactive detrusor muscle leads to increased pressure within bladder and inability to inhibit voiding	• Neurological disorders such as stroke, Parkinson's disease, multiple sclerosis; peripheral nervous system disorders • Detrusor muscle overactivity associated with bladder outlet obstruction, ageing or disorders such as diabetes
Overflow	Inability to empty bladder, resulting in overdistension and frequent loss of small amounts of urine	Outlet obstruction or lack of normal detrusor activity leads to overfilling of bladder and increased pressure	• Spinal cord injuries below S_2 • Diabetic neuropathy • Prostatic hypertrophy • Faecal impaction • Drugs, especially those with anticholinergic effect
Functional	Incontinence resulting from physical, environmental or psychosocial causes	Ability to respond to the need to urinate is impaired	• Confusion or dementia • Physical disability or impaired mobility • Therapy or sedation • Depression • Regression

- *Post-voiding residual (PVR) volume* is measured to determine how completely the bladder empties with voiding. Less than 50 mL PVR is expected; when 100 mL or more is obtained, further testing is indicated.
- *Cystometrography* is used to assess neuromuscular function of the bladder by evaluating detrusor muscle function, pressure within the bladder and the filling pattern of the bladder. The person describes sensations and any urge to void as sterile water or saline is instilled into the bladder. Normally, the urge to void is perceived at 150 to 450 mL and the bladder feels full at 300 to 500 mL. Bladder pressure and volume are recorded on a graph. When the bladder is full, the individual voids and intravesical pressure is noted during voiding.
- *Uroflowmetry* is a non-invasive test used to evaluate voiding patterns. The uroflowmeter, contained in a funnel, measures the rate of urine flow, the continuous flow time and the total voiding time.
- *IVP* may be ordered to evaluate structure and function of the upper and lower urinary tract.
- *Cystoscopy* or *ultrasonography* may be ordered to identify structural disorders contributing to incontinence, such as an enlarged prostate or a tumour.

Nursing implications for the specialised studies for urinary incontinence are outlined in Chapter 25.

Medications

Both stress and urge incontinence may improve with drug treatment.

When incontinence is associated with postmenopausal atrophic vaginitis, oestrogen therapy may be effective. Both systemic oestrogens and local creams are used.

People with urge incontinence may be treated with preparations that increase bladder capacity. The primary drugs used to inhibit detrusor muscle contractions and increase bladder capacity include oxybutynin (Ditropan and Ditropan XL), an anticholinergic drug, and tolterodine (Detrol and its longer-acting form, Detrol LA), a more specific antimuscarinic agent. These drugs can be taken once or twice a day and have fewer side effects than less specific anticholinergic drugs. Drugs with anticholinergic effects are contraindicated for the person with acute glaucoma. Urinary retention is a potential side effect that must be considered when these drugs are used (see the 'Medication administration' box above).

Surgery

Surgery may be used to treat stress incontinence associated with cystocele or urethrocele and overflow incontinence associated with an enlarged prostate gland.

Suspension of the bladder neck, a technique that brings the angle between the bladder and urethra closer to normal, is effective in treating stress incontinence associated with urethrocele in 80–95% of individuals. A laparoscopic, vaginal or abdominal approach may be used to perform this surgery. Care of the person with a bladder neck suspension is outlined below.

Prostatectomy, using either the transurethral or suprapubic approach, is indicated for the person who is experiencing overflow incontinence as a result of an enlarged prostate gland and urethral obstruction. Care of the person with a prostatectomy is outlined in Chapter 47.

Other surgical procedures of potential benefit in the treatment of incontinence include implantation of an artificial sphincter, formation of a urethral sling to elevate and compress

Your Daily Voiding Diary **Date** _____

This diary will help you and your healthcare team identify factors causing bladder control problems. Choose a 24-hour period when you can record your fluid intake (type and amount), urine output and episodes of urine leakage, any strong urge to void just prior to leaking, and your activity when leak episodes occur. The line below illustrates how to use your diary.

Time	Fluid Intake		Urine Output	Leaks	Urge	Activity
	Amount	Type			Yes No	
7 am	2 cups	coffee	sm (med) lg	(sm) med lg	Yes	walking
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		
			sm med lg	sm med lg		

I used ___ pads today. I used ___ nappies today.

Questions to ask my healthcare team: _____

_____

_____

FIGURE 26.8 ■ A sample voiding diary

Source: Adapted from *Your daily bladder diary*, National Kidney and Urologic Diseases Information Center, National Institute of Diabetes and Digestive and Kidney Disease (NIDDK), National Institutes of Health.

the urethra, and augmentation of the bladder with bowel segments to increase bladder capacity.

Complementary therapies

Biofeedback and relaxation techniques may help reduce episodes of urinary incontinence. Biofeedback uses electronic monitors to teach conscious control over physiological responses of which the individual is not normally aware. Developing awareness of perceptible information allows the person to gain voluntary control over urination. Biofeedback is widely used to manage urinary incontinence.

Nursing care

Health promotion

Although urinary incontinence rarely causes serious physical effects, it frequently has significant psychosocial effects and can lead to lowered self-esteem, social isolation and even institutionalisation (Lauver et al., 2004). Get the word out—inform all people that UI is not a normal consequence of ageing and that treatments are available. To reduce the incidence of UI, teach all women to perform pelvic floor muscle (Kegel) exercises (see Box 26.3) to improve perineal muscle tone. Advise women to seek advice from their women's healthcare or primary care practitioner about using topical or systemic hormone therapy during menopause to maintain perineal tissue integrity. Advise older men to have routine prostate examinations to prevent urethral obstruction and overflow incontinence. Pelvic floor muscle exercises also may benefit men who experience UI following prostatectomy, but evidence supporting this is limited (Moore & Gray, 2004).

Assessment

Nursing assessment for the person with urinary incontinence includes both subjective and objective data:

- *Health history:* voiding diary; frequency of incontinent episodes, amount of urine loss and activities associated with incontinence; methods used to deal with incontinence; use of Kegel exercises or medications; any chronic

TRANSLATION TO PRACTICE Evidence-based practice: urinary incontinence

While an accurate diagnosis of stress urinary incontinence often is made based on clinical data, motor urge incontinence has been more difficult to diagnose accurately without urodynamic investigations. This presents difficulty for nurses and nurse practitioners planning care for incontinent individuals when urological testing is not feasible or readily available. A model developed by Gray et al. (2001) may be useful to address this problem in cognitively intact adults. By comparing personal data with urodynamic testing results, this team of researchers identified factors predictive of motor urge incontinence. These factors included age, gender and three key symptoms: diurnal frequency (urinating more often than every 2 hours while awake), nocturia (awakening with urge to urinate more than once per night if under age 65, twice per night if over age 65) and urge incontinence (urine loss associated with a strong desire to urinate). The presence of all three symptoms was more than 92% predictive of motor urge incontinence in study participants of all ages (range 18 to 89; median 61) and both genders.

IMPLICATIONS FOR NURSING

Asking specific questions about urinary tract symptoms can facilitate accurate identification of the nursing diagnosis *Urinary incontinence: urge*. Accurate diagnosis is vital to planning and implementing appropriate care measures and achieving the desired outcome of continence. Successful treatment promotes self-esteem and provides positive reinforcement for continuing planned strategies.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. What nursing care measures and person-centred teaching will you provide for the person with stress incontinence that may not be appropriate or necessary for the person with urge incontinence but not stress incontinence?
2. Identify circumstances in which it may not be possible or feasible to have the person undergo urodynamic testing to differentiate stress, urge or mixed (stress and urge) incontinence.
3. The individuals in this study lived independently in the community and were cognitively intact. Can the data in this study be generalised to people residing in a long-term care facility? Can the results be applied to all types of incontinence? Why or why not?

Source: A model for predicting motor urge urinary incontinence by Gray et al., (2001). *Nursing Research*, *50*(2), 116–122.

BOX 26.3 Pelvic floor muscle (Kegel) exercises

Particularly important for postpartum, menopausal or post-vaginal hysterectomy women.

- Identify the pelvic muscles with these techniques:
  a. Stop the flow of urine during voiding and hold for a few seconds.
  b. Tighten the muscles at the vaginal entrance around a gloved finger or tampon.
  c. Tighten the muscles around the anus as though resisting defecation.
- Perform exercises by tightening pelvic muscles, holding for 10 seconds and relaxing for 10 to 15 seconds. Continue the sequence (tighten, hold, relax) for 10 repetitions.
- Keep abdominal muscles and breathing relaxed while performing exercises.
- Initially, exercises should be performed twice per day, working up to four times a day.
- Encourage exercising at a specific time each day or in conjunction with another daily activity (such as bathing or watching the news). Establish a routine because these exercises should be continued for life.

diseases, related surgeries; effects of incontinence on usual activities, including social activities.

- *Physical examination:* physical and mental status, including any physical limitations or impaired cognition; inspect, palpate and percuss abdomen for bladder distension; inspect perineal tissues for redness, irritation or tissue breakdown; observe for bulging of bladder into vagina when bearing down; assess pelvic muscle tone as indicated.

Nursing diagnoses and interventions

In planning nursing care, consider the person's mental status, mobility and motivation. Behavioural techniques can be effective, but require long-term commitment and the physical and mental capability to use them.

Nursing care and modification of routines can restore continence fully or partially even in the institutionalised person. Scheduled toileting, bladder training and prompted voiding, combined with positive reinforcement such as praise, can reduce the need for nappies, incontinence pads and indwelling catheters.

See the nursing care plan that follows for additional nursing diagnoses and interventions for the person with urinary incontinence.

Urinary incontinence: stress and/or urge

Exercises to strengthen pelvic floor muscles, dietary modifications and bladder training programs often are effective to restore and maintain continence.

- Instruct to keep a voiding diary, recording the time and amount of all fluid intake and urinary output, status at the time of voiding (dry or wet) and on arising from sleep and

NURSING CARE OF THE PERSON having a bladder neck suspension

PREOPERATIVE CARE

- Provide routine preoperative care and teaching as outlined in Chapter 3.
- Discuss the need to avoid straining and the Valsalva manoeuvre postoperatively. Suggest measures such as increasing fluid and fibre intake and using a stool softener to prevent postoperative constipation. *Straining and increased abdominal pressure during the Valsalva manoeuvre may place excessive stress on suture lines and interfere with healing.*

POSTOPERATIVE CARE

- Provide routine postoperative care as outlined in Chapter 3.
- Monitor urine output, including quantity, colour and clarity. Expect urine to be pink initially, gradually clearing. *Bright red urine, excessive vaginal drainage or incisional bleeding may indicate haemorrhage. Instrumentation of the urinary tract increases the potential for UTI; cloudy urine may be an early sign.*
- Maintain stability and patency of suprapubic and/or urethral catheters. Secure catheters in position. *Maintaining bladder decompression eliminates pressure on suture lines. Preventing movement or pulling of catheters reduces the risk of resultant pressure on surgical incisions.*
- Carefully monitor urine output after catheter removal. *Difficulty voiding is common following catheter removal. Early intervention to prevent bladder distension is important to prevent pressure on suture lines.*
- If the urethral or suprapubic catheter will remain in place on discharge, teach proper care to the person and family members as needed. *Appropriate self-care and early recognition of problems reduce the risk of significant complications.*

activities. *Voiding diaries provide valuable information for identifying the type of incontinence and possible measures to reduce or eliminate incontinent episodes.*

- Teach pelvic floor muscle exercises (see Box 26.3). Instruct to consciously tighten pelvic muscles when the need to void is perceived and to relax the abdomen while walking to the bathroom. *Improved pelvic muscle strength helps retain urine and prevent stress incontinence by increasing urethral pressure. Exercises also decrease abnormal detrusor muscle contractions, decreasing pressure within the bladder.*

CONSIDERATION FOR PRACTICE

Do not advise people who have difficulty emptying the bladder completely to stop urine flow while voiding to identify pelvic floor muscles. Repeated interruption of micturition can interfere with complete bladder emptying and increase the risk of UTI.

- Using the person's voiding diary, suggest dietary and fluid intake modifications to reduce stress and urge incontinence. Include limiting caffeine, alcohol, citrus juice and artificial sweetener consumption; limiting fluid intake to no less than 1.5 to 2.0 L per day; and limiting evening fluid intake. *Caffeine, alcohol and citrus juices are bladder irritants and tend to promote detrusor instability, increasing the risk of urge incontinence. Artificial sweeteners may also irritate the bladder. Fluid intake of 1.5 to 2.0 L per day is adequate to maintain health for most people; excess fluid may increase stress incontinence if bathroom facilities are not readily available.*

CONSIDERATION FOR PRACTICE

Limiting total fluid intake to less than 1.5 to 2.0 L per day is not recommended for those with urinary incontinence. Inadequate fluid increases urine concentration, leading to bladder wall irritation and possibly increasing problems of urge incontinence.

Self-care deficit: toileting

Functional incontinence may be the predominant problem in an institutionalised older adult. Limited mobility, impaired vision, dementia, lack of access to facilities and privacy, and tight staffing patterns increase the risk of incontinence in previously continent residents. The primary problem in functional incontinence is an outside factor that interferes with the ability to respond normally to the urge to void. An immobilised person may wet the bed if a call light is not within reach; a person with Alzheimer's disease may perceive the urge to void but be unable to interpret its meaning or respond by seeking a bathroom. For these people, self-care deficit in toileting is a primary problem.

- Assess physical and mental abilities and limitations, usual voiding pattern and ability to assist with toileting. *A thorough assessment allows planned interventions to address specific needs and promote independence.*
- Provide assistive devices as needed to facilitate independence, such as raised toilet seats, grab bars, a bedside commode or night-lights. *Fostering independence in toileting bolsters self-concept and maintains a positive body image.*
- Plan a toileting schedule based on the person's normal elimination patterns to achieve approximately 300 mL of urine output with each voiding. *Allowing the bladder to fill to a point at which the urge to void is experienced and then emptying it completely helps maintain normal bladder capacity and bacteriostatic functions.*
- Position for ease of voiding—sitting for females, standing for males—and provide privacy. *Normal positioning, usual toileting facilities and privacy enhance the ability to void on schedule and empty the bladder completely.*
- Adjust fluid intake so that the majority of fluids are consumed during times of the day when the person is most able to remain continent. Unless fluids are to be restricted, maintain a fluid intake of at least 1.5 to 2.0 L per day. *An adequate fluid intake is vital to promote hydration and*

urinary function. Overly concentrated urine can irritate the bladder, increasing incontinence.

- Assist with clothing that is easily removed (e.g. elastic-waist pants or loose dresses). Velcro and zipper fasteners may be easier to use than snaps and buttons. *Clothing that is difficult to remove can increase the risk of incontinence for those with mobility problems or impaired dexterity.*

Social isolation

Urinary incontinence increases the risk of social isolation due to embarrassment, fear of not having ready access to a bathroom, body odour or other factors. Social isolation can, in turn, increase problems of incontinence because normal cues and relationships are lost and the need to remain dry is less strongly felt.

- Assess reasons for and extent of social isolation. Verify the degree of social isolation with the person or significant other. Do not assume that social isolation is only related to urinary incontinence. *Other problems frequently associated with ageing (such as a hearing deficit) may be primary or contributing factors.*
- Refer the person for urological examination and incontinence evaluation. *People who assume that urinary incontinence is a normal part of the ageing process may not be aware of treatment options.*
- Explore alternative coping strategies with the person, significant other, staff and other healthcare team members. *Protective pads or shields, good perineal hygiene, scheduled voiding and clothing that does not interfere with toileting can enhance continence.*

Community-based care

Because urinary incontinence is a contributing factor in the institutionalisation of many older people, person-centred teaching and family teaching can have a significant impact on maintaining independence and residence in the community. Address possible causes of incontinence and appropriate treatment measures. Refer for urological examination if not already completed. Discuss fluid intake management, perineal care and products for clothing protection.

NURSING CARE PLAN A person with urinary incontinence

Anna Giovanni, a 76-year-old retired teacher, has been widowed for 10 years and lives alone. Mrs Giovanni's eldest daughter expresses concern that her mother seems increasingly reluctant to leave her apartment to visit friends and family. She reports a strong odour of urine throughout her mother's apartment and that her mother's bed is often wet. She is worried about needing to place her mother in a nursing home if she cannot continue to live independently.

ASSESSMENT

The community nurse visits Mrs Giovanni to assess her situation. Mrs Giovanni admits that she has problems with urine leakage when laughing and coughing and has a strong urge to void on hearing the sound of running water. At night, her urge to void is so strong that she often cannot reach the bathroom in time. Mrs Giovanni does not have a history of UTIs, neurological disorders or difficulty with her bowels. She had a hysterectomy at age 52 and was on hormone replacement therapy for about 10 years afterwards. She is taking digoxin 0.125 mg daily, frusemide 40 mg twice daily and potassium chloride 20 mEq three times daily for mild heart failure.

Physical assessment reveals a moderate cystourethrocele and atrophy of vaginal and vulvar tissues. Moderate perineal dermatitis is also noted and her pelvic floor strength is weak. Urinalysis is within normal limits and postvoiding residual urine is 5 mL.

Analysis of Mrs Giovanni's voiding diary shows moderate consumption of tea and juices throughout the day, nine day-time voidings and four night voidings with an average volume of about 250 mL per void. She notices urine leakage most often in the late afternoon and at night. The community nurse identifies stress incontinence with an urgency component and decides to try a conservative approach before referring Mrs Giovanni for further testing and possible doctor referral for cystourethrocele repair. Oestrogen cream, tolterodine (Detrol) and a barrier cream to treat Mrs Giovanni's vulvitis are prescribed.

DIAGNOSES

- *Stress urinary incontinence* related to weak pelvic floor musculature and tissue atrophy.
- *Urge urinary incontinence* related to excess intake of caffeine and citrus juices.
- *Impaired skin integrity* related to constant contact of urine with perineal tissues.
- *Ineffective coping* related to inability to control urine leakage.

PLANNING

- Plan interventions to facilitate a return of normal bladder function.
- Plan interventions that foster a resumption of previous levels of social activity.

Expected outcomes

- Remain dry between voidings and at night.
- Demonstrate improved perineal muscle strength.
- Regain and maintain perineal skin integrity.
- Return to her previous level of social activity.

IMPLEMENTATION

- Teach how to identify pelvic floor muscles and how to perform Kegel exercises.
- Suggest drinking decaffeinated tea and non-citrus fruit juices (grape, apple and cranberry).
- Encourage to minimise fluid intake after evening meal.

NURSING CARE PLAN A person with urinary incontinence (continued)

- Change evening dose of frusemide from 9 pm to 3 pm.
- Instruct to void by the clock, gradually increasing intervals from every 45 to 60 minutes to every 2 to 2.5 hours. Advise to maintain shorter voiding intervals for 2 to 3 hours after frusemide doses.
- Teach to how to cleanse perineal area, wiping front to back after each voiding or incident of urine leakage.
- Introduce commercial products available for clothing and furniture protection, encouraging experimentation to identify the most helpful product(s).
- Provide a commode for bedside at night and adequate lighting to prevent injury.
- Schedule follow-up visits and evaluations to reinforce teaching.

EVALUATION

Three months after her initial visit, Mrs Giovanni states that she is doing very well, experiencing only occasional leakage of small amounts of urine, primarily when sneezing, coughing or laughing. She finds a minipad adequate for protection and is often able to remain dry all day. She has had no further problems with enuresis since changing her evening frusemide dose to late afternoon and limiting her fluids after dinner. She can make it to the bathroom and no longer needs the bedside commode. Her perineal tissue is intact and she demonstrates improved muscle strength. Anna's daughter says her mother is beginning to resume her normal social activities and that she is no longer worried about her mother's ability to care for herself independently.

CRITICAL THINKING IN THE NURSING PROCESS

1 What factors in Mrs Giovanni's medical history and current medication regimen contributed to her nighttime incontinence?
2 What is the rationale for including an intervention to teach Mrs Giovanni about perineal cleansing as part of her care plan?

REFLECTION ON THE NURSING PROCESS

1 Outline what you have learned from Mrs Giovanni's situation that you could apply to your future practice.
2 What educational strategies would you give Mrs Giovanni's family to prevent her developing social isolation related to the urinary incontinence?

CHAPTER HIGHLIGHTS

- Urinary tract infections are very common and are a leading complication among hospitalised individuals. Short-course antibiotic therapy is appropriate for uncomplicated infections of the lower urinary tract that are not associated with the presence of an indwelling urinary catheter.
- Teach people about perineal hygiene and the importance of maintaining adequate fluid intake as measures to help prevent UTI.
- Urinary calculi can obstruct the urinary tract at any level and cause significant pain as they move from the kidney through the ureter. Instruct those who have had a renal stone to maintain a generous fluid intake particularly during exercise and warm weather, to reduce the risk of further stone formation.
- The risk of bladder cancer is greater among men than women and cigarette smoking is the most significant risk factor for bladder cancer. Most tumours can be resected transurethrally if diagnosed early, before spreading to deeper layers of the bladder wall, the lymph nodes and adjacent tissue.
- When resection of the urinary bladder is necessary, a urinary diversion is created to collect urine. A collection appliance must be worn constantly on an ileal conduit; when a continent urinary diversion is created, the pouch is emptied by intermittent catheterisation of the stoma.
- Urinary retention may occur as a result of some medications, neurological damage or disease, or obstruction (e.g. an enlarged prostate gland). If the underlying condition cannot be treated, medications or intermittent catheterisation are used to promote bladder emptying.
- Older adults, in particular, are at risk of urinary incontinence, a treatable condition. A health history, voiding diary and diagnostic testing are used to establish the type of urinary incontinence and direct treatments such as surgery, pelvic floor muscle exercises, medications and scheduled toileting.

CONCEPT CHECK

1 A 30-year-old male presents to the urgency clinic complaining of concentrated bright yellow urine and of discomfort in his lower back area. The assessment reveals no significant nursing history. Additional questions the nurse should ask include:
   1 'Have you had any injury to your abdominal area?'
   2 'When did you first notice your urine's colour?'
   3 'How often do you void?'
   4 'How much fluid do you drink each day?'

2 The man volunteers the information that he has just started cycling, has been following a strict high-level exercise program and has a high-protein diet. His fluid intake is about 1 L per day. The nurse should then ask:
   1 'Do you take any prescribed medications?'
   2 'How much exercise do you have each day?'
   3 'How long have you been on this exercise program?'
   4 'Do you take any additional supplements, e.g. vitamins?' (Multi-vitamins will colour urine orange.)

3 After an assessment and a nursing history nothing of significance is noted except mild tenderness over the lower back area. The man admits he has been taking multivitamins because he wanted to improve his physical performance. The nurse recommends the man:
   1 reduces the amount of exercise he does on a daily basis
   2 applies a warm pack to his lower back when he rests
   3 stops taking the multivitamin medication until his urine returns to a normal colour
   4 increases his fluid intake to at least 3 L per day

4 A 58-year-old woman presents at her doctor's with symptoms of frequency, urgency, nocturia, dysuria and cloudy, rust-coloured urine for the third time in the past 2 years. The nurse should plan to include which of the following in her teaching for this woman? (Select all that apply.)
1 return to the office in 10 days for follow-up culture
2 preprocedure instruction for an IVP
3 the potential benefits of oestrogen vaginal cream
4 recommendations for perineal cleansing
5 recommendations for screening cystoscopy

5 Recognising the risk of urolithiasis in the immobilised person, the nurse appropriately plans to:
1 administer a calcium supplement
2 regularly monitor urine pH
3 maintain an indwelling urinary catheter
4 increase fluid intake to 3000 mL per day

6 A person admitted with possible kidney stones develops sudden complaints of acute crampy pain on the left side that radiates into the groin. He is nauseated and vomits clear fluid. On voiding, his urine is pink. The nurse should:
1 obtain a bladder scan to assess for residual urine
2 administer the prescribed narcotic analgesic
3 notify the doctor
4 strain all urine

7 The nurse teaching a group of community members about wellness and disease prevention includes which of the following as a measure to reduce the risk of bladder cancer?
1 Do not start smoking. If you smoke, stop.
2 Avoid using hair dyes and pesticides in the home.
3 Limit your intake of coffee and other caffeinated beverages.
4 Empty your bladder every 2 hours.

8 A person remarks to the nurse that his urine occasionally appears pink. He wonders if this is anything to be concerned about. The nurse should:
1 instruct the man to notify his doctor if he develops pain or difficulty voiding
2 advise the man to make an appointment to see his doctor
3 instruct the man to record if the urine is pink following exercise
4 tell the man to increase his fluid intake to 4 L per day

9 The nurse caring for a person in the spinal shock phase following spinal cord injury appropriately plans to:
1 insert a Foley catheter to accurately measure output
2 stimulate voiding using Credé's method
3 assess for urinary retention following each voiding
4 catheterise with a straight catheter every 3 to 4 hours

10 The nurse implements a nursing care necessary for this individual with a Foley catheter in place to:
1 maintain the closed system of drainage tubing and collection bag to prevent ascending infection
2 irrigate the person with 1% neosporin solution three times a daily
3 clamp the catheter for 1 hour every 4 hours to maintain the bladder's elasticity
4 maintain the drainage tubing and collection bag below bladder level to facilitate drainage by gravity

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CHAPTER 27

NURSING CARE OF PEOPLE WITH KIDNEY DISORDERS

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LEARNING OUTCOMES

- Discuss the course, implications and health management options for a person with acute kidney injury.
- Relate the pathophysiology, clinical manifestations and possible interventions required for a person with chronic kidney disease to the pathophysiology of the condition.
- Discuss the risk factors, implications and health management and treatment options for a person who has end-stage kidney disease.

CLINICAL COMPETENCIES

- Assess the functional health status of people with kidney disease.
- Monitor, document and report unexpected or abnormal manifestations in people with kidney disease.
- Provide appropriate and effective nursing care for people undergoing dialysis, surgery involving the kidneys or kidney transplant.
- Based on assessment data, determine priority nursing diagnoses and interventions for people with kidney disease.
- Plan and implement evidence-based nursing care for people with kidney disease using research and best practices.
- Collaborate with the person and other members of the interprofessional team to prioritise and implement care.
- Provide teaching appropriate to both people with kidney disease and their personal circumstances.
- Evaluate responses to care, revising the plan of care as needed to promote, maintain or restore functional health status for people with kidney disease.

KEY TERMS

The internal environment of the body normally remains in a relatively constant or *homeostatic* state. One of the easiest ways to reflect on the nursing roles necessary when looking after a person with renal insufficiency is to recall the functions of the kidney (see Table 27.1) and then consider how we support the loss of those functions.

RENAL INSUFFICIENCY: TOWARDS A NEW UNDERSTANDING

Renal insufficiency is a broad term that describes any condition in which the kidneys are unable to remove accumulated metabolites from the blood, leading to altered fluid, electrolyte and acid–base balance. The cause may be an acute insult or a primary kidney disorder, or may be secondary to a systemic disease or urological defects.

Before we continue to explore the many facets of renal insufficiency it is important to understand the disease trajectory of any kidney-related disorder and the meanings of the terms used to describe them. One point to note when reviewing the renal literature is that there has been a change in the past few years as to how we describe renal insufficiency. You may note that some literature uses the term 'chronic renal failure' or 'acute renal failure' whereas more modern texts use the terms 'chronic kidney disease' and 'acute kidney injury or dysfunction', respectively. These changes in terminology reflect the changes in our understanding of both the illness trajectory and the pathophysiology of renal insufficiency, and that in fact, more often than not, there is a disease process occurring and not merely 'renal failure'.

The sub-types of renal insufficiency are described as either acute, chronic or end-stage. **Acute kidney injury (AKI)** is characterised by a rapid onset of symptoms that are potentially reversible with prompt intervention that addresses the initial cause of the injury. Chronic kidney disease, by contrast, is a largely silent disease that presents as an insidious irreversible decline in renal function. A more detailed definition of chronic kidney disease (CKD) will be discussed later in this chapter. People with CKD are also at high risk of acute episodes, which is referred to as 'acute on chronic kidney disease'. As the phrase suggests, an acute injury is superimposed on a background of CKD. For example, a person with a history of CKD may become dehydrated and experience a rapid decline in renal function which may be reversible with adequate rehydration.

TABLE 27.1 Functions of the kidney

EXCRETORY	REGULATORY	HORMONAL/ METABOLIC
Removal of metabolic waste products	Maintain fluid and electrolyte balance Maintain acid–base balance Regulate blood pressure	Activation of vitamin D Erythropoietin production Renin production

There is often some confusion in the literature when the terms 'chronic kidney disease' and 'end-stage kidney disease' are used interchangeably. This use is incorrect, as the final stage of chronic kidney disease, when the glomerular filtration rate falls below 15 mL/min, sees its description change to 'end-stage kidney disease' to denote the terminal phase of the disease trajectory. It is at this stage that the person must choose to undergo a kidney replacement therapy or to withdraw from active treatment.

FAST FACTS

- Acute kidney injury has a sudden onset and is often reversible with prompt treatment.
- Chronic kidney disease is a slowly progressive and often silent disease that is irreversible.
- End-stage kidney disease is the final stage of the CKD continuum.

AGE-RELATED CHANGES IN KIDNEY FUNCTION

Structural and functional changes occur in the ageing kidney. Structurally, the number of nephrons decreases. Glomeruli in the renal cortex (see Chapter 25 for a review of normal kidney structure and function) are lost with ageing, reducing kidney mass. Because of the large functional reserve of the kidneys, however, renal function remains adequate unless additional stressors affect the renal system. Any additional stressors such as hypotension, exposure to nephrotoxic drugs or an inflammatory process such as glomerulonephritis may precipitate an acute episode in the older adult. The **glomerular filtration rate (GFR)**, the amount of filtrate made by the kidneys per minute, declines due to age-related factors affecting the renovascular system (such as arteriosclerosis, decreased renal vascularity and decreased cardiac output). By age 80, the GFR may be less than half of what it was at age 30. Serum creatinine levels may rise slowly. Because older adults have less muscle mass, they produce less creatinine. Likewise, the urea may remain within normal limits.

Age-related changes in renal function have significant implications. The kidneys are less able to concentrate urine and compensate for increased or decreased salt intake. When combined with the diminished effectiveness of antidiuretic hormone (ADH) and a reduced thirst response, both common in ageing, this decreased ability to concentrate urine increases the risk of dehydration. Potassium excretion may be decreased because of lower aldosterone levels. As a result, fluid and electrolyte imbalances are more common and potentially critical in older people.

Decreased GFR in the older adult also reduces the clearance of drugs excreted through the kidneys. This reduced clearance prolongs the half life of drugs and may necessitate lower drug doses and longer dosing intervals. Common medications affected by decreased GFR include:

- cardiac drugs: digoxin
- antibiotics: aminoglycosides, tetracyclines, cephalosporins
- histamine H_2 antagonists: cimetidine
- hypoglycaemic agents: metformin.

TABLE 27.2 Nursing implications of age-related changes in kidney function

FUNCTIONAL CHANGE	EFFECT	IMPLICATIONS
Decreased GFR	Decreased clearance of drugs excreted primarily through the kidneys increases drug half life and blood levels and risk of drug toxicity.	Monitor carefully for signs of toxicity, especially when administering contrast media, amphotericin B, digoxin, aminoglycoside antibiotics, tetracyclines, vancomycin, cimetidine and cephalosporin antibiotics.
Decreased number of functional nephrons; lower levels of aldosterone; increased resistance to ADH	Decreased ability to conserve water and sodium; impaired potassium excretion; and decreased hydrogen ion excretion, resulting in reduced ability to compensate for acidosis.	Monitor for dehydration and hyponatraemia; maintain fluid intake of 1500 to 2500 mL/day unless contraindicated; monitor for hyperkalaemia, especially if taking a potassium-sparing diuretic, heparin, ACE inhibitor, beta-blocker or NSAID; increased risk of acidosis.
Reduced numbers of functional nephrons	Decreased renal reserve with increased risk of acute injury.	Avoid giving nephrotoxic drugs if possible; monitor urine output and blood chemistries for early signs of renal failure.

When caring for older adults or any person at risk of chronic kidney disease, it is especially important to monitor drugs that are toxic to the renal tubules. Age-related changes in renal function and related nursing implications are summarised in Table 27.2.

THE PERSON WITH ACUTE KIDNEY INJURY

The use of the term 'AKI' suggests some form of structural injury to the renal parenchyma, yet classifications of AKI such as RIFLE (Risk, Injury, Failure, Loss, End-stage kidney disease) and the Acute Kidney Injury Network (AKIN) criteria (see Table 27.3) define AKI in terms of functional decreases. In basic terms, AKI manifests as a rapid decline in renal function with uraemic symptoms and fluid and electrolyte imbalances. The deterioration in renal function sees an increase in serum creatinine levels. However, what is important in terms of outcomes for people experiencing AKI is the context and environment in which the insult to the kidney occurs as this is directly associated with mortality and morbidity.

AKI is a broad clinical syndrome, which includes both direct injury to the kidney and the acute impairment of function. Even an increase in serum creatinine of only 26.5 µmol/L (0.3 mg/dL) in a 48-hour period is associated with higher mortality rates (Kellum & Lameire, 2013).

Incidence and risk factors

Approximately 5–7% of all people hospitalised develop AKI; the incidence jumps to as high as 30% in critical care units, with an associated mortality rate as high as 60% (Fauci et al., 2008). The reason these rates are so high has previously been attributed to the populations affected by AKI (i.e. older people and the critically ill), but recent evidence suggests that AKI itself may have an independent and deleterious effect on morbidity and mortality (Obermüller et al., 2014). However, a longitudinal Australian study has reported that although the incidence of AKI is increasing, mortality rates are steadily declining (Bagshaw, George & Bellomo, 2007).

Major trauma or surgery, infection, haemorrhage, severe heart failure, severe liver disease and lower urinary tract obstruction are risk factors for AKI. Drugs and radiological contrast media that are toxic to the kidney (*nephrotoxic*) also increase the risk of AKI. Older adults develop AKI more frequently due to a higher incidence of serious illness, hypotension, major surgeries, diagnostic procedures and treatment with nephrotoxic drugs. The older adult also may have some degree

TABLE 27.3 Comparison of AKI staging by RIFLE and AKIN systems

RIFLE STAGES[a]	RIFLE SERUM CREATININE INCREASE[b]	RIFLE AND AKIN URINE OUTPUT CRITERIA[c]	AKIN SERUM CREATININE INCREASE[b]	AKIN STAGES
Risk	≥ 150–200%	< 0.5 mL/kg per h for > 6 h	≥ 0.3 mg/dL or ≥ 150%	1
Injury	> 200–300%	< 0.5 mL/kg per h for >12 h	> 200–300%	2
Failure	> 300%	< 0.3 mL/kg per h for ≥ 24 h or anuria ≥ 12 h	≥ 300%[d] or acute RRT	3

[a] The remaining RIFLE stages are Loss (persistent acute renal failure = complete loss of kidney function > wk) and ESKD (> 3 mo).
[b] Serum creatinine increase from baseline.
[c] Urine output criteria are identical in the corresponding RIFLE and AKIN stages.
[d] Or if baseline > 4 mg/dL then increase ≥ 0.5 mg/dL; RRT = renal replacement therapy.

Source: Nephrology self-assessment program: Acute kidney injury and critical care nephrology by P. M. Palevsky & P.T. Murray (2009). *Journal of the American Society of Nephrology, 8*(3), 169–238. Copyright 2009 by American Society of Nephrology. Reproduced with permission of American Society of Nephrology via Copyright Clearance Center.

of pre-existing renal insufficiency associated with ageing (Chronopoulos, Cruz & Ronco, 2010).

The most common causes of AKI are ischaemia and exposure to nephrotoxic agents. The kidney is particularly vulnerable to both due to the amount of blood that passes through it. A fall in blood pressure or volume can result in ischaemic injury to the kidney tissues. Alternatively, nephrotoxins in the blood damage kidney tissue directly.

Physiology review

The functional unit of the kidneys, the nephron (see Figure 25.3), produces urine through three processes: glomerular filtration, tubular reabsorption and tubular secretion. In the *glomerulus*, a filtrate of water and small solutes is formed. The solute concentration of this filtrate is equal to that of plasma, with the exception of large molecules such as plasma proteins and blood cells. The GFR is affected by blood volume and pressure, the autonomic nervous system and other factors. From the glomerulus, the filtrate flows into the *tubules*, where its composition is changed by the processes of *tubular reabsorption* and *tubular secretion*. Most water and many filtered solutes such as electrolytes and glucose are reabsorbed. Metabolic waste products such as urea, hydrogen ion, ammonia and some creatinine are secreted into the tubule for elimination. By the time urine exits the collecting duct into the renal pelvis, 99% of the filtrate has been reabsorbed.

Pathophysiology

Acute kidney injury is categorised as being prerenal, intrinsic or postrenal. The incidence of prerenal AKI accounts for about 40–55% of all AKI cases (Sharfuddin et al., 2011). In *prerenal AKI*, hypoperfusion leads to kidney dysfunction without directly affecting the integrity of kidney tissues. *Intrinsic* (or *intrarenal*) *AKI*, due to direct damage to functional kidney tissue, is responsible for approximately 50% of reportable cases. Urinary tract obstruction with resulting kidney damage is the precipitating factor for *postrenal AKI* and is the least common form (~5%) (Sharfuddin et al., 2011). Table 27.4 summarises the causes of AKI. See also 'Pathophysiology illustrated: Acute kidney injury' below.

Prerenal AKI

Prerenal AKI results from any condition that decreases renal blood flow, causing subsequent hypoperfusion of the kidney; as a result, it is also termed 'volume responsive AKI'. Renal blood flow can be decreased though renovascular obstruction (e.g. renal artery stenosis), during surgical procedures or as a result of any condition that significantly decreases vascular volume, cardiac output or systemic vascular resistance. The kidney's natural response is to use its own autoregulatory system in an attempt to restore renal blood flow. The afferent and efferent arterioles dilate and constrict, respectively, while the enzyme angiotensinogenase (more commonly known as renin) is simultaneously released to activate the renin–angiotensin system to stimulate the production of the potent vasoconstrictor angiotensin II. Angiotensin II has multiple functions, including the constriction of the efferent arterioles, which increases the pressure within the glomeruli. It also facilitates the release of aldosterone from the adrenal cortex to increase sodium and chloride reabsorption. The ensuing increase in plasma sodium stimulates the secretion of antidiuretic hormone from the posterior pituitary gland, which increases water reabsorption and assists peripheral vasoconstriction. The sum effect of these autoregulatory features of the kidney is a decrease in urine production and an increase in circulating blood volume, which collectively increase blood pressure. The unfortunate by-product of this autoregulation is that urea reabsorption also occurs, which sees a corresponding elevation in serum urea levels. If these autoregulatory mechanisms are unsuccessful and renal perfusion is not restored, the continuing ischaemia can lead to acute tubular necrosis.

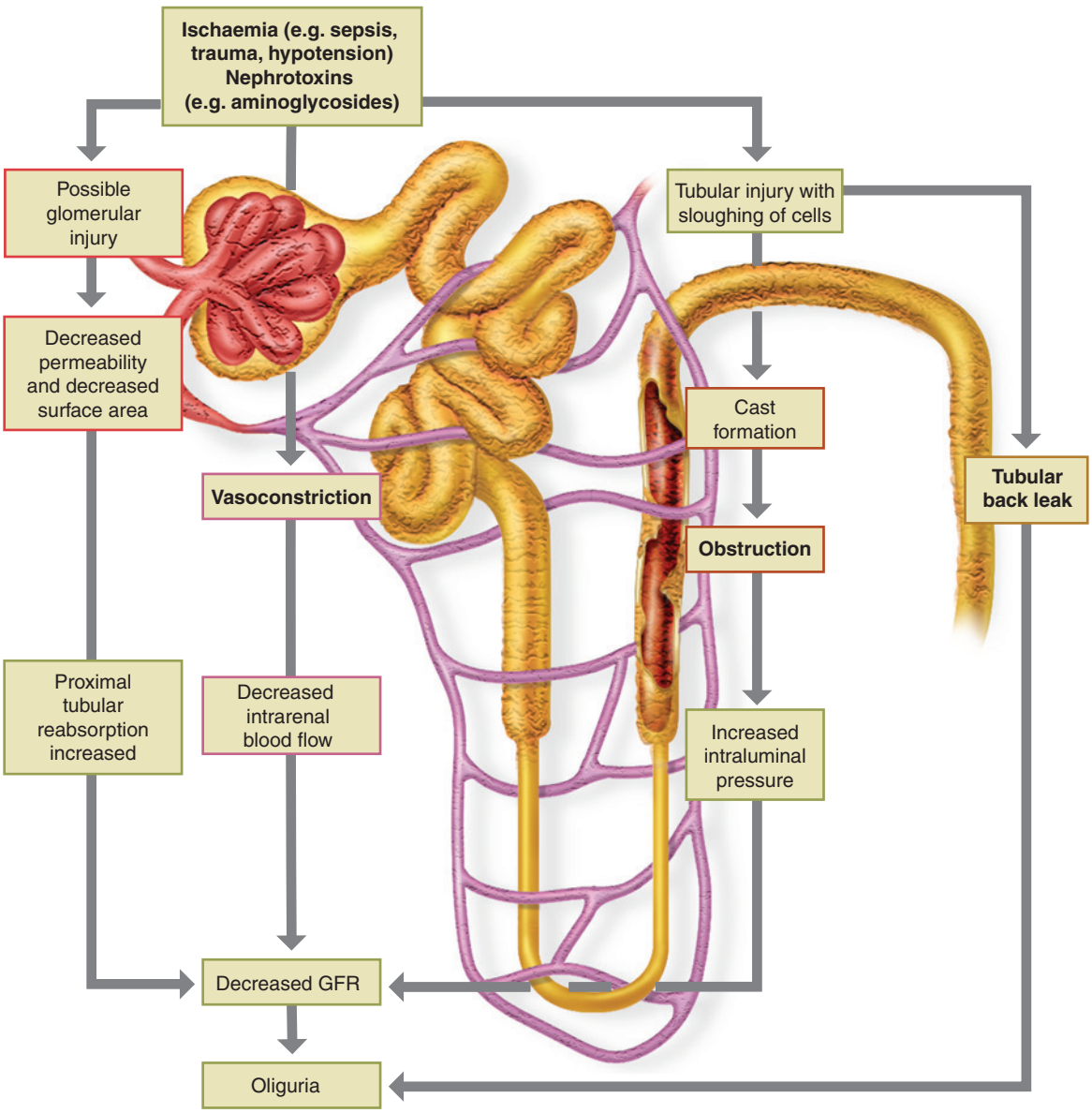
The kidneys normally receive 20–25% of the cardiac output to maintain the GFR. A drop in renal blood flow to less than 20% of normal causes the GFR to fall. As the filtration of substances by the glomeruli is reduced, less reabsorption of substances in the tubule is required. As a result, the kidney cells require less energy and oxygen, their metabolism slows and they effectively go to sleep (Grossman & Porth, 2014).

TABLE 27.4 Causes of acute kidney injury

	CAUSE	EXAMPLES
Prerenal	Hypovolaemia	Haemorrhage, dehydration, excess fluid loss from GI tract, burns, wounds, sepsis
	Decreased cardiac output	Heart failure, cardiogenic shock
	Altered vascular resistance	Sepsis, anaphylaxis, vasoactive drugs
	Renovascular obstruction	Renal artery stenosis, aneurysm, trauma, emboli, thrombi
Intrarenal	Glomerular/microvascular injury	Glomerulonephritis, disseminated intravascular coagulation, vasculitis, hypertension, toxaemia of pregnancy, haemolytic uraemic syndrome
	Acute tubular necrosis	Ischaemia due to conditions associated with prerenal failure; toxins such as drugs, heavy metals; haemolysis, rhabdomyolysis (muscle cell breakdown)
	Interstitial nephritis	Acute pyelonephritis, toxins, metabolic imbalances, idiopathic
Postrenal	Ureteral obstruction	Calculi, carcinoma, external compression Prostatic hypertrophy, calculi, carcinoma, stricture, blood clot, neuropathy

Acute kidney injury

The initial kidney injury is usually associated with an acute condition such as sepsis, trauma and hypotension, or the result of treatment for an acute condition with a nephrotoxic medication. Injury to the kidney can occur because of glomerular injury, vasoconstriction of capillaries or tubular injury. All consequences of injury lead to decreased glomerular filtration and oliguria.



FAST FACTS

- Prerenal AKI is common, particularly in trauma, surgical and critically ill people.
- Restoration of blood pressure and blood flow to the kidneys rapidly reverses prerenal AKI.
- If not promptly identified and treated, prerenal AKI leads to ischaemic acute tubular necrosis and intrinsic AKI.

Intrinsic (intrarenal) AKI

Intrinsic or intrarenal failure is characterised by acute damage to the renal parenchyma and nephrons. The major structures of the kidney that may be affected are the blood vessels, the glomerulus, the tubules and the interstitium. Intrarenal causes include diseases of the kidney itself, such as acute glomerulonephritis and **acute tubular necrosis (ATN)**, the most common intrinsic cause of AKI.

Acute glomerulonephritis

Acute **glomerulonephritis** can be caused by a number of different factors; however, the key feature is inflammation of the glomerulus which can decrease renal blood flow and cause AKI. Acute glomerulonephritis can result from infection, immunological abnormalities, drugs, toxins and systemic diseases such as lupus erythematosus. Acute post-streptococcal glomerulonephritis (also known as acute proliferative glomerulonephritis) is a common cause of glomerulonephritis. Infection of the pharynx or skin with group A beta-haemolytic *Streptococcus* is the usual initiating event for this disorder. Staphylococcal or viral infections, such as hepatitis B, mumps or varicella (chickenpox), can lead to a similar post-infectious acute glomerulonephritis (Huether, 2010). This primarily childhood disease can also affect adults. See 'Pathophysiology illustrated: acute glomerulonephritis' below.

MANIFESTATIONS AND COMPLICATIONS Post-infectious acute glomerulonephritis is characterised by an abrupt onset of **haematuria**, **proteinuria**, salt and water retention, and evidence of uraemic symptomology occurring 10 to 14 days after the initial infection. The urine often appears brown or cola-coloured. Salt and water retention increase extracellular fluid volume, leading to hypertension and oedema. The oedema is primarily noted in the face, particularly around the eyes (*periorbital* oedema). Dependent oedema, affecting the hands and upper extremities in particular, may also be noted. Other manifestations may include fatigue, anorexia, nausea and vomiting, and headache. See the 'Manifestations' box below.

The older adult may have less apparent symptoms. Nausea, malaise, arthralgias and proteinuria are common manifestations; hypertension and oedema are seen less often. Pulmonary infiltrates may occur early in the disorder, often due to worsening of a pre-existing condition such as heart failure.

The prognosis for adults with acute post-infectious glomerulonephritis is less favourable than it is for children. The symptoms may resolve spontaneously within 10 to 14 days. Full recovery is usual in children, whereas approximately only 60% of adults recover completely, decreasing to 25% in the older population. The remainder have persistent symptoms and some have permanent kidney damage (Rodriguez-Iturbe et al., 2007; Nasr et al., 2011).

ACUTE TUBULAR NECROSIS ATN is characterised pathologically by structural damage to the tubular epithelial cells and cell death if the insult is not reversed promptly. Nephrons are particularly susceptible to injury from ischaemia or exposure to nephrotoxic agents. Prolonged ischaemia is the primary cause of ATN. When ischaemia and nephrotoxin exposure occur concurrently, the risk of ATN and tubular dysfunction increases. See Figure 27.1 for the pathogenesis of acute kidney injury due to ATN. Risk factors for ischaemic ATN include major surgery, severe hypovolaemia, sepsis, trauma and burns. The impact of ischaemia resulting from vasodilation and fluid loss in sepsis, trauma and burns is often compounded by toxins and inflammatory markers released by bacteria or from damaged tissue (Koyner & Murray, 2009).

Ischaemia lasting more than 2 hours causes severe and irreversible damage to kidney tubules with patchy cellular necrosis and sloughing. The GFR is significantly reduced as a result of (1) ischaemia, (2) activation of the renin–angiotensin system, and (3) tubular obstruction by cellular debris, which raises the pressure in the glomerular capsule.

Common nephrotoxic agents associated with ATN include the aminoglycoside antibiotics and radiological contrast media. Research has demonstrated AKI to be a common problem in the hospitalised and ambulatory population and the term contrast-induced AKI (CI-AKI) is proposed for people who develop AKI following intravenous radiological contrast media. Many other drugs (e.g. NSAIDs, antineoplastics and some immunosuppressants), heavy metals such as mercury and gold, and some common chemicals such as ethylene glycol (antifreeze) are also potentially toxic to the renal tubule. The risk of ATN is higher when nephrotoxic drugs are given to older people or those with pre-existing renal insufficiency and when used concomitantly with other nephrotoxic agents. Dehydration also increases the risk by increasing the toxin concentration in nephrons (Kidney Disease: Improving Global Outcomes (KDIGO), 2012a).

Nephrotoxins destroy tubular cells by both direct and indirect effects. As tubular cells are damaged and lost through necrosis and sloughing, the tubule becomes more permeable. This increased permeability results in filtrate reabsorption, further reducing the ability of the nephron to eliminate wastes.

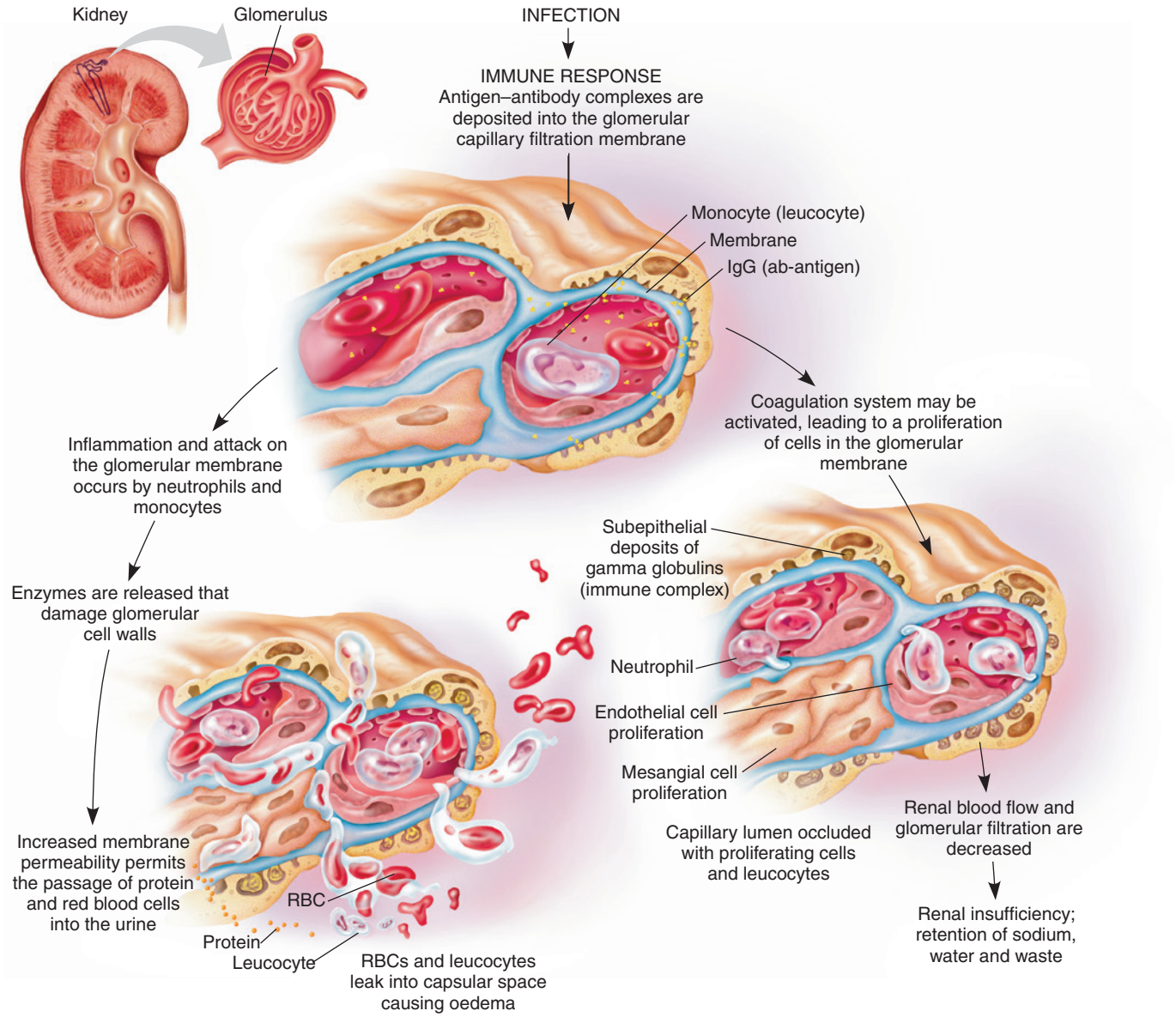
Rhabdomyolysis is a pathological syndrome that is characterised by damage to skeletal muscle fibres, which causes the release of excess myoglobin. Myoglobin is a protein that acts as the oxygen reservoir for muscle fibres, much as haemoglobin does for the blood. Muscle trauma and other factors can precipitate rhabdomyolysis. The myoglobin obstructs renal tubules, causing ischaemic injury, and contains an iron pigment that directly damages the tubules. Common causes of rhabdomyolysis include crush injuries, strenuous exercise, drug overdose and infection. Although no epidemiological data exists in Australia, 15–33% of people experiencing rhabdomyolysis will develop AKI. It accounts for between 7% and 15% of all cases of AKI in the United States (Melli, Chaudhry & Comblath, 2005; Russell, 2005). Renal injury occurs secondary to the myoglobin-induced tubule obstruction, renal hypoperfusion and intratubular cast formation. Prompt intervention with fluid resuscitation and urinary alkalinisation will minimise the impact of AKI and contribute to positive outcomes.

MANIFESTATIONS Acute glomerulonephritis

- Haematuria, cola-coloured urine
- Proteinuria
- Salt and water retention
- Oedema, periorbital and facial, dependent oedema
- Hypertension
- Uraemia
- Fatigue
- Anorexia, nausea and vomiting
- Headache

Acute glomerulonephritis

Infection from group A beta-haemolytic *Streptococcus* causes an immune response that causes inflammation and damage to the glomeruli. Protein and red blood cells are allowed to pass through the glomeruli. Blood flow to the glomeruli is reduced due to obstruction with damaged cells and renal insufficiency results, leading to the retention of sodium, water and waste.



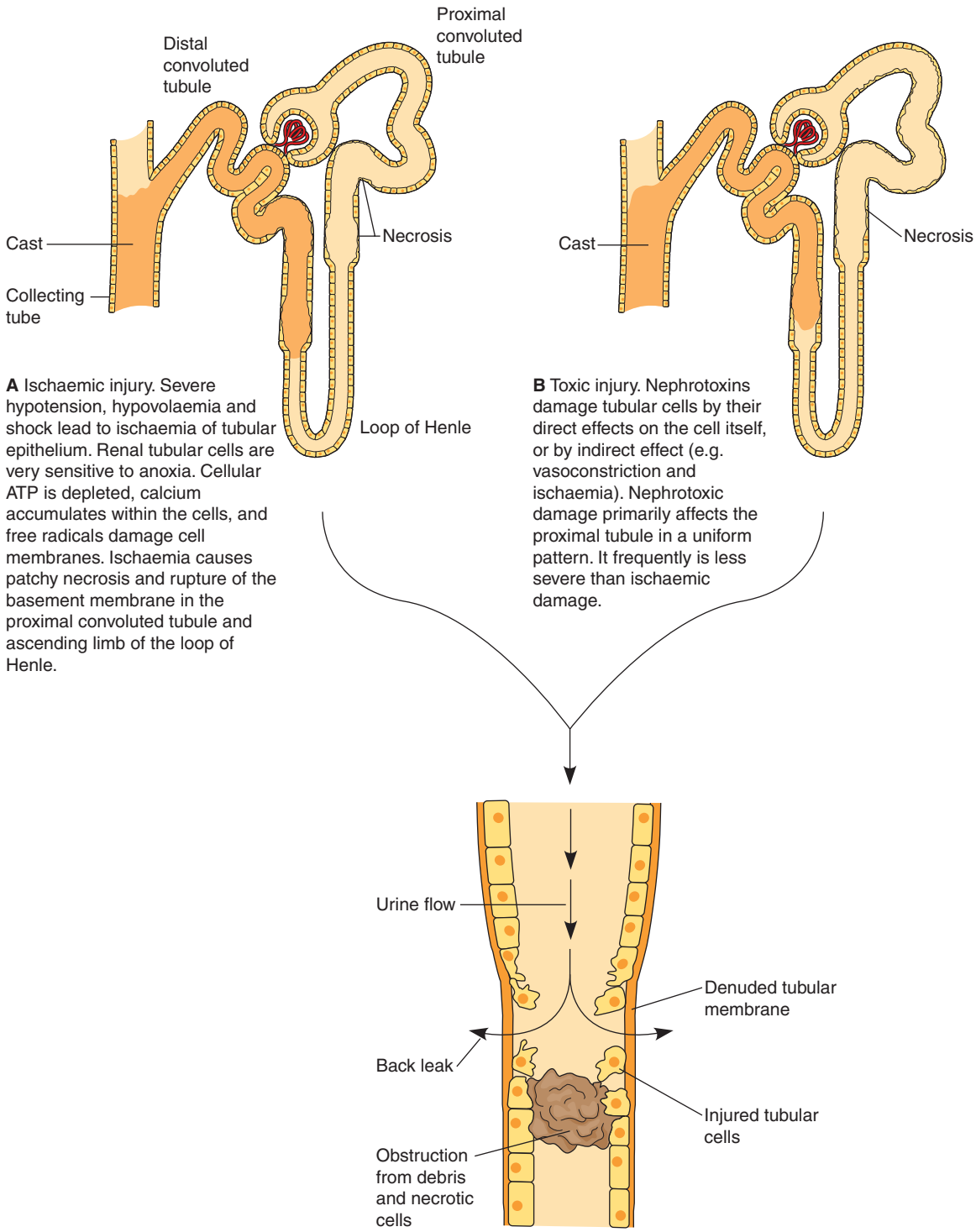
Postrenal AKI

Obstructive causes of acute kidney injury are classified as postrenal. Any condition that prevents urine excretion can lead to postrenal AKI. Benign prostatic hypertrophy is the most common precipitating factor. Others include renal or urinary tract calculi and tumours. See Chapter 26 for further discussion of urinary tract obstruction.

FAST FACTS

Clues for identifying pre-existing CKD in people with suspected AKI:

- Review biochemistry history: does the person currently have an elevated parathyroid hormone or is there a record of elevated serum creatinine levels anywhere in their patient history?
- Renal ultrasound revealing small kidneys with decreased cortical width is consistent with a history of CKD.
- Does the person have a long-standing history of uraemic symptoms?



A Ischaemic injury. Severe hypotension, hypovolaemia and shock lead to ischaemia of tubular epithelium. Renal tubular cells are very sensitive to anoxia. Cellular ATP is depleted, calcium accumulates within the cells, and free radicals damage cell membranes. Ischaemia causes patchy necrosis and rupture of the basement membrane in the proximal convoluted tubule and ascending limb of the loop of Henle.

B Toxic injury. Nephrotoxins damage tubular cells by their direct effects on the cell itself, or by indirect effect (e.g. vasoconstriction and ischaemia). Nephrotoxic damage primarily affects the proximal tubule in a uniform pattern. It frequently is less severe than ischaemic damage.

C Injured tubular cells release intracellular debris, which combines with proteins within the tubules to form casts. These casts, together with sloughed necrotic cells, occlude the tubular lumen, increasing tubular pressure and disrupting the flow of glomerular filtrate. Glomerular filtration slows. The increased pressure pushes filtrate out of the damaged tubule into interstitial tissues (back leak). Renal blood flow and glomerular filtration may be further reduced by intrarenal angiotension II release and vasoconstriction.

FIGURE 27.1 ■ Acute tubular necrosis. In ATN, tubular epithelial cells are destroyed by either ischaemic or toxic injury

Clinical course and manifestations of acute kidney injury

Not all people with AKI experience an identical clinical course; however, more often than not it progresses as a continuum through four phases: initiation, maintenance, diuretic and recovery.

Initiation phase

The *initiation phase* may last hours to days. It begins with the initiating event (e.g. haemorrhage) and ends when tubular injury occurs. If AKI is recognised and the initiating event is effectively treated during this phase, the prognosis is good. The initiation phase of AKI has few manifestations; in fact, it is often identified only when manifestations of the maintenance phase develop.

Maintenance phase

The *maintenance phase* of AKI is characterised by a significant fall in GFR and tubular necrosis. **Oliguria** may develop, although many people continue to produce normal or near-normal amounts of urine (non-oliguric AKI). Even though urine may be produced, the kidney cannot efficiently eliminate metabolic wastes, water, electrolytes and acids from the body during the maintenance phase of AKI. Uraemic symptoms, fluid retention, electrolyte imbalances and metabolic acidosis develop. These abnormalities are more severe in oliguric AKI than non-oliguric AKI. Up to 50% of AKI is non-oliguric, which generally reflects a less severe insult and is associated with a more favourable prognosis than in people with oliguric AKI.

During the maintenance phase, salt and water retention cause oedema, increasing the risk of heart failure and pulmonary oedema. Impaired potassium excretion leads to hyperkalaemia. When the serum potassium level is greater than 6.0 to 6.5 mmol/L, manifestations of its effect on neuromuscular function develop. These include muscle weakness, nausea and diarrhoea, electrocardiographic changes, arrhythmias and possible cardiac arrest. Other electrolyte imbalances include hyperphosphataemia and hypocalcaemia. Metabolic acidosis results from impaired hydrogen ion elimination by the kidneys.

Anaemia develops after several days of AKI due to suppressed erythropoietin secretion by the kidneys. Immune function is often impaired, increasing the risk of infection, and is one of the leading causes of mortality in AKI. Other manifestations of the maintenance phase include:

- oedema and hypertension due to salt and water retention
- confusion, disorientation, agitation or lethargy, hyperreflexia and possible seizures or coma due to uraemic toxicity and electrolyte and acid–base imbalances
- anorexia, nausea, vomiting and decreased or absent bowel sounds
- uraemic syndrome if AKI is prolonged (see the section on chronic kidney disease that follows).

Diuretic phase

The diuretic phase, if it occurs, can last up to 1 to 2 weeks. An increase in diuresis occurs because patency within the renal tubules is re-established. Nephrons have not recovered functionally and still cannot concentrate urine; and the solutes previously retained, such as urea and sodium, promote an osmotic effect, thus increasing urine output. It is crucial during this phase that the nurse ensures that fluid intake matches urine output so that renal perfusion is maintained. As the diuretic phase ends, urea and creatinine levels begin to decrease and electrolyte imbalances begin to resolve, and the kidneys start to regain their ability to regulate hydrogen and bicarbonate ion exchange. This sees the commencement of the recovery phase.

Recovery phase

The recovery phase of AKI is characterised by the fibrous scar tissue laid down within the glomerular basement membrane during the initial insult being replaced with a more contractile scar tissue. This process of tubular cell regeneration and repair sees a gradual return of the GFR to normal or pre-AKI levels. Serum creatinine, urea, potassium and phosphate levels remain high and may continue to rise in spite of increasing urine output. Renal function improves rapidly during the first 5 to 25 days of the recovery phase and continues to improve for up to a year.

FAST FACTS

The classification of urine output:

- Anuria: no urine output
- Oliguria: less than 400 mL/day
- Polyuria: greater than 2500 mL/day

Diagnostics

Diagnostic tests are used to identify the cause of acute kidney injury and monitor its effects on homeostasis:

- *Urinalysis* often shows the following abnormal findings in acute kidney injury:
  a. Urine-specific gravity is typically higher in prerenal AKI as the kidney attempts to conserve water, and lower in intrinsic AKI as the tubules are unable to concentrate the filtrate. However, urine osmolality provides a more accurate measurement.
  b. Proteinuria if glomerular damage is the cause of AKI.
  c. The presence of RBCs (due to glomerular dysfunction), WBCs (related to inflammation) and renal tubular epithelial cells (indicating ATN).
  d. Cell casts, which are protein and cellular debris moulded in the shape of the tubular lumen. (In AKI, RBCs, WBCs and renal tubular epithelial casts may be present. Brownish pigmented casts and positive tests for occult blood indicate haemoglobinuria or myoglobinuria.)
- *Serum creatinine* and *urea* are used to evaluate renal function. In AKI, serum creatinine levels increase rapidly, within 24 to 48 hours of the onset. Creatinine levels generally peak within 5 to 10 days. Creatinine and urea levels tend to increase more slowly when urine output is maintained. The onset of recovery is marked by a halt in the rise of the serum creatinine and urea. eGFR is an inaccurate marker to evaluate kidney function in AKI as the formula used to determine GFR relies on a steady-state creatinine level and does not account for

fluctuating or rapid changes in creatinine levels. It is also important to note that eGFR has not been validated in children or the Indigenous population.

- *Serum electrolytes* are monitored to evaluate the fluid and electrolyte status. The serum potassium rises at a moderate rate and is often used to indicate the need for dialysis. Hyponatraemia is common, due to the water excess associated with AKI.
- *Arterial blood gases* often show a metabolic acidosis due to the kidneys' inability to adequately eliminate metabolic wastes and hydrogen ions (see Chapter 9).
- *FBC* shows reduced RBCs, moderate anaemia and a low haematocrit. AKI affects erythropoietin secretion and RBC production. Iron and folate absorption may also be impaired, further contributing to anaemia.

INTERPROFESSIONAL CARE

Identifying people at risk and preventing acute kidney injury should be a goal for health professionals, especially for people in high-risk groups. Treatment goals for acute kidney injury are to: (1) identify and correct the underlying cause, (2) prevent additional kidney damage, (3) restore the urine output and kidney function, and (4) compensate for renal impairment until kidney function is restored.

Maintaining an adequate vascular volume, cardiac output and blood pressure is vital to preserve kidney perfusion. The nursing team is ideally placed to facilitate the coordination of interprofessional care and is able to notify the treating team of any changes in status and to identify the need for timely referral to other members of the healthcare team. AKI is a serious life-threatening illness that requires multidisciplinary collaboration, agreed guidelines and implementation to deliver high-quality care (Sedgewick, 2011).

Medications

The primary focus in drug management for acute kidney injury is to restore and maintain renal perfusion and volume homeostasis, correct biochemical imbalances and eliminate nephrotoxic agents from the treatment regimen.

Intravenous fluids and blood volume expanders are given as needed to restore renal perfusion. Low or 'renal' dose dopamine has been historically administered via intravenous infusion to increase renal blood flow. Dopamine is a sympathetic neurotransmitter that improves cardiac output and dilates blood vessels of the mesentery and kidneys when given in low therapeutic doses. However, recent research has shown that low-dose dopamine may worsen renal perfusion in critically ill people (Lauschke et al., 2006).

If restoration of renal blood flow does not improve urinary output, a loop diuretic such as frusemide may be given with intravenous fluids. The purpose is twofold. First, if nephrotoxins are present, the combination of fluids and potent diuretics may, in effect, 'wash out' the nephrons, reducing toxin concentration. Second, re-establishing urine output may prevent oliguria and reduce the degree of uraemic toxin accumulation and fluid and electrolyte imbalances. Frusemide also may be used to manage salt and water retention associated with AKI.

Aggressive hypertension management limits renal injury when AKI is associated with disorders such as toxaemia and pregnancy-induced hypertension. ACE inhibitors or other antihypertensive medications are used to control arterial pressures.

All drugs that are either directly nephrotoxic or that may interfere with renal perfusion may be discontinued. NSAIDs, nephrotoxic antibiotics and other potentially harmful drugs are avoided throughout the course of acute kidney injury. When a nephrotoxic drug or substance must be used, the risk of AKI can be reduced by using the minimum effective dose, maintaining hydration and eliminating other known nephrotoxins from the medication regimen.

People with kidney injury are at an increased risk of GI bleeding; this is related to the stress response and impaired platelet function. The cause is likely due to the combined effect of uraemic toxins, medications like warfarin or heparin, or coexisting GI tract pathologies. Histamine H_2-receptor antagonists or proton pump inhibitors are used to prevent GI haemorrhage. These drug classes protect the gastric mucosa by profoundly reducing gastric acid secretion. Antacids are used infrequently due to the inconvenience of dosing, whereas the other drug classes afford more convenient administration routes and less dosing intervals. It is worth noting that there has been an increase in reported cases linking episodes of acute interstitial nephritis with the use of proton pump inhibitors (Brewster & Perazella, 2007).

Hyperkalaemia may require active intervention in combination with the restriction of dietary potassium intake. Serum levels of greater than 6.0–6.5 mmol/L are treated to prevent cardiac effects of hyperkalaemia. With significant hyperkalaemia, calcium chloride, sodium bicarbonate or insulin and glucose may be given intravenously to reduce serum potassium levels by moving potassium into the cells. A potassium-binding exchange resin such as calcium resonium may be given orally or by enema. This agent removes potassium from the body by exchanging sodium for potassium, primarily in the large intestine. When given orally, it is often co-administered with an aperient to prevent constipation. Rectally, it is instilled as a retention enema, allowed to remain in the bowel for approximately 30 to 60 minutes and then irrigated out using a tap-water enema.

Hyperphosphataemia is generally managed conservatively in AKI unless it is severe and there is a clinically significant reduction in serum calcium. Generally it is only when the episode of AKI is protracted that oral phosphate binders are initiated (see the CKD section on medications later in this chapter for a more comprehensive review of phosphate binders).

Because many drugs are eliminated from the body by the kidney, drug dosages may need to be adjusted. Doses within the usual range can lead to potentially toxic blood levels, because their elimination is slowed and half life prolonged. Nursing implications for medications commonly prescribed for the person with AKI are summarised in the 'Medication administration' box below.

MEDICATION ADMINISTRATION The person with acute kidney injury

LOOP DIURETICS

Frusemide
Ethacrynic acid

The loop diuretics, named for their primary site of action in the loop of Henle, are *high-ceiling diuretics*: the response increases with increasing doses. These are highly effective diuretics used in early AKI to re-establish urine flow and convert oliguric renal failure to non-oliguric renal failure. Loop diuretics may be given with intravenous dopamine to promote renal blood flow. In ATN secondary to exposure to a nephrotoxic agent, loop diuretics are often used to clear the toxin from the nephrons more rapidly. Loop diuretics cause potassium wasting, which is generally not a concern in AKI because renal dysfunction impairs normal potassium elimination.

Nursing responsibilities

- Assess weight and vital signs for baseline data.
- Monitor intake and output, daily weight (or more frequently as ordered), vital signs, skin turgor and other indicators of fluid volume status frequently.
- Assess for postural hypotension because these potent diuretics can lead to hypovolaemia.
- Monitor laboratory results, especially serum electrolyte, glucose, urea and creatinine levels.
- Administer by mouth or, if ordered, by intravenous injection:
  a. Frusemide undiluted at a rate of no more than 20 mg per minute
  b. Ethacrynic acid 50 mg diluted with 50 mL of normal saline at a rate of no more than 10 mg per minute
- Assess response. Urine output typically increases within 10 minutes after intravenous administration.
- Monitor hearing and for complaints such as tinnitus. High doses of loop diuretics increase the risk of ototoxicity, especially with ethacrynic acid. These effects may be reversible if detected early and the drug is discontinued.
- Avoid administering concurrently with other ototoxic agents, such as aminoglycoside antibiotics and cisplatin.

Health education for the person and family

- Unless contraindicated, maintain a fluid intake that is equal to the previous day's urine output plus 500–700 mL to allow for insensible losses.
- Rise slowly from lying or sitting positions, because a fall in blood pressure may cause light headedness.
- Administer in the morning and at lunchtime if ordered twice a day, to avoid sleep disturbance.
- Take with food or milk to prevent gastric distress.
- NSAIDs interfere with the effectiveness of loop diuretics and should be avoided.

ELECTROLYTES AND ELECTROLYTE MODIFIERS

Calcium chloride
Calcium gluconate
Sodium bicarbonate
Calcium polystyrene sulfonate

Calcium chloride or gluconate and sodium bicarbonate are administered intravenously in the initial management of hyperkalaemia. Calcium can also be administered to correct hypocalcaemia and reduce hyperphosphatemia. (Calcium and phosphate have a reciprocal relationship in the body; as the level of one rises, the level of the other falls.) Sodium bicarbonate helps to correct acidosis and move potassium back into the intracellular space. Calcium polystyrene sulfonate is used to remove excess potassium from the body by exchanging calcium ions for potassium in the large intestine.

Nursing responsibilities

- Assess serum electrolyte levels prior to and during therapy. Report rapid shifts or adverse responses to the treating team.
- Administer as appropriate:
  a. Intravenous calcium chloride at less than 1 mL per minute; intravenous calcium gluconate at 0.5 mL per minute. Inject into a large vein through a small-bore needle; avoid infiltration because extravasation of intravenous solution will cause tissue necrosis.
  b. Intravenous sodium bicarbonate infusion over 4 to 8 hours; oral tablets as prescribed.
  c. Calcium polystyrene sulfonate as an oral solution can be taken with a laxative to prevent constipation or as a retention enema mixed with warm water. Leave in the bowel for 30 to 60 minutes; irrigate using a small tap-water enema.
- Monitor for adverse reactions, such as arrhythmias, electrolyte imbalances and metabolic alkalosis.

Health education for the person and family

- Intravenous calcium may make you light headed; remain in bed for at least 30 minutes after administration.
- Do not take sodium bicarbonate tablets with milk.
- Retain the calcium polystyrene sulfonate enema as long as possible.

Fluid management

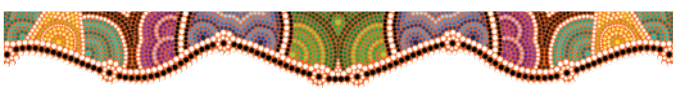
Once vascular volume and renal perfusion is restored, fluid intake is usually restricted. The allowed daily fluid intake is calculated by allowing 500–700 mL for insensible losses (respiration, perspiration, bowel losses) and adding the amount excreted as urine (or lost in vomitus) during the previous 24 hours. For example, if a person with AKI excretes 325 mL of urine in 24 hours, they are allowed a fluid intake allowance (including oral and intravenous fluids) of 825 mL for the next 24 hours. Fluid balance is carefully monitored, using accurate weight measurements and strict fluid balance charts as the primary indicators.

Nutrition

Nutritional management is critical in supporting the person with AKI. AKI is associated with the accumulation of electrolytes and fluids and the accelerated breakdown of body proteins (*catabolism*) which creates a negative nitrogen balance. Poor nutritional status is associated with increased mortality and morbidity; people with malnutrition are at higher risk of sepsis and

haemorrhage. Managing nutritional intake remains a challenge in which the determinants of treatment requirements are based not on the degree of renal insufficiency, but on the cause of the AKI and the degree of catabolism experienced. People in hypercatabolic states experience multiple metabolic sequelae, including insulin resistance, hormonal alterations and metabolic acidosis. The goal of nutritional management is to maintain lean body mass through the provision of adequate kilojoule and nutritional intake, and to restore immunocompetence and an anabolic state (Fiaccadori & Cremaschi, 2009). Early referral to the dietitian is essential in order to provide comprehensive and timely management of the individual's nutritional status. Nutritional requirements are highly individualised and are influenced by the cause and treatment of the AKI. Due to the well-established association of malnutrition with morbidity and mortality, protein restriction is not recommended. It is recommended that a non-catabolic person with AKI who is not on dialysis receive between 0.8 and 1.0 g/kg/day of protein. For those receiving kidney replacement therapy higher amounts are recommended (KDIGO, 2012b). Dietary proteins should be of high biological value (rich in essential amino acids). Carbohydrates are increased to maintain adequate kilojoule intake and provide a protein-sparing effect.

Parenteral nutrition providing amino acids, concentrated carbohydrates and fats may be instituted when the individual with AKI cannot tolerate enteral nutrition or consume an adequate diet (e.g. due to impaired gut function, nausea, vomiting or underlying critical illness). The disadvantages of parenteral nutrition in these instances are the high volume of fluid required and the risk of infection through the central venous access.



Nursing care

Health promotion

Acute kidney injury often can be prevented by measures that maintain fluid volume and cardiac output and reduce the risk of exposure to nephrotoxins.

- Carefully monitor critically ill, postoperative and other people who are at high risk of early signs of hypovolaemia (low urine output, altered mental status or changes in vital signs, skin colour or temperature).
- Report significant decreases in urine output and other evidence of decreased cardiac output.
- Maintain intravenous fluids as ordered.
- Alert the treating team if you note that the individual is receiving more than one nephrotoxic drug or if a nephrotoxic drug is ordered for a person who is dehydrated.
- Closely observe people receiving blood or blood cells for early signs of transfusion reaction and intervene appropriately.

Assessment

Both subjective and objective data are useful when assessing the person with acute kidney injury:

- *Health history:* complaints of anorexia, nausea, weight gain or oedema; recent exposure to a nephrotoxin such as an aminoglycoside antibiotic or radiological procedure using an injected contrast medium; previous transfusion reaction; chronic diseases such as diabetes, heart failure or kidney disease.
- *Physical examination:* vital signs, including temperature; urine output (amount, colour, clarity, specific gravity, presence of blood cells or protein); weight; skin colour, peripheral pulses; presence of oedema (periorbital or dependent); lung sounds, heart sounds and bowel tones.

Nursing diagnoses and interventions

The person with acute kidney injury has numerous nursing care needs related not only to the renal dysfunction but also to the underlying condition that precipitated it. Priority nursing care needs relate to fluid volume alterations, appetite and nutrition, and teaching/learning. For additional nursing diagnoses and interventions, see the nursing care plan below.

Excess fluid volume

In AKI, the kidneys often cannot excrete adequate urine to maintain a normal extracellular fluid balance. Fluid retention is greater in oliguric renal failure than in non-oliguric failure. Rapid weight gain and oedema indicate fluid retention. In addition, heart failure and pulmonary oedema may develop.

- Maintain hourly intake and output records. *Accurate intake and output records help guide therapy, especially fluid restrictions.*
- Weigh daily. Use standard technique (same scale, time, clothing or coverings) to ensure accuracy. *Rapid weight changes are an accurate indicator of fluid volume status, particularly in the oliguric person.*
- Assess vital signs at least every 4 hours. *Hypertension, tachycardia and tachypnoea may indicate excess fluid volume.*

CONSIDERATION FOR PRACTICE

Frequently assess breath and heart sounds, neck veins for distension and back and extremities for oedema. Report abnormal findings. Adventitious breath sounds (crackles), abnormal heart sounds such as an S_3 or S_4 gallop, distended neck veins and peripheral oedema may indicate hypervolaemia, heart failure or pulmonary oedema.

- If not contraindicated, place in semi-Fowler's position *to enhance cardiac and respiratory function.*
- Report abnormal serum electrolyte values and manifestations of electrolyte imbalance. People with AKI are at particular risk of the following electrolyte imbalances:
  a. *Hyperkalaemia* due to impaired potassium excretion. Manifestations include irritability, nausea, diarrhoea, abdominal cramping, muscle weakness, cardiac arrhythmias and ECG changes.
  b. *Hyponatraemia* due to water retention. Manifestations include nausea, vomiting and headache, with possible central nervous system (CNS) manifestations of lethargy, confusion, seizures and coma. The body's natural physiological response post surgery is to retain sodium and water.

c. *Hyperphosphataemia* due to decreased phosphate excretion. Manifestations include hyperreflexia, paraesthesias and possible tetany secondary to hypocalcaemia.

AKI impairs electrolyte and water excretion, causing multiple electrolyte imbalances.

- Restrict fluids as ordered. Provide frequent mouth care and strategies to decrease thirst drive (chewing gum, boiled lollies). If ice chips are allowed, include the water content (approximately one-half of the total volume) as intake. *Fluids are restricted to minimise fluid retention and complications of fluid volume excess.*
- Administer medications with meals. *Giving oral medications with meals minimises ingestion of excess fluids.*
- Turn frequently and provide good skin care. *Oedema decreases tissue perfusion and increases the risk of skin breakdown.*

Imbalanced nutrition: less than body requirements

Anorexia and nausea associated with any form of renal insufficiency often interfere with oral intake and nutrition. In addition, the disease process leading to AKI may contribute to increased nutritional needs for healing and decreased food intake.

- Monitor and record food intake, including the amount and type of food consumed. *A detailed intake record helps guide decisions about nutritional status and necessary supplements.*
- Weigh daily. Weight changes over time (days to weeks) reflect nutritional status, while rapid weight changes are more reflective of fluid volume status. *In AKI, weight may remain stable or increase due to fluid retention even though tissue mass is being lost.*
- Arrange for dietary consultation to plan meals within prescribed limitations that consider individual food preferences. *Diets restricted in protein, salt and potassium can be unpalatable; intake and appetite improve when preferred foods are included as allowed.*
- Engage in planning daily menus in line with dietary allowances. *Participation in meal planning increases the individual's sense of control and autonomy.*
- Allow family members to prepare meals within dietary restrictions. Encourage family members to eat with the person. *Familiar foods and social interaction encourage eating and increase enjoyment of meals.*
- Provide frequent, small meals or between-meal snacks. *These measures promote food intake in people who are fatigued or anorexic.*
- Administer anti-emetics as ordered and provide mouth care prior to meals. *Nausea and a metallic taste in the mouth, common manifestations of uraemia, can decrease food intake.*
- Administer parenteral nutrition as ordered if the person is unable to eat or tolerate enteral nutrition. *Preventing or slowing tissue catabolism is important for the people with AKI.*

CONSIDERATION FOR PRACTICE

Intravenous lines and parenteral nutrition solutions can increase the risk of infection. Monitor sites carefully for signs of infection or inflammation and adhere to local policy for line replacement.

Deficient knowledge

The person with AKI has multiple learning needs. These include information about AKI, diagnostic and laboratory studies, management strategies and implications for the recovery period.

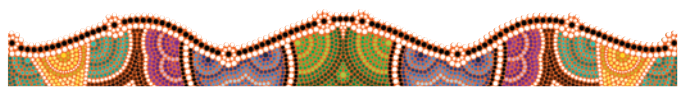
- Assess anxiety level and ability to comprehend instruction. Tailor information and presentation to developmental level and physical, mental and emotional status. *The person with AKI may be critically ill or have uraemic effects that hinder learning. During the initial stages of AKI it may be necessary to limit information to immediate concerns.*
- Assess knowledge and understanding. *To enhance understanding and retention, relate information presented to previous learning.*
- Teach about diagnostic tests and therapeutic procedures. *Teaching reduces anxiety and improves understanding and cooperation.*
- Discuss dietary and fluid restrictions. *These measures may be continued after discharge.*
- If discharge is planned prior to the recovery phase of AKI, teach the signs and symptoms of complications, such as fluid volume excess or deficit, heart failure and electrolyte imbalances. *As kidney function returns, urine output increases, but the concentrating ability of the nephrons and electrolyte excretion remain impaired. This impaired function increases the risk of excess fluid loss, possible dehydration, postural hypotension and electrolyte imbalance.*
- Teach how to monitor weight, blood pressure and pulse. *These are important means of assessing fluid status.*
- Instruct to avoid nephrotoxic agents for up to 1 year following an episode of AKI. *During recovery, nephrons remain vulnerable to damage by nephrotoxins such as NSAIDs, some antibiotics, radiological contrast media and heavy metals. Because alcohol can increase the nephrotoxicity of some materials, discourage alcohol intake.*

Community-based care

Quite often people develop AKI when they are critically ill. Critical illness and the resulting state of personal and family crisis can impair learning and retention of information. Include family members in teaching during the initial stages to promote understanding of what is happening and the reasons for specific treatment measures. Inclusion of the family reduces their anxiety and provides a valuable resource for reinforcing key health education points about care after discharge.

Teaching needs for home care include:

- avoiding exposure to nephrotoxins, particularly those in over-the-counter products
- preventing infection and other major stressors that can slow healing
- monitoring weight, blood pressure and pulse
- manifestations of relapse
- continuing dietary restrictions
- knowing when to contact the GP or present to the local emergency department.



NURSING CARE PLAN A person with acute kidney injury

Sharon Jones is driving home late one evening when she loses control of her car trying to avoid hitting a kangaroo on the road. Her car strikes a tree and rolls into a deep ditch beside the road, out of sight of passing cars. The wreck is not discovered until 8 hours later. On arrival at the accident scene, the paramedics find Ms Jones hypotensive: BP 90/60, P 120, R 24 and GCS 11/15. She appears disorientated and in severe pain, with a fractured right femur and multiple facial fractures. 1 L of crystalloid is infused, analgesia administered and oxygen applied. After immobilising Ms Jones's neck and back and extricating her from the car, they apply a traction splint to her leg and transport her to the local hospital.

ASSESSMENT

Upon arriving at the high dependency unit Ms Jones is more responsive, her pain score is 4/10 and GCS is 14/15. Lexi Ballinger, RN, obtains a nursing history on Ms Jones's admission and notes no significant medical history and nil known allergies. Ms Jones is not currently taking prescription or non-prescription drugs. Physical assessment findings on admission to the unit include T 36.3°C, P 100, R 18 and BP 124/68. Skin pale, cool and dry, with multiple scrapes and contusions on her face and extremities. A linear bruise is noted on her chest and abdomen from the seat belt. Lung sounds clear, heart tones normal and abdomen tender but soft to palpation. Right leg alignment maintained with skeletal traction. One unit of whole blood was infused in the emergency department. An indwelling urinary catheter and nasogastric tube were in situ.

During the first few hours after admission, Ms Ballinger notes that Ms Jones's hourly output has dropped from 55 mL to 45 mL to 28 mL of clear yellow urine. The resident medical officer is notified and orders a 500 mL intravenous fluid challenge, 40 mg frusemide IV, urinalysis, serum urea, creatinine and electrolytes. The fluid challenge and frusemide sees only a slight increase in urine output. Urinalysis results show a specific gravity of 1.010 and the presence of WBCs, red and white cell casts, and tubular epithelial cells in the sediment. Ms Jones's blood results reveal: urea 10 mmol/L; creatinine 133 µmol/L and potassium 5.9 mmol/L. The resident medical officer diagnoses probable acute kidney injury and orders sodium polystyrene sulfonate 15 g TDS. In addition, the physician orders aluminium hydroxide, 10 mL every 2 hours per nasogastric tube and ranitidine 50 mg intravenously every 8 hours.

DIAGNOSES

- *Acute pain* related to injuries sustained in accident.
- *Anxiety* related to admission to the high dependency unit.
- *Risk of excess fluid volume* related to impaired renal function.
- *Impaired physical mobility* related to skeletal traction.
- *Ineffective protection* related to injuries and invasive procedures.

PLANNING

- Plan for effective pain management.
- Plan for effective psychological care.
- Plan to monitor for bleeding, fluid balance and respiratory distress.
- Plan for skin care, pressure ulcer prevention and to prevent infection.

Expected outcomes

- Report adequate pain control.
- Verbalise reduced anxiety.
- Maintain stable weight and vital signs within normal range.
- Maintain skin integrity.
- Use the trapeze appropriately to adjust position in bed while maintaining body alignment.
- Remain free of infection, bleeding or respiratory distress.

IMPLEMENTATION

- Maintain PCA.
- Assess frequently for pain control and response to analgesia.
- Encourage expression of thoughts, feelings and fears about condition and placement in the critical care unit.
- Document vital signs and heart and lung sounds at least every 4 hours.
- Weigh daily as tolerated by pain.
- Document hourly intake and output.
- Restrict fluids as ordered, including diluents for all intravenous medications as intake.
- Assist with mouth care every 3 to 4 hours; allow frequent rinsing of mouth, and ice chips as allowed.
- Assist with position changes at least every 2 hours; teach use of the overhead trapeze.
- Monitor frequently for signs of infection, bleeding or respiratory distress.

EVALUATION

After just over 3 days of oliguria, Ms Jones's urine output increases. By the end of the fourth day she is excreting 60 to 80 mL/h of urine. Although her urea and creatinine levels remain slightly elevated, they never reach a critical point and dialysis is not required. She is transferred from the high dependency unit on the fourth day after admission. When Ms Jones is able to begin eating, she is placed on a low-potassium diet, restricted to 60 g of protein. Her renal function gradually improves. By discharge, the results of her renal function studies, including urea and serum creatinine, are nearly normal. Ms Jones verbalises an understanding of the need to avoid nephrotoxic agents such as NSAIDs in order to prevent another insult to her kidneys and agrees to be followed up by her GP in 2 weeks.

CRITICAL THINKING IN THE NURSING PROCESS

1. What was the most likely specific precipitating factor for Ms Jones's acute kidney injury? Did anything else contribute to her risk?
2. Why did the physician prescribe aluminium hydroxide and ranitidine? Consider both the acute kidney injury and Ms Jones's placement in the high dependency unit.
3. Ms Jones is at risk of respiratory distress related to potential fluid volume excess. How does her fractured femur further contribute to risk of respiratory distress?
4. Develop a care plan for Ms Jones for the nursing diagnosis of *Deficient diversional activity*.

REFLECTION ON THE NURSING PROCESS

1. Consider the case study above and identify what new knowledge you have gained that will enhance the care you deliver to a person with AKI.
2. Reflect on a time you have provided education in the clinical setting. What are some of the key factors you would need to consider when educating Ms Jones about avoiding nephrotoxic agents?

THE PERSON WITH KIDNEY TRAUMA

Despite the kidneys being relatively well protected by the rib cage and back muscles, kidney trauma due to blunt force or penetrating injury remains the most frequent urological trauma (Santucci & Bartley, 2010). The majority of kidney trauma is managed successfully without surgery, but prompt diagnosis and treatment is essential and can be lifesaving in the event of major damage.

Pathophysiology and manifestations

Blunt force is the most common cause of kidney injury. Falls, motor vehicle accidents and sports injuries can damage the kidney. The injury may be minor, resulting in a contusion or small haematoma, or more serious, resulting in laceration or other damage. The kidney may fragment or 'shatter', causing significant blood loss and urine extravasation. Tearing of the renal artery or vein may cause rapid haemorrhage, with shock and possible death.

Gunshot wounds, knife wounds, impalement injuries and fractured ribs are more likely to require surgical intervention including nephrectomy (Santucci et al., 2004). Minor penetrating injuries may lacerate the capsule or renal cortex. Major injuries include laceration or destruction of renal parenchyma or the vascular supply.

The primary manifestations of kidney trauma are haematuria (gross or microscopic), flank or abdominal pain, and oliguria or anuria. There may be localised swelling, tenderness or ecchymoses in the flank region. Retroperitoneal bleeding from the kidney may cause Turner's sign, a bluish discolouration of the flank. Signs of shock may be present, including hypotension, tachycardia, tachypnoea, cool and pale skin, and an altered level of consciousness.

INTERPROFESSIONAL CARE

The radiology team plays a crucial role in differentiating between cases that require immediate surgical intervention or conservative management. In blunt force trauma, renal imaging is indicated if there is suspected trauma (e.g. following a blunt injury or falling from a height) associated with either gross haematuria or microhaematuria with concurrent shock. Renal imaging is used immediately in penetrating trauma unless severe haemodynamic instability warrants urgent surgical intervention (Heyns, 2004). Haemoglobin and haematocrit levels fall in significant renal injury with haemorrhage. Haematuria is typically noted on urinalysis and aspartate transaminase (AST) levels rise within 12 hours of significant renal trauma. Renal ultrasound is used to diagnose bleeding and kidney damage. It is non-invasive but lacks the resolution afforded by a CT scan. An abdominal CT scan with contrast medium may be done to visualise renal structures to establish a definitive diagnosis. Renal angiogram has been replaced by CT as a diagnostic tool but may be performed for suspected renal artery thrombosis where stenting or percutaneous embolisation may be required (Heyns, 2004).

Treatment of minor kidney injuries is generally conservative, including bed rest, regular observation of vital signs, abdominal symptomology and full blood count. In these injuries, bleeding is typically minor and self-limiting, as opposed to major or critical trauma where immediate treatment focuses on controlling haemorrhage and treating or preventing shock. Major lacerations may require surgical repair or partial or total **nephrectomy** (removal) of the affected kidney (McCombie et al., 2014).

Nursing care

Nursing care for people who have experienced renal trauma focuses on timely and accurate assessment and appropriate intervention to preserve life and prevent complications. A urine specimen for analysis should be obtained when kidney trauma is suspected. Monitor level of consciousness, vital signs, skin colour and temperature, and urine output for possible signs of shock. See Chapter 10 for additional nursing care measures for the person who has had a traumatic injury or who develops shock.

THE PERSON WITH A RENAL TUMOUR

Renal tumours are classified as benign or malignant, primary or metastatic. Benign renal tumours are infrequent and are often found only on autopsy. In 2011, renal cell carcinoma accounted for 2.4% of all adult cancers in Australia (Australian Institute of Health and Welfare (AIHW), 2015a). Most primary renal tumours arise from renal cells; a primary tumour also may develop in the renal pelvis, although less frequently. Metastatic lesions to the kidney are associated with lung and breast cancer, melanoma and malignant lymphoma.

Males are affected by renal cancer more than females by a 2:1 ratio. The highest incidence is seen in people over the age of 55 years. Risk factors include smoking, obesity, prolonged time on dialysis, environmental exposure and genetic predisposition. The chronic irritation associated with renal calculi may also contribute (Kidney Health Australia (KHA), 2015).

Pathophysiology and manifestations

The most prevalent renal tumours are renal cell carcinomas, which develop in the renal cortex. The tumour, which can range in size up to several centimetres, has clearly defined margins and contains areas of ischaemia, necrosis and haemorrhage. Renal tumours tend to invade the renal vein and often have metastasised when first identified. Metastases tend to occur in the lungs, bone, lymph nodes, liver and brain (Fauci et al., 2008; Grossman & Porth, 2014).

Renal tumours are often silent, with few manifestations. The classic triad of symptoms—gross haematuria, flank pain and a palpable abdominal mass—is seen in only about 10% of people

with renal cell carcinoma. Haematuria, often microscopic, is the most consistent symptom. Systemic manifestations include fever without infection, fatigue and weight loss; see the box below.

The tumour may produce hormones or hormone-like substances, including parathyroid hormone, prostaglandins, prolactin, renin, gonadotropins and glucocorticoids. These substances produce *paraneoplastic syndromes*, with additional manifestations such as hypercalcaemia, hypertension and hyperglycaemia. The progression of renal cell carcinomas varies from prolonged periods of stable disease to very aggressive. Table 27.5 outlines the staging and prognosis for renal cell cancers.

TABLE 27.5 Renal cell cancer staging

STAGE	EXTENT OF TUMOUR	PROGNOSIS
I	Confined to the kidney capsule	> 90% 5-year survival
II	Invasion through the capsule but confined to local fascia	85% 5-year survival
III	Renal vein or inferior vena cava involvement (IIIA), local lymph node (IIIB) and local lymph nodes and vessels (IIIC)	60% 5-year survival
IV	Spread to nearby organs or distant metastases	≤ 10% 5-year survival

Source: Adapted from *Harrison's principles of internal medicine* (17th ed.) by A. S. Fauci et al. (eds) (2008). New York: McGraw-Hill.

INTERPROFESSIONAL CARE

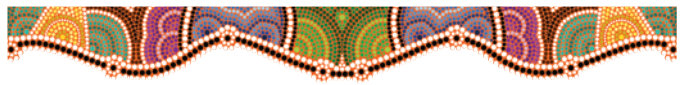
Haematuria is often the only initial manifestation of renal cancer; its presence indicates a need for further diagnostic studies. Commonly used imaging studies are discussed later in this chapter.

Radical nephrectomy is the treatment of choice for kidney tumours. In a radical **nephrectomy**, the adrenal gland, upper ureter, fat and fascia surrounding the kidney, as well as the entire kidney, are removed. Regional lymph nodes may also be resected. Although nephrectomy can be done using a laparoscopic approach, laparotomy primarily is used for radical nephrectomy. Nursing care for the person having a nephrectomy is summarised in the box below.

No effective treatment is available for advanced renal carcinoma with metastases. Biological therapies such as interferon or interleukin-2 have been used, but rarely achieve a durable effect. No chemotherapy drug consistently causes tumour regression in more than 20% of individuals (Fauci et al., 2008).

MANIFESTATIONS Renal tumours

- Microscopic or gross haematuria
- Flank pain
- Palpable abdominal mass
- Fever
- Fatigue
- Weight loss
- Anaemia or polycythaemia



Nursing care

Nursing diagnoses and interventions

Nursing care focuses on needs related to the cancer diagnosis and to the surgical intervention. Postoperative pain may be significant and the risk of respiratory complications is high. The remaining kidney must be protected from damage to preserve renal function. Psychologically, the person may grieve the loss of a major organ and the diagnosis of cancer.

Pain

The size and location of the incision used for a radical nephrectomy (see Figure 27.2) make pain management a challenge. Intercostal blocks, patient-controlled analgesia (PCA) or routine analgesic administration can effectively relieve the discomfort. Nursing care focuses on assessing pain relief, providing supportive measures to enhance analgesia and ensuring that pain or the fear of pain does not lead to respiratory complications.

- Assess frequently for adequate pain relief. Use a standard pain scale and non-verbal signs such as grimacing, tense body position, apparent dozing, elevated pulse, change of blood pressure or rapid, shallow respirations. Notify the physician of inadequate pain relief. *The person may assume that pain is to be expected or may fear becoming addicted to analgesics. Careful questioning and assessment allow effective pain management. Responses to analgesics are individual and the prescribed dose may need to be adjusted.*
- Assess the incision for inflammation or swelling, and drainage catheters and tubes for patency. *An obstructed catheter can lead to hydronephrosis, haematoma or abscess, increasing incisional pain.*
- Use adjunctive pain relief measures such as positioning, diversional activities, management of environmental stimuli, guided imagery and relaxation techniques. *These can enhance the effects of analgesia.*

CONSIDERATION FOR PRACTICE

Assess for abdominal distension, tenderness and bowel sounds. Intra-abdominal bleeding, peritonitis or paralytic ileus can cause pain that may be confused with incisional pain.

Ineffective breathing pattern

The location of the incision combined with the respiratory depressant effects of narcotic analgesics increases the risk of respiratory complications in the person who has had a nephrectomy.

- Position to promote respiratory excursion, using semi-Fowler's position and side-lying positions as allowed and tolerated. *Lung expansion is improved in semi-Fowler's and Fowler's positions.*

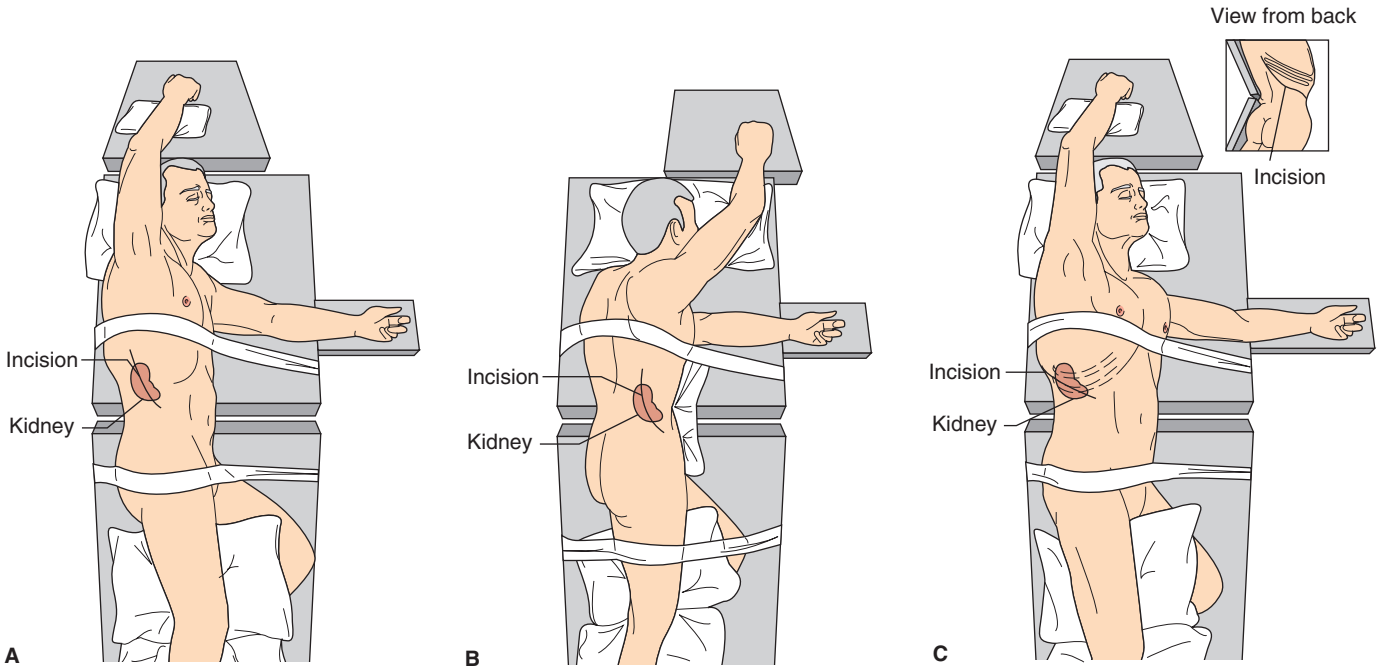


FIGURE 27.2 ■ Incisions used for kidney surgery: *A*, flank; *B*, lumbar; and *C*, thoracoabdominal

NURSING CARE OF THE PERSON having a nephrectomy

PREOPERATIVE CARE

- Provide routine preoperative care as outlined in Chapter 3.
- Report abnormal laboratory values to the surgical team. *Bacteriuria, blood coagulation abnormalities or other significant abnormal values may affect surgery and postoperative care.*
- Discuss operative and postoperative expectations as indicated, including the location of the incision and anticipated tubes, stents and drains. *Preoperative teaching about postoperative expectations reduces anxiety for the person and family during the early postoperative period.*

POSTOPERATIVE CARE

- Provide routine postoperative care as described in Chapter 3.
- Frequently assess urine colour, amount and character, noting any haematuria, pyuria or sediment. Promptly report any significant decreases in urine output or changes in colour or clarity. *Preserving function of the remaining kidney is critical; frequent assessment allows early intervention for potential problems.*
- Note the placement, status and drainage from ureteral catheters, stents, nephrostomy tubes or drains. Label each clearly. Maintain gravity drainage; irrigate only as ordered. *Maintaining drainage tube patency is vital to prevent potential hydronephrosis. Bright bleeding or unexpected drainage may indicate a surgical complication.*
- Support the grieving process and adjustment to the loss of a kidney. Loss of a major organ leads to a body image change and grief response. *When renal cancer is the underlying diagnosis, the person may also grieve the loss of health and potential loss of life.*
- Provide the following home care instructions for the person and family:
  a. The importance of protecting the remaining kidney by preventing UTI, renal calculi and trauma. See Chapter 26 for measures to prevent UTI and calculi. *Damage to the remaining kidney by UTI, renal calculi or trauma can lead to renal failure.*
  b. Maintain a fluid intake of 2000 to 2500 mL per day. *This important measure helps prevent dehydration and maintain good urine flow.*
  c. Gradually increase exercise to tolerance, avoiding heavy lifting for a year after surgery. Participation in contact sports is not recommended to reduce the risk of injury to the remaining kidney. Lifting is avoided to allow full tissue healing. *Trauma to the remaining kidney could seriously jeopardise renal function.*
  d. Care of the incision and any remaining drainage tubes, catheters or stents. *This routine postoperative instruction is vital to prepare the person for self-care and prevent complications.*
  e. Report abnormal signs and symptoms to the treating team, including manifestations of UTI (dysuria, frequency, urgency, nocturia, cloudy, malodorous urine) or systemic infection (fever, general malaise, fatigue), redness, swelling, pain or drainage from the incision or any catheter or drain tube site. *Prompt treatment of postoperative infection is vital to allow continued healing and prevent compromise of the remaining kidney.*

- Change position frequently, ambulate as soon as possible. *These measures promote lung expansion and the movement of mucus out of airways.*
- Encourage frequent (every 1 to 2 hours) deep breathing, spirometer use and coughing. *Assist to splint the incision. These measures promote alveolar ventilation, gas exchange and airway clearance.*

CONSIDERATION FOR PRACTICE

Assess respiratory status frequently, including rate and depth, cough, breath sounds, oxygen saturation and temperature. Pneumothorax on the operative side is common. Early identification and intervention can prevent major respiratory complications.

Risk of impaired urinary elimination

Surgery involving the urinary tract increases the risk of altered renal function and urine elimination. In addition, removal of one kidney dictates extra caution to maintain renal circulation, a sterile urinary tract and free urine flow.

- Monitor vital signs, central venous pressure (CVP) and urine output every 1 to 2 hours initially, then every 4 hours. *Hypovolaemia due to haemorrhage, diuresis or fluid sequestering (third spacing) reduces blood flow to the kidney and increases the risk of renal ischaemia with possible acute tubular necrosis and acute kidney injury.*
- Frequently assess the amount and nature of drainage on surgical dressings and from drainage tubes, stents and catheters. Measure and record output from each drain or catheter separately. *Frequent and accurate assessment of drainage helps to identify excess bleeding, abnormal fluid loss, infection or other potential surgical complications.*

CONSIDERATION FOR PRACTICE

Prevent kinking, twisting or tension on drains and tubes. Do not clamp. Notify the surgical team immediately if any tube becomes dislodged. It is vital to maintain the patency of drains, particularly any affecting the remaining kidney, to prevent the excess pressure of hydronephrosis.

- Maintain fluid intake with intravenous fluids until oral intake is resumed. Encourage an intake of 2000 to 2500 mL per day if not contraindicated. *A liberal fluid intake prevents dehydration, helps to dilute any nephrotoxic substances and promotes good urinary output.*
- Use strict aseptic technique in caring for all urinary catheters, tubes, stents, drains and incisions. *Asepsis is vital to prevent infection and possible compromise of the remaining kidney.*
- Following catheter removal, assess frequently for urinary retention. Notify the physician if the person is unable to void within 4 to 6 hours or if manifestations of retention (distended bladder, discomfort, urinary dribbling) develop. *Maintenance of urine output is vital to prevent stasis and possible complications such as infection and hydronephrosis.*
- Monitor laboratory results, including urinalysis, serum urea, creatinine and electrolytes. Report abnormal findings to the treating team. *Abnormal values may indicate early acute kidney injury; prompt intervention is necessary to preserve renal function.*

Anticipatory grieving

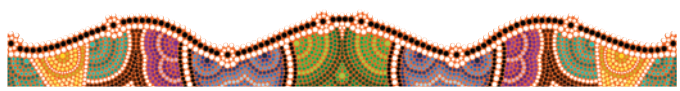
The person having a radical nephrectomy for renal cancer not only loses a major organ but also has to adjust to the diagnosis of cancer. Although the prognosis for recovery may be good, many people perceive cancer as always fatal. Providing support during the initial stages of grieving can improve physical recovery, psychological coping and eventual adaptation.

- Work to develop a trusting relationship with the person and family. *Trust increases the nurse's effectiveness in helping them work through the process of grieving.*
- Listen actively, encouraging the person and family to express fears and concerns. *As they begin to express their concerns, person and family can begin to deal more effectively with them.*
- Assist the person and family to identify strengths, past experiences and support systems. *These resources can be employed in working through the grieving process.*
- Demonstrate respect for cultural, spiritual and religious values and beliefs; encourage use of these resources to cope with losses. *Value and belief systems can provide a structure and form for dealing with the grieving process.*
- Encourage discussion of the potential impact of loss on the person and the family structure and function. Assist family members to share concerns with one another. *Sharing of fears and concerns among family members promotes involvement and support of the entire family unit so that the individual is not left to cope alone.*
- Refer to cancer support groups, social services or counselling as appropriate. *Support groups and counselling services provide additional resources for coping.*

Community-based care

If renal cancer was detected at an early stage and cure is anticipated, teaching for home care focuses on protecting the remaining kidney. Include the following measures to prevent infection, renal calculi, hydronephrosis and trauma:

- Maintain a fluid intake of 2000 to 2500 mL per day, increasing the amount during hot weather or strenuous exercise.
- Urinate when the urge is perceived and before and after sexual intercourse.
- Properly clean the perineal area.
- Watch for symptoms of UTI and understand the importance of early and appropriate evaluation and intervention.
- If the person is an older adult male, he should watch for symptoms of prostatic hypertrophy, a major cause of urinary tract obstruction. Stress the importance of routine screening examinations.
- Avoid contact sports such as football or hockey; use measures to prevent motor vehicle accidents and falls, which could damage the kidney.



THE PERSON WITH A CONGENITAL KIDNEY MALFORMATION

Congenital kidney disorders can affect the form and/or function of the kidney. Functional congenital kidney disorders are usually identified in childhood or adolescence. If function is not affected, congenital malformations may be detected only coincidentally. Malformations include agenesis, hypoplasia, alterations in kidney position and fusion anomalies such as horseshoe kidney.

Agenesis, absence of the kidney, and *hypoplasia*, underdevelopment of the kidney, typically affect only one of these paired organs. Renal function remains normal unless the unaffected kidney is compromised. Abnormal kidney position affects the ureters and urine flow, potentially leading to urinary stasis, increased risk of UTI and lithiasis or stone formation (see Chapter 26).

One in every 400 to 500 people has *horseshoe kidney*, making it one of the most common renal malformations (Kumar & Burton, 2007). Failure of the embryonic kidneys to ascend normally can result in a single, horseshoe-shaped organ. The two kidneys are fused at either the upper or lower pole (usually the lower). This malformation does not typically affect renal function; however, because the ureters cross the fused poles, there is an increased risk of *hydronephrosis* or distension of the renal pelvis and calyces with urine (see Chapter 26). Recurrent UTI and renal calculi are also common in people with horseshoe kidney.

Renal ultrasound and intravenous pyelography are used to diagnose horseshoe kidney. Correction of the abnormality is rarely necessary, although surgical resection of the isthmus (connection between the kidneys) may be done to relieve ureteral obstruction or allow access to the abdominal aorta, which lies behind it.

Nursing care for people with horseshoe kidney or other congenital malformations is primarily educational. Because abnormal kidney shape or position increases the risk of infection and stone formation, teach the person to maintain a fluid intake of at least 2500 mL per day. Emphasise the importance of avoiding dehydration by increasing fluids during hot weather and strenuous exercise. Teach hygiene practices such as perineal cleansing and voiding before and after intercourse to help prevent UTI. Teach the early manifestations of UTI and instruct to seek treatment promptly to prevent infection of the kidney. See Chapter 26.

THE PERSON WITH CHRONIC KIDNEY DISEASE

Although the kidneys usually recover from acute injury, many chronic conditions lead to progressive renal tissue destruction and loss of function. Nephron units are lost and renal mass decreases, with progressive deterioration of glomerular filtration, tubular secretion and reabsorption. This process of chronic kidney disease (CKD) may progress slowly for many years without being recognised. Eventually, the kidneys are unable to excrete metabolic wastes and regulate fluid and electrolyte balance adequately, a condition known as end-stage kidney disease (ESKD), the final stage of CKD.

A person is described as having **chronic kidney disease (CKD)** if they have a GFR < 60 mL/min/1.73 m^2 for more than 3 months with or without evidence of kidney damage, *or* evidence of kidney damage (with or without decreased GFR) for more than 3 months including microalbuminuria, proteinuria, glomerular haematuria or any anatomical or pathological abnormality. There are five stages of chronic kidney disease (see Figure 27.3) and, in Australia, one in 10 adults has indications of CKD including decreased GFR and microalbuminuria (Australian Bureau of Statistics, 2012).

Conditions causing CKD typically involve diffuse, bilateral disease of the kidneys with progressive destruction and scarring of the entire nephron. As indicated in Table 27.6, diabetic nephropathy continues to be the leading cause of ESKD in all population groups in Australia and New Zealand, and in fact in most of the Western world. Glomerulonephritis and hypertension follow as the next two main causes of ESKD, and in many people these disorders coexist (ANZDATA Registry, 2015).

CKD is a growing public health concern in Australia and New Zealand (KHA, 2015). Although data exist that enable us to report the incidence and prevalence of ESKD, it is much harder to report the same with CKD. This is due largely to its asymptomatic nature and, as such, incidence and prevalence

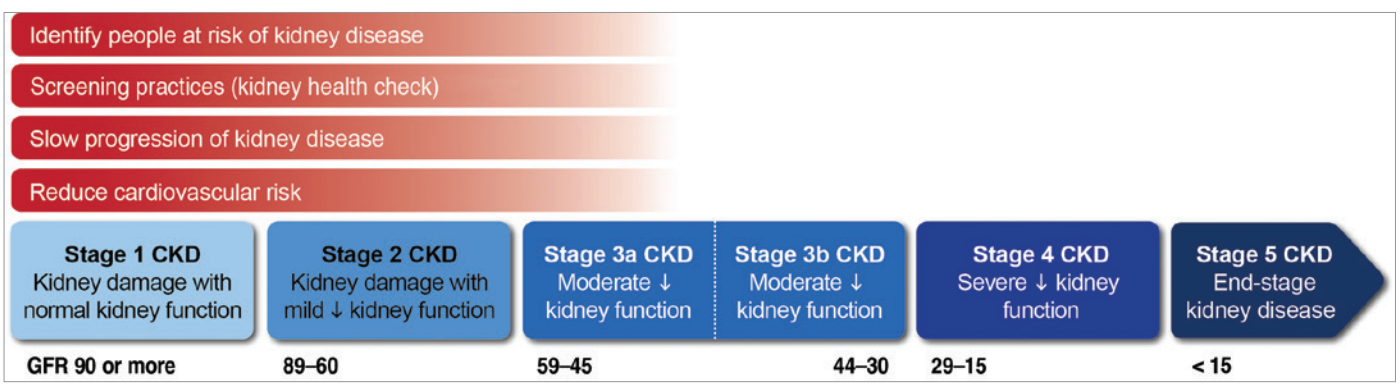


FIGURE 27.3 ■ Stages of chronic kidney disease

Source: Nephrology Educators Network.

TABLE 27.6 Pathophysiology of primary disease leading to end-stage kidney disease in the Australian population (2014)

CAUSE (% OF PEOPLE)	EXAMPLES
Diabetic nephropathy (35%)	Multifactorial. Changes in the glomerular basement membrane lead to sclerosis of the glomerulus and gradual destruction of the nephron.
Glomerulonephritis (19%)	Bilateral inflammatory process of the glomeruli leads to ischaemia, nephron loss and shrinkage of the kidney.
Hypertension (14%)	Long-standing hypertension damages the vasculature supplying the kidneys and leads to renal arteriosclerosis and ischaemia, resulting in glomerular destruction and tubular atrophy.
Polycystic kidney disease (6%)	Multiple bilateral cysts gradually destroy normal renal tissue by compression.
Reflux nephropathy (2%)	Results from chronic infection (pyelonephritis) commonly associated with an obstructive or neurological process and vesicoureteral reflux. The end result is renal scarring, atrophy and dilated calyces.

estimations are made from epidemiology studies and national self-reported surveys (Green & Ryan, 2009).

CKD was the leading cause of 2678 deaths in Australia in 2012 (AIHW, 2013). These figures do not accurately reflect the burden of CKD as the majority of people with CKD die of a cardiovascular-related illness rather than CKD per se. The burden of CKD in Australia has led to the development of a national CKD strategy with six national priority actions (see Box 27.1). Many area health services have now put into action CKD management programs to identify and screen people at risk, implement risk reduction and prevention strategies, and prevent its progression while managing the co-morbid associations of CKD.

The incidence of **end-stage kidney disease (ESKD)**, the terminal phase of CKD, is increasing, particularly in older adults. In 2013, 2544 and 546 people in Australia and New Zealand respectively began receiving treatment for ESKD. That same year, in Australia, a total of 11 774 people with ESKD underwent dialysis, with a further 9696 people maintaining functioning kidney transplants (ANZDATA Registry, 2015). The cumulative cost of treatment for all current and new cases of ESKD from 2009 to 2020 is projected to be between approximately $11.3 billion and $12.3 billion (Cass et al., 2006). This cost excludes not only the financial costs incurred by people undertaking kidney replacement therapy, but also the immeasurable costs and burden placed on their lifestyle.

BOX 27.1 National CKD strategy and national priority actions

- Risk reduction and prevention of CKD
- Early detection and management of CKD
- Management of advanced CKD
- Dialysis
- Organ donation and transplantation
- Indigenous Australians

Source: Kidney Health Australia (2008).

The burden of kidney disease on Indigenous peoples

Chronic kidney disease (CKD) exemplifies the differences in health equality with CKD death rates seven and 11 times higher for male and female Indigenous Australians than for non-Indigenous Australians (AIHW, 2009). Although exact comparisons are confounded by different age distributions, the incidence of CKD is still much higher in Indigenous than non-Indigenous Australians. In the more remote Indigenous communities, the incidence of ESKD is more prevalent than the national incidence; this often requires the need to relocate in order to receive dialysis treatment (Preston-Thomas, Cass & O'Rourke, 2007).

The burden of chronic illness on Indigenous Australians is reflected in the burden of kidney replacement therapy being required 10 years earlier than for non-Indigenous Australians, and death rates at an earlier age being more likely the result of cardiovascular, endocrine, metabolic, respiratory and nutrition-related illness than for non-Indigenous Australians (McDonald & Russ, 2003). Several factors contribute to this increased burden of kidney disease, including poorer socioeconomic conditions, higher rates of physical and behavioural risk factors, and poorer educational attainment. Indigenous Australians have additional risk factors that predispose them to CKD more than non-Indigenous Australians. These include higher rates of post-streptococcal childhood infections and prenatal elements that lead to lower nephron numbers due to reduced nephron endowment (Hoy et al., 2006; White, Hoy & McCredie, 2001).

The burden for Indigenous people can be increased when they are required to leave their lands for complex healthcare treatment in a distant Australian city. Nurse researchers Burnette and Kickett (2009) found that Aboriginal people undertaking dialysis away from their homes had overwhelming feelings of disempowerment related to their dependence on health professionals and dialysis treatment, perceived lack of education and the requirements of the foreign hospitalisation experience. Thus, the prevention or identification of early-stage

CKD in Indigenous populations is critical to ensure people receive appropriate management to delay, minimise or halt disease progression.

Physiology review

The glomeruli are the filtering units of the kidney, with each glomerulus consisting of a tuft of capillaries surrounded by a thin, double-walled capsule (Bowman's capsule) (see Figure 25.3). About 20% of the resting cardiac output flows through the glomeruli of the kidneys, forming approximately 180 L of plasma ultrafiltrate each day. More than 99% of this filtrate is reabsorbed in the renal tubules. The rate of glomerular filtration is controlled by opposing forces: the pressure and amount of blood flowing through the glomeruli promote filtration, and the pressure in Bowman's capsule and colloid osmotic (*oncotic*) pressure of the blood oppose it. The total surface area of glomerular capillaries also affects the GFR. The glomerular capillary membrane has three layers: the capillary endothelial layer, the basement membrane and the capsule epithelial layer. Water and the smallest solutes (such as electrolytes) pass freely across this membrane, whereas larger molecules (such as plasma proteins) are retained in the blood.

Pathophysiology

In the early stages of CKD, as nephrons are damaged the remaining functional nephrons hypertrophy. Glomerular capillary flow and pressure increase in these nephrons and more solute particles are filtered to compensate for lost renal mass. This increased demand predisposes the remaining nephrons to glomerular sclerosis (scarring), resulting in their eventual destruction. This process of continued loss of nephron function may continue even after the initial disease process has resolved (Longo et al., 2012).

Haematuria, microalbuminuria and proteinuria are manifestations of glomerular capillary membrane damage, which allows blood cells and proteins to escape from the blood into the glomerular filtrate. Haematuria may be either gross or microscopic. Microalbuminuria (30–300 mg/day), the precursor to proteinuria, may be detected early on in the disease process but this is highly reliant on screening practices and the type of testing methodology utilised. The degree of albuminuria can be measured with a simple early morning spot urine test to determine the albumin:creatinine ratio (ACR). Proteinuria is measured similarly with a protein:creatinine ratio (PCR) and is considered an important indicator of glomerular injury, because it increases progressively with increased glomerular damage. Loss of plasma proteins leads to hypoalbuminaemia (low serum albumin levels), which in turn reduces the plasma oncotic pressure (osmotic pressure created by plasma proteins), leading to oedema.

As plasma proteins are lost, the forces opposing filtration diminish and the amount of filtrate increases. The increased flow of filtrate stimulates the renin–angiotensin–aldosterone mechanism (see Chapter 25), producing vasoconstriction and a fall in GFR. Increased aldosterone production causes salt and water retention, which further contributes to oedema. As the GFR falls, filtration and elimination of nitrogenous wastes, including urea, decrease. Oliguria, urine output of less than 400 mL in 24 hours, may result from the decreased GFR. Hypertension results from fluid retention and disruption of the renin–angiotensin system, a key regulator of blood pressure.

Table 27.6 outlines common pathological processes causing CKD that lead to ESKD in Australia.

The course of CKD is variable, progressing over a period of months to many years. In stage 1 the GFR remains essentially normal or slightly elevated as a result of a hyperperfusive state whereby unaffected nephrons compensate for the lost nephrons. As the disease progresses to stage 2 the GFR falls to between 89 and 60 mL/min and the individual remains asymptomatic. Any further insult to the kidneys at this stage (such as infection, dehydration, exposure to nephrotoxins or urinary tract obstruction) can further reduce function and precipitate the onset of an acute decline in renal function. Stage 3 is characterised by a moderate decrease in renal function with a GFR of between 59 and 30 mL/min. The serum creatinine and urea may begin to rise and the person may begin to notice some feelings of tiredness, itchiness or loss of appetite, but it is not unusual to remain asymptomatic. Stage 4 sees a severe decrease in renal function with the GFR declining to between 28 and 15 mL/min. Symptomology of this decreased function, including increased tiredness, shortness of breath and general malaise, may be enough to prompt the individual to schedule a visit to their general practitioner if they have not already been diagnosed with CKD. In end-stage kidney disease, the final stage of CKD, the GFR is less than 15 mL/min and kidney replacement therapy is necessary to sustain life (see Figure 27.3).

Diabetic nephropathy

Diabetic nephropathy is one of the main microvascular complications of diabetes mellitus (DM) which sees the combination of haemodynamic, genetic and metabolic sequelae contribute to the development of increased vascular permeability at the glomerular basement membrane. Diabetic nephropathy is the leading cause of ESKD in Australia and New Zealand, with type 2 representing the majority of diabetic nephropathy cases requiring kidney replacement therapy (ANZDATA Registry, 2015). ESKD resulting from diabetic nephropathy is seen more frequently in Indigenous populations and those with type 2 DM due to a higher prevalence of type 2 DM in the community.

The characteristic lesion of diabetic nephropathy is glomerulosclerosis and thickening of the glomerular basement membrane. Hyperglycaemia impairs the ability of afferent and efferent arterioles and glomerular capillaries to regulate glomerular pressure and respond to systemic changes in blood pressure. As the disease progresses, the glomerular capillary lumen narrows, reducing the surface area for glomerular filtration. Arteriosclerosis, a common feature of long-term diabetes and hypertension, contributes to the disease process, as do nephritis and tubular lesions. Pyelonephritis, inflammation of the kidney, is also implicated in the development of diabetic nephropathy. A further discussion is found in Chapter 19.

Diabetic nephropathy progresses from a stage of hyperfiltration to early stage glomerular damage signified by microalbuminuria, which is typically seen within 10 to 15 years after the

onset of diabetes. If this damage is not detected early or is poorly controlled it will lead to overt proteinuria (within 15 to 20 years of the initial diagnosis) and decreased GFR, and may also see the concomitant development of hypertension. Eventually ESKD will develop and the individual will require kidney replacement therapy to sustain life.

Glomerulonephritis

Glomerulonephritis (GN) affects both the structure and function of the glomerulus, disrupting glomerular filtration, and is the second leading cause of ESKD in Australia (ANZDATA Registry, 2015). Histologically, glomerular involvement may be diffuse, involving all glomeruli, or focal, involving some glomeruli while others remain essentially normal. GN can present as either an acute or chronic condition and describes any disease that is mediated by either an inflammatory or immune mechanism that affects the glomerulus. Circulating antigen–antibody immune complexes formed during the initiating event become trapped in the glomerular membrane, leading to an inflammatory response. The complement system is activated and vasoactive substances and inflammatory mediators are released. Endothelial cells proliferate and the glomerular membrane swells and becomes permeable to plasma proteins and blood cells (see Figure 27.4). This increased permeability causes the manifestations common to glomerular disorders: haematuria, proteinuria and oedema.

The taxonomy of GN sometimes causes confusion as it attempts to classify the histopathological characteristics of the disease (e.g. focal segmental GN), the clinical presentation (e.g. nephrotic syndrome) or the aetiology of the disease (e.g. autoimmune) (Chadban & Atkins, 2005). From an aetiological perspective, GN is described as either primary, where the disease is immunological or idiopathic in origin, or secondary, where it is associated with a multisystem disease or hereditary condition. Systemic lupus erythematosus (SLE) and Goodpasture's syndrome are frequently implicated in secondary glomerular disorders. The most appropriate way to classify GN and avoid any potential confusion is according to its clinical presentation (Chadban & Atkins, 2005). In this way the treating team is able to identify the priorities for management.

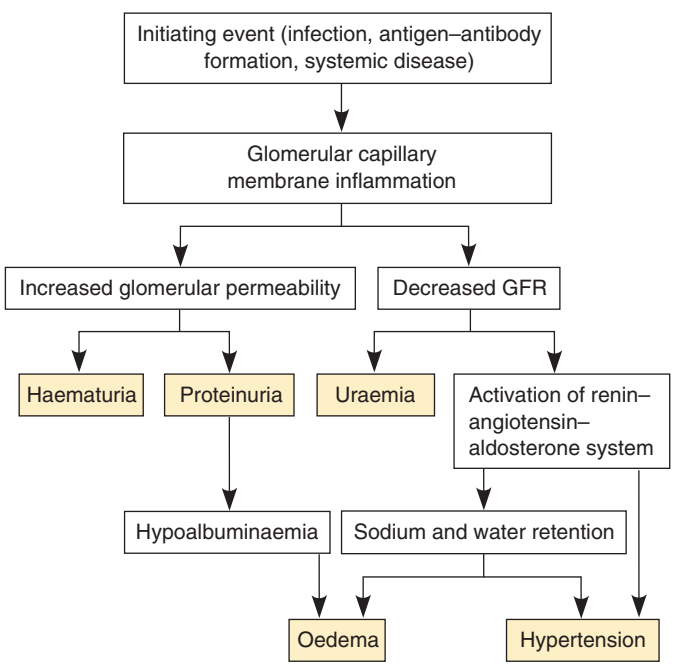


FIGURE 27.4 ■ The pathogenesis of glomerulonephritis

FAST FACTS

- Glomerulonephritis is the second leading cause of CKD in Australia.
- Haematuria and microalbuminuria are early manifestations of CKD.
- IgA mesangioproliferative GN is the most common histologically proven form of glomerulonephritis in Australia, followed by focal sclerosing GN.

In Australia, the major primary glomerular disorders include IgA mesangioproliferative GN, rapidly progressive glomerulonephritis, focal sclerosing glomerulonephritis and nephrotic syndrome (ANZDATA Registry, 2015). Goodpasture's syndrome and lupus nephritis are the most common secondary forms of glomerular disease.

IGA MESANGIOPROLIFERATIVE GLOMERULONEPHRITIS IgA mesangioproliferative glomerulonephritis, or IgA nephropathy (IgAN) as it is more commonly known, is characterised by the deposition of the antibody immunoglobulin A in the glomerular mesangium. IgAN has a highly variable course which can range from a benign condition to either a chronically slow or rapidly progressive disease. Children and young adults generally present with haematuria post respiratory tract infection or gastrointestinal illness; adults present later in the disease trajectory with proteinuria, haematuria and hypertension (Wyatt & Julian, 2013). Renal biopsy is diagnostic of IgAN and therefore early biopsy is essential in order to implement appropriate treatment to retard the progression of the disease.

RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS *Rapidly progressive glomerulonephritis (RPGN)* is characterised by manifestations of severe glomerular injury without a specific, identifiable cause. This type of glomerulonephritis often progresses to renal failure within months. It may be idiopathic (primary) or secondary to a systemic disorder such as SLE or Goodpasture's syndrome. It affects people of all ages.

In RPGN, glomerular cells proliferate and, together with macrophages, form crescent-shaped lesions that obliterate Bowman's space (Porth, 2011). Glomerular damage is diffuse, leading to a rapid, progressive decline in renal function. Irreversible renal failure often develops over weeks to months (Longo et al., 2012).

People with RPGN typically present with complaints of weakness, nausea and vomiting. Some may relate a history of flu-like illness preceding the onset of the glomerulonephritis. Other symptoms include oliguria and abdominal or flank pain. On urinalysis, haematuria and massive proteinuria are noted. People with RPGN are often treated with plasma exchange as an adjunct to immunosuppressive therapy (see Box 27.2).

BOX 27.2 Therapeutic plasma exchange

Therapeutic plasma exchange (**plasmapheresis**), a procedure performed by highly trained nurses to remove disease-mediating antibodies from the plasma, is used in conjunction with immunosuppressive therapy to treat RPGN and Goodpasture's syndrome. Blood is removed from the person via a large vein or central venous device and then the plasma containing the glomerular-damaging antibodies is separated and discarded using either centrifugal or membrane separation technologies. The RBCs are then returned to the person along with albumin or human plasma to replace the plasma removed. This procedure is usually done in a series of treatments. It is not without risk and informed consent is required. Procedural complications are mainly associated with the type of technology used to achieve plasma separation. The most common complications are transfusion reactions, citrate toxicity (with centrifugal methods), altered coagulation status, vascular access infection and hypotension secondary to fluid volume shifts.

Nephrotic syndrome

Nephrotic syndrome is a group of clinical findings as opposed to a specific disorder. It is characterised by massive proteinuria, hypoalbuminaemia, hyperlipidaemia and oedema. A number of disorders can affect the glomerular capillary membrane, changing its porosity and allowing plasma proteins to escape into the urine.

With plasma protein loss in the urine and resulting hypoalbuminaemia, the oncotic pressure of the plasma falls. Fluid shifts from the vascular compartment to interstitial spaces, causing the oedema characteristic of nephrotic syndrome. Salt and water retention, possibly due to activation of the renin–angiotensin system, contribute to the oedema. Oedema may be severe, affecting the face and periorbital area as well as dependent tissues (see Figure 27.5).

Loss of plasma proteins stimulates the liver to increase albumin production and lipoprotein synthesis. As a result, serum triglyceride and low-density lipoprotein (LDL) levels increase, as do urine lipids (*lipiduria*). Hyperlipidaemia increases the risk of atherosclerosis in people with nephrotic syndrome.

Thromboemboli (mobilised blood clots) are a relatively common complication of nephrotic syndrome. Loss of clotting and anti-clotting factors along with plasma proteins disrupts coagulation pathways and increases the risk of renal venous thrombosis, deep venous thrombosis and pulmonary embolism. Renal venous thrombosis can cause flank or groin pain on one or both sides, gross haematuria and a reduced GFR (Porth, 2011).

Nephrotic syndrome usually resolves without long-term effects in children. The prognosis for adults is less optimistic because the syndrome often occurs secondarily to another disorder. Many adults do not recover completely, experiencing persistent proteinuria and, potentially, progressive renal impairment.

Goodpasture's syndrome

Goodpasture's syndrome is a rare autoimmune disorder of unknown aetiology. It is characterised by formation of antibodies to the glomerular basement membrane. These antibodies may also bind to alveolar basement membranes, damaging alveoli and causing pulmonary haemorrhage. Goodpasture's syndrome usually affects young men aged between 18 and 35, although it can occur at any age and affect women as well.

FIGURE 27.5 ■ Severe oedema characteristic of nephrotic syndrome

Source: © MedicImage/Alamy Stock Photo.

Although the glomeruli may be nearly normal in appearance and function in Goodpasture's syndrome, extensive cell proliferation and crescent formation characteristic of rapidly progressive glomerulonephritis are more common. Renal manifestations include haematuria, proteinuria and oedema. Rapid progression through the CKD continuum may occur. Alveolar membrane damage can lead to mild or life-threatening pulmonary haemorrhage. Cough, shortness of breath and haemoptysis (bloody sputum) are early respiratory manifestations.

Lupus nephritis

Systemic lupus erythematosus (SLE) is an inflammatory autoimmune disorder affecting the connective tissue of the body. Between 60% and 80% of people with SLE develop manifestations of nephritis (Longo et al., 2012). Immune complexes that form within the glomerular capillary wall are the usual trigger for glomerular injury in SLE. Manifestations of lupus nephritis range from microscopic haematuria to massive proteinuria. Its progression may be slow and chronic or *fulminant*, with a sudden onset and the rapid development of renal disease. Most people with minimal or mild lesions survive for at least 10 years. Improved management of the underlying disease, immunotherapy, dialysis and renal transplantation have significantly improved the prognosis in recent years.

THE PERSON WITH A RENOVASCULAR DISORDER

Renal function is dependent on an adequate supply of blood. Blood supports renal cell metabolism and is vital to kidney function, the nephron in particular. The kidney can regulate fluid, electrolyte and acid–base balance, and serve as a major organ of excretion, only when its blood supply is sufficient. Vascular disorders, therefore, can have a significant impact on renal function.

Hypertension

Hypertension, sustained elevation of the systemic blood pressure, can result from or cause kidney disease. In 2013, it was the cause of 14% of all cases of end-stage kidney disease in Australia (ANZDATA Registry, 2015).

Prolonged hypertension damages the walls of arterioles and accelerates the process of atherosclerosis. This damage primarily affects the heart, brain, kidneys, eyes and major blood vessels. In the kidney, arteriosclerotic lesions develop in the *afferent* (leading into) and *efferent* (going out of) arterioles and the glomerular capillaries. The glomerular filtration rate declines and tubular function is affected, resulting in proteinuria and microscopic haematuria.

Malignant hypertension is a rapidly progressive form of hypertension that can develop in people with untreated primary hypertension. Here, the diastolic pressure is in excess of 120 mmHg and may be as high as 150 to 170 mmHg. Malignant hypertension affects less than 1% of people with hypertension; it is more common in African Americans than in people of European ancestry. Untreated, malignant hypertension causes a rapid decline in renal function due to vessel changes, renal ischaemia and infarction.

Approximately 5–10% of hypertension is a manifestation of an underlying disease and is termed 'secondary hypertension'. Renal vascular disease and diseases of the renal parenchyma, such as diabetic nephropathy, are commonly associated with secondary hypertension. Ensuring blood pressure lies within normal limits is vital to prevent kidney damage. When hypertension is secondary to kidney disease, adequate blood pressure control is an integral step in attempting to retard the decline in kidney function. Hypertension and its management are discussed in depth in Chapter 31.

Renal artery occlusion

Renal arteries can be occluded by either a primary process affecting the renal vessels or by emboli, clots or other foreign material. Risk factors for acute renal artery thrombosis (formation of a blood clot in the renal artery) include severe abdominal trauma, vessel trauma from surgery or angiography, aortic or renal artery aneurysms, and severe aortic or renal artery atherosclerosis. Emboli from the left side of the heart can travel via the aorta to occlude the renal artery. Emboli may form as a result of atrial fibrillation (irregular and uncoordinated electrical activity of the atria), following myocardial infarction, as vegetative growths on heart valves associated with bacterial endocarditis or from fatty plaque in the aorta.

Renal arterial occlusion may be asymptomatic when the occlusion develops slowly and the affected vessels are small. Acute occlusion leading to ischaemia and infarction typically causes sudden, severe localised flank pain, nausea and vomiting, fever and hypertension. Haematuria and oliguria may occur. In the older person, the new onset of hypertension or worsening of previously controlled hypertension may signal renal artery thrombosis.

Laboratory studies reveal leucocytosis (elevated WBCs) and elevated renal enzyme levels, including aspartate transaminase (AST) and lactic dehydrogenase (LDH). These enzymes, normally present in renal cells, are released into the circulation when cells necrose and die. With bilateral arterial occlusion and infarction, renal function deteriorates rapidly, leading to acute kidney injury (Longo et al., 2012).

Surgery to restore blood flow to the affected kidney may be indicated for acute occlusion. Management usually is more conservative, using anticoagulant therapy, hypertension control and supportive treatment.

Renal vein occlusion

A thrombus (clot) formed in a renal vein can occlude the vessel. The cause of the thrombus often is unclear. In adults, renal venous thrombosis usually occurs with nephrotic syndrome. Other predisposing factors include pregnancy, oral contraceptive use and certain malignancies.

Gradual or acute deterioration of renal function may be the only manifestation of renal vein occlusion. If the thrombus breaks loose, it can become a pulmonary embolism. The definitive diagnosis is made by visualising the thrombus through renal venography.

Thrombolytic drugs such as streptokinase or tissue plasminogen activator (tPA) may be given to dissolve or break up the thrombus. Anticoagulant therapy also is used to prevent further clotting and pulmonary emboli. Renal function often improves with treatment.

Renal artery stenosis

Renal artery stenosis is narrowing of the artery that supplies blood to the kidney; it can be uni- or bilateral. Quite often a person can go through life with unilateral renal artery stenosis undetected; however, this is not always the case.

Atherosclerosis with gradual occlusion of the renal artery lumen by plaque is the primary cause of renal artery stenosis in men. In younger women, the most common cause is fibromuscular dysplasia, structural abnormalities involving the intimal, medial or adventitial layers of the arterial wall.

Renal artery stenosis is suspected when hypertension develops before age 30 or after age 50 with no prior history of high blood pressure. An epigastric bruit (murmur) and other manifestations of vascular insufficiency may also be present. The affected kidney appears small and atrophied on renal ultrasound. Renal angiography uses radiological contrast dye injected into the renal arteries to allow visualisation of renal blood vessels.

Polycystic kidney disease

Polycystic kidney disease (PKD), a hereditary disease characterised by cyst formation and massive kidney enlargement, affects both children and adults. This disease has two forms: the autosomal dominant form, which affects adults; and the autosomal recessive form, which is present at birth (Porth, 2011). Autosomal recessive PKD is rare. It usually is diagnosed prenatally or in infancy. Autosomal dominant PKD is relatively common and accounts for approximately 6% of

people with ESKD in Australia (ANZDATA Registry, 2015). This section focuses on autosomal dominant PKD, the more common adult form of the disorder.

Pathophysiology

Renal cysts are fluid-filled sacs affecting the nephron, the functional unit of the kidneys. The cysts may range in size from microscopic to several centimetres in diameter and affect the renal cortex and medulla of both kidneys. As the cysts fill, enlarge and multiply, the kidneys also enlarge. Renal blood vessels and nephrons are compressed and obstructed and functional tissue is destroyed (see Figure 27.6).

Although the name suggests the disease affects the kidneys, polycystic kidney disease is actually a systemic disease process and manifests with both renal and non-renal abnormalities. Often cysts develop elsewhere in the body, including the liver, spleen, pancreas and other organs. Diverticular disease of the colon is common and may lead to perforation of the bowel (Longo et al., 2012). Up to 10% of people affected experience subarachnoid haemorrhage from a type of congenital intracranial aneurysm.

Manifestations

Polycystic kidney disease is slowly progressive. Symptoms usually develop by age 40 to 50. Common manifestations include flank pain, microscopic or gross haematuria, proteinuria, and *polyuria* and *nocturia*, as the concentrating ability of the kidney is impaired. Urinary tract infection and renal calculi are common, as cysts interfere with normal urine drainage. Most people will develop hypertension from disruption of renal vessels. The kidneys become palpable, enlarged and knobby. Symptoms of renal insufficiency typically develop in approximately 60% of people by the age of 70. The progression to ESKD tends to occur more rapidly in men than in women.

FIGURE 27.6 ■ A polycystic kidney. The functional tissue of the kidneys is gradually destroyed and replaced with fluid-filled cysts

Source: © Arthur Glauberman/Getty Images.

GENETIC CONSIDERATIONS
Adult polycystic kidney disease

- In approximately 50% of cases, parents with autosomal dominant PKD will pass on the faulty gene to their offspring.
- Approximately 90% of cases are inherited as an autosomal dominant trait; the remaining 10% are due to spontaneous mutations.
- In autosomal recessive PKD, both parents must carry the defective gene and have a 25% chance of their child inheriting this disease.

MANIFESTATIONS AND COMPLICATIONS OF CKD

Uraemia

Uraemia, which literally means 'urine in the blood', refers to a group of symptoms associated with the decline in renal function generally in the final stages of the disease trajectory. In uraemia, fluid and electrolyte balance is altered, the regulatory and endocrine functions of the kidney are impaired, and accumulated metabolic waste products affect essentially every other organ system (Longo et al., 2012; Porth, 2011).

Early manifestations of uraemia include nausea, apathy, weakness and fatigue, symptoms that may be dismissed as a viral infection or just being run down. The multisystem effects of uraemia are illustrated below.

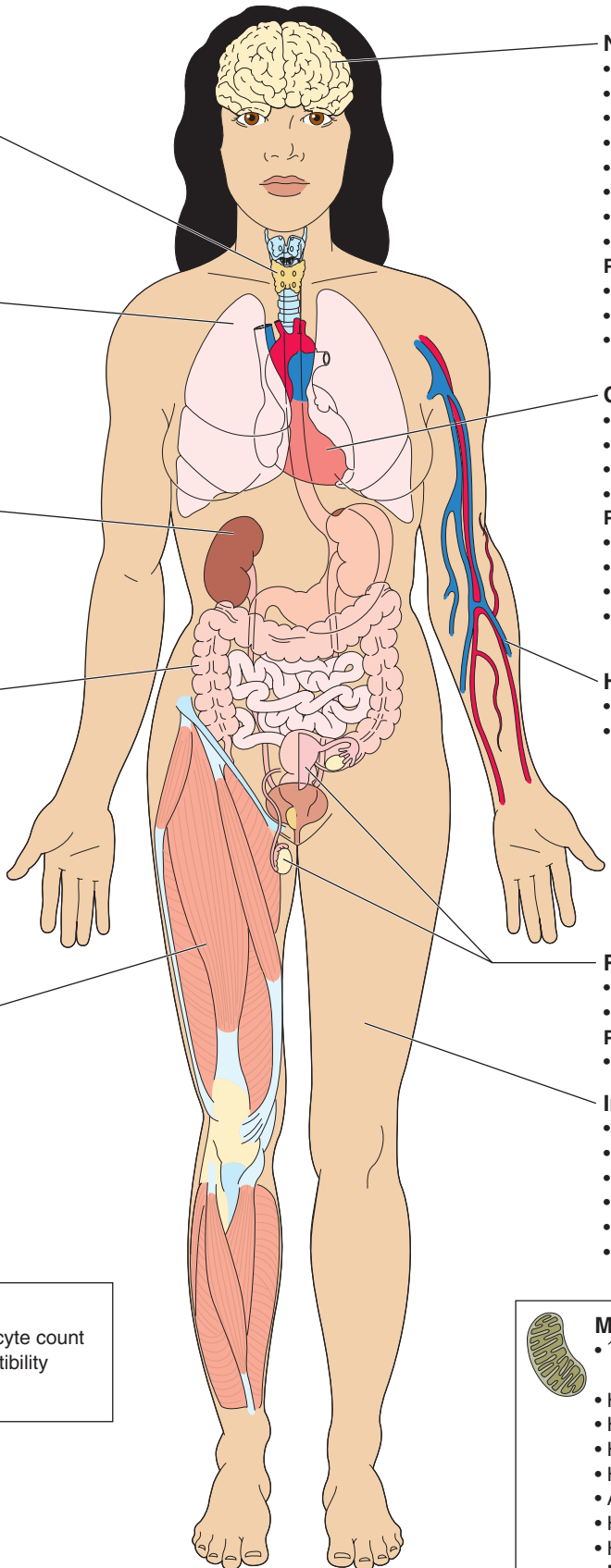
Fluid and electrolyte effects

Loss of kidney function impairs its ability to regulate fluid, electrolyte and acid–base balance. In the early stages of CKD, impaired filtration and reabsorption lead to proteinuria, haematuria and decreased urine-concentrating ability. Salt and water are poorly conserved and the risk of dehydration increases. Polyuria, nocturia and a fixed specific gravity of 1.008 to 1.012 are common (Porth, 2011). As the GFR decreases and renal function deteriorates further, sodium and water retention are common, necessitating salt and water restrictions.

Hyperkalaemia develops as kidney disease progresses. Manifestations of hyperkalaemia, such as muscle weakness, paraesthesias and ECG changes, are not usually seen until the GFR is less than 5–10 mL/min. Phosphate excretion is also impaired, leading to hyperphosphataemia and hypocalcaemia. Reduced calcium absorption due to impaired vitamin D activation also contributes to hypocalcaemia. Hypermagnesaemia develops with advancing kidney disease; magnesium-containing antacids are avoided for this reason.

As kidney disease advances, hydrogen-ion excretion and buffer production are impaired, leading to metabolic acidosis. Respiratory rate and depth increase (Kussmaul's respirations) to compensate for metabolic acidosis. Although metabolic acidosis is often asymptomatic, other possible manifestations include general malaise, weakness, headache, nausea and vomiting, and abdominal pain (see Chapter 9).

MULTISYSTEM EFFECTS OF URAEMIA



Endocrine
- Hyperparathyroidism
- Glucose intolerance

Respiratory
- Pulmonary oedema
- Pleuritis
- Kussmaul's respirations

Urinary
- Proteinuria
- Haematuria
- Fixed specific gravity
- Nocturia
- Oliguria, anuria

Gastrointestinal
- Anorexia
- Nausea and vomiting
- Gastroenteritis
- Hiccups
- Abdominal pain
- Uremic fetor

Potential complications
- Peptic ulcer
- GI bleeding

Musculoskeletal
- Osteodystrophy
- Bone pain
- Spontaneous fractures

Neurological
- Apathy
- Lethargy
- Headache
- Impaired cognition
- Insomnia
- Restless leg syndrome
- Gait disturbances
- Paraesthesias

Potential complications
- Seizures
- Decreased LOC
- Coma

Cardiovascular
- Hypertension
- Oedema
- Coronary heart disease
- Arrhythmias

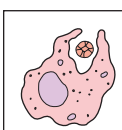
Potential complications
- Pericarditis
- Pericardial effusion
- Cerebrovascular disease
- Heart failure

Hematological
- Anaemias
- Impaired clotting

Reproductive
- Amenorrhoea (female)
- Impotence (male)

Potential complication
- Spontaneous abortion

Integumentary
- Pallor
- Uraemic skin colour (yellow-green)
- Dry skin, poor turgor
- Pruritis
- Ecchymoses
- Uraemic 'frost'



Immune system
- Diminished leucocyte count
- Increased susceptibility to infection

Metabolic processes
- ↑ urea and serum creatinine
- Hyperkalaemia
- Hyperphosphataemia
- Hypocalcaemia
- Hypermagnesia
- Acidosis
- Hyperlipidaemia
- Hyperuricaemia
- Malnutrition

Cardiovascular effects

In 2013, cardiovascular causes were attributed as the leading cause of deaths in dialysis-dependent people in Australia and New Zealand (ANZDATA Registry, 2015). A combination of malnutrition, inflammatory processes, atherosclerosis, hypertension, hyperlipidaemia and glucose intolerance all contribute to the increased cardiovascular burden on people with kidney disease (Tonbul et al., 2006). Cerebral and peripheral vascular manifestations of atherosclerosis are also seen.

Systemic hypertension is a common complication of ESKD. Hypertension results from excess fluid volume, increased renin–angiotensin activity, increased peripheral vascular resistance and decreased prostaglandins. Increased extracellular fluid volume also can lead to oedema and heart failure. Pulmonary oedema may result from heart failure and increased permeability of the alveolar capillary membrane.

Retained metabolic toxins can irritate the pericardial sac, causing an inflammatory response and signs of pericarditis. *Cardiac tamponade*, a potential complication of pericarditis, occurs when inflammatory fluid in the pericardial sac interferes with ventricular filling and cardiac output.

Haematological effects

Anaemia is a common sequela in CKD and its cause is multifactorial. The kidneys produce erythropoietin, a hormone that controls RBC production. In kidney disease, erythropoietin production declines. Retained metabolic toxins further suppress RBC production and contribute to a shortened RBC lifespan. Nutritional deficiencies (iron and folate) and increased risk of blood loss from the GI tract also contribute to anaemia. (See Chapter 32 for more information on anaemia.)

Anaemia contributes to symptoms such as fatigue, weakness, depression and impaired cognition. It also affects cardiovascular function, as the heart's natural response to anaemia is to increase cardiac output which leads to increased cardiac remodelling and left ventricular hypertrophy. As such, anaemia is associated with an increased risk of cardiovascular disease.

People with kidney disease are at increased risk of bleeding disorders such as ecchymoses, epistaxis and GI bleeding. The combination of anaemia, platelet abnormalities, drug interactions, impaired platelet–vessel wall interaction and circulating uraemic toxins in the blood are all associated with this increased risk.

Immune system effects

Uraemia increases the risk of infection. High levels of urea and retained metabolic wastes impair all aspects of inflammation and immune function. The WBC declines, humoral and cell-mediated immunity are impaired and phagocyte function is defective. Both the acute inflammatory response and delayed hypersensitivity responses are affected (Porth, 2011). Fever is suppressed, often delaying the diagnosis of infection.

Gastrointestinal effects

Anorexia, nausea and vomiting are the most common early symptoms of uraemia. Hiccups also are commonly experienced. Gastroenteritis is frequent. Ulcerations may affect any level of the GI tract and contribute to an increased risk of GI bleeding. Peptic ulcer disease is particularly common in people with uraemia *Uraemic fetor*, a urine-like breath odour often associated with a metallic taste in the mouth, may develop. Uraemic fetor can further contribute to anorexia.

Neurological effects

Uraemia alters both central and peripheral nervous system function. CNS manifestations occur early and include changes in mentation, difficulty concentrating, fatigue and insomnia. Psychotic symptoms, seizures and coma are associated with advanced uraemic encephalopathy.

Peripheral neuropathy is also common in advanced uraemia. Both the sensory and motor tracts are involved. The lower limbs are initially affected. 'Restless leg syndrome', sensations of crawling or creeping, prickling or itching of the lower legs with frequent leg movement, increases during rest. Paraesthesias and sensory loss typically occur in a 'stocking-glove' pattern. As uraemia progresses, motor function is also impaired, causing muscle weakness, decreased deep tendon reflexes and gait disturbances.

Musculoskeletal effects

Hyperphosphataemia and hypocalcaemia associated with uraemia stimulate parathyroid hormone secretion. Parathyroid hormone causes increased calcium resorption from bone. In addition, osteoblast (bone-forming) and osteoclast (bone-destructing) cell activity is affected. This bone resorption and remodelling, combined with decreased vitamin D synthesis and decreased calcium absorption from the GI tract, lead to *renal osteodystrophy*. Osteodystrophy is characterised by oste*omalacia,* softening of the bones, and *osteoporosis*, decreased bone mass. Bone cysts may develop. Manifestations of osteodystrophy include bone tenderness, pain and muscle weakness, which all increase the risk of spontaneous fractures (Porth, 2011).

Endocrine and metabolic effects

Accumulated waste products of protein metabolism are a primary factor involved in the effects and manifestations of uraemia. Serum creatinine and urea levels are significantly elevated. Uric acid levels are increased, contributing to an increased risk of gout.

Tissues become resistant to the effects of insulin in uraemia, leading to glucose intolerance. High blood triglyceride levels and lower than normal high-density lipoprotein (HDL) levels contribute to the accelerated atherosclerotic process.

Reproductive function is affected. Pregnancies are rarely carried to term and menstrual irregularities are common. Reduced testosterone levels, low sperm counts and impotence affect men with ESKD.

Dermatological effects

Anaemia and retained pigmented metabolites cause pallor and a yellowish hue to the skin in uraemia. Dry skin with poor turgor, a result of dehydration and sweat gland atrophy, is common. Bruising and excoriations are frequently seen. Metabolic wastes not eliminated by the kidneys may be deposited in the skin, contributing to itching or pruritus. In advanced uraemia, high levels of urea in the sweat may result in *uraemic frost*, crystallised deposits of urea on the skin.

INTERPROFESSIONAL CARE

Management of chronic kidney disease regardless of its aetiology focuses on identifying and slowing the progression of the underlying disease process, preserving renal function, preventing and managing complications, and providing psychological support. This aim is achieved through the collaboration of all members of the healthcare team.

Diagnostics

Laboratory and diagnostic testing are valuable to identify the cause of CKD, monitor kidney function and evaluate the efficacy of treatment. A number of tests and procedures may be performed to determine the underlying renal disease. (See Table 27.7 for normal laboratory values for commonly requested blood and urine tests.) Once the diagnosis is established, renal function is monitored primarily through blood levels of metabolic wastes and electrolytes. Imaging investigations provide information about the size and shape of the kidneys, possible tumours or cysts, the nature of the renal parenchyma and the presence of any obstruction. See Chapter 25 for the nursing implications of selected tests.

The following studies may be ordered to help identify the underlying cause or aetiology:

- *eGFR* provides an estimation of the glomerular filtration rate. eGFR is used to evaluate renal function and may be reduced despite serum creatinine levels being in the normal range.
- *Urea* is the end product of protein metabolism. It is created by the breakdown and metabolism of both dietary and body proteins. Urea is eliminated from the body by filtration in the glomerulus; minimal amounts are reabsorbed in the renal tubules. Glomerular diseases interfere with filtration and elimination of urea nitrogen, causing blood levels to rise. Increased protein catabolism (destruction), which may occur with GI bleeding or tissue breakdown, can also raise the urea level.
- *Serum creatinine* measures the amount of creatinine in the blood. Creatinine is the metabolic by-product of muscle metabolism and is produced in relatively constant amounts by skeletal muscles. It is excreted entirely by the kidneys, making serum creatinine a good indicator of kidney function. Normal values are lower in the older adult because of decreased muscle mass.
- *Serum electrolytes* are monitored throughout the course of CKD because impaired kidney function alters their excretion. The serum sodium may be within normal limits or low because of water retention. Potassium levels are elevated but usually remain below 6.5 mmol/L. Serum phosphate is elevated and the calcium level is decreased. Metabolic acidosis is identified by a low pH, low CO_2 and low bicarbonate levels.
- *Full blood count (FBC)* provides information about the degree of anaemia, with most people with stage 4 and 5 CKD

TABLE 27.7 Changes in laboratory values associated with kidney disease

TEST	NORMAL VALUE	ASSOCIATED WITH KIDNEY DISEASE
Urea, serum	3.5–7.2 mmol/L Slightly higher in older adults	Elevated in hypovolaemia, AKI, later stages of CKD Decreased in hypervolaemia and malnutrition
Creatinine, serum	Female: 50–110 µmol/L Male: 60–120 µmol/L Slightly lower in older adults	Elevated in hypovolaemia, AKI, later stages of CKD
eGFR	> 90 mL/min (young adults) eGFR declines naturally with age	See Figure 27.3 eGFR is not validated for use in children, Indigenous populations or AKI
Serum albumin	33–41 g/L	Decreased in nephrotic syndrome
Serum electrolytes	Potassium 3.7–5.2 mmol/L Sodium 136–144 mmol/L Calcium 2.18–2.49 mmol/L Ionised calcium 1.16–1.30 mmol/L Phosphorus 0.88–1.46 mmol/L	Increased in renal insufficiency Decreased in nephrotic syndrome Decreased in later stages of CKD and malabsorption Elevated in hypocalcaemia and later stages of CKD
Haemoglobin	115–165 g/L	Decreased in later stages of CKD. Target concentration of 110 g/L for stage 4 and 5 CKD/ESKD
Red blood cell count	Female: 3.8–5.8 x 10^{12}/L Male: 4.5–6.5 x 10^{12} /L	Decreased in later stages of CKD
Urinary ACR	Female: 3.5–35 mg/mmol Male: 2.6–25 mg/mmol Female: > 35 mg/mmol Male: > 25 mg/mmol	Microalbuminuria Macroalbuminuria
Urinary PCR	> 30 mg/mmol	Proteinuria
Urine red blood cells	< 2–3/HPF; no RBC casts	Present in glomerular disorders

showing decreased haematocrit, haemoglobin, RBCs and platelets.

- *Erythrocyte sedimentation rate (ESR)* is a general indicator of inflammatory response. It is generally elevated in conditionals such as vasculitis, acute post-streptococcal glomerulonephritis and lupus nephritis.
- *C-reactive protein (CRP)* is a non-specific inflammatory marker and is used to indicate the presence of inflammatory states. It may be elevated in infective processes and in lupus nephritis.
- *Urinalysis* often reveals red blood cells (RBCs) and proteins in the urine of people with CKD. These substances, which normally are too large to enter glomerular filtrate, escape due to the increased permeability of the glomerular basement membrane.

In CKD, urinary specific gravity may be fixed at approximately 1.010, equivalent to that of plasma. This fixed specific gravity is due to impaired tubular secretion, reabsorption and urine concentrating ability. Abnormal proteins (e.g. Bence-Jones proteins), blood cells and cellular casts may also be noted in the urine.

- *Albumin:creatinine (ACR)* and *protein:creatinine ratio (PCR)* evaluates the urinary excretion of either albumin or protein and is considered best practice in the primary screening of CKD.
- *24-hour timed urine* enables the measurement of urinary protein and/or creatinine excretion over a 24-hour time period. Urine creatinine levels decrease when renal function is impaired because it is not effectively eliminated from the body.
- *Urine microbial culture and sensitivity* may be ordered to identify any urinary tract infection that may hasten the progress of CKD.
- *Throat or skin cultures* detect infection by group A beta-haemolytic *Streptococcus.* Although post-streptococcal glomerulonephritis typically follows the acute infection by 1 to 2 weeks, treatment to eradicate any remaining organisms is initiated to minimise antibody production.
- *Antistreptolysin O (ASO) titre* and other tests detect streptococcal exoenzymes (bacterial enzymes that stimulate the immune response in acute post-streptococcal glomerulonephritis). Other titres such as antistreptokinase (ASK) or antideoxyribonuclease B (ADNAase B) may be obtained as well.
- *Kidney biopsy*, microscopic examination of kidney tissue, assists in determining the cause of CKD, its prognosis and appropriate treatment. It is usually done percutaneously, by inserting a biopsy needle through the skin into the kidney to obtain a tissue sample. Open biopsy, which requires surgery, may also be done. Biopsy is not justified when clinical evidence makes diagnosis almost certain—for example, polycystic kidney disease. Other contraindications include small kidneys, single kidney, uncontrolled blood pressure and/or uraemia.
- *Kidney, ureter, bladder (KUB) ultrasound* is considered a first-line imaging technique and is used to evaluate kidney size and identify potential obstructions. It is a useful non-invasive technique to differentiate acute kidney injury from chronic kidney disease. In AKI, the kidneys may be enlarged, whereas they typically appear small and shrunken with decreased cortical width in CKD. Ultrasound can also detect renal masses and differentiate polycystic kidney disease from renal carcinoma.
- *Kidney scan*, a nuclear medicine procedure, allows visualisation and functional assessment of the kidneys after intravenous administration of a radioisotope.
- *Intravenous pyelogram (IVP)*, *retrograde pyelography* or *antegrade pyelography* may also be used to evaluate kidney structure and function. Radiological contrast media are used with extreme caution because of their potential nephrotoxicity. Retrograde pyelography, in which contrast dye is injected into the ureters, and antegrade pyelography, in which the contrast medium is injected percutaneously into the renal pelvis, are preferred because they have fewer nephrotoxic effects than IVP.
- *Renal angiography* is used to investigate and evaluate renal blood flow and the major vasculature of the kidney and affords the opportunity for simultaneous interventional procedures such as stenting or angioplasty.
- *Computed tomography (CT) scan* is used to detect and differentiate renal masses such as cystic disease or tumours. CT can determine tumour density, local extension of the tumour and any regional lymph node or vascular involvement.
- *Bone density scan* is used to assess bone mineral density; see Chapter 37 for more information.

See Chapter 25 for nursing implications of tests used in renal medicine.

CONSIDERATION FOR PRACTICE

People with pre-existing kidney disease, volume depletion or advanced age are at high risk of contrast nephropathy if exposed to radiocontrast. Alert the treating team prior to any imaging procedure that requires radiocontrast if person is in high-risk category. Preventative methods surround adequate hydration. If not contraindicated, ensure the person remains well hydrated pre and post procedure.

Medications

Chronic kidney disease affects both the pharmacokinetic and pharmacodynamic effects of drug therapy. Many medications are excreted primarily by the kidney. The half life and plasma levels of many drugs increase in chronic kidney disease. Drug absorption may be decreased when phosphate-binding agents are administered concurrently. Proteinuria can significantly reduce plasma protein levels, leading to toxicity when highly protein-bound drugs are given. In addition, any potentially nephrotoxic agent is avoided or used with extreme caution in people with CKD. Dose reductions may be required and/or dosing intervals are lengthened in CKD according to individual GFR.

As renal function declines, medications become an integral component of replacing diminished renal function. Medications are used to treat underlying disorders, reduce inflammatory processes, manage symptoms and attempt to retard the progression of CKD.

Diuretics such as frusemide may be prescribed to reduce extracellular fluid volume and oedema. Diuretic therapy can also reduce hypertension and lower potassium levels. Thiazides such as hydrochlorothiazide, which are commonly used in combination with some antihypertensive classes, are less effective as diuretics when the eGFR falls below 25–30 mL/min but still remain effective in reducing hypertension as they reduce blood pressure through a vasodilatory effect.

ANTIHYPERTENSIVE AGENTS Antihypertensive agents are used to maintain the blood pressure within normal levels, slow the progression of CKD and prevent complications of coronary heart disease and cerebral vascular disease. Angiotensin-converting-enzyme (ACE) inhibitors are preferred, although any class of antihypertensive agent may be prescribed (see Chapter 31). ACE inhibitors may be ordered to reduce protein loss associated with nephrotic syndrome. These drugs reduce proteinuria and slow the progression of renal disease in the kidney. Angiotensin receptor blockers such as irbesartan have also been shown to have a renoprotective benefit independent of blood pressure control, particularly in the reduction of microalbuminuria in people with diabetic nephropathy (Ravera et al., 2005).

DRUGS FOR ACID–BASE IMBALANCE AND ELECTROLYTE DISTURBANCES Other drugs may be used to manage electrolyte imbalances and acid–base imbalances. Sodium bicarbonate may be used to correct mild acidosis.

If the serum potassium rises to dangerously high levels, a combination of bicarbonate, insulin and glucose may be given intravenously to promote potassium movement into the cells. Sodium polystyrene sulfonate (Resonium), a potassium-ion exchange resin, can be given either orally or rectally (as an enema).

PHOSPHATE BINDERS AND AGENTS FOR RENAL OSTEODYSTROPHY Oral phosphate binding agents such as calcium carbonate are taken with food to lower serum phosphate levels and before or after food to normalise serum calcium levels. Aluminium hydroxide has a stronger phosphate binding potency but its use is limited by complications such as encephalopathy and osteodystrophy associated with long-term administration (McPhee, Papadakis & Tierney, 2008). Non-calcium phosphate binders such as sevelamer and lanthanum have the advantage that they are not themselves absorbed from the gut, so have less potential to cause problems with accumulation. They have shown benefits in reducing coronary calcification (Block et al., 2007). Vitamin D analogues such as calcitriol may be given to improve gastrointestinal calcium absorption and normalise calcium levels. Bisphosphonates such as alendronate are contraindicated when the eGFR falls below 35 mL/min and are also contraindicated in hypocalcaemia. Cinacalcet belongs to a drug class termed 'calcimimetic' as it imitates calcium at the parathyroid gland to decrease output of parathyroid hormone due to secondary hyperparathyroidism. This slows down excessive bone turnover and remodelling and lowers the chance of harmful metastatic calcification.

ERYTHROPOIETIN STIMULATING AGENTS AND MEDICATIONS USED IN ANAEMIA Folic acid and iron supplements are given to combat anaemia associated with chronic kidney disease due to impaired erythropoiesis and shortened red blood cell survival. A multivitamin preparation is also often prescribed because anorexia, nausea and dietary restrictions may limit nutrient intake. Recombinant human erythropoietin stimulates the proliferation and differentiation of erythroid progenitors, which in turn increase haemoglobin synthesis and accelerate the release of reticulocytes (young RBCs) from the bone marrow. There are five erythropoietin stimulating agents (ESAs) currently on the Australian market. These are epoetin alfa (Eprex), epoetin beta (NeoRecormon), epoetin lambda (Novicrit), methoxy polyethylene glycol-epoetin beta (Mircera) and darbepoetin (Aranesp). There is little difference between the agents in terms of efficacy and the choice is governed by factors such as frequency and route of administration.

ANTIBIOTICS Antibiotics, usually from the penicillin family, are prescribed for the person with post-streptococcal glomerulonephritis to eradicate any remaining bacteria, removing the stimulus for antibody production. Nephrotoxic antibiotics such as aminoglycosides are avoided but will be administered in cases where the benefit outweighs the risk. Both vancomycin and gentamicin are renally cleared so redosing is titrated according to serum levels.

ANALGESIA Non-steroidal anti-inflammatory medications such as ibuprofen are avoided due to their nephrotoxic nature. Pethidine is avoided altogether as its metabolite is renally cleared and accumulation will cause seizures. Morphine is also avoided due its metabolite, M6G, accumulating in kidney disease but may be given once or twice for acute pain episodes such as chest pain. Fentanyl and oxycodone are generally preferred due to the liver metabolising them into inactive metabolites.

IMMUNOSUPPRESSANTS Aggressive immunosuppressive therapy is used to treat acute inflammatory processes such as rapidly progressive glomerulonephritis, Goodpasture's syndrome and exacerbations of SLE. When begun early, immunosuppressive therapy significantly reduces the risk of end-stage kidney disease. Corticosteroids such as prednisolone are often used in large doses for several months, sometimes in combination with other immunosuppressants such as azathioprine and cyclophosphamide. Corticosteroids may also be used to induce remission of nephritic syndrome. However, in post-streptococcal glomerulonephritis they may actually worsen the condition so are usually avoided. See the transplant section of this chapter and Chapter 12 for more information about corticosteroids and other immunosuppressive drugs.

Nutrition and fluid management

As renal function declines, the elimination of water, solutes and metabolic wastes is impaired. Accumulation of these wastes in the body leads to uraemic symptoms. Instituted early in the course of CKD, dietary modifications can reduce uraemic symptoms and help prevent complications.

TABLE 27.8 Foods high in potassium or phosphate and some low-value alternatives

HIGH POTASSIUM	LOW POTASSIUM
Fruit and vegetable juices (except cranberry, lemon), bananas, apricots, plums, dried fruit, nuts and seeds, legumes, potatoes, avocados, tomatoes and tomato paste	Cranberry juice, apples, pears, blueberries, lychees, green beans, zucchinis, onions, strawberries, raspberries, lettuce, lemons
HIGH PHOSPHATE	**LOW PHOSPHATE**
Milk, eggs, cheese, meats, offal, poultry, fish, cola drinks and soy	Pasta, rice, corn cereals, popcorn, green beans, non-cola-based drinks and cordial

Unlike carbohydrates and fats, the body is unable to store excess proteins. Unused dietary proteins are degraded into urea and other nitrogenous wastes, which are then eliminated by the kidneys. Protein-rich foods also contain inorganic ions such as hydrogen ion, phosphate and sulfites that are eliminated by the kidneys. Consequently, dietary protein intake should be assessed by a renal dietitian with a bias towards optimising nutritional status.

A daily protein intake of 0.6 g/kg of ideal body weight provides the amino acids necessary for tissue repair in order to maintain body composition for people with CKD, provided the protein is of high biological value (Fouque & Mitch, 2014). Carbohydrate and fat intake may need to be increased to maintain ideal body weight and energy requirements, and is dependent on the rate of physical activity.

Water and sodium intake are regulated to maintain the extracellular fluid volume at normal levels. Water intake of 1000 to 2000 mL per day is generally recommended to maintain water balance. Sodium is restricted to 100 mmol or 2300 mg per day initially and equates to the equivalent of a 'no added salt' diet. More stringent water and sodium restrictions may be necessary as CKD progresses. The person is instructed to monitor weight daily and report any weight gain in excess of 2 kg over a 2-day period.

When the GFR falls to less than 10 to 20 mL/min, potassium and phosphorus intake may also be restricted. Potassium intake is generally limited to 1 mmol/kg of ideal body weight per day. The person is cautioned to avoid using salt substitutes, which typically contain high levels of potassium chloride. See Table 27.8 for foods high in potassium and phosphorus and some low-potassium and low-phosphorus alternatives.

Nursing care

The clinical setting, whether it is in the community or a secondary or tertiary referral hospital, determines the nursing care.

Upon diagnosis of CKD, regardless of the clinical setting, the teaching of self-management principles begins. The principal nursing diagnosis at this time may be *Deficient knowledge* in relation to understanding of disease process and measures to help preserve kidney function. The nurse, in partnership with the person, should establish priorities for management and collaborate with other members of the interprofessional team as required. Ensure that the person and family understand and are able to discuss the following topics:

- information about the disease and the prognosis
- prescribed treatment, including activity and diet restrictions; the use and potential effects, both beneficial and adverse, of all medications
- risks, manifestations, prevention and management of complications such as oedema and infection
- signs, symptoms and implications of declining renal function
- measures to prevent further kidney damage, including eliminating modifiable risk factors and avoiding nephrotoxic drugs
- community resources, such as home care providers and support groups.

Teach people with CKD about the underlying pathology of their specific disease process, its treatment and usual course. Discuss measures to maintain optimal renal function and the importance of avoiding additional insults to their kidneys. Include extra information about preventing UTI (such as hygiene measures) and early manifestations of UTI. Stress the importance of seeking treatment to prevent further kidney damage. Advise to avoid drugs that are potentially toxic to the kidneys and to check with the primary care provider before taking any new drug, including over-the-counter and complementary medicines.

For those with polycystic kidney disease, discuss the potential benefits of genetic counselling and screening of family members for evidence of the disease. This is particularly important if renal transplantation is contemplated and family members are potential donors.

Health promotion

Measures to reduce the risk of CKD focus on preventing kidney disease, particularly in those with diabetes and hypertension. Deal with modifiable risk factors promptly, including the maintenance of a healthy BMI and smoking cessation. Promote early and effective treatment of all infections, particularly skin and pharyngeal infections caused by streptococcal bacteria. Discuss measures to reduce the risk of urinary tract infections and stress the importance of prompt treatment to eradicate the infecting organism. Discuss the relationship between diabetes, hypertension and kidney disease. The rates of obesity in Australia are of particular concern given that increased BMI is linked as an independent risk factor for type 2 diabetes, hypertension and cardiovascular disease. Emphasise that maintaining blood glucose levels and blood pressure within the recommended ranges reduces the risk of adverse effects on the kidneys. Reiterate the importance of avoiding drugs and

substances that are potentially toxic to the kidneys. Ensure that all people with reduced renal function are well hydrated, particularly when a nephrotoxic drug is prescribed or anticipated. Finally, encourage people approaching ESKD to investigate options for early transplantation to avoid long-term dialysis. In liaison with the 'living related' transplant team discuss the option of a living donor and renal allotransplantation.

Assessment

Review Chapter 25 for complete assessment of the renal and urinary systems. Focused assessment data related to CKD include the following:

- *Health history:* complaints of facial or peripheral oedema or weight gain, fatigue, nausea and vomiting, headache, general malaise, abdominal or flank pain; cough or shortness of breath; changes in amount, colour or character of urine (e.g. frothy urine); history of skin or pharyngeal streptococcal infection, diabetes, SLE or kidney disease; current medications.
- *Physical examination:* general appearance; vital signs; weight; presence of periorbital, facial or peripheral oedema; skin for lesions, infection; inspect throat, obtain culture as indicated; urine specimen for colour, character, odour.

Nursing diagnoses and interventions

Nursing care is supportive and educational. Monitoring renal function and fluid volume status is a key component of care, as is protecting the person from infection. Both manifestations of CKD and its treatment can interfere with a person's ability to maintain usual roles and responsibilities.

Excess fluid volume

Excess fluid volume and resulting oedema are common manifestations of glomerular disorders. When proteins are lost in the urine, the oncotic pressure of plasma falls and fluid shifts into the interstitial spaces. The body responds to this fluid shift by retaining sodium and water to maintain intravascular volume, leading to excess fluid volume.

- Monitor vital signs, including blood pressure, apical pulse, respirations and breath sounds, at least every 4 hours. Report significant changes. *Excess fluid increases the cardiac workload and the blood pressure. Tachycardia may result. Associated electrolyte imbalances can cause arrhythmias. Increased pulmonary vascular pressure can lead to pulmonary oedema, tachypnoea, dyspnoea and crackles (rales) in the lungs.*
- Record fluid intake and output as indicated. *Accurate fluid balance records help determine fluid volume status.*

CONSIDERATION FOR PRACTICE

Weigh daily, using consistent technique (time of day, scale and clothing). Accurate daily weights are the best indicator of approximate fluid balance.

- Monitor serum electrolytes, haemoglobin and haematocrit, urea, creatinine and eGFR. *Glomerular disorders affect fluid balance and may alter electrolyte balance as well, potentially leading to complications such as cardiac arrhythmias (see Chapter 9). Increased intravascular volume can result in low haemoglobin and haematocrit values. Urea, creatinine and eGFR provide information about renal function.*
- Maintain fluid restriction as ordered. Offer ice chips (in limited and measured amounts) and frequent mouth care to relieve thirst. With the person, develop a fluid intake schedule. *Fluids may be restricted to reduce fluid overload, oedema and hypertension. Ice chips and frequent mouth care moisten mucous membranes and help relieve thirst while maintaining oral tissue integrity. Including the person in planning fluid intake promotes a sense of control and understanding of the treatment regimen.*

CONSIDERATION FOR PRACTICE

Carefully monitor and regulate intravenous infusions; include fluid used to dilute IV medications as intake. Significant 'hidden' fluid intake can occur with intravenous medication administration.

- Arrange dietary consultation regarding sodium- or protein-restricted diets. Include the person and dietitian in planning to allow individualisation of the diet to person preferences. *The glomerular disorder may reduce appetite; considering food preferences can help maintain adequate nutrition.*
- Monitor for desired and adverse effects of prescribed medications. *Diuretic therapy helps reduce excess fluid volume; however, glomerular disorders can affect the person's response to treatment. In addition, diuretics can exacerbate the electrolyte imbalances and muscle weakness often associated with glomerular disorders.*
- Provide frequent position changes and good skin care. *Perfusion may be altered by tissue oedema, increasing the risk of breakdown.*

Fatigue

Fatigue is a common manifestation of CKD. Anaemia, loss of plasma proteins, headache, anorexia and nausea compound this fatigue. The ability to maintain usual physical and mental activities may be impaired.

- Schedule activities and procedures to provide adequate rest and energy conservation. Prevent unnecessary fatigue. *Adequate rest and energy conservation reduce fatigue and improve the person's ability to tolerate and cope with required treatments and activities.*
- Assist with ADLs as needed. *The goal is to conserve limited energy reserves.*
- Discuss the relationship between fatigue and the disease process with the person and family. *Understanding the nature of the disease and associated fatigue helps the person and family cope with reduced energy and comply with prescribed rest.*
- Reduce energy demands with frequent, small meals and short periods of activity. Limit the number of visitors and visit length. *Small, frequent meals reduce the energy*

needed for eating and digestion. Limiting visitors and visit length helps conserve energy. In addition, nurses can assist the fatigued person who may be reluctant to ask visitors to leave.

Ineffective protection

People with CKD are at high risk of infection. The use of some drug classes in specific treatments can depress the immune system further. The anti-inflammatory effect of corticosteroids may also mask early symptoms of infection.

CONSIDERATION FOR PRACTICE

Monitor vital signs, temperature and mental status every 4 hours. An elevated temperature may indicate infection; anti-inflammatory drugs may moderate this response, however. Tachycardia, increasing lethargy or confusion may be the initial signs of infection.

- Assess frequently for other signs of infection such as purulent wound drainage, productive cough, adventitious breath sounds and red or inflamed lesions. Monitor for manifestations of UTI, such as dysuria, frequency and urgency, and cloudy, foul-smelling urine. *Early identification and treatment of infection is important to prevent systemic complications in the susceptible person.*
- Monitor FBC, focusing on the WCC and differential. *An elevated WCC and increased numbers of immature white cells in the blood (left shift) may be early indicators of infection.*
- Use standard precautions and good handwashing technique. Protect from cross-infection by providing a private room and restricting ill visitors. *People with decreased resistance to infection need increased protection.*
- Avoid or minimise invasive procedures. *Maintaining the protective skin barrier is especially important for the person with altered immune status.*
- If catheterisation is required, use sterile intermittent straight catheterisation or maintain a closed drainage system for an indwelling catheter. Prevent urine reflux from the drainage system to the bladder or the bladder to the kidneys by ensuring a patent, gravity flow system. *The urinary tract is a frequent entry point for infection, particularly in the hospitalised person. Maintaining strict asepsis during catheterisation is vital. Intermittent catheterisation is associated with a lower risk of UTI than an indwelling catheter.*
- Provide a nutritionally sound diet with complete proteins. *A well-balanced, nutritionally sound diet is important to maintain nutritional status and support immune function.*
- Teach measures to prevent infection. *Care is often provided in the home, requiring the person and family to use appropriate infection control measures.*

Ineffective role performance and anticipatory grieving

Strategies to support people with ineffective role performance and anticipatory grieving are similar and revolve around support processes. The manifestations and treatment of CKD can affect the ability to maintain usual roles and activities. In addition, the person may express feelings associated with the loss of function and lifestyle. Fatigue and muscle weakness may limit physical and social activities. If uraemia is present, malaise, nausea and mental status changes can interfere with role function. Facial and periorbital oedema affect the person's self-esteem and may lead to isolation.

- Establish a strong therapeutic relationship. *It is important to gain the person's trust and confidence to afford them the opportunity to share their feelings.*
- Encourage self-care and participation in decision making. *Increased autonomy helps to restore self-confidence and reduce powerlessness.*
- Provide time for verbalisation of thoughts and feelings; listen actively, acknowledging and accepting fears and concerns. *Adequate time and active listening encourage expression of concerns and the effect of the disease or treatments on daily life. This helps the person deal with the illness, its treatment and associated losses.*
- Support coping skills, helping the person identify personal strengths. *This support helps the person gain confidence.*
- When possible, enlist the support of family, other people and friends. *These people can provide physical, psychological, emotional and social support.*
- Discuss the effect of the disease and treatments on roles and relationships, helping identify potential changes in roles, relationships and lifestyle. Help the person and family develop a plan for alternative behaviours and relationships, encouraging the person to maintain usual roles to the extent possible. *Developing a plan helps reduce the strain of role changes and maintain a sense of dignity and control.*
- Provide accurate and optimistic information about the disorder and its short- and long-term effects. *The person and family need accurate information to plan for the future.*
- Evaluate the need for additional support and social services for the person and family. Provide referrals as indicated. *Depending on the person and family strengths, the severity of the disorder and its treatment and prognosis, ongoing social support services may be necessary to facilitate coping and adaptation.*

Community-based care

Whereas hospital-based care is focused on treating the complications of CKD symptomology, community care is focused on primary prevention, screening practices and the teaching of self-management principles. The cornerstone of any primary prevention program is the screening of people in 'at-risk' categories (see Box 27.3). Screening programs, whether they are opportunistic or systematic, assist in identifying people with CKD and afford the opportunity to initiate timely intervention to prevent the progression to ESKD. Nurses are ideally placed to identify at-risk people and implement screening strategies to identify people with CKD. One of the wonders of screening strategies for CKD is that they are predominantly non-invasive and non-expensive (see Box 27.4). Urinalysis and blood pressure measurements are key screening techniques for detecting

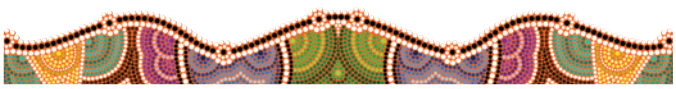
BOX 27.3 Major risk factors for chronic kidney disease (CKD)

- Diabetes
- High blood pressure
- Aged over 60 years
- Smoking
- Obesity
- Family history of kidney disease
- Aboriginal or Torres Strait Islander origin
- Established cardiovascular disease

BOX 27.4 The kidney health check

1. Blood test for eGFR to check kidney function
2. Urine test for albumin:creatinine ratio (ACR)
3. Check blood pressure for presence of hypertension

glomerular dysfunction (i.e. microalbuminuria or proteinuria) and hypertension. Kidney function can also be measured through eGFR from a simple blood test. CKD is progressive in nature; its course is generally lengthy but may also progress rapidly in some cases.



THE PERSON WITH END-STAGE KIDNEY DISEASE

Kidney replacement therapy

Kidney replacement therapy (KRT) (previously referred to as renal replacement therapy (RRT)), used to treat end-stage kidney disease (ESKD), is provided through haemodialysis, peritoneal dialysis or kidney transplantation. ESKD is reached when glomerular function decreases to a level of less than 15 mL/min (AIHW, 2015d). In Australia, dialysis and transplantation have been available since the early 1960s. The first dialysis was performed in Brisbane in 1954 (Dique, 1955). The first successful transplant was performed in Adelaide in 1965 (Lawrence, 1994).

KRT involves considerable individual and healthcare burdens. Australia has followed the worldwide trend and seen an approximate annual increase of 6% in the number of people receiving dialysis treatment, although this has stabilised somewhat from 2006 to 2013 (ANZDATA Registry, 2015). The greatest increase since 1989 has been in the elderly with a 13-fold increase in the number of people over 75 years of age receiving dialysis (AIHW, 2012). In 2013, a record high 882 kidney transplants were performed in Australia; however, Australia's organ donation rates remain relatively low compared with countries such as Spain, Portugal and the United States (ANZDATA Registry, 2015). The numbers of people receiving KRT will likely increase by 80% from 2011 to 2020 (AIHW, 2012) placing an enormous burden on future Australian healthcare resources.

Dialysis

Dialysis is the diffusion of solute molecules across a semipermeable membrane from an area of higher solute concentration to one of lower concentration. It is used to remove excess fluid and metabolic waste products in kidney failure. During dialysis, blood is separated from a dialysis solution by a semipermeable membrane. Both haemodialysis and peritoneal dialysis rely on the principle of diffusion to remove wastes from people during KRT. For the person who is not a candidate for kidney transplantation, or who has had a transplant failure, dialysis is life sustaining.

Of the 11 774 people receiving dialysis at the end of 2013, 80% were receiving haemodialysis and 20% were receiving peritoneal dialysis (ANZDATA Registry, 2015). Care involving dialysis is by far the most common reason for same-day separations in Australia (AIHW, 2012). Unfortunately, there are significantly higher rates of admission for dialysis in the Indigenous and lower socioeconomic populations (AIHW, 2012).

Both peritoneal dialysis and haemodialysis can be undertaken safely at home. Although home dialysis therapy is the preferred treatment of clinicians and resources have been increased (Lauder et al., 2011; Ludlow et al., 2011), rates have not increased significantly over the past 10 years (Fortnum & Ludlow, 2014). Home dialysis can be performed more frequently and can suit the person's lifestyle (Chow & Tran, 2012). In saying this, factors such as employment and availability of a dialysis centre become the primary factors influencing the choice of haemodialysis or peritoneal dialysis (Bennett, Schatell & Shah, 2015; Sinclair, 2008).

People on long-term dialysis have a higher risk of complications and death than the general population. Mortality rates are generally higher with older age, diabetes, Indigenous status and coronary artery disease; however, crude 1-year mortality rates in this group have decreased from 0.14 to 0.10 over the past 10 years (ANZDATA Registry, 2015). Although dialysis and transplantation can provide lifesaving treatment they are not the only options for people with ESKD, with supportive care more frequently being provided.

Supportive care

The decision to initiate dialysis is not easy as dialysis only manages the symptoms of ESKD and is not a cure. Although many people on dialysis still maintain jobs, dialysis requires considerable support from healthcare professionals and significant others as people can be challenged with the day-to-day stress that accompanies dialysis treatment (Josland, 2013). Many people live with constant lethargy, never feeling truly well and may feel powerless because of their dependence on others for treatment (Tallis, 2005). In the end, the person may choose supportive care rather than undergoing dialysis or transplantation.

Supportive care in the ESKD context describes the active therapy that is delivered without dialysis or transplantation. Given the often high symptom burden in ESKD, renal supportive care includes appropriate symptom management, care coordination and ongoing education for patients, families and health professional staff (Josland, 2013). Although supportive care is increasingly being offered in Australia and globally,

patients commonly report a greater emphasis on dialysis and transplant over supportive care (Moustakas, Bennett & Tranter, 2015). Given that the quality of life is often compromised by dialysis and the mortality rates over 65 years are comparable to supportive care (Jassal & Watson, 2011), supportive care is a legitimate therapy option for any person with ESKD.

Haemodialysis

Haemodialysis uses the principles of diffusion and ultrafiltration to remove electrolytes, waste products and excess water from the body. Blood is pumped from the person via a vascular access to the dialyser (see Figure 27.7). The porous membranes of the dialyser allow small molecules such as water, glucose and electrolytes to pass through, but block larger molecules such as serum proteins and blood cells. The **dialysate**, a solution of approximately the same composition and temperature as normal extracellular fluid, passes along the other side of the membrane allowing small solute molecules to move freely across the membrane by diffusion.

The direction of movement for any substance is determined by the concentrations of that substance in the blood and the dialysate. Electrolytes and waste products such as urea and creatinine diffuse from the blood into the dialysate. Essential electrolytes such as calcium or potassium may be added to the dialysate to reduce excessive removal. Excess water is removed by creating a negative pressure on to the blood moving through the dialyser. This process is known as **ultrafiltration**.

Haemodialysis is predominantly performed three times a week, varying from 3 to 8 hours per treatment with the majority of people receiving 4 to 5 hours per treatment or 12 to 15 hours per week (ANZDATA Registry, 2015). Haemodialysis is performed most frequently in satellite, or community, settings that are nurse run and require little nephrologist management input (Bennett, 2011). The most frequent complications during dialysis (intradialytic) are hypotension and muscle cramps (Bradshaw, Ockerby & Bennett, 2011). Infection, malnutrition and vascular access problems are common long-term complications of haemodialysis.

Following are complications associated with haemodialysis:

- Hypotension, the most frequent complication during haemodialysis, is related to changes in serum osmolality, rapid removal of fluid from the vascular compartment, vasodilation and other factors.
- Bleeding is related to altered platelet function associated with uraemia and the use of heparin during dialysis.
- Infection (local or systemic) is related to WBC damage and immune system suppression. *Staphylococcus aureus* septicaemia is commonly associated with contamination of the vascular access site.

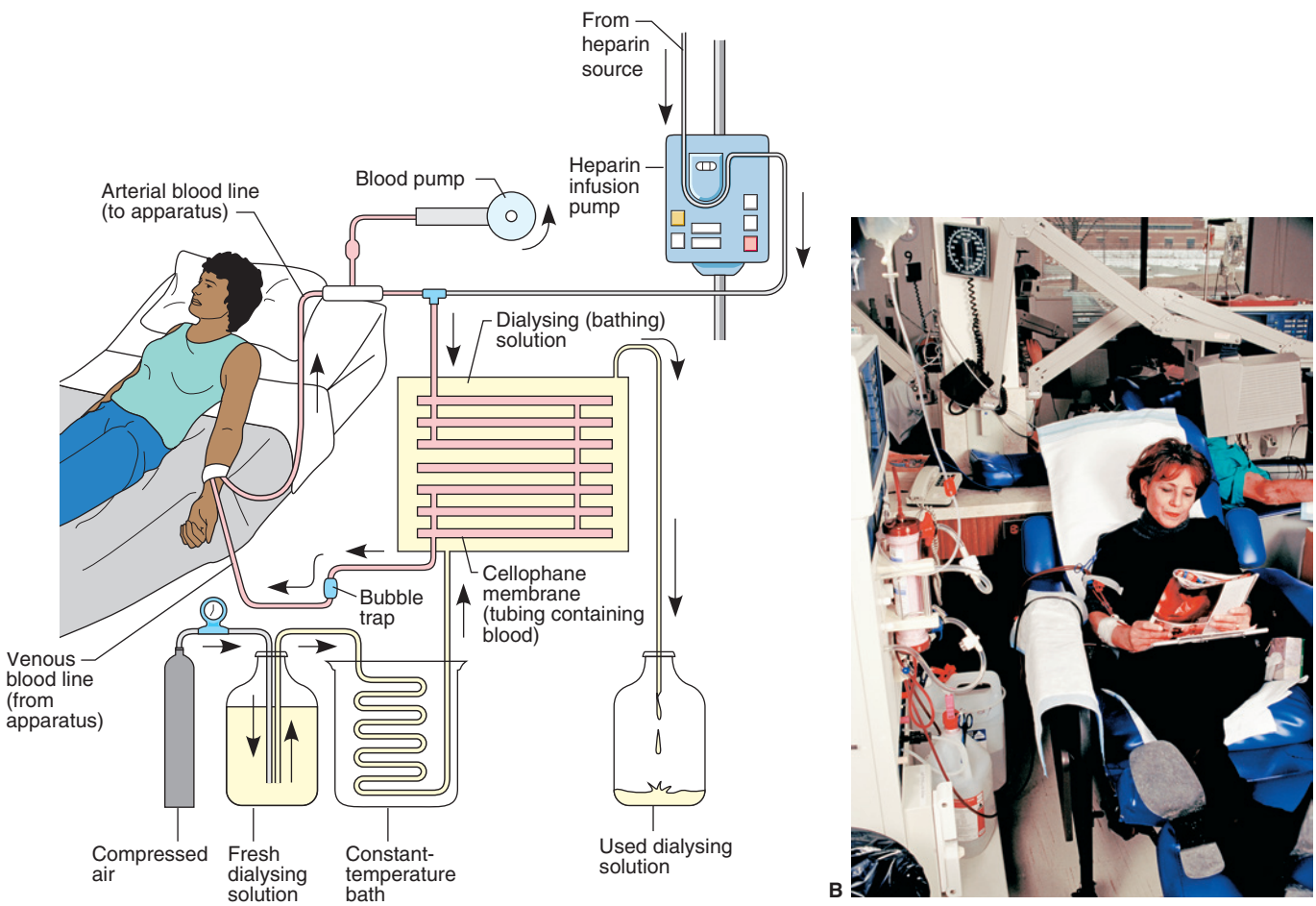


FIGURE 27.7 ■ *A*, The components of a haemodialysis system. *B*, A person receiving haemodialysis

Source: B, © Carolyn A. McKeone/Science Source.

See the box below for nursing care for the person undergoing intermittent haemodialysis.

Haemodiafiltration combines the processes of haemodialysis and greater ultrafiltration in an attempt to better mimic the function of the healthy kidney (Hill et al., 2015). Large amounts of fluid are infused into the dialysis blood circuit and then removed through the haemofilter. Thus both diffusion and convection occurs in this process. The advantage of haemodiafiltration is the greater amount of wastes that can be removed through increased convection. Continuous haemodiafiltration, known as CVVHDF, is used in acutely ill people through continuous therapy.

Vascular access

Access to the blood is required for haemodialysis and is commonly termed 'vascular access'. Vascular access can be in the form of arteriovenous fistula (AVF), arteriovenous graft (AVG) or central venous dialysis catheter (CVDC). AVF is the preferred vascular access with decreased complications compared with grafts and catheters. In 2013 in Australia, prevalent haemodialysis access comprises AVFs 78%, grafts 7% and CVDCs 15%; however, over 50% of all people commencing haemodialysis commence with a CVDC (ANZDATA Registry, 2015).

Arteriovenous fistulae are created by surgically creating an opening (fistula), from an artery to a vein. In preparation for fistula formation, the access limb should not be used for venipuncture prior to the surgical construction of the AVF. The most common vessels used are the radial artery and cephalic vein; however, upper arm veins and arteries can be used. It takes approximately 4 to 6 weeks for the pressure in the artery to engorge or enlarge the veins. Once the veins are large enough and are providing an adequate blood flow, the fistula can be used

NURSING CARE OF THE PERSON undergoing intermittent haemodialysis

PRE-DIALYSIS CARE

- Assess vital signs, including the most appropriate blood pressures (lying, sitting and standing), pulse, respirations and lung sounds. *These data provide baseline information to help evaluate the effects of haemodialysis. Hypertension or hypotension may indicate excess fluid volume. The person who is hypotensive may not tolerate rapid fluid volume changes during dialysis. Abnormal heart sounds (e.g. a gallop or murmur) and changes in heart rate or rhythm may indicate excess fluid volume or electrolyte imbalance. Fluid overload may also cause dyspnoea, tachypnoea and rales or crackles in the lungs.*
- Record weight. *Weight changes are an indicator of fluid volume and nutritional status and assist in determining amount of ultrafiltration required.*
- Assess vascular access site for a palpable pulsation or vibration and an audible bruit and for inflammation. *Infection and thrombus formation are the most common problems affecting the access site in haemodialysis people.*
- Alert all staff to avoid using the extremity with the vascular access site (or the non-dominant arm, if long-term access has not been established) for blood pressures or venipuncture. *These procedures may damage vessels and lead to failure of the arteriovenous fistula.*

INTRADIALYTIC CARE

- Assess and document vital signs during dialysis treatment. *Fluid removal may lead to hypotension.*
- Assess and treat other signs of volume depletion such as cramp, nausea and vomiting. Reset fluid loss as required. *Hypotension may lead to cramp and/or nausea and vomiting.*
- Respond to dialysis machine blood circuit alarms. *Arterial pressure and/or venous pressure may indicate access or volume status complications.*
- Assess access sites for signs of bleeding. *Dialysis anticoagulation may contribute to bleeding around access sites.*
- Use aseptic technique during cannulation technique and when accessing the blood circuit. *Poor aseptic technique may lead to infection.*

POST-DIALYSIS CARE

- Assess and document vital signs, weight and vascular access site condition. *Rapid fluid and solute removal during dialysis may lead to orthostatic hypotension, cardiopulmonary changes and weight loss.*
- Review urea, serum creatinine, serum electrolyte and haemoglobin levels between dialysis treatments. *These values help determine the effectiveness of the treatment, the need for fluid and diet restrictions, and the timing of future dialysis sessions. If the anaemia associated with kidney failure does not improve with dialysis, erythropoietin stimulating agents (ESAs), iron and folate supplements may be needed.*
- Assess for dialysis disequilibrium syndrome, with headache, nausea and vomiting, altered level of consciousness and hypertension. *Rapid changes in urea, pH and electrolyte levels during dialysis may lead to cerebral oedema and increased intracranial pressure.*
- Assess for other adverse responses to dialysis, such as dehydration, nausea and vomiting, or muscle cramps. *Excess fluid removal and rapid changes in electrolyte balance can cause fluid deficit, nausea, vomiting and seizure activity.*
- Assess for bleeding at the access site or elsewhere. Use standard precautions at all times. *Kidney failure and heparinisation during dialysis increase the risk of bleeding.*
- Provide psychological support and listen actively. Address concerns and accept responses such as anger and depression. Reinforce person and family strengths in coping with kidney failure and haemodialysis. *Grieving is a normal response to loss of organ function. The person may feel hopeless or helpless and resent dependence on a machine. The nurse can help the person and family work through these responses and focus on positive aspects of living.*
- Refer to social services and counselling as indicated. *People with kidney failure may need additional support services to help them adapt to and live with their disease.*

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 9:
Recognising and Responding to Clinical Deterioration in Acute Health Care

'The intention of this standard is to ensure a patient's deterioration is recognised promptly, and appropriate action is taken.' (ACSQHC, 2011, p. 60)

Implementing this standard is achieved by the establishment of systems to assist with recognition, initiating appropriate responses and ensuring meaningful and appropriate communication between all individuals involved in a person's care (including patients, families and carers).

Comprehensive and ongoing assessment of a person's renal function should be undertaken and documented as frequently as clinically necessary to facilitate rapid, responsive action in the event of a person's deterioration. Treatment may be pharmacological, but may also include acute renal replacement therapy. Advanced planning should be undertaken prior to the potential rapid renal deterioration to ensure treatment decisions are patient centred.

Source: © Australian Commission on Safety and Quality in Health Care.

for taking and replacing blood during dialysis. A functional AVF has a palpable pulsation and a bruit on auscultation. Accurate measurement of blood flow through the fistula can be assessed using ultrasound techniques (Schoch et al., 2008).

Historically the recommended cannulation technique for AVFs was the 'rope-ladder' technique where, ideally, a new site was used up and down the fistula to avoid fistula aneurysm, creating a rope-ladder look. Recent shifts in cannulation techniques have occurred with the increased popularity of the 'buttonhole' or 'constant site' technique. This technique involves the repeated use of the same track or tunnel to access blood from the AVF, culminating in the use of blunt needles. Although not appropriate for all people on dialysis (Moist & Nesrallah, 2014), the buttonhole technique may decrease pain and anxiety related to cannulation (Verhallen, Kooistra & van Jaarsveld, 2007).

Arteriovenous graft is the alternative if AVF construction is not possible, most commonly due to poor blood vessel condition. AVGs are most commonly made of polytetrafluoroethelene (PTFE) material. The graft is surgically implanted and connects the artery and the vein. Blood flows through the graft from the artery to the vein, providing sufficient blood flow for haemodialysis. AVGs can be constructed in the arm or leg. Unlike AVFs, grafts can often be used soon after surgery but have a higher infection and thrombosis rate than AVFs (Moist & Al-Jaishi, 2013). The HeRO Graft is a further option that is showing early successes; it involves traversing higher vein stenosis with the outflow directly into the central venous circulation (Nassar et al., 2014).

Central venous dialysis catheters are predominantly double-lumen catheters inserted into the jugular, subclavian or femoral vein (see Figure 27.8). If prolonged vascular access is required, a double-cuffed tunnelled catheter may be inserted (Hill et al., 2015). Design of CVDCs is constantly evolving; however, in all CVDCs blood is drawn into the catheter through small openings in the proximal portion of the catheter and returned to the circulation through an opening in the distal end of the catheter to minimise recirculation of the blood that has just been dialysed.

The two most frequent complications of a CVDC are infection and clotting. To reduce the risk of infection, strict aseptic technique is vital in caring for CVDC sites (Chu, Adams and Crawford, 2013). To reduce intraluminal clotting, insertion of anticoagulant (heparin, sodium citrate) following the dialysis procedure is required. Importantly, the anticoagulant lock needs to be removed prior to the next treatment to avoid unnecessary anticoagulation of the already acutely ill person.

Peritoneal dialysis

Peritoneal dialysis is more frequently being used as the first treatment of choice. This is related to the increased independence and increased maintenance of residual kidney function (McCarthy et al., 2010). Peritoneal dialysis uses the highly vascular peritoneal membrane, which surrounds the surfaces of the peritoneal cavity, as the dialysing surface (see Figure 27.9). Warmed sterile dialysate is instilled into the peritoneal cavity through a Tenckhoff catheter inserted into the peritoneal cavity. Metabolic waste products and excess electrolytes diffuse into the dialysate while it remains in the abdomen. Water movement is

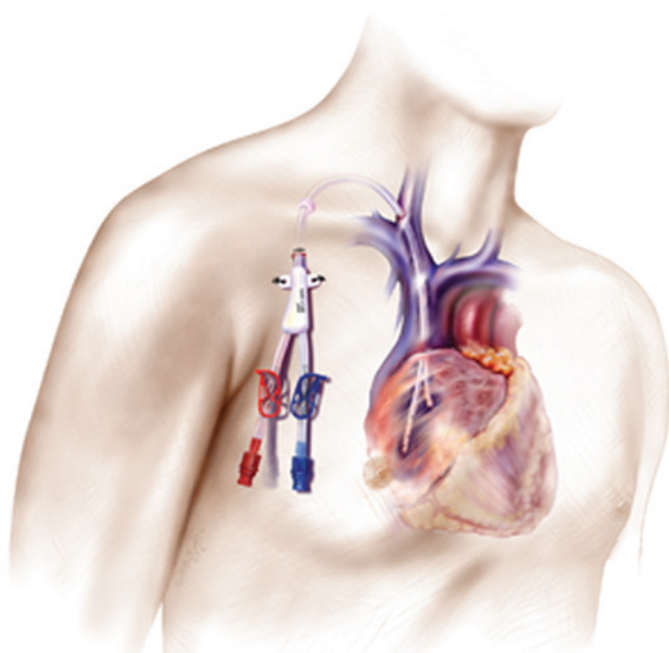


FIGURE 27.8 ■ Central venous dialysis catheter

controlled using dextrose-based fluids as osmotic agents to draw it into the dialysate, a process known as ultrafiltration. The two most common forms of peritoneal dialysis are continuous ambulatory peritoneal dialysis and automated peritoneal dialysis.

In peritoneal dialysis, heparinisation and vascular complications associated with an arteriovenous fistula are avoided and often residual renal function is better maintained. The clearance of metabolic wastes is slower but more continuous, avoiding rapid fluctuations in extracellular fluid composition and associated symptoms. More liberal intake of fluids and nutrients is often possible for the person with peritoneal dialysis. While glucose absorbed from dialysate can increase blood glucose levels in the person with diabetes, regular insulin can be added to the infusion to manage hyperglycaemia. The majority of people on peritoneal dialysis are able to self-manage the treatment regimen, affording a greater sense of autonomy and reducing any potential feelings of helplessness (Al Wakeel et al., 2009).

The major complication of peritoneal dialysis is peritonitis, where infection of the peritoneum occurs. This can severely limit the effect of peritoneal dialysis, decrease the life of the peritoneum and cause life-threatening infection if not treated early (Evans, 2012). Other long-term complications include exit site infection, mechanical flow restrictions and abdominal pain. (See the box below for nursing care for the person undergoing peritoneal dialysis.) The presence of an indwelling peritoneal catheter may cause a body image disturbance; however, the presence of an AVF in haemodialysis may also be a disturbance.

Continuous ambulatory peritoneal dialysis (CAPD) is performed manually and is most commonly performed at home. Two- to three-litre bags of dialysate fluid are infused into the abdomen and left to dwell for prescribed intervals. The fluid is then drained by gravity out of the peritoneal cavity into a sterile bag. This process of dialysate infusion, dwell time of the solution in the abdomen and drainage is repeated four to five times per day and is often referred to as 'exchanges'. No special equipment is needed. Excess fluid and solutes are removed more gradually in CAPD, minimising large blood pressure and solute variations. Depending on comfort, clearance and ultrafiltration measures, people receiving CAPD may leave a bag of dialysate fluid in their peritoneum overnight.

Automated peritoneal dialysis (APD) uses a machine (see Figure 27.10) to cycle dialysate fluid in and out of the peritoneum over a continuous period of 8 to 12 hours. This is most commonly undertaken overnight and at home. This allows the person receiving APD to cap off their catheter during the day. CAPD and APD can be combined, with some people undertaking one or two exchanges in the middle of the day while still receiving APD overnight (George, 2009).

FAST FACTS

- Infective complications of peritoneal dialysis are the second most common cause of technique failure after social reasons.
- A diagnosis of peritonitis is established if the person presents with two or more of the following: cloudy effluent with a WCC > 100 µL (with a neutrophil count > 50%), abdominal pain, fever or positive Gram stain.
- Fungal peritonitis generally requires the removal of the Tenckhoff catheter and temporary haemodialysis.

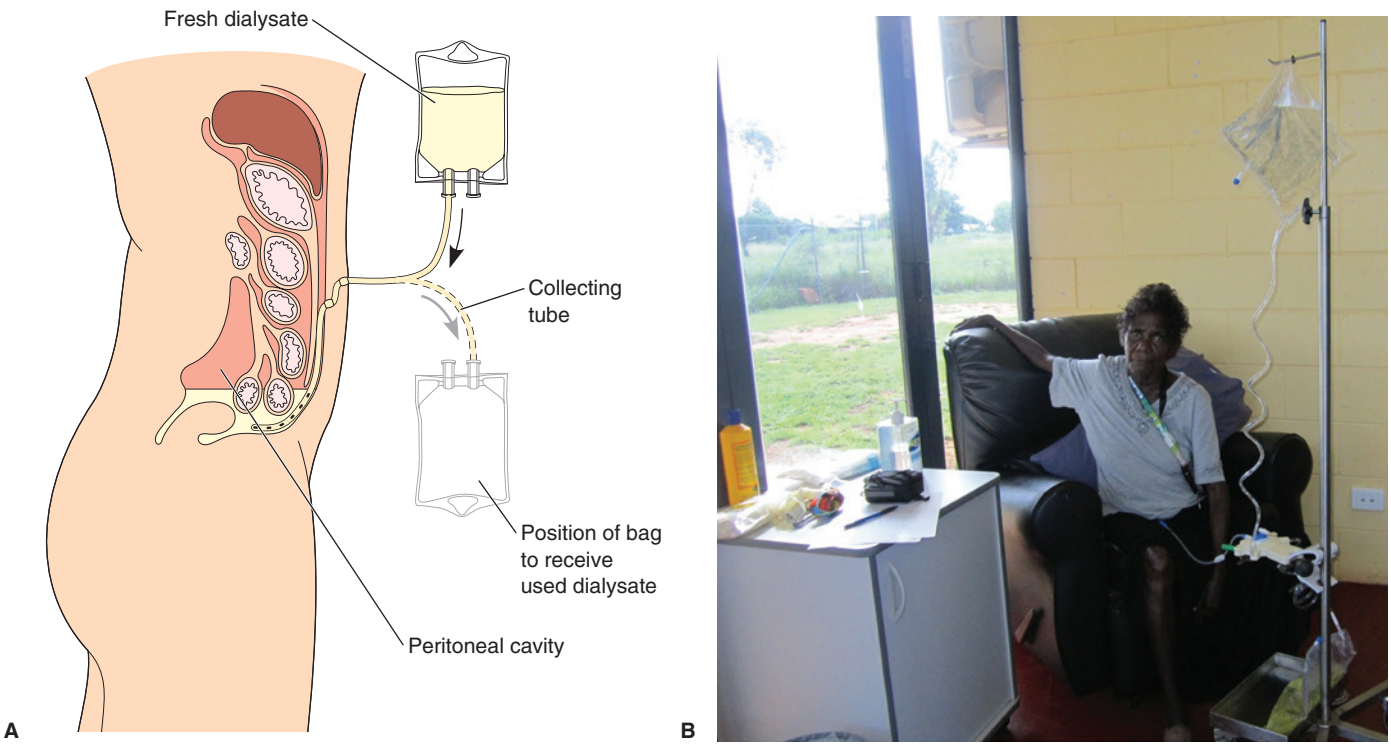


FIGURE 27.9 ■ *A,* Peritoneal dialysis. *B,* A person receiving peritoneal dialysis

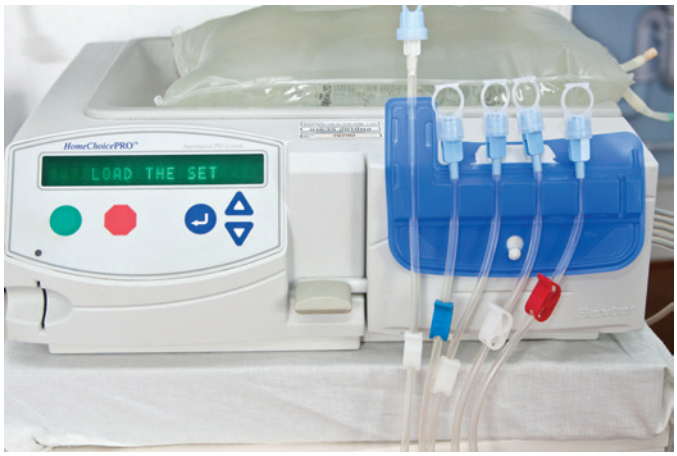


FIGURE 27.10 ■ Automated peritoneal dialysis machine

Source: © Life in View/Science Photo Library.

Continuous renal replacement therapy

Continuous renal replacement therapy (CRRT) is used for people with acute kidney injury (AKI) and is most commonly undertaken in a critical care or high-dependency setting. Initially, people with AKI typically undergo continuous haemodialysis, then intermittently as indicated. Intermittent haemodialysis is not used if the person is haemodynamically unstable (e.g. with hypotension or low cardiac output). Access to a blood flow of at least 200 mL/min is required for CRRT (see Figure 27.11), predominantly using a double-lumen venous catheter.

In CRRT, blood is continuously circulated through a highly porous haemofilter from artery to vein, or vein to vein, for a period of 12 or more hours. CRRT is used for people with acute kidney dysfunction who are often unable to tolerate haemodialysis and rapid fluid removal if their cardiovascular status is unstable (e.g. due to trauma, major surgery, heart failure, septicaemia), as it allows more gradual fluid and solute removal. Excess water and solutes such as electrolytes, urea, creatinine, uric acid and glucose are removed, and fluid may be replaced with normal saline or a balanced electrolyte solution as needed.

CRRT is a slower process than maintenance haemodialysis and helps maintain haemodynamic stability and avoid complications associated with rapid changes in extracellular fluid composition. Continuous therapies include continuous venovenous haemofiltration (CVVH), continuous venovenous haemodialysis (CVVHD), continuous venovenous haemodiafiltration (CVVHDF) and slow continuous ultrafiltration (SCUF). Intermittent therapies gaining popularity include sustained low-efficiency dialysis (SLED) and intermittent haemodiafiltration (IHD). See Table 27.9.

Collaborative care

A number of considerations affect the choice of long-term kidney replacement therapy. Haemodialysis and peritoneal dialysis each have advantages and disadvantages. Establishing vascular access for haemodialysis may take several months; therefore, planning ahead to develop the access before dialysis is necessary can ease the transition to dialysis (Morton et al., 2009). Established access

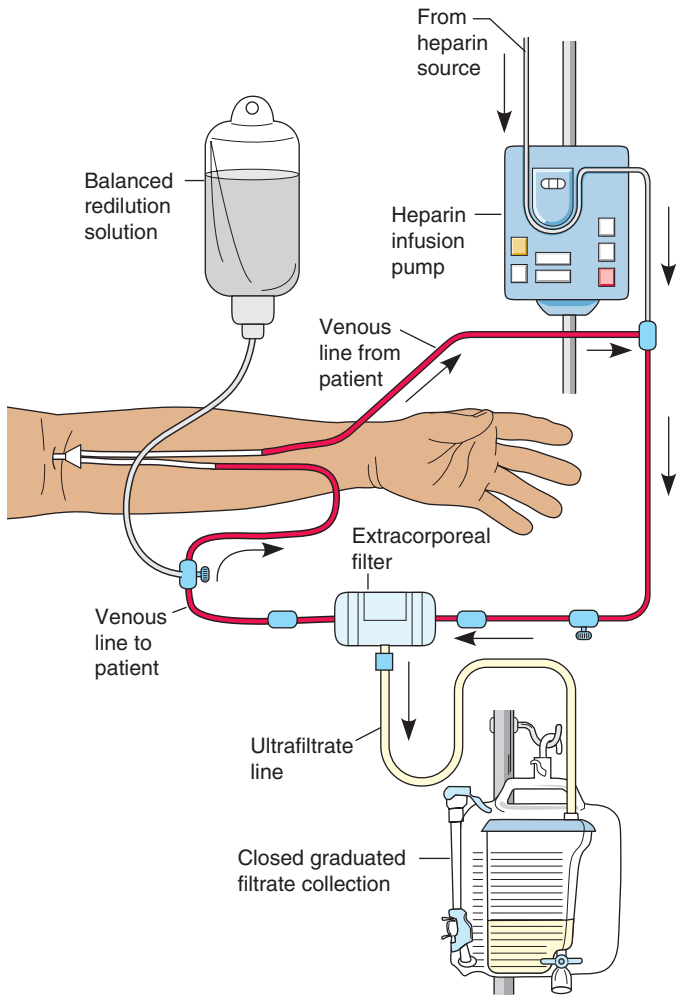


FIGURE 27.11 ■ Continuous renal replacement therapy

is not a consideration for peritoneal dialysis. The peritoneal catheter can be placed and treatment initiated as soon as it is indicated. When dialysis treatments will be performed at home, initiating instruction before it is required can result in more effective learning. If a family member or other care partner can serve as a dialysis helper, training begins prior to the onset of uraemia.

If transplantation is considered, tissue typing and identification of potential living related donors can be done prior to the onset of ESKD. To make an informed decision, both the person and the potential donor need to understand the risks, benefits and options available. If the decision for transplant is made early, dialysis can potentially be avoided. The person's age, concurrent health problems, donor availability and personal preference influence the choice of KRT.

KIDNEY TRANSPLANTATION

The first kidney transplant was performed in 1954 in Seattle, in the US; the donor and recipient were identical twins. Not long after, the first kidney transplants in Australia were performed in Melbourne and Adelaide. Unfortunately, kidney transplantation as a treatment for ESKD is limited primarily by the

NURSING CARE OF THE PERSON undergoing peritoneal dialysis

CONTINUOUS CARE

- Document vital signs including temperature, orthostatic blood pressures (lying, sitting and standing), pulse, respirations and lung sounds. *Data help assess fluid volume status and tolerance of the dialysis procedure. Hypertension, abnormal heart or lung sounds, or dyspnoea may indicate excess fluid volume. Poor respiratory function may affect the ability to tolerate peritoneal dialysis. Temperature measurement is vital because infection is the most common complication of peritoneal dialysis.*
- Assess weight and effluent drain volume between exchanges. *Weight is an accurate indicator of fluid volume status and allows assessment of ultrafiltration achieved from each exchange.*
- Monitor urea, serum electrolyte and creatinine levels. *These values are used to assess the effectiveness of dialysis.*
- Encourage healthy eating while assisting the person to maintain fluid and dietary restrictions as ordered. *Healthy eating while maintaining fluid and diet restrictions helps nutritional status and reduces hypervolaemia.*
- Warm the prescribed dialysate solution to body temperature (approx. 37°C) using a warm designated heating pad on low setting. *Dialysate is warmed to prevent hypothermia.*
- Explain all procedures and expected sensations. *Knowledge helps reduce anxiety and elicit cooperation.*
- Use strict aseptic technique during the exchange procedure and when caring for the Tenckhoff catheter. *Peritonitis is a common complication of peritoneal dialysis; sterile technique reduces the risk.*
- Add prescribed medications to the dialysate as required; prime the tubing with solution and connect it to the peritoneal catheter, avoiding kinks. *This allows dialysate to flow freely into the abdominal cavity and prevents leaking or contamination.*
- Instil dialysate into the abdominal cavity over a period of approximately 10 minutes. Clamp tubing and allow the dialysate to remain in the abdomen for the prescribed dwell time. Keep drainage tubing clamped at all times during instillation and dwell time. *Dialysate should flow freely into the abdomen if the peritoneal catheter is patent.*
- During instillation and dwell time, observe closely for signs of respiratory distress, such as dyspnoea, tachypnoea or crackles. Place in Fowler's or semi-Fowler's position and slow the rate of instillation slightly to relieve respiratory distress if it develops. *Respiratory compromise may result from overly rapid filling or overfilling of the abdomen or from a diaphragmatic defect that allows fluid to enter the thoracic cavity.*
- After prescribed dwell time, open drainage tubing clamps and allow dialysate to drain. Note the clarity, colour and odour of returned dialysate. *Cloudy dialysate may indicate an infection.*
- Accurately record amount and type of dialysate instilled (including any added medications), dwell time and amount and character of the drainage. *When more dialysate drains than has been instilled, excess fluid has been lost (output), the positive net difference between dialysate infused and effluent returned is termed the 'ultrafiltration volume'. If less dialysate is returned than has been instilled, a fluid gain has occurred (intake) and is referred to as 'fluid retained'.*
- Troubleshoot for possible problems during dialysis:
  a. Slow dialysate instillation. Increase the height of the dialysate bag and reposition the person. Check tubing and catheter for kinks. Check exit site dressing for wetness, indicating leakage around the catheter. *Slow dialysate flow may be related to a partially obstructed tube or catheter.*
  b. Excess dwell time. *Prolonged dwell time may lead to water depletion or hyperglycaemia.*
  c. Poor dialysate drainage. Lower the drainage container, reposition, check for tubing kinks. Check abdominal dressing. *Tubing or catheter obstruction can also interfere with dialysate drainage.*
- Time meals to correspond with dialysis drain. *Scheduling meals while the abdomen is empty of dialysate enhances intake and reduces nausea.*

TABLE 27.9 Continuous renal replacement therapies

TYPE	INDICATIONS	DESCRIPTION
Slow continuous utrafiltration (SCUF)	Remove mainly fluid	Blood circulates through dialyser and fluid is removed through negative pressure.
Continuous venovenous haemofiltration (CVVH)	Remove fluid and some solutes	Blood circulates through a haemofilter, then returns to person; ultrafiltrate collects in a drainage bag through convection.
Continuous arteriovenous haemodialysis (CVVHD)	Remove fluid and waste products	Venous blood circulates through a haemofilter surrounded by dialysate, then returns to the person through a double-lumen venous catheter; ultrafiltrate collects in a drainage bag following diffusion and osmosis.
Continuous venovenous haemodialysis (CVVHDF)	Remove fluid and waste products	Venous blood circulates through a haemofilter surrounded by dialysate, then returns to the person through a double-lumen venous catheter; ultrafiltrate collects in a drainage bag. Sterile dialysate is infused and removed through convection and diffusion.

availability of organs. In Australia, at the beginning of 2014 a total of 1056 people were on the transplant waiting list (ANZDATA Registry, 2015).

Kidney transplantation has become the treatment of choice for people with ESKD because it improves both survival and quality of life. In Australia, a person who has received a transplant has a far higher survival rate than those on dialysis (ANZDATA Registry, 2015). The person with a kidney transplant is no longer attached to a dialysis catheter, machine or centre. Dietary and fluid restrictions are reduced and the body image is more 'whole'. Importantly, medication and lifestyle regimes still have to be adhered to (Anderson et al., 2012).

Transplanted kidneys are obtained from living donors and cadaver donors. In Australia in 2013, 29% of transplanted kidneys came from living donors, most of whom were related to the recipient (ANZDATA Registry, 2015). Living donor transplants have been falling as a proportion of all transplants since 2008 (ANZDATA Registry, 2015). With both cadaver and living donor transplants, a close match between blood and tissue type is desired. Human leucocyte antigens (HLAs) are compared between the donor and recipient, with six antigens in common being considered to be a 'perfect' match. Close tissue matching may account for the better outcomes with living donors. Pre-emptive transplants (prior to commencement of dialysis) have maintained stable in Australia over the past 5 years with 38% of all living donors pre-emptive (ANZDATA Registry, 2015).

People with normal kidneys who are in good physical health may donate a kidney. Pre-donation counselling is vital because a nephrectomy (removal of the kidney) is major surgery and involves a small risk. If the transplant fails, the psychological impact on the donor can be significant.

Cadaver kidneys are obtained from people who meet the criteria for brain death and who are generally free of systemic disease, malignancy or infection. Kidneys are removed after brain death has been determined and are preserved by hypothermia or a technique called continuous hypothermic pulsatile perfusion. A kidney preserved by hypothermia is transplanted within 24 to 48 hours. Continuous pulsatile perfusion allows up to 3 days before transplantation. The system used to allocate cadaver kidneys for transplantation is outlined in Box 27.5.

In the surgical operation, the donor kidney is placed in the lower abdominal cavity of the recipient and the renal artery, vein and ureter are anastomosed (see Figure 27.12). The renal artery of the donor kidney is connected to the hypogastric artery and the renal vein to the iliac vein. The ureter is connected to one of the recipient's ureters or directly to the bladder, using a tunnel technique to prevent reflux.

Nursing care for the person receiving a kidney transplant is outlined in the box below.

BOX 27.5 How cadaver kidneys are allocated for transplant in Australia

The scarcity of organs for transplant raises questions about how cadaver kidneys are allocated. Past inequities in the allocation process (e.g. more men than women, more Caucasians than people of colour, more rich than poor and more young than old) led to the development of the United Network for Organ Sharing (UNOS) in 1986. UNOS developed policies for organ distribution, including kidneys, hearts, livers and other transplanted organs. The Transplantation Society of Australia and New Zealand (TSANZ) has developed Australian processes based on UNOS principles.

In Australia, the allocation of kidneys from a deceased donor to people on the waiting list is determined by a computer program called the National Organ Matching System (NOMS) which is administered by the Australian Red Cross. Mostly, only people who have commenced dialysis will be eligible to be listed on the waiting list. The main criteria that are used by NOMS to decide who will receive the kidneys are:

- the blood group (most kidneys go to somebody who is the same blood group as the donor)
- how long the person has been on dialysis
- the tissue typing or 'matching' with the donor
- whether the person has a lot of antibodies against other people's tissue types
- whether the person is a child; children get priority (Transplant Society of Australia and New Zealand (TSANZ), 2009).

NOMS runs a national matching program, which looks at all the people that are waiting in Australia to identify people who have antibodies against other people's tissue types. This is important because these people need a very well-matched kidney. It is much harder to find a suitable kidney for them than for people who do not have these antibodies. If somebody with a lot of these antibodies comes up as a very close match, the kidney can be sent to them from anywhere in Australia. This scheme covers people who have high levels of antibodies and only 0, 1 or 2 mismatches with the donor. It also allocates kidneys to people who have a perfect match with the donor, even if they have no antibodies. The exchange program also allows for kidneys to be sent from one state/territory to another to maintain a balance between the jurisdictions, so that an excess of kidneys does not move out of any one state/territory (TSANZ, 2009).

About 20% (1 in 5) of all kidneys are transferred according to this exchange program. The remaining 80% (4 in 5) kidneys are transplanted in the same state/territory where they were donated. NOMS calculates who should receive the kidneys in each state/territory, according to the jurisdiction's allocation formula. Each state/territory transplant service uses a slightly different formula to take into account differences in the number of people waiting for a transplant and other factors (TSANZ, 2009).

As long as the demand for kidneys exceeds the supply of donor organs, it is likely that controversy will exist regarding their allocation. Nurses can help by informing the public about organ donation and the allocation system and encouraging donation.

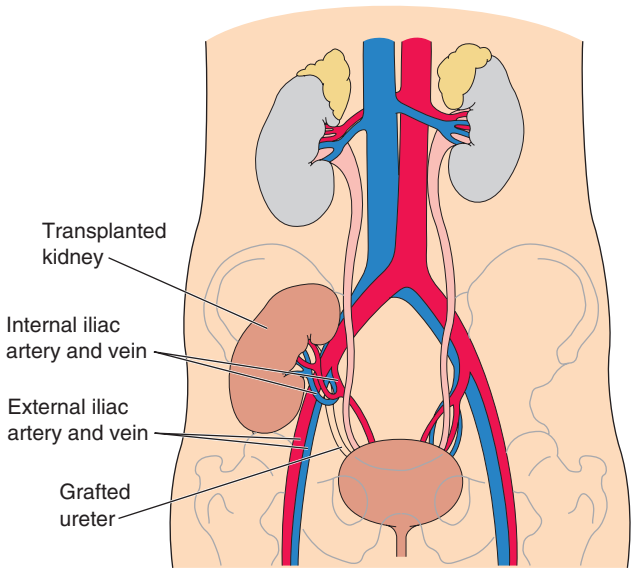


FIGURE 27.12 ■ Placement of a transplanted kidney in the iliac fossa with anastomosis to the hypogastric artery, iliac vein and bladder

The grafted kidney may stimulate an immune response to reject the transplanted kidney. Immunosuppressive drugs, such as tacrolimus, sirolimus, cyclosporin, azathioprine or mycophenolate mofetil, are commonly used to minimise the immune response. These drugs suppress a portion of the immune system and the inflammatory response, increasing the risk of infections and cancers with long-term therapy. The nursing implications of immunosuppressive therapy are outlined in Chapter 12.

During the period between 1990 and 2005 in Australia there was a sharp increase in the use of tacrolimus, mycophenolates and anti-CD25 antibodies (Chang et al., 2008). Glucocorticoids such as prednisone and methylprednisolone are used for both maintenance immunosuppression and to treat acute rejection episodes. Side effects of long-term corticosteroid use include impaired wound healing, emotional disturbances, osteoporosis and Cushingoid effects on glucose, protein and fat metabolism.

Azathioprine inhibits both cellular and humoral immunity. Because this drug is rapidly metabolised by the liver, the dose may not need to be altered in the presence of CKD. Bone marrow suppression, abnormalities of liver function and alopecia are the primary significant adverse effects for azathioprine. The action of mycophenolate mofetil is similar to that of azathioprine. Its advantages are minimal bone marrow suppression and increased potency in preventing or reversing rejection of the transplanted organ (Fauci et al., 2008). Sirolimus is not nephrotoxic and inhibits the response to interleukin-2 (IL-2), blocking activation of T and B cells.

Cyclosporin primarily affects cellular immunity, the T helper cells in particular. Among its many adverse effects, which include hepatotoxicity and hirsutism, nephrotoxicity is a primary concern for the kidney transplant recipient.

Even with immunosuppressive therapy, the transplanted kidney can be rejected at any time. Either acute or chronic rejection may develop. *Acute rejection* develops within months of the transplant. It is caused by a cellular immune response with T-lymphocyte proliferation (Porth & Matfin, 2009). Few manifestations may be apparent other than a rise in serum creatinine and possible oliguria. *Chronic rejection*, which may develop months to years following the transplant, is a major cause of graft loss. Both humoral and cellular immune responses are involved in chronic rejection. It does not respond to increased immunosuppression. The presenting manifestations of chronic rejection—progressive uraemia, proteinuria and hypertension—are those of progressive CKD.

Hypertension is a complication of kidney transplant, resulting from graft rejection, renal artery stenosis or renal vasoconstriction. People may develop glomerular lesions and manifestations of nephrosis. Hypertension and altered blood lipids (increased LDLs and decreased HDLs) increase the risk of death from myocardial infarction and stroke following transplant (Fauci et al., 2008).

Long-term immunosuppression has adverse effects. Infection is a continuing threat. Bacterial and viral infections may develop, as well as fungal infections of the blood, lungs and CNS. Tumours are also common, with carcinoma in situ of the cervix, lymphomas and skin cancers most prevalent. The risk of congenital anomalies is increased in infants whose mothers have undergone immunosuppressive therapy. Corticosteroid use may lead to bone problems, gastrointestinal disorders such as peptic ulcer disease and cataract formation.

Nursing care

Assessment

Both subjective and objective data are used to assess the person with CKD leading to ESKD:

- *Health history:* complaints of anorexia, nausea, weight gain or oedema; current treatment (if any), including type and frequency of dialysis or previous kidney transplant; chronic diseases such as diabetes, heart failure or kidney disease.
- *Physical examination:* mental status; vital signs, including temperature, heart and lung sounds, and peripheral pulses; urine output (if any); weight; skin colour, moisture, condition; presence of oedema (periorbital or dependent); bowel sounds; presence and location of an arteriovenous fistula or graft or peritoneal catheter.

Nursing diagnosis and interventions

Whether the person with ESKD is facing long-term dialysis, kidney transplantation or supportive care, a number of nursing care needs can be identified. This section focuses on nursing care related to impaired kidney function, nutritional deficits due to dietary restrictions and nausea, increased risk of

NURSING CARE OF THE PERSON receiving a kidney transplant

PREOPERATIVE CARE

- Provide routine preoperative care as outlined in Chapter 3.
- Assess knowledge and feelings about the procedure, answering questions and clarifying information as needed. Listen and address concerns about surgery, the source of the donor organ and possible complications. *Addressing concerns and reducing preoperative anxiety improve postoperative recovery.*
- Continue dialysis as ordered. *Intermittent or continuous renal replacement therapy may be necessary to manage fluid and electrolyte balance and prevent uraemia prior to surgery.*
- Administer immunosuppressive drugs as ordered before surgery. *Immunosuppression is initiated before transplantation to prevent immediate graft rejection.*

POSTOPERATIVE CARE

- Provide routine postoperative care as outlined in Chapter 3
- Maintain urinary catheter patency and a closed system. *Catheter patency is vital to keep the bladder decompressed and prevent pressure on suture lines. A closed drainage system minimises the risk of urinary tract infection.*
- Measure urine output every 30 to 60 minutes initially. Careful assessment of urine output helps determine fluid balance and transplant function. *Acute tubular necrosis is a common early complication, usually due to tissue ischaemia during the period between removal of the kidney from the donor and transplantation. Oliguria is an early sign.*
- Monitor vital signs and haemodynamic pressures closely. *Diuresis may occur immediately, resulting in hypervolaemia, low cardiac output and impaired perfusion of the transplanted kidney.*
- Maintain fluid replacement, generally calculated to replace urine output over the previous 30 or 60 minutes. *Fluid replacement is vital to maintain vascular volume and tissue perfusion.*
- Administer diuretics as ordered. *Loop and/or osmotic diuretics may be used to promote postoperative diuresis.*
- Remove the catheter within 2 to 3 days or as ordered. Encourage to void every 1 to 2 hours and assess frequently for signs of urinary retention following catheter removal. *The bladder may have atrophied prior to surgery, reducing its capacity. Urinary retention places stress on suture lines and increases the risk of infection.*
- Monitor serum electrolytes and renal function tests. *These tests are used to monitor graft function and fluid and electrolyte status. Electrolyte imbalances may develop as the transplanted kidney begins to function and diuresis occurs. Elevated serum creatinine and urea levels may be early signs of rejection or graft failure.*
- Monitor for possible complications:
  a. *Haemorrhage* from an arterial or venous anastomosis can be either acute or insidious. Indicators include swelling at the operative site, increased abdominal girth and signs of shock, including changes in vital signs and level of consciousness. *Haemorrhage is a surgical emergency, requiring prompt recognition and treatment to preserve the graft.*
  b. *Ureteral anastomosis failure* causes urine leakage into the peritoneal cavity. It may be marked by decreased urine output with abdominal swelling and tenderness. *Failure of the ureteral anastomosis requires surgical intervention.*
  c. *Renal artery thrombosis* is characterised by an abrupt onset of hypertension and reduced GFR. *Renal artery thrombosis can result in transplant failure.*
  d. *Infection* due to immunosuppression is an immediate and continuing risk. The inflammatory response is blunted and infection may not significantly elevate the temperature. Monitor for signs such as change in level of consciousness, cloudy or malodorous urine, or purulent drainage from the incision. *Prevention and prompt treatment of infections is particularly important in the immunosuppressed person.*
- Include the following in pre-discharge teaching for the person and family:
  a. The use and effects of prescribed medications, including antihypertensive medications, immunosuppressive agents, prophylactic antibiotics and others as ordered.
  b. Monitoring vital signs (including temperature) and weight.
  c. Manifestations of organ rejection, such as swelling and tenderness over the graft site, fever, joint aching, weight gain and decreased urinary output. Stress the importance of promptly reporting signs and symptoms to the person's nephrologist.
  d. Ordered or recommended dietary restrictions such as restricted carbohydrate and sodium intake and increased protein intake. Encourage good nutritional habits.
  e. Measures to prevent infection, such as avoiding crowds and obviously ill individuals.

  The person and family will manage care after discharge and therefore need a good understanding of what to expect, how to monitor graft status and measures to reduce the adverse effects of medications.
- Provide psychological support, address concerns and provide information as needed. The person has often been managing a chronic disease independently and is used to having a degree of control. *Providing information and allowing the person to retain control relieves anxiety and improves recovery.*

infection and changes in body image. Also see the nursing care plan below for nursing care of the person with ESKD.

Ineffective kidney perfusion

Capillaries are an integral part of the nephron. As nephrons are destroyed, kidney perfusion progressively declines. As kidney perfusion and nephron function fall, the kidney is less able to maintain fluid and electrolyte balance and eliminate waste products from the body.

- Monitor intake and output, vital signs including orthostatic blood pressures and weight. *These provide important data to identify changes in fluid volume.*

- Restrict fluids as ordered. *As kidney function declines, the ability to eliminate excess fluid is impaired.*

CONSIDERATION FOR PRACTICE

Weight changes are a more accurate indicator of fluid volume status in the oliguric or anuric person than intake and output measurements.

- Monitor respiratory status, including lung sounds as indicated. *Fluid volume overload may lead to heart failure and possible pulmonary oedema.*
- Monitor urea, serum creatinine, pH, electrolytes and FBC. Report significant changes. As kidney function declines, urea and serum creatinine increase. *Metabolic acidosis develops as the kidney is unable to eliminate hydrogen ions and conserve bicarbonate. Hyponatraemia, hyperkalaemia, hyperphosphataemia and hypocalcaemia are associated with kidney failure. The RBC count and haemoglobin decline due to deficient erythropoietin to stimulate cell production in the bone marrow. An acute fall in haemoglobin may also indicate GI bleeding, a risk in people with ESKD.*
- Report manifestations of electrolyte imbalances, such as cardiac arrhythmias and other ECG changes, muscle tremors and possible tetany and Kussmaul's respirations. *Manifestations of electrolyte imbalance may indicate the need for intervention.*

CONSIDERATION FOR PRACTICE

Monitor clarity of dialysate return. Dialysate should return clear in the person undergoing peritoneal dialysis. Cloudy dialysate may indicate peritonitis, the most common complication of peritoneal dialysis, and should be reported and cultured.

- Administer medications to treat electrolyte imbalances as ordered. *Medications may be prescribed to help maintain electrolyte and acid–base balance and prevent adverse effects of imbalances.*
- Administer antihypertensive medications as ordered. *Hypertension management is an important factor in slowing the progression of CKD.*
- Time activities and procedures to allow rest periods. *Anaemia associated with CKD may cause significant fatigue and activity intolerance.*

Imbalanced nutrition: less than body requirements

Anorexia, nausea and vomiting are common manifestations of ESKD and uraemia. The person often has a metallic taste and bad breath, which also diminish appetite. A diet restricted in protein and sodium will compound these problems. Food intake may be insufficient to meet metabolic needs. Catabolism, the breakdown of body proteins to meet energy needs, exacerbates uraemia.

- Monitor food and nutrient intake as well as episodes of vomiting. *Careful monitoring helps determine the adequacy of intake.*
- Weigh daily before breakfast. This provides the most accurate measurement. *Remember that a gain of 1 kg or more over a 24-hour period is more likely to reflect fluid retention than a gain in body mass.*
- Administer anti-emetic agents 30 to 60 minutes before eating. *Anti-emetics reduce nausea and the risk of vomiting with food intake.*
- Assist with mouth care prior to meals and at bedtime. *Mouth care improves taste, stimulates the appetite and maintains the integrity of oral mucous membranes.*
- Serve small meals and provide between-meal snacks. *Small meals are less likely to prompt nausea and help improve food intake*.
- Arrange for a dietary consultation. Provide preferred foods to the extent possible and involve the person in planning daily menus. Encourage family members to bring food as dietary restrictions allow. *Providing preferred foods within restrictions promotes intake.*
- Monitor nutritional status by tracking weight, laboratory values such as serum albumin, and urea and anthropometric measurements (see Chapters 20 and 21). *Indicators of impaired nutrition develop gradually and may be subtle. Careful assessment is important.*

Risk of infection

People with end-stage kidney disease have altered immune systems and leucocyte function and have increasing susceptibility to infection. Invasive devices required for haemodialysis or peritoneal dialysis add to this risk. The person who has had a kidney transplant remains on immunosuppressive therapy for life, further depressing the immune system and increasing the risk of infection.

- Use standard precautions and good handwashing technique at all times. Handwashing is a primary means of preventing the transfer of organisms. *People who are on haemodialysis have an increased risk of vancomycin-resistant* Enterococcus *(VRE), methicillin-resistant* Staphylococcus aureus *(MRSA), hepatitis B, hepatitis C and HIV infection.*
- Monitor temperature and vital signs at least every 4 hours. *A low-grade fever or increased pulse rate may indicate an infection in the immunosuppressed person.*
- Monitor WBC count and differential. *Increased WBCs may indicate a bacterial infection; decreased WBCs may indicate a viral infection. A shift in the differential showing more immature WBCs (bands) in circulation is another indicator of infection.*

CONSIDERATION FOR PRACTICE

Use strict aseptic technique when managing ports, catheters and incisions, to reduce the risk of introducing infectious organisms when immune responses are impaired.

- Culture peritoneal dialysis fluid and other drainage as indicated. *Culture is done to verify the presence of pathogens and actively treat early signs of peritonitis.*
- Provide good respiratory hygiene, including position changes, coughing and deep breathing. *These measures improve clearance of respiratory secretions, reducing the risk of infection.*

NURSING CARE PLAN A person with end-stage kidney disease

Walter Cohen, 45 years old, is the librarian at a local university. He has had type 1 diabetes since the age of 20 and was diagnosed with diabetic nephropathy 10 years ago. Despite blood pressure control, ACE inhibitor medications and frequent blood glucose monitoring with insulin coverage, he developed overt proteinuria 5 years ago and has now progressed to end-stage kidney disease. He is admitted to the nephrology ward and will undertake haemodialysis while waiting for a Tenckhoff catheter to be inserted to do CAPD. Mr Cohen's desire to continue working is the primary factor in his choice of CAPD over haemodialysis.

ASSESSMENT

Richard Gray, Mr Cohen's nurse, obtains a nursing assessment. Mr Cohen states that his diabetes has always been difficult to control. He has had numerous hypoglycaemic episodes and has been hospitalised 'four or five times' for ketoacidosis. Recently he has developed symptoms of peripheral neuropathy and increasing retinopathy. He attributed his lack of appetite, nausea, vomiting and fatigue over the past month to 'a touch of the flu'. His weight remained stable, so he did not worry about not eating much.

Physical assessment findings include T 36.5°C, P 96, R 20 and BP 178/100. His skin is cool and dry, with minor excoriations on forearms and lower legs. His breath has a strong offensive odour. Scattered fine rales noted in bilateral lung bases. He has bilateral pitting oedema of lower extremities to just below the knees; fingers and hands also oedematous. Abdominal assessment essentially normal, with hypoactive bowel sounds. Urinalysis shows a specific gravity of 1.011, gross proteinuria and multiple cell casts. FBC results: RBC 2.9 million/mm^3; haemoglobin 9.4 g/dL. Blood chemistry abnormalities include urea 32 mmol/L; creatinine 621 µmol/L; eGFR 9 mL/min; sodium 125 mmol/L; potassium 6.2 mmol/L; albumin 31 g/L; calcium 2.3 mmol/L; phosphate 3.5 mmol/L. A temporary jugular central venous dialysis catheter will be inserted for haemodialysis the next day, followed by a Tenckhoff catheter insertion later in the week.

DIAGNOSES

- *Excess fluid volume* related to failure of kidneys to eliminate excess body fluid.
- *Imbalanced nutrition: less than body requirements* related to effects of uraemia.
- *Impaired skin integrity* of lower extremities related to dry skin and itching.
- *Risk of infection* related to invasive catheters and impaired immune function.

PLANNING

- Plan fluid balance to a restriction of 750 mL per day.
- Plan a nutrition and fluid education program.
- Plan skin care program.
- Plan peritoneal exit site and catheter care education.

Expected outcomes

- Adhere to the prescribed fluid restriction of 750 mL per day.
- Demonstrate reduced extracellular fluid volume by weight loss, decreased peripheral oedema, clear lung sounds and normal heart sounds.
- Consume and retain 100% of prescribed diet, including snacks.
- Demonstrate healing of lower extremity skin lesions.
- Remain free of infection.
- Demonstrate appropriate peritoneal exit site and catheter care.

IMPLEMENTATION

- Space person's fluid intake. For example, 400 mL from 0700 to 1500, 200 mL from 1500 to 2300, and 100 mL from 2300 to 0700.
- Encourage and provide mouth care at least every 4 hours and before every meal.
- Weigh daily before breakfast; monitor vital signs every 4 hours.
- Document intake and output accurately.
- Arrange dietary consultation for menu planning.
- If required, administer prescribed anti-emetic 1 hour before meals.
- Monitor food intake, noting percentage and types of food consumed.
- Clean lesions on lower extremities every 8 hours and assess healing.
- If possible, begin to teach CAPD procedure and peritoneal exit site care.
- Assist to identify strengths and needs in health regimen management.

EVALUATION

Mr Cohen was hospitalised for 2 weeks, undergoing four haemodialysis sessions to reduce uraemic symptoms. An arteriovenous fistula was created in his left arm in case he should need haemodialysis in the future. He begins peritoneal dialysis the second week and by discharge he is able to manage the catheter care and dialysis runs with the help of his wife. His heart and lung sounds are normal and he has minimal peripheral oedema on discharge. The excoriations on his legs have healed. His temperature is normal and no evidence of infection is noted. Mr Cohen remains anorexic and slightly nauseated, but is eating most of his prescribed diet and snacks. He has lost 5 kg with excess fluid removal by dialysis, but his weight remains stable during the second week. Mr Cohen and his wife have been introduced to another person who has been on CAPD for several years and promises to help them with problem solving.

CRITICAL THINKING IN THE NURSING PROCESS

1. How does diabetes mellitus damage the kidneys and lead to ESKD? Why is this more significant for a person with type 1 diabetes than for someone with type 2 diabetes (see Chapter 19)?
2. Why do high levels of urea in the blood often cause changes in cognition and mental status? What manifestations of encephalopathy would you expect to see?
3. How might Mr Cohen's insulin dosage and diet need to be changed with the institution of peritoneal dialysis? Why?
4. Develop a care plan for the nursing diagnosis *Disturbed body image*.

REFLECTION ON THE NURSING PROCESS

1. What impact does Mr Cohen's cognitive status have on his ability to contribute to a planned education program?
2. How has the nursing process influenced Mr Cohen's nutrition status?

CONSIDERATION FOR PRACTICE

Monitor carefully for desired and adverse effects of all medications. Impaired kidney function affects drug elimination and increases the risk of toxic effects.

- Restrict visits from people who may be infective. Teach the person and family about the risk of infection and measures to reduce the spread of infection. *The person's resistance to infection is impaired, necessitating extra caution in preventing unnecessary exposures.*

Disturbed body image

Chronic disease and impaired kidney function can affect the person's body image. Haemodialysis requires an arteriovenous fistula or shunt; a permanent peritoneal catheter is required for peritoneal dialysis. While kidney transplant can restore an image of wholeness, a visible scar remains and the organ may be perceived as 'foreign'.

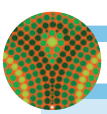
- Involve the person in care, including meal planning, dialysis, and catheter, port or incision care, to the extent possible. *Involvement improves acceptance and stimulates discussion about the effect of the disease and treatment measures on the person's life.*
- Encourage expression of feelings and concerns, accepting perceptions and feelings without criticism. *Self-expression enhances the person's self-worth and acceptance.* See the 'Translation to practice' box below.
- Include the person in decision making and encourage self-care. *Increased autonomy enhances the person's sense of control, independence and self-worth.*
- Support positive gains, but do not support denial. *The person may have difficulty accepting the kidney failure, but adaptation to the loss is important.*
- Help the person develop and achieve realistic goals. *Realistic goals allow the person to see progress.*
- Provide positive reinforcement and feedback. *These measures support growth and adaptation.*
- Reinforce effective coping strategies. *Reinforcement helps the person develop positive versus negative strategies for coping.*
- Facilitate contact with a support group, such as Kidney Health Australia or other community members. *The person benefits by providing and receiving support in a group of people going through similar circumstances.*
- Refer for mental health counselling as indicated or desired. *Counselling can help the person develop effective coping and adaptation strategies.*

Emerging nursing roles

Nurses have undertaken various roles in caring for people with kidney disease. Traditionally, these have involved renal ward care, haemodialysis care, peritoneal dialysis care and transplant care. Various specialty nursing roles have emerged to address the many areas that the person with ESKD has to address that have included vascular access, anaemia and transplant liaison (Schoch et al., 2014). These roles can be undertaken at an advanced practice nurse or nurse practitioner level (Bonner & Douglas, 2011).

The vascular access nurse, also known as renal access coordinator, has become a vital member of the renal team and can increase the proportion of people starting dialysis therapy with an AVF (Polkinghorne, Seneviratne & Kerr, 2009). The role includes organising timely surgical referral for AVF or AVG creation for haemodialysis or Tenchkoff insertion for peritoneal dialysis (Schoch et al., 2014). The vascular access nurse can also coordinate renal access surgical lists, access surveillance and vascular access education. Primarily the nurse provides a fluent communication channel between the renal services department, vascular surgery, allied health and other relevant units within the healthcare system.

The renal anaemia nurse, also known as the renal anaemia coordinator, provides specialised anaemia-related support for people living with CKD and ESKD. This support includes education for people with kidney disease, monitoring and



TRANSLATION TO PRACTICE Evidence-based practice: minimising hypotension during dialysis

Australian researchers explored the effects of pre-emptively pausing fluid removal to minimise dialysis hypotension (Bradshaw et al., 2011). Given that a sudden drop in blood pressure occurs in between 15% and 55% of all treatments, this is the most common serious side effect seen in haemodialysis. In an interventional pre- and post-test study involving 864 individual dialysis treatments, fluid removal was paused for a minimum of 10 minutes if the following criteria were met: (1) mean arterial pressure was less than or equal to 70 mmHg, or (2) the mean arterial pressure dropped by 30 mmHg or more. The results demonstrated that a pre-emptive nursing intervention decreased the odds of an intradialytic episode by 61% (Bradshaw et al., 2011).

IMPLICATIONS FOR NURSING

Pre-emptively pausing fluid removal according to specific mean arterial pressure parameters is a method to decrease intradialytic hypotensive episodes. This may be particularly useful for dialysis units without biofeedback or profiling machine technology.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Identify blood pressure assessment that is required throughout the dialysis treatment tools.
2. Identify a contextually appropriate intervention based on evidence to minimise hypotension.
3. Develop a teaching plan for people receiving dialysis, families and significant others to help them develop strategies to identify the early signs of hypotension.

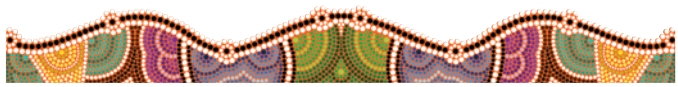
management of renal anaemia, administration of IV iron and blood transfusions, liaison with physicians, GPs and community nursing support, and education for nurses and other health professionals (Lee, 2009).

The transplant liaison nurse coordinates the evaluation and preparation of the person who has identified the request for a kidney transplant. Evaluation for kidney transplantation involves complete physical assessment to determine that the person is suitable to undergo a surgical procedure and to identify potential issues that will need to be managed post transplant (Wilden, 2008). Cardiovascular evaluation is especially important, as cardiovascular disease is the leading cause of death post transplant of people with functioning renal grafts (Siddiqu, Sundaram & Danovitch, 2005). It is also during the workup that contraindications to transplantation, such as malignancy, are identified. Preparation involves focused education, as the person's understanding, willingness and ability to manage their own care throughout the entire transplant process will have a major impact on the success and outcome of their transplant. Preparation also involves blood group typing and tissue typing. The transplant liaison nurse plays a pivotal role in encouraging the person to engage in healthy behaviours. Encouraging the person to be infection-free, adherent to medication and maintain a healthy body mass index is important to prevent post-transplant complications.

Community-based care

CKD and ESKD are long-term conditions that require self-management. No matter what treatment option is chosen (haemodialysis, peritoneal dialysis, kidney transplantation, supportive care), day-to-day management falls to the person and family. Teaching for home care includes the following topics:

- Nature of the kidney disease and kidney failure, including expected progression and effects.
- Monitoring weight, vital signs and temperature.
- Prescribed dietary and fluid restrictions. (Involve the person, a dietitian and the family member usually responsible for cooking. Include strategies to manage nausea and relieve thirst within allowed fluid limits.)
- How to assess and protect a fistula or graft for haemodialysis (or the extremity to be used if one is anticipated).
- Tenckhoff catheter care and the procedure for peritoneal dialysis as indicated. (Include a family member or significant other, in case the person is unable to perform the procedure independently at some time.)
- Following kidney transplant, prescribed medications, adverse effects and their management, infection prevention, graft protection and manifestations of organ rejection.
- Refer to a dietitian for diet planning and counselling. If home haemodialysis is planned, refer the designated dialysis helper for formal training. Kidney Health Australia can provide support and educational materials for the person with ESKD (visit www.kidney.org.au). Local and state trained representatives of these organisations can provide additional support.



CHAPTER HIGHLIGHTS

- Glomerulonephritis, inflammation of the glomerulus of the kidney, leads to loss of proteins and blood cells in the urine, a decrease in the glomerular filtration rate and severe oedema.
- The renal and cardiovascular systems are closely interrelated. Vascular disorders, such as hypertension, renal artery stenosis or obstruction of the renal artery or vein, can have serious consequences in terms of kidney function.
- Acute kidney injury is a frequent complication of critical illnesses, typically occurring in people with no prior history of kidney disorders. Ischaemic and nephrotoxic damage to the kidney are the most common precipitating factors for AKI.
- Chronic kidney disease is the end-stage of numerous systemic and kidney disorders, such as diabetes mellitus, systemic lupus erythematosus and chronic glomerulonephritis.
- When the kidneys are no longer able to maintain homeostasis, kidney replacement therapies are necessary to eliminate metabolic waste products and sustain life. Dialysis and kidney transplant are the primary kidney replacement therapies used.
- Home dialysis therapy is the preferred treatment for people with ESKD.

CONCEPT CHECK

1 The physician orders digoxin 0.125 mg 3 times per week for an 82-year-old person with heart failure. The nurse should:

1. question the order because older people frequently require larger doses of the drug due to impaired ability of the kidneys to concentrate urine
2. administer the drug as ordered, monitoring the person for manifestations of toxicity
3. assess the person's urine specific gravity and pH before administering the drug at this dose
4. use 0.25 mg digoxin tablets, cutting the tablet in half to save money for the person

2 A person newly diagnosed with polycystic kidney disease asks if there is anything his children need to know about their risk of getting the disorder. The appropriate response by the nurse is:

1. because the condition was just diagnosed, it is due to a new genetic mutation and there is no risk of passing the condition on to his children
2. when his children prepare to marry, they and their potential partners should undergo genetic testing to determine if their children will be at risk
3. the adult form of this disorder is transmitted as a dominant gene; each child has a 50% risk of having inherited the defective gene

4 his children would have developed symptoms of the disorder in utero or shortly after birth if they had inherited the defective gene

3 In obtaining a nursing history from a 22-year-old person admitted with a diagnosis of acute glomerulonephritis, the nurse specifically asks the person about a recent history of which of the following?
1 urinary tract infection
2 strep throat
3 x-ray using contrast media
4 illicit drug use

4 The nurse evaluates his teaching for a person with acute glomerulonephritis as effective when the person:
1 chooses soy or animal proteins for allowed grams of protein in diet
2 states the need to remain on bed rest until his urine returns to clear yellow
3 demonstrates care for the vascular shunt or peritoneal catheter
4 limits fluid intake to less than 1500 mL per day

5 Appropriate postoperative nursing interventions for the person who has had a partial or total nephrectomy include:
1 connecting all catheters and drains to a single collection device
2 routine irrigation of all catheters with sterile normal saline
3 administering cough suppressant medication as needed
4 labelling and securing all catheters, tubes and drains

6 Important nursing interventions to prevent acute kidney dysfunction in the critically ill person include:
1 maintaining fluid volume and cardiac output
2 avoiding all potentially nephrotoxic drugs
3 administering antihypertensive drugs
4 assessing for a history of diabetes or systemic lupus erythematosus

7 The nurse evaluates their teaching as effective when the person recovering from acute kidney dysfunction states that he will:
1 limit his fluid intake to 1500 mL or less per day
2 consume only vegetable proteins
3 avoid taking drugs that may be nephrotoxic
4 self-catheterise for residual urine at least once a week

8 The nurse caring for a person preparing to undergo haemodialysis includes which of the following in the plan of care? (Select all that apply.)
1 Obtain weight and orthostatic vital signs.
2 Assess blood pressure of extremity where fistula has been created.
3 Monitor serum creatinine, urea and haemoglobin levels.
4 Determine urine specific gravity and pH.
5 Restrict fluid and protein intake.

9 An appropriate goal of nursing care for a person with end-stage kidney disease is the person will be able to:
1 identify a live-in caregiver
2 state the advantages and disadvantages of haemodialysis, peritoneal dialysis and kidney transplant as kidney replacement therapies
3 demonstrate the ability to independently perform haemodialysis in the home
4 relate the hospice philosophy and identify indicators of the need for hospice care

10 Following a kidney transplant, the nurse notes that the person's urine is cloudy. The most appropriate response by the nurse is to:
1 record the finding
2 increase the intravenous flow rate
3 irrigate the urinary catheter
4 notify the physician

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UNIT 7 BUILDING CLINICAL COMPETENCE

Responses to altered urinary elimination

CLINICAL SCENARIO

You have been assigned to work with the following four people for the 0700 shift on a renal medical–surgical unit. Significant data obtained during report are as follows:

- Phillip Connor is a 45 year old who was admitted 2 days ago after a fall from a ute. He experienced a bruised right kidney and numerous ecchymotic areas on his right side from the fall. His vital signs are T 37.2°C, P 98, R 28, BP 110/68. He is complaining of abdominal pain and difficulty urinating.
- Agnes Gibson is an 84 year old who was admitted 2 hours ago with manifestations of urinary incontinence, anorexia, confusion and lethargy. Her vital signs on admission were T 36.1°C, P 88, R 20, BP 148/90. The physician ordered trimethoprim-sulfamethoxazole (Bactrim) to be started as soon as possible.
- Joseph Rouse is a 45 year old who is to undergo surgery for removal of uric acid stones after having a failed lithotripsy. His vital signs are T 37.5°C, P 94, R 24, BP 112/68. His skin is pale, cool and clammy. He is complaining of nausea, severe left-sided flank pain with spasms and light headedness.
- Angela Baldwin is a 34 year old who has a medical history of systemic lupus erythematosus (SLE). She was admitted with complaints of left flank pain and generalised oedema. Urinalysis results indicate haematuria and proteinuria. Vital signs are T 37.8°C, P 88, R 26, BP 144/90. She is admitted for aggressive immunosuppressive therapy.

Critical thinking questions

1 In what order would you visit these people after report?

1. _____
2. _____
3. _____
4. _____

2 What top two priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Phillip Connor		
Agnes Gibson		
Joseph Rouse		
Angela Baldwin		

3 The physician ordered trimethoprim-sulfamethoxazole (Bactrim) for Agnes Gibson's uncomplicated cystitis. Mrs Gibson understands the length of antibiotic therapy when she verbalises which statement?

1. 'I will need to complete the full course of Bactrim.'
2. 'I can be discharged after 5 days of antibiotics.'
3. 'I need to stay in the hospital for 1 week to finish the antibiotics.'
4. 'I can stay in the hospital for 5 days of antibiotics and take 5 days of antibiotics at home.'

4 The physician orders phenazopyridine (Pyridium) for relief of pain and burning with cystitis. Which does the nurse teach the person about the use of this medication?

1. Take the medication with antacids to prevent stomach upset.
2. Drink less fluid to allow the drug to concentrate in the bladder.
3. Wear a sanitary pad to protect your clothing from stains while taking this drug.
4. Stop taking the drug if nausea and diarrhoea occur.

5 The person diagnosed with uric acid stones is ordered to follow a diet low in purines. Which is the meal plan lowest in purines?

1. liver with onions and potatoes
2. chicken burger with chips
3. spaghetti with bolognese sauce
4. macaroni and cheese with stewed tomatoes

6 When assessing a person with glomerulonephritis, which manifestations are indicative of an early disease process?

1. pyuria, leucocytosis and hyperthermia
2. haematuria, proteinuria and hypertension
3. dysuria, hyperglycaemia and hypertension
4. oliguria, flank pain and hypotension

7 Which are risk factors of urinary tract infections? (Select all that apply.)

1. circumcision in males
2. decreased cervicovaginal antibodies
3. sexual intercourse in women
4. short urethra in men
5. ageing in men
6. urinary catheterisation

8 Which is the most accurate indicator of fluid volume status in the oliguric or anuric person?

1. intake and output
2. weight changes
3. restricted fluids
4. urea and creatinine levels

9 The most reliable diagnostic procedure to determine glomerular disorders is which diagnostic test?

1. kidney scan
2. antistreptolysin O titre
3. kidney biopsy
4. blood urea nitrogen

10 A female postoperative person is complaining of inability to void. A bladder ultrasound indicates that there is a significant amount of urine in the bladder. Which interventions does the nurse perform?

1. Insert a urinary catheter and completely drain the bladder at once.
2. Insert a urinary catheter and drain urine in 500 mL increments.

3. Ambulate person to the bathroom to try to void and run water in the sink.
4. Give the person a glass of water to drink to encourage voiding.

11 Which nursing actions are instituted for the person with kidney trauma?

1. Monitor level of consciousness and urine output.
2. Monitor vital signs for hypotension and bradycardia.
3. Observe for hypertension and check urine for haematuria.
4. Observe urine for oliguria and proteinuria.

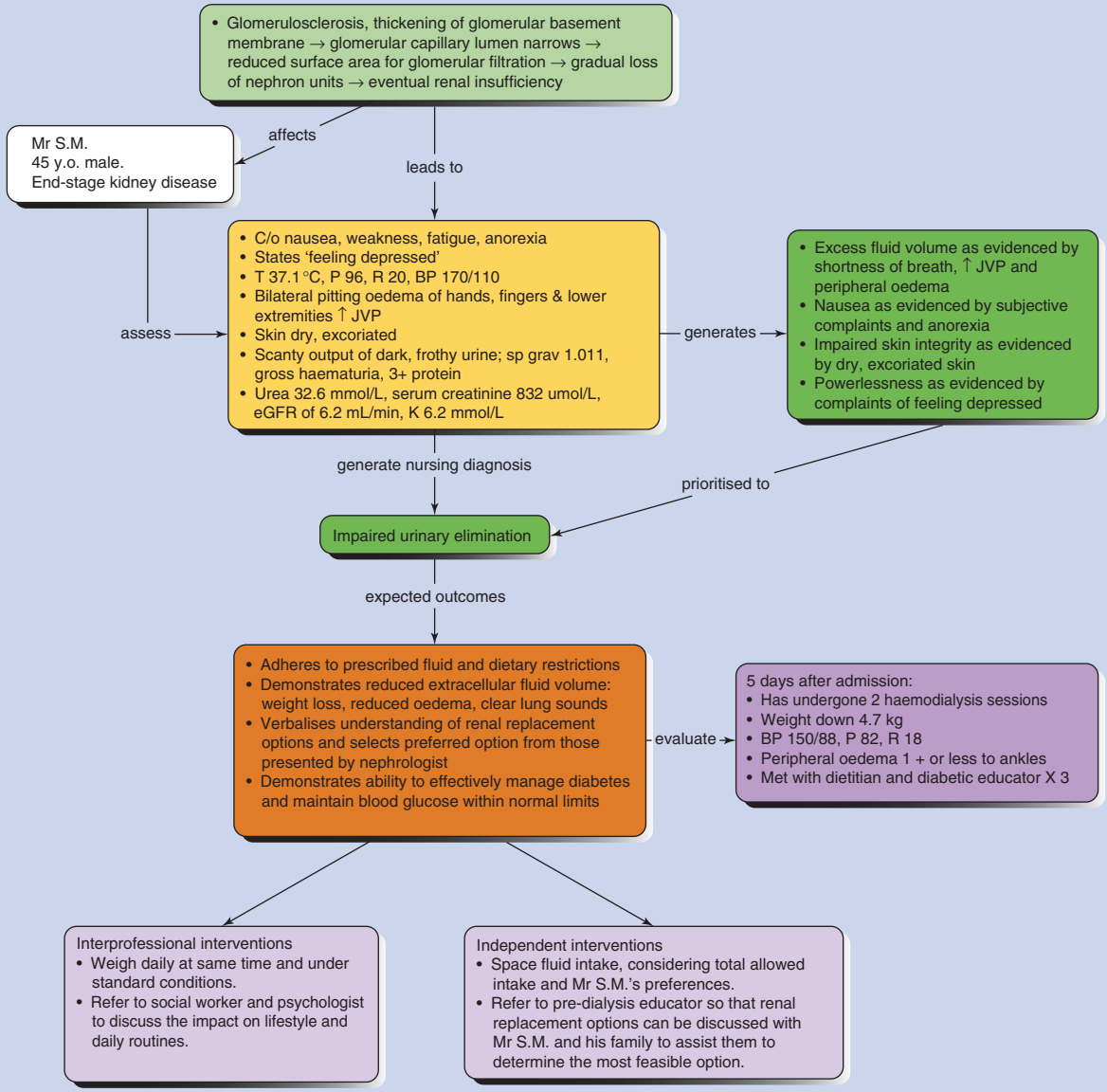
12 After a person has returned from surgery, the nurse needs to report which urinary output?

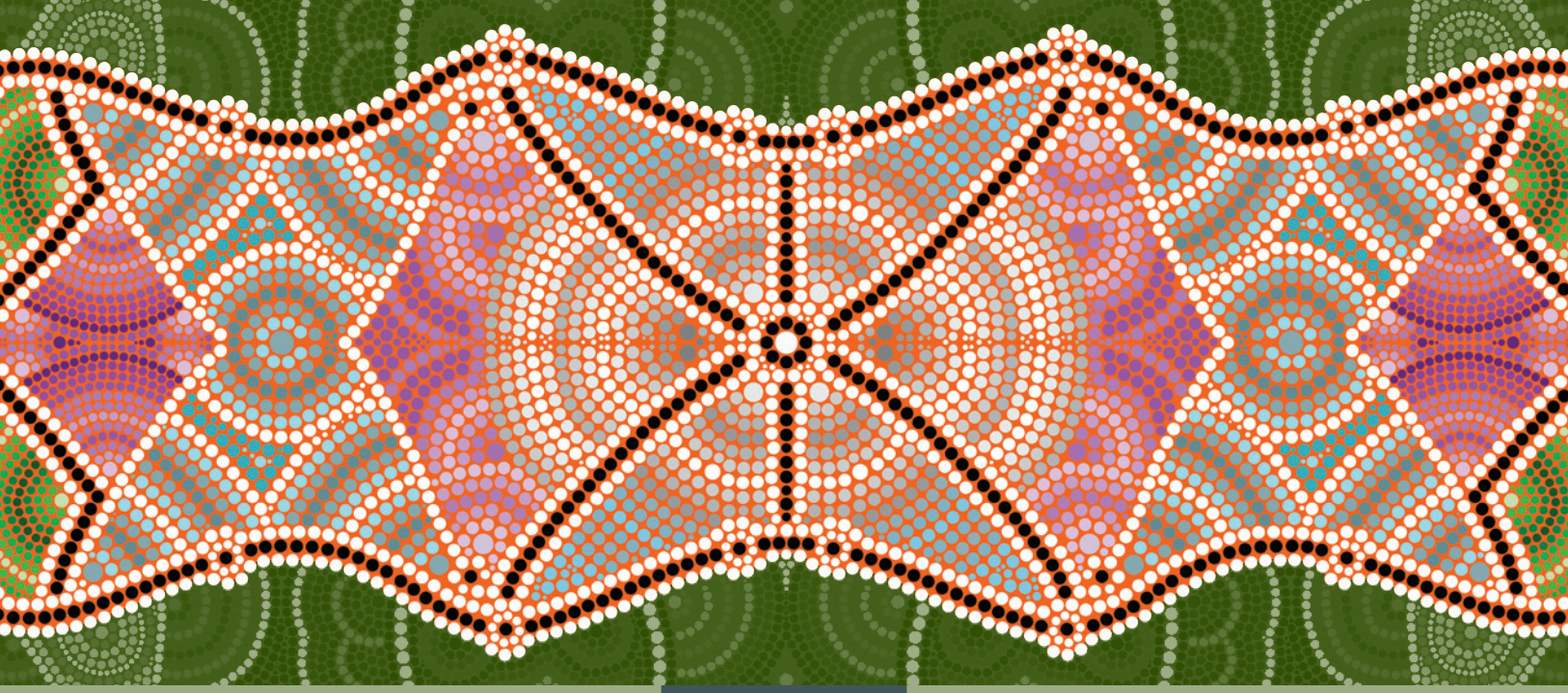
1. 20 mL per hour
2. 40 mL per hour
3. 300 mL per 8 hours
4. 400 mL per 8 hours

CASE STUDY

Steven McEvoy is a 45-year-old Indigenous male who has been a truck driver for the past 20 years. Increasing shortness of breath has prompted him to attend the ED. He is admitted to the hospital with a further history of nausea for several weeks, weakness, fatigue and loss of appetite. He has been feeling very depressed. He has a past medical history of type 1 diabetes mellitus, hypertension and diabetic nephropathy. On admission, his vital signs are T 37.1°C, P 96, R 20, BP 170/110. He has bilateral pitting oedema of the lower extremities. His fingers and hands are also oedematous. He complains of dry and itching skin. His urine is dark, frothy and scanty. A specimen is collected for a urinalysis and blood is taken and sent to the laboratory. Results returned with the urinalysis showing a specific gravity of 1.011, gross haematuria and 3+ protein. His blood work reveals a urea of 32.6 mmol/L, creatinine of 832 µmol/L and eGFR of 6.2 mL/min. Based on his past medical history of diabetes, hypertension and diabetic nephropathy and the current findings, a medical diagnosis of end-stage kidney disease is established.

Based on Mr McEvoy's assessment and past medical history, the nursing diagnosis of *Impaired urinary elimination* is identified as one of the high priorities for planning nursing care.





UNIT 8

RESPONSES TO ALTERED CARDIOVASCULAR FUNCTION

CHAPTER 28

A PERSON-CENTRED APPROACH TO ASSESSING THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

PENNY PALIADELIS

KEY TERMS

LEARNING OUTCOMES

- Describe the anatomy and physiology of the cardiovascular and lymphatic systems.
- Examine investigations and observations important for assessing a person's cardiovascular and lymphatic system function.
- Accurately interpret normal and aberrant data gained from assessment of a person's cardiovascular and lymphatic system.
- Discuss manifestations of impaired cardiovascular and lymphatic systems.

CLINICAL COMPETENCIES

- Assess an ECG strip and identify normal cardiac function and abnormal rhythm.
- Conduct and document a health history for people having or at risk of alterations in the structure and function of the cardiovascular, haematological or lymphatic systems.
- Conduct and document a physical assessment of cardiovascular, haematological and lymphatic status.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Stethoscope with diaphragm and bell
- Blood pressure cuff
- Tape measure
- Metric ruler
- Doppler ultrasound device (if needed) and transducer gel

The cardiovascular system is comprised of the heart (the system's pump), the peripheral vascular system (a network of arteries, veins and capillaries) and the haematological system (blood and blood components). The lymphatic system (the lymph, lymph nodes and spleen) is a special vascular system that helps maintain sufficient blood volume in the cardiovascular system by picking up excess tissue fluid and returning it to the bloodstream. The heart, a muscular pump, beats an average of 70 times per minute, or once every 0.86 seconds, every minute of a person's life. This continuous pumping moves blood through the body, nourishing tissue cells and removing wastes. Deficits in the structure or function of the heart affect all body tissues. Changes in cardiac rate, rhythm or output may limit almost all human functions, including self-care, mobility, and the ability to maintain tissue perfusion, fluid volume status, respirations and comfort. Cardiac changes may also affect self-concept, sexuality and role performance.

STRUCTURE AND FUNCTION OF THE CARDIOVASCULAR SYSTEM

The heart is a hollow, cone-shaped organ approximately the size of an adult's fist, weighing less than 500 grams. It is located in the mediastinum of the thoracic cavity, between the vertebral column and the sternum and is flanked laterally by the lungs. Two-thirds of the heart mass lies to the left of the sternum; the upper base lies beneath the second rib, and the pointed apex is approximate with the fifth intercostal space, midpoint to the clavicle (see Figure 28.1).

The heart is covered by the pericardium, a double layer of fibroserous membrane (see Figure 28.2). The pericardium encases the heart and anchors it to surrounding structures, forming the pericardial sac. The snug fit of the pericardium prevents the heart from overfilling with blood. The outermost layer is the parietal pericardium, and the visceral pericardium (or epicardium) adheres to the heart surface. The small space between the visceral and parietal layers of the pericardium is called the pericardial cavity. A serous lubricating fluid produced in this space cushions the heart as it beats.

The heart wall consists of three layers of tissue: the epicardium, the myocardium and the endocardium (see Figure 28.2). The epicardium covers the entire heart and great vessels and then folds over to form the parietal layer that lines the pericardium and adheres to the heart surface. The myocardium, which is the middle layer of the heart wall, consists of specialised cardiac muscle cells (myofibrils) that provide the bulk of contractile heart muscle. The endocardium, which is the innermost layer, is a thin membrane composed of three layers; the innermost layer is made up of smooth endothelial cells that line the inside of the heart's chambers, the great vessels and the valves.

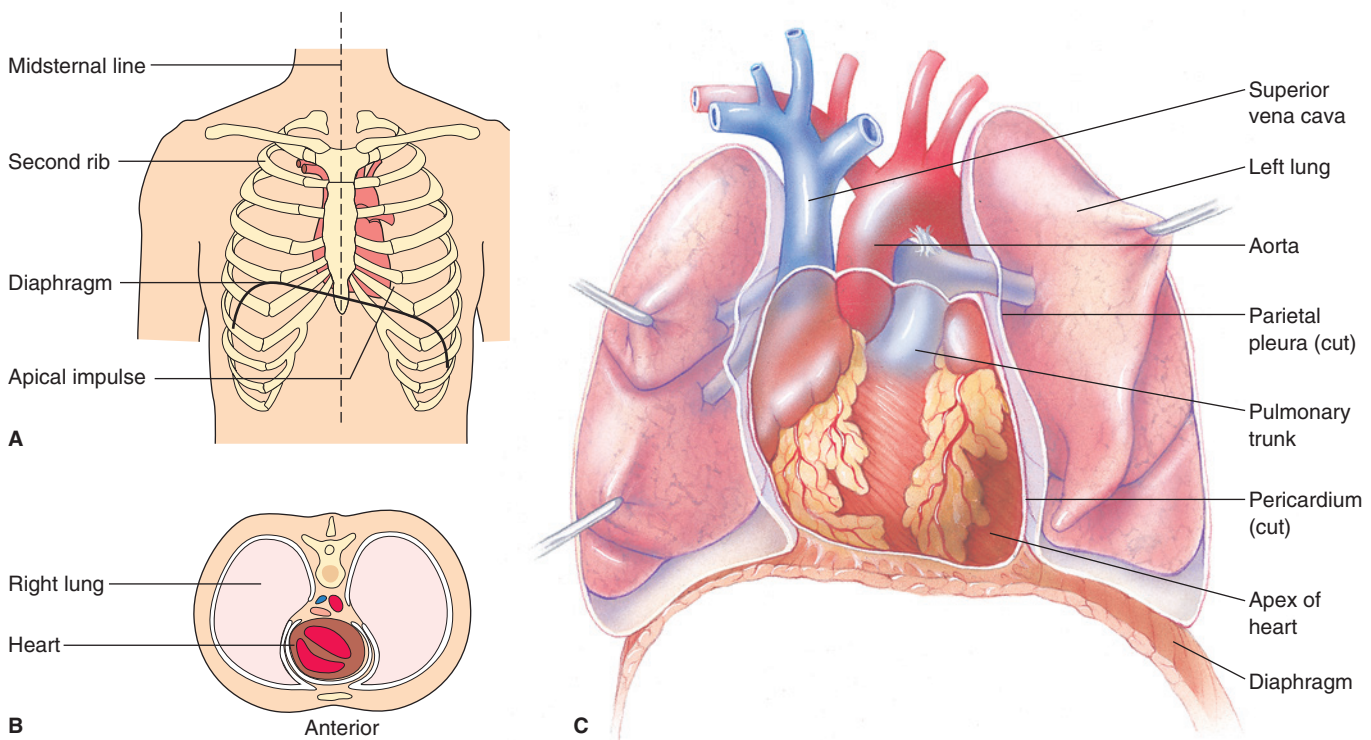


FIGURE 28.1 ■ Location of the heart in the mediastinum of the thorax. *A*, Relationship of the heart to the sternum, ribs and diaphragm. *B*, Cross-sectional view showing relative position of the heart in the thorax. *C*, Relationship of the heart and great vessels to the lungs

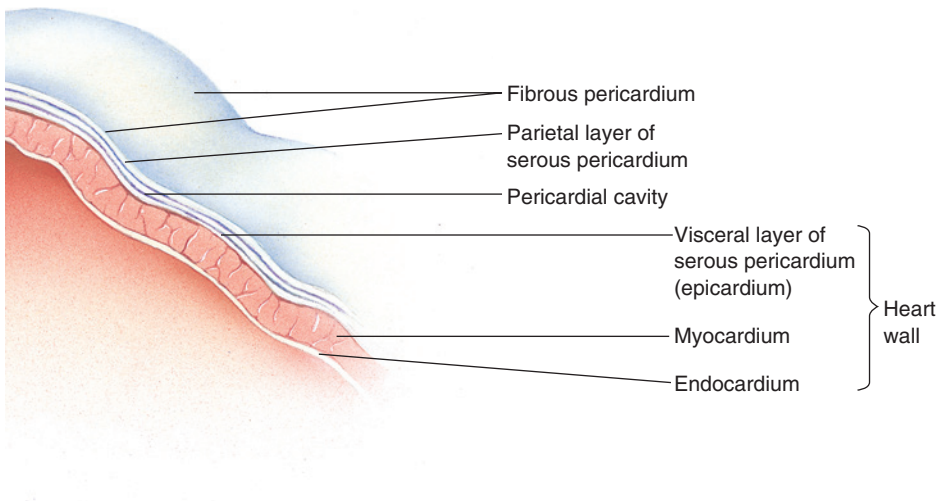


FIGURE 28.2 ■ Coverings and layers of the heart

Chambers and valves of the heart

The heart has four hollow chambers, two upper atria and two lower ventricles. They are separated longitudinally by the interventricular septum (see Figure 28.3).

The right atrium receives oxygen-poor blood from the veins of the body. (Note that the terms 'deoxygenated' and 'oxygen-poor' are often used interchangeably; however, 'oxygen-poor' is more technically correct. Haemoglobin will never become fully devoid of all oxygen—i.e. 'deoxygenated'—even at the tissues.) The superior vena cava returns blood from the body area above the diaphragm, the inferior vena cava returns blood from the body below the diaphragm, and the coronary sinus drains blood from the heart. The left atrium receives freshly oxygenated blood from the lungs through the pulmonary veins.

The right ventricle receives oxygen-poor blood from the right atrium and pumps it through the pulmonary artery to

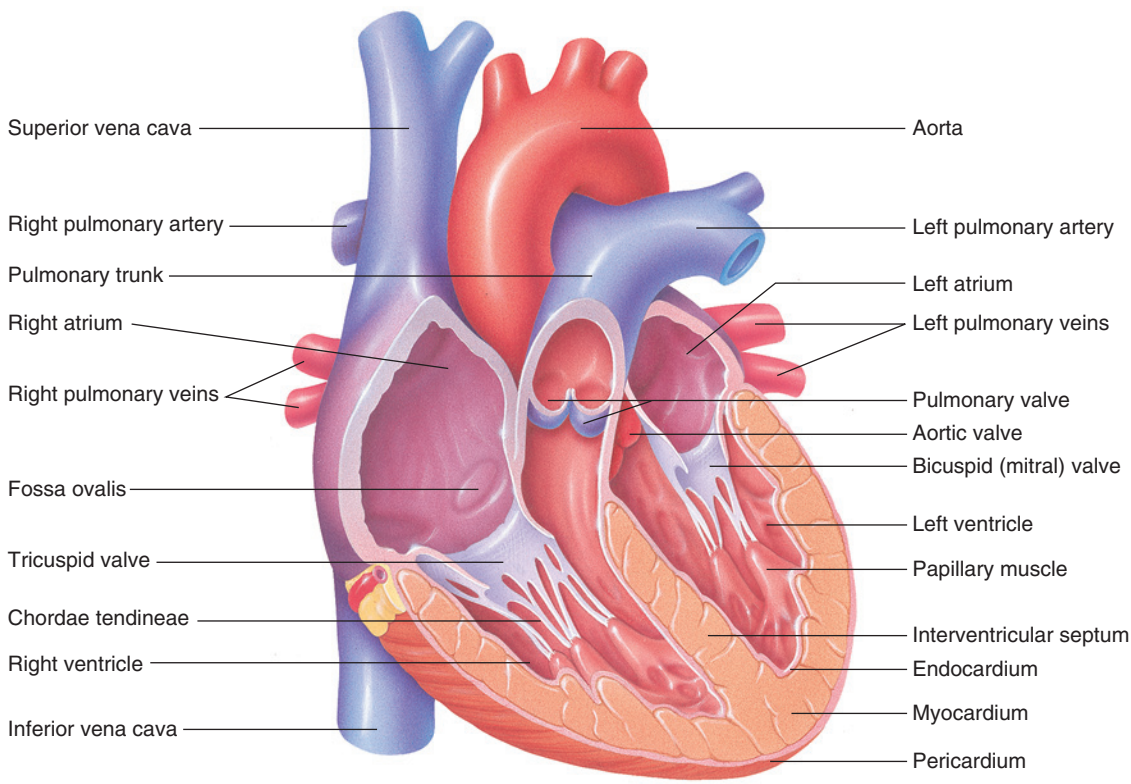


FIGURE 28.3 ■ The internal anatomy of the heart, frontal section

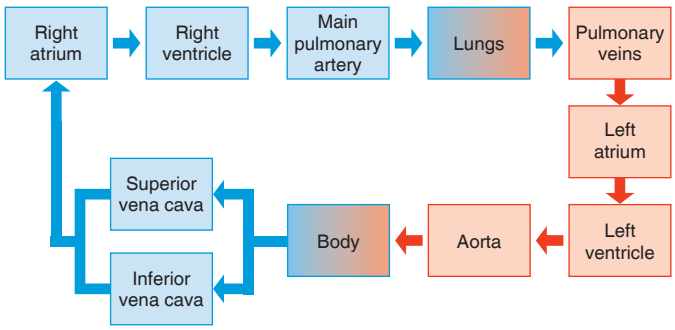


FIGURE 28.4 ■ Blood flow around the systemic circuit

the pulmonary capillary bed for oxygenation. The newly oxygenated blood then travels through the pulmonary veins to the left atrium. Blood enters the left atrium and crosses the mitral (bicuspid) valve into the left ventricle. Blood is then pumped out of the aorta to the arterial circulation (see Figure 28.4).

The chambers of the heart are separated by valves that allow unidirectional blood flow to the next chamber or great vessel. The atria are separated from the ventricles by the two atrioventricular (AV) valves; the tricuspid valve is on the right side and the bicuspid (or mitral) valve is on the left. The flaps of each of these valves are anchored to the papillary muscles of the ventricles by the chordae tendineae. These structures control the movement of the AV valves to prevent backflow of blood. The ventricles are connected to their great vessels by the semilunar valves. On the right, the pulmonary (pulmonic) valve joins the right ventricle with the pulmonary artery. On the left, the aortic valve joins the left ventricle to the aorta (see Figure 28.5).

Closure of the AV valves at the onset of contraction (systole) produces the first heart sound, or S_1 (characterised by the syllable 'lub'); closure of the semilunar valves at the onset of relaxation (diastole) produces the second heart sound, or S_2 (characterised by the syllable 'dub').

Systemic, pulmonary and coronary circulation

Because each side of the heart both receives and ejects blood, the heart is often described as a double pump. Blood enters the right atrium and moves to the pulmonary bed at almost the exact same time that blood is entering the left atrium. The circulatory system has two parts: the pulmonary circulation (moving blood through the capillary bed surrounding the lungs to link with the gas exchange system of the lungs) and the systemic circulation, which supplies blood to all other body tissues. In addition, the heart muscle itself is supplied with blood via the coronary circulation.

Systemic circulation

The systemic circulation consists of the left side of the heart, the aorta and its branches, the capillaries that supply the brain and peripheral tissues, the systemic venous system and the vena cavae. The systemic system, which must move blood to peripheral areas of the body, is a high-pressure system.

Pulmonary circulation

The pulmonary circulation consists of the right side of the heart, the pulmonary artery, the pulmonary capillaries and the pulmonary veins. Because it is located in the thorax near the heart, the pulmonary circulation is a low-pressure system. Pulmonary circulation begins with the right side of the heart. Oxygen-poor blood from the venous system enters the right atrium through two large veins, the superior and inferior venae cavae, and is transported to the lungs via the pulmonary artery and its branches (see Figure 28.6). After oxygen and carbon dioxide are exchanged in the pulmonary capillaries, oxygen-rich blood returns to the left atrium through several pulmonary veins. Blood is then pumped out of the left ventricle through the aorta and its major branches to supply all body tissues. This second circuit of blood flow is called the systemic circulation.

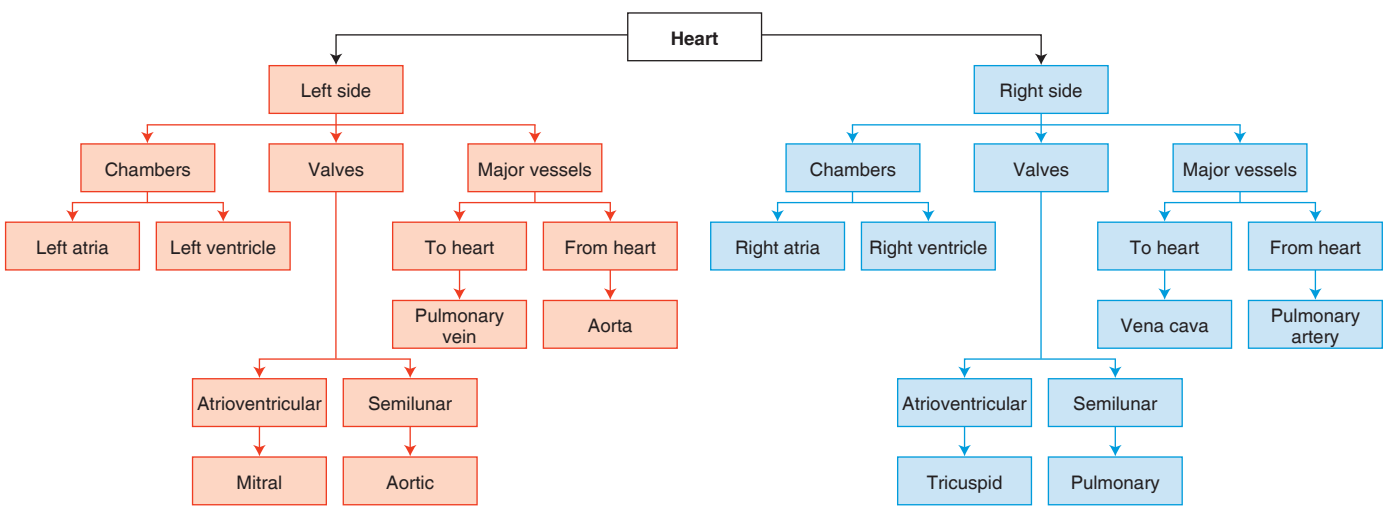


FIGURE 28.5 ■ Summary of key components of the heart

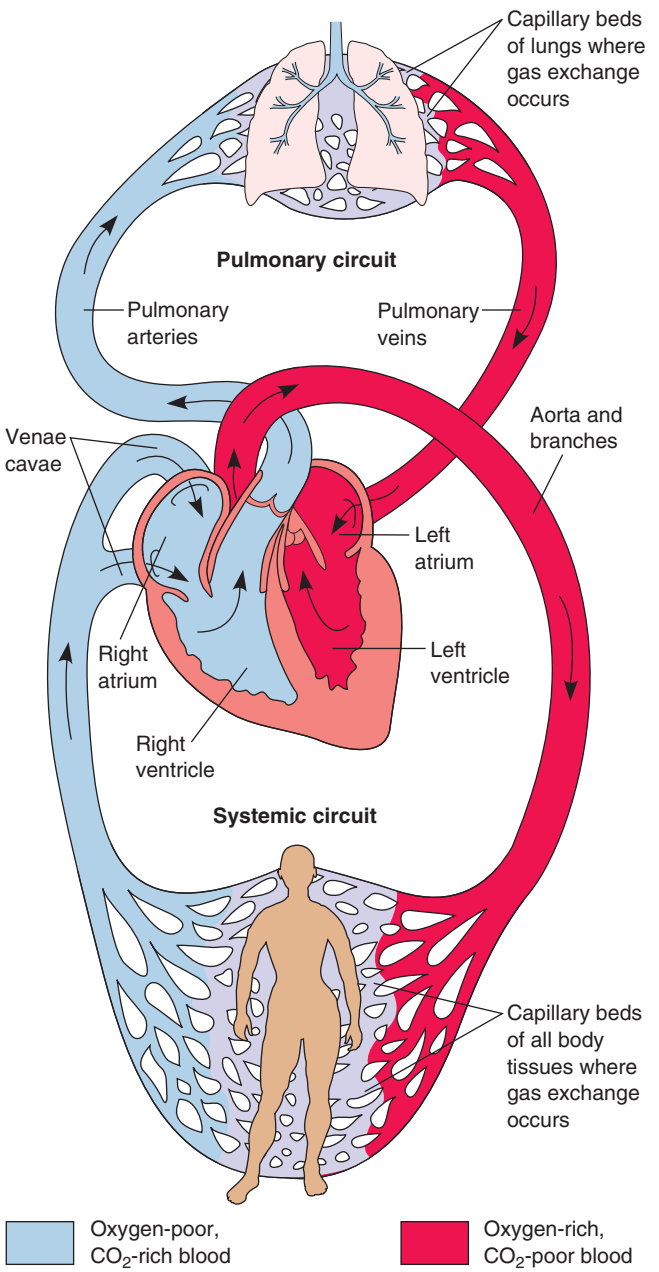


FIGURE 28.6 ■ Pulmonary and systemic circulation

Coronary circulation

The heart muscle itself is supplied by its own network of vessels through the coronary circulation. The left and right coronary arteries originate at the base of the aorta and branch out to encircle the myocardium (see Figure 28.7A), supplying blood, oxygen and nutrients to the myocardium. The left main coronary artery divides to form the anterior descending and circumflex arteries. The anterior descending artery supplies the anterior interventricular septum and the left ventricle. The circumflex branch supplies the left lateral wall of the left ventricle. The right coronary artery supplies the right ventricle and forms the posterior descending artery. The posterior descending artery supplies the posterior portion of the heart. While ventricular contraction delivers blood through the pulmonary circulation and the systemic circulation, it is during ventricular relaxation that the coronary arteries fill with oxygen-rich blood. After the blood perfuses the heart muscle, the cardiac veins drain the blood into the coronary sinus, which empties into the right atrium of the heart (see Figure 28.7B).

Blood flow through the coronary arteries is regulated by several factors. Aortic pressure is the primary factor. Other factors include the heart rate (most flow occurs during diastole, when the muscle is relaxed), metabolic activity of the heart and blood vessel tone (constriction).

The cardiac cycle and cardiac output

The contraction and relaxation of the heart constitutes one heartbeat and is called the cardiac cycle (see Figure 28.8). Ventricular filling is followed by ventricular systole, a phase during which the ventricles contract and eject blood into the pulmonary and systemic circuits. Systole is followed by a relaxation phase known as diastole, during which the ventricles refill, the atria contract and the myocardium is perfused. Normally, the complete cardiac cycle occurs about 70 to 80 times per minute, measured as the heart rate (HR).

During diastole the volume in the ventricles is increased to about 120 mL (the end-diastolic volume), and at the end of systole about 40 mL of blood remains in the ventricles (the end-systolic volume). The difference between the end-diastolic volume and the end-systolic volume is called the **stroke volume (SV)** (see Figure 28.9). Stroke volume ranges from 60 to 100 mL/beat and averages about 80 mL/beat in an adult. The **cardiac output (CO)** is the amount of blood pumped by the ventricles into the pulmonary and systemic circulations in 1 minute. Multiplying the stroke volume (SV) by the heart rate (HR) determines the cardiac output: CO = SV × HR. The **ejection fraction** is the stroke volume divided by the end-diastolic volume and represents the fraction or percentage of the diastolic volume that is ejected from the heart during systole (Mohrman & Heller, 2010). For example, an end-diastolic volume of 120 mL divided by a stroke volume of 80 mL equals an ejection fraction of 66% (see Figure 28.9). The normal ejection fraction ranges from 50% to 70%.

The average person's cardiac output ranges from 4 to 8 L/min. Cardiac output is an indicator of how well the heart is functioning as a pump. If the heart cannot pump effectively, cardiac output and tissue perfusion are decreased. Body tissues that do not receive enough blood and oxygen (carried in the blood on haemoglobin) become **ischaemic** (deprived of oxygen). If the tissues do not receive enough blood flow to maintain the functions of the cells, the cells die. (Cellular death results in necrosis or infarction.)

Activity level, metabolic rate, physiological and psychological stress responses, age and body size all influence cardiac output. In addition, cardiac output is determined by the interaction of four main factors: heart rate, preload, afterload and contractility. Changes in each of these variables influence cardiac output intrinsically and each can also be manipulated to affect cardiac output. The heart's ability to respond to the body's changing need for cardiac output is called **cardiac reserve**.

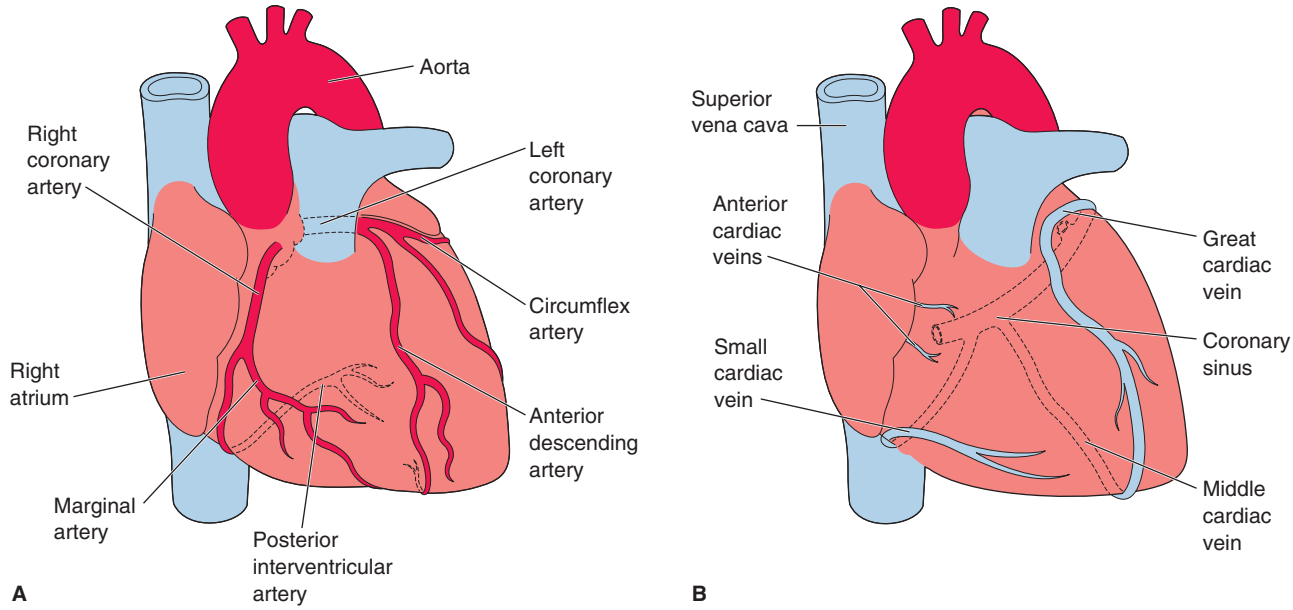


FIGURE 28.7 ■ Coronary circulation. *A*, Coronary arteries; *B*, coronary veins

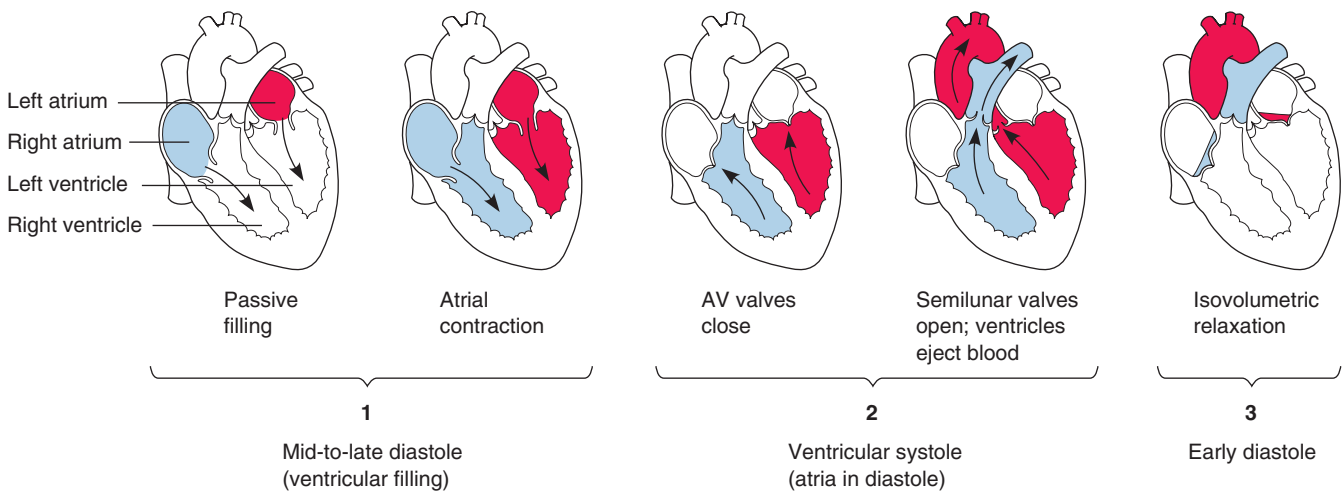


FIGURE 28.8 ■ The cardiac cycle has three events: (1) ventricular filling in mid-to-late diastole, (2) ventricular systole, and (3) isovolumetric relaxation in early diastole

Heart rate

Heart rate is affected by both direct and indirect autonomic nervous system stimulation. Direct stimulation is accomplished through the innervation of the heart muscle by sympathetic and parasympathetic nerves. The sympathetic nervous system increases the heart rate, whereas the parasympathetic vagal tone slows the heart rate. Reflex regulation of the heart rate in response to systemic blood pressure also occurs through activation of sensory receptors known as baroreceptors or pressure receptors located in the carotid sinus, aortic arch, venae cavae and pulmonary veins.

If heart rate increases, cardiac output increases (up to a point) even if there is no change in stroke volume. However, rapid heart rates decrease the amount of time available for ventricular filling during diastole. Cardiac output then falls because decreased filling time decreases stroke volume. Coronary artery perfusion also decreases because the coronary arteries fill primarily during diastole. Cardiac output decreases during bradycardia if stroke volume stays the same because the number of cardiac cycles is decreased.

Contractility

Contractility is the inherent capability of the cardiac muscle fibres to shorten. Poor contractility of the heart muscle reduces the forward flow of blood from the heart, increases the ventricular pressures from accumulation of blood volume and reduces cardiac output. Increased contractility may stress the heart.

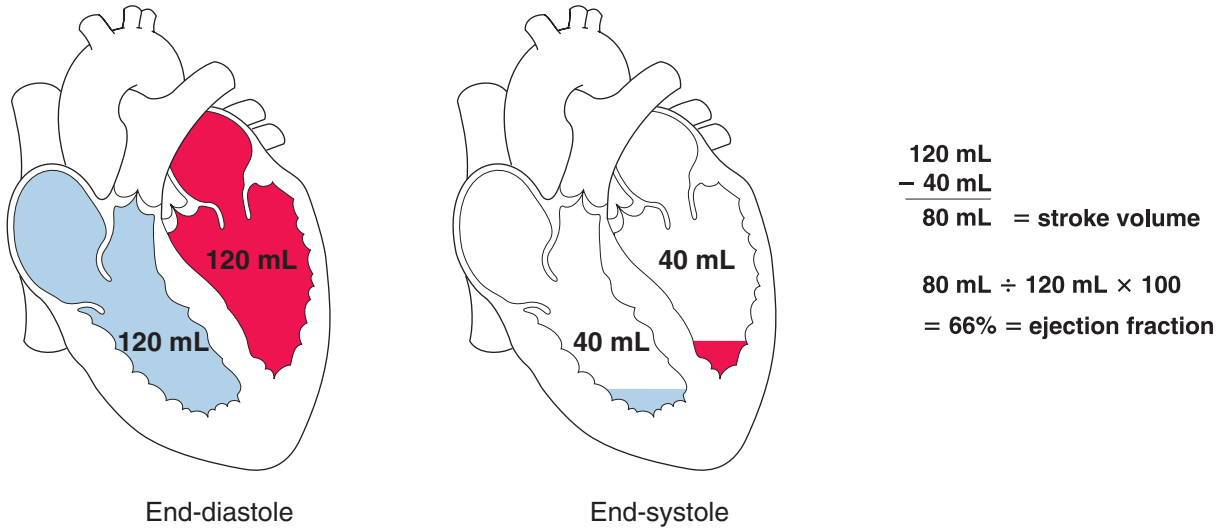


FIGURE 28.9 ■ Stroke volume is end-diastole volume minus end-systole volume; ejection fraction is stroke volume divided by end-diastole volume, expressed as a percentage

Preload

Preload is the amount of cardiac muscle fibre tension or stretch that exists at the end of diastole, just before contraction of the ventricles. Preload is influenced by venous return and the compliance of the ventricles. It is related to the total volume of blood in the ventricles: the greater the volume, the greater the stretch of the cardiac muscle fibres and the greater the force with which the fibres contract to accomplish emptying. This principle is called Starling's law of the heart.

This mechanism has a physiological limit. Just as continuous overstretching of a rubber band causes the band to relax and lose its ability to recoil, overstretching of the cardiac muscle fibres eventually results in ineffective contraction. Disorders such as kidney disease and congestive heart failure result in sodium and water retention and increased preload. Vasoconstriction also increases venous return and preload.

Too little circulating blood volume results in a decreased venous return and therefore a decreased preload. A decreased preload reduces stroke volume and thus cardiac output. Decreased preload may result from haemorrhage or maldistribution of blood volume, as occurs in 'third spacing' (see Chapter 9).

Afterload

Afterload is the force the ventricles must overcome to eject their blood volume. It is the pressure in the arterial system ahead of the ventricles. The right ventricle must generate enough tension to open the pulmonary valve and eject its volume into the low-pressure pulmonary arteries. Right ventricle afterload is measured as peripheral vascular resistance (PVR). The left ventricle, in contrast, ejects its load by overcoming the pressure behind the aortic valve. Afterload of the left ventricle is measured as systemic vascular resistance (SVR). Arterial pressures are much higher than pulmonary pressures; thus, the left ventricle has to work much harder than the right ventricle.

Alterations in vascular tone affect afterload and ventricular work. As the pulmonary or arterial blood pressure increases (e.g. through vasoconstriction), PVR and/or SVR increases and the work of the ventricles increases. As workload increases, consumption of myocardial oxygen also increases. A compromised heart cannot effectively meet this increased oxygen demand and a vicious cycle ensues. By contrast, a very low afterload decreases the forward flow of blood into the systemic circulation and the coronary arteries.

Clinical indicators of cardiac output

For many people who are critically ill, invasive haemodynamic monitoring catheters are used to measure cardiac output in quantifiable numbers. However, advanced technology is not the only way to identify and assess compromised blood flow. Because cardiac output perfuses the body's tissues, clinical indicators of low cardiac output may be manifested by changes in organ function that result from compromised blood flow. For example, a decrease in blood flow to the brain presents as a change in level of consciousness. Other manifestations of decreased cardiac output are discussed in Chapters 9 and 29.

Cardiac index (CI) is the cardiac output adjusted for the person's body size, also called the person's body surface area (BSA). Because it takes into account the person's BSA, the cardiac index provides more meaningful data about the heart's ability to perfuse the tissues and therefore is a more accurate indicator of the effectiveness of the circulation.

BSA is stated in square metres (m^2), and cardiac index is calculated as CO divided by BSA. Cardiac measurements are considered adequate when they fall within the range of 2.5 to 4.2 L/min/m^2. For example, two people are determined to have a cardiac output of 4 L/min. This parameter is within normal limits. However, one person is 157 cm tall and weighs 54.5 kg, with a BSA of 1.54 m^2. This person's cardiac index is 4 ÷ 1.54, or 2.6 L/min/m^2. The second person is 188 cm tall and weighs 81.7 kg, with a BSA of 2.52 m^2. This person's cardiac index is 4 ÷ 2.52, or 1.6 L/min/m^2.

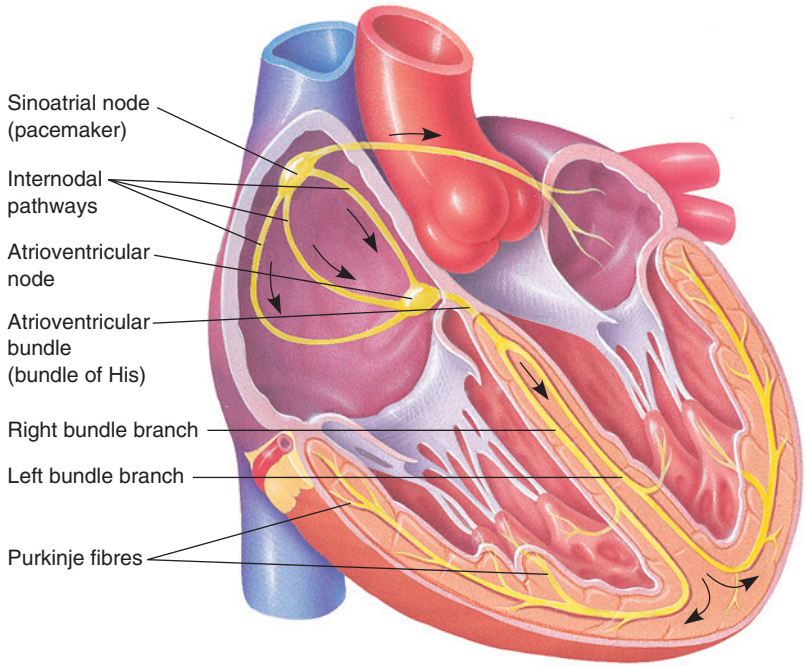


FIGURE 28.10 ■ The intrinsic conduction system of the heart

The cardiac index results show that the same cardiac output of 4 L/min is adequate for the first person but grossly inadequate for the second person.

The conduction system of the heart

The cardiac cycle is perpetuated by a complex electrical circuit commonly known as the intrinsic conduction system of the heart. Cardiac muscle cells possess an inherent characteristic of self-excitation, which enables them to initiate and transmit impulses independent of a stimulus. However, specialised areas of myocardial cells typically exert a controlling influence in this electrical pathway.

One of these specialised areas is the sinoatrial (SA) node, located at the junction of the superior vena cava and right atrium (see Figure 28.10). The SA node acts as the normal 'pacemaker' of the heart, usually generating an impulse 60 to 100 times per minute. This impulse travels across the atria via internodal pathways to the atrioventricular (AV) node, in the floor of the interatrial septum. The very small junctional fibres of the AV node slow the impulse, slightly delaying its transmission to the ventricles. It then passes through the bundle of His at the atrioventricular junction and continues down the interventricular septum through the right and left bundle branches and out to the Purkinje fibres in the ventricular muscle walls (Mohrman & Heller, 2010).

THE PERIPHERAL VASCULAR SYSTEM

The two main components of the peripheral vascular system are the arterial network and the venous network. The arterial network begins with the major arteries that branch from the aorta. The major arteries of the systemic circulation are illustrated in Figure 28.11. These major arteries branch into successively smaller arteries, which in turn subdivide into the smallest of the arterial vessels, called *arterioles*. The smallest arterioles feed into beds of hair-like capillaries in the body's organs and tissues.

In the capillary beds, oxygen and nutrients are exchanged for metabolic wastes, and deoxygenated blood begins its journey back to the heart through venules, the smallest vessels of the venous network. Venules join the smallest of veins, which in turn join larger and larger veins. The blood transported by the veins empties into the superior and inferior venae cavae entering the right side of the heart. The major veins of the systemic circulation are shown in Figure 28.12.

Structure of blood vessels

The structure of blood vessels reflects their different functions within the circulatory system (see Figure 28.13). Except for the tiniest vessels, blood vessel walls have three layers: the tunica intima, the tunica media and the tunica adventitia. The tunica intima, the innermost layer, is made of simple squamous epithelium (the endothelium); this provides a slick surface to facilitate the flow of blood. In arteries the middle layer, or tunica media, is made of smooth muscle and is thicker than the tunica media of veins. This makes arteries more elastic than veins and allows the arteries to alternately expand and recoil as the heart contracts and relaxes with each beat, producing a pressure wave, which can be felt as a **pulse** over an artery. The smaller arterioles are less elastic than arteries but contain more smooth muscle, which promotes their constriction (narrowing) and dilation (widening). In fact, arterioles exert the major control over arterial blood pressure. The tunica adventitia, or outermost layer, is made of

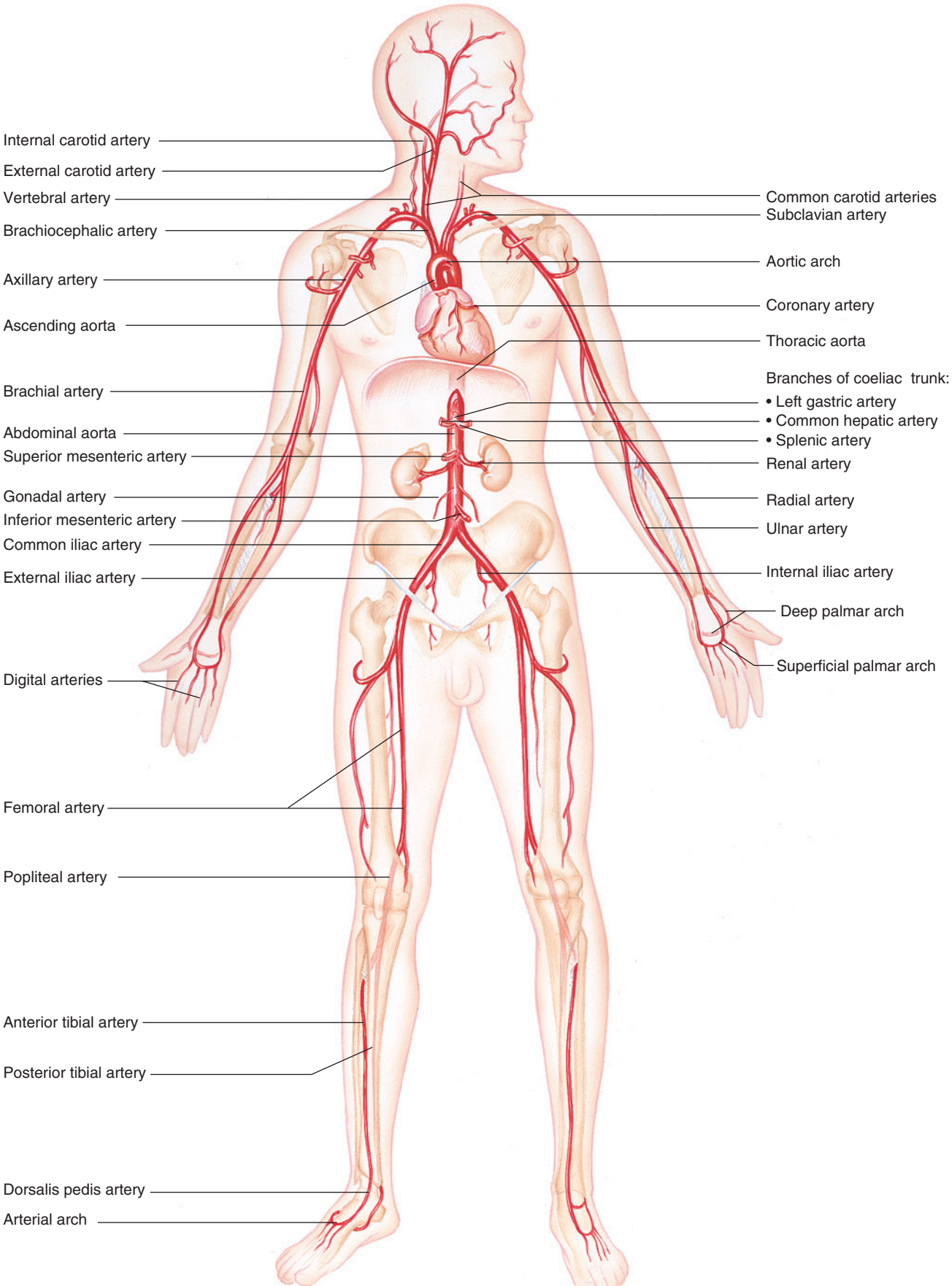


FIGURE 28.11 ■ Major arteries of the systemic circulation

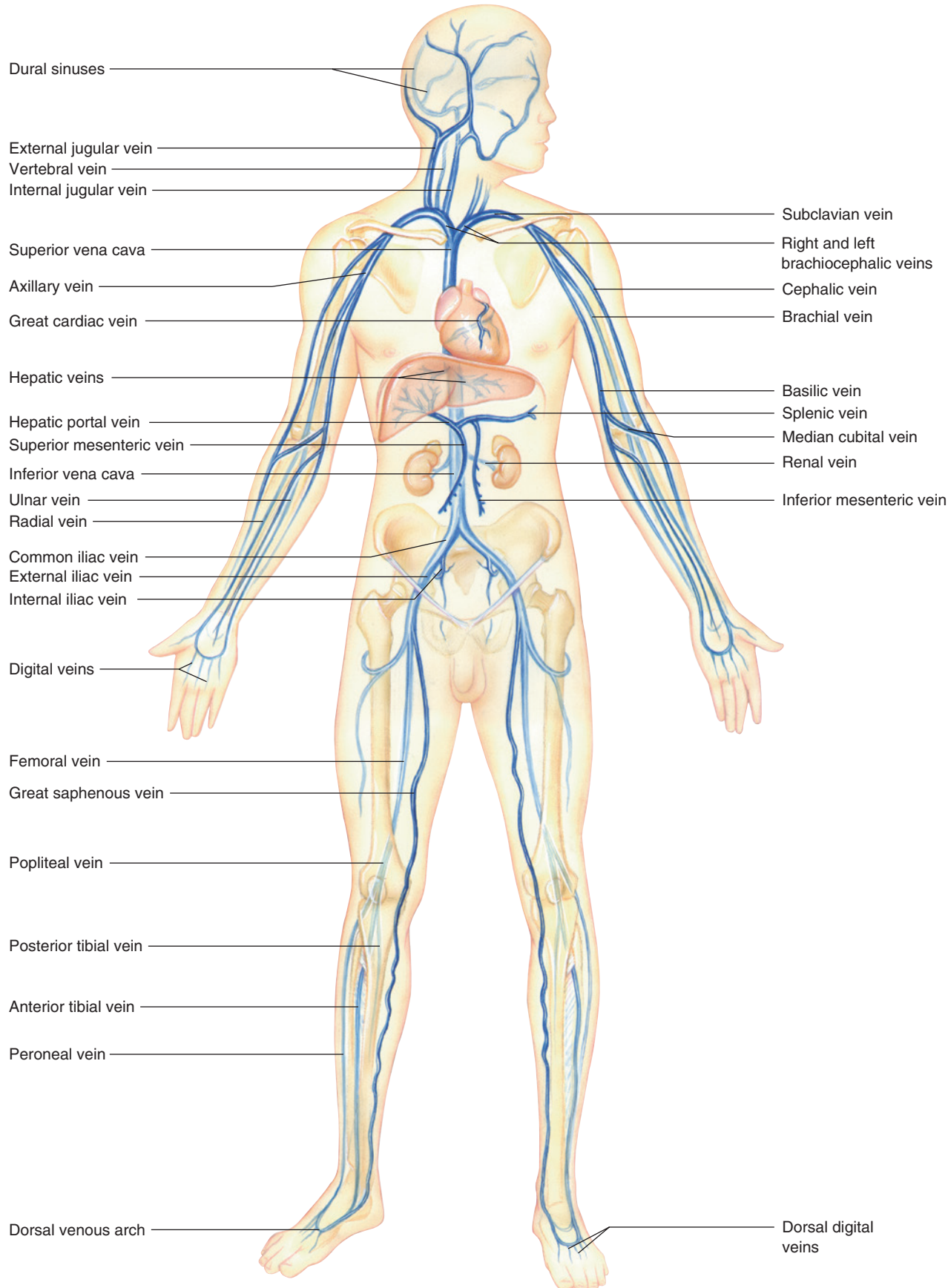


FIGURE 28.12 ■ Major veins of the systemic circulation

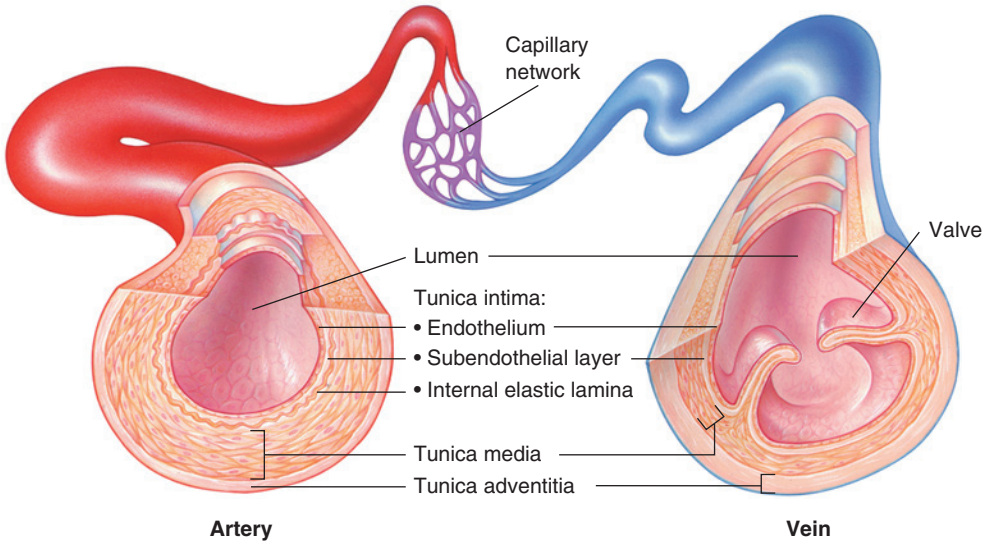


FIGURE 28.13 ■ Structure of arteries, veins and capillaries. Capillaries are composed of only a fine tunica intima. Notice that the tunica media is thicker in arteries than in veins

connective tissue and serves to protect and anchor the vessel. Veins have a thicker tunica adventitia than do arteries.

Blood in the veins travels at a much lower pressure than blood in the arteries. Veins have thinner walls, a larger lumen and greater capacity, and many are supplied with valves that help blood flow against gravity back to the heart. The 'milking' action of skeletal muscle contraction (called the muscular pump) also supports venous return. When skeletal muscles contract against veins, the valves proximal to the contraction open and blood is propelled towards the heart. The abdominal and thoracic pressure changes that occur with breathing (called the respiratory pump) also propel blood towards the heart.

The tiny capillaries that connect the arterioles and venules contain only one thin layer of tunica intima, which is permeable to the gases and molecules exchanged between blood and tissue cells. Capillaries typically are found in interwoven networks. They filter and shunt blood from precapillary arterioles to postcapillary venules.

Physiology of arterial circulation

The factors that affect arterial circulation are blood flow, peripheral vascular resistance and blood pressure. **Blood flow** refers to the volume of blood transported in a vessel, in an organ or throughout the entire circulation over a given period of time. It is commonly expressed as litres or millilitres per minute or cubic centimetres per second.

Peripheral vascular resistance (PVR) refers to the opposing forces or impedance to blood flow as the arterial channels become more and more distant from the heart. Peripheral vascular resistance is determined by three factors:

1. *Blood viscosity:* the greater the viscosity, or thickness, of the blood, the greater its resistance to moving and flowing.
2. *Length of the vessel:* the longer the vessel, the greater the resistance to blood flow.
3. *Diameter of the vessel:* the smaller the diameter of a vessel, the greater the friction against the walls of the vessel and, thus, the greater the impedance to blood flow.

Blood pressure is the force exerted against the walls of the arteries by the blood as it is pumped from the heart. It is most accurately referred to as **mean arterial pressure (MAP)**. The highest pressure exerted against the arterial walls at the peak of ventricular contraction (systole) is called the systolic blood pressure. The lowest pressure exerted during ventricular relaxation (diastole) is the diastolic blood pressure.

Mean arterial blood pressure is regulated mainly by cardiac output (CO) and peripheral vascular resistance (PVR), as represented in this formula: MAP = CO × PVR. For clinical use, the MAP may be estimated by calculating the diastolic blood pressure plus one-third of the pulse pressure (the difference between the systolic and diastolic blood pressure).

Factors influencing arterial blood pressure

Blood flow, peripheral vascular resistance and blood pressure, which influence arterial circulation, are in turn influenced by various factors, as follows:

- The sympathetic and parasympathetic nervous systems are the primary mechanisms that regulate blood pressure. Stimulation of the sympathetic nervous system exerts a major effect on peripheral resistance by causing vasoconstriction of the arterioles, thereby increasing blood pressure. Parasympathetic stimulation causes vasodilation of the arterioles, lowering blood pressure.
- Baroreceptors and chemoreceptors in the aortic arch, carotid sinus and other large vessels are sensitive to pressure and chemical changes and cause reflex sympathetic stimulation, resulting in vasoconstriction, increased heart rate and increased blood pressure.
- The kidneys help maintain blood pressure by excreting or conserving sodium and water. When blood pressure

decreases, the kidneys initiate the renin–angiotensin mechanism. This stimulates vasoconstriction, resulting in the release of the hormone aldosterone from the adrenal cortex, increasing sodium ion reabsorption and water retention. In addition, pituitary release of antidiuretic hormone (ADH) promotes renal reabsorption of water. The net result is an increase in blood volume and a consequent increase in cardiac output and blood pressure.

- Temperatures may also affect peripheral resistance: cold causes vasoconstriction, whereas warmth produces vasodilation. Many chemicals, hormones and drugs influence blood pressure by affecting CO and/or PVR. For example, epinephrine causes vasoconstriction and increased heart rate; prostaglandins dilate blood vessel diameter (by relaxing vascular smooth muscle); endothelin, a chemical released by the inner lining of vessels, is a potent vasoconstrictor; nicotine causes vasoconstriction; and alcohol and histamine cause vasodilation.
- Dietary factors, such as intake of salt, saturated fats and cholesterol, elevate blood pressure by affecting blood volume and vessel diameter.
- Race, gender, age, weight, time of day, position, exercise and emotional state may also affect blood pressure. These factors influence the arterial pressure. Systemic venous pressure, though it is much lower, is also influenced by such factors as blood volume, venous tone and right atrial pressure.

STRUCTURE AND FUNCTION OF BLOOD

Blood is an exchange medium between the external environment and the body's cells. Blood consists of plasma, solutes (e.g. proteins, electrolytes and organic constituents), red blood cells, white blood cells and platelets (which are fragments of cells). The haematopoietic (blood-forming) system includes the bone marrow (myeloid) tissues, where blood cells form, and the lymphoid tissues of the lymph nodes, where white blood cells mature and circulate. All blood cells originate from cells in the bone marrow called **stem cells** or *haemocytoblasts*. The origin of the cellular components of blood is illustrated in Figure 28.14.

Regulatory mechanisms cause stem cells to differentiate into families of parent cells, each of which gives rise to one of the formed elements of the blood (red blood cells, platelets and white blood cells). The functions of blood include transporting oxygen, nutrients, hormones and metabolic wastes; protecting against invasion of pathogens; maintaining blood coagulation; and regulating fluids, electrolytes, acids, bases and body temperature.

Red blood cells

Red blood cells (RBCs, erythrocytes) and the haemoglobin molecules they contain are required to transport oxygen to body tissues. Haemoglobin also binds with some carbon dioxide, carrying it to the lungs for excretion. Abnormal numbers of RBCs, changes in their size and shape, or altered haemoglobin content or structure can adversely affect health. Anaemia, the most common RBC disorder, is an abnormally low RBC count or reduced haemoglobin content. Polycythaemia is an abnormally high RBC count.

The red blood cell is shaped like a biconcave disk (see Figure 28.15). This unique shape increases the surface area of the cell and allows the cell to pass through very small capillaries without disrupting the cell membrane. RBCs are the most common type of blood cell.

Haemoglobin is the oxygen-carrying protein within RBCs. It consists of the haem molecule and globin, a protein molecule. Globin is made of four polypeptide chains—two alpha chains and two beta chains (see Figure 28.16). Each of the four polypeptide chains contains a haem unit containing an iron atom. The iron atom binds reversibly with oxygen, allowing it to transport oxygen as *oxyhaemoglobin* to the cells. Haemoglobin is synthesised within the RBCs. The rate of synthesis depends on the availability of iron (Bullock & Hales, 2012).

Normal adult laboratory values for red blood cells are defined and identified in Table 28.1. The size, colour and shape of stained RBCs also may be analysed. RBCs may be normocytic (normal size), smaller than normal (microcytic) or larger than normal (macrocytic). Their colour may be normal (normochromic) or diminished (hypochromic).

Red blood cell production and regulation

In adults, RBC production (**erythropoiesis**) (see Figure 28.17) begins in red bone marrow of the vertebrae, sternum, ribs and pelvis, and is completed in the blood or spleen. Erythroblasts begin forming haemoglobin while they are in the bone marrow, a process that continues throughout the RBC lifespan. Erythroblasts differentiate into *normoblasts*. As these slightly smaller cells mature, their nucleus and most organelles are ejected, eventually causing normoblasts to collapse inward and assume the characteristic biconcave shape of RBCs. The cells enter the circulation as *reticulocytes*, which fully mature in about 48 hours. The complete sequence from stem cell to RBC takes 3 to 5 days.

The stimulus for RBC production is tissue hypoxia. The hormone erythropoietin is released by the kidneys in response to hypoxia. It stimulates the bone marrow to produce RBCs. However, the process of RBC production takes about 5 days to maximise. During periods of increased RBC production, the percentage of reticulocytes (immature RBCs) in the blood exceeds that of mature cells.

Red blood cell destruction

RBCs have a life span of about 120 days. Old or damaged RBCs are lysed (destroyed) by phagocytes in the spleen, liver, bone marrow and lymph nodes. The process of RBC destruction is called **haemolysis**. Phagocytes save and reuse amino acids and iron from haem units in the lysed RBCs. Most of the haem unit is converted to bilirubin, an orange-yellow pigment that is removed from the blood by the liver and excreted in the bile. During disease processes causing increased haemolysis or impaired liver function, bilirubin accumulates in the serum, causing a yellowish appearance of the skin and sclera (jaundice).

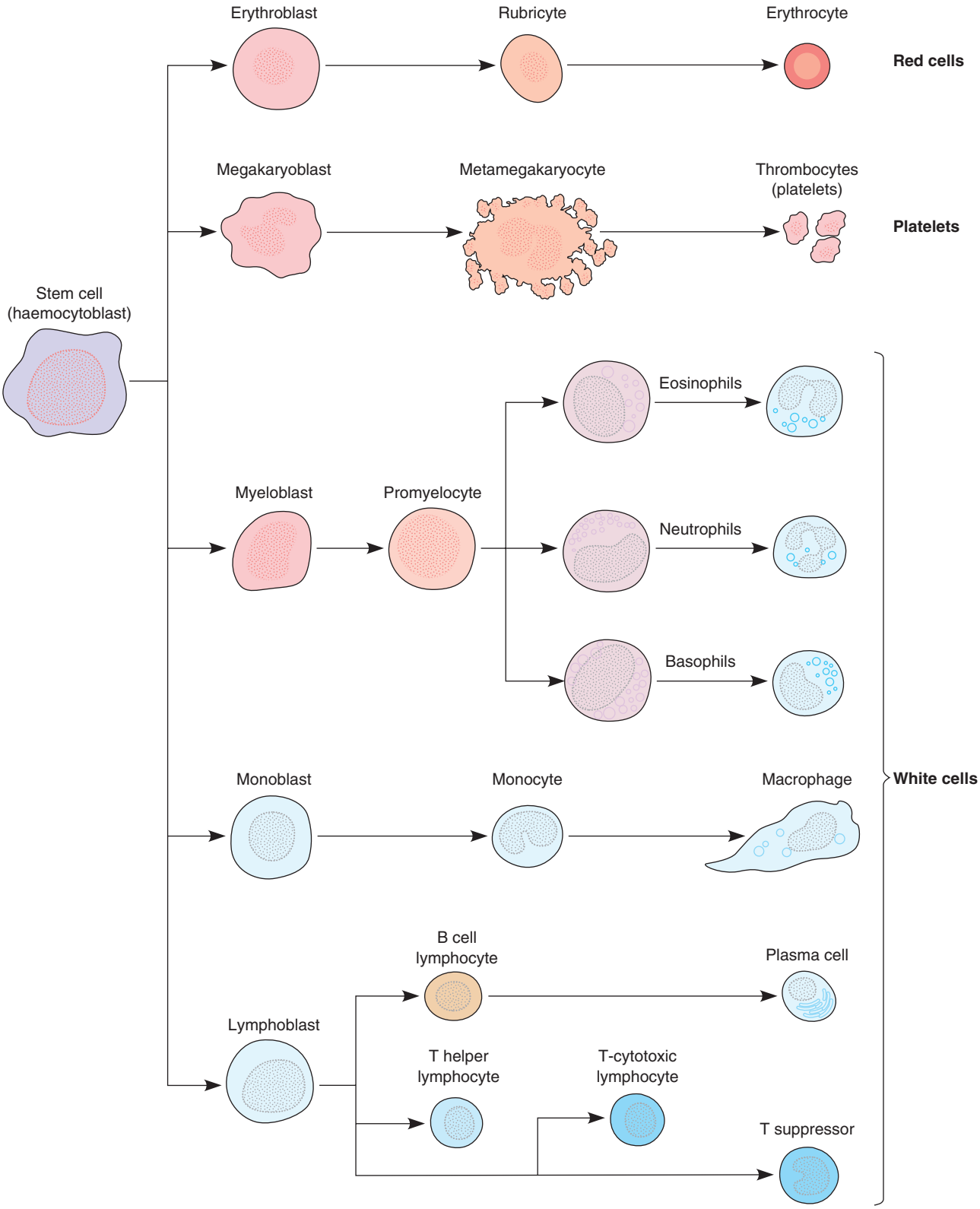


FIGURE 28.14 ■ Blood cell formation from stem cells. Regulatory factors control the differentiation of stem cells into blasts. Each of the five kinds of blasts is committed to producing one type of mature blood cell. Erythroblasts, for example, can differentiate only into RBCs; megakaryoblasts can differentiate only into platelets

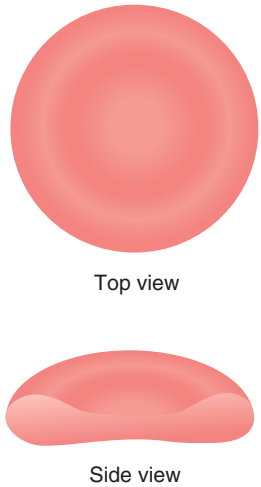


FIGURE 28.15 ■ Top and side view of a red blood cell (erythrocyte). Note the distinctive biconcave shape

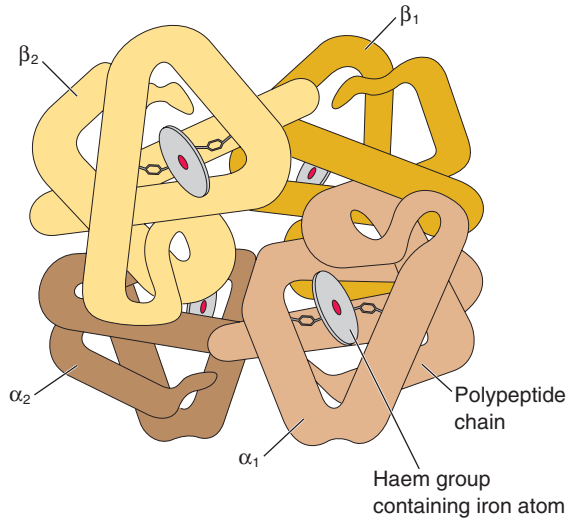


FIGURE 28.16 ■ The haemoglobin molecule includes globin (a protein) and haem, which contains iron. Globin is made of four subunits, two alpha and two beta polypeptide chains. A haem disk containing an iron atom (red dot) nests within the folds of each protein subunit. The iron atoms combine reversibly with oxygen, transporting it to the cells

TABLE 28.1 Full blood count (FBC)

COMPONENT	PURPOSE	NORMAL VALUES
Haemoglobin (Hb)	Measures the capacity of the haemoglobin to carry gases	Women: 12–17.5 g/dL Men: 13.6–17.5 g/dL
Haematocrit (HCT)	The haematocrit represents the percentage of whole blood volume composed of erythrocytes	Women: 35–45% Men: 39–49%
Total RBC count	Counts number of circulating RBCs	Women: 4–5 × 10^6 /μL Men: 4.5–6 × 10^6 /μL
Red cell indices:		
MCV	Determines relative size of MCV (mean corpuscular volume)	76–96 fL
MCH	Mean corpuscular haemoglobin	27–33 pg
MCHC	Evaluates RBC saturation with Hb (MCHC = mean corpuscular haemoglobin concentration)	32–36%
WBC count (leucocytes)	Measures total number of leucocytes (total count) and whether each kind of WBC is present in proper proportion (differential)	Total WBC count: 4300–10 800/ μL (4.3–10.8 × 10^9 /L) WBC differential: Neutrophils: 50–70% Eosinophils: 2–4% Basophils: 0–2% Lymphocytes: 20–40% Monocytes: 4–8%
Platelets	Measures number of platelets available to maintain clotting functions	150 000–400 000 /μL (150–400 × 10^9/L)

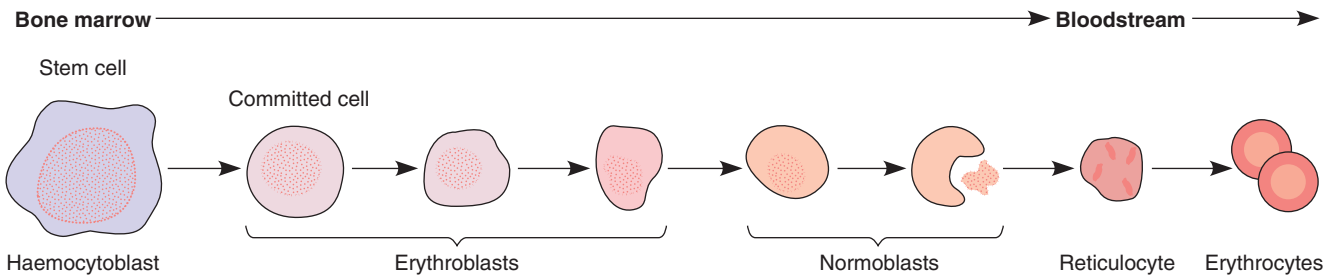


FIGURE 28.17 ■ Erythropoiesis. RBCs begin as erythroblasts within the bone marrow, maturing into normoblasts, which eventually eject their nucleus and organelles to become reticulocytes. Reticulocytes mature within the blood or spleen to become erythrocytes

White blood cells

White blood cells (WBCs, leucocytes) are a part of the body's defence against microorganisms. On average, there are 5000 to 10 000 WBCs per microlitre of blood, accounting for about 1% of total blood volume. **Leucocytosis** is a higher than normal WBC count; **leucopenia** is a WBC count that is lower than normal.

WBCs originate from haemopoietic stem cells in the bone marrow. These stem cells differentiate into the various types of white blood cells (see Figure 28.14).

The two basic types of WBCs are granular leucocytes (or granulocytes) and non-granular leucocytes. Granulocytes have horseshoe-shaped nuclei and contain large granules in the cytoplasm. Stimulated by granulocyte-macrophage colony-stimulating factor (GM-CSF) and granulocyte colony-stimulating factor (G-CSF), granulocytes mature fully in the bone marrow before being released into the bloodstream. The three types of granulocytes are as follows:

- Neutrophils (also called *polymorphonuclear (PMN)* or segmented (*segs*) leucocytes) comprise 60–70% of the total circulating WBCs. Their nuclei are divided into three to five lobes. Neutrophils are active phagocytes, the first cells to arrive at a site of injury. Their numbers increase during inflammation. Immature forms of neutrophils (bands) are released during inflammation or infections and are referred to as having a shift to the left (so named because immature cell frequencies appear on the left side of the graph) on a differential blood count. Neutrophils have a lifespan of only about 10 hours and are constantly being replaced.
- Eosinophils comprise 1–3% of circulating WBCs, but are found in large numbers in the mucosa of the intestines and lungs. Their numbers increase during allergic reactions and parasitic infestations.
- Basophils, which comprise less than 1% of the WBC count, contain histamine, heparin and other inflammatory mediators. Basophils increase in number during allergic and inflammatory reactions.

Non-granular WBCs (agranulocytes) include the monocytes and lymphocytes. They enter the bloodstream before final maturation.

- Monocytes are the largest of the WBCs. They comprise approximately 3–8% of the total WBC count. Monocytes contain powerful bactericidal substances and proteolytic enzymes. They are phagocytic cells that mature into macrophages. Macrophages dispose of foreign and waste material, especially in inflammation. They are an active part of the immune response.
- Lymphocytes comprise 20–30% of the WBC count. Lymphocytes mature in lymphoid tissue into B cells and T cells. B cells are involved in the humoral immune response and antibody formation, whereas T cells take part in the cell-mediated immunity process (see Chapter 12). Plasma cells (which arise from B cells) are lymphoid cells found in bone marrow and connective tissue; they also are involved in immune reactions.

Platelets

Platelets (thrombocytes) are cell fragments that have no nucleus and cannot replicate. They are metabolically active, however, producing ATP and releasing mediators required for clotting. Platelets are formed in the bone marrow as pinched-off portions of large megakaryocytes (see Figure 28.14). Platelet production is controlled by *thrombopoietin*, a protein produced by the liver, kidney, smooth muscle and bone marrow. The number of circulating platelets controls thrombopoietin release. Once released from the bone marrow, platelets remain in the spleen for about 8 hours before entering the circulation. Platelets live up to 10 days in circulation. There are about 250 000 to 400 000 platelets in each microlitre of blood. An excess of platelets is *thrombocytosis*. A deficit of platelets is *thrombocytopenia*.

Haemostasis

Platelet and coagulation disorders affect haemostasis, the control of bleeding. Haemostasis is a series of complex interactions between platelets and clotting mechanisms that maintains a relatively steady state of blood volume, blood pressure and blood flow through injured vessels. The five stages of haemostasis are (1) vessel spasm, (2) formation of the platelet plug, (3) development of an insoluble fibrin clot, (4) clot retraction, and (5) clot dissolution.

Vessel spasm

When a blood vessel is damaged, thromboxane A_2 (TXA_2) is released from platelets and cells, causing *vessel spasm*. This spasm constricts the damaged vessel for about 1 minute, reducing blood flow.

Formation of the platelet plug

Platelets attracted to the damaged vessel wall change from smooth disks to spiny spheres. Receptors on the activated platelets bind with *von Willebrand's factor*, a protein molecule and exposed collagen fibres at the site of injury to form the platelet plug (see Figure 28.18). The platelets release adenosine diphosphate (ADP) and TXA_2 to activate nearby platelets, adhering them to the developing plug. Activation of the clotting pathway on the platelet surface converts fibrinogen to fibrin. Fibrin, in turn, forms a meshwork that binds the platelets and other blood cells to form a stable plug (see Figure 28.19).

Development of the fibrin clot

The process of coagulation creates a meshwork of fibrin strands that cements the blood components to form an insoluble clot. Coagulation requires many interactive reactions and two clotting pathways (see Figure 28.20). The slower intrinsic pathway is activated when blood contacts collagen in the injured vessel wall; the faster extrinsic pathway is activated when blood is exposed to tissues. The final outcome of both pathways is fibrin clot formation. Each procoagulation substance is activated in sequence; the activation of one coagulation factor activates another in turn. Table 28.2 lists known factors, their origin and their function or pathway. A deficiency of one or

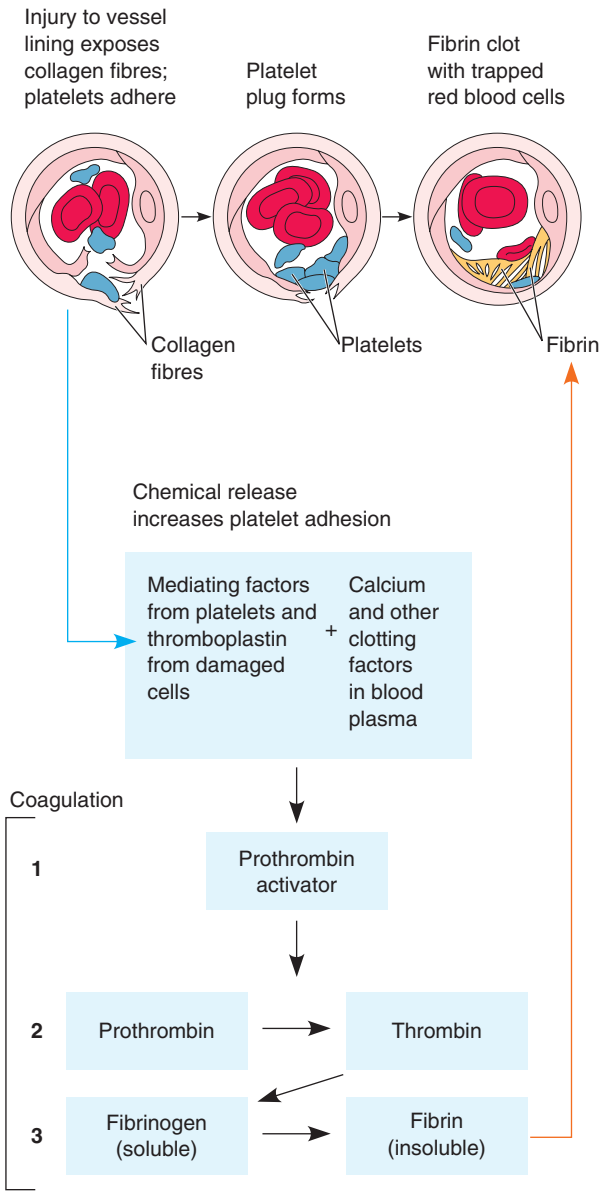


FIGURE 28.18 ■ Platelet plug formation and blood clotting. The flow diagram summarises the events leading to fibrin clot formation

more factors or inappropriate inactivation of any factor alters normal coagulation.

Clot retraction

After the clot is stabilised (within about 30 minutes), trapped platelets contract, much like muscle cells. Platelet contraction squeezes the fibrin strands, pulling the broken portions of the ruptured blood vessel closer together. Growth factors released by the platelets stimulate cell division and tissue repair of the damaged vessel.

Clot dissolution

Fibrinolysis, the process of clot dissolution, begins shortly after the clot has formed, restoring blood flow and promoting tissue repair. Like coagulation, fibrinolysis requires a sequence of interactions between activator and inhibitor substances. Plasminogen, an enzyme that promotes fibrinolysis, is converted into plasmin, its active form, by chemical mediators released from vessel walls and the liver. Plasmin dissolves the clot's fibrin strands and certain coagulation factors. Stimuli such as exercise, fever and vasoactive drugs promote plasminogen activator release. The liver and endothelium also produce fibrinolytic inhibitors.

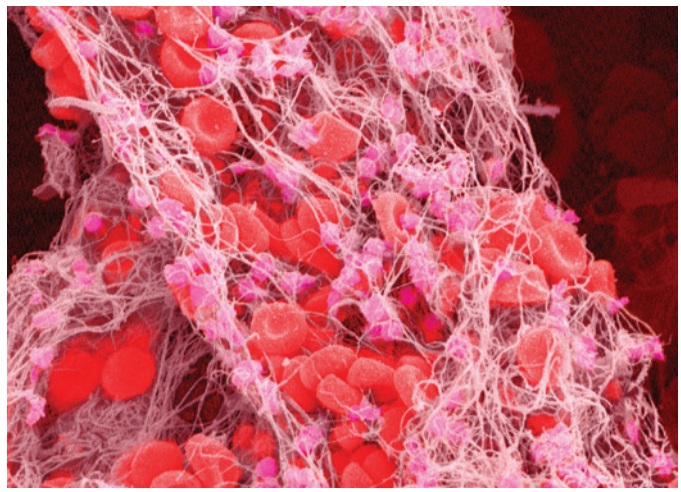


FIGURE 28.19 ■ Scanning electron micrograph of an RBC trapped in a fibrin mesh

Source: Micro Discovery/Corbis.

STRUCTURE AND FUNCTION OF THE LYMPHATIC SYSTEM

The structures of the lymphatic system include the lymphatic vessels and several lymphoid organs (see Figure 28.21). The organs of the lymphatic system are the lymph nodes, the spleen, the thymus, the tonsils and the Peyer's patches of the small intestine. Lymph nodes are small aggregates of specialised cells that assist the immune system by removing foreign material, infectious organisms and tumour cells from lymph. Lymph nodes are distributed along the lymphatic vessels, forming clusters in certain body regions, such as the neck, axilla and groin (see Figure 28.21). The spleen, the largest lymphoid organ, is in the upper left quadrant of the abdomen under the thorax. The main function of the spleen is to filter the blood by breaking down old red blood cells and storing or releasing to the liver their by-products (such as iron). The spleen also synthesises lymphocytes, stores platelets for blood clotting and serves as a reservoir of blood. The thymus gland is in the lower throat and is most active in childhood, producing hormones (such as thymosin) that facilitate the immune action of lymphocytes. The tonsils of the pharynx and Peyer's patches of the small intestine are lymphoid organs that protect the upper respiratory and digestive tracts from foreign pathogens.

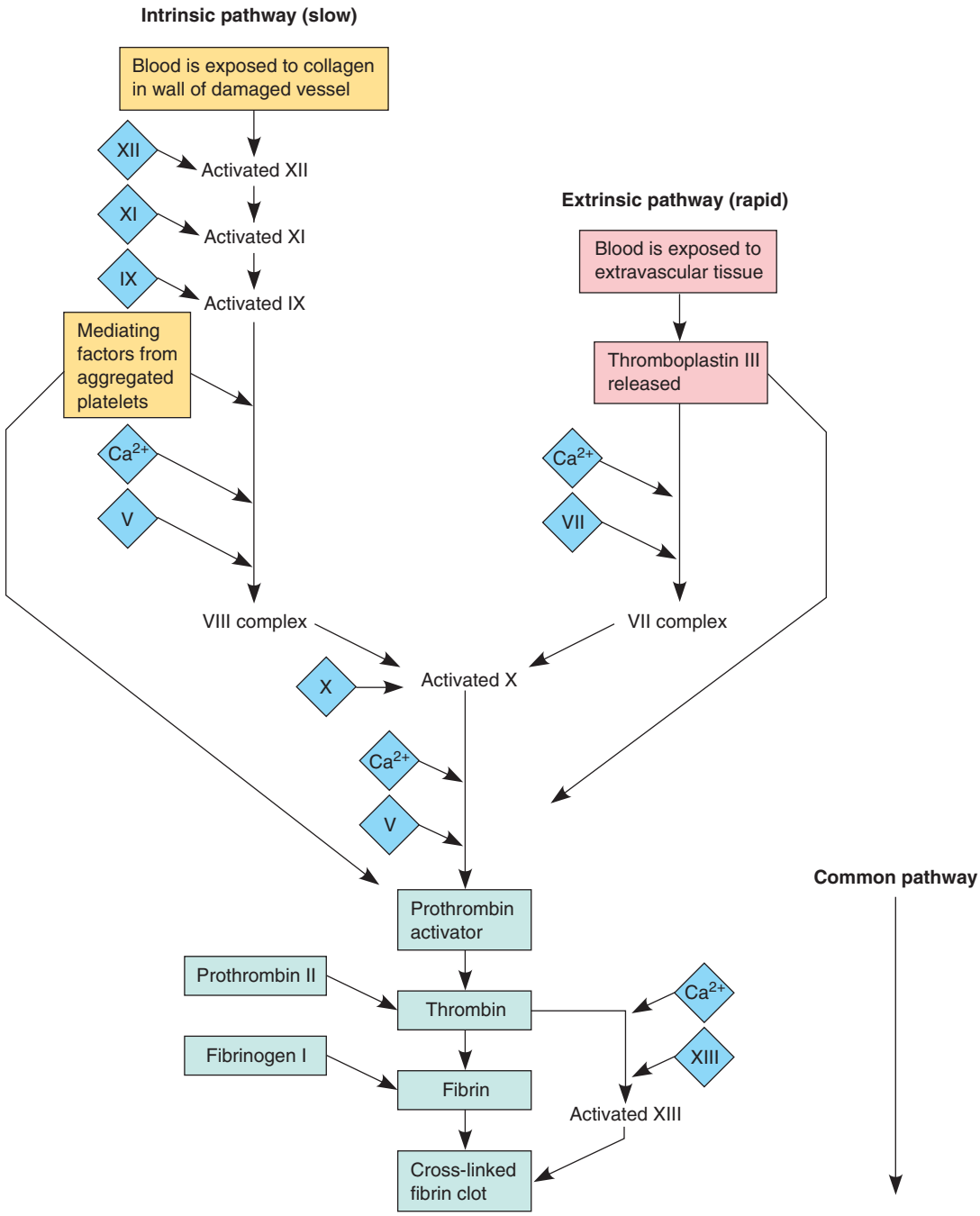


FIGURE 28.20 ■ Clot formation. Both the slower intrinsic pathway and the more rapid extrinsic pathway activate factor X. Factor X then combines with other factors to form prothrombin activator. Prothrombin activator transforms prothrombin into thrombin, which then transforms fibrinogen into long fibrin strands. Thrombin also activates factor XIII, which draws the fibrin strands together into a dense meshwork. The complete process of clot formation occurs within 3 to 6 minutes after blood vessel damage

The lymphatic vessels, or lymphatics, form a network around the arterial and venous channels and interweave at the capillary beds. They collect and drain excess tissue fluid, called *lymph*, that 'leaks' from the cardiovascular system and accumulates at the venous end of the capillary bed. The lymphatics return this fluid to the heart through a one-way system of lymphatic venules and veins that eventually drain into the right lymphatic duct and left thoracic duct, both of which empty into their respective subclavian veins. Lymphatics are a low-pressure system without a pump; their fluid transport depends on the rhythmic contraction of their smooth muscle and the muscular and respiratory pumps that assist venous circulation.

TABLE 28.2 Blood coagulation factors

FACTOR	NAME	FUNCTION OR PATHWAY
I	Fibrinogen	Converted to fibrin strands
II	Prothrombin	Converted to thrombin
III	Thromboplastin	Catalyses conversion of thrombin
IV	Calcium ions	Needed for all steps of coagulation
V	Proaccelerin	Extrinsic/intrinsic pathways
VI	Derived from proaccelerin	A hypothetical agent said to be derived from proaccelerin
VII	Serum prothrombin conversion accelerator	Extrinsic pathway
VIII	Antihaemophilic factor	Intrinsic pathway
IX	Plasma prothrombin component	Intrinsic pathway
X	Stuart factor	Extrinsic/intrinsic pathways
XI	Plasma prothrombin antecedent	Intrinsic pathway
XII	Hageman factor	Intrinsic pathway
XIII	Fibrin stabilising factor	Cross-links fibrin strands to form insoluble clot

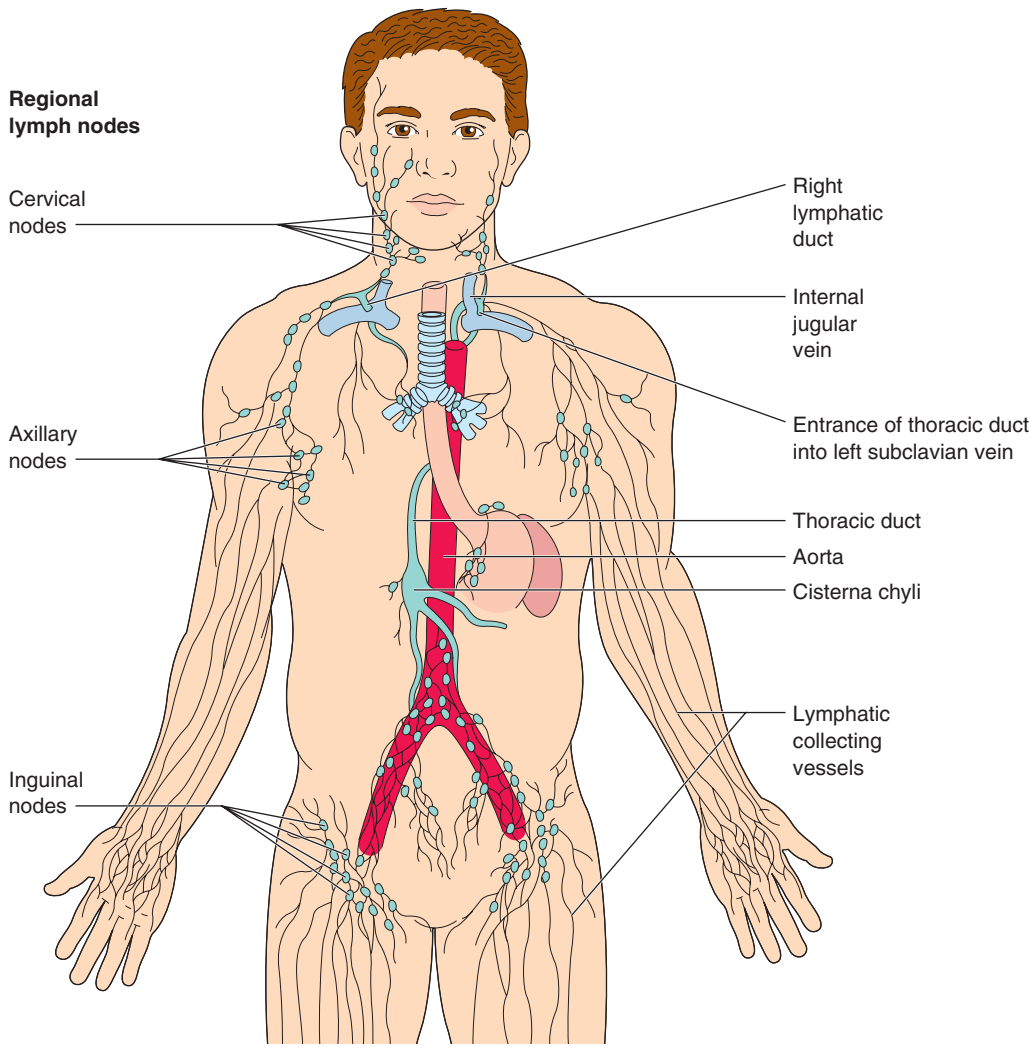


FIGURE 28.21 ■ The lymphatic system

HEALTH ASSESSMENT AND DOCUMENTATION

Cardiovascular, haematological and lymphatic function is assessed by findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests. Consideration of all the data which contributes to understanding a person's absolute cardiovascular disease risk (CVD) should be considered. The National Vascular Disease Prevention Alliance has produced comprehensive guidelines to assist clinicians with assessment of CVD risk. Sample documentation of an assessment of cardiovascular function is included in the boxes below.

Health assessment interview

A health assessment interview to determine problems with the structure and functions of the cardiovascular and/or lymphatic systems may be conducted during a health screening, may focus on a chief complaint (such as chest pain, fatigue and bleeding) or may be part of a total health assessment.

If the person has a problem with cardiovascular or lymphatic function, analyse its onset, characteristics, course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, ask the person:

- What is the location of the chest pain you experienced? Did it move up to your jaw or into your left arm?
- Describe the type of activity that brings on your chest pain.
- Have you noticed any changes in your energy level?
- Have you felt light-headed during the times your heart is racing?
- Have you noticed any glands that are sore and swollen? What do you think causes this?

The interview begins by exploring the person's chief complaint (e.g. chest pain, palpitations or fatigue). Describe the person's chest or leg pain in terms of location, quality or character, timing, setting or precipitating factors, severity, aggravating and relieving factors, and associated symptoms (see Table 28.3).

Explore the person's history for heart disorders such as angina, heart attack, congestive heart failure (CHF), hypertension (HTN) and valvular disease. Ask the person about previous heart surgery or illnesses, such as rheumatic fever, scarlet fever or recurrent streptococcal throat infections. Also ask about the presence and treatment of other chronic illnesses such as diabetes mellitus, bleeding disorders or endocrine disorders. Review the person's family history for coronary artery disease (CAD), HTN, stroke, hyperlipidaemia, diabetes, congenital heart disease or sudden death.

Ask the person about past or present occurrence of various cardiac symptoms, such as chest pain, shortness of breath, difficulty breathing, cough, palpitations, fatigue, light headedness or dizziness, fainting, heart **murmur**, blood clots or swelling. Because cardiac function affects all other body systems, a full history may need to explore other related systems, such as respiratory function and/or peripheral vascular function.

Review the person's personal habits and nutritional history, including body weight; eating patterns; dietary intake of fats, salt, fluids; dietary restrictions; hypersensitivities or intolerances to food or medication; and the use of caffeine and alcohol. If the person uses tobacco/nicotine products, ask about type

SAMPLE DOCUMENTATION
Assessment of altered cardiac function

28/2/2016 Nursing entry 1000 hrs	56-year-old male admitted to a coronary care unit from ED to rule out myocardial infarction. He states he has pain in the middle of his chest that is 'like a heavy pressure'; 6 on a 10-point scale. Skin cool, slightly moist. BP 190/94 right arm and 186/92 left arm (both reclining). Apical pulse 92, regular and strong. No pulse deficit. Respirations 28. Apical impulse non-palpable, no visible heaves or thrusts. S_1 and S_2 auscultated without murmurs or clicks. S_4 noted. ——————————— RN Peres (S PERES RN)

SAMPLE DOCUMENTATION
Assessment of the peripheral vascular system

28/2/2016 Nursing entry 1100hrs	Mr Alex Fraser is a 57 year old with medical history of type 1 diabetes for 15 years. Mr Fraser states he has smoked cigars for 20 years (1/day), but 'knows he shouldn't'. And that he 'can't walk more than 20 steps without pain in his legs and sometimes has pain in his feet at night'. Mr Fraser also admits to not adhering to a regular regimen of BGLs TDS and takes his insulin when he remembers to. As Mr Fraser is a rural fencing contractor, his symptoms are preventing him from carrying out his work. Pulses in lower extremities (dorsalis pedis, posterior tibial and popliteal): regular but weak. No bruits auscultated over femoral arteries. When legs elevated and then lowered, the skin was pale on elevation and dusky red when dependent in the sitting position with lower legs dangling. Skin on both lower extremities from knees to toes is cool, shiny and hairless. Capillary refill of toenails (which are thickened) on great toe is 6 seconds bilaterally. No ankle or tibial oedema. assessed.——————————— ——————————— ——————————— RN Peres (S PERES RN)

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 9:
Recognising and Responding to Clinical Deterioration in Acute Health Care

'The intention of this standard is to ensure a patient's deterioration is recognised promptly, and appropriate action is taken.' (ACSQHC, 2011, p. 60)

Implementing this standard is achieved by the establishment of systems to assist with recognition, initiating appropriate responses and ensuring meaningful and appropriate communication between all individuals involved in a person's care (including patients, families and carers).

Comprehensive and ongoing assessment of a person's cardiovascular system function should be undertaken and documented as frequently as clinically necessary to facilitate rapid, responsive action in the event of a person's deterioration.

Source: © Australian Commission on Safety and Quality in Health Care.

TABLE 28.3 Assessing chest pain (OLDCART)

CHARACTERISTIC	EXAMPLES
Onset	Onset: Sudden or gradual?
	Awake, at rest, sleep interrupted?
	With activity? With eating, exertion, exercise, elimination, emotional upset?
Location	Where is the pain?
	(Substernal, precordial, jaw, back)
	Is the pain localised or diffuse?
Duration	How many minutes does the pain last?
Characteristics	Pressure; tightness; stabbing; crushing, burning or aching quality; heaviness; dullness; 'heartburn' or indigestion
	Can range from 0 (no pain) to 10 (worst pain ever felt)
	Is the pain continuous or periodic?
	Is it worse with a deep breath?
	Is it worse with temperature change?
Associated symptoms	Fatigue, shortness of breath, palpitations, nausea and vomiting, sweating, anxiety, light headedness or dizziness
Relieving factors	What has been done to try to relieve the pain? Medication (glyceryl trinitrate (GTN), antacid), repositioning, rest; there may be no relieving factors
Treatment	What can be done further to relieve the pain? Rest; position; oxygen; medication (GTN, morphine)

(cigarettes, patches, pipe, cigars, snuff), duration, amount and efforts to quit. If the person uses street drugs, ask about type, method of intake (e.g. inhaled or injected), duration of use and efforts to quit. Include questions about the person's activity level and tolerance, recreational activities and relaxation habits. Assess the person's sleep patterns for interruptions in sleep due to dyspnoea, cough, discomfort, urination or stress. Ask how many pillows the person uses when sleeping.

Also consider psychosocial factors that may affect the person's stress level:

- What is the person's marital status, family composition and role within the family?
- Have there been any changes?
- What is the person's occupation, level of education and socioeconomic level?
- Are resources for support available?
- What is the person's emotional disposition and personality type?
- How does the person perceive their state of health or illness, and how able is the person to comply with treatment?

See the 'Functional health pattern interview' box below for a list of interview questions categorised by functional health patterns.

Diagnostic tests of the cardiovascular and lymphatic systems

The results of diagnostic tests of cardiovascular and lymphatic function are used to support the diagnosis of a specific disease, to provide information to identify or modify the appropriate medications or therapy used to treat the disease and to help nurses monitor the person's responses to treatment and nursing care interventions. Diagnostic tests to assess the structures and functions of the heart are described below and summarised in the following bulleted list. More information is included in the discussion of specific disorders in Chapters 29 and 30.

- The primary test used to identify the risk of coronary artery disease or to monitor treatment for alterations in lipid levels is a measurement of lipid components of cholesterol, triglycerides and lipoproteins in the blood.
- Non-invasive tests of cardiac structure and function include a chest x-ray and stress/exercise tests. The treadmill test is the most basic exercise test, with diagnostic ability to measure cardiac perfusion enhanced by administering IV radioisotopes during the test. A treadmill exercise test is often combined

FUNCTIONAL HEALTH PATTERN INTERVIEW Cardiovascular, haematological and lymphatic systems

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Have you ever had any problems with your heart, such as angina (pain), heart attack or disease of the valves? If so, describe. What was used to treat these problems? ■ Describe any problems you have had with bleeding, bruising, swollen glands and circulation (e.g. heart disease, hardening of the arteries, high blood pressure, stroke, clots, high cholesterol). ■ Have you been diagnosed with high blood pressure? If so, how is it treated? ■ Have you had your cholesterol checked recently? What is it? If you have high cholesterol, how is it treated? ■ Have you ever been diagnosed with a health problem involving the blood, heart, blood vessels or lymph glands? If so, what were they and how were they treated? ■ Is there a family history of bleeding, cancer or anaemia? Explain. ■ What medications, vitamins, dietary supplements, complementary theories or over-the-counter drugs do you take now? ■ Do you, or have you ever, smoked? If so, what, for how long and how many a day?
Nutritional–Metabolic	■ Describe your usual intake of food and fluids in a 24-hour period. ■ Do you drink coffee, tea, cola or other caffeinated drinks? If so, how much? ■ Do you drink alcohol? If so, what type, how much and how often? ■ Describe how much salt you use on your food. ■ Describe what type of fatty foods you eat. How often? ■ Have you had a recent weight gain or loss? Explain. ■ Have you noticed any change in the colour, temperature or appearance of the skin on your arms, hands, legs or feet? If so, what were they? ■ Have you noticed any glands that are sore and swollen? What do you think causes this? ■ Have you noticed an increase in the time it takes your blood to clot if you cut yourself or how easily you bruise? ■ Have you noticed loss of hair, bulging veins, sores that will not heal on your legs or thicker toenails? Have you ever worn support stockings? ■ Do your feet ever swell or your shoes feel tight? If so, when does this happen and what do you do to decrease the swelling?
Elimination	■ Has a health problem interfered with your usual bowel and bladder movements? Explain. ■ Have your bowel movements been a dark black colour?
Activity–Exercise	■ Has there been any change in your ability, energy or strength to perform your usual activities (such as bathing, housework, gardening, shopping)? If so, explain. ■ Describe your activities in a typical day. ■ Do you exercise regularly? Describe what you do when you exercise. ■ Have your activities or exercise abilities changed? If so, explain. ■ Do you notice shortness of breath with certain activities? If so, what are they? How long does this last? What do you do to breathe better? ■ Do you have leg pain when you walk? If so, where is it located? How far do you walk before you have pain? Describe the pain. What do you do to relieve it? ■ Do you ever have to stop and rest while doing daily activities? Explain. ■ Do you feel tired even after sleep and rest? Describe the feeling.

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Sleep–Rest	■ How much rest and sleep do you get each day? ■ Does your health problem interfere with your ability to sleep and rest? Explain. ■ Do leg cramps ever wake you at night? If so, describe the pain and what you do to relieve it. ■ Have you experienced any numbness or tingling, dizziness or light headedness, or palpitations? Describe if so. ■ Do you ever feel short of breath while you are resting or sleeping? Does this wake you up if so? Explain.
Cognitive–Perceptual	■ Describe any chest pain you have experienced. When did it occur? Where was it located? On a scale of 0 to 10, with 10 being the worst pain you have ever had, rate the pain and describe it (for example, burning, crushing, stabbing, squeezing, heavy, tight). ■ Did you have any other symptoms with the pain, such as nausea or vomiting, sweating, racing heart, pale skin, palpitations? ■ What made the pain worse? What did you do to try to relieve the pain? Did that work? ■ Do you have any of these sensations in your legs or feet: pain, cramps, burning, numbness, tingling? ■ If you have these sensations, when do they occur, how long do they last and what do you do to relieve them?
Self-perception–Self-concept	■ How does having this health problem make you feel about yourself?
Role–Relationships	■ How has having this health problem affected your relationships with others? ■ Has having this health problem interfered with your ability to work? Explain. ■ Does your work environment bring you into contact with any chemicals? Describe them.
Sexuality–Reproductive	■ Has this condition interfered with your usual sexual activity? ■ Have you ever had chest pain during sexual activity? What do you do for it? ■ *For women:* Have you noticed any changes in your menstrual cycle? (*For older women:* Have you gone through menopause?) If so, describe them.
Coping–Stress–Tolerance	■ Has having this condition created stress for you? ■ Have you experienced any kind of stress that makes the condition worse? Explain. ■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments (such as blood transfusions) that you would not use to treat this problem?

with other tests to evaluate cardiac function under stress. The exercise thallium or technetium test is probably the most useful non-invasive test to monitor and diagnose CAD.

- Abnormal areas of the heart may be identified and evaluated by an MRI to locate areas of myocardial infarction, a CT scan to quantify calcium deposits in coronary arteries, or a positron emission tomography (PET) test to evaluate myocardial perfusion and myocardial metabolic function.
- Echocardiograms (ECGs) are conducted in conjunction with Dopplers and colour flow imaging to produce audio and graphical data about the motion, wall thickness and chamber size of the heart, and of the blood flow and velocity.
- A transoesophageal echocardiogram (TOE) allows visualisation of structures adjacent to the oesophagus to visualise cardiac and extracardiac structures, including mitral valve and aortic valve pathology, left atrium intracardiac thrombosis, acute dissection of the aorta, endocarditis, and ventricular function during and after surgery.
- A cardiac catheterisation with coronary angiography may be performed to identify CAD or cardiac valvular disease,

to determine pulmonary artery or heart chamber pressures, to obtain a myocardial biopsy, to evaluate artificial valves, or to perform angioplasty or stent of an area of CAD.

- Pericardiocentesis is a procedure done to remove fluid from the pericardial sac for diagnostic or therapeutic purposes. It may also be an emergency procedure to treat cardiac tamponade.
- A physical assessment of the lymphatic system includes specific problems such as lymph node enlargement or swollen glands, as well as other more general complaints about infection or impaired immunity.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, for assessing for medication use that may affect the outcome of the tests, for supporting the person during the examination as necessary, for documenting the procedures as appropriate and for monitoring the results of the tests.

DIAGNOSTIC TESTS Cardiac disorders

NAME OF TEST Lipids

PURPOSE AND DESCRIPTION Blood lipids are cholesterol, triglycerides, and phospholipids. They circulate bound to proteins, and so are known as lipoproteins. Lipids are measured to evaluate risk of CAD and to monitor effectiveness of anti-cholesterol medications.

Normal values:
Cholesterol: 140–200 mg/dL
Triglycerides: 40–190 mg/dL
HDL: Men = 537–70 mg/dL
Women = 540–88 mg/dL
LDL: < 130 mg/dL
(*Note:* Normal values may vary by laboratory.)

RELATED NURSING CARE Cholesterol levels alone may be measured at any time of the day, regardless of food or fluid intake. When measuring triglycerides and lipoproteins (HDL and LDL), fasting for 12 hours (except for water) with no alcohol intake for 24 hours prior to the test is recommended.

NAME OF TEST Electrocardiogram (ECG)

PURPOSE AND DESCRIPTION See Boxes 28.1 and 28.2.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Chest x-ray

PURPOSE AND DESCRIPTION An x-ray of the thorax can illustrate the contours, placement and chambers of the heart. It may be done to identify heart displacement or hypertrophy, or fluid in the pericardial sac.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Stress/exercise tests

- Treadmill test

PURPOSE AND DESCRIPTION Stress testing is based on the theory that CAD results in depression of the ST segment with exercise. Depression of the ST segment and depression or inversion of the T wave indicates myocardial ischaemia. When the person is walking on a treadmill machine, the work rate of the heart is changed every 3 minutes for 15 minutes by increasing the speed and degree of incline by 3% each time. People exercise until they are fatigued, develop symptoms or reach their maximum predicted heart rate.

RELATED NURSING CARE *For all stress/exercise tests:* Ask the person to wear comfortable shoes, and to avoid food, fluids and smoking for 2 to 3 hours before the test. Assess for events that contraindicate the tests: recent myocardial infarction; severe, unstable angina; controlled arrhythmias; congestive heart failure or recent pulmonary embolism.

NAME OF TEST Thallium/technetium stress test (Myocardial perfusion scan, Cardiac blood pool imaging)

PURPOSE AND DESCRIPTION *Thallium stress test:* Thallium-201, a radioisotope that accumulates in myocardial cells, is used during the stress test to evaluate myocardial perfusion. Second scans are done 2 to 3 hours later when the heart is at rest; this is to differentiate between an ischaemic area and an infarcted or scarred area of myocardium.

Exercise technetium perfusion test: Technetium-99m-laced compounds are administered and a scan is done to evaluate cardiac perfusion, wall motion and ejection fraction. This is probably the most useful non-invasive test to diagnose and monitor CAD.

RELATED NURSING CARE Assess medications; those that affect the blood pressure or heart rate should be discontinued for 24 to 36 hours prior to the test (unless the test is being done to monitor the effectiveness of the medications).

NAME OF TEST Nuclear persantin (dipyridamole) stress test (Myocardial perfusion scan)

PURPOSE AND DESCRIPTION This test is used when the person is not physically able to walk on the treadmill. Persantin, given IV, dilates the coronary arteries and increases myocardial blood flow. Coronary arteries that are narrowed from CAD cannot dilate to increase myocardial perfusion.

RELATED NURSING CARE Person is nil by mouth (NBM) after midnight except for water. Food, fluids and drugs that contain caffeine should be avoided for 24 hours prior to the test, as should decaffeinated fluids. Some drugs, such as theophylline preparations, are discontinued for 36 hours prior to the test.

DIAGNOSTIC TESTS Cardiac disorders (continued)

NAME OF TEST Nuclear dobutamine stress test

PURPOSE AND DESCRIPTION Dobutamine is an adrenergic drug that increases myocardial contractility, heart rate and systolic blood pressure, which increases coronary oxygen consumption and thus increases coronary blood flow.

RELATED NURSING CARE Person is NBM after midnight except for water. Discontinue beta-blockers, calcium channel blockers and ACE inhibitors for 36 hours prior to the test. Do not administer nitrates for 6 hours prior to the test.

NAME OF TEST Magnetic resonance imaging (MRI)

PURPOSE AND DESCRIPTION An MRI may be used to identify and locate areas of myocardial infarction.

RELATED NURSING CARE Assess for any metallic implants (such as pacemaker, body piercing or artificial joint), which would contraindicate the test.

NAME OF TEST Computed tomography (CT) scan

PURPOSE AND DESCRIPTION A CT scan may be conducted to quantify calcium deposits in coronary arteries.

RELATED NURSING CARE Assess for allergy to iodine or seafood if contrast medium is to be administered.

NAME OF TEST Cardiolite—technetium-99m sestamibi (Myocardial perfusion scan)

PURPOSE AND DESCRIPTION Used to evaluate blood flow in different parts of the heart. Cardiolite (technetium-99m sestamibi) is injected IV. In a dipyridamole cardiolite scan, dipyridamole (Persantin) is injected to increase blood flow to coronary arteries. These scans may be done in conjunction with a treadmill test.

RELATED NURSING CARE As for stress/exercise tests, above. Instruct the person to avoid intake of caffeine for 12 hours before having a test with dipyridamole cardiolite.

NAME OF TEST Positron emission tomography (PET)

PURPOSE AND DESCRIPTION Two scans are performed following injection of radionuclides, and the resulting images compared for myocardial perfusion and myocardial metabolic function. A stress test (treadmill) may be a part of the test. If the myocardium is ischaemic or damaged, the images will be different. Normally, the images will be the same.

RELATED NURSING CARE Assess person's blood glucose: for accurate metabolic activity images, the blood glucose level must be between 60 and 140 mg/dL. If exercise is included in the test, the person will need to be NBM and avoid smoking and caffeine for 24 hours prior to the test.

NAME OF TEST Blood pool imaging

PURPOSE AND DESCRIPTION Following intravenous injection of technetium-99m pertechnetate, sequential evaluation of the heart can be performed for several hours. Useful for evaluation of cardiac status following myocardial infarction and congestive heart failure and assessing effectiveness of cardiac medications. Can be done at the person's bedside.

RELATED NURSING CARE No special preparation is needed. Following the procedure, explain that the dose if very low and the biological elimination of the technetium-99m is virtually complete by 6 hours. It is best to refrain from breastfeeding during this time.

NAME OF TEST Echocardiogram

- M-mode
- Two-dimensional (2-D)
- Cardiac Doppler
- Colour Doppler
- Stress echocardiogram

PURPOSE AND DESCRIPTION Echocardiograms use a transducer to record waves that are bounced off the heart, and to record the direction and flow of blood through the heart in audio and graphical data. An *M-mode (motion-mode) echocardiogram* records the motion, wall thickness and chamber size of the heart. A *2-D echocardiogram* provides a cross-sectional view of the heart. *Colour flow imaging* combines 2-D echocardiography and Doppler technology to evaluate the speed and direction of blood flow through the heart, which can identify pathology such as leaky valves. *Stress echocardiography* combines a treadmill test with ultrasound images to evaluate segmental function and wall motion. If the person is not physically able to exercise, IV dobutamine may be administered and ultrasound images taken.

RELATED NURSING CARE No special preparation is needed; see related nursing care for the person having a treadmill test for a stress echocardiogram.

NAME OF TEST Transoesophageal echocardiography (TOE)

PURPOSE AND DESCRIPTION Allows visualisation of adjacent cardiac and extracardiac structures to identify or monitor mitral and aortic valve pathology, left atrium intracardiac thrombus, acute dissection of the aorta, endocarditis, perioperative left ventricular function and intracardiac repairs during surgery. A transducer (probe) attached to an endoscope is inserted into the oesophagus and images are taken. Concurrent IV contrast medium, Doppler ultrasound and colour flow imaging may be used.

(continued)

DIAGNOSTIC TESTS Cardiac disorders (continued)

RELATED NURSING CARE A light sedative is often used and a local anaesthetic is applied to the oropharynx. It is important to ensure airway patency during this time. If constrast medium is to be administered, assess for allergy to iodine or seafood.

Following the procedure, explain that it may be difficult to swallow for a few hours and may be uncomfortable to swallow for a few days.

NAME OF TEST Cardiac catheterisation (Coronary angiography, Coronary arteriography)

PURPOSE AND DESCRIPTION A cardiac catheterisation may be performed to identify CAD or cardiac valvular disease, to determine pulmonary artery or heart chamber pressures, to obtain a myocardial biopsy, to evaluate artificial valves, or to perform angioplasty or stent an area of CAD. The test is performed by inserting a long catheter into a vein or artery (depending on whether the right side or the left side of the heart is being examined) in the arm or leg. Using fluoroscopy, the catheter is then threaded to the heart chambers or coronary arteries, or both. Contrast dye is injected and heart structures are visualised and heart activity is filmed. The test is done for diagnosis and before heart surgery.

Right cardiac catheterisation: The catheter is inserted into the femoral vein or antecubital vein and then threaded through the inferior vena cava into the right atrium to the pulmonary artery. Pressures are measured at each site, and blood samples can be obtained for the right side of the heart. The functions of the tricuspid and pulmonary valves can be observed.

Left cardiac catheterisation: The catheter is inserted into the brachial or femoral artery and advanced retrograde through the aorta to the coronary arteries and/or left ventricle. The patency of the coronary arteries and/or functions of the aortic and mitral valves and left ventricle can be observed.

RELATED NURSING CARE See 'Nursing care' box following.

NAME OF TEST Pericardiocentesis

PURPOSE AND DESCRIPTION This procedure is performed to remove fluid from the pericardial sac for diagnostic or therapeutic purposes. It may also be done as an emergency procedure for the person with cardiac tamponade (which may result in death). A large-gauge (16 to 18) needle is inserted to the left of the xiphoid process into the pericardial sac and excess fluid is withdrawn (see Figure 28.22). The needle is attached to an ECG lead to help determine if the needle is touching the epicardial surface, thus preventing piercing of the myocardium.

RELATED NURSING CARE See 'Nursing care' box following.

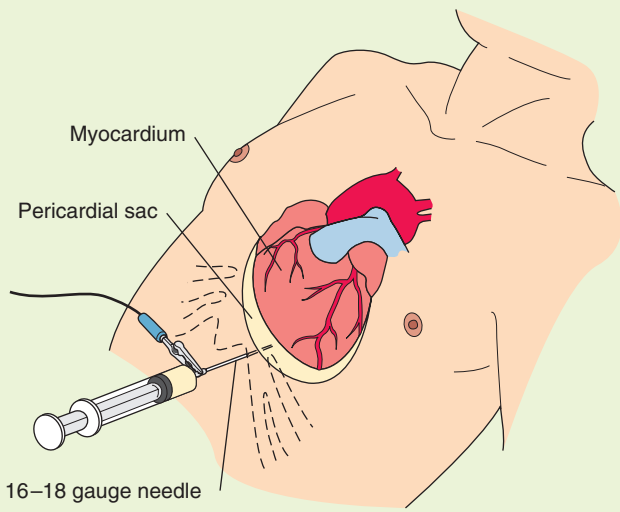


FIGURE 28.22 ■ Pericardiocentesis

BOX 28.1 Electrocardiogram

The *electrocardiogram (ECG)* is a graphical record of the heart's electrical activity. Electrodes applied to the body surface are used to obtain a graphical representation of cardiac electrical activity. These electrodes detect the magnitude and direction of electrical currents produced in the heart. They attach to the electrocardiograph by an insulated wire called a *lead*. The electrocardiograph converts the electrical impulses it receives into a series of waveforms that represent cardiac depolarisation and repolarisation. Placement of electrodes on different parts of the body allows different views of this electrical activity, much like turning the head while holding a camera provides different views of the scenery. ECG waveforms and patterns are examined to detect arrhythmias as well as myocardial damage, the effects of drugs and electrolyte imbalances.

ECG waveforms reflect the direction of electrical flow in relation to a positive electrode. Current flowing towards the positive electrode produces an upward (positive) waveform;

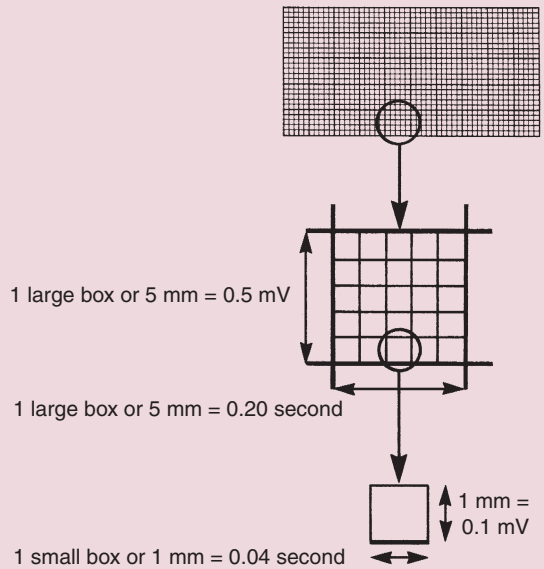
BOX 28.1 Electrocardiogram (continued)

current flowing away from the positive electrode produces a downward (negative) waveform. Current flowing perpendicular to the positive pole produces a biphasic (both positive and negative) waveform. Absence of electrical activity is represented by a straight line called the *isoelectric line*.

ECG waveforms are recorded by a heated stylus on heat-sensitive paper. The paper is marked at standard intervals that represent time and voltage or amplitude (see Figure 1). Each small box is 1 mm^2. The recording speed of the standard ECG is 25 mm/second, so each small box represents 0.04 second. Five small boxes horizontally and vertically make one large box, equivalent to 0.20 second. Five large boxes represent 1 full second. Measured vertically, each small box represents 0.1 mV.

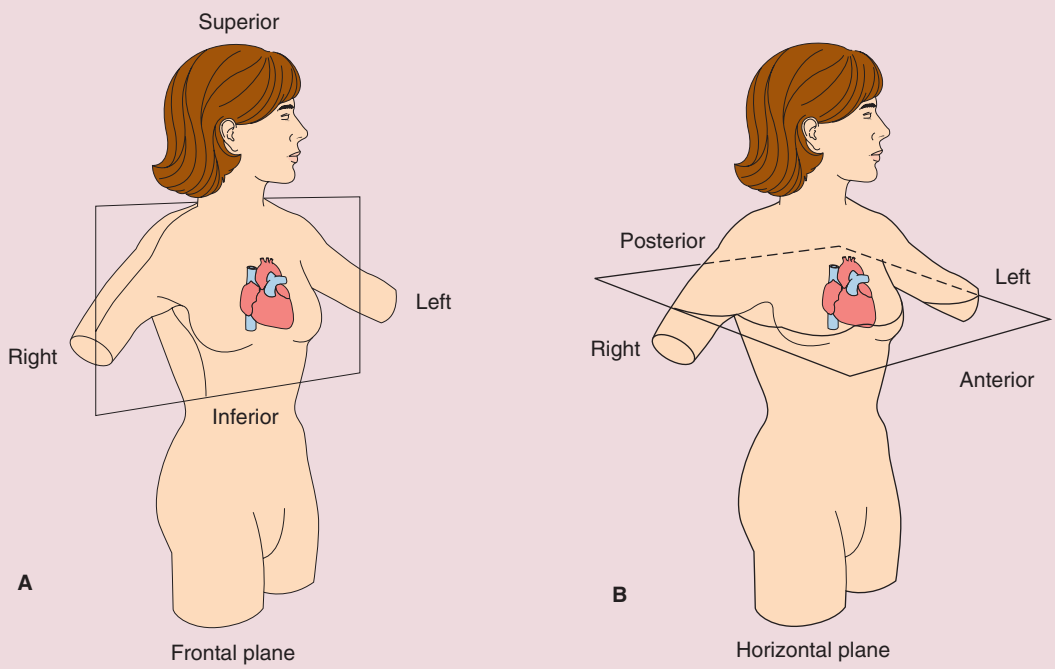
Both bipolar and unipolar leads are used in recording the ECG. A bipolar lead uses two electrodes of opposite polarity (negative and positive). In a *unipolar* lead, one positive electrode and a negative reference point at the centre of the heart are used. The electrical potential between the two monitoring points is graphically recorded as the ECG waveform.

The heart can be viewed from both the frontal plane and the horizontal plane (see Figure 2). Each plane provides a unique perspective of the heart muscle. The frontal plane is an imaginary cut through the body that views the heart from top to bottom (superior–inferior) and side to side (right–left). This perspective of the heart is analogous to a paper doll cut-out. It provides information about the inferior and lateral walls of the heart. The horizontal plane is a cross-sectional view of the heart from front to back (anterior–posterior) and side to side (right–left). Information regarding the anterior, septal and lateral walls of the heart, as well as the posterior wall, is obtained from this view.



(1) Time and voltage measurements on ECG paper at a recording speed of 25 mm/second

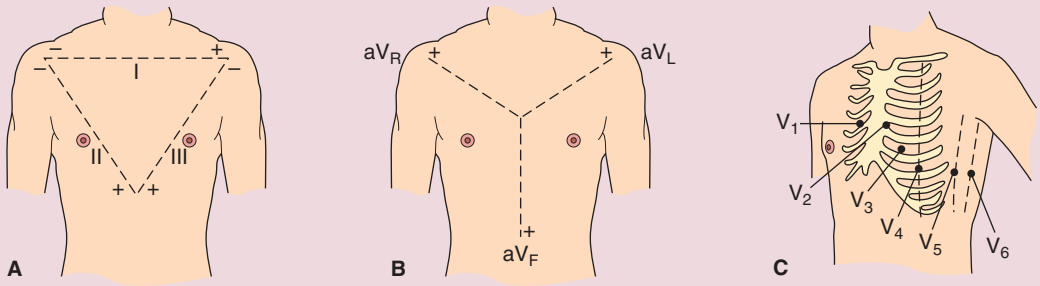
A standard 12-lead ECG provides a simultaneous recording of six limb leads and six precordial leads (see Figure 3). The limb leads provide information about the heart in the frontal plane and include three bipolar leads (I, II, III) and three unipolar leads (aV_R, aV_L and aV_F). The bipolar limb leads measure electrical activity between a negative lead on one extremity and a positive lead on another. The unipolar limb leads (called augmented leads) measure the electrical activity



(2) Planes of the heart. *A*, Frontal plane, *B*, Horizontal plane

(continued)

BOX 28.1 Electrocardiogram (continued)



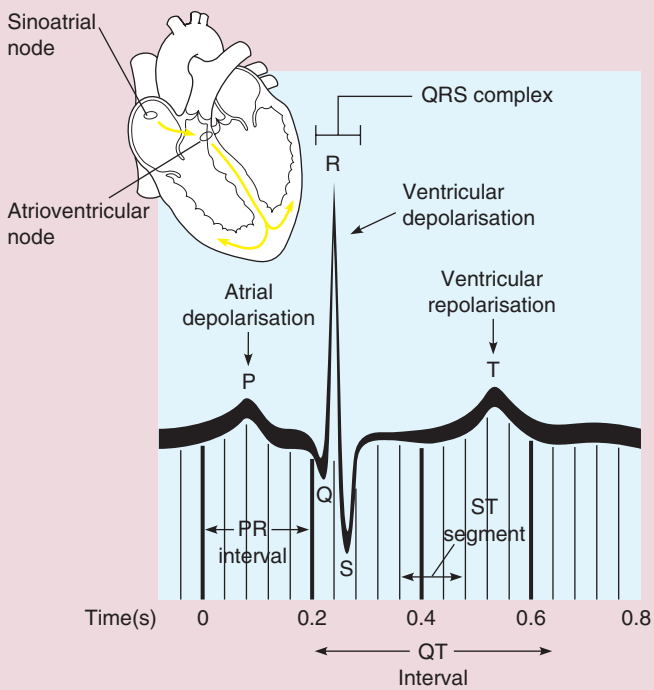
(3) Leads of the 12-lead ECG. *A*, Bipolar limb leads I, II, III; *B*, Unipolar limb leads aV_R, aV_L, aV_F; *C*, Unipolar precordial leads V_1 to V_6

between a single positive electrode on a limb (right arm (R), left arm (L) or left leg (F for foot)) and the centre of the heart.

The *precordial leads*, also known as chest leads or V leads, view the heart in the horizontal plane. They include six unipolar leads (V_1, V_2, V_3, V_4, V_5 and V_6), which measure electrical activity between the centre of the heart and a positive electrode on the chest wall.

The cardiac cycle is depicted as a series of waveforms, the P, Q, R, S and T waves (see Figure 4).

- The *P wave* represents atrial depolarisation and contraction. The impulse is from the sinoatrial (or sinus) node. The P wave precedes the QRS complex and is normally smooth, round and upright. P waves may be absent when the sinoatrial node is not acting as the pacemaker. Atrial repolarisation occurs during ventricular depolarisation and usually is not seen on the ECG.
- The *PR interval* represents the time required for the sinus impulse to travel to the atrioventricular node and into the Purkinje fibres. This interval is measured from beginning of P wave to beginning of QRS complex. If no Q wave is seen, the beginning of the R wave is used. The PR interval is normally 0.12 to 0.20 second. (Up to 0.24 second is considered normal in adults over age 65.) PR intervals greater than 0.20 second indicate a delay in conduction from the sinoatrial node to the ventricles.
- The *QRS complex* represents ventricular depolarisation and contraction. The QRS complex includes three separate waves: the Q wave is the first negative deflection, the R wave is the positive or upright deflection, and the S wave is the first negative deflection after the R wave. Not all QRS complexes have all three waves; nonetheless, the complex is called a QRS complex. The normal duration of a QRS complex is from 0.06 to 0.10 second. QRS complexes greater than 0.10 second indicate delays in transmitting the impulse through the ventricular conduction system.
- The *ST segment* signifies the beginning of ventricular repolarisation. The ST segment, the period from the end of the QRS complex to the beginning of the T wave, should be isoelectric. An abnormal ST segment is displaced (elevated or depressed) from the isoelectric line.
- The *T wave* represents ventricular repolarisation. It normally has a smooth, rounded shape that is usually less than 10 mm tall. It usually points in the same direction as the QRS complex. Abnormalities of the T wave may indicate myocardial ischaemia or injury or electrolyte imbalances.
- The *QT interval* is measured from the beginning of the QRS complex to the end of the T wave. It represents the total time of ventricular depolarisation and repolarisation. Its duration varies with gender, age and heart rate; usually, it is 0.32 to 0.44 second long. Prolonged QT intervals indicate a prolonged relative refractory period and a greater risk of arrhythmias. Shortened QT intervals may result from medications or electrolyte imbalances.
- The *U wave* is not normally seen. It is thought to signify repolarisation of the terminal Purkinje fibres. If present, the U wave follows the same direction as the T wave. It is most commonly seen in hypokalaemia.



(4) Normal ECG waveform and intervals

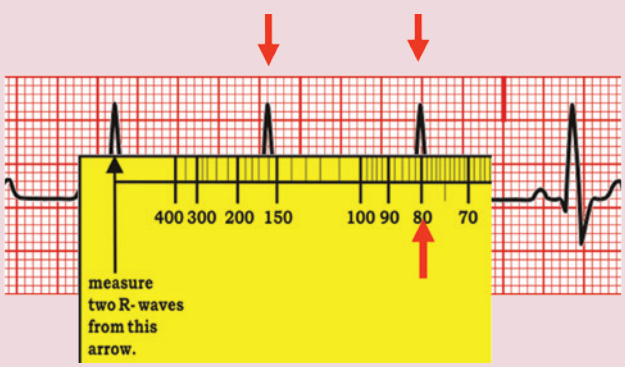
BOX 28.2 Interpreting an ECG

Interpreting an ECG strip to determine the cardiac rhythm is a skill that takes practice to learn and master. Many methods are used to analyse ECGs. It is important to use a consistent method for ECG analysis. Identifying and interpreting complex arrhythmias requires advanced skills and knowledge obtained through further training. One method follows.

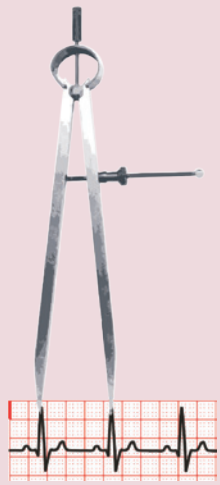
- **Step 1: Determine rate**. Assess heart rate. Use P waves to determine the atrial rate and R waves for the ventricular rate. Several approaches can be used to determine the heart rate.
  - Count the number of complexes in a 6-second rhythm strip (the top margin of ECG paper is marked at 3-second intervals) and multiply by 10. This provides an estimate of the rate and is particularly valuable if rhythms are irregular.
  - Count the number of large boxes between two consecutive complexes and divide 300 (the number of large boxes in 1 minute) by this number. For example, there are 6 large boxes between two R waves; 300 divided by 6 equals a ventricular rate of 50 bpm. Memorise the following sequence for rapid rate determination: 300, 150, 100, 75, 60, 50, 43. One large box between complexes equals a rate of 300; two, a rate of 150; three, a rate of 100; and so on.
  - Count the number of small boxes between two consecutive complexes and divide 1500 (the number of small boxes in 1 minute) by this number. For example, there are 19 small boxes between two R waves; 1500 divided by 19 equals a ventricular rate of 79 bpm. This is the most precise measurement of heart rate.
  - Use a commercially available rate ruler, following strictly the instructions on the device (see Figure 1).
- **Step 2: Determine regularity**. Regularity is the consistency with which the P waves or QRS complexes occur. In a regular rhythm, all waves occur at a consistent rate. Rhythm regularity is determined by measuring the interval between consecutive waves. Place one point of an ECG calliper (a measuring device) on the peak of the P wave (for atrial rhythm) or the R wave (for ventricular rhythm). Adjust the other point to the peak of the next wave, P to P or R to R (see Figure 2). Keeping the callipers set at this distance, evaluate intervals between consecutive waves. The rhythm is *regular* if all calliper points fall on succeeding wave peaks. Alternately, use a strip of blank paper on top of the ECG strip, marking the peaks of two or three consecutive waves. Then move the paper along the strip to consecutive waves. Wave peaks that vary by more than one to three small boxes (depending on the rate) are *irregular*. Irregular rhythms may be *irregularly irregular* (if the intervals have no pattern) or *regularly irregular* (if a consistent pattern to the irregularity can be identified).
- **Step 3: Assess P wave**. The presence or absence of P waves helps determine the origin of the rhythm. All the P waves should be alike in size and shape (*morphology*). If P waves are not seen or they differ in shape, the rhythm may not originate in the sinoatrial node.
- **Step 4: Assess P to QRS relationship**. Determine the relationship between P waves and QRS complexes. There should be one and only one P wave for every QRS complex, because the normal stimulus for ventricular contraction originates in the sinoatrial node.
- **Step 5: Determine interval durations**. To evaluate impulse transmission through the cardiac conduction system, measure the PR interval, QRS duration and QT interval. To measure, count the number of small boxes from the beginning of the interval to the end and multiply by 0.04 second. Then determine whether the interval duration is within its normal limits. For example, the PR interval is 3.5 small boxes wide, or 0.14 second. This is within the normal limits of 0.12 to 0.20 second. This interval should be consistent, not varying from beat to beat. A PR interval greater than 0.20 second or one that varies from beat to beat is abnormal.

  The QRS complex duration is normally between 0.06 and 0.10 second. A QRS complex greater than 0.12 second indicates delayed ventricular conduction.

  The QT interval is normally 0.32 to 0.44 second. It varies inversely with the heart rate: the faster the heart rate, the shorter the QT interval. As a general rule, the QT interval should be no more than half the previous R–R interval. A prolonged QT interval indicates a prolonged relative refractory period of the heart.
- **Step 6: Identify abnormalities**. Note the presence and frequency of ectopic (extra) beats, deviation of the ST segment above or below the baseline, and abnormalities in waveform shape and duration.



(1) Example of commercially available rate rule



(2) Use of callipers to determine rate

NURSING CARE OF THE PERSON having cardiac catheterisation

BEFORE THE PROCEDURE

- Explain the procedure to the person.
- No food or fluids are allowed for 6 to 8 hours before the test.
- Assess for allergies to seafood, iodine or iodine contrast dyes (if previous tests have been done). If an allergic response to the dye is possible, antihistamines (such as Benadryl) or steroids may be administered the evening before and the morning of the test.
- Assess for use of aspirin or NSAIDs (risk of bleeding), sildenafil (Viagra; risk of heart problems) or history of kidney disease (dye used may be toxic to the kidneys).
- Discontinue oral anticoagulant medications. Heparin may be ordered to prevent thrombi.
- An IV (with isotonic crystalloid solution) is started at a 'to-keep-vein-open' (TKVO) rate (in case intravenous access is needed for emergency drugs).
- Establish baseline of peripheral pulses.
- Take and record baseline vital signs.

PROCEDURE

- The person is positioned on a padded table that tilts. A local anaesthetic is used at the site of catheter insertion. ECG leads are applied and vital signs are monitored during the procedure. The person lies supine and is asked to cough and deep breathe frequently. The procedure takes ½ to 3 hours.
- Tell the person that a hot, flushing sensation may be felt for a minute or two when the dye is injected.

AFTER THE PROCEDURE

- Monitor vital signs every 15 minutes for the first hour and then every 30 minutes until stable. Assess cardiac rhythm and rate for alterations. Assess peripheral pulses distal to the insertion site.
- Assess the person for chest heaviness, shortness of breath, and abdominal or groin pain.
- Monitor catheter insertion site for bleeding or haematoma.
- Administer pain medications as prescribed.
- Instruct the person to remain on bed rest for 6 to 12 hours (or as ordered). If a collagen-like plug was inserted after removal of the catheter, only a 2- to 3-hour bed rest is necessary.
- Encourage oral fluids unless contraindicated (i.e. if the person has congestive heart failure).

NURSING CARE OF THE PERSON having pericardiocentesis

BEFORE THE PROCEDURE

- Gather all supplies:
  a. Pericardiocentesis tray
  b. ECG machine and electrode patches
  c. Emergency cart with defibrillator
  d. Dressing
  e. Culture bottles (if indicated).
- Reinforce teaching and answer questions about the procedure or associated care. Provide emotional support.
- Ensure that informed consent has been obtained.
- Provide for privacy.
- Obtain and document baseline vital signs.
- Connect the person to a cardiac monitor; obtain a baseline rhythm strip for comparison during and after the procedure.
- Connect the precordial ECG lead of the hub of the aspiration needle using an alligator clamp.

DURING THE PROCEDURE

- Follow standard precautions.
- Position seated at a 45- to 60-degree angle. Place a dry towel under the rib cage to catch blood or fluid leakage.
- Observe the ST segment for elevation and the ECG monitor for signs of myocardial irritability (PVCs) during the procedure. These indicate that the needle is touching the myocardium and should be withdrawn slightly.
- Notify the physician of changes in cardiac rhythm, blood pressure, heart rate, level of consciousness and urine output. These may indicate cardiac complications.
- Monitor central venous pressure (CVP) and blood pressure closely. As the effusion is relieved, CVP will decrease and BP will increase.

AFTER THE PROCEDURE

- Document the procedure and the person's response to and tolerance of the procedure.
- Continue to monitor vital signs and cardiac rhythm every 15 minutes during the first hour, every 30 minutes during the next hour, and every hour for the next 24 hours.
- Record the amount of fluid removed as output on the intake and output record.
- If indicated, send a sample of aspirated fluid for culture and sensitivity and laboratory analysis.
- Assess heart and breath sounds.

DIAGNOSTIC TESTS Haematological, peripheral vascular and lymphatic disorders

NAME OF TEST Full blood count (FBC)

PURPOSE AND DESCRIPTION This is a blood test involving several measurements of blood components. See Table 28.1.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Erythrocyte sedimentation rate (ESR)

PURPOSE AND DESCRIPTION This blood test is done as a measure of inflammation and is increased in many illnesses, including cancer, heart disease and kidney disease. This test is performed to detect and monitor inflammation in both acute and chronic medical conditions. The findings are used to assist in identifying the cause of inflammation, infections, cancers and autoimmune diseases. It is a non-specific test; therefore, it is typically used in conjunction with other tests, such as FBC, to determine a specific cause of ill health.

Normal values:
Women: 1–12 mm in 1 hour; men: 1–14 mm in 1 hour; pregnant women: > 30 mm in the first hour (due to elevated levels of plasma globulins and fibrinogen)—therefore, ESR cannot be used as an inflammatory marker in pregnancy.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Bone marrow biopsy

PURPOSE AND DESCRIPTION Conducted to evaluate blood-forming tissue; to diagnose multiple myeloma, leukaemia and some lymphomas; and to assess effectiveness of therapy for leukaemia. Bone marrow is removed from a site such as the posterior iliac crest with needle aspiration.

RELATED NURSING CARE See 'Nursing care' box below.

NAME OF TEST Magnetic resonance angiography (MRA)

PURPOSE AND DESCRIPTION Used to visualise vascular occlusive disease and aneurysms of the abdominal aorta. The procedure is done by using a non-iodine-based contrast medium injected IV.

RELATED NURSING CARE Assess for any metallic implants, such as a pacemaker or body piercings. (If present, test will not be performed.)

NAME OF TEST Magnetic resonance imaging (MRI)

PURPOSE AND DESCRIPTION A radiological study used to visualise liver, spleen and lymph nodes. Does not require injection of contrast medium.

RELATED NURSING CARE Assess for any metallic implants, such as a pacemaker or body piercings. (If present, the test will not be performed.)

NAME OF TEST Computed tomography (CT) scan

PURPOSE AND DESCRIPTION A radiological study used to evaluate the lymph nodes. Contrast medium may be used when assessing the nodes of the abdomen.

RELATED NURSING CARE Assess for allergy to iodine (such as shellfish) if contrast medium is to be administered.

NAME OF TEST Liver and/or spleen scan

PURPOSE AND DESCRIPTION A radiological study used to assess the liver and/or spleen. A radioisotope is injected IV prior to the scan.

RELATED NURSING CARE Assess for allergy to iodine (such as shellfish).

NAME OF TEST Lymphangiography (lymphangiogram)

PURPOSE AND DESCRIPTION This is an x-ray examination of the lymphatic vessels and lymph nodes, used to assess metastasis of the lymph nodes, to identify malignant lymphoma and to identify the cause of lymphoedema. An iodine contrast substance is injected at various sites and fluoroscopy is used to visualise lymphatic filling.

RELATED NURSING CARE Ask the person about allergies to seafood, iodine or contrast medium used in a previous x-ray test. Tell the person that the blue contrast dye discolours the urine and possibly the skin for a few days.

(continued)

DIAGNOSTIC TESTS Haematological, peripheral vascular and lymphatic disorders (continued)

NAME OF TEST Lymph node biopsy

PURPOSE AND DESCRIPTION Done to obtain tissue for histological examination for diagnosis and treatment. May be open (performed in the operating room) or closed (needle) by needle aspiration of tissue from a lymph node.

RELATED NURSING CARE Use sterile technique when changing dressings.

Disorders of the arteries and veins are diagnosed by various non-invasive examinations, including transoeosophageal echocardiography (TOE), ultrasound and Doppler studies. A magnetic resonance angiography (MRA) may be done to visualise vascular occlusive disease and abdominal aorta aneurysms.

Tests of the lymphatic system, including a lymphangiogram and a lymph node biopsy, may be done to identify malignancies, assess metastasis of cancer to lymph nodes, identify the causes of lymphoedema and obtain tissue for diagnosis and treatment.

NURSING CARE OF THE PERSON having bone marrow studies

Bone marrow specimens are obtained by either aspiration or biopsy. The preferred site for bone marrow aspiration is the posterior iliac crest; the anterior iliac crest or the sternum may also be used. The procedure is performed by inserting a needle into the bone and drawing out a sample of the blood in the marrow. A bone marrow biopsy is performed by making a small incision over the bone and screwing a core biopsy instrument into the bone to obtain a specimen. Bone marrow studies are used to diagnose leukaemias, metastatic cancer, lymphoma, aplastic anaemia and Hodgkin's disease.

PREPARATION OF THE PERSON

- Explain the purpose and procedure of the test.
- Ensure presence of a signed consent for the procedure.
- Offer sedation (neuroleptic or tranquillising) and/or pain relief before the procedure to alleviate discomfort.
- Record vital signs.
- Ask the person to void.
- Support person into the supine position if the specimen will be obtained from the sternum or anterior iliac crest; prone position if the posterior iliac crest will be used.
- Assist the person to remain still during the procedure.

AFTER THE PROCEDURE

- Apply pressure to the puncture site for 5 to 10 minutes.
- Assess vital signs and compare results with pre-procedure readings.
- Apply a dressing to the puncture site and monitor for bleeding and infection for 24 hours.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- The procedure (either aspiration or biopsy) takes about 20 minutes.
- A sedative may be given prior to the procedure.
- It is important the person remains very still during the procedure to prevent accidental injury.
- Although the area will be anaesthetised with a local anaesthetic, insertion of the needle will be painful for a short time. Taking deep breaths may make this part of the procedure less painful for the person. (In certain circumstances, such as with confused or paediatric patients, sedation or a short-acting general anaesthetic may be used to prevent excessive movement during the procedure.)
- The aspiration site may ache for 1 or 2 days.
- Ask the person to report any unusual bleeding immediately.

Physical assessment

Physical assessment of cardiovascular and lymphatic function may be performed either as part of a total assessment or alone for people with suspected or known problems with cardiovascular or lymphatic function. Assess the heart through inspection, palpation and auscultation over the precordium (the area of the chest wall overlying the heart). Normal age-related findings for the older person are summarised in Table 28.4.

The techniques used to assess these systems include inspection of the skin for such changes as oedema, ulcerations or alterations in colour and temperature; auscultation of blood pressure; and palpation of the major pulse points of the body (see Figure 28.23) and lymph nodes. The person may be assessed in the supine, sitting and standing positions. Normal age-related findings for the older adult are summarised in Table 28.5.

Before beginning the assessment, collect all required equipment and explain the techniques to the person to decrease anxiety. A quiet environment is essential to hear and assess heart sounds accurately.

The person may sit or lie in the supine position. Movements over the precordium may be more easily seen with tangential lighting (in which the light is directed at a right angle to the area being observed, producing shadows). Assess the following types of movements:

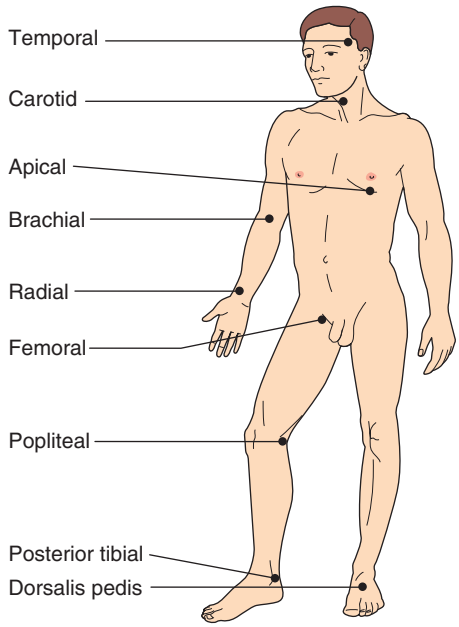


FIGURE 28.23 ■ Body sites at which peripheral pulses are most easily palpated

- The **apical impulse** is a normal, visible pulsation in the area of the midclavicular line in the left fifth intercostal space. It can be seen on inspection in about half of the adult population. (The apical impulse was previously called the point of maximal impulse (PMI), but this term is no longer used because a maximal impulse may occur in other areas of the precordium as a result of abnormal conditions.)
- Retraction is a pulling in of the tissue of the precordium; a slight retraction just medial to the midclavicular line at the area of the apical impulse is normal and is more likely to be visible in people who are thin.
- Pulsations (other than the normal apical pulsations), which may be called heaves or lifts, are considered abnormal. They may occur as the result of an enlarged ventricle.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of the person. During the health assessment interview, ask about family members with health problems affecting cardiovascular and lymphatic function, or about a family history of high cholesterol levels or early-onset

TABLE 28.4 Age-related changes in the heart

AGE-RELATED CHANGE	SIGNIFICANCE
Myocardium: ↓ efficiency and contractibility. Sinoatrial node: ↑ in thickness of shell surrounding the node and a ↓ in the number of pacemaker cells.	■ Decreased cardiac output when under physiological stress with resulting tachycardia that lasts longer. The person may require rest time between physical activities.
Left ventricle: slight hypertrophy, prolonged isometric contraction phase and relaxation time; ↑ time for diastolic filling and systolic emptying cycle.	■ Stroke volume may increase to compensate for tachycardia, leading to increased blood pressure.
Valves and blood vessels: aorta is elongated and dilated, valves are thicker and more rigid, and resistance to peripheral blood flow increases by 1% per year.	■ Blood pressure increases to compensate for increased peripheral resistance and decreased cardiac output.

TABLE 28.5 Age-related changes in the haematological, peripheral vascular and lymphatic systems

AGE-RELATED CHANGE	SIGNIFICANCE
Bone marrow: ↓ ability of bone marrow to respond to need for increased RBCs, WBCs and platelets.	■ Anaemia may result.
Blood vessels: ■ *Tunica intima:* fibrosis, calcium and lipid accumulation, cellular proliferation.	■ As a result of age-related changes, the systolic blood pressure rises. Decreased arterial elasticity results in vascular changes in the heart, kidneys and pituitary gland. Decreased baroreceptor function results in postural hypotension. Vessels in the head, neck and extremities are more prominent.
■ *Tunica media:* thins, elastin fibres calcify; increase in calcium results in stiffening. Baroreceptor function is impaired and peripheral resistance increases.	■ Inefficient vasoconstriction, decreased cardiac output and reduced muscle mass and subcutaneous tissue lead to a reduced ability to respond to cold temperatures. ■ With a decrease in blood pressure and changes in blood vessel walls, tissue perfusion may be inadequate, leading to oedema, inflammation, pressure ulcers and changes in effects of medications.
■ *Tunica adventitia:* no change.	
Immune system: ■ Impaired function of B and T lymphocytes. ■ Decreased production of antibodies. ■ Unable to distinguish 'self' from 'non-self'. ■ Phagocytic immune response delayed.	■ Increased risk of infection, with decreased manifestations of an actual infection. ■ Increased incidence of cancers. ■ Altered response to antigens (such as Mantoux tuberculin test). ■ May have reactivation of tuberculosis.

coronary artery disease. Ensure that the person considers both alive and deceased relatives in their family history. In addition, ask about a family history of high blood pressure, haemophilia, chronic myeloid leukaemia, porphyria and/or atherosclerosis. Depending on the racial and ethnic background of the person, ask about any family members with sickle cell anaemia or thalassaemia. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the 'Genetic considerations' box below). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

GENETIC CONSIDERATIONS Cardiovascular, haematological and lymphatic system disorders

Cardiovascular disorders

- Familial hypercholesterolaemia is a single gene disorder that results in atherosclerosis and CAD, which may occur at an earlier age than in the general population (i.e. before age 55 in men and age 65 in women). However, increased cholesterol levels may also be inherited and are a risk factor for CAD in both men and women.
- Marfan's syndrome is an autosomal dominant inherited disorder that affects the skeleton, the eyes and the cardiovascular system. The cardiovascular effects are a dilatation of the proximal aorta and aortic dissection associated with degeneration of the elastic fibres in the tunica media of the aorta. There may also be thoracic aortic aneurysms.
- Supraventricular aortic stenosis (SVAS) is a genetic vascular disorder resulting in an hourglass-shaped stenosis of the ascending aorta. It may also affect other major arteries, including the pulmonary, carotid, cerebral, renal and coronary arteries.
- Hypertropic cardiomyopathy, a disease of sarcomere proteins, has a genetic transmission.
- Williams syndrome is a rare genetic disorder characterised by characteristic 'elfin-like' features and heart and blood vessel problems (as well as other physical problems).
- Long QT syndrome (LQTS) is an inherited genetic disorder that results from structural abnormalities of the potassium channels in the heart, leading to arrhythmias. This can result in unconsciousness and may cause sudden cardiac death in teenagers and young adults when exposed to stressors ranging from stressors to loud sounds.

Haematological, peripheral vascular and lymphatic disorders

- There is a genetic link in 30–40% of people with primary hypertension.
- Sickle cell anaemia is an inherited genetic disorder seen in people of Mediterranean, Caribbean, Central and South American, Arab or East Indian ancestry. As Australia is an increasingly multicultural society, healthcare professionals need to be aware of its existence among people with these racial backgrounds.
- Gaucher disease, more common in descendants of Eastern European Jewish people, is an inherited illness caused by a gene mutation. The gene is responsible for an enzyme that breaks down a specific fat. When the fat is not broken down, it accumulates in the liver, spleen and bone marrow, causing pain, fatigue, jaundice, bone damage, anaemia and even death.
- Haemophilia A is a hereditary blood disorder, primarily affecting males, characterised by a deficiency of the blood clotting factor named factor VIII. Abnormal bleeding results.
- Chronic myeloid leukaemia (CML), a cancer of blood cells, is characterised by replacement of bone marrow with malignant, leukaemic cells. Leukaemic cells also circulate in the blood, causing enlargement of the spleen, liver and other organs. This leukaemia is the result of a chromosomal abnormality called the Philadelphia chromosome.
- Porphyria is a group of genetic blood diseases in which haem production is disrupted. When haem production is disrupted, porphyrins (a part of haem) are overproduced and cause illnesses; they also give urine a reddish-purple colour.
- Thalassaemia, an inherited disease of faulty haemoglobin synthesis, is more often found in descendants of people living near the Mediterranean Sea, Africa, the Middle East and Asia. It comprises a group of disorders that range from very mild blood abnormalities to severe or fatal anaemia.
- Atherosclerosis is characterised by narrowing of arteries by cholesterol-rich plaques of immune system cells. Risk factors may be genetic and/or environmental. Although it may affect people at any age, it usually does not cause health problems until people are in their forties and fifties.

INTERPRETATION OF NORMAL AND ABERRANT DATA OBTAINED FROM CARDIOVASCULAR AND LYMPHATIC SYSTEM ASSESSMENT

CARDIAC ASSESSMENTS

Technique/normal findings	Abnormal findings
Apical impulse assessment	
First using the palmar surface and then repeating with finger pads, palpate the precordium for symmetry of movement and the apical impulse for location, size, amplitude and duration. The sequence for palpation is shown in Figure 28.24. To locate the apical impulse, ask the person to assume a left lateral recumbent position. Simultaneous palpation of the carotid pulse may also be helpful. *The apical impulse is not palpable in all people. The apical impulse may be palpated in the mitral area and has only a brief small amplitude.*	■ An enlarged or displaced heart is associated with an apical impulse lateral to the midclavicular line (MCL) or below the fifth left intercostal space (ICS). ■ Increased size, amplitude and duration of the apical impulse are associated with left ventricular volume overload (increased afterload) in conditions such as hypertension (HTN) and aortic stenosis, and with pressure overload (increased preload) in conditions such as aortic or mitral regurgitation. ■ Increased amplitude alone may occur with hyperkinetic states, such as anxiety, hyperthyroidism and anaemia. ■ Decreased amplitude is associated with a dilated heart in cardiomyopathy. ■ Displacement alone may also occur with dextrocardia, diaphragmatic hernia, gastric distension or chronic lung disease. ■ A **thrill** (a palpable vibration over the precordium or an artery) may accompany severe valve stenosis. ■ A marked increase in amplitude of the apical impulse at the right ventricular area occurs with right ventricular volume overload in atrial septal defect. ■ An increase in amplitude and duration occurs with right ventricular pressure overload in pulmonic stenosis and pulmonary hypertension. A lift or heave may also be seen in these conditions (and in chronic lung disease). ■ A palpable thrill in this area occurs with ventricular septal defect.
Palpate the subxiphoid area with the index and middle finger. *No pulsations or vibrations should be palpated.*	■ Right ventricular enlargement may produce a downward pulsation against the fingertips. ■ An accentuated pulsation at the pulmonary area may be present in hyperkinetic states. ■ A prominent pulsation reflects increased flow or dilation of the pulmonary artery. ■ A thrill may be associated with aortic or pulmonary stenosis, aortic stenosis, pulmonary HTN or atrial septal defect. ■ Increased pulsation at the aortic area may suggest aortic aneurysm. ■ A palpable second heart sound (S_2) may be noted with systemic HTN.
Cardiac rate and rhythm assessment	
Auscultate heart rate. *The heart rate should be 60 to 100 beats per minute with regular rhythm.*	■ A heart rate exceeding 100 beats per minute (beats/min) is tachycardia. A heart rate less than 60 beats/min is bradycardia.
Simultaneously palpate the radial pulse while listening to the apical pulse. *The radial and apical pulses should be equal.*	■ If the radial pulse falls behind the apical rate, the person has a pulse deficit, indicating weak, ineffective contractions of the left ventricle.

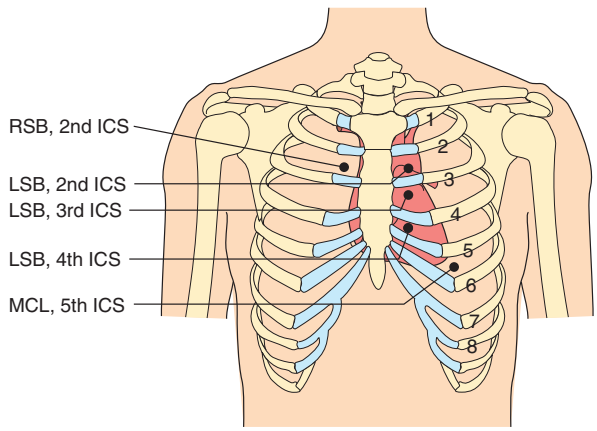


FIGURE 28.24 ■ Areas for inspection and palpation of the precordium, indicating the sequence for palpation (ICS = intercostal space; LSB = left sternal border; MCL = midclavicular line; RSB = right sternal border)

CARDIAC ASSESSMENTS (continued)

Technique/normal findings	Abnormal findings
Auscultate heart rhythm. *The heart rhythm should be regular.*	■ **Arrhythmias** (abnormal heart rate or rhythm) may be regular or irregular in rhythm; their rates may be slow or fast. Irregular rhythms may occur in a pattern (e.g. an early beat every second beat, called bigeminy), sporadically or with frequency and disorganisation (e.g. atrial fibrillation). A pattern of gradual increase and decrease in heart rate that is within the normal heart rate and that correlates with inspiration and expiration is called sinus arrhythmia.
Heart sounds assessment	
See guidelines for cardiac auscultation in Box 28.3.	
Identify S_1 (first heart sound) and note its intensity. At each auscultatory area, listen for several cardiac cycles. See Figure 28.25 for auscultation areas. *S_1 is loudest at the apex of the heart.*	■ An accentuated S_1 occurs with tachycardia, states in which cardiac output is high (fever, anxiety, exercise, anaemia, hyperthyroidism), complete heart block and mitral stenosis. ■ A diminished S_1 occurs with first-degree heart block, mitral regurgitation, CHF, CAD and pulmonary or systemic HTN. The intensity is also decreased with obesity, emphysema and pericardial effusion. Varying intensity of S_1 occurs with complete heart block and grossly irregular rhythms.
Listen for splitting of S_1. *Splitting of S_1 may occur during inspiration.*	■ Abnormal splitting of S_1 may be heard with right bundle branch block and premature ventricular contractions.
Identify S_2 (second heart sound) and note its intensity. *S_2 immediately follows S_1 and is loudest at the base of the heart.*	■ An accentuated S_2 may be heard with HTN, exercise, excitement, and conditions of pulmonary HTN such as CHF and cor pulmonale. ■ A diminished S_2 occurs with aortic stenosis, a fall in systolic blood pressure (shock) and increased anteroposterior chest diameter.
Listen for splitting of S_2. *No splitting of S_2 should be heard.*	■ Wide splitting of S_2 is associated with delayed emptying of the right ventricle, resulting in delayed pulmonary valve closure (e.g. mitral regurgitation, pulmonary stenosis and right bundle branch block).

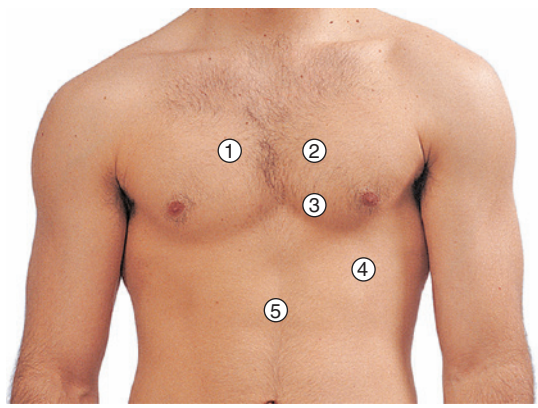


FIGURE 28.25 ■ Areas for auscultation of the heart

BOX 28.3 Guidelines for cardiac auscultation

1. Locate the major auscultatory areas on the precordium.
2. Choose a sequence of listening. Either begin from the apex and move upward along the sternal border to the base, or begin at the base and move downward to the apex.
3. Listen first with the person in the sitting or supine position. Then ask the person to lie on the left side and focus on the apex. Lastly, ask the person to sit up and lean forward. These position changes bring the heart closer to the chest wall and enhance auscultation. Carry out the following steps when the person assumes each of these positions:
   a. First, auscultate each area with the diaphragm of the stethoscope to listen for high-pitched sounds: S_1, S_2, murmurs, pericardial friction rubs.
   b. Next, auscultate each area with the bell of the stethoscope to listen for lower-pitched sounds: S_3, S_4, murmurs.
   c. Listen for the effect of respirations on each sound; while the person is sitting up and leaning forward, ask the person to exhale and hold the breath while you listen to heart sounds.

Technique/normal findings	Abnormal findings
	■ Fixed splitting occurs when right ventricular output is greater than left ventricular output and pulmonary valve closure is delayed (e.g. with atrial septal defect and right ventricular failure). ■ Paradoxical splitting occurs when closure of the aortic valve is delayed (e.g. left bundle branch block).
Identify extra heart sounds in systole. *Extra heart sounds are not present in systole.*	■ Ejection sounds (or clicks) result from the opening of deformed semilunar valves (e.g. aortic and pulmonary stenosis). ■ A midsystolic click is heard with mitral valve prolapse (MVP).
Identify the presence of extra heart sounds in diastole. *Extra heart sounds are not present in diastole.*	■ An opening snap results from the opening sound of a stenotic mitral valve. ■ A pathological S_3 (a third heart sound that immediately follows S_2, called a ventricular gallop) results from myocardial failure and ventricular volume overload (e.g. CHF, mitral or tricuspid regurgitation). ■ An S_4 (a fourth heart sound that immediately precedes S_1, called an atrial gallop) results from increased resistance to ventricular filling after atrial contraction (e.g. HTN, CAD, aortic stenosis and cardiomyopathy). ■ A combined S_3 and S_4 is called a summation gallop and occurs with severe CHF.
Identify extra heart sounds in both systole and diastole. *No extra heart sounds should be heard during systole and diastole.*	■ A pericardial friction rub results from inflammation of the pericardial sac, as with pericarditis.
Murmur assessment	
Identify any murmurs. Note location, timing, presence during systole or diastole, and intensity. Use the following scale to grade murmurs: I = Barely heard II = Quietly heard III = Clearly heard IV = Loud V = Very loud VI = Loudest; may be heard with stethoscope off the chest. A thrill may accompany murmurs of grade IV to grade VI. Note pitch (low, medium, high) and quality (harsh, blowing or musical). Note pattern/shape, crescendo, decrescendo and radiation/transmission (to axilla, neck). *No murmurs should be heard.*	■ Midsystolic murmurs are heard with semilunar valve disease (e.g. aortic and pulmonary stenosis) and with hypertrophic cardiomyopathy. ■ Pansystolic (holosystolic) murmurs are heard with AV valve disease (e.g. mitral and tricuspid regurgitation, ventricular septal defect). ■ A late systolic murmur is heard with MVP. ■ Early diastolic murmurs occur with regurgitant flow across incompetent semilunar valves (e.g. aortic regurgitation). ■ Mid-diastolic and presystolic murmurs, such as with mitral stenosis, occur with turbulent flow across the AV valves. ■ Continuous murmurs throughout systole and all or part of diastole occur with patent ductus arteriosus.
Blood pressure and pulse pressure assessment	
See Box 28.4 for blood pressure measurement guidelines.	
Auscultate blood pressure in each arm with the person seated. *The normal blood pressure is considered to be < 120/< 80, with readings of 120–139/80–89 diagnosed as prehypertension.*	■ Consistent BP readings over 140/90 in adults under age 40 are considered hypertension. ■ BP under 90/60 is considered hypotension. ■ An auscultatory gap—a temporary disappearance of sound between the systolic and diastolic BP—may be a normal variation or it may be associated with systolic HTN or a drop in diastolic BP due to aortic stenosis. ■ **Korotkoff's sounds** (see Box 28.4) may be heard down to zero with cardiac valve replacements, hyperkinetic states, thyrotoxicosis and severe anaemia, as well as after vigorous exercise. ■ The sounds of aortic regurgitation may obscure the diastolic BP. ■ A difference of over 10 mmHg between arms suggests arterial compression on the side of the lower reading, aortic dissection or coarctation of the aorta.

BOX 28.4 Guidelines for blood pressure assessment

Review of Korotkoff's sounds

The first sound heard is the systolic pressure; at least two consecutive sounds should be clear. If the sound disappears and then is heard again 10 to 15 mmHg later, an auscultatory gap is present; this may be a normal variant or it may be associated with hypertension. The first diastolic sound is heard as a muffling of the Korotkoff's sound and is considered the best approximation of the true diastolic pressure. The second diastolic sound is the level at which sounds are no longer heard.

The Heart Foundation of Australia's (2015) guidelines for assessing blood pressure include the following key issues.

Technique reminders

- Choose a cuff of an appropriate size: the cuff should snugly cover two-thirds of the upper arm and the bladder should completely encircle the arm. The bladder should be centred over the brachial artery, with the lower edge 2 to 3 cm above the antecubital space.
- The person's arm should be slightly flexed and supported (on a table or by the examiner) at heart level.
- To determine how high to inflate the cuff, palpate the brachial pulse and inflate the cuff to the point on the manometer at which the pulse is no longer felt; then, add 30 mmHg to this reading and use the sum as the target for inflation. Wait 15 seconds before reinflating the cuff to auscultate the BP.
- To recheck a BP, wait at least 30 seconds before attempting another inflation.
- Always inflate the cuff completely, then deflate it. Once deflation begins, allow it to continue; do not try to reinflate the cuff if the first systolic sound is not heard or if the cuff inadvertently deflates.
- The bell of the stethoscope more effectively transmits the low-pitched sounds of BP.

Sources of error

- Falsely high readings can occur if the cuff is too small or too loose, or if the person supports their own arm.
- Falsely low readings can occur if a standard cuff is used on a person with thin arms.
- Inadequate inflation may result in underestimation of the systolic pressure or overestimation of the diastolic pressure if an auscultatory gap is present.
- Rapid deflation and repeated or slow inflations (causing venous congestion) can lead to underestimation of the systolic BP and overestimation of the diastolic BP.

Factors altering blood pressure

- A change from the horizontal to upright position causes a slight decrease (5 to 10 mmHg) in systolic BP; the diastolic BP remains unchanged or rises slightly.
- BP taken in the arm is lower when the person is standing.
- If the BP is taken with the person in the lateral recumbent position, a lower BP reading may be obtained in both arms; this is especially apparent in the right arm with the person in the left lateral position.
- Factors that increase BP include exercise, caffeine, cold environment, eating a large meal, painful stimuli and emotions.
- Factors that lower BP include sleep (by 20 mmHg) and very fast, slow or irregular heart rates.
- BP tends to be higher in people who are taller or heavier.

Alternative methods of blood pressure measurement

- The palpatory method may be necessary if severe hypotension is present and the BP is inaudible. Palpate the brachial pulse and inflate the cuff 30 mmHg above the point where the pulse disappears; deflate the cuff and note the point on the manometer where the pulse becomes palpable again. Record this as the palpatory systolic BP.
- Leg BP measurement may be needed when there is injury of the arms or to rule out coarctation of the aorta or aortic insufficiency when arm diastolic BP is over 90 mmHg. Place the person in the prone or supine position with the leg slightly flexed. Place a large leg cuff on the thigh with the bladder centred over the popliteal artery. Place the bell of the stethoscope over the popliteal space. Normal leg systolic BP is higher than arm BP; diastolic BP should be equal to or lower than arm BP. Abnormally low leg BP occurs with aortic insufficiency and coarctation of the aorta.

Technique/normal findings	Abnormal findings
Auscultate blood pressure in each arm with the person standing. If orthostatic changes occur, measure the BP with the person supine, legs dangling, and again with the person standing, 1 to 3 minutes apart. *A decrease of systolic BP is expected, but should be < 10 mmHg; diastolic BP should not drop on standing.*	■ A decrease in systolic BP of over 10 to 15 mmHg and a drop in diastolic BP on standing is called **orthostatic hypotension**. Causes include antihypertensive medications, volume depletion, peripheral vascular disease, prolonged bed rest and ageing.

CONSIDERATION FOR PRACTICE

If unable to auscultate blood pressure or palpate pulses, a Doppler ultrasound device may be used to evaluate blood flow. Apply a five-cent-piece amount of gel over the blood vessel to be assessed and lightly place the probe over the gel. Listen for a whooshing (artery) or rushing (vein) sound.

Technique/normal findings	Abnormal findings
Observe the pulse pressure. The **pulse pressure** is the difference between the systolic and diastolic BP. For example, if the BP is 140/80, the pulse pressure is 60. *A normal pulse pressure is one-third the systolic measurement.*	■ A widened pulse pressure with an elevated systolic BP occurs with exercise, arteriosclerosis, severe anaemia, thyrotoxicosis and increased intracranial pressure. ■ A narrowed pulse pressure with a decreased systolic BP occurs with shock, cardiac failure and pulmonary embolus.
Skin assessment	
Inspect the colour of the skin. *The skin colour should be appropriate to the person's age and race.*	■ Pallor reflects constriction of peripheral blood flow (e.g. due to syncope or shock) or decreased circulating oxyhaemoglobin (e.g. due to haemorrhage or anaemia). ■ Central cyanosis of the lips, earlobes, oral mucosa and tongue suggests chronic cardiopulmonary disease. (See Box 28.5 for abnormal findings associated with peripheral vascular and lymphatic assessment.)
Inspect the skin of the extremities and over the regional lymph nodes, noting any oedema, erythema, red streaks or skin lesions. *There should be no oedema, redness or lesions over the regional lymph nodes.*	■ Lymphangitis (inflammation of a lymphatic vessel) may produce a red streak with induration (hardness) following the course of the lymphatic collecting duct; infected skin lesions may be present, particularly between the digits. ■ **Lymphoedema** (swelling due to lymphatic obstruction) occurs with congenital lymphatic anomaly (Milroy's disease) or with trauma to the regional lymphatic ducts from surgery or metastasis (e.g. arm lymphoedema after radical mastectomy with axillary node removal). ■ Oedema of lymphatic origin is usually not pitting and the skin may be thickened; one example is the taut swelling of the face and body that occurs with myxoedema, associated with hypothyroidism.

BOX 28.5 Abnormal findings associated with peripheral vascular and lymphatic assessment

- *Pallor* is an absence of colour of the skin. The degree of pallor depends on the person's normal skin colour and health status. Dark skin may appear ashen or have a yellowish tinge.
- *Cyanosis* is a bluish discolouration of the skin and mucous membranes in people with light skin. In people with dark skin, cyanosis may be difficult to observe. Inspect the nail beds and conjunctiva.
- *Oedema* is an abnormal accumulation of fluid in the interstitial spaces of body tissues. It is often most apparent in the lower extremities.
- *Varicose veins* are tortuous and dilated veins that have incompetent valves. The saphenous veins of the legs are most commonly affected.
- *Enlarged lymph nodes* result from infection or malignancy.
- *Atrophic changes* are changes in size or activity of body tissues as the result of pathology or injury. Decreased blood flow and oxygenation of the lower extremities often cause atrophic changes of loss of hair, thickened toenails, changes in pigmentation and ulcerations.
- *Gangrene* is the necrosis (or death) of tissue, most often the result of loss of blood supply and infection. Gangrene often begins in the most distal of the tissues of the extremities.
- *Pressure ulcers*, also called decubitus ulcers or bed sores, are the result of ischaemia and hypoxia of tissue following prolonged pressure. These ulcers often are located over bony prominences. If untreated, the tissue changes proceed from red skin to deep, crater-like ulcers.

Artery and vein assessment

Palpate the temporal arteries. *There should be no redness, swelling, nodules or variations in pulse amplitude.*

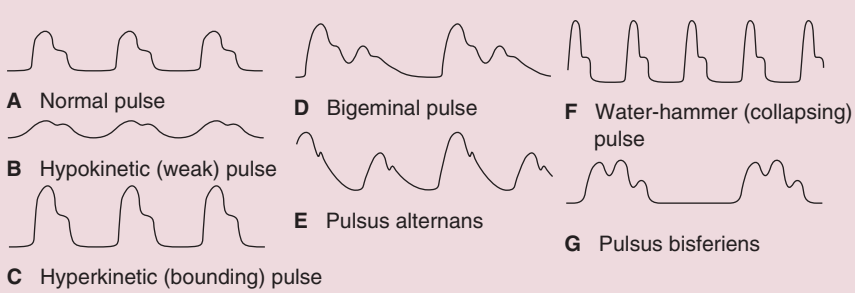
■ Redness, swelling, nodularity and variations in pulse amplitude may occur with temporal arteritis.

Inspect and palpate the carotid arteries. Note symmetry, the pulse rate, rhythm, volume and amplitude. Note any variation with respiration. Describe all pulses as increased, normal, diminished or absent. Scales ranging from 0 to 4+ are sometimes used as follows:

0 = Absent
1+ = Diminished
2+ = Normal
3+ = Increased
4+ = Bounding

Pulse waveforms are showne in Box 28.6.

BOX 28.6 Types of pulse patterns



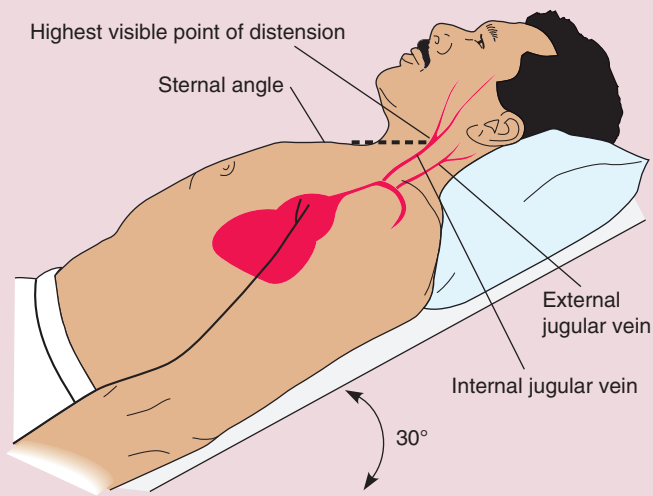
CARDIAC ASSESSMENTS (continued)

Technique/normal findings	Abnormal findings
Remember that carotid pulses should never be assessed simultaneously as this can cause vagal stimulation and potentially result in decreased cerebral perfusion. *The carotid pulses should be bilaterally equal in rate, rhythm, volume and amplitude.*	■ A unilateral pulsating bulge is seen with a tortuous or kinked carotid artery. ■ Alterations in pulse rate or rhythm are due to cardiac arrhythmias. ■ An absent pulse indicates arterial occlusion. ■ A hypokinetic (weak) pulse is associated with decreased stroke volume (see Box 28.6B). This may be due to congestive heart failure (CHF), aortic stenosis or hypovolaemia; to increased peripheral resistance, which may result from cold temperatures; or to arterial narrowing, commonly found with atherosclerosis. ■ A hyperkinetic (bounding) pulse occurs with increased stroke volume and/or decreased peripheral resistance (see Box 28.6C). This may result from states in which cardiac output is high or from aortic regurgitation. It also may occur with anaemia, hyperthyroidism, bradycardia or reduced compliance, as with atherosclerosis. ■ A bigeminal pulse is marked by decreased amplitude of every second beat (see Box 28.6D). This may be due to premature contractions (usually ventricular). ■ Pulsus alternans is a regular pulse with alternating strong and weak beats (see Box 28.6E). This may be due to left ventricular failure and severe HTN.
Auscultate the carotid arteries, using the bell of the stethoscope. *No bruits should be heard.*	■ A murmuring or blowing sound heard over stenosed peripheral vessels is known as a *bruit*. A bruit heard over the middle to upper carotid artery suggests atherosclerosis.
Inspect and palpate the internal and external jugular veins for venous pressure. *See Box 28.7 for guidelines for assessing jugular venous pressure (JVP).*	■ An increase in jugular venous pressure (JVP) over 3 cm and located above the sternal angle reflects increased right atrial pressure. This occurs with right ventricular failure or, less commonly, with constrictive pericarditis, tricuspid stenosis and superior venae cavae obstruction.
If venous pressure is elevated, assess the hepatojugular reflex. (Compress the liver in the right upper abdominal quadrant with the palm of the hand for 30 to 60 seconds while observing the jugular veins.)	■ A decrease in venous pressure reflects reduced left ventricular output or blood volume. ■ Unilateral neck vein distension suggests local compression or anatomic anomaly. ■ A rise in the column of neck vein distension over 1 cm with liver compression indicates right heart failure.
Upper extremity assessment	
Inspect and palpate the arms and hands, noting size and symmetry, skin colour and temperature. *Arms and hands should be symmetrical in size and shape, warm and of appropriate skin colour.*	■ Unilateral swelling with venous prominence occurs with venous obstruction. ■ Extreme localised pallor of the fingers is seen with Raynaud's disease. ■ Cyanosis of the nail beds reflects chronic cardiopulmonary disease. ■ Cold temperature of the hands and fingers occurs with vasoconstriction.
Palpate the nail beds for capillary refill. (Apply pressure to the person's fingertips. Watch for blanching of the nail beds. Release the pressure. Note the time it takes for capillary refill, indicated by the return of pink colour on release of the pressure.) *Capillary refill should be less than 2 seconds (i.e. immediate).*	■ Capillary refill that takes more than 2 seconds reflects circulatory compromise, such as hypovolaemia or anaemia.
Assess venous pattern and pressure. (Elevate one of the person's arms over the head for a few seconds. Slowly lower the arm. Observe the filling of the person's hand veins.) *Hand veins should fill equally and immediately.*	■ Distension of hand veins at elevations over 9 cm above heart level reflects an increase in systemic venous pressure
Palpate the radial and brachial pulses. Note rate, rhythm, volume amplitude, symmetry, variations with respiration. (See Box 28.6 for pulse patterns.) *Radial and brachial pulses should have equal and normal rate, be strong and not vary with respirations.*	■ Alterations in pulse rate or rhythm are due to cardiac arrhythmias (such as atrial fibrillation, atrial flutter and premature ventricular contractions). A pulse rate over 100 bpm is tachycardia; a pulse rate below 60 bpm is bradycardia. ■ A pulse deficit (slower radial rate than apical rate) occurs with arrhythmias and CHF.

BOX 28.7 Assessing jugular venous pressure

When a person with normal venous pressure lies in the supine position, full neck veins are normally visible, but as the head of the bed is elevated, the pulsations disappear. In the person with greatly elevated venous pressure, visible pulsations of the jugular vein are present even in the upright position. To conduct the inspection:

1. Remove clothing from the person's neck and chest. Elevate the head of the bed 30 to 45 degrees (semi-Fowler's position) and turn the person's head to the opposite side. As jugular venous pressure is the measurement of pressure in the right atrium, it is best to examine the right jugular vein. This is because it has a more direct anatomical path to the right atrium.
2. Make sure the neck and the upper thorax are exposed. Avoid neck flexion or hyperextension which may cause the jugular vein to become kinked or stretched, and thus cause compression pulsations.
3. Assess venous pressure by measuring the vertical distance between the angle of St Louis and the highest visible level of pulsation in the internal jugular vein. Using two rulers, line up the bottom edge with the top of the area of pulsation in the jugular vein. Then align another ruler perpendicular to the first at the level of the sternal angle. In centimetres, measure the distance between the second ruler and the sternal angle.
4. Repeat this technique on the other side. Bilateral pressures higher than 2.5 cm are considered elevated and are a sign of right-sided heart failure. One-sided pressure elevation can be a sign of obstruction.
5. If jugular distension is present, assess the JVP by measuring from the highest point of visible distension to the sternal angle (the point at which the clavicles meet) on both sides of the neck (see the accompanying figure). Bilateral measurements above 3 cm are considered elevated and indicate increased venous pressure; distension on only one side may indicate obstruction



Assessment of the highest point of jugular vein distension

Technique/normal findings	Abnormal findings
Colour should return within 3 to 5 seconds in both the ulnar and the radial arteries. See Box 28.8 for the Allen test.	■ Irregularities of rhythm produce early beats and pauses (skipped beats) in the pulse, which may be regular in pattern, sporadic or grossly irregular. ■ Diminished or absent radial pulses may be due to thromboangiitis obliterans (Buerger's disease) or acute arterial occlusion. ■ A weak and thready pulse, often with tachycardia, reflects decreased cardiac output. ■ A bounding pulse occurs with hyperkinetic states and atherosclerosis. ■ Unequal pulses between extremities suggest arterial narrowing or obstruction on one side. ■ In sinus arrhythmia (a normal variant, especially in young adults), the pulse rate increases with inspiration and decreases with expiration. ■ The normal ulnar artery may or may not have a palpable pulse. ■ Persistent pallor with the Allen test suggests ulnar artery occlusion.

BOX 28.8 Allen test

If arterial insufficiency is suspected, palpate the ulnar pulse and perform the Allen test:

- Have the person make a tight fist.
- Compress both the radial and ulnar arteries.
- Have the person open the hand to a slightly flexed position.
- Observe for pallor and manifestations of pain.
- Release the ulnar artery and observe for the return of pink colour within 3 to 5 seconds.
- Repeat the procedure on the radial artery.

Technique/normal findings	Abnormal findings
Lower extremity assessment	
Inspect and palpate each leg, noting size, shape and symmetry; arterial pattern; skin colour, temperature and texture; hair pattern; pigmentation; rashes; ulcers, sensation; and capillary refill. *Legs should be symmetrical in size and shape, arterial pattern, appropriate colour, warm, without lesions. Capillary refill on toenails should be immediate.*	■ Chronic arterial insufficiency may be due to arteriosclerosis or autonomic dysfunction, or to acute occlusion resulting from thrombosis, embolus or aneurysm. ■ Signs of arterial disruption include pallor, dependent rubor (dusky redness); cool to cold temperature; and atrophic changes, such as hair loss with shiny and smooth texture, thickened nails, sensory loss, slow capillary refill and muscle atrophy. ■ Ulcers with symmetric margins, a deep base, black or necrotic tissue, and absence of bleeding may occur at pressure points on or between the toes, on the heel, on the lateral malleolar or tibial area, over the metatarsal heads or along the side or sole of the foot. ■ Gangrene due to complete arterial occlusion presents as black, dry, hard skin; pregangrenous colour changes include deep cyanosis and purple-black discolouration.
With the person supine, assess the venous pattern of the legs. Repeat with the person standing. *Venous pattern on both legs should be symmetrical and there should be no oedema, cyanosis or lesions.*	■ Signs of venous insufficiency include swelling, thickened skin, cyanosis, stasis dermatitis (brown pigmentation, erythema and scaling) and superficial ankle ulcers located predominantly at the medial malleolus with uneven margins, ruddy granulation tissue and bleeding. ■ Varicose veins appear as dilated, tortuous and thickened veins, which are more prominent in a dependent position.
Palpate the femoral, popliteal, posterior tibial and dorsalis pedis pulses for volume, amplitude and symmetry (see Figure 28.23). *All lower extremity pulses should be strong and equal in amplitude.*	■ Diminished or absent leg pulses suggest partial or complete arterial occlusion of the proximal vessel and are often due to arteriosclerosis obliterans. ■ Increased and widened femoral and popliteal pulsations suggest aneurysm. ■ Absence of a posterior tibial pulse with signs and symptoms of arterial insufficiency is usually due to acute occlusion by thrombosis or embolus. ■ Diminished or absent pedal pulses are often due to popliteal occlusion associated with diabetes mellitus.
If pulses are diminished, observe for postural colour changes. Elevate both legs 60 degrees and observe the colour of the soles of the feet. Have the person sit and dangle the legs; note the return of colour to the feet.	■ Extensive pallor on elevation is suggestive of arterial insufficiency. ■ Rubor (dusky redness) of the toes and feet along with delayed venous return (over 45 seconds) suggests arterial insufficiency.
If arterial insufficiency is suspected, auscultate the femoral arteries. *No bruits should be heard.*	■ Femoral bruits suggest arterial narrowing due to arteriosclerosis.
Inspect and gently palpate the calves. *There should be no redness or swelling, heat or pain in the calves of the legs.*	■ Redness, warmth, swelling, tenderness and cords along a superficial vein suggest thrombophlebitis or deep venous thrombosis.

Inspect and palpate for oedema. Use your thumb to compress the dorsum of the person's foot, around the ankles and along the tibia (see Figure 28.26A). *A depression in the skin that does not immediately refill is called pitting oedema. Normally, there is no oedema.*	■ Oedema can be graded on a scale from 1+ to 4+ (Figure 28.26B):
	  ■ 1+ (–2 mm depression)—No visible change in the leg; slight pitting
	  ■ 2+ (–4 mm depression)—No marked change in the shape of the leg; pitting slightly deeper
	  ■ 3+ (–6 mm depression)—Leg visibly swollen; pitting deep
	  ■ 4+ (–8 mm depression)—Leg very swollen; pitting very deep
	■ Oedema may be caused by disease of the cardiovascular system such as CHF; by renal, hepatic or lymphatic problems; or by infection.
	■ Venous distension suggests venous insufficiency or incompetence.

Technique/normal findings **Abnormal findings**

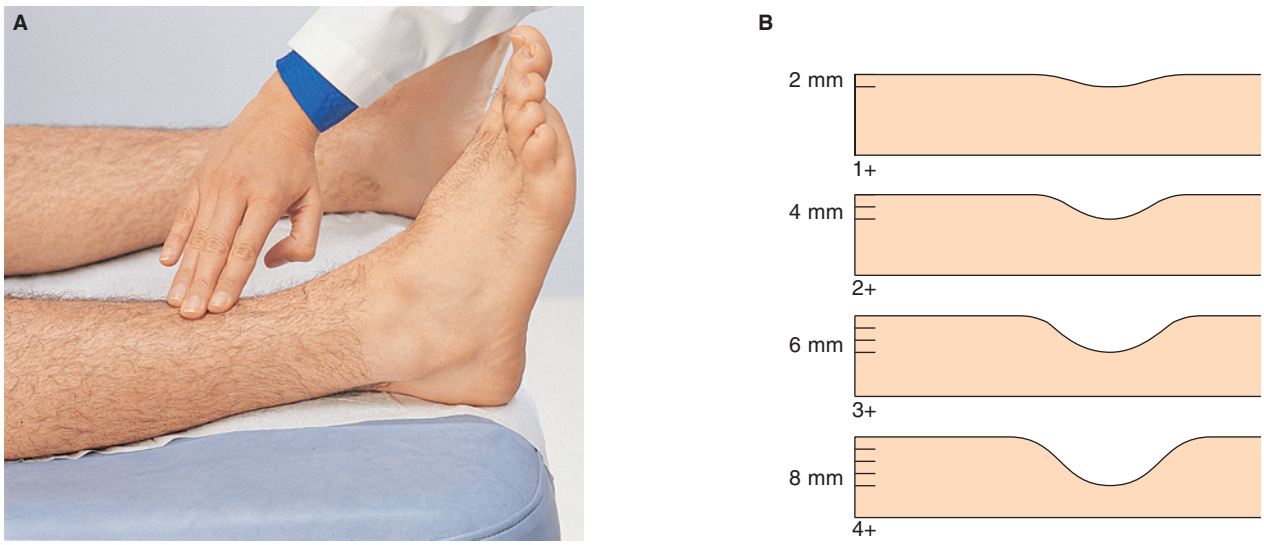


FIGURE 28.26 ■ Evaluation of oedema. *A*, Palpating for oedema over the tibia. *B*, Four-point scale for grading oedema

Abdominal assessment

Inspect and palpate the abdominal aorta. Note size, width and any visible pulsations or bulging. *Abdominal aorta should be of appropriate size without visible pulsations or bulging.*

- A pulsating mass in the upper abdomen suggests an aortic aneurysm, particularly in the older adult.
- An aorta greater than 2.5 to 3 cm in width reflects pathological dilation, most likely due to arteriosclerosis.

Auscultate the epigastrium and each abdominal quadrant, using the bell of the stethoscope (see Figure 28.27). *No bruits should be heard over the abdominal aorta.*

- Abdominal bruits reflect turbulent blood flow associated with partial arterial occlusion.
- A bruit heard over the aorta suggests an aneurysm.
- A bruit heard over the epigastrium and radiating laterally, especially with HTN, suggests renal artery stenosis.
- Bruits heard in the lower abdominal quadrants suggest partial occlusion of the iliac arteries.

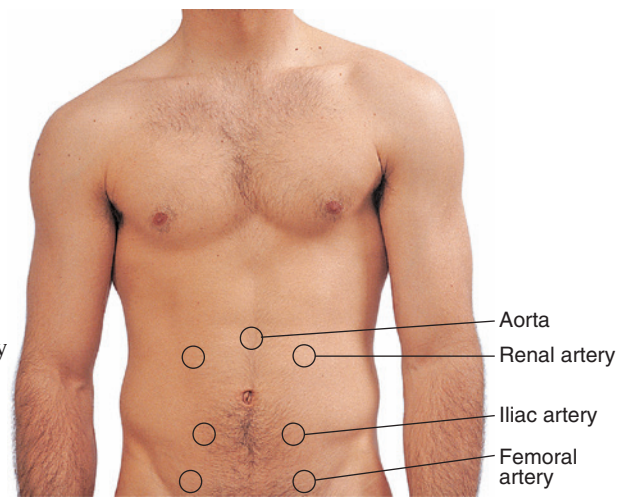


FIGURE 28.27 ■ Auscultation sites of the abdominal aorta and its branches

Lymph node assessment

Palpate the regional lymph nodes of the head and neck, axillae, arms and groin. Use firm, circular movements of the finger pads and note size, shape, symmetry, consistency, delineation, mobility, tenderness, sensation and condition of overlying skin. *Nodes should not be enlarged or painful.*

- **Lymphadenopathy** refers to the enlargement of lymph nodes (over 1 cm) with or without tenderness. It may be caused by inflammation, infection or malignancy of the nodes or the regions drained by the nodes.
- Lymph node enlargement with tenderness suggests inflammation (*lymphadenitis*). With bacterial infection, the nodes may be warm and matted with localised swelling.
- Malignant or metastatic nodes may be hard, indicating lymphoma; rubbery, indicating Hodgkin's disease; or fixed to adjacent structures. Usually they are not tender.
- Ear infections and scalp and facial lesions, such as acne, may cause enlargement of the pre-auricular and cervical nodes.

CARDIAC ASSESSMENTS (continued)

Technique/normal findings	Abnormal findings
	■ Anterior cervical nodes are enlarged and infected with streptococcal pharyngitis and mononucleosis. ■ Lymphadenitis of the cervical and submandibular nodes occurs with herpes simplex lesions. ■ Enlargement of supraclavicular nodes, especially the left, is highly suggestive of metastatic disease from abdominal and thoracic cancer. ■ Axillary lymphadenopathy is associated with breast cancer. ■ Lesions of the genitals may produce enlargement of the inguinal nodes. ■ Persistent generalised lymphadenopathy is associated with acquired immune deficiency syndrome (AIDS) and AIDS-related complex.
Spleen assessment	
Palpate for the spleen, in the upper left quadrant of the abdomen. *The spleen is normally not palpable.*	■ A palpable spleen in the left upper abdominal quadrant of an adult may indicate abnormal enlargement (splenomegaly) and may be associated with cancer, blood dyscrasias and viral infection, such as mononucleosis.
Percuss for splenic dullness in the lowest left intercostal space (ICS) at the anterior axillary line or in the ninth to tenth ICS at the midaxillary line (see Figure 28.28). *Normally, tympany is heard.*	■ A dull percussion note in the lowest left ICS at the anterior axillary line or below the tenth rib at the midaxillary line suggests splenic enlargement.

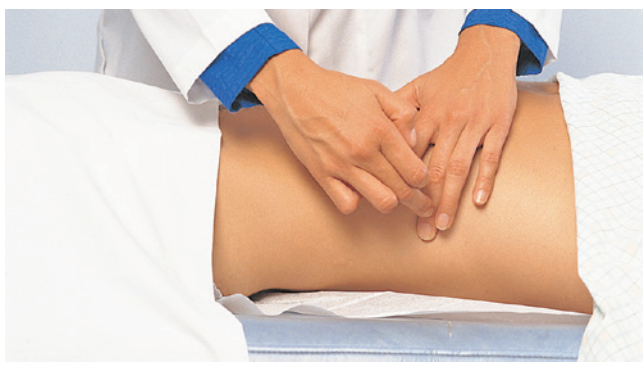


FIGURE 28.28 ■ Percussing the spleen

TRANSLATION TO PRACTICE

Evidence-based practice: core components of cardiovascular disease secondary prevention and cardiac rehabilitation 2014

Appropriate assessment and short-term monitoring are pivotal components to decrease morbidity and mortality rates of individuals experiencing cardiovascular disease. An article by Woodruffe et al. (2015) reported on the convening of a nationally represented, expert panel to review research evidence to guide practice in cardiovascular health care. Five core components were identified and recommended as necessary for secondary prevention and cardiac rehabilitation:

1. equity and access to services
2. assessment and short-term monitoring
3. recovery and longer-term maintenance
4. lifestyle/behavioural modification and medication adherence
5. evaluation and quality improvement (Woodruffe et al., 2015).

IMPLICATIONS FOR NURSING

This article highlights evidence-based recommendations essential for improving healthcare provision for users of cardiovascular health services. The core components are explained and advice regarding data collection, key performance indicators and methods to promote change are described. Specific considerations warranting attention are also emphasised for Aboriginal and Torres Strait Islander peoples.

CRITICAL THINKING IN PERSON-CENTRED CARE

1 How does appropriate, individualised assessment of people with cardiovascular disease decrease mortality and morbidity?

2 Consider all the data that should be collected when assessing an individual with cardiovascular disease. What assessment data are critical and what other data may influence outcomes for individuals experiencing cardiovascular disease?

3 How could information in this review improve your practice when caring for individuals experiencing cardiovascular disease?

CONCEPT CHECK

1 The amount of blood pumped by the ventricles in 1 minute is known as:
1 heart rate
2 ventricular contraction
3 stroke volume
4 cardiac output

2 During what part of the cardiac cycle is the myocardium perfused?
1 prior to atrial filling
2 prior to ventricular relaxation
3 during diastole
4 during pulmonary perfusion

3 What physiological process is responsible for the electrical impulse that stimulates myocardial contraction?
1 action potential
2 cardiac reserve
3 cardiac potential
4 ventricular contraction

4 The intensity of chest pain may be assessed by asking which question?
1 'Did the pain move into your left arm?'
2 'Was your pain relieved by resting or worse when you were busy?'
3 'On a scale of 0 (no pain) to 10 (worst pain), what number was your pain?'
4 'Was the pain a pressure, a burning or a tightness?'

5 At what anatomical location would you assess the apical impulse?
1 left midclavicular, fifth intercostal space
2 left substernal, sixth intercostal space
3 right midaxillary, second intercostal space
4 right nipple line, any intercostal space

6 A person's heart rate is 50. You would document this as:
1 tachycardia
2 bradycardia
3 hypertension
4 hypotension

7 A person has a very low RBC count. What subjective manifestation would you expect to find during a health history?
1 sore throat
2 chest pain
3 fatigue
4 nausea

8 A person has a low platelet count. What would you likely find on physical assessment?
1 enlarged lymph nodes
2 excessive bruising
3 varicose veins
4 changes in pulse pressure

9 An older person is severely dehydrated and, as a result, has increased blood viscosity. How will this affect the peripheral vascular resistance (PVR)?
1 increased PVR
2 decreased PVR
3 no change
4 depends on gender

10 When auscultating the abdominal aorta, you hear a murmuring or blowing sound. You would document this sound as a:
1 hypokinetic pulse
2 bigeminal pulse
3 bruit
4 arrhythmia

11 Swelling of a body part as a result of lymphatic obstruction is labelled:
1 lymphoedema
2 lymphadenopathy
3 atrophic change
4 central cyanosis

12 You are assessing a man who has severe leg pain. The leg is cool and cyanotic. You are unable to palpate a femoral pulse. What would be your priority intervention based on these assessments?
1 Document your findings.
2 Ask the family about this problem.
3 Teach the man relaxation techniques.
4 Notify the medical officer immediately.

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CHAPTER 29

NURSING CARE OF PEOPLE WITH CORONARY HEART DISEASE

MAJELLA HALES

LEARNING OUTCOMES

- Discuss how alterations to myocardial perfusion contribute to the development of a person's heart disease.
- Relate interprofessional care principles for a person with angina to the course and manifestations of the condition.
- Summarise the course, management and outcomes when caring for a person with acute coronary syndrome.
- Compare and contrast the potential complications for a person experiencing an acute myocardial infarction.
- Differentiate between various cardiac arrhythmia in the context of influence on lifestyle and management principles.
- Discuss the interprofessional care and implications for a person with sudden cardiac death.

CLINICAL COMPETENCIES

- Assess functional health status of the person with coronary heart disease and/or an arrhythmia, including the impact of the disorder on the person's ability to perform activities of daily living and usual tasks.
- Apply knowledge of the normal anatomy and physiology of the heart to improve care of the person with coronary heart disease.
- Monitor the person with coronary heart disease or arrhythmias for expected and unexpected manifestations, reporting and recording findings as indicated.
- Interpret assessment data, determine priorities of care and develop and implement individualised nursing interventions for the person with coronary heart disease and arrhythmias.
- Administer medications and treatments to the person with coronary heart disease and arrhythmias safely and knowledgeably.
- Integrate multidisciplinary care into nursing care planning and implementation for the person with coronary heart disease and arrhythmias.
- Provide appropriate teaching for prevention, health promotion and self-care related to coronary heart disease and arrhythmias.
- Evaluate the effectiveness of nursing interventions, revising or modifying the plan of care as needed to promote, maintain or restore functional health for the person with coronary heart disease or arrhythmias.

KEY TERMS

Irrespective of the underlying cause, impaired cardiac function affects the person's ability to participate in exercise and activities of daily living. When the functioning of other systems becomes affected by problems such as impaired blood flow to the myocardium, changes in the conduction of electrical impulses through the heart or structural changes in the heart itself, it becomes unable to pump enough blood to meet the body's demand for oxygen and nutrients. Death may result.

Cardiovascular disease (CVD) is a generic term for disorders of the heart and blood vessels. CVD is the leading cause of death and disability in many developed countries and affects over 3.7 million Australians. In Australia, CVD kills one person every 12 minutes and represented 26% of all deaths in 2010. Nationally, almost $7.7 billion is spent on CVD annually, representing 10.4% of the total allocated health expenditure (Australian Institute of Health and Welfare (AIHW), 2014). Indigenous Australians are twice as likely to die from CVD (AIHW, 2014).

Coronary heart disease (CHD) refers more specifically to an ischaemic pathology related to disease of the blood vessels, causing myocardial oxygenation issues. Angina and myocardial infarction are the two main clinical forms of this disease. Cardiovascular disease is a National Health Priority and through increased funding, research and public education campaigns, deaths from CHD have reduced by 73% since 1980 (AIHW, 2014). Although obesity is still a major concern, education involving the reduction of fat intake, increasing exercise and lowering cholesterol levels have made Australians more aware of risk factors associated with CHD.

This chapter focuses on disorders of myocardial blood flow (coronary heart disease) and cardiac rhythm. Disorders of cardiac structure and function are discussed in Chapter 30. Review the normal anatomy and physiology and nursing assessment of the heart in Chapter 28 before proceeding with this chapter.

FAST FACTS

- Heart disease accounts for over 21 500 deaths annually in Australia.
- 15% of Australians aged over 70 have CHD.
- Aboriginal and Torres Strait Islander people are twice as likely as non-Indigenous Australians to experience CHD.

Source: AIHW (2014).

DISORDERS OF MYOCARDIAL PERFUSION

THE PERSON WITH CORONARY HEART DISEASE

Coronary heart disease or *coronary artery disease (CAD)* is caused by impaired blood flow to the myocardium. Accumulation of atherosclerotic plaque in the coronary arteries is the usual cause. Coronary heart disease may be asymptomatic or may lead to angina pectoris, acute coronary syndrome, myocardial infarction (MI), arrhythmias, heart failure and even sudden death.

Incidence and prevalence

Many risk factors for CHD can be controlled through lifestyle modification. In fact, with increased public awareness of risk factors related to CHD, mortality rates have declined by over 40% since the 1970s. Nevertheless, CHD remains a major public health problem. Nurses are in a prime position to encourage and support positive lifestyle changes by teaching and promoting healthy living practices.

Both men and women are affected by CHD in women, however, the onset is about 10 years later because of the heart-protective effects of oestrogen. After menopause, women's risk is equal to that of men.

Physiology review

The two main coronary arteries, the left and the right, supply blood, oxygen and nutrients to the myocardium. They originate in the root of the aorta, just outside the aortic valve. The *left main coronary artery* divides to form the anterior descending and circumflex arteries. The *anterior descending* artery supplies the anterior interventricular septum and the left ventricle, including the apex of the heart. The *circumflex* branch supplies the lateral wall of the left ventricle. The *right coronary artery* supplies the right ventricle and forms the posterior descending artery. The *posterior descending artery* supplies the posterior portion of the heart (see Figure 28.7).

Blood flow through the coronary arteries is regulated by several factors. Aortic pressure is the primary factor. Other factors include the heart rate (most flow occurs during diastole, when the muscle is relaxed), metabolic activity of the heart, blood vessel tone (constriction) and collateral circulation. Although there are no connections between the large coronary arteries, small arteries are joined by **collateral vessels**. If large vessels are gradually occluded, the collateral vessels enlarge, providing alternative routes for blood flow.

Pathophysiology

Coronary atherosclerosis is the most common cause of reduced coronary blood flow.

Atherosclerosis

Atherosclerosis is a progressive disease characterised by *atheroma* (plaque) formation, which affects the intimal and medial layers of large and midsize arteries. See 'Pathophysiology illustrated: coronary heart disease' below.

Atherosclerosis is initiated by unknown precipitating factors that cause lipoproteins and fibrous tissue to accumulate in the arterial wall. Although the precise mechanisms are

unknown, endothelial dysfunction, abnormal lipid metabolism and injury to or inflammation of endothelial cells lining the artery appear to be key to its development.

Endothelial dysfunction is mediated by various factors including endothelial tone and vascular remodelling. An imbalance of endothelial-derived relaxing factors (EDRFs) and endothelial-derived constricting factors (EDCFs) results in augmented constriction intake (Bleakley et al., 2015). This continued workload results in arterial hypertrophy within the media and perpetuates local hypertension, increasing shear stress and contributing to further endothelial damage and remodelling.

In the bloodstream, lipids are transported attached to proteins called apoproteins. High levels of certain *lipoproteins*, a type of apoprotein, increase the risk of atherosclerosis. *Low-density lipoproteins*, which are high in cholesterol, carry cholesterol to peripheral tissues where some of it is released to be taken up and incorporated into cells for use in producing energy. *Very-low-density lipoproteins*, large molecules primarily composed of triglycerides and cholesterol, carry triglycerides to muscle and fat cells. When the triglycerides are released into these tissues, the remainder of the molecule is a low-density lipoprotein. *High-density lipoproteins*, in contrast, attract cholesterol, returning it from peripheral tissues to the liver (Bullock & Hales, 2012).

Hyperlipidaemia itself may damage arterial endothelium. Endothelial damage promotes platelet adhesion and aggregation and attracts leucocytes to the area. At the site of injury, *atherogenic* (atherosclerosis-promoting) lipoproteins collect in the intimal lining of the artery. These lipoproteins appear to actually bind with the extracellular portion of the vessel endothelium. Macrophages migrate to the injured site as part of the inflammatory process. Contact with platelets, cholesterol and other blood components stimulates smooth muscle cells and connective tissue within the vessel wall to proliferate abnormally. Although blood flow is not affected at this stage, this early lesion appears as a yellowish fatty streak on the inner lining of the artery. Fibrous plaque develops as smooth muscle cells enlarge, collagen fibres proliferate and blood lipids accumulate. The lesion protrudes into the arterial lumen and is fixed to the inner wall of the intima. It may invade the muscular media layer of the vessel as well. The developing plaque not only gradually occludes the vessel lumen but also impairs the vessel's ability to dilate in response to increased oxygen demands. Fibrous plaque lesions often develop at arterial bifurcations or curves or in areas of narrowing. As the plaque expands, it can produce severe stenosis or total occlusion of the artery.

The final stage of the process is the development of *atheromas*, complex lesions consisting of lipids, fibrous tissue, collagen, calcium, cellular debris and capillaries. These calcified lesions can ulcerate or rupture, stimulating thrombosis. The vessel lumen may be rapidly occluded by the thrombus or it may embolise to occlude a distal vessel.

Plaque formation may be *eccentric*, located in a specific, asymmetric region of the vessel wall, or *concentric*, involving the entire vessel circumference. Manifestations of the process usually do not appear until about 75% of the arterial lumen has been occluded.

Atherosclerosis tends to develop where arteries bifurcate or branch which encourages atheroma-prone conditions such as high shear forces, less ordered endothelial cell alignment and inferior shear-resistant endothelial cell morphology, ultimately promoting a pro-inflammatory microenvironment (Tabas, García-Cardeña & Owens, 2015). Certain vessels have a higher likelihood of being affected, including the coronary arteries (the left anterior descending artery, in particular), the renal arteries, the bifurcation of the carotid arteries and branching sections of peripheral arteries. In addition to obstructing or occluding blood flow, atherosclerosis weakens arterial walls and is a major cause of aneurysm in vessels such as the aorta and iliac arteries.

Myocardial ischaemia

Myocardial cells become ischaemic when the oxygen supply is inadequate to meet metabolic demands. The critical factors in meeting metabolic demands of cardiac cells are coronary perfusion and myocardial workload. Coronary perfusion can be affected by several different mechanisms:

- One or more vessels may be partially occluded by large, stable areas of plaque.
- Platelets can aggregate in narrowed vessels, forming a thrombus.
- Normal or already narrowed vessels may spasm.
- A drop in blood pressure may lead to inadequate flow through coronary vessels.
- Normal autoregulatory mechanisms that increase flow to working muscles may fail.

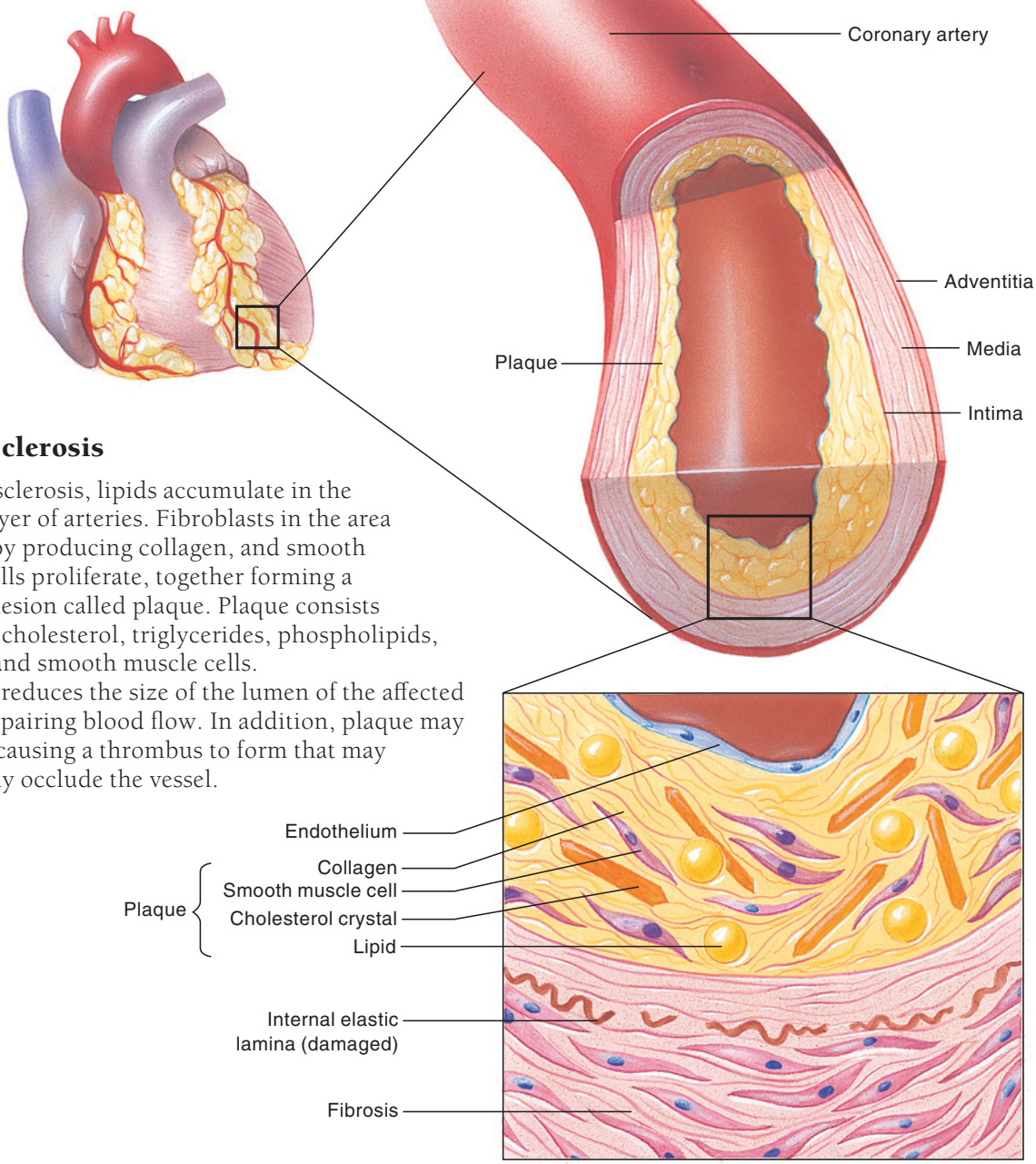
Workload is affected by the heart rate, myocardial contractility, preload (the amount of blood in the ventricles just prior to systole) and afterload (the peripheral pressure that must be overcome to move blood out of the heart into the circulation). The oxygen content of the blood and haematocrit are contributing factors to myocardial ischaemia. Table 29.1 lists factors that may lead to myocardial ischaemia.

TABLE 29.1 Factors contributing to myocardial ischaemia

CORONARY PERFUSION	MYOCARDIAL WORKLOAD	BLOOD OXYGEN CONTENT
• Atherosclerosis • Thrombosis • Vasospasm • Poor perfusion pressure	• Rapid heart rate • Increased preload, afterload or contractility • Increased metabolic demands (e.g. hyperthyroidism)	• Reduced atmospheric oxygen pressure • Impaired gas exchange • Low red blood cells and haemoglobin content

Coronary heart disease

Coronary heart disease usually is due to *atherosclerosis*, occlusion of the coronary arteries by fibrous, fatty plaque. Coronary heart disease is manifested by *angina pectoris, acute coronary syndrome* and/or *myocardial infarction*. Risk factors for coronary heart disease include age (over 50 years), heredity, smoking, obesity, high serum cholesterol levels, hypertension and diabetes mellitus. Other factors, such as diet and lack of exercise, also contribute to the risk of CHD.



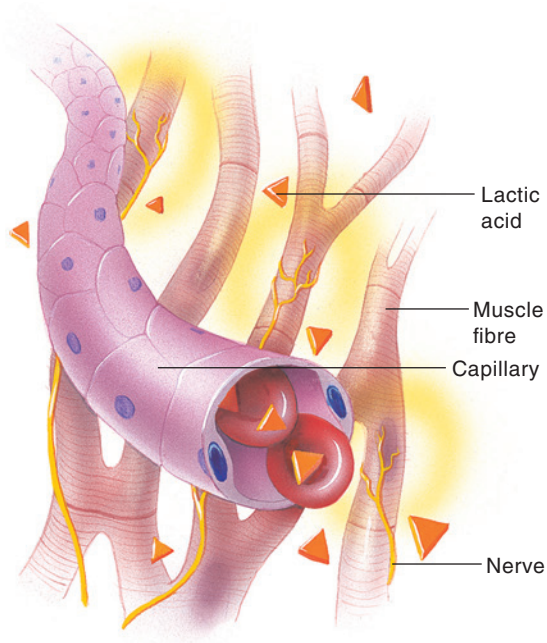
Atherosclerosis

In atherosclerosis, lipids accumulate in the intimal layer of arteries. Fibroblasts in the area respond by producing collagen, and smooth muscle cells proliferate, together forming a complex lesion called plaque. Plaque consists mostly of cholesterol, triglycerides, phospholipids, collagen and smooth muscle cells.

Plaque reduces the size of the lumen of the affected artery, impairing blood flow. In addition, plaque may ulcerate, causing a thrombus to form that may completely occlude the vessel.

Angina pectoris

Angina is characterised by episodes of chest pain, usually precipitated by exercise and relieved by rest. When myocardial oxygen needs are greater than partially occluded vessels can supply, myocardial cells become ischaemic and shift to anaerobic metabolism. Anaerobic metabolism produces lactic acid that stimulates nerve endings in the muscle, causing pain. The pain subsides when the oxygen supply again meets myocardial demand.



Myocardial infarction

Myocardial infarction occurs when complete obstruction of a coronary artery interrupts blood supply to an area of myocardium. Affected tissue becomes ischaemic and eventually dies (infarcts) if the blood supply is not restored. The necrotic area is bordered by an area of injured or damaged tissue, which is in turn surrounded by an area of ischaemic tissue.

As myocardial cells die, they lyse and release various cardiac isoenzymes into the circulation. Elevated serum levels of creatine kinase (CK) and cardiac-specific troponins are specific indicators of myocardial infarction.

Myocardial cells have limited supplies of adenosine triphosphate (ATP) for energy storage. When myocardial workload increases or the supply of blood and oxygen falls, cellular ATP stores are quickly depleted, affecting their contractility. Cellular metabolism switches from an efficient aerobic process to anaerobic metabolism. Lactic acid accumulates and cells are damaged. If blood flow is restored within 20 minutes, aerobic metabolism and contractility are restored and cellular repair begins (Bullock & Hales, 2012). Continued ischaemia results in cell necrosis and death (infarction).

Coronary heart disease is generally divided into two categories: chronic ischaemic heart disease and acute coronary syndromes. *Chronic ischaemic heart disease* includes stable and unstable angina pectoris and silent myocardial ischaemia. In women, angina is the most common presenting symptom of CHD. *Acute coronary syndromes* range from unstable angina to myocardial infarction (Coven, 2015). Acute coronary syndromes and myocardial infarction are the most common presenting symptoms of CHD in men. These disorders are discussed in the following sections of this chapter.

Risk factors

The causes of atherosclerosis are not known, but certain risk factors have been linked with the development of atherosclerotic plaques. The Framingham Heart Study (FHS) provided vital research into the relationship between risk factors and the development of heart disease (see the 'Translation to practice' box below). Research into CHD is ongoing, looking at causative factors, manifestations and protective measures for many populations. Risk factors for CHD are frequently classified as *non-modifiable*, factors that cannot be changed, and *modifiable*, those factors that can be changed (see Table 29.2).

Non-modifiable risk factors

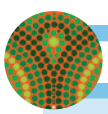
Age is a non-modifiable risk factor. In Australia, approximately 6.7% of all male deaths and 2.7% of all female deaths attributable to CHD occur in individuals under 55. By contrast, over 68% of all male deaths and 87% of all female deaths can be attributable to CHD in individuals 75 years or older (Nichols et al., 2014). *Genetic factors* and *gender* also are non-modifiable risk factors for CHD. A family history of CHD is considered a strong risk factor of the development of coronary artery disease. Men are affected by CHD at an earlier age than women, yet women are still at risk of CHD despite the perception otherwise. In a recent study by Cainzos-Achirica and Blaha (2015), a discordance in CHD risk perception existed in over 60% of the female study cohort. Results such as these reinforce the need for better CHD information campaigns encompassing factors such as risk, prevention and identification.

Modifiable risk factors

Modifiable risk factors include lifestyle factors and pathological conditions that predispose the person to developing CHD. Disease conditions that contribute to CHD include hypertension, diabetes mellitus and hyperlipidaemia. Although these conditions are not a matter of choice, they are modifiable risk factors that can often be controlled through medication, weight control, diet and exercise.

Behavioural or lifestyle factors can be controlled or completely eliminated. Lifestyle changes require significant commitment by the person; ongoing support from the healthcare team is vital for success.

HYPERTENSION *Hypertension* is consistent blood pressure readings greater than 140 mmHg systolic or 90 mmHg



TRANSLATION TO PRACTICE Evidence-based practice: Framingham Heart Study

The Framingham Heart Study (FHS) is an ongoing, significant clinical research study that has provided data about cardiovascular disease for over 60 years. The study was initiated in 1948 with an original study group of 5209 participants in the town of Framingham, Massachusetts, in the United States (Framingham Heart Study, 2015). Every 2 years, this original group is evaluated for cardiovascular 'events' via their medical history, physical findings and diagnostic testing. Children and grandchildren of the original group have also been studied as part of the Framingham Offspring Study. It was in reports of the Framingham study that the term 'risk factor' first appeared.

IMPLICATIONS FOR NURSING

Data collected from the Framingham Heart Study and the Framingham Offspring Study provide a rich database from which to develop evidence-based approaches for people with heart disease. A major application of these research findings to practice is in primary preventive education—for example, through community cardiovascular health programs. Although research shows that increased public awareness of cardiovascular risk factors has lowered morbidity and mortality from heart disease, it remains a leading cause of premature death in Australia. For cardiovascular disease to be reduced on a large scale, education about the effects of lifestyle on the cardiovascular system must begin in the early school years and be reinforced throughout the formative years where healthy choices can become habit.

A second application of these findings is in interprofessional treatment. Nurses should keep up to date on the latest strategies for medical treatment so that they can provide accurate rationales to individuals and formulate effective nursing care plans that complement medical management strategies. The result is better communication, a sense of collegiality and teamwork, and positive health outcomes.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. What kinds of strategies can be used in primary school settings to teach cardiovascular health in a fun, informative manner?
2. Which healthcare providers should be included in a multidisciplinary effort to encourage individuals to modify their lifestyles?
3. What lifestyle changes does one need to make to role model healthy living for optimal cardiovascular health?

TABLE 29.2 Risk factors for coronary heart disease

NON-MODIFIABLE	MODIFIABLE	PATHOPHYSIOLOGICAL
Age	Cigarette smoking	Hyperlipidaemia
Men ≥ 45 years	Obesity	Elevated LDL cholesterol
Women ≥ 55 years	Sedentary behaviour	Elevated triglycerides
Gender	Low fruit and vegetable intake	Low HDL cholesterol
Heredity	Excessive alcohol intake	Hypertension
	Women only: use of oral contraceptives, hormone replacement therapy	Diabetes mellitus
		Kidney disease
		Women only: premature menopause
		Emerging risk factors:
		Elevated homocysteine levels
		Thrombogenic factors
		Inflammatory factors
		Impaired fasting glucose

diastolic. Hypertension affects approximately 30% of Australians. Indigenous Australians experience higher rates of hypertension than non-Indigenous Australians. Aboriginal and Torres Strait Islander people develop hypertension earlier (Australian Bureau of Statistics (ABS), 2014). Hypertension damages arterial endothelium. Inflammatory responses stimulate the development of atherosclerotic plaque. Individuals can reduce hypertension through lifestyle changes.

DIABETES *Diabetes mellitus* contributes to CHD in several ways. Diabetes is associated with several risk factors, including hyperlipidaemia, a higher incidence of hypertension and obesity. In addition, diabetes affects vascular endothelium, which contributes to the development of atherosclerosis. Hyperglycaemia and hyperinsulinaemia, altered platelet function, elevated fibrinogen levels and inflammation also are thought to play a role in the development of atherosclerosis in people with diabetes.

ABNORMAL BLOOD LIPIDS *Hyperlipidaemia* is an abnormally high level of blood lipids and lipoproteins. Lipoproteins carry cholesterol in the blood. Low-density lipoproteins (LDLs) are the primary carriers of cholesterol. High levels of LDL promote atherosclerosis because LDLs deposit cholesterol on artery walls. By contrast, high-density lipoproteins (HDLs) help clear cholesterol from the arteries by transporting it to the liver for excretion. HDL levels above 0.4 mmol/L have a protective effect, reducing the risk of CHD; by contrast, HDL levels lower than 0.4 mmol/L are associated with an increased risk of CHD. Triglycerides are compounds of fatty acids bound to glycerol. They are used for fat storage by the body and are carried on very low-density lipoprotein (VLDL) molecules. Elevated triglycerides also contribute to the risk of CHD. Table 29.3 lists the aims for lipid lowering therapy as recommended by National Vascular Disease Prevention Alliance.

CIGARETTE SMOKING *Cigarette smoking* is an independent risk factor for CHD. The effects of nicotine and other toxins from cigarette smoke negatively affect the cardiovascular system. Individuals who smoke are significantly more at risk of developing CHD than non-smokers (Mons et al., 2015). Research suggests that stopping smoking by the age of 30 removes almost all mortality risks associated with smoking (National Drug Strategy (NDS), 2012) Cigarette smoking promotes CHD in several ways. The carbon monoxide damages vascular endothelium, promoting cholesterol deposition. Nicotine stimulates catecholamine release and increases blood pressure, heart rate and myocardial oxygen demand. Nicotine also causes arterial vasoconstriction, which reduces tissue perfusion. Nicotine also reduces HDL levels, increases platelet aggregation and increases the risk of thrombus formation.

TABLE 29.3 Serum cholesterol and triglyceride targets

Total cholesterol	< 4 mmol/L
High-density lipoprotein cholesterol (HDL-C)	≥ 1 mmol/L
Low-density lipoprotein cholesterol (LDL-C)	< 2 mmol/L
Non-high-density lipoprotein cholesterol (Non-HDL-C)	< 2.5 mmol/L
Triglycerides (TG)	< 2 mmol/L

Source: National Vascular Disease Prevention Alliance (NVDPA) (2012).

FAST FACTS

- Cigarette smoking is the leading independent risk factor for coronary heart disease and a primary target of risk factor management.

OBESITY *Obesity* (excess adipose tissue) is generally defined as a body mass index (BMI) of 30 kg/m^2 or greater. Obese people have higher rates of hypertension, diabetes and hyperlipidaemia. In the Framingham Study (FHS, 2015),

obese men over age 50 had twice the incidence of CHD and acute myocardial infarction (MI) of those who were within 10% of their ideal weight. Central obesity, or intra-abdominal fat, is associated with an increased risk of CHD. The best indicator of central obesity is the waist circumference. A waist-to-hip ratio of greater than 0.8 (women) or 0.9 (men) increases the risk of CHD.

PHYSICAL INACTIVITY *Physical inactivity* is associated with a higher risk of CHD. Research data indicate that people who maintain a regular program of physical activity are less prone to developing CHD than sedentary people. Cardiovascular benefits of exercise include increased availability of oxygen to the heart muscle, decreased oxygen demand and cardiac workload, and increased myocardial function and electrical stability. Other positive effects of regular physical activity include decreased blood pressure and blood lipids, and a decrease in insulin resistance, platelet aggregation and weight (Rahmati-Najarkolaei et al., 2015).

DIET *Diet* is a risk factor for CHD, independent of fat and cholesterol intake. Diets high in fruits, vegetables, whole grains and unsaturated fatty acids appear to have a protective effect. The underlying factors are not clear, but probably relate to nutrients such as antioxidants, folic acid, other B vitamins, omega-3 fatty acids and other unidentified micronutrients (Cespedes & Hu, 2015; Reidlinger et al., 2015).

Emerging risk factors

Recent research demonstrates a link between *elevated serum homocysteine levels* and CHD. Until menopause, women have lower homocysteine levels than men, which may partially explain their lower risk of CHD. Increased homocysteine levels negatively correlate with serum folate and dietary folate intake. Increasing folate intake can reduce homocysteine levels. However, currently, studies do not show benefits of folic acid supplementation. As an alternative, a balanced diet including two serves of fruit and five serves of vegetables a day is recommended to provide adequate vitamin and nutrient supply.

Based on evidence that aspirin and antiplatelet therapies reduce the risk of MI, *clot-promoting factors* are identified as CHD risk factors. *Inflammation* has also recently been identified as a risk factor. Inflammatory processes may increase the development of atherosclerotic plaque (Bullock & Hales, 2012). Inflammation also promotes clot formation at the site of ruptured plaque.

Metabolic syndrome

Metabolic syndrome, a group of metabolic risk factors occurring in an individual, is a strong risk factor for CHD (see Box 29.1). Metabolic syndrome has emerged as a risk factor for premature CHD that is equal to cigarette smoking. Three underlying causes of metabolic syndrome have been identified: overweight/obesity, physical inactivity and genetic factors. It is closely associated with *insulin resistance*, where tissue responds poorly to insulin signalling. Both genetic and acquired factors play a role in insulin resistance, including the presence of abdominal obesity, physical inactivity, and dietary and lifestyle choices.

BOX 29.1 Characteristics of the metabolic syndrome

- Abdominal obesity
- Abnormal blood lipids (low HDL, high triglycerides)
- Hypertension
- Elevated fasting blood glucose
- Clotting tendency
- Inflammatory factors

Risk factors unique to women

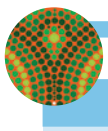
Risk factors unique to women include *premature menopause*, *oral contraceptive use* and *hormone replacement therapy (HRT)*. At menopause, serum HDL levels drop and LDL levels rise, increasing the risk of CHD (see the 'Translation to practice' box below. Early menopause (natural or surgically induced) increases the risk of CHD and MI. Women who have bilateral oophorectomy before age 35 without hormone replacement are eight times more likely to have an MI than women experiencing natural menopause. Oestrogen replacement therapy reduces the risk of CHD and MI in these women. Oral contraceptives with unopposed oestrogen (oestrogen without progestogen), by contrast, increase the risk of MI, particularly in women who also smoke. This increased risk is due to the tendency of oral contraceptives to promote clotting and their effects on blood pressure, serum lipids and glucose tolerance (Fallah et al., 2012; Andersson et al., 2012).

FAST FACTS

- Risk factors for coronary heart disease are those factors that promote atherosclerosis and plaque development.
- Angina pectoris, acute coronary syndromes and myocardial infarction are the manifestations of myocardial ischaemia and coronary heart disease due to atherosclerosis.
- Atherosclerosis also is the primary underlying cause of stroke and peripheral vascular disease; therefore, the risk factors for atherosclerosis are also the risk factors for coronary heart disease, including angina, acute coronary syndromes and myocardial infarction.

Risk factors unique to indigenous Australians

Some significant cardiovascular risk factors include obesity, diabetes, smoking, inactivity and chronic kidney disease. Indigenous Australians experience CVD-related risk factors more than non-Indigenous Australians. Prevalence of type 2 diabetes is three times greater and chronic kidney disease is four times greater in Indigenous Australians than in non-Indigenous Australian populations (AIHW, 2015c). There are stark demographic differences between Indigenous and non-Indigenous Australians (see Table 29.4 and the 'Focus on cultural diversity' box below).



TRANSLATIONTO PRACTICE Evidence-based practice: HRT and cardiovascular disease prevention in postmenopausal women

Numerous research projects have attempted to ascertain the benefit of hormone replacement therapy in the prevention of cardiovascular disease in postmenopausal women. In a recent Cochrane review by Boardman et al. (2015), 19 trials with a combined total of over 40 000 women found strong evidence to suggest that HRT is of little benefit in the context of preventing cardiovascular disease events. Furthermore, HRT demonstrated an increase in the risk of stroke and thromboembolic events.

IMPLICATIONS FOR NURSING

Nurses often are in the position of advising women about menopause, its manifestations and hormone replacement therapy. While HRT does reduce unpleasant menopausal effects such as night sweats and hot flushes, and it reduces the risk of osteoporosis and subsequent fractures, it carries associated risks. Advise each woman about the risks and benefits of HRT, clearly presenting the evidence. Encourage measures such as weight-bearing exercise, calcium supplements and a diet high in fibre to reduce the risks for osteoporosis, fracture and colorectal cancer. Ultimately, each woman will make her own decision about postmenopausal HRT.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. What are some common effects of menopause and how does HRT influence these effects?
2. What are the risks and benefits of HRT consumption in women after menopause?
3. What factors might you suggest an individual consider when deciding whether to use HRT for menopausal manifestations?

TABLE 29.4 Comparison of demographic characteristics between Indigenous and non-Indigenous Australians

	INDIGENOUS AUSTRALIANS	NON-INDIGENOUS AUSTRALIANS
Percentage of population	3%	75%
Current smokers—% of population (15 years and above)	44%	17%
Median age	22 years old	38 years old
Life expectancy (years)	Male—69, Female—74	Male—84, Female—87
Obesity	1.6% higher	
Hypertension	1.5% higher	
CVD-related deaths	25%	12%

Sources: Adapted from data from Australian Indigenous HealthInfoNet (2012; 2015); AIHW (2015a).

INTERPROFESSIONAL CARE

Care of people with coronary heart disease focuses on aggressive risk factor management to slow the atherosclerotic process and maintain myocardial perfusion. Until manifestations of chronic or acute ischaemia are experienced, the diagnosis often is presumptive, based on history and the presence of risk factors.

Diagnosis

Laboratory testing is used to assess for risk factors such as an abnormal blood lipid profile (elevated triglyceride and LDL levels and decreased HDL levels).

- *Total serum cholesterol* is elevated in hyperlipidaemia. A *lipid profile* includes triglyceride (TG), HDL and LDL levels. Calculations are made of the ratio of HDL to total cholesterol. The ratio should be at least 1:5, with 1:3 being the ideal ratio. Elevated lipid levels are associated with an increased risk of atherosclerosis. In people with a strong family history of premature CHD or familial hypercholesterolaemia, *lipoprotein (a)* (Lp(a)) may also be measured. Elevated levels of Lp(a) may independently increase the risk of CHD. Other subsets of blood lipids may also be measured in selected individuals. See the 'Diagnostic tests' boxes in Chapter 28 for nursing care related to lipid profile studies.

Diagnostic tests to identify subclinical (asymptomatic) CHD may be indicated when multiple risk factors are present.

- *C-reactive protein* is a serum protein associated with inflammatory processes. Recent evidence suggests that elevated blood levels of this protein may be predictive of CHD.
- *The ankle–brachial blood pressure index (ABI)* is an inexpensive, non-invasive test for peripheral vascular disease that may be predictive of CHD. The systolic blood pressure in the brachial, posterior tibial and dorsalis pedis arteries is measured by Doppler. An ABI of < 0.9 in either leg indicates the presence of peripheral arterial disease and a significant risk of CHD.
- *Exercise ECG testing* may be performed. ECGs are used to assess the response to increased cardiac workload induced by exercise. The test is considered 'positive' for CHD if myocardial ischaemia is detected on the ECG (depression of

FOCUS ON CULTURAL DIVERSITY Aboriginal and Torres Strait Islander people

Cardiovascular mortality in Aboriginal and Torres Strait Islander people is double that of non-Indigenous Australians and constitutes a significant health burden on Indigenous Australians. Myocardial infarctions account for almost 66% of cardiovascular-related deaths in males and almost 50% of cardiovascular-related deaths in females. All cardiovascular disease-associated risk factors are higher in Aboriginal and Torres Strait Islander Australians than they are for non-Indigenous Australians.

Ethnicity has a strong role in the development of cardiovascular disease. People from some countries experience fewer risk factors by virtue of their diet and genetics. Table 29.5 compares selected risk factors associated with CVD for non-Indigenous Australians against those in some other countries.

Implications for nursing

Australia is a multicultural country and an understanding of how ethnicity may increase or decrease an individual's risk factors for coronary artery disease is important in not only the assessment of an individual presenting with symptoms of cardiovascular dysfunction, providing holistic care and administering medications, but also in individualised planning of education sessions.

Critical thinking in person-centred care

1. It is well established that an individual's diet will influence cardiovascular risk factors. Choose four different cultures/ethnicities commonly found in Australia. Research a typical diet of these four different cultures.
2. Of the four cultures/ethnicities that you've chosen, predict how the diets might influence cardiovascular risk factors. Pay particular attention to fat, cholesterol and triglycerides.
3. Genetics can play an influence on the pharmacology of antihyperlipidaemic agents. Research the influence of genetics/ethnicity on lipid-lowering medications. Relate this to the answers you have given for question 2.

TABLE 29.5 Comparison of risk factors for CVD related to ethnicity

ETHNICITY (PEOPLE FROM)	RISK FACTOR COMPARED TO NON-INDIGENOUS AUSTRALIANS							
	HTN	Smoking	ETOH	Obesity	Physical inactivity	LDLs	HDLs	TG
New Zealand	↓	↑	↑	↑	↑	↑		
South Asia	↓				↑	↑		≅
China	↓	↑						≅
Middle East	↓	↑		↑	↑		↑	
UK and Ireland	↓	↑	↑		↑			
Greece	↓	↑		↑			↓	
Italy	↓		↑				↓	

↓ = lower risk; ↑ = higher risk; ≅ = approximately equal risk; HTN = hypertension; ETOH = drinks excessive alcohol; LDLs = low-density lipoproteins; HDLs = high-density lipoproteins; TG = triglycerides

Sources: Data compiled from various resources, including Foulds, Bredin & Warburton (2012); and Dassanayake et al. (2011).

the ST segment by greater than 3 mm) (see Figure 29.1 below), the person develops chest pain or the test is stopped due to excess fatigue, arrhythmias or other symptoms before the predicted maximal heart rate is achieved.

- *Electron beam computed tomography (EBCT)* creates a three-dimensional image of the heart and coronary arteries that can reveal plaque and other abnormalities. This non-invasive test requires no special preparation and can identify individuals at risk of developing myocardial ischaemia.
- *Myocardial perfusion imaging* (scintigraphy), see the section on angina that follows, may be used to evaluate myocardial blood flow and perfusion, both at rest and during stress testing (exercise or mental stress). These diagnostic tests are further explained in Chapter 28 and the section on angina. Perfusion imaging studies are costly and therefore are not recommended for routine CHD risk assessment.

Risk factor management

Conservative management of CHD focuses on risk factor modification, including smoking, diet, exercise and management of contributing conditions.

SMOKING Individuals who smoke have 20 times more angina attacks and are up to four times more at risk of dying from coronary artery disease than non-smokers (Australian National Preventive Health Agency, 2012). Smoking cessation reduces the risk of CHD within months after quitting and improves cardiovascular status (Zwar et al., 2011). In addition, stopping smoking improves HDL levels, lowers LDL levels and reduces blood viscosity. All smokers are advised to quit. Health promotion activities focus on preventing children, teenagers and adults from starting to smoke. Specific attention should also be paid to pregnant women in order to assist them to quit smoking.

DIET Dietary recommendations for cardiovascular health should include strategies to lower LDL levels and the need to reduce saturated fat and cholesterol intake. Most fats are a mixture of saturated and unsaturated fatty acids. The highest proportions of saturated fat are found in whole-milk products, red meats and coconut oil. Non-fat dairy products, fish and poultry as primary protein sources are recommended. Solidified vegetable fats (e.g. margarine, shortening) contain *trans* fatty acids, which behave more like saturated fats. Soft margarines and vegetable oil spreads contain low levels of trans fatty acids and should be used instead of butter, margarine and shortening. Monounsaturated fats, found in olive, canola and peanut oils, actually lower LDL and cholesterol levels. Certain cold-water fish, such as tuna, salmon and mackerel, contain high levels of omega-3 fatty acids, which help raise HDL levels and decrease serum triglycerides, total serum cholesterol and blood pressure (State Government of Victoria, 2015).

In addition, increased intake of soluble fibre (found in oats, psyllium, pectin-rich fruit and beans) and insoluble fibre (found in whole grains, vegetables and fruit) is recommended. Folic acid and vitamins B_6 and B_{12} affect homocysteine metabolism, reducing serum levels. Leafy green vegetables (e.g. spinach and broccoli) and legumes (e.g. black-eyed peas, dried beans and lentils) are rich sources of folate. Meat, fish and poultry are rich in vitamins B_6 and B_{12}. Vitamin B_6 also is found in soy products; B_{12} is in fortified cereals. Increased intake of antioxidant nutrients (vitamin E, in particular) and foods rich in antioxidants (fruits and vegetables) appear to increase HDL levels and have a protective effect on CHD (State Government of Victoria, 2015).

In middle-aged and older adults, low levels of alcohol consumption (½ standard drink a day) may reduce the risk of CHD. Consumption of no more than two drinks per day for men or one drink per day for women is recommended. People who do not drink alcohol, however, should not be encouraged to start consuming it as a heart-protective measure as any benefit from alcohol consumption can be gained more readily from healthy diet and exercise (National Health and Medical Research Council (NHMRC), 2009).

People who are overweight or obese are encouraged to lose weight through a combination of reduced kilojoule intake (maintaining a nutritionally sound diet) and increased exercise. High-protein, high-fat weight-loss programs are not recommended for weight reduction.

EXERCISE Regular physical exercise reduces the risk of CHD in several ways. It lowers VLDL, LDL and triglyceride levels and raises HDL levels. Regular exercise reduces blood pressure and insulin resistance. National guidelines for physical activity for adults between 18 and 64 years of age have recently changed to accumulate between 150 and 300 minutes of moderate intensity or 75–150 minutes of intense physical intensity (or a combination) each week. Several themes are fostered in the new guidelines, including doing any physical activity is better than none, trying to be active most days and gradually building to the recommended amount (Department of Health, 2014).

HYPERTENSION Although hypertension often cannot be prevented or cured, it can be controlled. Hypertension control (maintaining a blood pressure lower than 140/90 mmHg) is vital to reduce its atherosclerosis-promoting effects and to reduce the workload of the heart. Management strategies include reducing sodium intake, increasing calcium intake, regular exercise, stress management and medications. Hypertension management is discussed in Chapter 31.

DIABETES Diabetes increases the risk of CHD by accelerating the atherosclerotic process. Weight loss (if appropriate), reduced fat intake and exercise are particularly important for the person with diabetes. Because hyperglycaemia also contributes to atherosclerosis, consistent blood glucose management is vital. See Chapter 19 for a detailed discussion of diabetes and blood glucose management.

Medications

Drug therapy to lower total serum cholesterol and LDL levels and to raise HDL levels is now an integral part of CHD management. It is used in conjunction with diet and other lifestyle changes and is based on the individual's overall risk of CHD.

Drugs used to treat hyperlipidaemia act specifically by lowering LDL levels and increasing HDLs. The goal of treatment is to achieve an LDL level of < 1.8 mmol/L and an HDL of > 1.0 mmol/L (NHFA/CSANZ, 2012). Medications to treat hyperlipidaemia are not inexpensive; the cost–benefit ratio needs to be considered because long-term treatment may be required. The four main classes of cholesterol-lowering drugs are statins, bile acid sequestrants, nicotinic acid and fibrates (see 'Medication administration' box).

People at high risk of MI are often started on prophylactic low-dose aspirin therapy. The usual dose is 100 mg/day. In women, the benefit of low-dose aspirin in reducing the risk of CHD is not clear prior to age 65. Aspirin is contraindicated for individuals who have a history of aspirin sensitivity, bleeding disorders or active peptic ulcer disease. Angiotensin-converting-enzyme (ACE) inhibitors or angiotensin receptor blockers also may be prescribed for high-risk individuals, including diabetics with other CHD risk factors (Bullock & Manias, 2013).

THE PERSON WITH ANGINA PECTORIS

Angina pectoris, or *angina*, is chest pain resulting from reduced coronary blood flow, which causes a temporary imbalance between myocardial blood supply and demand. The imbalance may be due to coronary heart disease, atherosclerosis or vessel constriction that impairs myocardial blood supply. Hypermetabolic conditions such as exercise, thyrotoxicosis, stimulant abuse (e.g. cocaine), hyperthyroidism and emotional stress can increase myocardial oxygen demand, precipitating angina. Anaemia, heart failure, ventricular hypertrophy or pulmonary diseases may affect blood and oxygen supplies as well, causing angina.

Pathophysiology

The imbalance between myocardial blood supply and demand causes temporary and reversible myocardial ischaemia. **Ischaemia**, deficient blood flow to tissue, may be caused by partial obstruction of a coronary artery, coronary artery spasm or a

MEDICATION ADMINISTRATION Cholesterol-lowering drugs

STATINS

Statins inhibit the enzyme HMG-CoA reductase in the liver, lowering LDL synthesis and serum levels. The statins are first-line treatment for elevated LDL, used in conjunction with diet and lifestyle changes. Although their side effects are minimal, they may cause increased serum liver enzyme levels and myopathy.

Nursing responsibilities

- Monitor serum cholesterol and liver enzyme levels before and during therapy. Report elevated liver enzyme levels.
- Assess for muscle pain and tenderness. Monitor creatine phosphokinase (CPK) level if present.
- If taking digoxin concurrently, monitor for and report digoxin toxicity.

Health education for the person and family

- Promptly report muscle pain, tenderness or weakness; skin rash or hives or changes in skin colour; abdominal pain, nausea or vomiting.
- Drugs should not be used when pregnant or if pregnancy is planned.
- Inform the medical officer if taking any other medications concurrently.

BILE ACID SEQUESTRANTS

Bile acid sequestrants lower LDL levels by binding bile acids in the intestine, reducing LDL reabsorption and cholesterol production in the liver. They are used in combination therapy regimens and for women who are considering pregnancy. Their primary disadvantages are inconvenience of administration due to bulk and gastrointestinal side effects such as constipation.

Nursing responsibilities

- Store in a tightly closed container.

Health education for the person and family

- Promptly report constipation, severe gastric distress with nausea and vomiting, unexplained weight loss, black or bloody stools, or sudden back pain.
- Drinking ample amounts of fluid while taking these drugs reduces problems of constipation and bloating.
- Do not omit doses as this may affect the absorption of other drugs you are taking.

NICOTINIC ACID

Nicotinic acid in both prescription and non-prescription forms lowers total and LDL cholesterol and triglyceride levels. The crystalline form and the extended-release tablet also raise HDL levels. Because the doses required to achieve significant cholesterol-lowering effects are associated with multiple side effects, nicotinic acid generally is used in combination therapy, particularly with statins.

Nursing responsibilities

- Give oral preparations with meals and accompanied by a cold beverage to minimise GI effects.
- Administer with caution to individuals with active liver disease, peptic ulcer disease, gout or type 2 diabetes.
- Monitor blood glucose, uric acid levels and liver function tests during treatment.

Health education for the person and family

- Flushing of face, neck and ears may occur within 2 hours following dose; these effects generally subside as treatment continues. Alcohol use during nicotinic acid therapy may worsen this effect.
- Report weakness or dizziness with changes in posture (lying to sitting; sitting to standing). Change positions slowly to reduce the risk of injury.

FIBRIC ACID DERIVATIVES

The fibrates are used to lower serum triglyceride levels; they have only a slight to modest effect on LDL. They affect lipid regulation by blocking triglyceride synthesis. They are used to treat very high triglyceride levels and may be used in combination with statins.

Nursing responsibilities

- Monitor serum LDL and VLDL levels, electrolytes, glucose, liver enzymes, renal function tests and full blood count (FBC) during therapy. Report abnormal values.
- Up to 2 months of treatment may be required to achieve a therapeutic effect; rebound, with decreasing benefit, may occur in the second or third month of treatment.

Health education for the person and family

- Take with meals if the drug causes gastric distress.
- Promptly report flu-like symptoms (fatigue, muscle aching, soreness or weakness).
- Drug should not be used in pregnancy. Use reliable birth control measures while taking this drug.
- Consultation with a medical officer is required before stopping this drug and before taking any over-the-counter preparations.

thrombus. Obstruction of a coronary artery deprives cells in the region of the heart normally supplied by that vessel of oxygen and nutrients needed for metabolic processes. Cellular processes are compromised as ATP stores are depleted. Reduced oxygen causes cells to switch from aerobic metabolism to anaerobic metabolism. Anaerobic metabolism causes lactic acid to build up in the cells. It also affects cell membrane permeability, releasing substances such as histamine, kinins and specific enzymes that stimulate terminal nerve fibres in the cardiac muscle and send pain impulses to the central nervous system. The pain radiates to the upper body because the heart shares the same dermatome as this region. Return of adequate circulation provides the nutrients needed by cells and clears the waste products. More than 30 minutes of ischaemia irreversibly damages myocardial cells (necrosis).

Three types of angina have been identified:

1. *Stable angina* is the most common and predictable form of angina. It occurs with a predictable amount of activity or stress and is a common manifestation of CHD. Stable angina usually occurs when the work of the heart is increased by physical exertion, exposure to cold or by stress. Stable angina is relieved by rest and nitrates.

2. *Prinzmetal's (variant) angina* is atypical angina that occurs unpredictably (unrelated to activity) and often at night. It is caused by coronary artery spasm with or without an atherosclerotic lesion. The exact mechanism of coronary artery spasm is unknown. It may result from hyperactive sympathetic nervous system responses, altered calcium flow in smooth muscle or reduced prostaglandins that promote vasodilation.
3. *Unstable angina* occurs with increasing frequency, severity and duration. Pain is unpredictable and occurs with decreasing levels of activity or stress and may occur at rest. People with unstable angina are at risk of myocardial infarction. Unstable angina is discussed further in the section on acute coronary syndromes that follows.

Silent myocardial ischaemia, or asymptomatic ischaemia, is thought to be common in people with CHD, especially with the co-morbidity diabetes mellitus. Silent ischaemia may occur with either activity or mental stress. Mental stress increases the heart rate and blood pressure, increasing myocardial oxygen demand (Bullock & Hales, 2012). Like symptomatic angina, silent myocardial ischaemia is associated with an increased chance of myocardial infarction and death.

FAST FACTS

- Stable angina occurs with a predictable amount of activity or stress.
- Unstable angina occurs with increasing frequency and severity; it may occur at times unrelated to activity or stress.
- Prinzmetal's angina is the only type of angina not necessarily related to coronary heart disease and atherosclerosis; it develops due to coronary artery spasm.

Course and manifestations

The cardinal manifestation of angina is chest pain. The pain typically is precipitated by an identifiable event, such as physical activity, strong emotion, stress, eating a heavy meal or exposure to cold. The classic sequence of angina is activity–pain, rest–relief. The person may describe the pain as a tight, squeezing, heavy pressure or constricting sensation. It characteristically begins beneath the sternum and may radiate to the jaw, neck, shoulder or arm. Less characteristically, the pain may be felt in the jaw, epigastric region or back. Anginal pain usually occurs in a crescendo–decrescendo pattern (increasing to a peak, then gradually decreasing) and typically lasts 2 to 5 minutes. It generally is relieved by rest. Additional manifestations of angina include dyspnoea, pallor, tachycardia, and great anxiety and fear.

Women frequently present with atypical symptoms of angina, including indigestion or nausea, vomiting and upper back pain. The manifestations of angina are summarised in the accompanying box.

The severity of angina can be graded by the degree to which it limits the individual's activities. Class I angina does not occur with ordinary physical activities. It is prompted by strenuous, rapid or prolonged physical exertion. Class II angina may develop with rapid or prolonged walking or stair climbing, whereas class III angina significantly limits ordinary physical activities. The person with class IV angina may have angina at rest, as well as with any physical activity (Alaeddini, 2014).

MANIFESTATIONS Angina

- *Chest pain:* substernal or precordial (across the chest wall); may radiate to neck, arms, shoulders or jaw.
- *Quality:* tight, squeezing, constricting or heavy sensation; may also be described as burning, aching, choking, dull or constant.
- *Associated manifestations:* dyspnoea, pallor, tachycardia, anxiety and fear.
- *Atypical manifestations:* indigestion, nausea, vomiting, upper back pain.
- *Precipitating factors:* exercise or activity, strong emotion, stress, cold, heavy meal.
- *Relieving factors:* rest, position change; glyceryl trinitrate.

INTERPROFESSIONAL CARE

The management of stable angina focuses on maintaining coronary blood flow and cardiac function. Stable angina often can be managed by medical therapy. Measures to restore coronary blood flow are discussed in the section on acute coronary syndrome. As for CHD, risk factor management is a vital component of care for the person with angina (see the preceding section of this chapter).

Diagnosis

The diagnosis of angina is based on past medical history and family history, a comprehensive description of the chest pain and physical assessment findings. Laboratory tests may confirm the presence of risk factors, such as an abnormal blood lipid profile and elevated blood glucose. Diagnostic tests provide information about overall cardiac function.

Common diagnostic tests to assess for coronary heart disease and angina include electrocardiography, stress testing, nuclear medicine studies, echocardiography (ultrasound) and coronary angiography.

ELECTROCARDIOGRAPHY A resting ECG may be normal, may show non-specific changes in the ST segment and T wave, or may show evidence of previous myocardial infarction. Characteristic ECG changes are seen during anginal episodes. During periods of ischaemia, the ST segment is depressed or downsloping and the T wave may flatten or invert (see Figure 29.1). These changes reverse when ischaemia is relieved. For more details about the ECG, its waveforms and its uses, see Chapter 28.

STRESS ELECTROCARDIOGRAPHY Stress electrocardiography (exercise stress test) uses ECGs to monitor the cardiac response to an increased workload during progressive exercise.

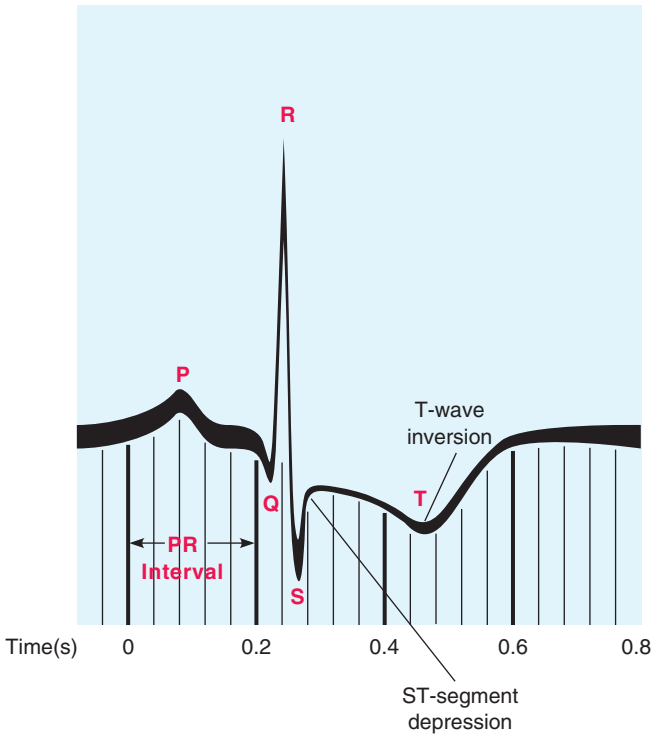


FIGURE 29.1 ■ ECG changes during an episode of angina. Note characteristic T-wave inversion and ST-segment depression of myocardial ischaemia

See the 'Diagnostic tests' box in the previous chapter for more information about exercise stress tests.

RADIONUCLIDE TESTING Radionuclide testing is a safe, non-invasive technique to evaluate myocardial perfusion and left ventricular function. The amount of radioisotope injected is very small; no special radiation precautions are required during or after the scan. Thallium-201 or a technetium-based radio compound is injected intravenously and the heart is scanned with a radiation detector. Ischaemic or infarcted cells of the myocardium do not take up the substance normally, appearing as a 'cold spot' on the scan. If the ischaemia is transient, these spots gradually fill in, indicating the reversibility of the process. With severe ischaemia or a myocardial infarction, these areas may remain devoid of radioactivity.

Left ventricular function can also be evaluated. Whereas the ejection fraction or portion of blood ejected from the left ventricle during systole normally increases during exercise, it may actually decrease in coronary heart disease and stress-induced ischaemia.

Radionuclide testing may be combined with pharmacological stress testing for individuals who are physically unable to exercise or to detect subclinical myocardial ischaemia. A vasodilator is injected to induce the same ischaemic changes that occur with exercise in the diseased heart. Coronary arteries unaffected by atherosclerosis dilate in response to the drugs, increasing blood flow to already well-perfused tissue. This reduces flow to ischaemic muscle, called *myocardial steal syndrome*.

ECHOCARDIOGRAPHY *Echocardiography* is a non-invasive test that uses ultrasound to evaluate cardiac structure and function. It may be done at rest, during supine exercise or immediately following upright exercise to evaluate movement of the myocardial wall and assess for possible ischaemia or infarction.

Transoesophageal echocardiography (TOE) uses ultrasound to identify abnormal blood flow patterns as well as cardiac structures. In TOE, the probe is on the tip of an endoscope inserted into the oesophagus, positioning it close to the posterior heart (especially the left atrium and the aorta). It avoids interference by breasts, ribs or lungs.

See the 'Diagnostic tests' box in Chapter 28 for more information about nursing care of the person undergoing these tests.

CORONARY ANGIOGRAPHY *Coronary angiography* is the gold standard for evaluating the coronary arteries. Guided by fluoroscopy, a catheter introduced into the femoral or brachial artery is threaded into the coronary artery. Dye is injected into each coronary opening, allowing visualisation of the main coronary branches and any abnormalities, such as stenosis or obstruction. Narrowing of the vessel lumen by more than 50% is considered significant; most lesions that cause symptoms involve more than 70% narrowing. Vessel obstructions are noted on a coronary artery 'map' that provides a guide for tracking disease progression and for elective treatment with angioplasty or cardiac surgery. See the 'Diagnostic tests' box in Chapter 28 for the nursing care of the person undergoing coronary angiography. See the 'Translation to practice' box below regarding recent research related to angiography alternatives.

Medications

Drugs may be used for both acute and long-term relief of angina. The goal of drug treatment is to reduce oxygen demand and increase oxygen supply to the myocardium. Three main classes of drugs are used to treat angina: nitrates, beta-blockers and calcium channel blockers.

NITRATES Nitrates, including glyceryl trinitrate (GTN) and longer-acting nitrate preparations, are used to treat acute anginal attacks and prevent angina.

Sublingual glyceryl trinitrate (Anginine) is the drug of choice to treat acute angina. It acts within 1 to 2 minutes, decreasing myocardial work and oxygen demand through venous and arterial dilation, which in turn reduces preload and afterload. It may also improve myocardial oxygen supply by dilating collateral blood vessels and reducing stenosis. Rapid-acting glyceryl trinitrate is also available as a buccal spray in a metered system. For some individuals, this may be easier to handle than small glyceryl trinitrate tablets. The spray also stores longer, reducing the need to discard and repurchase fresh medication every 3 months.

CONSIDERATION FOR PRACTICE

Sublingual glyceryl trinitrate tablets and glyceryl trinitrate spray are the only medications appropriate to treat an acute anginal attack.

TRANSLATION TO PRACTICE Possible non-invasive alternative to angiography to determine the presence of coronary artery disease

A group based at the Monash Centre of Cardiovascular Research Education in Therapeutics is investigating whether measurement of 5-minute heart rate variability can assist with risk stratification in people with cardiovascular disease. In a prospective observational study of over 450 people, it was determined that low heart rate variability is a strong predictor of coronary artery disease in people with sinus rhythm (Kotecha et al., 2012).

IMPLICATIONS FOR NURSING

Nurses are often required to care for individuals following coronary angiography, which is an invasive procedure used to quantify the presence of cardiovascular disease. This research may have identified a simple, non-invasive method of risk stratification that may be plausible for use in determining management or drug therapies.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. What are the risks and benefits associated with reliance on heart rate variability as opposed to the current gold standard for determining coronary artery disease (angiography)?
2. How will this potential change in method of cardiac investigation impact on provision of care, nursing workload and education of individuals with CVD?

Longer-acting glyceryl trinitrate preparations (oral tablets, ointment or transdermal patches) are used to prevent attacks of angina, not to treat an acute attack. The primary problem with long-term nitrate use is the development of *tolerance*, a decreasing effect from the same dose of medication. Tolerance can be limited by a dosing schedule that allows a nitrate-free period of at least 8 to 10 hours daily. This is usually scheduled at night, when angina is less likely to occur.

Headache is a common side effect of nitrates and may limit their usefulness. Nausea, dizziness and hypotension are also common effects of therapy.

BETA-BLOCKERS Beta-blockers, including propranolol, metoprolol and atenolol, are considered first-line drugs to treat stable angina. They block the cardiac-stimulating effects of noradrenaline and adrenaline, preventing anginal attacks by reducing heart rate, myocardial contractility and blood pressure, thus reducing myocardial oxygen demand. Beta-blockers may be used alone or with other medications to prevent angina.

Beta-blockers are contraindicated for people with asthma or severe COPD (see Chapter 36) because they may cause severe bronchospasm. They are not used in people with significant bradycardia or atrioventricular (AV) conduction blocks and are used cautiously in heart failure. Beta-blockers are not used to treat Prinzmetal's angina because they may make it worse.

CALCIUM CHANNEL BLOCKERS Calcium channel blockers reduce myocardial oxygen demand and increase myocardial blood and oxygen supply. These drugs, which include verapamil, diltiazem and nifedipine, lower blood pressure, reduce myocardial contractility and, in some cases, lower the heart rate, decreasing myocardial oxygen demand. They are also potent coronary vasodilators, effectively increasing oxygen supply. Like beta-blockers, calcium channel blockers act too slowly to effectively treat an acute attack of angina; they are used for long-term prophylaxis. Because they may actually increase ischaemia and mortality in people with heart failure or left ventricular dysfunction, these drugs are not usually prescribed in the initial treatment of angina. They are used cautiously in individuals with arrhythmias, heart failure or hypotension.

The nursing implications of antianginal medications are summarised in the 'Medication administration' box below.

ASPIRIN The person with angina, particularly unstable angina, is at risk of myocardial infarction because of significant narrowing of the coronary arteries. Low-dose aspirin (100 mg/day) is often prescribed to reduce the risk of platelet aggregation and thrombus formation.

Nursing care

The focus of nursing care for the person with angina is similar to the interprofessional care focus: to reduce myocardial oxygen demand and improve the oxygen supply. Angina usually is treated in community settings; the primary nursing focus is education.

Health promotion

In addition to health promotion measures identified for CHD, emphasise the importance of active CHD risk factor management to slow progression of the disease. Encourage the person to stop smoking. Discuss the use of cholesterol-lowering drug therapy with people who have hypercholesterolaemia. Encourage regular aerobic exercise and a diet based on the National Heart Foundation guidelines.

Assessment

Focused assessment data for the person with angina include the following:

- *Health history:* chest pain—Onset, Location, Duration, Characteristics, Aggravating symptoms, methods to Relieve the pain, current Treatment (OLDCART); history of other cardiovascular disorders, peripheral vascular disease or stroke; current medications and treatment; usual diet, exercise and alcohol intake patterns; smoking history; use of other recreational drugs.
- *Physical assessment:* vital signs and heart sounds; strength and equality of peripheral pulses; skin colour and

MEDICATION ADMINISTRATION Antianginal medications

ORGANIC NITRATES

Nitrates dilate both arterial and venous vessels, depending on the dose. Coronary artery vasodilation increases myocardial oxygen supply. Venous dilation allows peripheral blood pooling, reducing venous return, preload and cardiac work. Arterial dilation reduces vascular resistance and afterload, also reducing cardiac work. Sublingual glyceryl trinitrate (GTN) is used to treat and prevent acute anginal attacks (when taken prophylactically before activity). Nitrates are administered sublingually by buccal spray, or intravenously for immediate effect; or orally or topically for sustained effect.

Nursing responsibilities

- Dilute intravenous glyceryl trinitrate before infusing; use only glass bottles for the mixture. Glyceryl trinitrate will adsorb onto many infusion lines and PVC bags, affecting the amount of drug that is delivered. Special GTN infusion tubing must be used.
- Wear gloves when opening ampoule and drawing up glyceryl trinitrate infusions to prevent inadvertent administration to self by absorbing through the skin.
- Remove glyceryl trinitrate patches daily (or when required) reducing tolerance by providing GTN-free time.

Health education for the person and family

- Use only the sublingual, buccal and spray forms of nitrates to treat acute angina.
- If the first nitrate dose does not relieve angina within 5 minutes, take a second dose. After 5 more minutes, you may take a third dose if needed. If the pain is unrelieved, seek medical assistance immediately.
- Advise the person to carry a supply of glyceryl trinitrate tablets with them. Dissolve sublingual glyceryl trinitrate tablets under the tongue or between the upper lip and gum. Do not eat, drink or smoke until the tablet is completely dissolved.
- Keep sublingual tablets in their original amber glass bottle to protect them from heat, light and moisture. Replacement should occur every 3 months after opening the supply (tablets). Spray can be kept for a year.
- Burning or tingling sensation may be felt under the tongue and the individual may develop a transient headache on taking the drug. These effects are expected; the headache will diminish over time.
- Use caution when standing from a sitting position; glyceryl trinitrate may cause light headedness.
- Rotate the application sites of the transdermal patches. Apply to a hairless area so that the drug can reach the skin. Remove the patch at bedtime daily (or as ordered). Apply a fresh dose in the morning (or as ordered).
- If using a long-acting nitrate, a supply of immediate-acting nitrates should be kept to treat acute angina.

BETA-BLOCKERS

Beta-blockers decrease cardiac workload by blocking beta receptors on the heart muscle, thus decreasing heart rate, contractility, myocardial oxygen consumption and blood pressure. Beta-blockers also reduce *reflex tachycardia* (an increased heart rate in response to stimuli such as increased sympathetic nervous system activity or vasodilation), which may develop with other antianginal drugs. Beta-blockers are frequently prescribed as antianginal and antihypertensive agents. Some examples include metoprolol and carvedilol.

Nursing responsibilities

- Document heart rate and blood pressure before administering the medication. Withhold drug if the heart rate is below 50 bpm or the blood pressure is below prescribed limits. Notify the doctor.
- Assess for and report possible contraindications to therapy, including heart failure, bradycardia, AV block, asthma or chronic obstructive pulmonary disease (COPD).
- Concurrent use of beta-blockers and calcium channel blockers increases the risk of heart failure; notify the doctor if these drugs are prescribed together.
- Do not abruptly discontinue these drugs after long-term therapy, as this can increase heart rate, contractility and blood pressure, and cause fatal arrhythmia, myocardial infarction or stroke.

Health education for the person and family

- Beta-blockers help prevent angina but will not relieve an acute attack. Keep a supply of fast-acting nitrates on hand for acute anginal attacks.
- Do not suddenly stop taking this medication. Discuss discontinuing medication with medical officers.
- Take pulse before administration. Do not take the drug and contact the doctor if the heart rate is below 50 bpm. Check blood pressure frequently.
- Report a slow or irregular pulse, swelling or weight gain, or difficulty breathing.

CALCIUM CHANNEL BLOCKERS

Calcium channel blockers are used to control angina, hypertension and arrhythmias. By blocking the entry of calcium into cells, these drugs reduce contractility, slow the heart rate and conduction, and cause vasodilation. Calcium channel blockers increase myocardial oxygen supply by dilating the coronary arteries; they decrease the workload of the heart by lowering vascular resistance and oxygen demand. Calcium channel blockers are often prescribed for people with coronary artery spasm (Prinzmetal's angina). Some examples include verapamil and nifedipine.

Nursing responsibilities

- Do not mix verapamil in any solution containing sodium bicarbonate. Administer IV push verapamil over 2 to 3 minutes.
- Document blood pressure and heart rate before administering the drug. Withhold the drug if the heart rate is below 50 bpm. Notify the doctor.
- The nifedipine capsule may be punctured and administered by extracting the liquid with a syringe and squirting the dose under the person's tongue. (Discard the needle first!)

MEDICATION ADMINISTRATION Antianginal medications (continued)

- Use caution when giving a calcium channel blocker with other cardiac depressants, such as beta-blockers. Concomitant administration with nitrates may cause excessive vasodilation.
- Manifestations of toxicity include nausea, generalised weakness, signs of decreased cardiac output, hypotension, bradycardia and AV block. Report these findings immediately. Maintain intravenous access and slowly administer intravenous calcium chloride. Do not infuse large volumes of fluid to treat hypotension as heart failure may result.

Health education for the person and family

- Take pulse before taking the drug. Do not take the drug and notify doctor if the heart rate drops below 50 bpm.
- Keep a fresh supply of immediate-acting nitrate available to treat acute anginal attacks. Calcium channel blockers will not work fast enough to relieve an acute attack.

temperature (central and peripheral); physical appearance during pain episode (e.g. shortness of breath, apparent anxiety, colour, diaphoresis).

Nursing diagnoses and interventions

High-priority nursing problems for individuals with angina include ineffective cardiac tissue perfusion and management of the prescribed therapeutic regimen.

Ineffective tissue perfusion: cardiac

The pain of angina results from impaired blood flow and oxygen supply to the myocardium. Nursing interventions can both prevent ischaemia and shorten the duration of pain.

- Keep prescribed glyceryl trinitrate tablets (or spray) at the person's side so that this can be taken at the onset of pain. Anginal pain indicates *myocardial ischaemia. Glyceryl trinitrate causes vasodilation improving myocardial oxygen supply, relieving ischaemia and pain.*
- Start oxygen at 4 to 6 L/min per nasal prongs or as prescribed. *Supplemental oxygen reduces myocardial hypoxia by increasing oxygen supply.*
- Plan activities to allow rest between them. *Activity increases myocardial oxygen demand and may precipitate angina. Spacing activities reduces the risk of exceeding myocardial oxygen supply.*
- Teach about prescribed medications to maintain myocardial perfusion and reduce cardiac work. Emphasise that beta-blockers, calcium channel blockers and long-acting nitrates are used to *prevent* anginal attacks, not to *treat* an acute attack. *It is important for the person to understand the purpose and use of prescribed drugs to maintain optimal myocardial perfusion.*
- Instruct to take sublingual glyceryl trinitrate before engaging in activities that precipitate angina (e.g. climbing stairs, sexual intercourse). *This prophylactic dose of glyceryl trinitrate helps maintain cardiac perfusion (by improving myocardial oxygen supply) when increased work (myocardial oxygen demand) is anticipated; this reduces ischaemia and chest pain.*
- Encourage the person to implement and maintain a progressive exercise program under the supervision of the primary care provider or a cardiac rehabilitation professional. *Exercise slows the atherosclerotic process and helps develop collateral circulation to the heart muscle.*
- Refer individual to a smoking cessation program as indicated. *Nicotine causes vasoconstriction, increases the heart rate, decreases myocardial perfusion and increases cardiac workload.*

Risk of ineffective therapeutic regimen management

Denial may be strong in the person with angina pectoris. Because many people think of the heart as the locus of life itself, problems such as angina remind people of their mortality, an uncomfortable fact. Denial may lead to 'forgetting' to take prescribed medications or to attempting activities that will precipitate angina. Some people, by contrast, may become 'cardiac cripples', afraid to engage in activities because of anticipated chest pain. Their inactivity may actually hasten the atherosclerotic process and inhibit collateral circulation development, worsening angina.

- Assess knowledge and understanding of angina. *Assessment allows tailoring of teaching and interventions to the needs of the person.*
- Teach the individual about angina and atherosclerosis, building on current knowledge base. *This can help the person understand that angina is a manageable disease and that pain can usually be controlled and the disease progress slowed.*
- Provide written and verbal instructions about prescribed medications and their use. *Written instructions reinforce teaching and are available to the person for future reference.*
- Stress the importance of taking chest pains seriously while maintaining a positive attitude. *Although it is vital to recognise the significance of chest pain and deal with it appropriately, it is also important to maintain a positive outlook.*
- Refer to a cardiac rehabilitation program or other organised activities and support groups for individuals with coronary heart disease. *Programs such as these help the person develop risk factor management strategies, maintain a program of supervised activity and gain coping skills.*

Community-based care

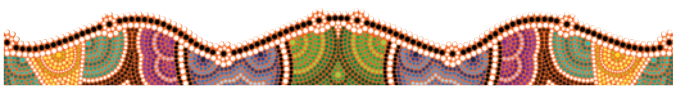
Many people with stable angina manage their pain effectively, continuing to live active and productive lives. To promote

effective management of this disorder, include the following topics in teaching for home care:

- coronary heart disease and the processes that cause chest pain, including the relationship between the pain and reduced blood flow to the heart muscle
- use and effects (desired and adverse) of prescribed medications; importance of not discontinuing medications abruptly
- glyceryl trinitrate use for acute angina: always carry tablets; use prophylactically before activities that often cause chest pain; take tablet at first indication of pain rather than waiting to see if the pain develops; seek immediate medical assistance if three glyceryl trinitrate tablets over 15 minutes do not relieve the pain
- the importance of calling 000 or going to the emergency department immediately for unrelieved chest pain
- appropriate storage of glyceryl trinitrate: this unstable compound needs to be stored in a cool, dry, dark place; no more than a 3-month supply should be kept on hand.

For the person who has undergone cardiac surgery, also include the following:

- respiratory care, activity and pain management
- the importance of actively participating in rehabilitation
- manifestations of infection or other potential complications and their management.



THE PERSON WITH ACUTE CORONARY SYNDROME

Acute coronary syndrome (ACS) is a condition of unstable cardiac ischaemia. ACS includes unstable angina and acute myocardial ischaemia with or without significant injury of myocardial tissue. Although the term ACS may, in some cases, be applied to acute myocardial infarction (AMI) (myocardial tissue death), myocardial infarction is discussed separately in the next section of this chapter.

FAST FACTS

- Acute coronary syndrome (severe cardiac ischaemia), a common cause of hospital admission, includes unstable angina and acute myocardial infarction.
- Unstable angina is characterised by injury to myocardial cells. With prompt restoration of blood flow, muscle tissue recovers.
- Myocardial infarction is characterised by necrosis and death of myocardial cells; scar tissue forms and functional muscle is lost.

Pathophysiology

ACS is a dynamic state in which coronary blood flow is acutely reduced, but not fully occluded. Myocardial cells are injured by the acute ischaemia that results. Most people affected by ACS have significant stenosis of one or more coronary arteries.

ACS is precipitated by any number of pathophysiological causes, including partial occlusion and clot formation of myocardial blood vessels, an oxygen demand versus supply issue or acute changes in the patency of critical myocardial vessels (Coven, 2015). Changes in haemodynamic factors such as increased heart rate, blood flow and blood pressure occur in response to a surge of sympathetic nervous system (SNS) activity. Increased SNS activity also is thought to contribute to the higher incidence of plaque rupture (van Rosendael, de Graaf & Scholte, 2015).

When atherosclerotic plaque ruptures or erodes, the exposed lipid core of the plaque stimulates platelet aggregation and the extrinsic clotting pathway. Thrombin is generated and fibrin is deposited, forming a clot that severely impairs or obstructs blood flow to tissue distal to the area of plaque rupture. As a result, these cells become ischaemic.

Injured myocardial cells contract less effectively, potentially reducing cardiac output if a large area of myocardium is affected. Lactic acid released from ischaemic cells stimulates pain receptors, causing chest pain. Ischaemia and injury affect electrical impulse conduction, producing inversion of the T wave and possibly elevation of the ST segment on the ECG.

Manifestations

The cardinal manifestation of ACS is chest pain, usually substernal or epigastric. The pain often radiates to the neck, left shoulder and/or left arm. The pain may occur at rest and typically lasts longer than 10 to 20 minutes. In ACS, the chest pain is more severe and prolonged than that previously experienced by the individual. It may be a new onset of pain or may represent a pattern of increasing frequency and severity of anginal pain. Dyspnoea, diaphoresis, pallor and cool skin may be present. Tachycardia and hypotension may occur. The person may be nauseated or feel light headed.

INTERPROFESSIONAL CARE

The person with ACS generally presents at the emergency department or doctor's office with complaints of severe chest pain. The pain may be unrelieved by glyceryl trinitrate or may be more severe and of longer duration than previous anginal episodes. The ECG is used in conjunction with blood levels of cardiac markers to differentiate between unstable angina and acute myocardial infarction. People with unstable angina generally are admitted to the acute care unit on bed rest with cardiac monitoring for 12 to 24 hours. Coronary revascularisation procedures may be performed within 48 hours if significant CHD is identified.

Diagnosis

The ECG and serum cardiac markers are the primary tests used to establish the diagnosis of ACS. Serum cardiac markers, proteins released from injured and necrotic heart muscle, can be measured. (See the following section on acute myocardial infarction and Table 29.6 for more information about serum cardiac markers.)

- Cardiac muscle troponins, *cardiac-specific troponin T* (cT_nT) and *cardiac-specific troponin I* (cT_nI), are sensitive indicators of myocardial damage. Troponins may be elevated in

TABLE 29.6 Serum cardiac markers

MARKER	NORMAL LEVEL	PRIMARY TISSUE LOCATION	SIGNIFICANCE OF ELEVATION	CHANGES OCCURRING WITH MI		
				APPEARS	PEAKS	DURATION
CK (CPK)	< 270 U/L	Cardiac muscle, skeletal muscle, brain	Injury to muscle cells	3 to 6 hours	12 to 24 hours	24 to 48 hours
CK-MB	< 10 U/L	Cardiac muscle	MI, cardiac ischaemia, myocarditis, cardiac contusion, defibrillation	4 to 8 hours	18 to 24 hours	72 hours
cT_nT	< 0.1 µg/L	Cardiac and skeletal muscle	Acute MI, unstable angina	2 to 4 hours	24 to 36 hours	10 to 14 days
cT_nI	0.4 µg/L (ACS) > 01.5 µg/L (AMI)	Cardiac muscle	Acute MI, unstable angina	2 to 4 hours	24 to 36 hours	7 to 10 days

ACS or may be within normal limits if chest pain is due to unstable angina.

- Creatine kinase (CK) and CK-MB (specific to myocardial muscle) levels are likely to be within normal limits or demonstrate transient elevation, returning to normal levels within 12 to 24 hours.

The ECG, particularly when done during the acute episode of chest pain, is a valuable diagnostic tool for ACS. ST-segment changes (elevation or depression) during chest pain that resolve when the pain abates usually indicate acute myocardial ischaemia and severe underlying CHD.

Medications

Medications include drugs to reduce myocardial ischaemia and to reduce the risk of blood clotting. Thrombolytic drugs (i.e. drugs that break down the fibrin in blood clots) may be given prior to or on admission to the emergency department. These drugs restore blood flow to ischaemic cardiac muscle and can prevent permanent damage. See the section on myocardial infarction for more information about thrombolytic drugs and their nursing implications.

Nitrates and beta-blockers are used to restore blood flow to the ischaemic myocardium and reduce the workload of the heart. Glyceryl trinitrate is given by sublingual tablet or buccal spray. If chest pain is unrelieved after three doses 5 minutes apart, an intravenous glyceryl trinitrate infusion is initiated. The infusion may be continued until the chest pain is relieved or for 12 to 24 hours. Topical or oral nitrates are then initiated. Beta-adrenergic blockers are initially given intravenously, followed by oral beta-blockers. See the 'Medication administration' box above for the nursing implications of these drugs.

Aspirin, other antiplatelet drugs and heparin are given to inhibit blood clotting and reduce the risk of thrombus formation. Aspirin and clopidogrel are given to people with ACS who do not have an excessive bleeding risk. Aspirin and clopidogrel suppress platelet aggregation, interrupting the process of forming a stable blood clot. Both increase the risk of serious haemorrhage; for most people, however, the benefit outweighs the risk. Intravenous antiplatelet drugs such as abciximab, eptifibatide or tirofiban may be used when an invasive coronary revascularisation procedure is anticipated in the immediate or near future. Nursing implications for the antiplatelet drugs are outlined in the 'Medication administration' box below.

Revascularisation procedures

Several procedures may be used to restore blood flow and oxygen to ischaemic tissue. Non-surgical techniques include transluminal coronary angioplasty, laser angioplasty, coronary atherectomy and intracoronary stents. Coronary artery bypass grafting (CABG) is a surgical procedure that may be used.

PERCUTANEOUS TRANSLUMINAL CORONARY REVASCULARISATION

Percutaneous transluminal coronary revascularisation (PTCA) procedures are used to restore blood flow to the ischaemic myocardium in the person with CHD. There are over 37 600 percutaneous coronary interventions a year performed in Australia (AIHW, 2015b). PTCA is used to treat the person with:

- moderately severe, chronic stable angina unrelieved by medical therapy
- unstable angina
- acute myocardial infarction
- significant stenosis of the left anterior descending coronary artery
- stenosis of a coronary artery bypass graft.

PTCA procedures are similar to the procedure used for coronary angiography. A catheter introduced into the arterial circulation is guided into the opening of the narrowed coronary artery. A flexible guide wire is inserted through the catheter lumen into the affected vessel. The guide wire is then used to thread an angioplasty balloon, arterial stent or other therapeutic device into the narrowed segment of the artery (see Figure 29.2). The procedure is performed in the cardiac catheterisation laboratory using local anaesthesia. The hospital stay is short (1 to 2 days), minimising costs.

Intracoronary stents are metallic scaffolds used to maintain an open arterial lumen. Stents are now used in the majority of all PTCA procedures. Some stents are drug-eluting and release chemicals known to reduce restenosis (Shurmur, 2014). The stent is placed over a balloon catheter, guided into position and expanded

MEDICATION ADMINISTRATION Antiplatelet drugs

ORAL ANTIPLATELET DRUGS

Antiplatelet drugs suppress platelet aggregation in arteries, preventing the development of an arterial thrombus. Aspirin and clopidogrel block different platelet activation pathways to inhibit platelet aggregation and clot formation. The dose of aspirin given to achieve antiplatelet effects is low, 100 mg/day.

Nursing responsibilities

- Inquire about a history of intracranial haemorrhage, upper gastrointestinal bleeding, peptic ulcer disease or known bleeding tendency.
- Observe for and report increased bruising, petechiae, purpura, apparent or occult bleeding (e.g. melaena, haematemesis).
- Do not administer concurrently with warfarin.

Health education for the person and family

- Take as directed. Take aspirin with food or milk; clopidogrel may be taken at any time of day.
- Do not use non-steroidal anti-inflammatory drugs (NSAIDs) or other over-the-counter drugs that may contain aspirin or an NSAID unless prescribed by the doctor.
- Check with your doctor before taking any herbal remedies such as evening primrose oil, feverfew, garlic, ginkgo biloba or grapeseed extract while taking these medications.
- Report unusual bruising or excessive bleeding.
- Inform all care providers (including dental professionals) of use of these drugs.

INTRAVENOUS ANTIPLATELET DRUGS

The intravenously administered antiplatelet drugs, abciximab, epifibatide and tirofiban, block the final common pathway of platelet activation and thus are more effective. However, the risk of bleeding is greater than with the orally administered antiplatelet drugs.

Nursing responsibilities

- Determine history of bleeding disorders, intracranial haemorrhage, recent trauma or surgery.
- Inquire about recent use of oral antiplatelet or anticoagulant drugs.
- Monitor FBC, including haemoglobin, haematocrit and platelet count; clotting studies, including prothrombin time (PT), International Normalized Ratio (INR), activated partial thromboplastin time (aPTT); vital signs; and ECG during therapy.
- Maintain a separate intravenous line for blood draws and administration of other drugs during infusion.
- Closely observe for and immediately report anaphylaxis or bleeding uncontrolled by pressure. Keep resuscitation equipment readily available.
- Maintain bed rest during infusion.

Health education for the person and family

- This drug is given to reduce the risk of clotting and myocardial infarction. It helps maintain blood flow through the affected vessel following angioplasty and stent placement.
- Immediately report any chest tightness, difficulty breathing, shortness of breath or itching that develops during the infusion.
- The risk of bleeding should return to normal within about 2 days following the infusion.
- Immediately report any unusual bruising or bleeding.

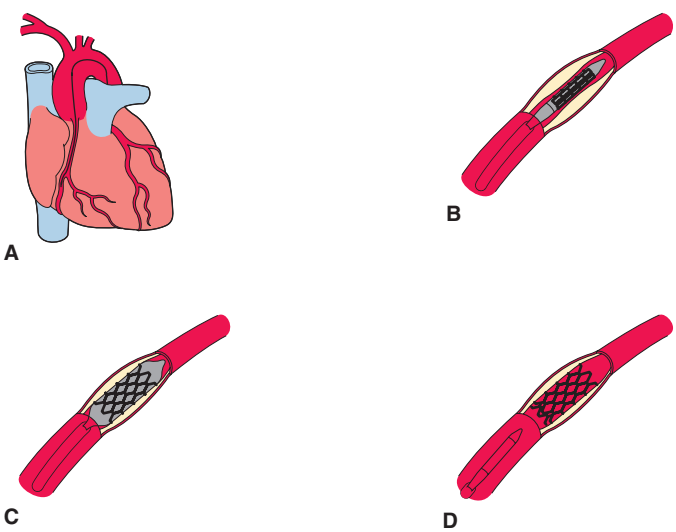


FIGURE 29.2 ■ Percutaneous coronary revascularisation. *A*, The balloon catheter with the stent is threaded into the affected coronary artery. *B*, *C*, The stent is positioned across the blockage and expanded. *D*, The balloon is deflated and removed, leaving the stent in place

as the balloon is inflated. It then remains in the artery as a prop after the balloon is removed. Endothelial cells will completely line the inner wall of the stent to produce a smooth inner lining. Antiplatelet medications (aspirin and ticlopidine) are given following stent insertion to reduce the risk of thrombus formation at the site.

In contrast to stent procedures, which enlarge the artery by displacing plaque, atherectomy procedures remove plaque from the identified lesion. The directional atherectomy catheter shaves the plaque off vessel walls using a rotary cutting head, retaining the fragments in its housing and removing them from the vessel. Rotational atherectomy catheters pulverise plaque into particles small enough to pass through the coronary microcirculation. Laser atherectomy devices use laser energy to remove plaque.

Complications following PTCA procedures include haematoma at the catheter insertion site, pseudoaneurysm, embolism, hypersensitivity to contrast dye, arrhythmias, bleeding and vessel perforation. Other complications include restenosis or reocclusion of the treated vessel.

Nursing care of the person undergoing PTCA is outlined in the box below.

CORONARY ARTERY BYPASS GRAFTING Surgery for coronary heart disease involves using a section of a vein or an

NURSING CARE OF THE PERSON having PTCA

BEFORE THE PROCEDURE

- Assess knowledge of the procedure and expectations of treatment. *This allows information to be tailored to the person's needs and provides an opportunity to clarify misconceptions.*
- Describe the cardiac catheterisation laboratory and the planned PTCA procedure, including:
  - preoperative preparation (see Chapter 3)
  - planned anaesthesia or sedation to be used
  - drugs that may be given during the procedure, such as anticoagulants to reduce the risk of thrombus formation; intravenous glyceryl trinitrate; and a calcium channel blocker to dilate coronary arteries and prevent anginal pain.
- Discuss possible sensations during the procedure, including flushing or warmth and a metallic taste in the mouth as the contrast dye is injected, and a feeling of pressure or chest pain during balloon inflation. *Advanced preparation for expected sensations reduces anxiety and improves outcomes.*
- Perform a comprehensive assessment, including hydration status (skin and mucous membrane moisture, turgor) and peripheral circulation (colour, warmth, sensation, pulses and capillary refill).

AFTER THE PROCEDURE

- Complete a head-to-toe assessment. Note any complaints of chest pain or evidence of decreased cardiac output or myocardial infarction. *Assessment provides a baseline for subsequent assessments and allows early identification of possible complications.*
- Monitor vital signs and cardiac rhythm continuously. Treat arrhythmias as ordered. Obtain a 12-lead ECG if signs of ischaemia develop and notify doctor. *Vital signs reflect cardiac output. Arrhythmias may develop with reperfusion of the ischaemic myocardium. ECG changes may indicate infarction or restenosis of the affected vessel.*
- Maintain intravenous glyceryl trinitrate infusion. Administer anticoagulant and antiplatelet medications, nitrates and calcium channel blockers as ordered. *These drugs decrease oxygen demand and increase oxygen supply by dilating the coronary arteries and systemic vasculature. They also reduce the risk of thrombus formation.*
- Monitor for and treat or report chest pain as indicated. *Chest pain may indicate ischaemia and possible myocardial infarction.*
- Maintain bed rest as ordered with the head of the bed at 30 degrees or less. Prevent flexion of the leg on the affected side. Following sheath removal, follow protocol for pressure dressing or device or sandbag placement. *A large puncture wound occurs at the insertion site. Immobilisation allows the wound to seal; a pressure dressing helps prevent bleeding.*
- Monitor distal pulses, colour, movement, sensation and temperature of the affected leg and insertion site every 15 minutes for the first hour, every 30 minutes for the next hour, every hour for the next 8 hours, then every 4 hours. A clot may form at the site, reducing perfusion of the affected leg. The site and dressing are monitored for excessive bleeding, haematoma formation or pseudoaneurysm. *Pseudoaneurysm occurs as a result of inadequate haemostasis after catheter removal.*
- Monitor intake and output, serum electrolytes, urea, creatinine, full blood count (FBC), activated partial thromboplastin time (aPTT) and cardiac enzymes. Report abnormal results to the medical officer. *Contrast dye causes osmotic diuresis and may cause kidney damage or a hypersensitivity reaction. Electrolyte imbalances increase the risk of arrhythmias. Cardiac enzymes are monitored for indications of possible myocardial damage during the procedure. The aPTT monitors the effectiveness of heparin therapy.*
- Monitor for bradycardia, light headedness, hypotension, diaphoresis and loss of consciousness during sheath removal. Keep atropine at bedside during sheath removal. Bradycardia and signs of decreased cardiac output may occur during sheath removal because of a vasovagal reaction. *Atropine decreases vagal tone and increases heart rate.*

artery to create a connection (or bypass) between the aorta and the coronary artery beyond the obstruction (see Figure 29.3). This then allows blood to perfuse the ischaemic portion of the heart. The internal mammary artery in the chest and the saphenous vein from the leg are the vessels most commonly used for CABG.

Bypass grafts are safe and effective. Angina is totally relieved or significantly reduced in 90% of people who undergo complete revascularisation. While anginal pain may recur within 3 years, it rarely is as severe as before surgery. Coronary artery bypass graft has a positive effect on mortality in many cases. It is recommended for people who have multiple vessel disease and impaired left ventricular function or diabetes, and for people who have significant obstruction of the left main coronary artery (Kalyanasundaram, 2014).

A median sternotomy commonly is used to access the heart. The heart is usually stopped during surgery. The *cardiopulmonary bypass (CPB) pump* is used to maintain perfusion to the rest of the organs during open-heart surgery. Venous blood is removed from the body through a cannula placed in the right atrium or the superior and inferior venae cavae. Blood then circulates through the CPB pump, where it is oxygenated, its temperature is regulated and it is filtered. Oxygenated blood is returned to the body through a cannula in the ascending aorta (see Figure 29.4). Cardiopulmonary bypass enables surgeons to operate on a still heart and a relatively bloodless field. Hypothermia can be maintained to reduce the metabolic rate and decrease oxygen demand during surgery.

Newer techniques have been developed that allow surgeons to perform CABG without cardioplegia (stopping the heart) and CPB. Off-pump coronary artery bypass (OPCAB) allows use of a smaller incision for access. Although cardiopulmonary bypass is employed for the majority of coronary artery bypass procedures, OPCAB is a promising alternative. OPCABs reduce mortality and morbidity rates and result in faster recovery for

the person undergoing OPCAB as compared to CABG with cardiopulmonary bypass (Kalyanasundaram, 2014).

Even more recently, a technique known as robotic CABG has emerged. The surgeon uses a computer to manipulate robotic arms to undertake surgery on anterior vessels in a more endoscopic approach (Kalyanasundaram, 2014). Early evidence is suggesting that this intervention may become a valuable and less traumatic option for selected individuals.

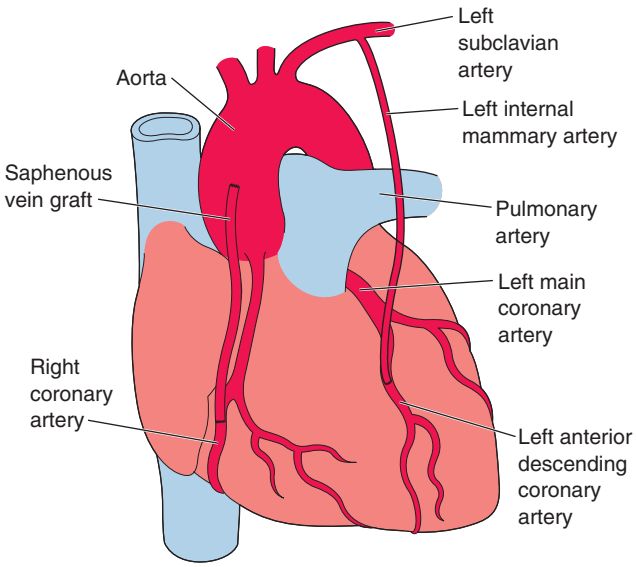


FIGURE 29.3 ■ Coronary artery bypass grafting using the internal mammary artery and a saphenous vein graft

When the saphenous vein is used, it is excised from its normal attachments in the leg and flushed with a cold heparinised saline solution. It is then reversed so that its valves do not interfere with blood flow. When appropriate, a laparoscopic approach may be used to remove the vein. The vein is *anastomosed* (grafted) to the aorta and the coronary artery, distal to the occlusion (see Figure 29.3). This provides a bridge or conduit for blood flow past the obstruction. If the internal mammary artery (IMA) is used, its distal end is excised and anastomosed to the coronary artery distal to the obstruction. The IMA often is used to revascularise the left coronary artery because of the greater oxygen demand of the left ventricle.

Once grafting is completed, cardiopulmonary bypass is discontinued and the person is rewarmed. Rewarming stimulates the heart to resume beating. Temporary pacing wires are sutured in place and passed through the chest wall in case temporary pacing is necessary. Chest tubes are placed in the pleural space and mediastinum to drain blood and re-establish negative pressure in the thoracic cavity, and to and drain bleeding as a result of the operation. The sternum is closed using heavy wires and bone wax, the skin is closed with sutures or staples, and sterile dressings are applied over sternal and leg incisions.

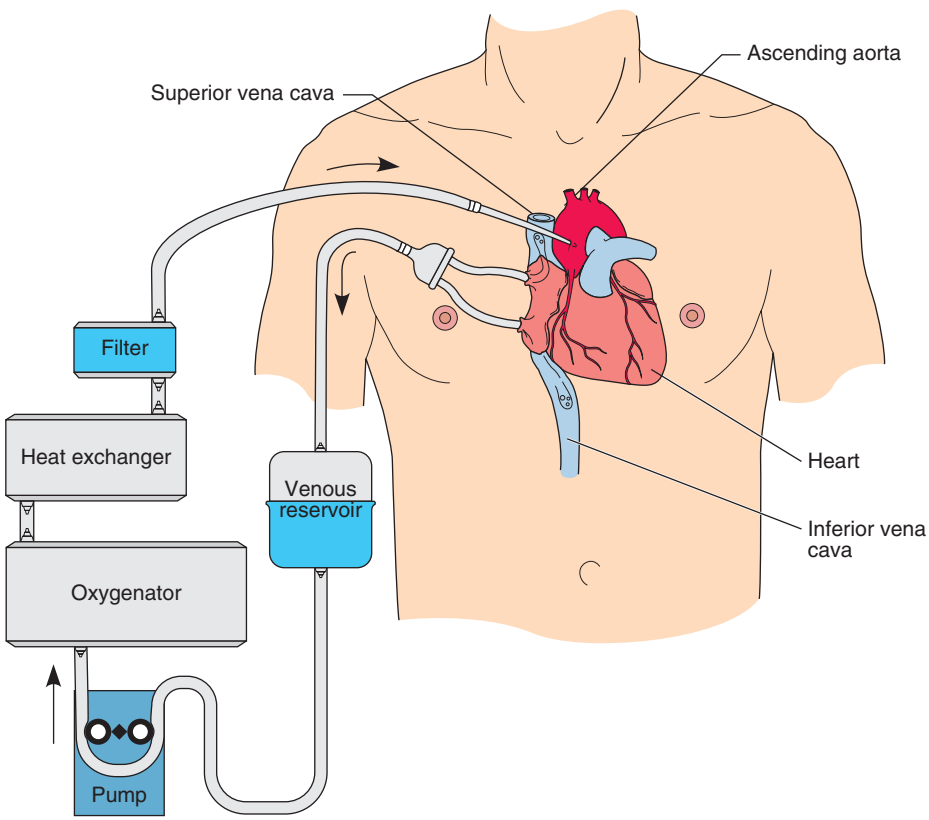


FIGURE 29.4 ■ A diagrammatic representation of cardiopulmonary bypass. A cannula in the superior and inferior venae cavae removes venous blood, which is then pumped through an oxygenator and heat exchanger. After filtering, oxygenated blood is returned to the ascending aorta

Pre- and postoperative nursing care and teaching for the person having a coronary artery bypass graft or other open-heart surgery are outlined in the following 'Nursing care' box.

MINIMALLY INVASIVE CORONARY ARTERY SURGERY

Minimally invasive coronary artery surgery is a potential future alternative to CABG. Two approaches may be used. *Port-access coronary artery bypass* uses several small holes, or 'ports', in the chest wall to access vessels for connection to the CPB pump and the surgical site. Alternatively, the femoral artery and femoral vein may be used for CPB. CPB is avoided altogether using the *minimally invasive direct coronary artery bypass (MIDCAB)* approach. With MIDCAB, a small surgical incision and several chest wall ports are used to graft a chest wall artery to the affected coronary vessel while the heart continues to beat. Beta-blockers are often used to reduce the heart rate to reduce movement in the surgical site.

TRANSMYOCARDIAL LASER REVASCULARISATION

A new development in myocardial revascularisation techniques is called *transmyocardial laser revascularisation (TMLR)*. In this procedure, a laser is used to drill tiny holes into the myocardial muscle itself to provide collateral blood flow to ischaemic muscle. Individuals whose coronary artery obstructions are too diffuse to bypass are candidates for this new surgical treatment.

NURSING CARE OF THE PERSON having coronary artery bypass grafts

PREOPERATIVE CARE

- Provide routine preoperative care and teaching as outlined in Chapter 3.
- Verify presence of laboratory and diagnostic test results in the chart, including FBC, coagulation profile, urinalysis, chest x-ray and coronary angiogram. *These baseline data are important for comparison of postoperative results and values.*
- Type and crossmatch four or more units of blood as ordered. *Blood is made available for use during and after surgery as needed.*
- Provide the person and their family with specific teaching related to procedure and postoperative care. Include the following topics:
  - cardiac recovery unit; sensory stimuli, personnel; noise and alarms; visiting policies
  - tubes, drains and general appearance
  - monitoring equipment, including cardiac and haemodynamic monitoring systems
  - respiratory support: ventilator, endotracheal tube, suctioning; communication while intubated
  - incisions and dressings
  - pain management.

  Preoperative teaching reduces anxiety and prepares the person and family for the postoperative environment and expected sensations.

POSTOPERATIVE CARE

- Provide routine postoperative care as outlined in Chapter 3. In addition to the care needs of all individuals having major surgery, the person having cardiac surgery has specific care needs related to open-heart and thoracic surgery. These are outlined under the nursing diagnoses identified below.

Decreased cardiac output

Cardiac output may be compromised postoperatively due to bleeding and fluid loss; depression of myocardial function by drugs, hypothermia and surgical manipulation; arrhythmias; increased vascular resistance; and a potential complication, *cardiac tamponade*, compression of the heart due to collected blood or fluid in the pericardium.

- Monitor vital signs, oxygen saturation and haemodynamic parameters every 15 minutes. Note trends and report significant changes to the doctor. *Initial hypothermia and bradycardia are expected; the heart rate should return to the normal range with rewarming. The blood pressure may fall during rewarming as peripheral vasodilation occurs. Hypotension and tachycardia, however, may indicate low cardiac output. Pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP), cardiac output and oxygen saturation are often monitored to evaluate fluid volume, cardiac function and gas exchange.* Haemodynamic monitoring is further discussed in Chapter 30.
- Auscultate heart and breath sounds on admission and at least every 4 hours. *A ventricular gallop, or S_3, is an early sign of heart failure; an S_4 may indicate decreased ventricular compliance. Muffled heart sounds may be an early indication of cardiac tamponade. Adventitious breath sounds (wheezes or rales—crackles) may be a manifestation of heart failure or respiratory compromise.*
- Assess skin colour and temperature, peripheral pulses and level of consciousness with vital signs. *Pale, mottled or cyanotic colouring, cool and clammy skin, and diminished pulse amplitude are indicators of decreased cardiac output.*
- Continuously monitor and document cardiac rhythm. *Arrhythmias are common and may interfere with cardiac filling and contractility, decreasing the cardiac output.*
- Measure intake and output hourly. Report urine output less than 30 mL/h for 2 consecutive hours. *Intake and output measurements help evaluate fluid volume status. A fall in urine output may be an early indicator of decreased cardiac output.*
- Record chest tube output hourly. *Chest tube drainage greater than 70 mL/h or that is warm, bright red and free flowing indicates haemorrhage and may necessitate a return to surgery. A sudden drop in chest tube output may indicate impending cardiac tamponade.* Monitoring for the presence of pulsus paradoxis and pulsus alternans should occur.
- Monitor haemoglobin, haematocrit and serum electrolytes. *A drop in haemoglobin and haematocrit may indicate haemorrhage that is not otherwise obvious. Electrolyte imbalances—potassium, calcium and magnesium, in particular—affect cardiac rhythm and contractility.*
- Administer intravenous fluids, fluid boluses and blood transfusions as ordered. *Fluid and blood replacement helps ensure adequate blood volume and oxygen-carrying capacity.*

(continued)

NURSING CARE OF THE PERSON having coronary artery bypass grafts (continued)

- Administer medications as ordered. *Medications ordered in the early postoperative period to maintain the cardiac output include inotropic drugs (e.g. dopamine, dobutamine) to increase the force of myocardial contractions; vasodilators (e.g. nitroprusside or glyceryl trinitrate) to decrease vascular resistance and afterload; and antiarrhythmic agents to correct arrhythmias that affect cardiac output.*
- Keep a temporary pacemaker at the bedside; initiate pacing as indicated. *Temporary pacing may be needed to maintain the cardiac output with bradyarrhythmias, such as high-level AV blocks.*

CONSIDERATION FOR PRACTICE

Assess for signs of cardiac tamponade: increased heart rate, decreased BP, decreased urine output, increased central venous pressure, a sudden decrease in chest tube output, muffled/distant heart sounds and diminished peripheral pulses. Notify medical officer immediately. Cardiac tamponade is a life-threatening complication that may develop postoperatively. Cardiac tamponade interferes with ventricular filling and contraction, decreasing cardiac output. Untreated, cardiac tamponade leads to cardiogenic shock and possible cardiac arrest.

Hypothermia

Hypothermia is maintained during cardiac surgery to reduce the metabolic rate and protect vital organs from ischaemic damage. Although rewarming is instituted on completion of the surgery, the person often remains hypothermic on admission to cardiac recovery. Gradual rewarming is necessary to prevent profound and rapid peripheral vasodilation and hypotension.

- Monitor core body temperature for the first 8 hours following surgery. Often temperature probes will be put in beside the nasogastric tubes. *Oral temperature measurements are not reliable indicators of core body temperature during this period. Tympanic temperature monitoring is indicated.*
- Institute rewarming measures (e.g. warmed intravenous solutions or blood transfusion, warm blankets, warm inspired gases, radiant heat lamps) as needed to maintain a temperature above 36°C. Administer pethidine to relieve shivering (as ordered). *Low body temperature may cause shivering, increasing oxygen demand. Hypothermia also increases the risk of hypoxia, metabolic acidosis, vasoconstriction and increased cardiac work, altered clotting and arrhythmias. If shivering cannot be controlled with narcotic agents, paralysing agents may be needed if the individual's haemodynamic stability is compromised. This intervention is not desirable, but is occasionally necessary.*

Acute pain

Following a CABG, pain is experienced due to both the thoracic incision and removal of the saphenous vein from the leg. Dissection of the internal mammary artery (usually the left IMA) from the chest wall also causes chest pain on the affected side. Chest tube sites are also uncomfortable. The leg from which the saphenous vein graft was obtained may be more painful than the chest incision.

- Frequently assess for pain, including its location and character. Document its intensity using a standard pain scale. Assess for verbal and non-verbal indicators of pain. Validate pain cues with the individual. *Pain is subjective and differs among individuals. Incisional pain is expected; however, anginal pain also may develop. It is important to differentiate the type of pain.*
- Administer analgesics on a scheduled basis, by patient-controlled analgesia (PCA), or by continuous infusion for the first 24 to 48 hours. *Research demonstrates that adequate pain management in the immediate postoperative period reduces complications from sympathetic stimulation and allows faster recovery. Pain causes muscle tension and vasoconstriction, impairing circulation and tissue perfusion, slowing wound healing and increasing cardiac work.*
- Premedicate 30 minutes before activities or planned procedures. *Premedication and the subsequent reduction of pain improve the person's participation and cooperation with care.*

CONSIDERATION FOR PRACTICE

Promptly report anginal or cardiac pain. Cardiac pain may indicate a perioperative or postoperative myocardial infarction.

Ineffective airway clearance/Impaired gas exchange

Atelectasis due to impaired ventilation and airway clearance is a common pulmonary complication of cardiac surgery. Gas exchange may also be affected by blood loss and decreased oxygen-carrying capacity following surgery. Phrenic nerve paralysis is a potential complication of cardiac surgery which may also contribute to impaired ventilation and gas exchange.

- Evaluate respiratory rate, depth, effort, symmetry of chest expansion and breath sounds frequently. *Pain, anxiety, excess fluid volume, surgical injury, narcotics and anaesthesia, and altered homeostasis can affect respiratory rate, depth and effort postoperatively. Decreased chest expansion or asymmetrical movement may indicate impaired ventilation of one lung and needs further evaluation.*
- Note endotracheal tube (ETT) placement on chest x-ray. Mark tube position and secure in place. *The chest x-ray documents correct ETT placement above the carina (bifurcation to the right and left mainstem bronchus). Marking its appropriate placement allows evaluation of potential tube movement. Secure the tube firmly in place to prevent slippage or inadvertent removal.*
- Maintain ventilator settings as ordered. Monitor arterial blood gases (ABGs) as ordered. *Mechanical ventilation promotes optimal lung expansion and oxygenation postoperatively. ABGs are used to evaluate oxygenation and acid–base balance.*
- Suction as needed. *Suctioning is performed only as indicated to clear airway secretions.*
- Prepare for ventilator weaning and extubation, as appropriate. *The individual is removed from the ventilator and*

NURSING CARE OF THE PERSON having coronary artery bypass grafts (continued)

extubated as soon as possible to reduce complications associated with mechanical ventilation and intubation.

- After extubation, teach use of the incentive spirometer and encourage use every 2 hours. Encourage deep breathing; advise against vigorous coughing. Teach use of a 'cough pillow' to splint chest incision and decrease pain. Assist in frequent pressure area care and encourage movement. *Deep breathing, controlled coughing and position changes improve ventilation and airway clearance and help prevent complications. Vigorous coughing may excessively increase intrathoracic pressure and cause sternal instability.*

Risk of infection

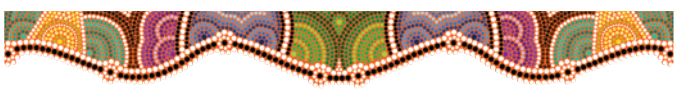
Following an open chest procedure, a sternal infection may develop that can progress to involve the mediastinum. Incisions for removal of the saphenous vein also may become infected. People with IMA grafts who are diabetic, older or malnourished are at high risk: harvesting of IMA disrupts blood supply to the sternum, and these individuals have impaired immune responses and healing.

- Assess sternal incision and leg wounds every shift. Document redness, warmth, swelling and/or drainage from the site. Note wound approximation. *These assessments provide indicators of inflammation and healing.*
- Depending on the type of dressing applied in theatre, it might be necessary to maintain a sterile dressing for the first 48 hours and then leave the incision open to air. Use Steri-Strips® as needed to maintain approximation of the wound edges. Alternatively, an occlusive dressing may be left in situ longer. *Care to reduce the risk of wound infection is paramount.*
- Report signs of wound infection: a swollen, erythematous area that is hot and painful to the touch; drainage from the wound; impaired healing or healed areas that reopen. *Evidence of infection or impaired healing requires further evaluation and treatment. Individuals with a subclinical infection will often report greater pain from the surgical wound.*
- Culture wound drainage as indicated. *Identifying the infective organism facilitates appropriate antibiotic therapy.*
- Collaborate with the dietitian to promote nutrition and fluid intake. *Good nutritional status is vital to healing and immune function.*

Disturbed thought processes

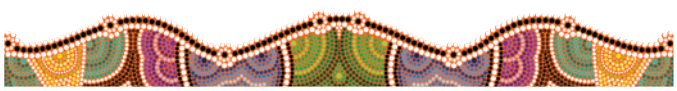
Many factors affect neuropsychological function after CABG, including the length of cardiopulmonary bypass, age, presurgery organic brain dysfunction, severity of illness and decreased cardiac output. Sensory overload and deprivation, sleep disruption and numerous drugs also affect thinking and mental clarity.

- Frequently reorient during initial recovery period. State that surgery is over and that the person is in the recovery area. *Frequent reorientation provides emotional support and reality checks.*
- Explain all procedures before performing them. Speak in a clear, calm voice. Encourage questions and give honest answers. *These measures provide information, decrease anxiety and establish trust.*
- Secure all intravenous lines and invasive catheters/tubes (e.g. ETT, urinary catheter, nasogastric tube). *Disoriented individuals may tug or pull at invasive equipment, disrupting them and increasing the risk of injury.*
- Note verbal responses to questions. Correct misconceptions immediately. *False beliefs or confused impressions regarding care may result in further confusion or acceptance of necessary care.*
- Orientate the individual to person, place and time (PPT). *This reduces confusion and assists the person to participate more in recovery.*
- Involve family members in providing reorientation. Place familiar objects and photographs within view. Encourage family presence. *The family provides reassurance and contact with the familiar, assisting with orientation.*
- Promote participation in their own care and decision making as appropriate. *This allows the person to maintain a degree of power and control, and enables the individual to take an active role in recovery.*
- Report signs of hallucinations, delusions, depression or agitation. *These may indicate progressive deterioration of mental status.*
- Administer sedatives cautiously. *Mild sedation may help prevent injury. Some sedatives may, however, have adverse effects, increasing confusion and disorientation.*
- Re-evaluate neurological status every shift. *These data allow evaluation of the effect of interventions.*



Nursing care

Health promotion, assessment, nursing diagnoses and interventions for the person with ACS are similar to those identified for people with angina and with acute myocardial infarction. See the preceding and subsequent sections of this chapter for specific nursing care activities, as well as the nursing care plan that follows.



THE PERSON WITH ACUTE MYOCARDIAL INFARCTION

An **acute myocardial infarction (AMI)**—necrosis (death) of myocardial cells—is a life-threatening event. If circulation to the affected myocardium is not promptly restored, loss of functional myocardium affects the heart's ability to maintain an effective cardiac output. This may ultimately lead to cardiogenic shock and death.

Many deaths from MI occur during the first few hours after symptoms begin. Heightening public awareness of the manifestations of MI, the importance of seeking immediate medical assistance and training in cardiopulmonary resuscitation (CPR) techniques are vital to decrease deaths due to MI.

NURSING CARE PLAN A person having coronary artery bypass surgery

Six weeks ago, John Clements, aged 50, was discharged from the hospital after emergency triple bypass surgery. Despite having emergency surgery, his postoperative recovery was uneventful and he was discharged 6 days after admission. He returns to the clinic for a postoperative stress test and to discuss his cardiac rehabilitation program.

ASSESSMENT

Mr Clements's medical history reveals significant CHD, an anterior wall myocardial infarction that led to his emergency triple bypass, and hyperlipidaemia. Current medications include diltiazem, isosorbide mononitrate, aspirin and a glyceryl nitrate patch. The ECG reveals sinus rhythm with some ST segment and T wave flattening, resting heart rate 68 and blood pressure 136/84.

Mr Clements has a strong family history of CHD. He does not smoke and uses alcohol occasionally in social situations. He enjoys takeaway fried chicken and watching television. Mr Clements states his only regular exercise used to be an evening of dancing with his wife and friends about once a month, 'But I get short of breath walking around the block now, so I guess I can't go dancing anymore!'

Mr Clements owns his own contracting business and states that he typically works about 50 to 60 hours per week. He doesn't know what the cardiac rehabilitation program is supposed to do for him. 'I have got to get back to work! You just can't sit around in my business—you have to make sure that the work is getting done on time and you have to check on supplies and equipment and the like. But I feel like a weakling—I need to get my energy back!'

DIAGNOSIS

- *Activity intolerance* related to general weakness and fatigue and manifested by inability to perform activities.
- *Ineffective role performance* related to health crisis manifested by inability to return to work.

PLANNING

- Define the purpose and components of a cardiac rehabilitation program.
- Enrol in 'cardiac rehabilitation' classes, including cardiac anatomy, physiology and coronary heart disease; exercise and activity prescriptions; lifestyle modifications, including diet counselling and stress management; emotional reactions to CAD; sexual activity; use of cardiac medications; and self-responsibility for health.
- Plan an exercise program based on stress test results, physical examination and interview.
- Encourage to schedule rest periods before and after activity/exercise.
- Review signs and symptoms of overexertion.
- Provide information about community resources for emotional and educational support.
- Assist to identify strategies for dealing with concerns about his business role.

Expected outcomes

- Verbalise an understanding of the definition and components of his structured cardiac rehabilitation program.
- Verbalise a desire to make lifestyle changes.
- Identify resources available in the community to assist with lifestyle changes.

IMPLEMENTATION

- Participate in his activity program without suffering any complications.
- Verbalise an increase in energy after 6 weeks on the program.
- Accept the reality of the temporary change in his usual work responsibilities.

EVALUATION

Mr Clements decides to 'give the rehab program a try'. Together, the cardiac rehabilitation team work with him to plan an individualised exercise/activity program. A dietitian provides dietary counselling. Stress management strategies are emphasised. Mr Clements is able to list manifestations of overexertion and states that he realises the need for gradual activity progression.

After 6 weeks, Mr Clements has reported a significant increase in energy and strength. 'I am feeling much stronger and have been sleeping better. Mary and I are taking evening walks around the neighbourhood. My chest soreness is also gone.' He has completed the 12-week cardiac rehabilitation program and another stress test indicates that his cardiac function is adequate. Mr Clements has joined the local National Heart Foundation 'Just Walk It' group and states that he is now incorporating 'heart-healthy' considerations into his daily routines.

CRITICAL THINKING IN THE NURSING PROCESS

1. Develop a personalised risk factor reduction plan for Mr Clements.
2. How might denial affect Mr Clements's ability to (a) accept the need for cardiac rehabilitation, (b) comply with the proposed lifestyle changes, and (c) make permanent adjustments to his daily life?
3. How does spousal support influence an individual's engagement with and adherence to a structured cardiac rehabilitation program?
4. Mr Clements tells you that since the surgery, his wife has been afraid that sexual activity will induce another heart attack. How would you respond to these concerns?

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Given the significance of cardiovascular function on exercise tolerance, is there a risk that Mr Clements may further damage his myocardium if demand outweighs supply? If so, what are the risks and how can they be avoided?
2. Outline what you have learned from this case study that you will apply to your clinical practice.

Myocardial infarction rarely occurs in people without pre-existing coronary heart disease. While no specific cause has been identified, the risk factors for MI are those for coronary heart disease: age, gender, heredity, race, smoking, obesity, hyperlipidaemia, hypertension, diabetes, sedentary lifestyle, diet and others. See the previous section of this chapter on coronary heart disease for further discussion of these risk factors.

Pathophysiology

Atherosclerotic plaque may form stable or unstable lesions. *Stable* lesions progress by gradually occluding the vessel lumen, whereas *unstable* (or *complicated*) lesions are prone to rupture and thrombus formation (Bullock & Hales, 2012). Stable lesions often cause angina (discussed previously); unstable lesions often lead to acute coronary syndromes or acute ischaemic heart diseases. Acute coronary syndromes include unstable angina, myocardial infarction and sudden cardiac death.

Myocardial infarction occurs when blood flow to a portion of cardiac muscle is completely blocked, resulting in prolonged tissue ischaemia and irreversible cell damage. Coronary occlusion is usually caused by ulceration or rupture of a complicated atherosclerotic lesion. When an atherosclerotic lesion ruptures or ulcerates, substances are released that stimulate platelet aggregation, thrombin generation and local vasomotor tone. As a result, the vessel constricts and a thrombus (clot) forms, occluding the vessel and interrupting blood flow to the myocardium distal to the obstruction.

Cellular injury occurs when the cells are denied adequate oxygen and nutrients. When ischaemia is prolonged, lasting more than 20 to 45 minutes, irreversible hypoxaemic damage causes cellular death and tissue necrosis. Oxygen, glycogen and ATP stores of ischaemic cells are rapidly depleted. Cellular metabolism shifts to an anaerobic process, producing hydrogen ions and lactic acid. Cellular acidosis increases cells' vulnerability to further damage. Intracellular enzymes are released through damaged cell membranes into interstitial spaces.

Cellular acidosis, electrolyte imbalances and hormones released in response to cellular ischaemia affect impulse conduction and myocardial contractility. The risk of arrhythmias increases and myocardial contractility decreases, reducing stroke volume, cardiac output, blood pressure and tissue perfusion.

The subendocardium suffers the initial damage, within 20 minutes of injury, because this area is the most susceptible to changes in coronary blood flow. If blood flow is restored at this point, the infarction is limited to subendocardial tissue (a *subendocardial* or *non-Q-wave infarction*). The damage progresses to the epicardium within 1 to 6 hours. When all layers of the myocardium are affected, it is known as a *transmural infarction*. A significant Q wave develops with a transmural infarction, so this may also be called a *Q-wave MI*. Complications such as heart failure are more frequently associated with Q-wave MIs; however, individuals with non-Q-wave MIs frequently experience recurrent ischaemia or subsequent MI within weeks or months of the event (Zafari, 2015).

The necrotic, infarcted tissue is surrounded by regions of injured and ischaemic tissues. Tissue in this ischaemic area is potentially viable; restoration of blood flow minimises the amount of tissue lost. This surrounding tissue also undergoes metabolic changes. It may be *stunned*, its contractility impaired for hours to days following reperfusion, or *hibernating*, a process that protects myocytes until perfusion is restored. *Myocardial remodelling* also may occur, with cellular hypertrophy and loss of contractility in regions distant from the infarction. Rapid restoration of blood flow limits these changes (Bullock & Hales, 2012).

When a larger artery is compromised, *collateral vessels* connecting smaller arteries in the coronary system dilate to maintain blood flow to the cardiac muscle. The degree of collateral circulation helps determine the extent of myocardial damage from ischaemia. Acute occlusion of a coronary artery without any collateral flow results in massive tissue damage and possible death. Progressive narrowing of the larger coronary arteries allows collateral vessels to develop and enlarge, meeting the demand for blood flow. Good collateral circulation can limit the size of an MI.

Myocardial infarctions are described by the damaged area of the heart. The coronary artery that is occluded determines the area of damage. Myocardial infarction usually affects the left ventricle because it is the major 'workhorse' of the heart; its muscle mass is greater, as are its oxygen demands. Occlusion of the left anterior descending (LAD) artery affects blood flow to the anterior wall of the left ventricle (an *anterior MI*) and part of the interventricular septum. Occlusion of the left circumflex artery (LCA) causes a lateral MI. *Right ventricular, inferior* and *posterior infarcts* involve occlusions of the right coronary artery (RCA) and posterior descending artery (PDA). Occlusion of the left main coronary artery is the most devastating, causing ischaemia of the entire left ventricle and a grave prognosis. Identifying the infarct site helps predict possible complications and determine appropriate therapy.

Cocaine-induced MI

Acute myocardial infarction may develop due to cocaine intoxication. Cocaine increases sympathetic nervous system activity by both increasing the release of catecholamines from central and peripheral stores and interfering with the reuptake of catecholamines. This increased catecholamine concentration stimulates the heart rate and increases its contractility, increases the automaticity of cardiac tissues and the risk of arrhythmias, and causes vasoconstriction and hypertension. The person with cocaine-induced MI may present with an altered level of consciousness, confusion and restlessness, seizure activity, tachycardia, hypotension, increased respiratory rate and respiratory crackles.

Manifestations

Pain is a classic manifestation of myocardial infarction. Chest pain due to MI is more severe than anginal pain. However, it is not the intensity of the chest pain that distinguishes MI from angina or acute coronary syndrome, but its duration and its continuous nature. The onset of pain is sudden and usually is not associated with activity. In fact, most MIs occur in the early morning. People with a history of angina may have more frequent anginal attacks in the days or weeks prior to an MI (unstable angina or ACS). Chest pain may be described as crushing and severe; as a pressure, heavy or squeezing sensation; or as chest tightness or burning. The pain often begins in the centre of the chest (*substernal*) and may radiate to the shoulders, neck, jaw or arms. It lasts more than 15 to 20 minutes and is not relieved by rest or glyceryl trinitrate.

Women and older adults often experience atypical chest pain, presenting with complaints of indigestion, heartburn, nausea and vomiting (see the box below). The greater diversity

MEETING INDIVIDUALISED NEEDS Recognising acute myocardial infarction in women and older adults

Women and older adults often present with atypical manifestations of MI. However, heart disease is the number one cause of death in both groups, making early recognition and aggressive treatment vital.

Women are more likely than men to have a 'silent' or unrecognised heart attack or to present in cardiac arrest or with cardiogenic shock. Women often experience epigastric pain and nausea, causing them to blame their discomfort on heartburn. Shortness of breath is common, as is fatigue and weakness of the shoulders and upper arms.

Older people often seek treatment for vague complaints of difficulty breathing, confusion, fainting, dizziness, abdominal pain or cough. They often attribute their symptoms to a stroke. The prevalence of silent ischaemia is greater in older adults.

Stress the importance of seeking medical help promptly for atypical manifestations of MI. Prompt diagnosis and intervention reduces the mortality and morbidity of MI in women and older adults, just as it does in men. Despite this fact, both women and older adults are more likely to delay seeking treatment and are less likely to be accurately diagnosed and aggressively treated for CHD. Women are almost three times more likely to die from heart disease than from breast cancer (National Heart Foundation, 2015).

of clinical manifestations experienced by women having a cardiovascular event decreases the likelihood of healthcare professionals recognising an event, and also reduces the likelihood of the woman seeking assistance (Lichtman et al., 2015).

Compensatory mechanisms cause many of the other symptoms of MI. Sympathetic nervous system stimulation causes anxiety, tachycardia and vasoconstriction. This results in cool, clammy, mottled skin. Pain and blood chemistry changes stimulate the respiratory centre, causing tachypnoea. The person often has a sense of impending doom and death. Tissue necrosis causes an inflammatory reaction that increases the white blood cell count and elevates the temperature. Serum cardiac enzyme levels rise as enzymes are released from necrotic myocytes.

Other manifestations may vary, depending on the location and amount of infarcted tissue. Hypertension, hypotension or signs of heart failure may develop. Vagal stimulation may cause nausea and vomiting, bradycardia and hypotension. Hiccupping may develop due to diaphragmatic irritation. If a large vessel is occluded, the first sign of MI may be sudden death. Typical manifestations of MI are listed in the box below.

Complications

The risk of complications associated with myocardial infarction is related to the size and location of the MI.

MANIFESTATIONS Acute myocardial infarction

- Chest pain: substernal or precordial (across the entire chest wall); may radiate to neck, jaw, shoulder(s) or left arm
- Tachycardia, tachypnoea
- Dyspnoea, shortness of breath
- Nausea and vomiting
- Anxiety, sense of impending doom
- Diaphoresis
- Cool, mottled skin; diminished peripheral pulses
- Hypotension or hypertension
- Palpitations, arrhythmias
- Signs of left heart failure
- Decreased level of consciousness

Arrhythmias

Arrhythmias, disturbances or irregularities of heart rhythm, are the most frequent complication of MI. Arrhythmias are discussed in detail in the next section of this chapter.

Infarcted tissue is *arrhythmogenic*; that is, it affects the generation and conduction of electrical impulses in the heart, increasing the risk of arrhythmias. Premature ventricular contractions (PVCs) are common following an MI, and may be predictive of more dangerous arrhythmias such as ventricular tachycardia or ventricular fibrillation (Zafari, 2015). The risk of ventricular fibrillation is greatest the first hour after MI; it is a frequent cause of sudden cardiac death associated with acute MI. Its incidence declines with time. If the infarct affects a conduction pathway, electrical conduction may be affected. Any degree of AV block may occur following MI, especially when the anterior wall is infarcted. First-degree and Mobitz I (Wenckebach) blocks are most common, although complete heart block may develop. Bradyarrhythmias (abnormal slow rhythms) also may develop, particularly when the inferior wall of the ventricle is affected.

Pump failure

Myocardial infarction reduces myocardial contractility, ventricular wall motion and compliance. Impaired contractility and filling may produce heart failure. The risk of heart failure is greatest when large portions of the left ventricle are infarcted. Heart failure may be more severe with an anterior infarction. Loss of 20–30% of the left ventricular muscle mass may cause manifestations of left-sided heart failure, including dyspnoea, fatigue, weakness and respiratory crackles on auscultation. Inferior or right ventricular MI may lead to right-sided heart failure with manifestations such as neck vein distension and peripheral oedema. Haemodynamic monitoring is often initiated for people with evidence of heart failure. Heart failure and its manifestations are discussed in greater depth in Chapter 30.

CARDIOGENIC SHOCK *Cardiogenic shock*, impaired tissue perfusion due to pump failure, results when functioning myocardial muscle mass decreases by more than 40%. The heart is unable to pump enough blood to meet the needs of the

body and maintain organ function. Low cardiac output due to cardiogenic shock also impairs perfusion of the coronary arteries and myocardium, further increasing tissue damage. Mortality from cardiogenic shock is greater than 70%, although this can be reduced by prompt intervention with revascularisation procedures (Ren, 2014). See Chapter 10 for a more extensive discussion of cardiogenic shock.

Infarct extension

Approximately 10% of people experience extension or reinfarction in the area of the original infarction during the first 10 to 14 days after an MI. *Extension* of the MI is characterised by increased myocardial necrosis from continued blood flow impairment and ongoing injury. *Expansion* of the MI is described as a permanent expansion of the infarcted area from thinning and dilation of the muscle. Infarct extension and expansion may cause manifestations such as continuing chest pain, haemodynamic compromise and worsening heart failure.

Structural defects

Necrotic muscle is replaced by scar tissue that is thinner than the ventricular muscle mass. This can lead to such complications as ventricular aneurysm, rupture of the interventricular septum or papillary muscle, and myocardial rupture. A *ventricular aneurysm* is a weakening and bulging of the ventricular wall. It may develop when a large section of the ventricle is replaced by scar tissue. Because it does not contract during systole, stroke volume decreases. Blood may pool within the aneurysm, causing clots to form. Ischaemia of the papillary muscle or chordae tendineae may cause structural damage leading to papillary muscle dysfunction or rupture. This affects AV valve function (usually the mitral valve), causing *regurgitation*, backflow of blood into the atria during systole. The interventricular septum may perforate or rupture due to ischaemia and infarction. Myocardial rupture is a risk between days 4 and 7 after MI, when the injured tissue is soft and weak. This potential complication of MI is often fatal.

Pericarditis

Tissue necrosis prompts an inflammatory response. *Pericarditis*, inflammation of the pericardial tissue surrounding the heart, may complicate AMI, usually within 2 to 3 days. Pericarditis causes chest pain that may be aching or sharp and stabbing, aggravated by movement or deep breathing. A *pericardial friction rub* may be heard on auscultation of heart sounds.

Dressler's syndrome, thought to be a hypersensitivity response to necrotic tissue or an autoimmune disorder, may develop days to weeks after AMI. It is a symptom complex characterised by fever, chest pain and dyspnoea. Dressler's syndrome may spontaneously resolve or recur over several months, causing significant discomfort and distress.

FAST FACTS

- Arrhythmias are the most common complication of AMI.
- Heart failure also is a common complication or consequence of myocardial infarction, developing due to loss of functional muscle tissue.

INTERPROFESSIONAL CARE

Immediate treatment goals for the person with an MI are to:

- relieve chest pain
- reduce the extent of myocardial damage
- maintain cardiovascular stability
- decrease cardiac workload
- prevent complications.

Slowing the process of coronary heart disease and reducing the risk of future MI is a major long-term management goal for the person.

Rapid assessment and early diagnosis is important in treating AMI. 'Time is muscle' is a medical truism for the person with AMI. The evolution of an AMI is dynamic: the quicker the artery is reopened (medically, surgically or spontaneously), the more myocardium can be salvaged. Survival and long-term outcomes following AMI are improved by rapidly restoring blood flow to the 'stunned' myocardium surrounding the infarcted tissue, reducing myocardial oxygen demand and limiting the accumulation of toxic by-products of necrosis and reperfusion (Herrmann, Kaski & Lerman, 2012).

The major problem interfering with timely reperfusion is delay in seeking medical care following the onset of symptoms. Up to 44% of people with symptoms of chest discomfort or pain wait more than 4 hours before seeking treatment. Many factors are cited as reasons for treatment delay, including advanced age, the perception of the seriousness of symptoms, denial, access to medical care, the availability of an emergency response system and in-hospital delays. Immediate evaluation of the person presenting with manifestations of myocardial infarction is essential to early diagnosis and treatment.

Diagnosis

Diagnostic testing is used to establish the diagnosis of AMI.

- *Serum cardiac markers* are proteins released from necrotic heart muscle. The proteins most specific for diagnosis of MI are the cardiac-specific troponins and creatine kinase (CK or creatine phosphokinase, CPK) (see Table 29.6).
- *Creatine kinase* is an important enzyme for cellular function, found principally in cardiac and skeletal muscle and the brain. CK levels rise rapidly with damage to these tissues, appearing in the serum 4 to 6 hours after AMI, peaking within 12 to 24 hours, and then declining over the next 48 to 72 hours. The CK level correlates with the size of the infarction; the greater the amount of infarcted tissue, the higher the serum CK level.
- *CK-MB* (also called MB-bands) is a subset of CK specific to cardiac muscle. This isoenzyme of CK is considered the most sensitive indicator of MI. Elevated CK alone is not specific for MI; elevated CK-MB greater than 5% is considered a positive indicator of MI. CK-MB levels do not normally rise with chest pain from angina or causes other than MI.
- Cardiac muscle troponins, *cardiac-specific troponin T* (cT_nT) and *cardiac-specific troponin I* (cT_nI), are proteins released during myocardial infarction that are sensitive indicators of myocardial damage. These proteins are part of the actin–myosin unit in cardiac muscle and normally are not detectable in the

blood. With necrosis of cardiac muscle, troponins are released and blood levels rise. The specificity of cT_nT and cT_nI to cardiac muscle necrosis makes these markers particularly useful when skeletal muscle trauma contributes to elevated CK levels (e.g. when CPR has been performed or traumatic injury occurred at the time of the MI). They are sensitive enough to detect very small infarctions that do not cause significant CK elevation. Both cT_nT and cT_nI remain in the blood for 10 to 14 days after an MI, making them useful to diagnose MI when medical treatment is delayed.

Serum levels of cardiac markers are ordered on admission and for 3 succeeding days. Serial blood levels help establish the diagnosis and determine the extent of myocardial damage.

Other laboratory tests may include the following:

- *Myoglobin* is one of the first cardiac markers to be detectable in the blood after an MI. It is released within 2–4 hours of symptom onset. Its lack of specificity to cardiac muscle and rapid excretion (blood levels return to normal within 24–36 hours) limit its use, however (Schreiber, 2011).
- *Full blood count (FBC)* shows an elevated white blood cell (WBC) count due to inflammation of the injured myocardium. The erythrocyte sedimentation rate (ESR) also rises because of inflammation, although ESR is a non-specific marker of inflammation.
- *Arterial blood gases (ABGs)* may be ordered to assess blood oxygen levels and acid–base balance.

Electrocardiography, echocardiography and myocardial nuclear scans are the most common diagnostic tests performed when AMI is suspected. With the exception of the ECG, the timing of these tests depends on the individual's immediate condition. Haemodynamic monitoring may be initiated in the unstable person following MI.

- The *electrocardiogram* reflects changes in conduction due to myocardial ischaemia and necrosis. Classic ECG changes seen in AMI include T-wave inversion, ST-segment elevation and formation of a Q wave. Ischaemic changes in the heart are seen as depression of the ST segment or inversion of the T wave (see Figure 29.1). With myocardial injury, elevation of the ST segment occurs (see Figure 29.5A). Significant Q-wave development (see Figure 29.5B) indicates a transmural or full-thickness infarction. Myocardial damage can be localised using the 12-lead ECG. See Chapter 28 for more information about ECGs.
- *Echocardiography* is done to evaluate cardiac wall motion and left ventricular function. Stunned and infarcted tissue does not contract as effectively (if at all) as healthy myocardium.
- *Radionuclide imaging* may be done to evaluate myocardial perfusion. These studies cannot differentiate between an acute MI and old scar tissue, but do help identify the specific area of myocardial ischaemia and damage.
- *Haemodynamic monitoring* may be initiated when AMI significantly affects cardiac output and haemodynamic status. These invasive procedures are described in Chapter 30.

Medications

Aspirin, a platelet inhibitor, is now considered an essential part of treating AMI. A 300 mg aspirin tablet is given by emergency personnel, with the instruction that it is to be chewed (for buccal absorption). This initial dose is followed by a daily oral dose of 100 mg of aspirin.

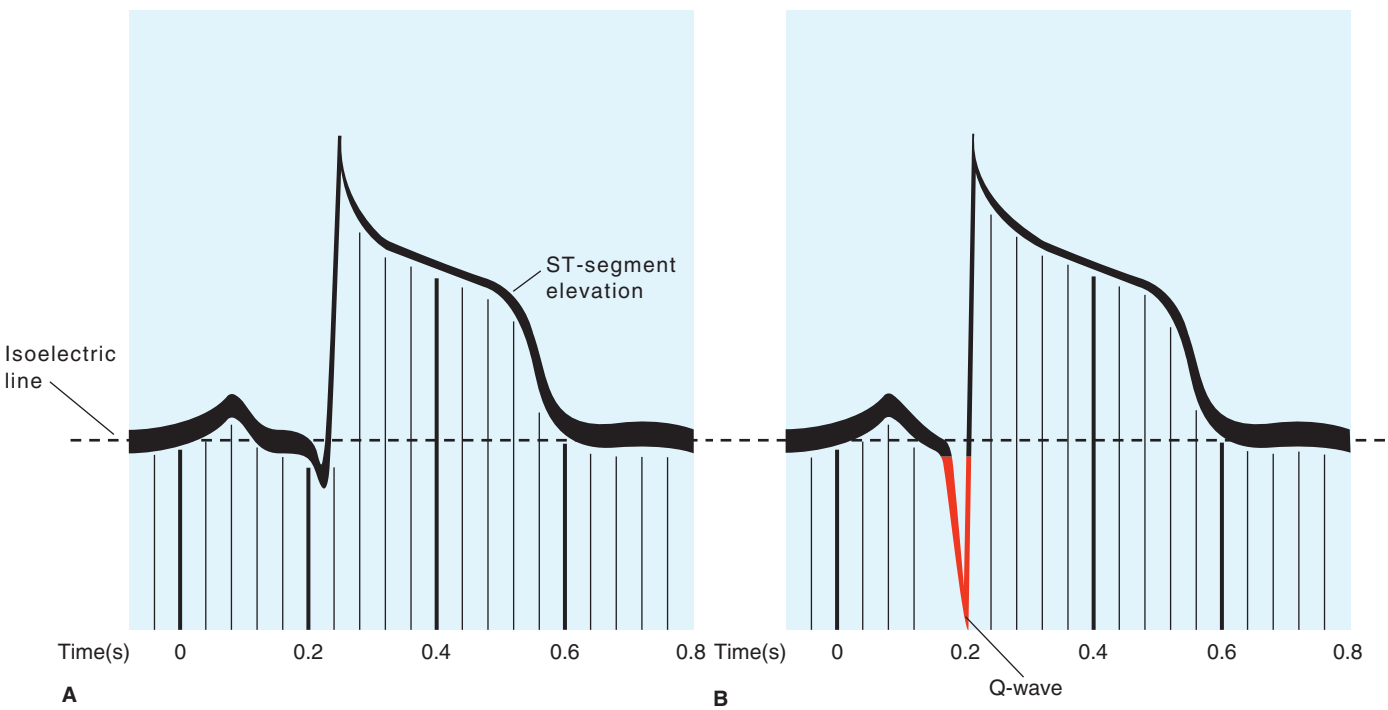


FIGURE 29.5 ■ ECG changes characteristic of MI. *A*, ST-segment elevation characteristic of myocardial injury. *B*, Clinically significant Q-wave characteristic of a transmural infarction

Thrombolytic agents, analgesics and antiarrhythmic agents are among the principal classes of drugs used in treating AMI.

ANALGESIA Pain relief is vital in managing the care of a person with AMI. Pain stimulates the sympathetic nervous system, increasing the heart rate and blood pressure and, in turn, myocardial workload. Sublingual glyceryl trinitrate may be given (up to three 0.4 mg doses at 5-minute intervals). Intravenous glyceryl trinitrate may be continued for the first 24 to 48 hours to reduce myocardial work. In addition to pain relief, glyceryl trinitrate decreases myocardial oxygen demand and may increase the supply of oxygen to the myocardium. Glyceryl trinitrate is a peripheral and arterial vasodilator that reduces afterload. It dilates coronary arteries and collateral channels in the heart, increasing coronary blood flow to save myocardial tissue at risk. Nitrates may, however, cause reflex tachycardia or excessive hypotension, so close monitoring is necessary during administration. It also is important to ask the person about use of sildenafil (Viagra) within the previous 24 hours before administering glyceryl trinitrate, as the combination can precipitate a significant drop in blood pressure. See the 'Medication administration' box on page 996 for the nursing implications of glyceryl trinitrate and other drugs given to reduce myocardial work following AMI.

Morphine sulfate is the drug of choice for pain unrelieved by glyceryl trinitrate and for sedation. Following an initial intravenous dose of 4 to 8 mg, small doses (2 to 4 mg) may be repeated intravenously every 5 minutes until pain is relieved. It is important to assess frequently for pain relief and possible adverse effects of analgesia, such as excessive sedation. Pain unrelieved by expected or usual doses should be reported to the doctor as it may indicate a complication such as extension of the infarct. See Chapter 8 for more details about morphine administration. Anxiolytic agents such as diazepam may also be administered to promote rest.

THROMBOLYTIC THERAPY Thrombolytic agents, drugs that dissolve or break up blood clots, are first-line drugs used to treat acute MI when access to a cardiac catheterisation lab for revascularisation procedures is not immediately available. Thrombolytic drugs activate the thrombolytic system to *lyse* or destroy the clot, restoring blood flow to the obstructed artery. Early thrombolytic administration (within the first 3 hours of MI onset) limits infarct size, reduces heart damage and improves outcomes. Activation of the thrombolytic system can cause multiple complications; approximately 0.5–5% of people receiving thrombolytic drugs experience serious bleeding complications. Not every person is a candidate for thrombolytic therapy; for example, it is contraindicated in people with known bleeding disorders, history of cerebrovascular disease, uncontrolled hypertension, pregnancy or recent trauma or surgery of the head or spine (Australian Resuscitation Council (ARC), 2011).

Several thrombolytic agents are commonly used today. Little difference in effectiveness has been demonstrated between these; there are, however, big differences in cost. Streptokinase, a biological agent derived from group C *Streptococcus* organisms, is the least expensive of the drugs. Its primary drawback is the risk of a severe hypersensitivity reaction, including anaphylaxis. Streptokinase is administered by intravenous infusion. Anisoylated plasminogen streptokinase activator complex (APSAC) is a related drug that can be administered by bolus over 2 to 5 minutes. It has many of the same effects as streptokinase, but is considerably more expensive. Tissue plasminogen activator (t-PA), tenecteplase (TNK) and reteplase (rPA) are more effective in re-establishing myocardial perfusion, especially when the pain developed more than 3 hours previously. These drugs, however, are the most expensive. Nursing care of the person receiving a thrombolytic agent is outlined in the following box.

ANTIARRHYTHMICS Arrhythmias are a common complication of AMI, particularly in the first 12 to 24 hours. Antiarrhythmic medications are used as needed to treat arrhythmias. They also may be given prophylactically to prevent arrhythmias. Ventricular arrhythmias are treated with a class I or class III antiarrhythmic drug (see the 'Medication administration' box on page 1029). Symptomatic bradycardia (bradycardia with associated hypotension and other signs of low cardiac output) is treated with intravenous atropine, 0.5 to 1 mg. Intravenous verapamil or the short-acting beta-blocker esmolol may be ordered to treat atrial fibrillation or other supraventricular tachyarrhythmias.

OTHER MEDICATIONS Beta-blockers such as propranolol, atenolol and metoprolol limit infarct size and decrease the incidence of serious ventricular arrhythmias in AMI. They may also reduce the risk of papillary muscle rupture. These drugs decrease the heart rate, reducing cardiac work and myocardial oxygen demand. Initial doses are given intravenously. Oral beta-blocker therapy is continued to reduce the risk of reinfarction and death related to cardiovascular causes (Bullock & Manias, 2013; Coven, 2015).

ACE inhibitors also reduce mortality associated with AMI. These drugs reduce ventricular remodelling following an MI, reducing the risk of subsequent heart failure. They also may reduce the risk of reinfarction (Bullock & Manias, 2013; Coven, 2015).

Anticoagulants and antiplatelet medications often are prescribed to maintain coronary artery patency following thrombolysis or a revascularisation procedure. Abciximab reduces platelet aggregation and the risk of reocclusion following angioplasty. It also improves vessel opening with thrombolytic therapy, permitting lower doses of thrombolytic drugs. Standard or low-molecular-weight heparin preparations often are given to person with AMI. Heparin helps establish and maintain patency of the affected coronary artery. It also is used, along with long-term warfarin, to prevent systemic or pulmonary embolism in people with significant left ventricular impairment or atrial fibrillation following AMI. See the 'Medication administration' box on page 1000 for the nursing implications of antiplatelet drugs and Chapter 32 for more information about anticoagulant therapy.

Individuals with pump failure and hypotension may receive intravenous dopamine, a vasopressor. At low doses

NURSING CARE OF THE PERSON receiving thrombolytic therapy

PRE-INFUSION CARE

- Obtain nursing history and perform a physical assessment. *Information obtained from the history and physical exam helps determine whether thrombolytic therapy is appropriate. The goal is to initiate thrombolytic therapy within 30 minutes of arrival.*
- Evaluate for contraindications to thrombolytic therapy: recent surgery or trauma (including prolonged CPR), bleeding disorders or active bleeding, cerebral vascular accident, neurosurgery within the last 2 months, gastrointestinal ulcers, diabetic haemorrhagic retinopathy and uncontrolled hypertension. *Thrombolytic agents dissolve clots and therefore may precipitate intracranial, internal or peripheral bleeding.*
- Inform the person of the purpose of the therapy. Discuss the risk of bleeding and the need to keep the extremity immobile during and after the infusion. *Minimal movement of the extremity is necessary to prevent bleeding from the infusion site.*

DURING THE INFUSION

- Assess and record vital signs and the infusion site for haematoma or bleeding every 15 minutes for the first hour, every 30 minutes for the next 2 hours and then hourly until the intravenous catheter is discontinued. Assess pulses, colour, sensation and temperature of both extremities with each vital sign check. *Vital signs and the site are frequently assessed to detect possible complications.*
- Remind the person to keep the extremity still and straight. Do not elevate head of bed above 15 degrees. *Extremity immobilisation helps prevent infusion site trauma and bleeding. Hypotension may develop; keeping the bed flat helps maintain cerebral perfusion.*
- Maintain continuous cardiac monitoring during the infusion. Keep antiarrhythmic drugs and the emergency trolley readily available for treatment of significant arrhythmias. *Ventricular arrhythmias commonly occur with reperfusion of the ischaemic myocardium.*

POST-INFUSION CARE

- Assess vital signs, distal pulses and infusion site frequently as needed. *The person remains at high risk of bleeding following thrombolytic therapy.*
- Evaluate response to therapy: normalisation of ST segment, relief of chest pain, reperfusion arrhythmias, early peaking of the CK and CK-MB. *These are signs that the clot has been dissolved and the myocardium is being reperfused.*
- Maintain bed rest for 6 hours. Keep the head of the bed at or below 15 degrees. Reinforce the need to keep the extremity straight and immobile. Avoid any injections for 24 hours after catheter removal. *Precautions such as these are important to prevent bleeding.*
- Assess puncture sites for bleeding. On catheter removal hold direct pressure over the site for at least 30 minutes. Apply a pressure dressing to any venous or arterial sites as needed. Perform routine care in a gentle manner to avoid bruising or injury. *Thrombolytic therapy disrupts normal coagulation. Peripheral bleeding may occur at puncture sites and there may not be sufficient fibrin to form a clot. Direct or indirect pressure may be needed to control the bleeding.*
- Assess body fluids, including urine, vomit and faeces, for evidence of bleeding; frequently assess for changes in level of consciousness and manifestations of increased intracranial pressure, which may indicate intracranial bleeding. Assess surgical sites for bleeding. Monitor haemoglobin and haematocrit levels, prothrombin time (PT) and activated partial thromboplastin time (aPTT). *These provide additional means of assessing for bleeding.*
- Administer platelet-modifying drugs (e.g. aspirin, dipyridamole) as ordered. *Platelet inhibitors decrease platelet aggregation and adhesion and are used to prevent reocclusion of the artery.*
- Report manifestations of reocclusion, including changes in the ST segment, chest pain or arrhythmias. *Early recognition of reocclusion is vital to save myocardial tissue.*

(less than 5 mg/kg/min), it improves blood flow to the kidneys, preventing renal ischaemia and possible acute kidney injury (see Chapter 27). With increasing doses, dopamine increases myocardial contractility and causes vasoconstriction, improving blood pressure and cardiac output.

Antihyperlipidaemic agents are used for the person with hyperlipidaemia. A stool softener such as docusate sodium is prescribed to maintain normal bowel function and reduce straining.

Treatments

The individual with a suspected or confirmed MI is monitored continuously. Care is provided in the intensive coronary care unit for the first 24 to 48 hours, after which time less intensive monitoring (e.g. telemetry) may be required. At least one intravenous line is established to allow rapid administration of emergency medications.

Bed rest is prescribed for the first 12 hours to reduce the cardiac workload. The bedside commode generally is allowed; studies have shown this to be less stressful than using a bedpan. If the person's condition is stable, sitting in a chair at the bedside is permitted after 12 hours. Activities are gradually increased as tolerated. A quiet, calm environment with limited outside stimuli is preferred. Visitors are limited to promote rest. Oxygen is administered by nasal prongs at 2 to 5 L/min to improve oxygenation of the myocardium and other tissues.

A liquid diet may be prescribed for the first 4 to 12 hours to reduce gastric distension and myocardial work. Following that, a low-fat, low-cholesterol, reduced-sodium diet is allowed. Sodium restrictions may be lifted after 2 to 3 days if no evidence of heart failure is present. Small, frequent feedings are often recommended. Drinks containing caffeine, and very hot and cold foods, may also be limited.

Revascularisation procedures

Many people with AMI are treated with immediate or early coronary revascularisation such as angioplasty and stent placement. Percutaneous transluminal coronary revascularisation (PCTA) may follow thrombolytic therapy or be used in place of thrombolytic therapy to restore blood flow to ischaemic myocardium. When compared with thrombolytic therapy, prompt PTCA reduces hospital mortality (Kalyanasundaram, 2014). In some cases, coronary artery bypass grafting (CABG) surgery may be performed. The choice of procedure depends on the person's age and immediate condition, the time elapsed from the onset of manifestations and the extent of myocardial disease and damage. These procedures and related nursing care are covered in more depth in the preceding section on acute coronary syndrome.

Other invasive procedures

For individuals with large MIs and evidence of pump failure, invasive devices may be used to temporarily take over the function of the heart, allowing the injured myocardium to heal. The intra-aortic balloon pump is widely used to augment cardiac output. Ventricular assist devices are indicated for people requiring more or longer-term artificial support than the intra-aortic balloon pump provides.

INTRA-AORTIC BALLOON PUMP The *intra-aortic balloon pump (IABP)*, also called intra-aortic balloon counterpulsation, is a mechanical circulatory support device that may be used after cardiac surgery or to treat cardiogenic shock following AMI. The IABP temporarily supports cardiac function, allowing the heart gradually to recover by decreasing myocardial workload and oxygen demand and increasing perfusion of the coronary arteries.

A catheter with a 30 to 40 mL balloon is introduced into the aorta, usually via the femoral artery. The balloon catheter is connected to a console that regulates the inflation and deflation of the balloon. The IABP catheter inflates during diastole, increasing perfusion of the coronary and renal arteries, and deflates just prior to systole, decreasing afterload and cardiac workload (see Figure 29.6). The inflation–deflation sequence is triggered by the ECG pattern. During the most acute period, the balloon inflates and deflates with each heartbeat (1:1 ratio), providing maximal assistance to the heart. As the person's condition improves, the IABP is weaned to inflate–deflate at varying intervals (e.g. 1:2, 1:4, 1:8). This provides a continually decreasing amount of support as the heart muscle recovers. When mechanical assistance is no longer required, the IABP catheter is removed.

VENTRICULAR ASSIST DEVICES Use of *ventricular assist devices (VADs)* to aid the failing heart is becoming more common with advances in technology. Whereas the IABP can supplement cardiac output by approximately 10–15%, the VAD temporarily takes partial or complete control of cardiac function, depending on the type of device used. VADs may be used as temporary or complete assist in AMI and cardiogenic shock when there is a chance for recovery of normal heart function after a period of cardiac rest. The device also may be used as a bridge in heart transplant. Nursing care for the person with a VAD is supportive and includes assessing haemodynamic status and for complications associated with the device. People with VADs *in situ* are at considerable risk of infection; strict aseptic technique is used with all invasive catheters and dressing changes. Pneumonia also is a risk due to immobility and ventilatory support. Mechanical failure of the VAD is a life-threatening event that requires immediate intervention (Kirkpatrick et al., 2015).

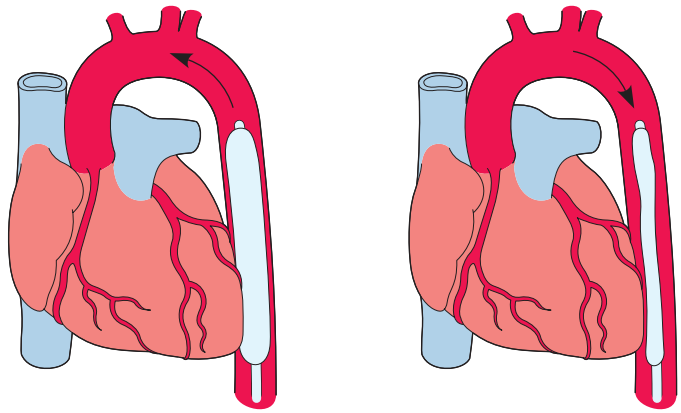


FIGURE 29.6 ■ The intra-aortic balloon pump. *A*, When inflated during diastole, the balloon supports cerebral, renal and coronary artery perfusion. *B*, The balloon deflates during systole, so cardiac output is unimpeded

Cardiac rehabilitation

Cardiac rehabilitation is a long-term program of medical evaluation, exercise, risk factor modification, education and counselling designed to limit the physical and psychological effects of cardiac illness and improve the person's quality of life. Cardiac rehabilitation begins with admission for a cardiac event such as AMI or a revascularisation procedure. Phase 1 of the program is the inpatient phase. A thorough assessment of the person's history, current status, risk factors and motivation is obtained. During this phase, activity progresses from bed rest to independent performance of activities of daily living (ADLs) and ambulation within the facility. Both subjective and objective responses to increasing activity levels are evaluated. Excess fatigue, shortness of breath, chest pain, tachypnoea, tachycardia or cool, clammy skin indicates activity intolerance. Phase 2, immediate outpatient cardiac rehabilitation, begins within 3 weeks of the cardiac event. The goals for the outpatient program are to increase activity level, participation and capacity; improve psychosocial status and treat anxiety or depression; and provide education and support for risk factor reduction. Continuation programs, phase 3 of cardiac rehabilitation, are directed at providing a transition to independent exercise and exercise maintenance. During this final phase, the person may 'check in' every 3 months to evaluate risk factors, quality of life and exercise habits.

Nursing care

Nursing care of the person with an acute myocardial infarction focuses on reducing cardiac work, identifying and treating complications in a timely manner, and preparing the person for rehabilitation. See the following nursing care plan for a person with an AMI.

Health promotion

Health promotion activities to prevent AMI are those outlined for coronary heart disease and angina in previous sections of this chapter. In addition, discuss risk factor management, use of prescribed medications and cardiac rehabilitation to reduce the risk of complications or future infarctions.

Assessment

Nursing assessment for the person with AMI must be both timely and ongoing. Assessment data related to AMI include the following:

- *Health history:* complaints of chest pain, including its location, intensity, character, radiation and timing; associated symptoms such as nausea, heartburn, shortness of breath and anxiety; treatment measures taken since onset of pain; past medical history, especially cardiac related; chronic diseases; current medications and any known allergies to medications; smoking history and use of recreational drugs and alcohol.
- *Physical examination:* general appearance, including obvious signs of distress; vital signs; peripheral pulses; skin colour, temperature, moisture; level of consciousness; heart and breath sounds; cardiac rhythm (on bedside monitor); bowel sounds, abdominal tenderness.

Nursing diagnoses and interventions

Priorities of nursing care include relieving chest pain, reducing cardiac work and promoting oxygenation. Psychosocial support is especially important because an acute myocardial infarction can be devastating, bringing the person face to face with their own mortality for the first time.

Acute pain

Chest pain occurs when the oxygen supply to the heart muscle does not meet the demand. Myocardial ischaemia and infarction causes pain, as does reperfusion of an ischaemic area following thrombolytic therapy or emergent PTCA. Pain stimulates the sympathetic nervous system, increasing cardiac work. Pain relief is a priority of care for the person with AMI.

- Assess for verbal and non-verbal signs of pain. Document characteristics and the intensity of the pain, using a standard pain scale. Verify non-verbal indicators of pain with the person. Frequent, careful pain assessment allows early intervention to reduce the risk of further damage. *Pain is a subjective experience; its expression may vary with location and intensity, previous experiences and cultural and social background. Pain scales provide an objective tool for measuring pain and a way to assess pain relief or reduction.*
- Administer oxygen at 2 to 5 L/min per nasal prongs. *Supplemental oxygen increases oxygen supply to the myocardium, decreasing ischaemia and pain.*
- Promote physical and psychological rest. Provide information and emotional support. *Rest decreases cardiac workload and sympathetic nervous system stimulation, promoting comfort. Information and emotional support help decrease anxiety and provide psychological rest.*
- Titrate intravenous glyceryl trinitrate as ordered to relieve chest pain, maintaining a systolic blood pressure greater than 100 mmHg. *Glyceryl trinitrate decreases chest pain by dilating peripheral vessels, reducing cardiac work and dilating coronary vessels, including collateral circulation, improving blood flow to ischaemic tissue.*

CONSIDERATION FOR PRACTICE

Intravenous glyceryl trinitate (GTN) causes peripheral vasodilation, which may lead to hypotension, reduced coronary blood flow and tachycardia. Reduce the GTN flow rate and notify the medical officer if this occurs.

- Administer 2 to 4 mg morphine by intravenous push for chest pain as needed. *Morphine is an effective narcotic analgesic for chest pain. It acts as a venodilator and decreases the respiratory rate, anxiety and the perception of pain. The resulting reduction in preload and sympathetic nervous system stimulation reduces cardiac work and oxygen consumption.*

CONSIDERATION FOR PRACTICE

Reassess for relief of chest pain. The goal of care is to achieve absolute pain relief, not simply a reduction in pain to a 'manageable' level. Any chest pain of cardiac origin indicates myocardial ischaemia.

Ineffective tissue perfusion

Cardiac muscle damage affects compliance, contractility and cardiac output. The extent of the effect on tissue perfusion depends on the location and amount of damage. Anterior wall infarcts have a greater effect on cardiac output than do right ventricular infarcts. Infarcted muscle also increases the risk of cardiac arrhythmias, which can also affect the delivery of blood and oxygen to the tissues.

- Assess and document vital signs. Report increases in heart rate and changes in rhythm, blood pressure and respiratory rate. *Decreased cardiac output activates compensatory mechanisms that may cause tachycardia and vasoconstriction, increasing cardiac work.*
- Assess for changes in level of consciousness (LOC); decreased urine output; moist, cool, pale, mottled or cyanotic skin; dusky or cyanotic mucous membranes and nail beds; diminished to absent peripheral pulses; delayed capillary refill. *These are manifestations of impaired tissue*

perfusion. A change in LOC is often the first manifestation of altered perfusion because brain tissue and cerebral function depend on a continuous supply of oxygen.

- Auscultate heart and breath sounds. Note abnormal heart sounds (e.g. an S_3 or S_4 gallop or a murmur) or adventitious lung sounds. *Abnormal heart sounds or adventitious lung sounds may indicate impaired cardiac filling or output, increasing the risk of decreased tissue perfusion.*
- Monitor ECG rhythm continuously. *Arrhythmias can further impair cardiac output and tissue perfusion.*

CONSIDERATION FOR PRACTICE

Obtain a 12-lead ECG to assess complaints of chest pain. Report marked changes to the doctor. Continued or unrelieved chest pain may indicate further myocardial ischaemia and extension of the infarct; an ECG during episodes of chest pain provides a valuable diagnostic tool to assess myocardial perfusion. A repeat ECG on subsidence of pain is also very valuable.

- Monitor oxygen saturation levels. Administer oxygen as ordered. Obtain and assess ABGs as indicated. *Oxygen saturation is an indicator of gas exchange, tissue perfusion and the effectiveness of oxygen administration. ABGs provide a more precise measurement of blood oxygen levels and allow assessment of acid–base balance.*
- Administer antiarrhythmic medications as needed. *Arrhythmias affect tissue perfusion by altering cardiac output.*
- Obtain serial CK, isoenzyme and troponin levels as ordered. *Levels of cardiac markers—CK isoenzymes, in particular—correlate with the extent of myocardial damage.*
- Plan for invasive haemodynamic monitoring. *Haemodynamic monitoring facilitates AMI management and treatment evaluation by providing a means of assessing pressures in the systemic and pulmonary arteries, the relationship between oxygen supply and demand, cardiac output and cardiac index.*

CONSIDERATION FOR PRACTICE

Continuously evaluate the response to interventions such as thrombolytic therapy, drugs to improve cardiac output and tissue perfusion, and drugs to reduce cardiac work. Adverse effects of therapy may reduce the effectiveness of treatment. Bleeding due to thrombolytic therapy may affect vascular volume and cardiac output; reperfusion arrhythmias also may affect cardiac output. Drugs used to improve cardiac output may also increase cardiac work, whereas those given to reduce cardiac work may significantly affect contractility and cardiac output.

Ineffective coping

Coping mechanisms help a person deal with a life-threatening event or with acute changes in health. However, certain coping mechanisms may be detrimental to restoring health, particularly if the person relies on them for a prolonged period. Denial, for example, is a common coping mechanism among people following an MI. In the initial stages, denial can reduce anxiety. Continued denial, however, can interfere with learning and compliance with treatment.

- Establish an environment of caring and trust. Encourage the person to express feelings. *Establishing a trusting relationship provides a safe environment for the person to discuss feelings of helplessness, powerlessness, anxiety and hopelessness. The nurse may then be able to provide additional resources to meet the person's needs.*
- Accept denial as a coping mechanism, but do not reinforce it. *Denial may initially help by diminishing the psychological threat to health, decreasing anxiety. However, its prolonged use can interfere with acceptance of reality and cooperation, possibly delaying treatment and hindering recovery.*
- Note aggressive behaviours, hostility or anger. *Document any failure to comply with treatments. These signs can indicate anxiety and denial.*
- Help the person identify positive coping skills used in the past (e.g. problem-solving skills, verbalisation of feelings, asking for help, prayer). *Reinforce use of positive coping behaviours. Coping behaviours that have been successful in the past can help the person deal with the current situation. These familiar methods can decrease feelings of powerlessness.*
- Provide opportunities for the person to make decisions about the plan of care, as far as possible. *This promotes self-confidence and independence. Participating in care planning gives the person a sense of control and the opportunity to use positive coping skills.*
- Provide privacy for the person and significant other to share their questions and concerns. *Privacy provides an opportunity for the person and their partner to share their feelings and fears, offer support and encouragement to one another, relieve anxiety and establish effective coping methods.*

Fear

The fear of death and disability can be a paralysing emotion that adversely affects the person's recovery from acute myocardial infarction.

- Identify the person's level of fear, noting verbal and non-verbal signs. This information enables the nurse to plan appropriate interventions. *Individuals may not voice concerns; attention to non-verbal indicators is important. Controlling fear helps decrease sympathetic nervous system responses and catecholamine release that may increase feelings of fear and anxiety.*
- Acknowledge the person's perception of the situation. Allow individuals to verbalise concerns. *A sudden change in health status causes anxiety and fear of the unknown. Verbalising these fears may help the person cope with change and allow the healthcare team to provide information and correct misconceptions.*

- Encourage questions and provide consistent, factual answers. Repeat information as needed. *Accurate and consistent information can reduce fear. Honest explanations help strengthen the patient–nurse relationship and help the person develop realistic expectations. Anxiety and fear decrease the ability to concentrate and retain information; therefore, information may need to be repeated.*
- Encourage self-care. Allow the individual to make decisions regarding the plan of care. *This promotes personal responsibility for health and allows some control over the situation. The person's confidence increases as their dependence decreases.*
- Administer anxiolytic medications as ordered. *These medications promote rest and relaxation and decrease feelings of anxiety, which may act as barriers to health restoration.*
- Teach non-pharmacological methods of stress reduction (e.g. relaxation techniques, mental imagery, music therapy, breathing exercises, meditation, massage). *Stress management techniques can help reduce tension and anxiety, provide a sense of control and enhance coping skills.*

NURSING CARE PLAN A person with acute myocardial infarction

Betty Williams, a 62-year-old office worker, is admitted to the emergency department with complaints of severe substernal chest pain. Mrs Williams states that the pain began after lunch, about 4 hours ago. She initially attributed the pain to indigestion. She described the pain, which now radiates to her jaw and left arm, as 'really severe heartburn'. It is accompanied by a 'choking feeling', severe shortness of breath and diaphoresis. The pain is unrelieved by rest, antacids or three sublingual glyceryl trinitrate tablets (0.4 mg).

Oxygen is started per nasal prongs at 5 L/min. Central and peripheral intravenous lines are inserted. A 12-lead ECG and the following labwork are obtained: cardiac troponins, CK and CK isoenzymes, ABGs, FBC and a chemistry panel. Morphine relieves Mrs Williams's pain.

Mrs Williams's medical history includes type 2 diabetes, angina and hypertension. She has a 45-year history of cigarette smoking, averaging 1.5 to 2 packets per day. Family history reveals that Mrs Williams's father died at age 42 of AMI and her paternal grandfather died at age 65 of AMI. Mrs Williams is taking the following medications: tolbutamide, hydrochlorothiazide and isosorbide mononitrate.

Based on ECG changes and cardiac markers, an acute anterior MI is diagnosed. Mrs Williams has no contraindications to thrombolytic therapy and is deemed a good candidate. Intravenous alteplase (t-PA) is given by bolus followed by intravenous infusions of alteplase and heparin. She is transferred to the coronary care unit (CCU).

ASSESSMENT

Mrs Williams is alert and oriented to person, place and time. Vital signs are T 37.5°C, P 118, R 24 with adequate depth and BP 172/92. Auscultation reveals an S_4 and fine crackles in the bases of both lungs. The ECG shows sinus tachycardia with occasional premature ventricular contractions (PVCs). Her skin is cool and slightly diaphoretic. Capillary refill is less than 3 seconds and peripheral pulses are strong and equal. Her nail beds are pink.

A triple-lumen central line is in place. Glyceryl trinitrate is infusing at 200 µg/min in the distal lumen; the alteplase infusion is in the middle lumen; and a heparin infusion is in the proximal lumen. The peripheral intravenous line has a saline lock. Mrs Williams states, 'The pain is better since the nurse in the ER gave me a shot. But it has been coming and going. I would rate it a 4 right now, but it was terrible before. The doctor told me that this drug I'm getting will quickly open up the artery that is blocked. I hope it works! Do many people get this drug?'

DIAGNOSIS

- *Acute pain* related to ischaemic myocardial tissue.
- *Anxiety* and *fear* related to change in health status.
- *Ineffective protection* related to the risk of bleeding secondary to thrombolytic therapy.
- *Risk of decreased cardiac output* related to altered cardiac rate and rhythm.

PLANNING

The following interventions are planned during the immediate phase of Mrs Williams's hospitalisation.

- Instruct her to report all chest pain. Monitor and evaluate pain using a scale of 0 to 10. Titrate intravenous glyceryl trinitrate infusion for chest pain; stop infusion if systolic BP is below 100 mmHg. Administer 2 to 4 mg of morphine intravenously for chest pain unrelieved by glyceryl trinitrate infusion.
- Encourage verbalisation of fears and concerns. Respond honestly and correct misconceptions about the disease, therapeutic interventions or prognosis.
- Assess knowledge of CHD. Explain the purpose of thrombolytic therapy to dissolve the fresh clot and reperfuse the heart muscle, limiting heart damage.
- Explain the need for frequent monitoring of vital signs and potential bleeding.
- Assess for manifestations of internal or intracranial bleeding: complaints of back or abdominal pain, headache, decreased level of consciousness, dizziness, bloody secretions or excretions, or pallor. Test all stools, urine and vomit for occult blood. Notify doctor immediately of any abnormal findings.

Expected outcomes

- Rate chest pain as 0 on a pain scale of 0 to 10.
- Verbalise reduced anxiety and fear.
- Demonstrate no signs of internal or external bleeding.
- Maintain an adequate cardiac output during and following reperfusion therapy.

IMPLEMENTATION

- Monitor for signs of reperfusion: decreased chest pain, return of ST segment to baseline, reperfusion arrhythmias (e.g. PVCs, bradycardia and heart block).
- Continuously monitor ECG for changes in cardiac rate, rhythm and conduction. Assess vital signs.
- Treat dangerous arrhythmias or other cardiac events per protocol. Notify the medical officer.
- Discuss continuing cardiac care and rehabilitation.

NURSING CARE PLAN A person with acute myocardial infarction (continued)

EVALUATION

The initial morphine dose reduces Mrs Williams's chest pain from a rating of 8 to 4. The glyceryl trinitrate infusion and thrombolytic therapy further reduce her pain to 2. The glyceryl trinitrate infusion is gradually discontinued after 24 hours. As her pain subsides, Mrs Williams states that she feels 'much better now that the pain is gone. I was afraid it would just get worse'. She verbalises an understanding of thrombolytic therapy to limit myocardial damage. No indication of bleeding problems are noted. Reperfusion is indicated by relief of chest pain, return of the ST segment to baseline on the ECG, early peaking of CK levels and increased frequency of PVCs but no significant arrhythmias. Mrs Williams remains in the CCU for 36 hours until she is transferred to the cardiac medical ward.

CRITICAL THINKING IN THE NURSING PROCESS

1 How would the initial plan of care have changed if Mrs Williams was not a candidate for thrombolytic therapy?
2 Two days after her initial therapy, Mrs Williams complains of palpitations. You notice frequent PVCs on the ECG monitor. What do you do?
3 What health promotion topics would you teach Mrs Williams before discharge?
4 Mrs Williams states, 'I've been smoking for 45 years and I'm not going to stop now! Besides, it calms me down when I'm anxious.' How would you respond to this statement?

RFLECTION ON THE NURSING PROCESS

1 Outline what you have learned from this case study that you will apply to your future practice.
2 Cardiovascular disease in women presents many challenges for assessment and diagnosis, resulting in higher mortality and morbidity statistics. Explore the factors that complicate the identification of cardiovascular disease in women. How will this information modify your practice when next assessing a woman presenting with chest pain?

Community-based care

Cardiac rehabilitation begins with admission to the healthcare facility and continues through the inpatient stay and after discharge into the rehabilitative period. The emphasis is on realistic application of information to maintain lifestyle changes.

Assessing readiness to learn is an important first step in preparing for home care. The person in strong denial may not identify any relevance to the information being taught. Evaluate ability to learn, assessing physiological and psychological health, beliefs regarding personal responsibility for health and expectations of the healthcare system. Also assess developmental level, ability to perform psychomotor skills, cognitive function, learning disabilities, existing knowledge base and the influence of previous learning experiences. Provide written material to supplement teaching and encourage questions.

Include the following topics in teaching for home care:

- the normal anatomy and physiology of the heart and the specific area of heart damage
- the process of CHD and implications of MI
- purposes and side effects of prescribed medications
- the importance of complying with the medical regimen and cardiac rehabilitation program and of keeping follow-up appointments
- information about community resources such as the National Heart Foundation.

After discharge, follow up by telephone within 1 week and periodically thereafter during the recovery period. Provide telephone numbers of resource personnel who are available to respond to questions and concerns after discharge. Research demonstrates the value of motivational and social support in adopting healthier behaviours after AMI (see the following 'Translation to practice' box).

Because the person who has had an MI is at high risk of sudden cardiac death, encourage family members to learn CPR and provide information about community resources for CPR training.

CARDIAC RHYTHM DISORDERS

Heart muscle contracts in response to electrical stimulation. In the normal heart, electrical stimulation produces a synchronised, rhythmic heart muscle contraction that propels blood into the vascular system. Changes in cardiac rhythm affect this synchronised activity and the heart's ability to effectively pump blood to body tissues.

THE PERSON WITH A CARDIAC ARRHYTHMIA

A cardiac arrhythmia is a disturbance or irregularity in the electrical system of the heart. Cardiac arrhythmias may be benign or have lethal consequences. Prompt recognition of a lethal arrhythmia and quick action can be lifesaving.

Arrhythmias develop for many reasons. Not all are pathological; some alterations in cardiac rhythm occur in response to events such as exercise or fear. For example, a rapid heart rate due to exercise, fever or excitement is a normal response to the body's demand for oxygen or to stimulation of the sympathetic nervous system. Slow heart rates also may be normal. Athletic heart syndrome, which results from long-term training on the heart muscle, allows the heart to beat more slowly and forcefully while maintaining cardiac output and tissue perfusion. Many athletes have a heart rate of less than 60 beats per minute (beats/min or bpm). Ageing affects cardiac rhythm as well (see the following 'Nursing care of the older adult' box).

TRANSLATION TO PRACTICE Evidence-based practice: the prevalence and pathophysiology of MINOCA

Most people who experience a myocardial infarction have angiographically recordable atherosclerotic disease. However, there are a small number of individuals who are diagnosed with an MI yet, strangely, demonstrate no evidence of obstructive atherosclerotic lesion. Consequently, a new diagnosis is emerging from the literature: myocardial infarction with non-obstructive coronary arteries (MINOCA).

In a systematic review by Pasupathy et al. (2015), a meta-analysis of 28 publications revealed that the prevalence of MINOCA is approximately 6%, with 60% of the individuals being male. However, when compared to myocardial infarction with associated coronary artery obstruction, the profile tended to favour younger females with less likelihood of having hyperlidipaemia. The mortality of the MINOCA group after 1 year was also lower.

IMPLICATIONS FOR NURSING

When working with younger females or individuals with no distinct hyperlipidaemia, the risk for developing non-obstructive coronary artery disease resulting in myocardial ischaemia or infarction should not be ignored. Education promoting healthy lifestyle choices, including nutrition and activity levels, remains paramount in the prevention of CHD.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Which clinical manifestations might a person experiencing a myocardial infarction experience?
2. How might a woman's description of clinical manifestations differ from a man's when experiencing a myocardial infarction?
3. Which assessments would you undertake and what data might you collect from a person you suspect is experiencing an MI?
4. How will the findings of this meta-analysis influence your clinical practice?

Source: Based on Pasupathy, S. et al. (2015). Systematic review of patients presenting with suspected myocardial infarction and nonobstructive coronary arteries. *Circulation*, *131*(10), 861–70. Doi: 10.1161/circulationaha.114.011201.

NURSING CARE OF THE OLDER ADULT Cardiac arrhythmias

Ageing affects the heart and the cardiac conduction system, increasing the incidence of arrhythmias and conduction defects. Older adults may experience arrhythmias even when no evidence of heart disease is found.

Older adults have a higher incidence of both ventricular and supraventricular arrhythmias without detrimental effects than younger people. Ectopic beats, including short runs of ventricular tachycardia, occur more commonly during exercise in older adults. These arrhythmias do not affect cardiac morbidity or mortality. Fibrosis of the bundle branches can lead to atrioventricular blocks; a prolonged PR interval is common in people over the age of 65. Older adults also have a higher incidence of diseases that may affect heart rhythm. An older adult person with hyperthyroidism, for example, may present with atrial fibrillation, syncope and confusion instead of the usual manifestations of goitre, tremor and exophthalmos.

ASSESSING FOR HOME CARE

Assessing older adults for problems related to cardiac arrhythmias focuses on the effect of the arrhythmia on functional health status.

- Ask about a history of cardiovascular disease and current medications.
- Inquire about symptoms such as episodes of presyncope, syncope, palpitations, chest pain or dyspnoea.
- Ask about relationship of symptoms such as palpitations to intake of certain foods and caffeine-containing beverages.
- Evaluate for other contributing factors such as smoking or alcohol intake.
- Inquire about a history of falls, particularly those occurring without apparent reason.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

Teach measures to reduce the risk of cardiac arrhythmias and potential adverse consequences of arrhythmias.

- Emphasise the importance of taking medications as prescribed. Discuss possible effects of over-the-counter medications on the heart.
- Encourage reducing or eliminating caffeine intake. Caffeine increases the risk of ectopic beats and rapid heart rates.
- Encourage participation in a smoking cessation program and reduction or elimination of alcohol intake if appropriate.
- Encourage engaging in regular exercise. Discuss the beneficial effects of exercise in maintaining muscle mass, including cardiac muscle and cardiovascular health.
- Instruct the individual to contact the primary healthcare provider for evaluation of symptoms such as dizziness, fainting, frequent palpitations, dyspnoea, unexplained falls or chest pain.

Regardless of cause, an arrhythmia can significantly affect cardiac performance, depending on heart muscle health. The person's response to the arrhythmia is key to determining the urgency and type of treatment needed.

Physiology review

The unique properties of cardiac cells allow effective heart function. Four properties are electrical; the fifth is cardiac muscle's mechanical response to electrical stimulation.

1. *Automaticity* is the ability of pacemaker cells to spontaneously initiate an electrical impulse (action potential). The sinoatrial (SA) node is the dominant pacemaker, generating impulses at 60 to 100 times a minute. Myocardial muscle cells do not possess this ability.
2. *Excitability* is the ability of myocardial cells to respond to stimuli generated by pacemaker cells.
3. *Conductivity* is the ability to transmit an impulse from cell to cell. When one cell is stimulated, the impulse rapidly spreads throughout the heart muscle.
4. *Refractoriness* is the inability of cardiac cells to respond to additional stimuli immediately following depolarisation. In the absolute refractory period, depolarisation will not occur in response to any stimulus. A stronger than normal stimulus is required to initiate depolarisation during the relative refractory period. This is followed by the supernormal period, during which a mild stimulus will cause depolarisation.
5. *Contractility* is the ability of myocardial fibres to shorten in response to a stimulus. Heart muscle responds in an all-or-nothing manner: stimulation of one muscle fibre causes the entire muscle mass to contract to its fullest extent as one unit.

Electrical activity of the heart is normally controlled by the cardiac conduction. The SA node, the primary pacemaker of the heart, usually generates impulses at a regular rate of 60 to 100 bpm. The impulse spreads through the atria, is briefly delayed at the AV node, then spreads through conduction pathways of the ventricles and to ventricular muscle. The AV nodal delay allows the atria to contract, delivering an extra bolus of blood to the ventricles before they contract (the atrial kick). The AV node also controls the number of impulses that reach the ventricles, preventing extremely rapid heart rates.

Pathophysiology

Arrhythmias arise through disruption of the very properties that stimulate and control the heartbeat: automaticity, excitability, conductivity and refractoriness.

Arrhythmias due to altered impulse formation include changes in rate and rhythm and the development of ectopic beats. This category includes *tachyarrhythmias* (rapid heart rates), *bradyarrhythmias* (slow heart rates) and ectopic rhythms. These arrhythmias result from a change in the automaticity of cardiac cells. The rate of impulse formation may abnormally increase or decrease. Aberrant (abnormal) impulses may originate outside normal conduction pathways, causing **ectopic beats**. Ectopic beats interrupt the normal conduction sequence and may not initiate a normal muscle contraction. Depending on the site and timing of abnormal impulses, they may have little effect on the person or pose a significant threat.

Ischaemia, injury and infarction of myocardial tissue affect its excitability and ability to conduct and respond to an electrical stimulus. Conduction abnormalities cause varying degrees of **heart block**, a block in the normal conduction pathways. Myocardial injury or infarction can obstruct or delay impulse conduction. Bundle branch blocks are common in acute myocardial infarction.

The *re-entry phenomenon*, a phenomenon of normal and slow conduction, is a major cause of tachyarrhythmias. A stimulus such as an ectopic beat triggers the re-entry phenomenon. The impulse is delayed in one area of the heart (e.g. an area of ischaemia or injury) but conducted normally through the rest. Muscle that has been depolarised by the normally conducted impulse is repolarised by the time the impulse travelling through the area of slow conduction reaches it, thus initiating another cycle of depolarisation (Grossman & Mattson, 2013). The result is an arrhythmia that propagates itself.

Several forms of re-entry may occur. The impulse may travel through a set pathway to re-enter repolarised tissue. Many atrial arrhythmias follow this pattern, including atrial flutter. In functional re-entry, local differences in the conduction of an impulse interrupt the normal wave of depolarisation, sending it back upon itself in a spiral pattern and setting up a permanent rotation. This type of pattern suppresses normal pacemaker activity and can lead to atrial fibrillation (Grossman & Mattson, 2013).

Cardiac rhythms are classified according to the site of impulse formation or the site and degree of conduction block. Supraventricular rhythms arise above the ventricles. These rhythms usually produce a QRS complex within the normal range. Sinus rhythms, atrial rhythms and junctional (arising from the AV junction) rhythms are all supraventricular rhythms. Ventricular rhythms originate in the ventricles and may prove fatal if left untreated. AV conduction blocks result from a defect in impulse transmission from the atria to the ventricles. The major normal and abnormal cardiac rhythms are summarised in Table 29.7.

FAST FACTS

- The normal sinus rhythm is 60 to 100 bpm. Each complex includes a P wave, QRS and T wave.
- Supraventricular arrhythmias arise in the sinus node or the atria. A P wave may be present; the QRS appears normal and a T wave may be seen.
- Junctional arrhythmias arise in tissue just above or just below the AV node. The P wave may be inverted and may precede, follow or be buried in the QRS complex. The QRS usually appears normal and is followed by a T wave.
- Ventricular arrhythmias arise in ventricular myocardium. They do not reset the SA node or activate the atria. QRS complexes are wide and bizarre.

Supraventricular rhythms

NORMAL SINUS RHYTHM **Normal sinus rhythm (NSR)** is the normal heart rhythm, in which impulses originate in the SA (sinus) node and travel through all normal conduction pathways without delay. All waveforms are of normal configuration, look alike and have consistent (fixed) durations. The rate is between 60 and 100 bpm.

TABLE 29.7 Characteristics of selected cardiac rhythms and arrhythmias

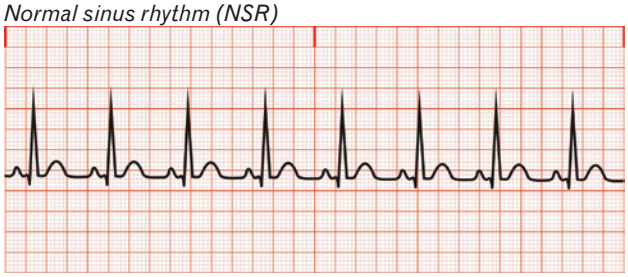
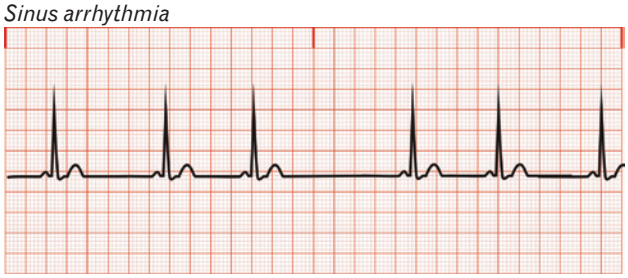
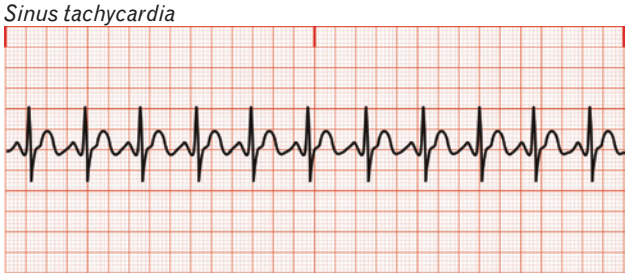
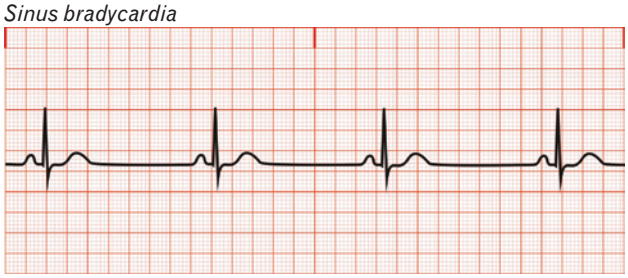
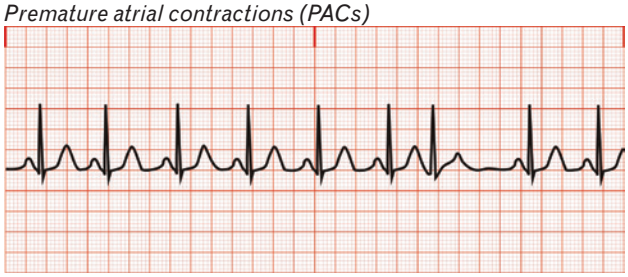
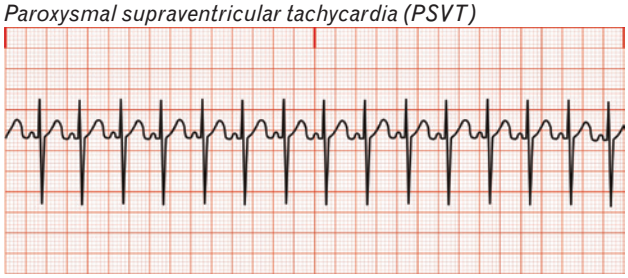
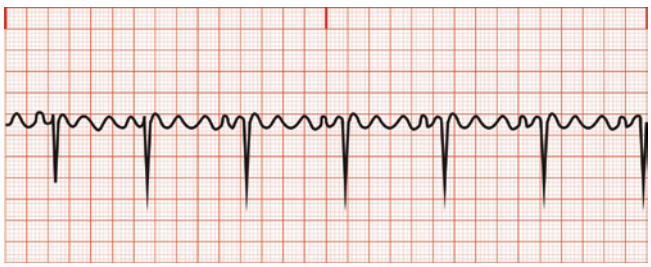
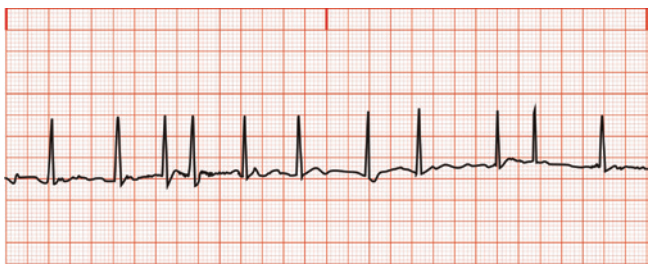
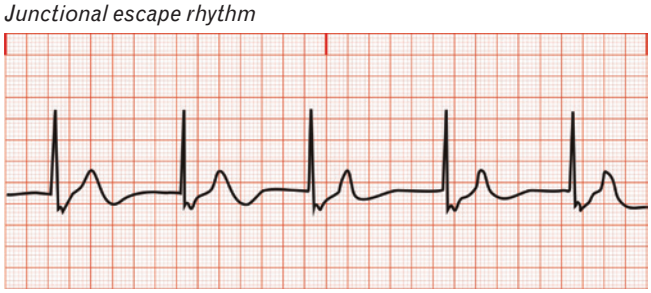
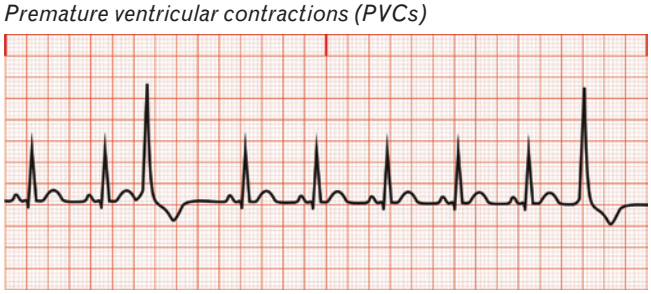
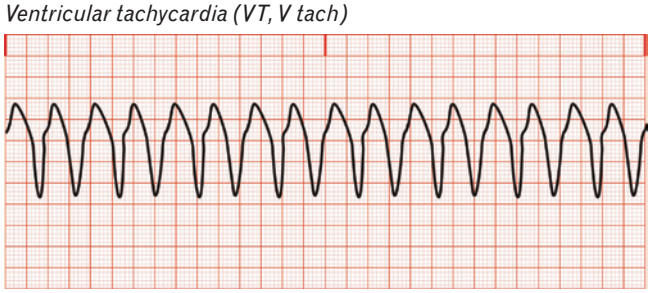
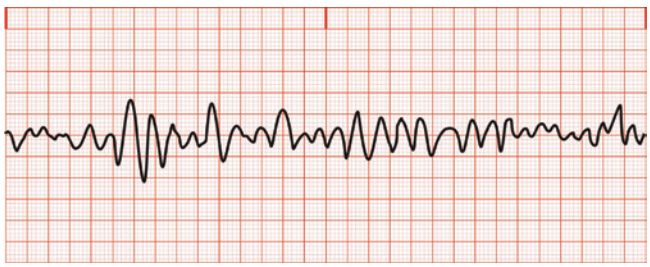
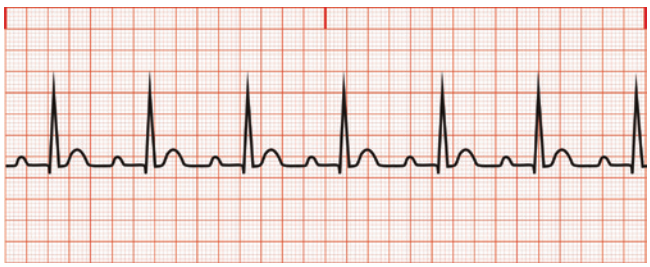
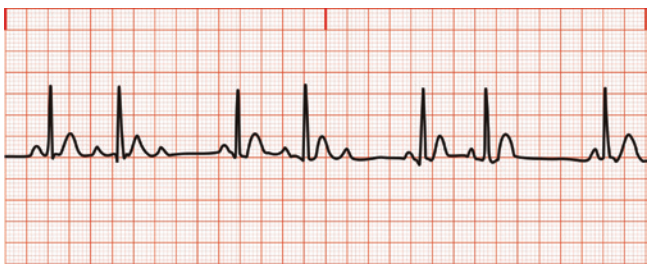
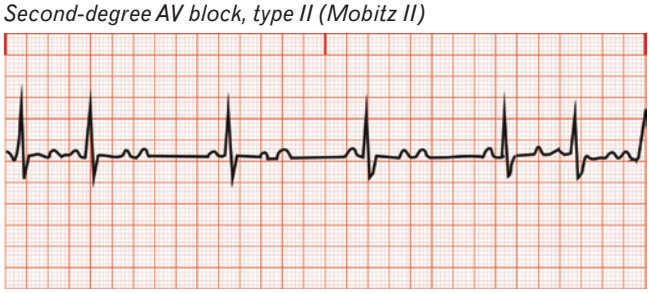
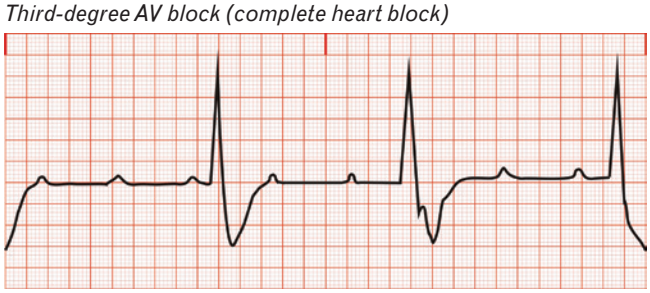
RHYTHM/ECG APPEARANCE	ECG CHARACTERISTICS	MANAGEMENT
Supraventricular rhythms		
Normal sinus rhythm (NSR) 	Rate: 60 to 100 beats/min Rhythm: regular P:QRS: 1:1 PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec	None; normal heart rhythm.
Sinus arrhythmia 	Rate: 60 to 100 beats/min Rhythm: irregular, varying with respirations P:QRS: 1:1 PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec	Generally none; considered a normal rhythm in the very young and very old.
Sinus tachycardia 	Rate: 101 to 150 beats/min Rhythm: regular P:QRS: 1:1 (With very fast rates, P wave may be hidden in preceding T wave) PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec	Treated only if symptomatic or person is at risk for myocardial damage. Treat underlying cause (e.g. hypovolaemia, fever, pain). Beta-blockers or verapamil may be used.
Sinus bradycardia 	Rate: < 60 beats/min Rhythm: regular P:QRS: 1:1 PR interval: 0.12 to 0.20 sec QRS complex: 0.6 to 0.10 sec	Treated only if symptomatic. Intravenous atropine or isoprenaline, and/or pacemaker therapy may be used.
Premature atrial contractions (PACs) 	Rate: variable Rhythm: irregular, with normal rhythm interrupted by early beats arising in the atria P:QRS: 1:1 PR interval: 0.12 to 0.20 sec, but may be prolonged QRS complex: 0.6 to 0.10 sec	Usually require no treatment. Advise to reduce alcohol and caffeine intake, to reduce stress and to stop smoking. Beta-blocker may be prescribed.
Paroxysmal supraventricular tachycardia (PSVT) 	Rate: 100 to 280 beats/min (usually 150 to 200 beats/min) Rhythm: regular P:QRS: P waves often not identifiable PR interval: not measured QRS complex: 0.6 to 0.10 sec	Treat if symptomatic. Treatment may include vagal manoeuvres (Valsalva, carotid sinus massage); oxygen therapy; adenosine or a beta-blocker; temporary pacing or synchronised cardioversion.

TABLE 29.7 Characteristics of selected cardiac rhythms and arrhythmias (continued)

RHYTHM/ECG APPEARANCE	ECG CHARACTERISTICS	MANAGEMENT
Atrial flutter 	Rate: atrial 240 to 360 beats/min, ventricular rate depends on degree of AV block and usually is < 150 beats/min Rhythm: atrial regular; ventricular usually regular P:QRS: 2:1, 4:1, 6:1; may vary PR interval: not measured QRS complex: 0.6 to 0.10 sec	Synchronised cardioversion; medications to slow ventricular response such as a beta-blocker or calcium channel blocker, followed by a class I antiarrhythmic agent or amiodarone.
Atrial fibrillation 	Rate: atrial 300 to 600 beats/min (too rapid to count); ventricular 100 to 180 beats/min in untreated people Rhythm: irregularly irregular P:QRS: variable PR interval: not measured QRS complex: 0.06 to 0.10 sec	Synchronised cardioversion; medications to reduce ventricular response rate: metaprolol, diltiazem or digoxin; anticoagulant therapy to reduce risk of clot formation and stroke.
Junctional escape rhythm 	Rate: 40 to 60 beats/min; junctional tachycardia 60 to 140 beats/min Rhythm: regular P:QRS: P waves may be absent, inverted and immediately preceding or succeeding QRS complex or hidden in QRS complex PR interval: < 0.10 sec QRS complex: 0.06 to 0.10 sec	Treat cause if symptomatic.
Ventricular rhythms		
Premature ventricular contractions (PVCs) 	Rate: variable Rhythm: irregular, with PVC interrupting underlying rhythm and followed by a compensatory pause P:QRS: No P wave noted before PVC PR interval: absent with PVC QRS complex: wide (> 0.12 sec) and bizarre in appearance; differs from normal QRS complex	Treat if symptomatic or in presence of severe heart disease. Advise against stimulant use (caffeine, nicotine). Beta-blockers or class I or III antiarrhythmic agents (see the box on page 1029) may be used in people with severe heart disease who are symptomatic.
Ventricular tachycardia (VT, V tach) 	Rate: 100 to 250 beats/min Rhythm: regular P:QRS: P waves usually not identifiable PR interval: not measured QRS complex: 0.12 sec or greater; bizarre shape	Treat if VT is sustained, symptomatic or associated with organic heart disease. Treatment includes DC cardioversion or intravenous procainamide, lignocaine or a class III antiarrhythmic agent if haemodynamic instability accompanies. Surgical ablation or pacing with an implanted cardioverter-defibrillator (ICD) for repeated episodes.

(continued)

TABLE 29.7 Characteristics of selected cardiac rhythms and arrhythmias (continued)

RHYTHM/ECG APPEARANCE	ECG CHARACTERISTICS	MANAGEMENT
Ventricular fibrillation (VF, V fib) 	Rate: no real rate to count Rhythm: grossly irregular P:QRS: no identifiable P waves PR interval: none QRS: bizarre, varying in shape and direction	Immediate cardioversion/ defibrillation.
Atrioventricular conduction blocks		
First-degree AV block 	Rate: usually 60 to 100 beats/min Rhythm: regular P:QRS: 1:1 PR interval: > 0.21 sec QRS complex: 0.06 to 0.10 sec	None required.
Second-degree AV block, type I (Mobitz I, Wenckebach) 	Rate: 60 to 100 beats/min Rhythm: atrial regular; ventricular irregular P:QRS: 1:1 until P wave blocked with no subsequent QRS complex PR interval: progressively lengthens in a regular pattern QRS complex: 0.06 to 0.10 sec; sudden absence of QRS complex	Monitoring and observation; rarely progresses to a higher degree of block or requires treatment.
Second-degree AV block, type II (Mobitz II) 	Rate: atrial 60 to 100 beats/min; ventricular < 60 beats/min Rhythm: atrial regular; ventricular irregular P:QRS: typically 2:1, may vary PR interval: constant PR interval for each conducted QRS complex QRS complex: 0.06 to 0.10 sec	Atropine or isoprenaline; pacemaker therapy.
Third-degree AV block (complete heart block) 	Rate: atrial 60 to 100 beats/min; ventricular 15 to 60 beats/min Rhythm: atrial regular; ventricular regular P:QRS: no relationship P waves and QRS complexes; independent rhythms PR interval: not measured QRS complex: 0.06 to 0.10 sec if junctional escape rhythm; > 0.12 sec if ventricular escape rhythm	Immediate pacemaker therapy.

SINUS NODE ARRHYTHMIAS *Sinus node arrhythmias* may occur as a normal compensatory response (e.g. to exercise) or because of altered automaticity. In these rhythms, as in NSR, the initiating impulse is from the sinus node. They differ from NSR in the rate or regularity of the rhythm. Sinus arrhythmias include sinus arrhythmia, sinus tachycardia and sinus bradycardia.

Sinus arrhythmia *Sinus arrhythmia* is a sinus rhythm in which the rate varies with respirations, causing an irregular rhythm. The rate increases during inspiration and decreases with expiration. Sinus arrhythmia is common in the very young and the very old. It can be caused by an increase in vagal tone, by digoxin toxicity or by morphine administration.

Sinus tachycardia *Sinus tachycardia* has all of the characteristics of NSR, except that the rate is greater than 100 bpm. Tachycardia arises from enhanced automaticity in response to changes in the internal environment. Sympathetic nervous system stimulation or blocked vagal (parasympathetic) activity increases the heart rate. Tachycardia is a normal response to any condition or event that increases the body's demand for oxygen and nutrients, such as exercise or hypoxia. If the person on bed rest or someone who has done little to increase oxygen demand has tachycardia, this is an ominous sign. Sinus tachycardia may be an early sign of cardiac dysfunction, such as heart failure. Tachycardia is detrimental in the person with cardiac disease because it increases oxygen demand and decreases oxygen supply (because of decreased diastole reducing coronary artery filling time).

Common causes of sinus tachycardia include exercise, excitement, anxiety, pain, fever, hypoxia, hypovolaemia, anaemia, hyperthyroidism, myocardial infarction, heart failure, cardiogenic shock, pulmonary embolism, caffeine intake and certain drugs, such as atropine, adrenaline or isoprenaline.

Manifestations of sinus tachycardia include a rapid pulse rate. The person may complain of feeling that the heart is 'racing', shortness of breath and dizziness. In the presence of heart disease, sinus tachycardia may precipitate chest pain.

Sinus bradycardia *Sinus bradycardia* has all of the characteristics of NSR, but the rate is less than 60 bpm. Sinus bradycardia may result from increased vagal (parasympathetic) activity or from depressed automaticity due to injury or ischaemia to the sinus node. Sinus bradycardia may be normal (e.g. in the person with athletic heart syndrome). The heart rate also normally slows during sleep because the parasympathetic nervous system is dominant at this time. Other causes of sinus bradycardia include pain, increased intracranial pressure, sinus node disease, AMI (especially with inferior wall damage), hypothermia, acidosis and certain drugs.

Sinus bradycardia may be asymptomatic; it is important to assess the person before treating the rhythm. Manifestations of decreased cardiac output, such as decreased level of consciousness, syncope (faintness) or hypotension, indicate a need for intervention.

Sick sinus syndrome *Sick sinus syndrome (SSS)* results from sinus node disease or dysfunction that causes problems with impulse formation, transmission and conduction. Sick sinus syndrome is often found in older adults. It may be caused by direct injury to sinus tissue, fibrosis of conduction fibres associated with ageing and such drugs as digoxin, beta-blockers and calcium channel blockers.

ECG characteristics of SSS include sinus bradycardia, sinus arrhythmia, sinus pauses or arrest, and atrial tachyarrhythmias such as atrial fibrillation, atrial flutter or atrial tachycardia. Bradycardia–tachycardia syndrome, characterised either by paroxysmal (abrupt onset and termination) atrial tachycardia followed by prolonged sinus pauses or alternating periods of bradycardia and tachycardia, also may indicate sinus node dysfunction.

Manifestations of sinus node dysfunction often are intermittent, related to a drop in cardiac output caused by the irregular rhythm. Fatigue, dizziness, light headedness and syncope are common. The heart rate may not increase in response to stressors such as exercise or fever.

SUPRAVENTRICULAR ARRHYTHMIAS When an action potential originates in atrial tissue outside the sinus node, the resulting rhythm is classified as a *supraventricular rhythm*. In these arrhythmias, an ectopic pacemaker takes over or overrides the SA node. They may also occur when the SA node fails; an *escape rhythm* develops as a fail-safe mechanism to maintain the heart rate. The most common supraventricular arrhythmias are premature atrial contractions, paroxysmal supraventricular tachycardia, atrial flutter and atrial fibrillation. These rhythms may be paroxysmal; that is, occur in bursts with an abrupt beginning and end.

Premature atrial contractions *A premature atrial contraction (PAC)* is an ectopic atrial beat that occurs earlier than the next expected sinus beat. PACs can arise anywhere in the atria. They are usually asymptomatic and benign, but they may initiate paroxysmal supraventricular tachycardia in susceptible individuals. PACs are common in older adults, often occurring without an obvious cause. Strong emotions, excessive alcohol intake, tobacco and stimulants such as caffeine can precipitate PACs. They also may be associated with myocardial infarction, heart failure and other cardiac disorders, hypoxaemia, pulmonary embolism, digoxin toxicity and electrolyte or acid–base imbalances. In people with underlying heart disease, PACs may precede a more serious arrhythmia.

The ECG tracing shows interruption of the underlying rhythm by a premature complex that looks similar to the underlying beats. The ectopic impulse of the PAC is usually conducted normally, leading to depolarisation of cardiac muscle and a normal QRS complex. Because the impulse arises above the ventricles, it follows normal conduction pathways through the ventricles. The QRS complex is narrow or matches those of the underlying rhythm. The shape of the P wave of a PAC differs from normal P waves because its impulse arises outside the sinus node. A *non-compensatory pause* usually follows, as the PAC resets the SA node rhythm. Occasionally, the ectopic impulse may not be conducted through the heart, resulting in a lone P wave without a QRS or a non-conducted PAC.

PACs cause few manifestations. If frequent, they may cause palpitations or a fluttering sensation in the chest. Early beats may be noted on auscultating or palpating the pulse.

Paroxysmal supraventricular tachycardia *Paroxysmal supraventricular tachycardia (PSVT)* is tachycardia of sudden onset and termination. PSVT is usually initiated by a re-entry loop in or around the AV node; that is, an impulse re-enters the same section of tissue over and over, causing repeated depolarisations.

PSVT occurs more frequently in women. Sympathetic nervous system stimulation and stressors such as fever, sepsis and hyperthyroidism may precipitate PSVT. It also may be associated with heart diseases such as CHD, myocardial infarction, rheumatic heart disease, myocarditis or acute pericarditis. Abnormal conduction pathways associated with Wolff–Parkinson–White (WPW) syndrome may account for PSVT.

PSVT affects ventricular filling and cardiac output and decreases coronary artery perfusion. Its manifestations include complaints of palpitations and a 'racing' heart, anxiety, dizziness, dyspnoea, anginal pain, diaphoresis, extreme fatigue and polyuria. (Urine output may reach up to 3 L in the first few hours after PSVT onset.)

Atrial flutter *Atrial flutter* is a rapid and regular atrial rhythm thought to result from an intra-atrial re-entry mechanism. Causes include sympathetic nervous system stimulation due to anxiety or caffeine and alcohol intake; thyrotoxicosis; coronary heart disease or myocardial infarction; pulmonary embolism; and abnormal conduction syndromes, such as WPW syndrome. Older people with rheumatic heart disease and/or valvular disease are especially vulnerable.

Two types of atrial flutter have been identified. Type I atrial flutter has an atrial rate of 240 to 340 bpm. It develops due to a re-entry mechanism in the right atrium. The mechanism leading to type II atrial flutter has not been identified. In this type of flutter, the atrial rate is faster, to 350 bpm.

People with atrial flutter may complain of palpitations or a fluttering sensation in the chest or throat. If the ventricular rate is rapid, manifestations of decreased cardiac output, such as decreased level of consciousness, hypotension, decreased urinary output and cool, clammy skin, may be noted. The atrial kick (additional ventricular filling with atrial contraction) is lost because of inadequate atrial filling.

ECG characteristics include a 'sawtooth' or 'picket fence' appearance of P waves, which are labelled flutter (F) waves. The atrial rate is rapid, often around 300 bpm. As a protective mechanism, many impulses are blocked at the AV node and the ventricular rate is rarely greater than 150 to 170 bpm. Usually, atrial impulses are evenly conducted through the AV node; for example, two impulses to one QRS complex (2:1), four impulses to one QRS complex (4:1) or six impulses to one QRS complex (6:1). A constant conduction ratio results in a regular ventricular rhythm; the ventricular rhythm is irregular if the conduction ratio varies. The ventricular rate usually ranges from 150 to 170 bpm in 2:1 conduction and from 60 to 75 bpm for lower conduction ratios. The T wave is usually hidden by overriding F waves; some F waves may be hidden in the QRS complex.

Atrial fibrillation *Atrial fibrillation* is a common arrhythmia characterised by disorganised atrial activity without discrete atrial contractions. Multiple small re-entry circuits develop in the atria. Atrial cells cannot repolarise in time to respond to the next stimulus (Grossman & Mattson, 2013). Extremely rapid atrial impulses bombard the AV node, resulting in an irregularly irregular ventricular response which may be rapid > 100 bpm or slow < 60 bpm. Atrial fibrillation may occur suddenly and recur or it may persist as a chronic arrhythmia. Atrial fibrillation is commonly associated with heart failure, rheumatic heart disease, coronary heart disease, hypertension and hyperthyroidism.

Manifestations of atrial fibrillation are dependent on the ventricular rate. With rapid ventricular response rates, manifestations of decreased cardiac output such as hypotension, dyspnoea, fatigue and angina may develop. People with extensive heart disease may develop syncope or heart failure. Peripheral pulses are irregular and of variable amplitude (strength).

The specific ECG characteristics of atrial fibrillation include an irregularly irregular rhythm and the absence of identifiable P waves. The atrial rate is so rapid that it is not measurable. The ventricular rate varies.

Atrial fibrillation increases the risk of formation of thromboemboli. Organ infarction may occur as a result; the incidence of stroke is high.

Junctional arrhythmias

Rhythms that originate in AV nodal tissue are termed *junctional*. The AV junction includes the AV node and the bundle of His, which branches into the right and left bundle branches. An impulse arising from the AV junction may occur in response to failure of higher pacemakers, as in a *junctional escape rhythm,* or may result from an abnormal mechanism, such as altered automaticity. An impulse arising from the AV junction may or may not be conducted back up to the atria. This conduction against the normal flow or pattern is called *retrograde conduction*. The resulting atrial wave, called a P wave, may be found before, during or after the QRS complex, depending on the speed of conduction. The P wave is inverted in some ECG leads because the impulse moves from the AV node up to the atria, instead of from the SA node down towards the AV node. In addition, the PR interval is shorter than normal (less than 0.12 sec). The QRS complex is typically narrow.

A junctional rhythm may be due to drug toxicity (e.g. digoxin, beta-blockers or calcium channel blockers) or other causes such as hypoxaemia, hyperkalaemia, increased vagal tone or damage to the AV node, myocardial infarction and heart failure. Loss of synchronised atrial contraction and the atrial kick may affect cardiac output, leading to manifestations of decreased cardiac output and impaired myocardial tissue perfusion. Heart failure may develop.

Premature junctional contractions (PJCs) occur before the next expected beat of the underlying rhythm. Isolated PJCs

may occur in healthy people and are insignificant. *Junctional tachycardia* is a junctional rhythm with a rate greater than 60 bpm. It is caused by increased automaticity of AV nodal tissue. The ventricular rate is usually less than 140 bpm. Both rhythms are most commonly associated with digoxin toxicity, hypoxia, ischaemia or electrolyte imbalances.

Ventricular arrhythmias

Ventricular arrhythmias originate in the ventricles. Because the ventricles pump blood into the pulmonary and systemic vasculature, any disruption of their rhythm can affect cardiac output and tissue perfusion. A wide and bizarre QRS complex (greater than 0.12 sec) is a characteristic feature of ventricular arrhythmias. This occurs because ventricular ectopic impulses begin and travel outside normal conduction pathways. Other characteristics include no relationship of the QRS complex to a P wave, increased amplitude of the QRS complex, an abnormal ST segment and a T wave deflected in the opposite direction from the QRS complex.

PREMATURE VENTRICULAR CONTRACTIONS *Premature ventricular contractions (PVCs)* are ectopic ventricular beats that occur before the next expected beat of the underlying rhythm. They usually do not reset the atrial rhythm and are followed by a full compensatory pause. PVCs often have no significance in people without heart disease. Frequent, recurrent or multifocal PVCs may be associated with an increased risk of lethal arrhythmias. PVCs result from either enhanced automaticity or a re-entry phenomenon. They may be triggered by anxiety or stress; tobacco, alcohol or caffeine use; hypoxia, acidosis and electrolyte imbalances; sympathomimetic drugs; coronary heart disease; heart failure; mechanical stimulation of the heart (e.g. the insertion of a cardiac catheter); or reperfusion after thrombolytic therapy. The incidence and significance of PVCs is greatest after myocardial infarction.

PVCs may be isolated or occur in a specific pattern. Two PVCs in a row are called a *couplet* or *paired* PVCs. Three consecutive PVCs (a *triplet* or *salvo*) is a short run of ventricular tachycardia. *Ventricular bigeminy* is characterised by a PVC following each normal beat; a PVC noted every third beat is called *ventricular trigeminy*. When the ventricular impulse arises from one ectopic site, all PVCs look the same (*monomorphic*) and are called *unifocal* PVCs. *Multifocal* PVCs arise from different ectopic sites and appear different from one another on the ECG (*polymorphic*).

The frequency and patterns of PVCs can be indicative of myocardial irritability and the risk of a lethal arrhythmia. The following are considered warning signs in the person with acute heart disease (e.g. an acute MI):

- PVCs that develop within the first 4 hours of an MI
- frequent PVCs (six or more per minute)
- couplets or triplets
- multifocal PVCs
- R-on-T phenomenon (PVCs or ventricular pacing falling on the T wave).

In people without heart disease, isolated PVCs usually are insignificant and do not require treatment. Individuals may complain of feeling that their hearts 'skip a beat' or of palpitations. In person with pre-existing heart disease, PVCs may indicate a drug toxicity or an increased risk of lethal arrhythmias and cardiac arrest. The risk is greatest following acute MI.

VENTRICULAR TACHYCARDIA *Ventricular tachycardia (VT, V tach)* is a rapid ventricular rhythm defined as three or more consecutive PVCs. Ventricular tachycardia may occur in short bursts or 'runs', or may persist for more than 30 seconds (sustained ventricular tachycardia). The rate is greater than 100 bpm and the rhythm is usually regular. Re-entry is the usual electrophysiological mechanism responsible for VT. Myocardial ischaemia and infarction are the most common predisposing factors for VT. It also is associated with cardiac structural disorders such as valvular disease, rheumatic heart disease or cardiomyopathy. It may occur in the absence of heart disease and with anorexia nervosa, metabolic disorders and drug toxicity.

Non-sustained VT may occur paroxysmally and convert back to an effective rhythm spontaneously. The person may experience a fluttering sensation in the chest or complain of palpitations and brief shortness of breath. People in sustained VT generally develop signs and symptoms of decreased cardiac output and haemodynamic instability, including severe hypotension, a weak or non-palpable pulse, and loss of consciousness. Allowed to continue, VT can deteriorate into ventricular fibrillation. Sustained ventricular tachycardia is a medical emergency that requires immediate intervention, particularly in people with cardiac disease.

Torsades de pointes is a type of ventricular tachycardia associated with *long QT syndrome*, a prolongation of the QT interval. Long QT syndrome may be genetic or acquired, occurring secondarily to electrolyte disruptions, myocardial infarction, cocaine use, liquid protein diets, medications or other conditions. In torsades de pointes, the QRS complexes vary in size, shape and amplitude (see Figure 29.7). Individuals with torsades de pointes may have multiple bursts or episodes of ventricular tachycardia or may develop ventricular fibrillation and sudden cardiac death (Dave, 2014).

VENTRICULAR FIBRILLATION *Ventricular fibrillation (VF, V fib)* is extremely rapid, chaotic ventricular depolarisation causing the ventricles to quiver and cease contracting; the heart

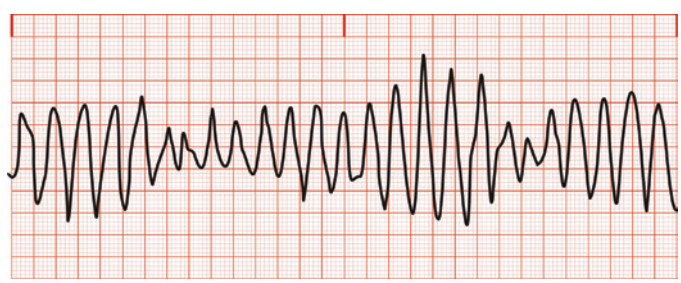


FIGURE 29.7 ■ Torsades de pointes. Note the wide and bizarre QRS complexes of varying size, shape (morphology) and amplitude

does not pump. This is known as **cardiac arrest**; it is a medical emergency requiring immediate intervention with cardiopulmonary resuscitation (CPR). VF is the most common initial rhythm and is found in 60–80% of all cardiac arrests. Survival rates are poor; however, strong predictors of a favourable outcome are the return of spontaneous circulation within 20 minutes and being less than 60 years of age (Wibrandt et al., 2015).

Ventricular fibrillation is usually triggered by severe myocardial ischaemia or infarction. It is the terminal event in many disease processes or traumatic conditions. Ventricular fibrillation may be precipitated by a single PVC or may follow VT. Other causes of VF include digoxin toxicity, reperfusion therapy, antiarrhythmic drugs, hypokalaemia and hyperkalaemia, hypothermia, metabolic acidosis, mechanical stimulation (as with the insertion of cardiac catheters or pacing wires) and electric shock.

Clinically, loss of ventricular contractions results in absence of a palpable or audible pulse. The person loses consciousness and stops breathing as perfusion ceases. The ECG shows grossly irregular, bizarre complexes with no discernible rate or rhythm.

Atrioventricular conduction blocks

Conduction defects that delay or block transmission of the sinus impulse through the AV node are called *atrioventricular conduction blocks*. Impaired conduction may result from tissue injury or disease, increased vagal (parasympathetic) tone, drug effects or a congenital defect. AV conduction blocks vary in severity from benign to severe.

FAST FACTS

- First-degree AV block = delayed conduction through the AV node and a long PR interval.
- Second-degree AV block = complete blockage of *some* impulses through the AV node; some P waves are not followed by a QRS complex.
- Third-degree AV block = complete blockage of *all* impulses through the AV node; no relationship between P waves and QRS complexes.

FIRST-DEGREE AV BLOCK *First-degree AV block* is a benign conduction delay that generally poses no threat, has no symptoms and requires no treatment. Impulse conduction through the AV node is slowed, but all atrial impulses are conducted to the ventricles. It may result from injury or infarct of the AV node, other cardiac diseases or drug effects. The ECG shows all characteristics of NSR, except the PR interval is greater than 0.20 second.

SECOND-DEGREE AV BLOCK *Second-degree AV block* is characterised by failure to conduct one or more impulses from the atria to the ventricles. Two patterns of second-degree AV block are seen, identified as type I and type II.

Second-degree AV block—type I *Type I second-degree AV block (Mobitz type I* or *Wenckebach phenomenon)* is characterised by a repeating pattern of increasing AV conduction delays until an impulse fails to conduct to the ventricles. On the ECG, PR intervals progressively lengthen until one QRS complex is not conducted or dropped. The ventricular rate remains adequate to maintain cardiac output and the person usually is asymptomatic. Mobitz type I AV block usually is transient, associated with acute MI or drug intoxication (e.g. digoxin, beta-blockers or calcium channel blockers). It rarely progresses to complete heart block.

Second-degree AV block—type II *Type II second-degree AV block (Mobitz type II)* involves intermittent failure of the AV node to conduct an impulse to the ventricles without preceding delays in conduction. The PR interval remains constant, but not all P waves are followed by QRS complexes (e.g. there may be two P waves for every QRS). Conduction through the His–Purkinje system usually is delayed as well, causing a widened QRS complex. Mobitz type II block is frequently associated with acute anterior wall MI and a high rate of mortality (Grossman & Mattson, 2013). Manifestations of Mobitz type II block depend on the ventricular rate. Pacemaker therapy may be required to maintain the cardiac output.

THIRD-DEGREE AV BLOCK *Third-degree AV block (complete heart block)* occurs when atrial impulses are completely blocked at the AV node and fail to reach the ventricles. As a result, the atria and ventricles are controlled by different and independent pacemakers, with separate rates and rhythms. The ventricular impulse arises from either junctional fibres (with a rate of 40 to 60 bpm) or a ventricular pacemaker at a rate of less than 40 bpm. The width of the QRS complex depends on the location of the escape pacemaker. The QRS is wide and the rate is slow when the rhythm arises distal to the bundle of His.

Third-degree block is frequently associated with an inferior or anteroseptal myocardial infarction. Other causes include congenital conditions, acute or degenerative cardiac disease or damage, drug effects and electrolyte imbalances. The slow escape rhythm significantly affects cardiac output, causing manifestations such as syncope (known as a *Stokes–Adams attack*), dizziness, fatigue, exercise intolerance and heart failure. Third-degree AV block is life threatening and requires immediate intervention to maintain adequate cardiac output.

AV DISSOCIATION Complete dissociation of atrial and ventricular rhythms can occur in conditions other than third-degree AV block. The two primary factors leading to AV dissociation are severe sinus bradycardia and a lower pacemaker (junctional or ventricular) that competes with or exceeds the normal sinus rhythm. AV dissociation may result from acute myocardial ischaemia or infarction, cardiac surgery or drug effects. The ECG shows separate and competing atrial (P waves) and ventricular (QRS complexes) rhythms.

Intraventricular conduction blocks

Once the impulse enters the ventricles, its conduction through the right and left bundle branches may be impaired (*bundle branch block*). As a result, the impulse is conducted more slowly than normal through the ventricles. On the ECG, the QRS complex is prolonged. Its appearance varies, depending on the affected bundle (right or left). Typically, no clinical

manifestations are associated with bundle branch block unless it occurs in conjunction with an AV block.

INTERPROFESSIONAL CARE

Cardiac arrhythmias may be either benign or critical: recognising lethal arrhythmias is a matter of life and death. Major goals of care include identifying the arrhythmia, evaluating its effect on physical and psychosocial wellbeing and treating underlying causes. This may involve correcting fluid and electrolyte or acid–base imbalances; treating hypoxia, pain or anxiety; administering antiarrhythmic medications; or mechanical and surgical interventions.

Diagnosis

Diagnostic tests for arrhythmias include the electrocardiogram, cardiac monitoring and electrophysiology studies. Laboratory tests such as serum electrolytes, drug levels and ABGs may be done to help identify the cause of the arrhythmia.

ELECTROCARDIOGRAM The 12-lead ECG may be required to accurately diagnose an arrhythmia. It also provides information about underlying disease processes, such as myocardial infarction or other cardiac disease. The ECG may also be used to monitor the effects of treatment. See Chapter 28 for more information about the 12-lead ECG.

CARDIAC MONITORING Cardiac monitoring allows continuous observation of the cardiac rhythm. It is used in many different circumstances (see Box 29.2). Different types of ECG monitoring are employed for different situations.

Continuous cardiac monitoring Continuous monitoring of the cardiac rhythm is provided by bedside and central monitoring stations. Electrodes placed on the person's chest attach to cables connected to a monitor. The heart rate and rhythm is displayed on a bedside monitor connected to a central monitoring station. The central station allows simultaneous monitoring of many individuals within a nursing unit. Alarms on both bedside and central monitors warn of potential problems such as very rapid or very slow heart rates. Alarm limits are preset by the nurse for the individual. Procedure 29.1 describes how to place a person on cardiac monitoring.

Telemetry may be used in acute care settings when the person is ambulatory. Chest electrodes are connected to a portable transmitter worn around the neck or waist; the ECG is transmitted electronically to a central monitoring station for continuous monitoring.

Holter monitoring Individuals often complain of palpitations or other heart symptoms but are asymptomatic during evaluation in a hospital or community-based setting. Ambulatory or Holter monitoring may be used to identify intermittent arrhythmias, to detect silent ischaemia, to monitor the effects of treatment and to assess pacemaker or automatic cardioverter-defibrillator function. Electrodes are applied and the leads attached to a portable telemetry monitor that records and stores all electrical activity. Individuals are instructed to leave the electrode pads in place during monitoring, record any cardiac symptoms or events in a journal (such as chest pain, palpitations, syncope) and are told when to return to the clinic. After the prescribed period, usually 48 to 72 hours, the person returns and the monitor is removed. Diary entries are compared to the recorded heart rhythms to identify the effects of arrhythmias.

ELECTROPHYSIOLOGY STUDIES *Diagnostic cardiac electrophysiology (EP) procedures* are used to identify arrhythmias and their causes. EP studies are used to analyse components of the conduction system, identify sites of ectopic stimulation and evaluate the effectiveness of treatment. EP procedures can be used both for diagnosis and as a therapeutic intervention.

In the electrophysiology laboratory, electrode catheters are guided by fluoroscopy into the heart through the femoral or brachial vein. The timing and sequence of electrical activation during normal and abnormal (aberrant) rhythms is observed and measured. Electrical stimulation may be used to induce arrhythmias similar to the person's clinical arrhythmia (Greenberg, 2015). Following diagnosis, an EP procedure may be used to treat the arrhythmia—for example, by overdrive pacing (stimulating the person's heart rate to a rate faster than that of the tachyarrhythmia) to break the arrhythmia's cycle or to perform ablative therapy to destroy the ectopic site. See the section on cardiac mapping and catheter ablation for further information.

Nursing care for the individual undergoing an EP procedure is similar to that for a percutaneous coronary revascularisation (see 'Nursing care of the person having PTCA' on page 1001). The procedure and expected sensations are explained. The person remains awake during the procedure; anxiolytic medications or sedatives are given to reduce apprehension. Intravenous heparin may be given during the procedure to reduce the risk of thromboembolism.

BOX 29.2 Indications for cardiac monitoring

- Perioperative monitoring of heart rate and rhythm
- Detecting and identifying arrhythmias
- Monitoring the effects of cardiac and non-cardiac diseases on the heart
- Monitoring people with potentially life-threatening conditions:
  a. Major trauma (especially cardiac trauma)
  b. Dissecting aneurysm
  c. Acute myocardial infarction
  d. Heart failure
  e. Shock
  f. Other emergency conditions
- Evaluating responses to procedures and interventions:
  a. Drug therapies
  b. Diagnostic procedures
  c. Ablative techniques
  d. Angioplasty or cardiac catheterisation
  e. Cardiac surgery
  f. Pacemaker function
  g. Automatic implantable cardioverter-defibrillator function

PROCEDURE 29.1 Initiating cardiac monitoring

GATHER SUPPLIES

- Bedside monitor and cable or telemetry unit with fresh battery
- Electrodes—self-adherent, pre-gelled, disposable
- Lead wires
- Washcloth, soap and towel
- Alcohol prep pads
- Dry gauze pads or ECG prep pads

BEFORE THE PROCEDURE

Explain the reason for ECG monitoring. Reassure person that changes in heart rhythm can be noted and immediately treated if necessary. Explain that loose or disconnected lead wires, poor electrode contact, excessive movement, electrical interference or equipment malfunction may trigger alarms and alert the staff, allowing correction of the problem. Reassure that movement is allowed, within activity restrictions, while on the monitor. Explain skin preparation procedure. Provide for privacy and drape appropriately.

PROCEDURE

1. Follow standard precautions.
2. Check equipment for damage (i.e. fraying, bent or broken wires). Connect lead wires to cable and secure connections.
3. Select electrode sites on the chest wall, avoiding areas of excessive movement, joints, skin creases, scar tissue or other lesions.
4. Clean sites with soap and water and dry thoroughly. Alcohol may be used to remove skin oils; allow the skin to dry for 60 seconds after use.
5. Gently rub the site with a dry gauze pad or ECG prep pad to remove dead skin cells, debris and residue.
6. Open the electrode package; peel the backing from the electrode and check to ensure that the centre of the pad is moist with conductive gel.
7. Apply electrode pads, pressing firmly to ensure contact.
8. Attach leads and position cable with sufficient slack for comfort. Place the telemetry unit (if used) in gown pouch or pocket.
9. Assess ECG tracing on the monitor, adjusting settings as needed.
10. Set monitor alarm limits typically at 20 bpm higher and lower than the person's baseline rate. Turn alarms on and leave on at all times. Assess immediately if an alarm is triggered.
11. Time and date pads with every change.

AFTER THE PROCEDURE

Monitor periodically for comfort. Assess electrode and lead wire connections as needed. Remove and apply new pads every 24 to 48 hours or whenever the pad becomes dislodged or non-adherent. Clean gel residue from previous site and document skin condition under the pads. Choose an alternative site if the skin appears irritated or blistered. Document ECG strips according to unit policy and/or doctor's order, as well as when the cardiac rhythm or the person's condition changes (especially with complaints of chest pain, decreased level of consciousness or changes in vital signs). Note the date, time, personal identification, monitor lead, duration of PR and QT intervals, and rhythm interpretation on each ECG strip.

Complications of EP procedures are infrequent, but include fatal ventricular fibrillation, cardiac perforation and major venous thrombosis (Greenberg, 2015). Careful post-procedure monitoring is vital.

Medications

The goal of drug therapy is to suppress arrhythmia formation. No drug has been found to be completely safe and effective. Antiarrhythmic drugs are primarily used for acute treatment of arrhythmias, although they may also be used to manage chronic conditions. The overall goal of therapy is to maintain an effective cardiac output by stabilising cardiac rhythm.

It is important to remember that virtually all antiarrhythmic drugs also have proarrhythmic effects; that is, they can worsen existing arrhythmias and precipitate new ones. Because of this tendency, the higher mortality rates demonstrated in people receiving antiarrhythmic medications, and the increasing safety and availability of interventional techniques, antiarrhythmic medications are used sparingly.

Most antiarrhythmic drugs are classified by their effects on the cardiac action potential. Most are class I drugs or fast sodium channel blockers. By blocking sodium channels, these drugs slow impulse conduction in the atria and ventricles. This class is further divided into subclasses A, B and C. Class II drugs are beta-blockers, which decrease SA node automaticity, AV conduction velocity and myocardial contractility. Class III agents block potassium channels, delaying repolarisation and prolonging the relative refractory period. Class IV drugs are calcium channel blockers. Their effect is similar to that of beta-blockers. Adenosine and digoxin do not fit within the major classes. Both drugs reduce SA node automaticity and slow AV conduction. Magnesium also falls outside the major classes, but is used to treat arrhythmias. The 'Medication administration' box below identifies common antiarrhythmic drugs within each class and the nursing implications in caring for people receiving these drugs.

Drugs that affect the autonomic nervous system may also be used to treat arrhythmias. Sympathomimetics, such as adrenaline, stimulate the heart, increasing both heart rate and contractility. Anticholinergic agents such as atropine are used to decrease vagal tone and increase the heart rate. Magnesium sulfate is an unclassified drug that has been shown to be safe and effective in the treatment of ventricular tachycardias.

MEDICATION ADMINISTRATION Antiarrhythmic drugs

CLASS I DRUGS: SODIUM CHANNEL BLOCKERS

Class IA

Class IA drugs decrease the flow of sodium into the cell and prolong the action potential. This decreases automaticity, slows the rate of impulse conduction and prolongs refractoriness. They are used to treat both supraventricular and ventricular tachycardias.

Class IB

Class IB, or lignocaine-like, drugs decrease the refractory period but have little effect on automaticity. Drugs in this class are used primarily to treat ventricular arrhythmias, including PVCs and ventricular tachycardia.

Class IC

Class IC drugs slow impulse conduction velocity but have little effect on refractoriness. They are used to reduce or eliminate tachyarrhythmias associated with re-entry. Their significant proarrhythmic effects limit their usefulness, but they may be used to treat supraventricular tachycardia.

CLASS II DRUGS: BETA-BLOCKERS

Class II drugs are beta-blockers that decrease automaticity and conduction through the AV node. They also reduce the heart rate and myocardial contractility. They are used to treat supraventricular tachycardia and to slow the ventricular response rate to atrial fibrillation. These drugs may cause bronchospasm and are contraindicated for people with asthma, chronic obstructive pulmonary disease (COPD) or other restrictive or obstructive lung diseases.

CLASS III DRUGS: POTASSIUM CHANNEL BLOCKERS

Class III drugs block potassium channels, prolonging repolarisation and the refractory period. Drugs in this class are used primarily to treat ventricular tachycardia and ventricular fibrillation. Amiodarone may also be used for supraventricular tachycardias.

CLASS IV DRUGS: CALCIUM CHANNEL BLOCKERS

Calcium channel blockers decrease automaticity and AV nodal conduction. They are used to manage supraventricular tachycardias. Like the beta-blockers, calcium channel blockers reduce myocardial contractility.

OTHER DRUGS

Adenosine and digoxin decrease conduction through the AV node and are used to treat supraventricular tachycardias.

Nursing responsibilities

- Obtain baseline data, including vital signs, cardiac rhythm (including rate, PR and QT intervals, and QRS duration) and physical assessment (especially cardiac, neurological and respiratory status).
- Assess medication regimen to identify drugs that may interfere with antiarrhythmic therapy.
- Monitor ECG to evaluate the effectiveness of therapy and to assess for possible arrhythmias precipitated by treatment.
- Immediately report manifestations of drug toxicity:
  - Procainamide—signs of heart failure; conduction delays or ventricular arrhythmias; skin rash, myalgias or arthralgias, flu-like symptoms.
  - Lignocaine—changes in neurological status, such as agitation, confusion, dizziness, nervousness.
  - Amiodarone—pulmonary fibrosis (increasing dyspnoea, cough, hepatic dysfunction—changes in liver function tests, jaundice); vision changes, photosensitivity.
  - Digoxin—anorexia, nausea, vomiting; blurred or double vision; yellow–green halos; new-onset arrhythmias.
- Use an infusion pump to administer intravenous infusions. Monitor the dose and assess its appropriateness (in mg/min or µg/kg/min).

Health education for the person and family

- Take the drug exactly as prescribed. Do not skip or double doses. Check with the medical officer regarding instructions for a missed dose.
- Pulse rates should be taken and recorded daily before rising. The record should be brought to all appointments with healthcare professionals.
- Report irregular pulse rates or rhythms, dizziness, eye pain, changes in vision, skin rashes or colour changes, wheezing or other respiratory problems, or changes in behaviour to the medical officer.

Countershock

Countershock is used to interrupt cardiac rhythms that compromise cardiac output and the person's welfare. Delivery of a direct current charge depolarises all cardiac cells at the same time. This simultaneous depolarisation may stop a tachyarrhythmia and allow the sinus node to recover control of impulse formation. There are two types of countershock: synchronised cardioversion and defibrillation.

SYNCHRONISED CARDIOVERSION

Synchronised cardioversion delivers direct electrical current synchronised with the person's heart rhythm. Synchronisation of the shock with the QRS complex prevents ventricular fibrillation by avoiding current delivery during the vulnerable period of repolarisation. Cardioversion is usually done as an elective procedure to treat supraventricular tachycardia, atrial fibrillation, atrial flutter or haemodynamically stable ventricular tachycardia.

The nurse assists with cardioversion by preparing the individual before the procedure; obtaining any laboratory tests ordered; obtaining and documenting ECG strips prior to, during and after treatment; setting up the equipment; and monitoring the person's response. Procedure 29.2 describes synchronised cardioversion.

Peoples in atrial fibrillation are at high risk of thromboembolism following cardioversion. Loss of atrial contractions with atrial fibrillation leads to blood pooling in the atria, increasing the risk of clot formation. When the atria begin to contract following successful cardioversion, clots may be dislodged, embolising to the pulmonary or systemic circulation. If possible, anticoagulants are given for several weeks before cardioversion is attempted.

PROCEDURE 29.2 Elective synchronised cardioversion

GATHER SUPPLIES

- Cardioverter-defibrillator with ECG cable and monitor
- Conductive gel pads or paste
- Dry gauze pads
- Emergency drug kit and resuscitation equipment
- IV supplies (catheter, solution, administration set)

BEFORE THE PROCEDURE

Explain the purpose of the procedure (to restore an effective cardiac rhythm). Describe the procedure in simple, non-threatening terms. Advise that some discomfort may be felt with each countershock, but a light anaesthetic will be given to minimise discomfort. Witness the signature on the procedure consent form. Document pre-procedure rhythm on an ECG strip. Ensure a patent intravenous access site for emergency drug administration. Keep nil by mouth (NBM) as specified prior to the procedure. Assess acid–base and electrolyte levels (especially potassium, magnesium and calcium) and drug levels if appropriate. Report abnormalities to the doctor prior to the procedure. Document vital signs, level of consciousness and peripheral pulses. Remove any medication patches from the chest and all metallic objects. Assist where necessary with the airway and medications for level of consciousness. Ensure privacy and position the person supine.

PROCEDURE

1. Use standard precautions.
2. Turn on the cardioverter-defibrillator and ECG monitor.
3. Connect the person's ECG cable to the cardioverter. Select a lead with prominent R waves for monitoring (generally lead II).
4. Set cardioverter to 'synchronise' mode. Observe the ECG waveform on the monitor for indications of synchronisation, such as a flashing bold line or a blip. Many units also display the message 'synchronised mode' on the monitor.
5. Place conductive pads on the chest below the right clavicle to the right of the sternum and in the midaxillary line on the left.
6. Turn on the ECG recording strip for a continuous printout during the procedure.
7. Turn oxygen off and remove it.
8. The paddles should be firmly applied to the chest over the conductive pads by the medical officer or the nurse qualified in advanced cardiac life support (ACLS).
9. Charge the paddles to the prescribed energy dose. The machine will beep to indicate that the selected energy level has been reached and that the paddles are ready for discharge.
10. Ensure that no one is touching the person or the bed prior to discharge of the electrical shock. There may be a slight delay in shock delivery as the machine synchronises with the R wave.
11. Assess person's status and ECG rhythm. Assure a patent airway and the presence of a pulse.
12. The procedure may be repeated if unsuccessful. The energy level may be increased with each attempt.
13. Remove conductive pads. Ensure that the skin is clean and dry.

AFTER THE PROCEDURE

Assess the individual for return of consciousness from sedative or cardioversion. Evaluate neurological, cardiovascular and respiratory status. Assess for possible complications, including emboli (especially cerebral), respiratory depression and arrhythmias. Document postcardioversion rhythm strip. Assess skin for burns. Document the procedure and the person's response in the medical record.

DEFIBRILLATION Unlike carefully synchronised cardioversion, *defibrillation* is an emergency procedure that delivers direct current without regard to the cardiac cycle. Ventricular fibrillation is immediately treated as soon as the arrhythmia is recognised. Early defibrillation has been shown to improve survival in people experiencing VF.

Defibrillation can be delivered by external or internal paddles or pads. Conductive gel pads or paste is applied and external paddles or pads are placed on the chest wall at the apex and base of the heart (see Figure 29.8). Internal paddles are applied directly on the heart and may be used in surgery, the emergency department or critical care. Internal defibrillation is done only by a doctor; external defibrillation may be performed by any healthcare provider who has been trained in the procedure. Automatic external defibrillators (AEDs) are available on most hospital units to allow early defibrillation for cardiac arrest (see Procedure 29.3).

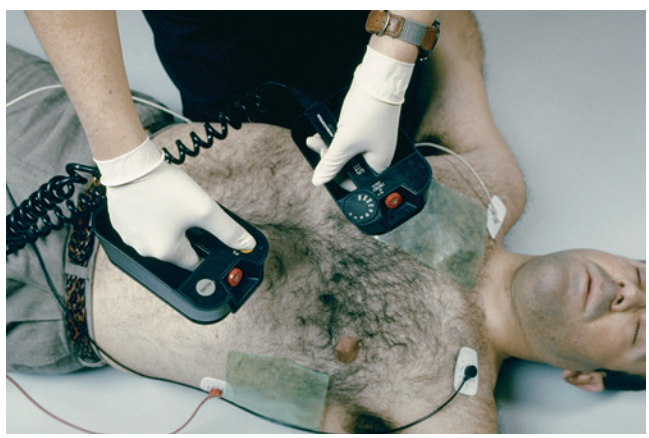


FIGURE 29.8 ■ Placement of pads for defibrillation

Source: Floyd Jackson/Pearson Education.

PROCEDURE 29.3 Emergency external defibrillation

GATHER SUPPLIES

- Automatic external defibrillator or defibrillator with ECG cable and monitor
- Conductive gel pads or paste
- Dry gauze pads
- Emergency medications and emergency trolley with pacemaker, airway management equipment and oxygen supplies

BEFORE THE PROCEDURE

Verify the lethal arrhythmia, such as pulseless VT, VF or asystole. Initiate the cardiac arrest (code) procedure and obtain the defibrillator. If one is not immediately available, begin CPR until the emergency cart and defibrillator are brought to the bedside. Place person in supine position on a firm surface.

PROCEDURE

1. Turn on the defibrillator. Set it in *defibrillation* mode.
2. Turn ECG recording on for a continuous printout of events during the procedure.
3. Set the energy level and charge the paddles. Monophasic—360 J for all shocks. Biphasic—200 J for all shocks.
4. Place conductive pads on the chest.
5. Position the paddles, holding them firmly on the chest wall.
6. Ensure that no one is touching the person or the bed. State, 'All clear.'
7. Depress the button on each paddle simultaneously to discharge the energy.
8. Immediately resume CPR.
9. Evaluate cardiac rhythm and for a pulse after approximately 2 minutes.
10. Implement ACLS protocols.

AFTER THE PROCEDURE

If the arrhythmia is successfully converted, evaluate and support neurological, cardiovascular and respiratory status. Monitor and titrate any intravenous infusions as ordered. Maintain ventilatory support as needed. Evaluate skin for burns. Obtain blood for laboratory analysis as ordered. Monitor vital signs and ECG continuously. Transfer to the critical care unit as indicated. Provide support and information to the individual and their family.

Pacemaker therapy

A **pacemaker** is a pulse generator used to provide an electrical stimulus to the heart when the heart fails to generate or conduct its own at a rate that maintains the cardiac output. The pulse generator is connected to *leads* (insulated wires) passed intravenously into the heart or sutured directly to the epicardium. The leads sense intrinsic electrical activity of the heart and provide an electrical stimulus to the heart when necessary (pacing).

Pacemakers are used to treat both acute and chronic conduction defects such as third-degree AV block. They also may be used to treat bradyarrhythmias and tachyarrhythmias.

Temporary pacemakers use an external pulse generator (see Figure 29.9) attached to a lead threaded intravenously into the right ventricle, to temporary pacing wires implanted during cardiac surgery or to external conductive pads placed on the chest wall for emergency pacing.

Permanent pacemakers use an internal pulse generator placed in a subcutaneous pocket in the subclavian space or abdominal wall. The generator connects to leads sewn directly onto the heart (*epicardial*) or passed transvenously into the heart (*endocardial*). Epicardial pacemakers (see Figure 29.10) require surgical exposure of the heart. Leads may be placed during cardiac surgery or using a small subxiphoid incision to expose the heart. Transvenous pacemaker leads are positioned in the right heart via the cephalic, subclavian or jugular vein (see Figure 29.11). Local anaesthesia can be used for permanent pacemaker insertion.

Pacemakers are programmed to stimulate the atria or the ventricles (*single-chamber pacing*) or both (*dual-chamber pacing*). Table 29.8 defines terms used to describe pacemaker modes and functions. The most commonly used pacemakers either: (1) sense activity in and pace the ventricles only; or (2) sense activity in and pace both the atria and the ventricles. Dual-chamber or *atrioventricular sequential pacing* stimulates both chambers of the heart in sequence. AV pacing imitates the normal sequence of atrial contraction followed by ventricular contraction, improving cardiac output.

Pacing is detected on the ECG strip by the presence of pacing artefacts (see Figure 29.12). A sharp spike is noted before the P wave with atrial pacing and before the QRS complex with ventricular pacing. Pacing spikes are seen before both the P wave and QRS complex in AV sequential pacing. Capture is noted if there is a contraction of the chamber immediately following the pacer spike. Problems in sensing, pacing and capture are noted in Table 29.9.

Care of the person with a temporary or permanent pacemaker focuses on monitoring for pacemaker malfunctioning, maintaining safety (see Box 29.3) and preventing infection and postoperative complications. Nursing care for the person having a pacemaker implant is outlined in the following box.

Implantable cardioverter-defibrillator

The true incidence of sudden cardiac death in Australia and New Zealand is unknown. The implantable cardioverter-defibrillator (ICD) detects life-threatening changes in the cardiac rhythm and automatically delivers an electric shock to convert the arrhythmia back into a normal rhythm. ICDs are used for sudden death survivors, people with recurrent

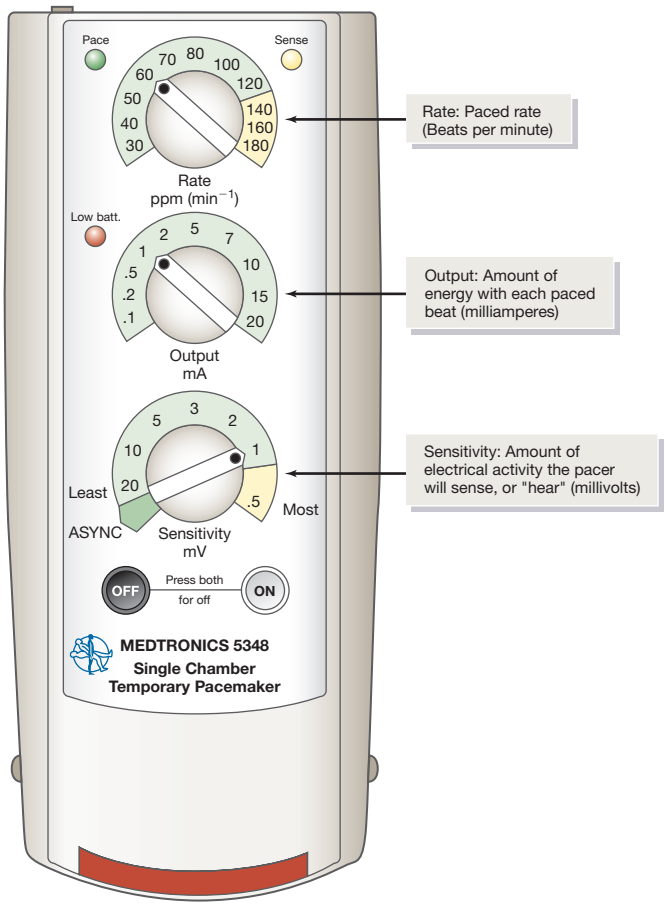


FIGURE 29.9 ■ Programmable settings on a temporary pacemaker

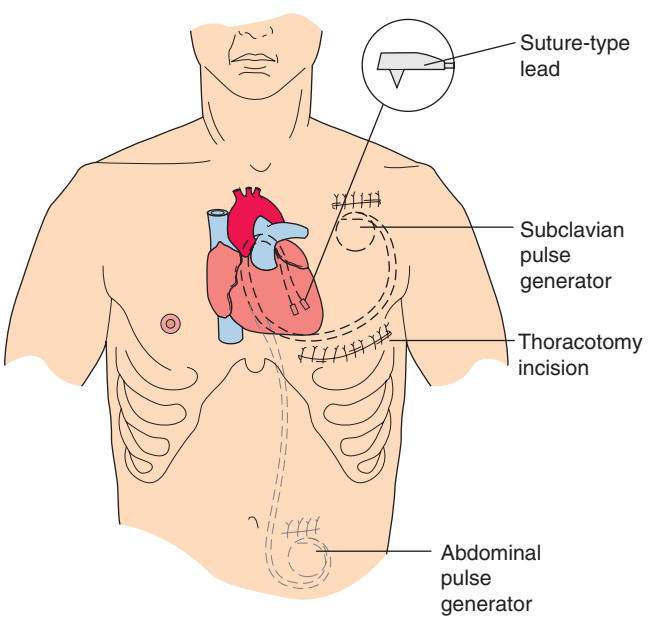


FIGURE 29.10 ■ A permanent epicardial pacemaker. The pulse generator may be placed in subcutaneous pockets in the subclavian or abdominal regions

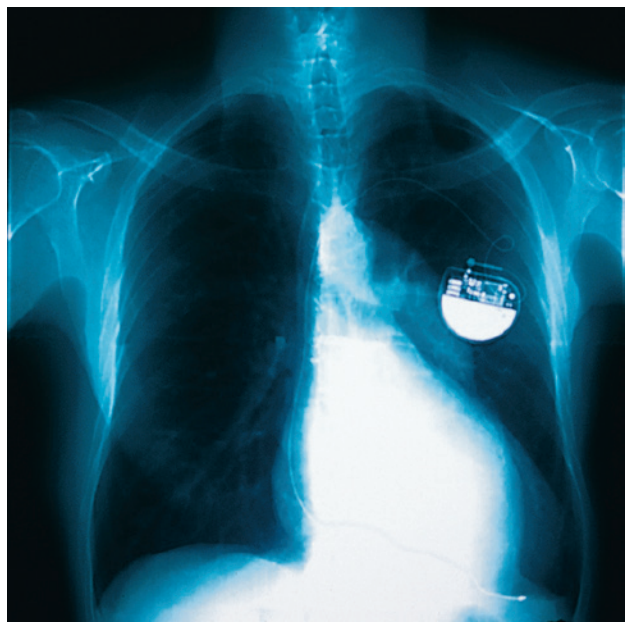


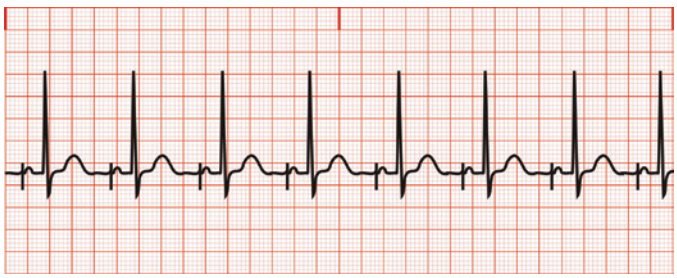
FIGURE 29.11 ■ A permanent transvenous (endocardial) pacemaker with the lead placed in the right ventricle via the subclavian vein

Source: © SPL/Science Source.

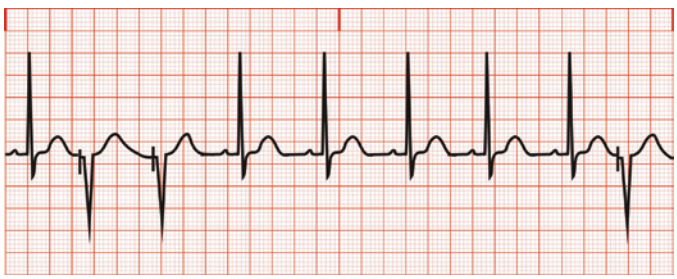
TABLE 29.8 Terms used to describe pacemaker functions

TERM	DEFINITION
Asynchronous pacing	A setting that results in delivery of a pacing stimulus at a set rate regardless of intrinsic cardiac activity.
Base rate	The rate at which the unit paces when no intrinsic cardiac activity is detected.
Capture	The ability of the stimulus to generate depolarisation through the myocardium.
Demand pacing	A setting that results in delivery of a pacing stimulus only when the intrinsic rate falls below the unit's base rate.
Dual-chamber pacing	The pacing of both atria and ventricles; mode most often used in permanent implanted pacing devices.
Lead	An insulated wire that is capable of sensing intrinsic cardiac activity and delivering a pacing stimulus.
Output	The electrical stimulus delivered by the unit.
Pacing spike	A vertical line occurring on the ECG with every pacemaker stimulus.
Sensing	The unit's ability to sense and respond to intrinsic cardiac activity.
Single-chamber pacing	The pacing of only atria or ventricles; mode most often used in the context of temporary pacing activities.

Source: Adapted from Kenny, T. (2015). *The Nuts and Bolts of Implantable Device Therapy Pacemakers.* Oxford, UK: John Wiley & Sons, Ltd.



A



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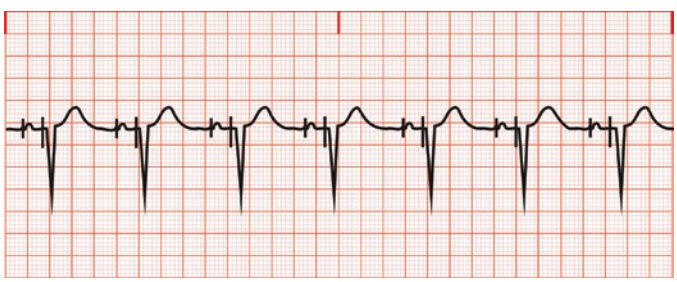


C

FIGURE 29.12 ■ Pacing artefacts. *A*, Atrial pacing and ventricular sensing. Note the pacer spike preceding the P wave. *B*, Ventricular demand pacing. Note the absence of pacer spikes when the person's natural rhythm predominates. *C*, Atrioventricular pacing. Note the pacer spikes preceding both P waves and QRS complexes

ventricular tachycardia and individuals with demonstrated risk factors for sudden death. ICDs can deliver a shock as needed, provide pacing on demand and can store ECG records of tachycardic episodes (Beyerbach, 2014).

A pulse generator connected to lead electrodes for rhythm detection and current delivery is implanted in the left pectoral region. The lead is threaded transvenously to the apex of the right ventricle. The ICD is programmed to sense a change in heart rate or rhythm. When it detects a potentially lethal rhythm, it shocks the heart to convert the rhythm. The device can be programmed or reprogrammed at the bedside as necessary. The ICD may be tested prior to discharge.

Local or general anaesthesia is used and the individual may be discharged within 24 hours. The lithium-powered battery must be surgically replaced every 5 years. Complications and nursing care are similar to that for an individual having a permanent pacemaker implant.

The person may briefly lose consciousness before the device discharges, typically regaining consciousness quickly after the episode. Some people report significant discomfort with ICD discharge (like a 'blow to the chest'). A person in direct contact with the individual when the device discharges may experience a tingling sensation.

BOX 29.3 Safety for the person with a temporary pacemaker

- Ensure that all electrical equipment in use has a grounded plug; do not use adapters or extension cords.
- Encourage the use of battery-powered equipment (e.g. electric razor).
- Remove any damaged electrical equipment from the unit, including equipment that:
  a. has been abused (e.g. has been dropped or in which liquid has been spilled)
  b. has given anyone a shock
  c. has damaged electrical cords or plugs
  d. has other evidence of impaired function, such as a hot smell during use, or control knobs that are loose or do not consistently produce the expected response.
- Wear gloves when handling the pacemaker electrodes or wires (to prevent microshock).
- Insulate pacemaker terminals and pacing wires with non-conductive, moisture-proof material (e.g. a rubber glove).
- Test the pacemaker battery prior to use.
- Keep a spare pacemaker, cable, batteries and battery tester available at all times.
- Immediately report any apparent deviation from expected pacemaker function.

Cardiac mapping and catheter ablation

Cardiac mapping and catheter ablation are used to locate and destroy an ectopic focus. These diagnostic and therapeutic measures use electrophysiology techniques and can be performed in the cardiac catheterisation laboratory. *Cardiac mapping* is used to identify the site of earliest impulse formation in the atria or the ventricles. Intracardiac and extracardiac catheter electrodes and computer technology are used to pinpoint the ectopic site on a map of the heart. These same catheters can be used to deliver the ablative intervention.

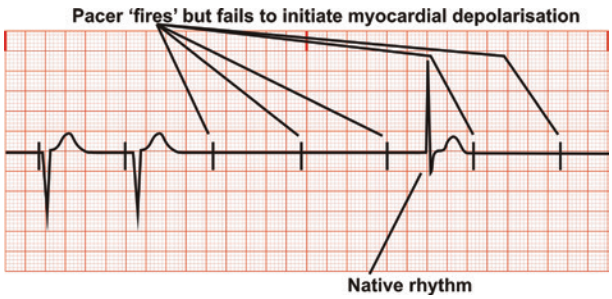
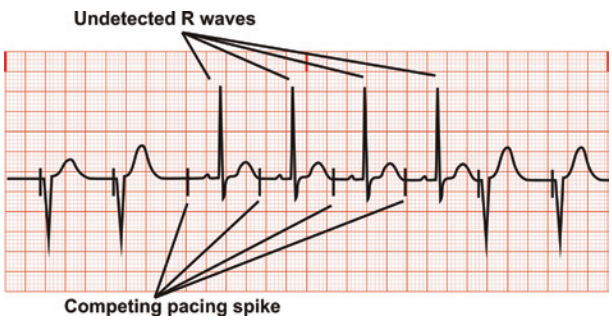
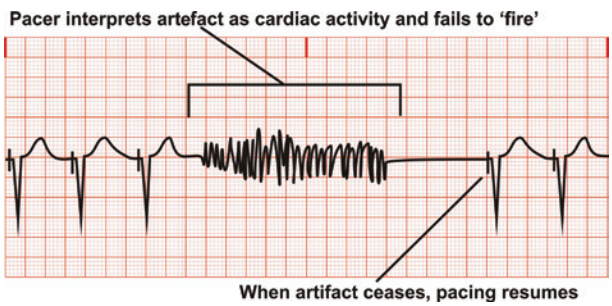
Ablation destroys, removes or isolates an ectopic focus. In most instances, radio-frequency energy produced by high-frequency alternating current is used to create heat as it passes through tissue. Catheter ablation is used to treat supraventricular tachycardias, atrial fibrillation and flutter, and, in some cases, paroxysmal ventricular tachycardia.

Anticoagulant therapy may be started after catheter ablation to reduce the risk of clot formation at the ablation site.

Other therapies

In addition to medications and interventional techniques, other measures may be used to treat selected arrhythmias. Vagal manoeuvres that stimulate the parasympathetic nervous system may be used to slow the heart rate in supraventricular

TABLE 29.9 Selected pacing problems

PROBLEM	POSSIBLE CAUSE	RESOLUTION
Failure to capture  Occurs when there is no atrial (or ventricular complex—whichever is appropriate) following the pacing spike. The pacing unit has delivered an impulse but the target myocardium did not achieve depolarisation.	The output mA* is set too low on the pacing unit.	Support HR & BP†. Increase the mA* on the unit until capture is obtained.
	The tip may have migrated or is not well enough in contact with the target myocardium.	Support HR & BP†. Reposition the individual lying on the other side (sometimes this will facilitate capture again). Observe for other signs that the tip may have migrated (i.e. hiccupping—the tip may have migrated to the diaphragm).
	The lead may have fractured.	Support HR & BP†. Change pacing leads.
	The threshold in the target myocardium may have increased due to chemical or metabolic changes making the cells less responsive to the mA* set.	Support HR & BP†. Increase the mA* on the unit until capture is obtained. Change the polarity of the pacing wires in the pacing box (place the negative where the positive had been and the positive where the negative wire had been).
	The pacemaker unit's battery may be low.	Change pacing box to programmed replacement unit and then replace batteries in old unit ready for use.
Failure to sense (undersensing)  Occurs when the pacing unit does not detect the intrinsic/native event (P wave or QRS complex) and delivers an impulse. If the pacing unit delivers an impulse onto the myocardium during repolarisation it can cause fibrillation in the target tissue (i.e. if it occurs during atrial repolarisation it can cause atrial fibrillation and if it occurs during ventricular repolarisation it can cause ventricular fibrillation).	The person's intrinsic/native myocardial voltage is low. The sensitivity is set too low (mV‡ is set too high) on the pacing unit.	Increase the sensitivity of the pacing unit (decrease mV‡) until sensing is identified.
	A wire or lead may be dislodged, loose or fractured, or the pacemaker may be malfunctioning.	Check all connections and change pacing leads. Try another pacing unit.
	The pacemaker unit's battery may be low.	Change pacing box to programmed replacement unit and then replace batteries in old unit ready for use.
	The person may be experiencing an electrolyte imbalance.	Check the person's electrolytes.
Failure to pace (oversensing)  Occurs when the pacing unit does not deliver an impulse even though the person's intrinsic/native rhythm is inadequate.	The sensitivity is set too high (mV‡ is set too low) on the pacing unit.	Support HR & BP†. Decrease the sensitivity of the pacing unit (increase mV‡) until appropriate sensing is identified.
	A wire or lead may be dislodged, loose or fractured, or the pacemaker may be malfunctioning.	Support HR & BP†. Check all connections and change pacing leads. Try another pacing unit.
	The pacemaker unit's battery may be low.	Support HR & BP†. Change pacing box to programmed replacement unit and then replace batteries in old unit ready for use.
	The person may be experiencing some cellular or electrolyte issues.	Support HR & BP†. Change the polarity of the pacing wires in the pacing box. Check the person's electrolytes.

* mA = milliamperes. † Support HR & BP (i.e. support the person's heart rate and blood pressure by other means where possible). ‡ mV = millivolts.

NURSING CARE OF THE PERSON having a permanent pacemaker implant

PREOPERATIVE CARE

- Provide routine preoperative care and teaching as outlined in Chapter 3.
- Assess knowledge and understanding of the procedure, clarifying and expanding on existing knowledge as needed. *Clarifying knowledge, providing information and conveying emotional support reduces anxiety and fear and allows the individual to develop a realistic outlook regarding pacer therapy.*
- Place ECG monitor electrodes away from potential incision sites. *This helps preserve skin integrity*.
- Teach range-of-motion (ROM) exercises for the affected side. *ROM exercises of the affected arm and shoulder prevent stiffness and impaired function following pacemaker insertion.*

POSTOPERATIVE CARE

- Provide postoperative monitoring, analgesia and care as outlined in Chapter 3.
- Obtain a chest x-ray as ordered. *A postoperative chest x-ray is used to identify lead location and detect possible complications, such as pneumothorax or pleural effusion.*
- Position for comfort. Minimise movement of the affected arm and shoulder during the initial postoperative period. *Restricting movement minimises discomfort on the operative side and allows the leads to become anchored, reducing the risk of dislodging.*
- Assist with gentle ROM exercises at least three times daily, beginning 24 hours after pacemaker implantation. *ROM exercises help restore normal shoulder movement and prevent contractures on the affected side.*
- Monitor pacemaker function with cardiac monitoring or intermittent ECGs. Report pacemaker problems to the doctor:
  - Failure to pace. *This may indicate battery depletion, damage or dislodgement of pacer wires or inappropriate sensing.*
  - Failure to capture (the pacemaker stimulus is not followed by ventricular depolarisation). The electrical output of the pacemaker may not be adequate or the lead may be dislodged.
  - Improper sensing (the pacemaker is firing or not firing, regardless of the intrinsic rate). *This increases the risk of decreased cardiac output and arrhythmias*.
  - Runaway pacemaker (a pacemaker firing at a rapid rate). *This may be due to generator malfunction or problems with sensing.*
  - Hiccups. A lead positioned near the phrenic nerve or diaphragm can stimulate it, causing hiccups. *Hiccups may occur in extremely thin individuals or may indicate a medical emergency with perforation of the right ventricle by the pacing electrode tip.*
- Assess for arrhythmias and treat as indicated. *Until the catheter is 'seated' or adheres to the myocardium, its movement may cause myocardial irritability and arrhythmias. Fibrotic tissue develops within 2 to 3 days.*
- Document the date of pacemaker insertion, the model and type, and settings. *This information is important for future reference.*
- Immediately report signs of potential complications, including myocardial perforation, cardiac tamponade, pneumothorax or haemothorax, emboli, skin breakdown, bleeding, infection, endocarditis or poor wound healing. (See Chapter 30 for more information about cardiac tamponade and endocarditis, and Chapter 35 for pneumothorax and haemothorax.) *Early identification of complications allows for aggressive intervention.*
- Provide a pacemaker identification card, including the manufacturer's name, model number, mode of operation, rate parameters and expected battery life. *This card provides a reference for the individual and future healthcare providers.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

Provide appropriate teaching for the person and their family about:

- Placement of the pacemaker generator and leads in relation to the heart.
- How the pacemaker works and the rate at which it is set.
- Battery replacement. Most pacemaker batteries last 6 to 12 years. Replacement requires an outpatient surgery to open the subcutaneous pocket and replace the battery.
- How to take and record the pulse rate. Instruct to assess pulse daily before arising and notify the doctor if 5 or more bpm slower than the preset pacemaker rate.
- Incision care and signs of infection. Bruising may be present following surgery.
- Signs of pacemaker malfunction to report, including dizziness, fainting, fatigue, weakness, chest pain or palpitations.
- Activity restrictions as ordered. This usually is limited to contact sports (which may damage the generator) and avoiding heavy lifting for 2 months after surgery.
- Resuming sexual activity as recommended by the medical officer. Avoid positions that cause pressure on the site.
- Avoiding tight-fitting clothing over the pacemaker site to reduce irritation and avoid skin breakdown.
- Carrying the pacemaker identification card at all times and wearing a MedicAlert® bracelet or tag.
- Notifying all care providers of the pacemaker.
- Not holding or using certain electrical devices over the pacemaker site, including household appliances or tools, garage door openers, antitheft devices or burglar alarms. Pacemaker will set off airport security detectors; notify security officials of its presence.
- Maintaining follow-up care with the doctor as recommended.

tachycardias. These manoeuvres include *carotid sinus massage* and the *Valsalva manoeuvre*. Carotid sinus massage is performed only by a medical officer during continuous cardiac monitoring. Excessive slowing of the heart rate may result. The Valsalva manoeuvre—forced exhalation against a closed glottis (e.g. bearing down)—increases intrathoracic pressure and vagal tone, slowing the pulse rate.

Nursing care

Caring for the person with cardiac arrhythmias requires the ability to recognise, identify and, in some cases, promptly treat the arrhythmia. The urgency of intervention is determined by the effects of the arrhythmia on the individual. Nursing care focuses on maintaining cardiac output, monitoring the response to therapy and teaching. See also the following nursing care plan for a person with superventricular tachycardia.

Health promotion

Health promotion measures to prevent coronary heart disease also reduce the risk of arrhythmias. In most cases, arrhythmias develop as a result of ischaemic or structural changes in the heart, rather than in isolation. Advise people who are at risk or who complain of occasional palpitations or 'flutters' in their chest to reduce their intake of caffeine and other sympathetic nervous system stimulants, such as excess chocolate.

Assessment

Assessment is vital before treating any suspected arrhythmia. What appears to be ventricular tachycardia on the monitor may be the individual scratching or brushing their teeth. Apparent asystole on the monitor may be due to a loose electrode patch. Similarly, a heart rate of 52 bpm may not affect the overall cardiac output in some individuals. Review Chapter 28 for complete assessment of the person with a cardiac problem.

- *Health history:* complaints of palpitations (ask for further definition of palpitations), 'fluttering' sensations or a sensation of the heart racing; episodes of dizziness, light headedness or syncope (fainting); timing (duration, time of day); correlation with food or beverage intake, activity; presence of chest pain, shortness of breath or other associated symptoms; history of heart or endocrine disease (such as hyperthyroidism); current medications.
- *Physical examination:* LOC; vital signs, including apical pulse for a full minute; regularity and amplitude of peripheral pulses; colour; presence of dyspnoea, adventitious lung sounds; ECG rhythm analysis; oxygen saturation levels.

Nursing diagnoses and interventions

The effect of the arrhythmia on cardiac output is the priority of nursing care. Other potential nursing diagnoses related to arrhythmias may include *Ineffective tissue perfusion, Activity intolerance*, and *Fear* or *Anxiety*.

Decreased cardiac output

Arrhythmias can affect cardiac output. Bradycardias decrease cardiac output if the stroke volume does not increase to compensate for the slow heart rate. Tachycardia reduces diastolic filling time, affecting stroke volume and coronary artery perfusion. Loss of the atrial kick in junctional rhythms, atrial fibrillation and AV blocks also decreases ventricular filling and cardiac output. In ventricular fibrillation, loss of ventricular contractions causes cardiac arrest and no cardiac output.

- Assess for decreased cardiac output: decreased LOC; tachycardia; tachypnoea; hypotension; low oxygen saturation; diaphoresis; low urine output; cool, clammy, mottled skin; pallor or cyanosis; diminished peripheral pulses. Initial signs of decreased cardiac output may be subtle, such as decreased LOC. *Early recognition of the arrhythmia's effect on cardiac output facilitates appropriate treatment and may prevent further adverse effects.*

CONSIDERATION FOR PRACTICE

Before treating any arrhythmia, assess the individual, not just the monitor! Loose electrode pads, disconnected leads or cables, and muscle movement can simulate critical arrhythmias. The person's condition is the best indicator of the need for treatment.

- Monitor ECG; obtain a rhythm strip and 'post event' ECG strip every shift and when rhythm changes occur. *Documenting cardiac rhythm provides a record of disease progression and treatment effectiveness.*
- Assess for underlying causes of arrhythmias, such as hypovolaemia, hypoxia, anaemia, vagal stimulation or medications. *Sinus tachycardia often develops in response to tissue hypoxia. Vagal stimulation (such as the Valsalva manoeuvre) can precipitate bradycardia.*
- Assess serum electrolytes (especially potassium, calcium and magnesium) and digoxin and antiarrhythmic drug levels as indicated. Report abnormal values. *Electrolyte imbalances affect cardiac depolarisation and repolarisation and may cause arrhythmias. Toxic levels of digoxin and antiarrhythmic drugs can precipitate further arrhythmias. Impaired renal or hepatic function increases the risk of toxicity, as does ageing.*

CONSIDERATION FOR PRACTICE

Assess vital signs, ECG and oxygen saturation every 5 to 15 minutes during acute arrhythmic episodes and during antiarrhythmic drug infusions. These data provide a record of cardiac output during the arrhythmia. Antiarrhythmic drugs can adversely affect heart rate, rhythm and blood pressure, further decreasing cardiac output.

- Be prepared to administer antiarrhythmic medications as indicated. Implement advanced cardiac life support (ACLS) protocols as needed. *Emergency drugs should be readily available, especially on units with individuals at high risk of a life-threatening cardiac event.* See Table 29.7 and the 'Medication administration: antiarrhythmic drugs' box on page 1029 for drugs used to treat common arrhythmias that may affect cardiac output.
- If appropriate, instruct to perform the Valsalva manoeuvre (bear down as if straining or coughing) for supraventricular tachycardia or ventricular tachycardia without angina.

NURSING CARE PLAN A person with supraventricular tachycardia

Sandra Banks, a 53-year-old woman from the Torres Strait Islands, is admitted to the cardiac unit with complaints of palpitations, light headedness and dyspnoea. Her history reveals rheumatic fever at age 12 with subsequent rheumatic heart disease and mitral stenosis. An intravenous line is in place and she is receiving oxygen.

ASSESSMENT

Ms Banks is moderately anxious. Her ECG shows supraventricular tachycardia (SVT) with a rate of 154. Vital signs: T 37.1°C, R 26, BP 95/60. Peripheral pulses weak but equal, mucous membranes pale pink, skin cool and dry. Fine crackles noted in both lung bases. A loud S_3 gallop and a diastolic murmur are noted. Ms Banks is still complaining of palpitations and states that she 'feels so nervous and weak and dizzy'. Adenosine was trialled with no effect followed by an order for verapamil 2.5 mg IV. Preparations for synchronised cardioversion may need to be organised if the drug therapy does not control the ventricular rate.

DIAGNOSES

- *Decreased cardiac output* related to inadequate ventricular filling associated with rapid tachycardia.
- *Ineffective tissue perfusion: cerebral/cardiopulmonary/peripheral* related to decreased cardiac output.
- *Anxiety* related to unknown outcome of altered health state.

PLANNING

- Prepare propofol infusion (or intraprocedure medications as ordered).
- Document pretreatment vital signs, level of consciousness and peripheral pulses.
- Place emergency trolley with drugs and airway management supplies in person's area.

Expected outcomes

- Maintain adequate cardiac output and tissue perfusion.
- Demonstrate a ventricular rate within normal limits and stable vital signs.
- Verbalise reduced anxiety.
- Verbalise an understanding of the rationale for the treatment measures to control the heart rate.

IMPLEMENTATION

- Assist with cardioversion as indicated.
- Document procedure and postcardioversion rhythm and response to intervention.
- Provide oxygen per nasal prongs at 4 L/min.
- Explain the importance of rapidly reducing the heart rate. Explain the cardioversion procedure and encourage questions.
- Encourage verbalisation of fears and concerns. Answer questions honestly, correcting misconceptions about the disease process, treatment or prognosis.

EVALUATION

- Assess LOC, level of sedation, cardiovascular and respiratory status and skin condition following cardioversion.
- Continuously monitor ECG for rate, rhythm and conduction. Assess vital signs and associated symptoms with changes in ECG. Report findings to medical officer.

Intravenous verapamil lowers Ms Banks's heart rate to 138 for a short time, after which it increases to 164 with BP of 82/64. Her cardiologist, Dr Mullins, performs carotid sinus massage. The ventricular rate slows to 126 for 2 minutes, revealing atrial flutter waves, and then returns to a rate of 150. Dr Mullins explains the treatment options, including synchronised cardioversion.

Ms Banks is lightly sedated and synchronised cardioversion is performed. One countershock converts Ms Banks to regular sinus rhythm at 96 bpm with BP 112/60.

Ms Banks is sleepy from the sedation but recovers without incident. She states that she feels 'much better', and her vital signs return to her normal levels. She remains in NSR with a rate of 86 to 92 for the remainder of her hospital stay. Dr Mullins places Ms Banks on frusemide to treat manifestations of mild heart failure.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the scientific basis for using carotid massage to treat supraventricular tachycardias? Was this an appropriate manoeuvre in the case of Ms Banks?
2. What other treatment options might the doctor have used to treat Ms Banks's supraventricular tachycardia if she had been asymptomatic with stable vital signs?
3. Why might the adenosine failed? What critical interventions are required to increase the likelihood of success with this drug? (Hint: related to drug half life.)
4. Develop a teaching plan for Ms Banks related to her prescription for frusemide.

REFLECTION ON THE NURSING PROCESS

1. What is the likelihood of Ms Banks experiencing SVT again? What education is required for an individual who presents with SVT?
2. Outline what you have learned from this case study that you will apply to your future practice.

Vagal manoeuvres stimulate the parasympathetic system and may terminate some arrhythmias. The Valsalva manoeuvre is contraindicated if chest pain occurs with the arrhythmia.

- Prepare to assist with cardioversion. Prepare the individual per orders or hospital protocol (see Procedure 29.2). Explain the procedure to reduce anxiety. Have emergency equipment readily available. *Elective or emergency cardioversion is a treatment of choice for certain arrhythmias.*

CONSIDERATION FOR PRACTICE

On recognising ventricular fibrillation and cardiac arrest, begin emergency basic life support procedures. Call for help. Initiate advanced cardiac life support (ACLS) protocols if it is within your scope and training to do so. Assist with resuscitation measures as directed. Cardiac output ceases with ventricular fibrillation. Immediate or early defibrillation has been shown to have the greatest impact on survival following cardiac arrest.

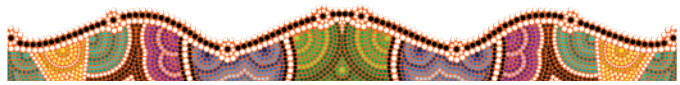
- After cardiac arrest, transfer to critical care. Perform and document head-to-toe assessment; obtain laboratory tests, 12-lead ECG and chest X-ray as ordered; monitor and maintain oxygenation and intravenous infusions; and monitor vital signs and cardiac rhythm. *The period following resuscitation is critical, necessitating careful monitoring. Post-arrest assessment allows comparison of the individual's condition with pre-arrest status and may identify CPR-related injuries. Correcting electrolyte disturbances, hypoxia and acid–base imbalances is important to prevent further arrhythmias and potential adverse effects on cardiac output. Intravenous access is crucial to maintain drug infusions. Haemodynamic monitoring may be instituted. The 12-lead ECG documents myocardial status and the chest x-ray provides information about pulmonary status and possible thoracic injury due to CPR.*
- Notify the family of significant changes in the person's condition, providing up-to-date information. Prepare family members prior to visits by explaining interventions (such as invasive tubes, a ventilator or additional equipment) implemented since the last visit. *Concern for the family and significant others is part of holistic nursing. Researchers studying the needs of families have found that one of the most important needs was information about their loved one's condition. Individuals and their families need and appreciate honest communication and compassionate care. Preparing the family for critical changes in the person's condition and plan of care helps them to cope with a situational crisis.*

Community-based care

Arrhythmias have a significant physical and psychological impact on the person and all their family members. Many of these individuals and their families are under a great deal of stress from frequent hospitalisations, experimentation with therapies, frustration and the fear of sudden cardiac death. A major teaching effort focuses on coping strategies and lifestyle changes as well as specific management of prescribed therapies. Include the following topics as appropriate when teaching the person and their family for home care:

- function, maintenance, precautions and signs of malfunction or complications of any implanted device such as a pacemaker or ICD
- monitoring pulse rate and rhythm
- activity or dietary restrictions, and any potential effects of the arrhythmia or its treatment on lifestyle
- medication management to reduce the risk of arrhythmias, including the desired and potential adverse effects of antiarrhythmic drugs
- specific instructions related to planned diagnostic tests or procedures
- the importance of follow-up visits with the cardiologist
- the importance of obtaining CPR training for the individual and their family members.

In addition, discuss fears related to treatment or implanted devices, such as that of shocking a significant other during close contact or sexual activity. Explain that if a shock occurs, the partner may feel a slight buzz or tingling but should not be harmed. Refer to and encourage the person and their family to attend a peer support group for the specific condition.



THE PERSON WITH SUDDEN CARDIAC DEATH

Sudden cardiac death (SCD) is defined as unexpected death occurring within 1 hour of the onset of cardiovascular symptoms. The presence of left ventricular dysfunction caused by any condition is the greatest predictor of SCD and usually results from ventricular fibrillation or pulseless ventricular tachycardia (Sovari, 2014). These rhythms are within the group of rhythms that cannot sustain life and can be called a cardiac arrest. *Cardiac arrest* is the sudden collapse, loss of consciousness and cessation of effective circulation that precedes biological death. Worldwide fewer than 6% of out-of-hospital cardiac arrest victims survive.

Almost 50% of all deaths due to coronary heart disease are attributed to SCD. A significant per cent of sudden cardiac deaths can be attributed to CHD. Other cardiac pathologies such as cardiomyopathy and valvular disorders also may lead to SCD. Non-cardiac causes of sudden death include electrocution, pulmonary embolism and rapid blood loss from a ruptured aortic aneurysm.

Ventricular fibrillation is the most common arrhythmia associated with sudden cardiac death, accounting for the majority of cardiac arrests. Sustained severe bradyarrhythmias, *asystole* or cardiac standstill, and pulseless electrical activity (organised cardiac electrical activity without a mechanical response) are responsible for most of the remaining SCDs. Selected cardiac and non-cardiac causes of sudden cardiac death are listed in Box 29.4.

Risk factors for SCD are those associated with coronary heart disease (see the first section of this chapter). Advancing age and male gender are powerful risk factors. After age 65, the gap between male and female incidence of SCD narrows. Individuals with arrhythmias such as recurrent ventricular tachycardia (VT) may have a higher risk of SCD. Women with acute myocardial infarction, however, are more likely to present with cardiac arrest and cardiogenic shock than with ventricular tachycardia. Recent Australian statistics demonstrate that in the first decade of the 21st century, even though deaths from cardiovascular diseases have decreased for both men and women, overall there are still more women dying of cardiac arrest and CVD than men (ABS, 2015).

Pathophysiology

Evidence of coronary heart disease with significant atherosclerosis and narrowing of two or more major coronary arteries is found in the majority of SCD victims. An acute change in cardiovascular status precedes cardiac arrest by up to 1 hour; however, often the onset is instantaneous or abrupt. An

BOX 29.4 Selected causes of sudden cardiac death

Cardiac causes

- Coronary heart disease
- Reperfusion following ischaemia
- Myocardial hypertrophy
- Cardiomyopathy
- Inflammatory myocardial disorders
- Valve disorders
- Primary electrical disorders
- Dissecting or ruptured aortic or ventricular aneurysm
- Cardiac drug toxicity

Non-cardiac causes

- Pulmonary embolism
- Cerebral haemorrhage
- Autonomic dysfunction
- Choking
- Electrical shock
- Electrolyte and acid–base imbalances

imbalance in electrolytes, or autonomic tone, irregular cellular activity, or functional blockages within the conduction system, may contribute to the development of ventricular tachycardiac or ventricular fibrillation (Sovari, 2014).

Various other factors can also contribute to the development of lethal arrhythmia, including abnormalities of myocardial structure or function. Structural abnormalities include infarction, hypertrophy and myopathy. Functional deviations are caused by such factors as ischaemia followed by reperfusion, altered homeostasis, pathological hormone interactions, and toxic effects. The interactions of the two cause myocardial instability and may precipitate fatal arrhythmias.

Manifestations

Sudden cardiac death may be preceded by typical manifestations of acute coronary syndrome or myocardial infarction, including severe chest pain, dyspnoea or orthopnoea, and palpitations or light headedness. The event itself is abrupt, with complete loss of consciousness and death within minutes. If ventricular tachycardia precedes cardiac arrest, consciousness and mentation may be impaired prior to collapse and loss of consciousness.

INTERPROFESSIONAL CARE

The goal of care is to restore cardiac output and tissue perfusion. Treatment measures are initiated as soon as clinical cardiac arrest is verified by the absence of respirations and carotid or femoral pulses. Basic life support and ACLS measures must be instituted within 2 to 4 minutes of cardiac arrest to prevent permanent neurological damage and ischaemic injury to other organs.

Providers trained in use of the *automated external defibrillator (AED)* should immediately defibrillate the person in ventricular fibrillation. Self-adhesive conductive pads attached to connecting cables are positioned on the chest (see Figure 29.13). The Australian Resuscitation Council supports the use of public access defibrillation in association with emergency services and locally provided training.

Cardiopulmonary resuscitation is a mechanical attempt to maintain tissue perfusion and oxygenation using oral resuscitation and external cardiac compressions. All healthcare providers need to be proficient in CPR. The technique should be performed according to the Australian Resuscitation Council's most recent procedures. Healthcare personnel need to be familiar with these protocols and maintain up-to-date knowledge and skills, as the techniques and theory do change.

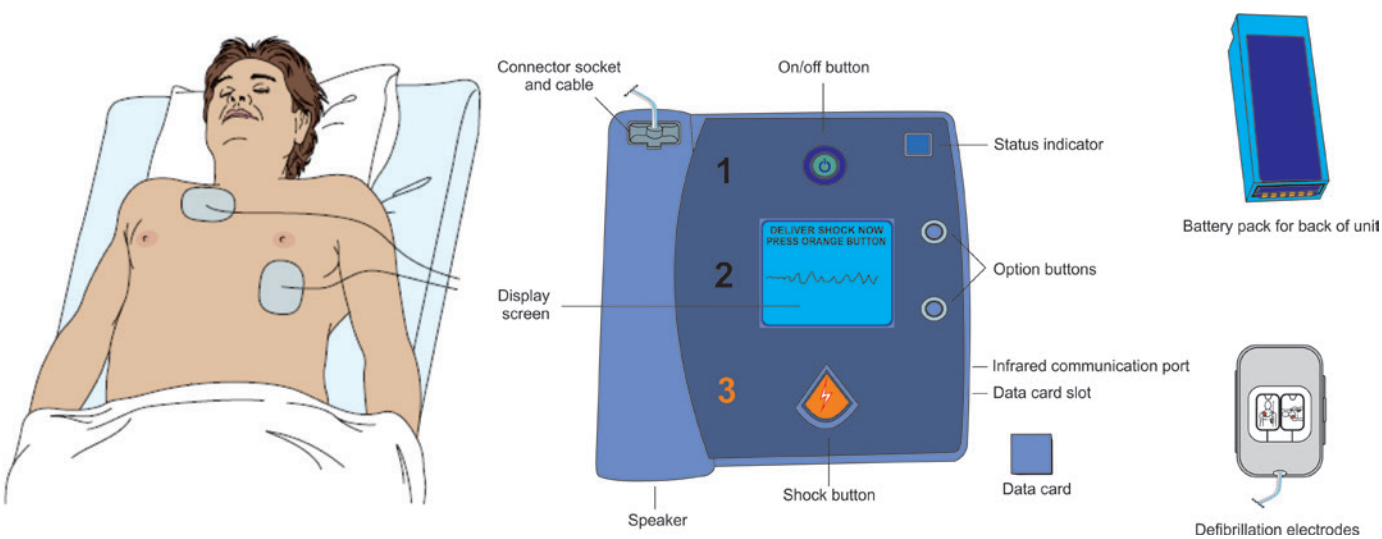


FIGURE 29.13 ■ Schematic of an automated external defibrillator (AED) attached to an individual

Nursing care

Nursing care of the person experiencing sudden cardiac death requires prompt recognition of the event and immediate initiation of basic lifesaving (BLS) and advanced lifesaving (ALS) protocols. As noted before, fast and effective cardiac compressions and early defibrillation of unstable VT and VF are the most important keys to survival of cardiac arrest victims. Important concepts of emergency cardiac care are:

- Treat the person, not the monitor. If a bizarre rhythm is displayed on the monitor, it is important to determine if this is artefact, clinical deterioration or emergency. It is critical to look at the individual. If they are talking, well perfused and in no apparent discomfort, there may be other explanations for what is occurring on the monitor. Recognise signs and symptoms of cardiac compromise early.
- Activate the emergency medical services system (i.e. call a 'code', call 000 or 112 from a mobile phone—even if it has no credit).
- Begin and continue basic cardiac life support principles throughout the resuscitation effort.
- Continually assess the effectiveness of emergency interventions.
- Defibrillate ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) as soon as possible.
- Initiate advanced cardiac life support protocols early if within scope of practice.

The family should not be forgotten during resuscitation. If the family is present, a staff member needs to support significant others and offer a private room in which to await the outcome. If the family is not present, they should be notified that their family member is not doing well and asked to come to the hospital as soon as possible. The outcome should be presented in a careful manner to prevent the family from racing to the hospital, possibly precipitating an automobile crash. Pastoral care or the family's choice of spiritual support is offered to help during this difficult time. Attendance of family members during resuscitation efforts is controversial and depends on institutional protocols and family desires.

After successful resuscitation, the nurse provides care specific to the individual's underlying disease processes and needs. Intravenous infusions such as lignocaine, bretylium or dopamine may be ordered to prevent further arrhythmias and maintain haemodynamic stability.

If the person does not survive the arrest, the nurse provides postmortem care and emotional and spiritual support to the family.

Nursing diagnoses to consider for the person experiencing SCD include the following:

- *Ineffective tissue perfusion: cerebral* related to ineffective cardiac output.
- *Impaired spontaneous ventilation* related to cardiac arrest.
- *Spiritual distress* related to unexplained sudden cardiac death.
- *Disturbed thought processes* related to compromised cerebral circulation.
- *Fear* related to risk of future episodes of sudden cardiac death.

The risk of a future episode of sudden cardiac death requires careful and effective teaching for home care prior to discharge. Discuss the following topics with the person and family:

- risk factor reduction for coronary heart disease
- planned diagnostic studies to identify the cause of SCD and possible interventions
- the risks and benefits of an ICD if appropriate
- the importance of carrying a card at all times listing all current medications and the healthcare provider
- early manifestations or warning signs of cardiac arrest
- the importance of CPR training and maintaining proficiency in performing CPR. (Provide referral to local CPR training providers.)

Nurses can affect death rates from cardiac arrest through community teaching as well. Survival rates from sudden cardiac death improve in communities in which a significant portion of the population is trained in CPR and early response by emergency services is stressed. Work with community groups and individuals can help create a population of people able to perform effective CPR.

CHAPTER HIGHLIGHTS

- Atherosclerosis is the primary underlying process in coronary heart disease, and impaired perfusion of myocardial tissue.
- The risk factors for coronary heart disease are those for atherosclerosis: age, gender and genetic factors; hypertension, diabetes, abnormal blood lipids; cigarette smoking, obesity, physical inactivity and diet; and emerging risk factors such as the metabolic syndrome and homocysteine levels.
- Smoking cessation, exercise, diet modification, weight loss, medications to achieve desired blood lipid values and effective hypertension and diabetes management are the primary treatment measures for coronary heart disease.
- Atherosclerosis of coronary vessels impairs the supply of blood, oxygen and nutrients to the myocardium. Myocardial ischaemia results in the manifestations of coronary heart disease: angina pectoris, acute coronary syndrome and myocardial infarction.
- Stable angina develops with a predictable amount of activity or stress and typically follows an activity–pain, rest–relief pattern. Stable angina often can be managed effectively by medications and risk factor modification. The nursing focus is on education.

- Acute coronary syndrome or unstable angina is characterised by increasingly severe chest pain that occurs unpredictably. Acute coronary syndrome often requires aggressive interventions such as percutaneous coronary revascularisation or coronary artery bypass surgery.
- Myocardial infarction, necrosis of myocardial tissue, results from complete blockage of a coronary artery, usually due to atherosclerotic plaque rupture and thrombus formation. Prompt restoration of blood flow through a revascularisation procedure or administration of a thrombolytic drug to dissolve the blood clot is necessary to preserve functional muscle tissue.
- The nursing focus for individuals with acute coronary syndrome and myocardial infarction is on reducing myocardial work through measures such as pain relief and activity limitation, promoting blood flow and oxygenation through medication and oxygen administration and positioning, and early recognition and treatment of complications.
- Cardiac arrhythmias may arise anywhere in conductive tissue of the myocardium. Arrhythmias may be either benign or fatal, depending on their effect on cardiac output.
- Tachycardias increase the workload of the heart and may interfere with cardiac output if ventricular filling is impaired by the rapid rate.
- Bradycardias can affect cardiac output when the rate is too slow to meet the metabolic needs of the body.
- Atrial fibrillation is a common arrhythmia that can lead to formation of blood clots within the heart and subsequent stroke if these clots lodge in cerebral blood vessels.
- Frequent ventricular arrhythmias may indicate an increased risk of ventricular fibrillation and cardiac arrest.
- AV conduction blocks interfere with conduction of the sinus or atrial impulse through the AV node and to the ventricles.
- Although many antiarrhythmic medications are available, all increase the risk of arrhythmia development, so they are used sparingly.
- The nurse's role in caring for a person with cardiac arrhythmias focuses on prompt identification of the rhythm disruption, assessment of its effect on the person, administration of medications and other treatment measures, and institution of life support procedures as indicated.

CONCEPT CHECK

1 The nurse evaluates her teaching as effective when an individual identifies which of the following modifiable risk factors for coronary heart disease (CHD) as contributing to the greatest extent?
1 obesity
2 diet
3 smoking
4 stress

2 When teaching an individual about simvastatin, the nurse instructs the person to:
1 promptly report muscle pain or tenderness to the doctor
2 consume a diet that includes no more than 20% of kilojoules from saturated fat
3 abstain from alcohol use while taking this drug
4 take the drug with meals to minimise gastric distress

3 When assessing a person with stable angina, the nurse would expect to find:
1 persistent ECG changes
2 correlation between activity level and pain
3 increasing nocturnal pain
4 evidence of impaired cardiac output such as weak peripheral pulses

4 The nurse caring for a person with acute coronary syndrome identifies which of the following nursing diagnoses to be of highest priority?
1 *Anxiety* related to unknown outcome of disorder.
2 *Ineffective health maintenance* related to lack of knowledge about coronary heart disease.
3 *Decreased cardiac output* related to myocardial ischaemia.
4 *Ineffective tissue perfusion: cardiopulmonary* related to underlying coronary heart disease.

5 The nurse caring for a person returning from a coronary angioplasty with stent placement plans which of the following interventions?
1 securing chest tubes to bedding
2 maintaining leg extension on the affected side
3 discontinuing intravenous lines when taking oral fluids
4 treating chest pain with intravenous morphine as needed

6 In planning care for the person with acute myocardial infarction (AMI), the nurse identifies the highest priority goal of care as:
1 stable ECG rhythm
2 ability to verbalise causes and effects of CHD
3 compliance with prescribed bed rest
4 relief of pain

7 Which of the following nursing diagnoses is of highest priority for the person undergoing thrombolytic therapy?
1 *Ineffective protection.*
2 *Ineffective health maintenance.*
3 *Risk of powerlessness.*
4 *Anxiety.*

8 In reviewing laboratory results for a person admitted with acute chest pain, the nurse is most concerned about which of the following?
1 haematocrit 35%
2 AST 65 U/L
3 CK 320 U/L
4 aPTT 35 seconds

9 The nurse recognises second-degree AV block, type II (Mobitz II) and intervenes appropriately when he:
1 records the finding in the chart
2 prepares for temporary pacemaker insertion
3 administers a class IB antiarrhythmic drug
4 places the person in Fowler's position

10 On identifying sinus bradycardia at a rate of 45 bpm, the nurse should:
1 assess mental status and blood pressure
2 assess peripheral pulses on all four extremities
3 determine if an apical–radial pulse deficit is present
4 prepare to administer intravenous atropine

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CHAPTER 30

NURSING CARE OF PEOPLE WITH CARDIAC DISORDERS

MAJELLA HALES

KEY TERMS

LEARNING OUTCOMES

- Relate the interprofessional care requirements for a person with heart failure to the pathophysiology of the condition.
- Explain the risk factors, course and interventions required to competently care for a person with pulmonary oedema.
- Differentiate between the various inflammatory and infective conditions resulting in a person's cardiac disorder.
- Compare and contrast the course and management principles for individuals experiencing various cardiomyopathies.

CLINICAL COMPETENCIES

- Apply knowledge of normal cardiac anatomy and physiology and assessment techniques in caring for the person with cardiac disorders.
- Assess functional health status of individuals with cardiac disorders, documenting and reporting deviations to expected findings.
- Plan, prioritise and provide evidence-based, individualised care for the person with cardiac disorders.
- Administer prescribed medications and treatments to individuals with cardiac disorders.
- Actively participate in planning and coordinating interprofessional care for the person with cardiac disorders.
- Provide appropriate teaching and community-based care for people with cardiac disorders and their families.
- Evaluate the effectiveness of nursing care, revising the plan of care as needed to promote, maintain or restore functional health status of individuals with cardiac disorders.

Cardiac disorders affect the structure and/or function of the heart. These disorders interfere with the heart's primary purpose: to pump enough blood to meet the body's demand for oxygen and nutrients. Disruptions in cardiac function affect the performance of other organs and tissues, potentially leading to organ system failure and death.

Heart failure is the most common cardiac disorder. Pulmonary oedema is also discussed in this chapter. The inclusion and organisation of the inflammatory and infective cardiac disorders proves difficult as there is usually an intimate relationship between these conditions, either as a cause or effect. As such, although conditions such as rheumatic heart disease, the various carditises and cardiomyopathy are often related, they are treated as separate entities in this chapter in order to facilitate sufficient coverage of content.

Before continuing with this chapter, please review the heart's anatomy and physiology, nursing assessment and diagnostic tests in Chapter 28.

HEART FAILURE

Heart failure is a complex syndrome caused by conditions that impair the ejection of oxygen- and nutrient-rich blood from the ventricles. The failure to pump sufficient blood into the systemic circulation results in an inability to meet the body's metabolic demands (Bullock & Hales, 2012). It is often a long-term effect of coronary heart disease (CHD) and myocardial infarction (MI) when left ventricular damage is extensive enough to impair cardiac output (see Chapter 29). Other diseases of the heart also may cause heart failure, including structural and inflammatory disorders. In normal hearts, failure can result from excessive demands placed on the heart. Heart failure may be acute or chronic.

THE PERSON WITH HEART FAILURE

When the heart cannot effectively fill or contract with adequate strength to meet the needs of the body, cardiac output falls and decreased tissue perfusion develops. Initially, compensatory mechanisms are activated to offset reduced cardiac output and restore tissue perfusion. The heart failure will often result in vascular congestion resulting in oedema in either the lungs or the periphery, and is therefore commonly called *congestive heart failure (CHF)*. As the compensatory mechanisms are exhausted, morbidity and mortality risks increase.

Reduced cardiac function may occur due to *impaired myocardial contraction*, from CHD, myocardial ischaemia or infarction, or from a primary cardiac muscle disorder such as cardiomyopathy or myocarditis. Structural cardiac disorders, such as valve disorders or congenital heart defects, and hypertension also can lead to heart failure when the heart muscle is damaged by the long-standing *excessive workload* associated with these conditions. Individuals without a primary abnormality of myocardial function may present with manifestations of heart failure due to *acute excess demands* placed on the myocardium, such as volume overload, hyperthyroidism and massive pulmonary embolus (see Table 30.1). Hypertension and coronary heart disease are the leading causes of heart failure in Australia.

Incidence, prevalence and risk factors

In Australia in 2014, 0.8% of male and 1.1% of female deaths resulted from heart failure. It has dropped to the 11th leading cause of death in males but remains the 8th leading cause of death in females (Australian Bureau of Statistics (ABS), 2015; Australian Institute of Health and Welfare (AIHW), 2014a). Some explanation of the gender disparity in the statistics may be related to greater life expectancy in women, as a significant percentage of heart-failure-related deaths occur in older women (see the 'Nursing care of the older adult' box below). In 2012–2013, 10.1% and 12.1% of all cardiovascular-related hospitalisations were as a result of heart failure for males and females respectively (AIHW, 2015).

Prevalence of heart failure in Aboriginal and Torres Strait Islander Australians is reportedly 1.7 times higher than in non-Indigenous Australians (AIHW, 2011). In a study by McGrady et al. (2012) risk factors for heart failure in Aboriginal and Torres Strait Islander Australians included a BMI > 30 kg/m^2, hypertension, diabetes mellitus, coronary artery disease, history of rheumatic fever or rheumatic heart disease, and asymptomatic left ventricular dysfunction (see the 'Focus on cultural diversity' box below).

The prognosis for an individual with heart failure depends on its underlying cause and how effectively precipitating factors can be treated.

In Aboriginal and Torres Strait Islander Australians, rheumatic heart disease (RHD) plays a significant role in mortality, more than in most other developed countries. Rheumatic heart disease

TABLE 30.1 Selected causes of heart failure

IMPAIRED MYOCARDIAL FUNCTION	INCREASED CARDIAC WORKLOAD	ACUTE NON-CARDIAC CONDITIONS
• Coronary heart disease	• Hypertension	• Volume overload
• Cardiomyopathies	• Valve disorders	• Hyperthyroidism
• Rheumatic fever	• Anaemias	• Fever, infection
• Infective endocarditis	• Congenital heart defects	• Massive pulmonary embolus

NURSING CARE OF THE OLDER ADULT with heart failure

Ageing affects cardiac function. Diastolic filling is impaired by decreased ventricular compliance. With ageing, the heart is less responsive to sympathetic nervous system stimulation. As a result, maximal heart rate, cardiac reserve and exercise tolerance are reduced. Concurrent health problems such as arthritis that affect stamina or mobility often contribute to a more sedentary lifestyle, further decreasing the heart's ability to respond to increased stress.

ASSESSING FOR HOME CARE

The older adult with heart failure may not be dyspnoeic, instead presenting with weakness and fatigue, somnolence, confusion, disorientation or worsening dementia. Dependent oedema and respiratory crackles may or may not indicate heart failure in older adults.

Assess the diet of the older adult. Decreased taste may lead to increased use of salt to bring out food flavours. Limited mobility or visual acuity may cause the older adult to rely on prepared foods that are high in sodium, such as canned soups and frozen meals. Discuss normal daily activities and assess sleep and rest patterns. It is also important to assess the environment for:

- safe roads or neighbourhoods for walking
- access to pharmacy, medical care and assistive services
- a cardiac rehabilitation program or structured exercise programs designed for older adults.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

Teaching for the older adult with heart failure focuses on maintaining function and promptly identifying and treating episodes of heart failure. Teach individuals how to adapt to changes in cardiovascular function associated with ageing, such as:

- allowing longer warm-up and cool-down periods during exercise
- engaging in regular exercise such as walking five or more times a week
- resting with feet elevated (e.g. in a recliner) when fatigued
- maintaining adequate fluid intake
- preventing infection through pneumococcal and influenza immunisations.

FOCUS ON CULTURAL DIVERSITY

Heart failure in the Aboriginal and Torres Strait Islander Australian populations

- Aboriginal and Torres Strait Islander Australians are 1.7 times more likely to experience CHF than non-Indigenous Australians.
- Manifestations of heart failure develop at an earlier age.
- The disease progresses more rapidly.
- More hospital visits are attributed to heart failure.
- The mortality rate is higher than in non-Indigenous Australians.

Heart failure in overseas-born individuals living in Australia

- In proportion to percentage of population, hospital admission rates of Australian residents born in Italy, Poland and Greece are higher than for Australian-born individuals.
- Hospital admission rates of Australian residents born in most other countries are lower than those of Australian-born individuals in proportion to percentage of population.

RHD in the Aboriginal and Torres Strait Islander Australian population

- Aboriginal and Torres Strait Islander Australians are 25 times more likely to develop RHD than non-Indigenous Australians.
- Aboriginal and Torres Strait Islander women are almost twice as likely to develop RHD than Aboriginal and Torres Strait Islander men.

prevalence in Aboriginal and Torres Strait Islander Australians is 26 times higher than in non-Indigenous Australians (AIHW, 2013).

Physiology review

The mechanical pumping action of cardiac muscle propels the blood it receives to the pulmonary and systemic vascular systems for reoxygenation and delivery to the tissues. *Cardiac output (CO)* is the amount of blood pumped from the ventricles in 1 minute. Cardiac output is used to assess cardiac performance, especially left ventricular function. Effective cardiac output depends on adequate functional muscle mass and the ability of the ventricles to work together. Cardiac output normally is regulated by the oxygen needs of the body: as oxygen use increases, cardiac output increases to maintain cellular function. *Cardiac reserve* is the ability of the heart to increase CO to meet metabolic demand. Ventricular damage reduces the cardiac reserve.

Cardiac output is a product of heart rate and stroke volume. *Heart rate (HR)* affects cardiac output by controlling the number of ventricular contractions per minute. It is influenced by the autonomic nervous system, catecholamines and thyroid hormones. Activation of a stress response (e.g. hypovolaemia or fear) stimulates the sympathetic nervous system, increasing heart rate and contractility. Elevated heart rates increase cardiac output. However, very rapid heart rates shorten ventricular filling time (diastole), reducing stroke volume and cardiac output. On the other hand, a slow heart rate reduces cardiac output simply because of fewer cardiac cycles.

Stroke volume, the volume of blood ejected with each heartbeat, is determined by preload, afterload and myocardial contractility. *Preload* is the volume of blood in the ventricles at end diastole (just prior to contraction). The blood in the ventricles exerts pressure on the ventricle walls, stretching muscle fibres.

BOX 30.1 Explaining physiological terms using practical examples

The concepts of preload, the Frank–Starling mechanism, compliance and afterload can be difficult to understand and to explain to people. Use common analogies to make these concepts easier to understand.

- *Preload:* Think about a new rubber band. The further a rubber band is stretched, the greater the force with which it snaps back.
- *Frank–Starling mechanism:* When a rubber band is repeatedly stretched beyond its limit, it loses some elasticity and fails to return to its original shape and size.
- *Compliance:* A new balloon is not very compliant—it takes a lot of work (force) to inflate it. As the balloon is repeatedly inflated and stretched, it becomes more compliant, expanding easily with less force.
- *Afterload:* When a hose is crimped or plugged, more force is required to eject a stream of water out its end.

The greater the blood volume, the greater the force with which the ventricle contracts to expel the blood. End-diastolic volume depends on the amount of blood returning to the ventricles (*venous return*) and the distensibility or stiffness of the ventricles (*compliance*). See Box 30.1.

Afterload is the force needed to eject blood into the circulation. This force must be great enough to overcome arterial pressures within the pulmonary and systemic vascular systems. Increased systemic vascular resistance (e.g. hypertension) increases afterload, impairing stroke volume and increasing myocardial work.

Contractility is the natural ability of cardiac muscle fibres to shorten during systole. Contractility is necessary to overcome arterial pressures and eject blood during systole. Impaired contractility affects cardiac output by reducing stroke volume. The *ejection fraction (EF)* is the percentage of blood in the ventricle that is ejected during systole. A normal ejection fraction is approximately 60%.

Pathophysiology

When the heart begins to fail, mechanisms are activated to compensate for the impaired function and to maintain the cardiac output. The primary compensatory mechanisms are (1) the Frank–Starling mechanism, (2) neuroendocrine responses including activation of the sympathetic nervous system and the renin–angiotensin system, and (3) myocardial hypertrophy. These mechanisms and their effects are summarised in Table 30.2.

Decreased cardiac output initially stimulates aortic baroreceptors, which in turn stimulate the sympathetic nervous system (SNS). SNS stimulation produces both cardiac and vascular responses through the release of noradrenaline. Noradrenaline increases heart rate and contractility by stimulating cardiac beta-receptors. Cardiac output improves as both heart rate and stroke volume increase. Noradrenaline also causes arterial and venous vasoconstriction, increasing venous return to the heart. Increased venous return increases ventricular filling and myocardial stretch, increasing the force of contraction (the Frank–Starling

TABLE 30.2 Compensatory mechanisms activated in heart failure

MECHANISM	PHYSIOLOGY	EFFECT	COMPLICATIONS
Frank–Starling mechanism	The greater the stretch of cardiac muscle fibres, the greater the force of contraction.	• ↑ contractile force leading to ↑ CO	• ↑ myocardial oxygen demand • Limited by overstretching
Neuroendocrine response	↓ CO stimulates the sympathetic nervous system and catecholamine release.	• ↑ HR, BP and contractility • ↑ vascular resistance • ↑ venous return	• ↑ vascular resistance • Tachycardia with ↓ filling time and ↓ CO • ↑ myocardial workload and oxygen demand
	↓ CO and ↓ renal perfusion stimulate renin–angiotensin system. Angiotensin stimulates aldosterone release from adrenal cortex.	• Vasoconstriction and ↑ BP	• Fluid retention • Pulmonary congestion • ↑ preload and afterload
	ADH is released from posterior pituitary. Atrial natriuretic peptide and brain natriuretic peptide are released.	• ↓ sodium excretion • ↓ diuresis • ↑ vascular volume	• Fluid retention • Pulmonary congestion • ↑ myocardial workload
	Blood flow is redistributed to vital organs (heart and brain).	• Vasodilation some locations • Vasoconstriction other locations • ↓ perfusion of other organ systems • ↓ perfusion of skin and muscles	• Renal vasoconstriction and ↓ renal perfusion • Renal failure • Anaerobic metabolism and lactic acidosis • ↑ preload and afterload
Ventricular hypertrophy	↑ cardiac workload causes myocardial muscle to hypertrophy and ventricles to dilate.	• ↑ contractile force to maintain CO	• ↑ myocardial oxygen demand • Myocyte enlargement

mechanism). An ineffective contraction results from overstretching the muscle fibres past their physiological limit.

Blood flow is redistributed to the brain and the heart to maintain perfusion of these vital organs. Decreased renal perfusion causes renin to be released from the kidneys. Activation of the renin–angiotensin system produces additional vasoconstriction and stimulates the adrenal cortex to produce aldosterone and the posterior pituitary to release antidiuretic hormone (ADH). Aldosterone stimulates sodium reabsorption in renal tubules, promoting water retention. ADH acts on the distal tubule to inhibit water excretion and also causes vasoconstriction. The effect of these hormones is significant vasoconstriction and salt and water retention, with a resulting increase in vascular volume. Increased ventricular filling increases the force of contraction, improving cardiac output. The effects of the renin–angiotensin–aldosterone system and ADH release are counterbalanced to a certain extent by two additional hormones. The increased vascular volume and venous return prompted by vasoconstriction and sodium and water retention increase the volume and pressures in the heart. Stimulation of stretch receptors in the atria and ventricles lead to the release of *atrial natriuretic peptide (ANP)* and *brain natriuretic peptide (BNP)* from stores in the atria (ANP and BNP) and ventricles (BNP). These hormones promote sodium and water excretion and inhibit the release of noradrenaline, renin and ADH, with resulting vasodilation. Although beneficial, the effects of these hormones are too weak to completely counteract the vasoconstriction and sodium and water retention that occurs in heart failure.

Ventricular remodelling occurs as the chambers and myocardium adapt to fluid volume and pressure increases. The chambers dilate to accommodate excess fluid resulting from increased vascular volume and incomplete emptying. Initially, this additional stretch causes more effective contractions. *Ventricular hypertrophy* occurs as existing cardiac muscle cells enlarge, increasing their contractile elements (actin and myosin) and force of contraction.

Although these responses may help in the short-term regulation of cardiac output, it is now recognised that they hasten the deterioration of cardiac function. The onset of heart failure is indicated by *decompensation*, the loss of effective compensation. Heart failure progresses due to the very mechanisms that initially maintained circulatory stability.

The rapid heart rate shortens diastolic filling time, compromises coronary artery perfusion and increases myocardial oxygen demand. Resulting ischaemia further impairs cardiac output. Beta-receptors in the heart become less sensitive to continued SNS stimulation, decreasing heart rate and contractility. As the beta-receptors become less sensitive, noradrenaline stores in the cardiac muscle become depleted. In contrast, alpha-receptors on peripheral blood vessels become increasingly sensitive to persistent stimulation, promoting vasoconstriction and increasing afterload and cardiac work.

Initially, ventricular hypertrophy and dilation increase cardiac output, but chronic distension causes the ventricular wall to eventually thin and degenerate. The purpose of hypertrophy is therefore defeated. In addition, chronic overloading of the dilated ventricle eventually stretches the fibres beyond the optimal point for effective contraction. The ventricles continue to dilate to accommodate the excess fluid, but the heart loses the ability to contract forcefully. The heart muscle may eventually become so large that the coronary blood supply is inadequate, causing ischaemia.

Chronic distension exhausts stores of ANP and BNP. The effects of noradrenaline, renin and ADH prevail and the renin–angiotensin pathway is continually stimulated. This mechanism ultimately raises the haemodynamic stress on the heart by increasing both preload and afterload. As heart function deteriorates, less blood is delivered to the tissues and to the heart itself. Ischaemia and necrosis of the myocardium further weaken the already failing heart and the cycle repeats.

In normal hearts, the cardiac reserve allows the heart to adjust its output to meet metabolic needs of the body, increasing the cardiac output by up to five times the basal level during exercise. People with heart failure have minimal to no cardiac reserve. At rest, they may be unaffected; however, any stressor (e.g. exercise, illness) tips the balance between oxygen demand and oxygen supply. Manifestations of activity intolerance when the person is at rest indicate a critical level of cardiac decompensation.

Classifications and manifestations of heart failure

Heart failure is commonly classified in several different ways, depending on the underlying pathology. Classifications include systolic versus diastolic failure, left-sided versus right-sided failure, high-output versus low-output failure, and acute versus chronic failure.

FAST FACTS

Terms used to describe or classify heart failure:

- systolic or diastolic failure
- left ventricular (or sided) or right ventricular (or sided) failure
- low-output or high-output failure
- acute or chronic failure
- forward or backward effects.

Systolic versus diastolic failure

Systolic failure occurs when the ventricle fails to contract adequately to eject a sufficient blood volume into the arterial system. Systolic function is affected by loss of myocardial cells due to ischaemia and infarction, cardiomyopathy or inflammation. The manifestations of systolic failure are those of decreased cardiac output: weakness, fatigue and decreased exercise tolerance.

Diastolic failure results when the heart cannot completely relax in diastole, disrupting normal filling. Passive diastolic filling decreases, increasing the importance of atrial contraction to preload. Diastolic dysfunction results from decreased ventricular compliance due to hypertrophic and cellular changes and impaired relaxation of the heart muscle. Its manifestations result from increased pressure and congestion behind the ventricle: shortness of breath, tachypnoea and respiratory crackles if the left ventricle is affected; distended neck veins, liver enlargement, anorexia and

nausea if the right ventricle is affected. Many individuals have components of both systolic and diastolic failure.

Left-sided versus right-sided failure

Depending on the pathophysiology involved, either the left or the right ventricle may be primarily affected. In chronic heart failure, however, both ventricles typically are impaired to some degree. Coronary heart disease and hypertension are common causes of *left-sided heart failure*, whereas *right-sided heart failure* often is caused by conditions that increase blood pressure in the pulmonary vasculature, such as acute or chronic pulmonary disease. Left-sided heart failure also can lead to right-sided failure as pressures in the pulmonary vascular system increase with congestion behind the failing left ventricle.

As left ventricular function fails, cardiac output falls. Pressures in the left ventricle and atrium increase as the amount of blood remaining in the ventricle after systole increases. These increased pressures impair filling, causing congestion and increased pressures in the pulmonary vascular system. Increased pressures in this normally low-pressure system increase fluid movement from the blood vessels into interstitial tissues and the alveoli (see Figure 30.1).

The manifestations of left-sided heart failure result from pulmonary congestion (*backward effects*) and decreased cardiac output (*forward effects*). Fatigue and activity intolerance are common early manifestations. Dizziness and syncope also may result from decreased cardiac output. Pulmonary congestion causes dyspnoea and cough. The person may develop orthopnoea (difficulty breathing while lying down), prompting use of two or three pillows or a recliner for sleeping. Cyanosis from impaired gas exchange may be noted. On auscultation of the lungs, inspiratory crackles (rales) and wheezes may be heard in lung bases. An S_3 gallop may be present, reflecting the heart's attempts to fill an already distended ventricle.

In right-sided heart failure, increased pressures in the pulmonary vasculature or right ventricular muscle damage impair the right ventricle's ability to pump blood into the pulmonary circulation. The right ventricle and atrium become distended and blood accumulates in the systemic venous system. Increased venous pressures cause abdominal organs to become congested and peripheral tissue oedema to develop (see Figure 30.2).

Dependent tissues tend to be affected because of the effects of gravity; oedema develops in the feet and legs or, if the person is bedridden, in the sacrum. Congestion of gastrointestinal tract

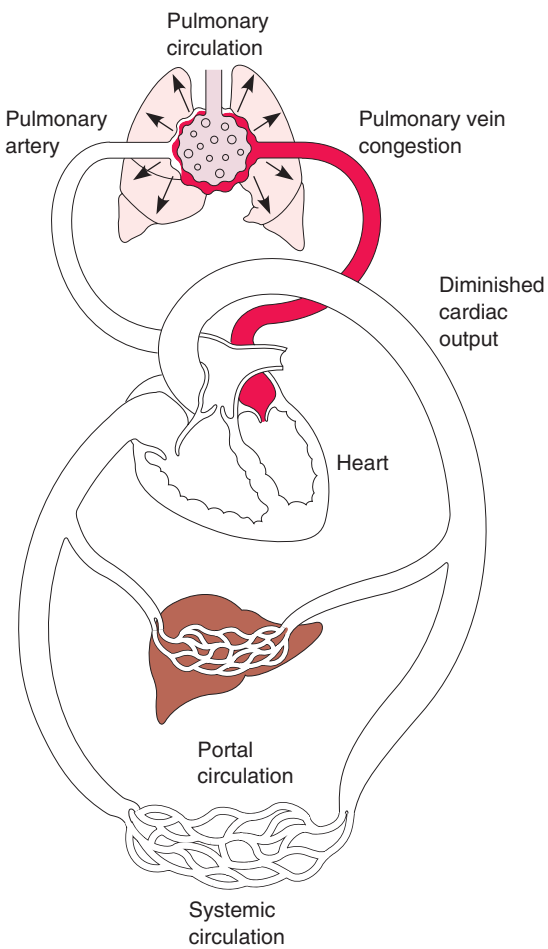


FIGURE 30.1 ■ The haemodynamic effects of left-sided heart failure

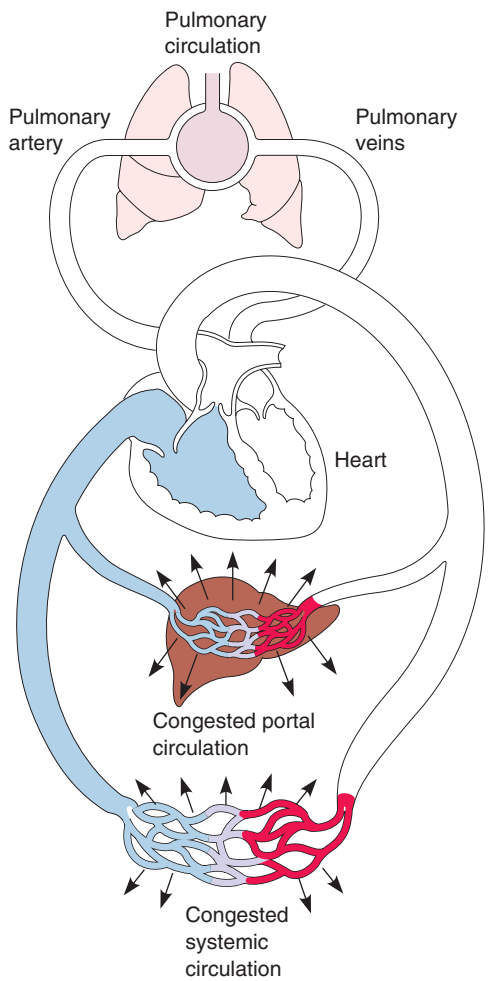


FIGURE 30.2 ■ The haemodynamic effects of right-sided heart failure

vessels causes anorexia and nausea. Right upper quadrant pain may result from liver engorgement. Neck veins distend and become visible even when the person is upright, due to increased venous pressure.

Low-output versus high-output failure

People with heart failure due to coronary heart disease, hypertension, cardiomyopathy and other primary cardiac disorders develop *low-output failure* and manifestations such as those previously described. Individuals in hypermetabolic states (e.g. hyperthyroidism, infection, anaemia or pregnancy) require increased cardiac output to maintain blood flow and oxygen to the tissues. If the increased blood flow cannot meet the oxygen demands of the tissues, compensatory mechanisms are activated to further increase cardiac output, which in turn further increases oxygen demand. Thus, even though cardiac output is high, the heart is unable to meet increased oxygen demands. This condition is known as *high-output failure*.

Acute versus chronic failure

Acute failure is the abrupt onset of a myocardial injury (such as a massive MI) resulting in suddenly decreased cardiac function and signs of decreased cardiac output. *Chronic failure* is a progressive deterioration of the heart muscle due to cardiomyopathies, valvular disease or CHD.

Other manifestations

In addition to the previous manifestations for the various classifications of heart failure, other signs and symptoms commonly are seen.

A fall in cardiac output activates mechanisms that cause increased salt and water retention. This causes weight gain and further increases pressures in the capillaries, resulting in oedema. *Nocturia*, voiding more than one time at night, develops as oedema fluid from dependent tissues is reabsorbed while the person is supine. **Paroxysmal nocturnal dyspnoea (PND)** may develop. PND is a frightening condition in which the person awakens at night acutely short of breath. PND occurs when fluid that has accumulated in the tissues during the day is reabsorbed into the circulation at night, causing fluid overload and pulmonary congestion. Severe heart failure with little or no cardiac reserve may cause dyspnoea at rest, as well as with activity. Both an S_3 and an S_4 gallop may be heard on auscultation.

See below for the multisystem effects of heart failure.

Complications

The compensatory mechanisms initiated in heart failure can lead to complications in other body systems. Congestive hepatomegaly and splenomegaly caused by engorgement of the portal venous system result in increased abdominal pressure, ascites and gastrointestinal problems. With prolonged right-sided heart failure, liver function may be impaired. Myocardial distension can precipitate arrhythmias, further impairing cardiac output. Pleural effusions and other pulmonary problems may develop. Major complications of severe heart failure are cardiogenic shock (described in Chapter 10) and acute pulmonary oedema, a medical emergency described in the next section of this chapter.

INTERPROFESSIONAL CARE

The main goals for care of heart failure are to slow its progression, reduce cardiac workload, improve cardiac function and control fluid retention. Treatment strategies are based on the evolution and progression of heart failure. The National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand (NHF Australia, 2011) produce guidelines for the prevention, detection and management of chronic heart failure. These guidelines are a comprehensive set of recommendations regarding the diagnosis and management of individuals with heart failure. A comprehensive online resource has also been developed for clinicians encompassing all aspects of heart failure assessment and management (National Heart Foundation of Australia, n.d.).

In Australia, symptom classification is still achieved using the New York Heart Association (NYHA) grading of symptoms in chronic heart failure (see Table 30.3).

Diagnosis

Diagnosis of heart failure is based on the history, physical examination and diagnostic findings. The National Heart Foundation of Australia recommends an echocardiogram, electrocardiogram, chest x-ray, full blood count (FBC), plasma, urea, creatinine and electrolytes in all individuals suspected of heart failure (NHF Australia, 2011) (see Table 30.4).

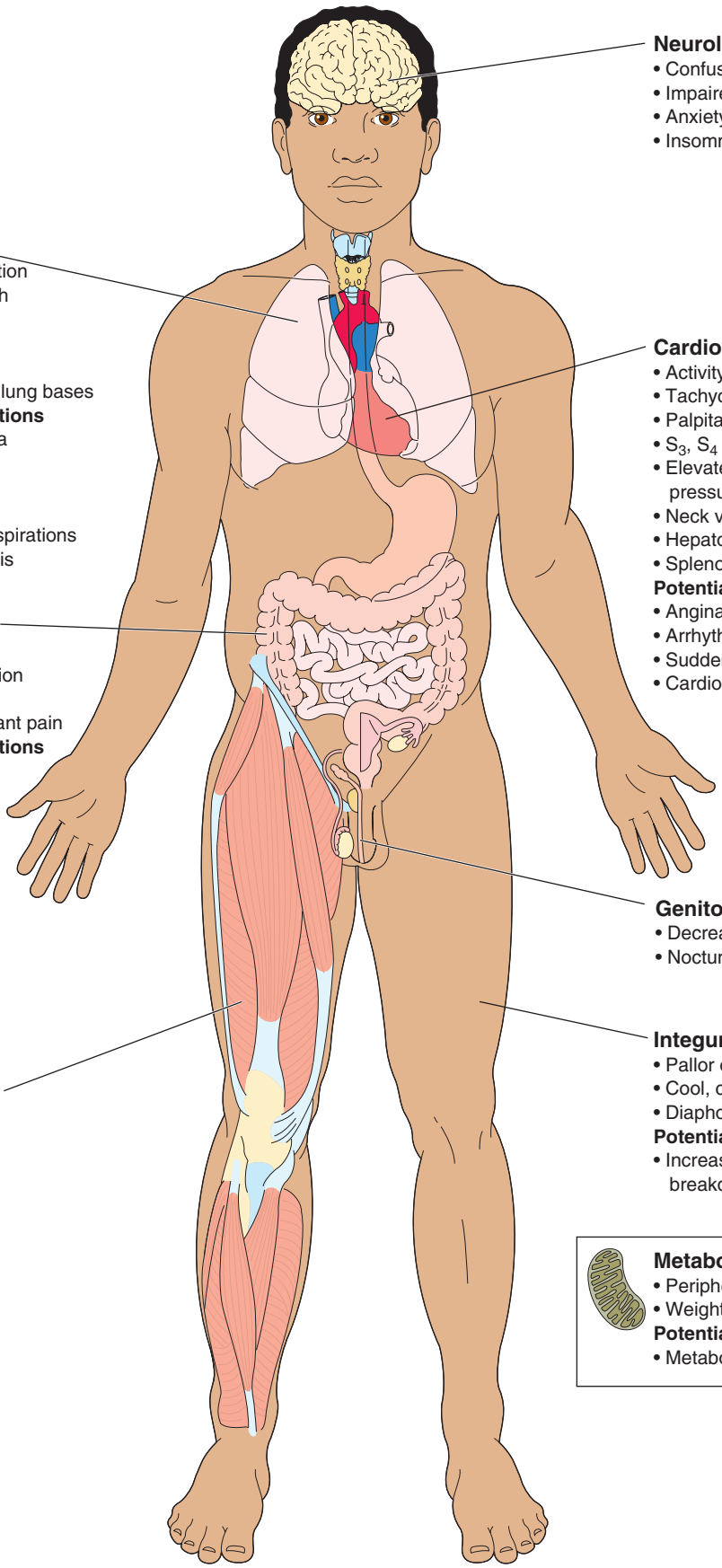
The arterial blood pressure reflects the cardiac output and resistance to blood flow created by the elastic arterial walls (*systemic vascular resistance, SVR*). Cardiac output is determined by the blood volume and the ability of the ventricles to fill and effectively pump that blood. Systemic vascular resistance is primarily determined by vessel diameter and distensibility (compliance). Factors such as SNS input, circulating hormones (e.g. adrenaline, noradrenaline, ANP and vasopressin) and the renin–angiotensin system affect SVR.

TABLE 30.3 NYHA grading of symptoms in chronic heart failure

NYHA Class I	No limitations. Ordinary physical activity does not cause undue fatigue, dyspnoea or palpitations (asymptomatic LV dysfunction).
NYHA Class II	Slight limitation of physical activity. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina pectoris (mild CHF).
NYHA Class III	Marked limitation of physical activity. Less than ordinary physical activity leads to symptoms (moderate CHF).
NYHA Class IV	Unable to carry on any physical activity without discomfort. Symptoms of CHF present at rest (severe CHF).

Source: National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand (Chronic Heart Failure Guidelines Expert Writing Panel) (2011). *Guidelines for the prevention, detection and management of chronic heart failure in Australia*, p. 8. © National Heart Foundation of Australia.

MULTISYSTEM EFFECTS OF HEART FAILURE



Neurological
- Confusion
- Impaired memory
- Anxiety, restlessness
- Insomnia

Respiratory
- Dyspnoea on exertion
- Shortness of breath
- Tachypnoea
- Orthopnoea
- Dry cough
- Crackles (rales) in lung bases

Potential complications
- Pulmonary oedema
- Pneumonia
- Cardiac asthma
- Pleural effusion
- Cheyne-Stokes respirations
- Respiratory acidosis

Cardiovascular
- Activity intolerance
- Tachycardia
- Palpitations
- S_3, S_4 heart sounds
- Elevated central venous pressure
- Neck vein distension
- Hepatojugular reflux
- Splenomegaly

Potential complications
- Angina
- Arrhythmias
- Sudden cardiac death
- Cardiogenic shock

Gastrointestinal
- Anorexia, nausea
- Abdominal distension
- Liver enlargement
- Right upper quadrant pain

Potential complications
- Malnutrition
- Ascites
- Liver dysfunction

Genitourinary
- Decreased urine output
- Nocturia

Integumentary
- Pallor or cyanosis
- Cool, clammy skin
- Diaphoresis

Potential complications
- Increased risk for tissue breakdown

Musculoskeletal
- Fatigue
- Weakness

Metabolic processes
- Peripheral oedema
- Weight gain

Potential complication
- Metabolic acidosis

TABLE 30.4 Tests indicated for individuals suspected of heart failure

Blood glucose	Metabolic disorders not only increase the risk of cardiovascular disease but complicate its course and management.
BNP and NT-pro BNP	Natriuretic peptides released by the atria may detect deterioration in pump function as increases in these values correlate with worsening heart failure.
Chest x-ray (CXR)	Assesses structure; some inferences regarding function may be made.
Echocardiogram (ECHO)	Assesses structure and function of the myocardium.
Electrocardiogram (ECG)	May demonstrate possible contributing factor or resulting consequence (arrhythmia or chamber enlargement).
Full blood count (FBC)	May detect anaemia.
Urea and electrolytes (U&Es)	Urea and creatinine are markers of renal function and can suggest renal failure or cause of fluid retention. Electrolyte measures may detect fluid and electrolyte imbalances.
Liver function tests (LFTs)	May detect alterations in liver function which can demonstrate possible cause or effect.
Arterial blood gases (ABGs)	May detect hypoxia as a cause or consequence of heart failure.
Myocardial perfusion scans (MPS or sestamibi)	May detect changes in ventricular wall function, such as wall motion or perfusion anomalies, as causes or consequences of heart failure.
Coronary angiogram	May quantify vessel occlusion as a cause of heart failure.

The systolic blood pressure, normally about 120 mmHg in healthy adults, reflects the pressure generated during ventricular systole. During diastole, elastic arterial walls keep a minimum pressure within the vessel (diastolic blood pressure) to maintain blood flow through the capillary beds. The average diastolic pressure in a healthy adult is 80 mmHg. The mean arterial pressure (MAP) is the average pressure in the arterial circulation throughout the cardiac cycle. It reflects the driving pressure or perfusion pressure, an indicator of tissue perfusion. The formula MAP = CO × SVR is often used to show the relationships between factors determining the blood pressure. Mean arterial pressure can be calculated by adding two times the diastolic blood pressure to the systolic blood pressure and dividing by 3 (MAP = [(2 × diastolic) + systolic]/3). For example, a blood pressure of 120/80 results in a MAP of 93. Mean arterial pressures of 70 to 90 mmHg are desirable. Perfusion to vital organs is severely jeopardised at MAPs of 50 or less; MAPs greater than 105 mmHg may indicate hypertension or vasoconstriction.

Medications

People with heart failure often receive multiple medications to reduce cardiac work and improve cardiac function. The main drug classes used to treat heart failure are the angiotensin-converting-enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), beta-blockers, diuretics, inotropic medications (including digoxin, sympathomimetic agents and phosphodiesterase inhibitors), direct vasodilators and antiarrhythmic drugs. Nursing implications for ACE inhibitors and ARBs, diuretics and inotropic medications are found in the 'Medication administration' box below.

Surgery

Nursing care of the person who has had a heart transplant (see Figure 30.3) is similar to care for a person who has had cardiac surgery (see box on the nursing care of person having a coronary artery bypass in Chapter 29). Bleeding is a major concern in the early postoperative period. Chest tube drainage is frequently monitored (initially every 15 minutes), as are the cardiac output, pulmonary artery pressures and CVP. Cardiac tamponade (compression of the heart) can develop, presenting as either a sudden event or a gradual process. Chest tubes are gently milked (not stripped) as needed to maintain patency.

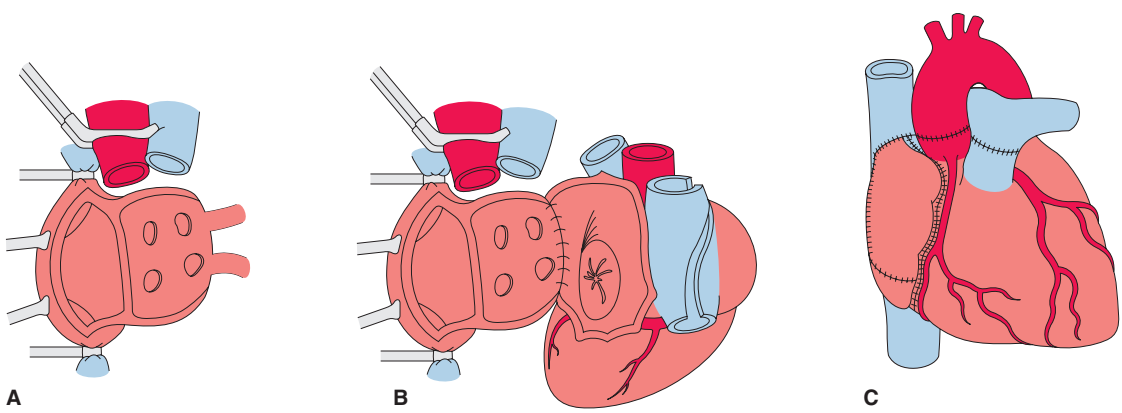


FIGURE 30.3 ■ Cardiac transplantation. *A*, The person's heart is removed, leaving the posterior walls of the atria intact. The donor heart is anastomosed to the atria, *B*, and the great vessels, *C*

MEDICATION ADMINISTRATION Heart failure

ACE INHIBITORS AND ARBs

Angiotensin-converting-enzyme inhibitors (ACE inhibitors) and angiotensin II receptor blockers (ARBs) prevent acute coronary events and reduce mortality in heart failure. ACE inhibitors interfere with production of angiotensin II, resulting in vasodilation and reduced circulating blood volume, ultimately reducing blood pressure. In heart failure, ACE inhibitors reduce afterload, improve cardiac output and increase renal perfusion. They also reduce pulmonary congestion and peripheral oedema. ACE inhibitors suppress myocyte growth and reduce ventricular remodelling in heart failure. While the pharmacological effect of ARBs is similar, they block the action of angiotensin II at the receptor rather than interfering with its production. This mechanism of action results in fewer side effects as ACE is also required in the lungs to break down the substances responsible for increasing airway hyperreactivity, tachykinins and bradykinins.

Nursing responsibilities

- Do not administer these drugs to women in the second and third trimesters of pregnancy.
- Carefully monitor individuals who are volume depleted or who have impaired renal function.
- Use an infusion pump when administering ACE inhibitors intravenously.
- Monitor blood pressure closely for 2 hours following first dose and as indicated thereafter.
- Monitor serum potassium levels; ACE inhibitors can cause hyperkalaemia. (This is less of a concern with ARBs.)
- Monitor white blood cell (WBC) count for potential neutropenia. Report to the doctor.

Health education for the person and family

- Take the drug at the same time every day to ensure a stable blood level.
- Monitor blood pressure and weight weekly. Report significant changes to the doctor.
- Avoid making sudden position changes; for example, rise from bed slowly. Lie down if feeling dizzy or light headed, particularly after the first dose.
- Report any signs of easy bruising and bleeding, sore throat or fever, oedema or skin rash. Immediately report swelling of the face, lips or eyelids, and itching or breathing problems.
- A persistent, dry cough may develop if taking an ACE inhibitor. Contact the doctor if this becomes a problem.

DIURETICS

Diuretics act on different portions of the kidney tubule to inhibit the reabsorption of sodium and water and promote their excretion. With the exception of the potassium-sparing diuretics—spironolactone and amiloride—diuretics also promote potassium excretion, increasing the risk of hypokalaemia. Spironolactone, an aldosterone receptor blocker, reduces symptoms and slows progression of heart failure. Aldosterone receptors in the heart and blood vessels promote myocardial remodelling and fibrosis, activate the sympathetic nervous system and promote vascular fibrosis (which decreases compliance) and baroreceptor dysfunction.

Nursing responsibilities

- Obtain baseline weight and vital signs.
- Monitor blood pressure, intake and output, weight, skin turgor and oedema as indicators of fluid volume status.
- Assess for volume depletion, particularly with loop diuretics (frusemide and ethacrynic acid—ethacrynic acid is primarily restricted to people who are either allergic to, or refractory to, frusemide or bumetanide): dizziness, orthostatic hypotension, tachycardia, muscle cramping.
- Report abnormal serum electrolyte levels to the doctor. Replace electrolytes as indicated.
- Do not administer potassium replacements to individuals receiving a potassium-sparing diuretic.
- Evaluate renal function by assessing urine output and serum urea and creatinine.
- Administer intravenous frusemide slowly, no faster than 20 mg/minute. Evaluate for signs of ototoxicity. Do not administer this drug or ethacrynic acid concurrently with aminoglycoside antibiotics (e.g. gentamicin), which are also ototoxic.

Health education for the person and family

- Drink at least 6 to 8 glasses of water per day.
- Take your diuretic at times that will be the least disruptive to your lifestyle, usually in the morning and early afternoon if a second dose is ordered. Take with meals to decrease gastric upset.
- Monitor your blood pressure, pulse and weight weekly. Report significant weight changes to your doctor.
- Report any of the following to your doctor: severe abdominal pain, jaundice, dark urine, abnormal bleeding or bruising, flu-like symptoms, signs of hypokalaemia, hyponatraemia and dehydration (thirst, salt craving, dizziness, weakness, rapid pulse). See Chapter 9 for manifestations of electrolyte imbalances.
- Avoid sudden position changes. They may cause dizziness, light headedness or feelings of faintness.
- Unless taking a potassium-sparing diuretic, integrate foods rich in potassium into your diet (see Chapter 9). Limit sodium use.

POSITIVE INOTROPIC AGENTS

Digoxin glycosides

Digoxin (Lanoxin)

Digoxin improves myocardial contractility by interfering with ATPase in the myocardial cell membrane and increasing the amount of calcium available for contraction. The increased force of contraction causes the heart to empty more completely, increasing stroke volume and cardiac output. Improved cardiac output improves renal perfusion, decreasing renin secretion. This decreases preload and afterload, reducing cardiac work. Digoxin also has electrophysiological effects, slowing conduction through the AV node decreasing heart rate and reducing oxygen consumption.

Nursing responsibilities

- Assess apical pulse before administering. Withhold digoxin and notify the doctor if heart rate is below 60 bpm

(continued)

MEDICATION ADMINISTRATION Heart failure (continued)

and/or manifestations of decreased cardiac output are noted. Record apical rate on medication record.
- Evaluate electrocardiogram (ECG) for scooped (spoon-shaped) ST segment, AV block, bradycardia and other arrhythmias (especially PVCs and atrial tachycardias).
- Report manifestations of digoxin toxicity: anorexia, nausea, vomiting, abdominal pain, weakness, vision changes (diplopia, blurred vision, yellow-green or white halos seen around objects) and new-onset arrhythmias.
- Assess potassium, magnesium, calcium and serum digoxin levels before giving digoxin. Hypokalaemia can precipitate toxicity even when the serum digoxin level is in the 'normal' range.
- Monitor individuals with renal insufficiency or renal failure and older adults carefully for digoxin toxicity.
- Prepare to administer digoxin immune Fab (Digibind) for digoxin toxicity.

Health education for the person and family

- Take pulse daily before taking the digoxin. Do not take the digoxin if the pulse is below 60 bpm or if weak, fatigued, light headed, dizzy, short of breath or having chest pain. Notify the doctor immediately.
- Contact the doctor if any manifestations of digoxin toxicity occur: palpitations, weakness, loss of appetite, nausea, vomiting, abdominal pain, blurred or coloured vision, double vision.
- Avoid using antacids and laxatives; they decrease digoxin absorption.
- Notify the doctor immediately if any manifestations of potassium deficiency occur: weakness, lethargy, thirst, depression, muscle cramps or vomiting.
- Incorporate foods high in potassium into your diet: fresh orange or tomato juice, bananas, raisins, dates, figs, prunes, apricots, spinach, cauliflower and potatoes.

SYMPATHOMIMETIC AGENTS

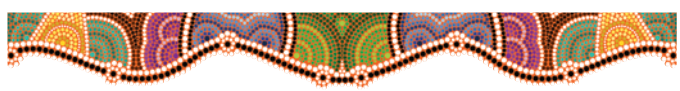
Sympathomimetic agents stimulate the heart, improving the force of contraction. Dobutamine is preferred in managing heart failure because it does not increase the heart rate as much as dopamine and it has a mild vasodilatory effect. These drugs are given by intravenous infusion and may be titrated to obtain their optimal effects.

Atrial arrhythmias are relatively common following cardiac transplant. Temporary pacing wires are placed during surgery as the conduction system may be disrupted by surgical manipulation or postoperative swelling. Hypothermia is induced during surgery; postoperatively, the person is gradually rewarmed over a 1- to 2-hour period. Prevention of rapid rewarming and shivering are important to maintain haemodynamic stability and reduce oxygen consumption. Cardiac function is impaired in up to 50% of transplanted hearts during the early postoperative period. Inotropic agents such as low-dose dopamine or dobutamine may be required to support cardiac function and circulation.

Infection and rejection are major postoperative concerns; these are the chief causes of mortality in people who have had a heart transplant. Rejection may develop immediately after transplant (rarely) or within weeks to months or even years after the transplant. Acute rejection usually presents within weeks of the transplant, developing when the transplanted organ is recognised by the immune system as foreign. Lymphocytes infiltrate the organ and myocardial cell necrosis can be detected on biopsy. Acute rejection often can be treated using immunosuppressive drugs. These drugs also are given to prevent rejection of the transplanted organ, even when the tissue match is good (see Chapter 12). Although immunosuppressive medications help prevent organ rejection, they impair the person's defences against infection. Early postoperative infections commonly are bacterial or fungal (*Candida*). Multiple invasive lines, prolonged ventilator support and immunosuppressive therapy contribute to the transplant recipient's risk of infection. Nursing care directed at prevention of infection is vital and includes strict aseptic technique, early extubation to reduce the risk of ventilator-associated infections, limitation of individuals who may present an infection risk and early ambulation (Elliot, Aitken & Chaboyer, 2012).

The donor heart is denervated during the transplant procedure and therefore the person's heart rate is commonly between 90–100 bpm. This occurs because the slowing effects of the parasympathetic nervous system are lost. The endocrine system, position changes, exercise and certain drugs moderate heart rate following a heart transplant.

OTHER PROCEDURES A person with heart failure may benefit from coronary revascularisation, left ventricular aneurysmectomy or mitral valvuloplasty (NHF Australia, 2011). Other surgical procedures such as cardiomyoplasty and ventricular reduction surgery are not recommended because of unfavourable results.



Nursing care

Health promotion

Health promotion activities to reduce the risk and incidence of heart failure should be directed at the risk factors. Teach individuals about coronary heart disease, the primary underlying cause of heart failure. Discuss CHD risk factors and ways to reduce those risk factors (see Chapter 29).

Hypertension also is a major cause of heart failure. Routinely screen individuals for hypertension and refer them

to a medical officer as required. Discuss the importance of effectively managing hypertension to reduce the future risk of heart failure. Likewise, stress the relationship between effective diabetes management and reduced risk of heart failure.

Assessment

Obtain both subjective and objective data when assessing the person with heart failure:

- *Health history:* complaints of increasing dyspnoea, decreasing activity tolerance or paroxysmal nocturnal dyspnoea; number of pillows used for sleeping; recent weight gain; presence of a cough; chest or abdominal pain; anorexia or nausea; history of cardiac disease, previous episodes of heart failure; other risk factors such as hypertension or diabetes; current medications; usual diet and activity and recent changes.
- *Physical examination:* general appearance; ease of breathing, conversing, changing positions; apparent anxiety; vital signs including apical pulse; colour of skin and mucous membranes; neck vein distension, peripheral pulses, capillary refill, presence and degree of oedema; heart and breath sounds; abdominal contour, bowel sounds, tenderness; right upper abdominal tenderness, liver enlargement.
- *Diagnostic tests:* serum electrolyte, urea, creatinine and digoxin levels; arterial blood gas (ABG) results; ECG, echocardiogram and chest x-ray reports.

Nursing diagnosis and interventions

Heart failure impacts on quality of life and interferes with activities of daily living (ADLs). Reducing myocardial oxygen demand is a major nursing care goal for the individual in acute heart failure. This includes providing rest and undertaking the prescribed interventions to reduce cardiac work, improve contractility and manage symptoms. See also the accompanying nursing care plan for additional nursing diagnoses and interventions for the individual with heart failure.

- Auscultate the person's heart and breath sounds regularly. *S_1 and S_2 may be diminished if cardiac function is poor. A ventricular gallop (S_3) is an early sign of heart failure; atrial gallop (S_4) may also be present. Crackles are often heard in the lung bases; increasing crackles and dyspnoea indicate worsening failure.*

Decreased cardiac output

Pump failure results in decreased upstroke volume and tissue perfusion.

- Monitor the person's vital signs and oxygen saturation as indicated. *Decreased cardiac output stimulates the SNS to increase the heart rate in an attempt to restore CO. Tachycardia at rest is common. Diastolic blood pressure may initially be elevated because of vasoconstriction; in late stages, compensatory mechanisms fail and the person's BP will fall. Oxygen saturation levels provide a measure of gas exchange and tissue perfusion.*

CONSIDERATION FOR PRACTICE

Report manifestations of decreased cardiac output and tissue perfusion including changes in mentation, decreased urine output, cool, clammy skin, diminished pulses, pallor or cyanosis, and arrhythmias.

- Administer supplemental oxygen as needed. *This improves oxygenation of the blood, decreasing the effects of hypoxia and ischaemia.*
- Administer prescribed medications as ordered. *Drugs are used to decrease the cardiac workload and increase contractility.*
- Encourage rest, explaining the rationale. Elevate the head of the bed to reduce the work of breathing. Provide a bedside commode and assist with ADLs. Instruct the person to avoid the Valsalva manoeuvre and encourage the use of stool softeners to reduce strain. *These measures reduce cardiac workload.*

CONSIDERATION FOR PRACTICE

Encourage the person to rest and teach methods to decrease anxiety. Maintain a quiet environment and encourage expression of fears and feelings. Explain care measures and their purpose. Reducing anxiety diminishes the sympathetic nervous system effects, resulting in an improved balance between oxygen demand and supply.

Excess fluid volume

As cardiac output falls, compensatory mechanisms cause salt and water retention, increasing blood volume. This increased fluid volume places additional stress on the already failing ventricles, making them work harder to move the fluid load.

- Assess respiratory status and auscultate lung sounds at least every 4 hours. Notify the doctor of significant changes in condition. *Declining respiratory status indicates worsening left heart failure.*

CONSIDERATION FOR PRACTICE

Immediately notify the doctor if the person develops dyspnoea, tachypnoea, severe orthopnoea, a cough productive of large amounts of pink, frothy sputum, or an overwhelming sense of impending doom or panic. Acute pulmonary oedema is a medical emergency and can develop rapidly. Immediate intervention is required to preserve life.

- Monitor the person's intake and output. Notify the doctor if urine output is less than 30 mL/h. Weigh daily. *Careful monitoring of fluid volume is important during treatment of heart failure. Diuretics may reduce circulating volume, producing hypovolaemia despite persistent peripheral oedema. A fall in urine output may indicate significantly reduced cardiac output and renal ischaemia. Weight is an objective measure of fluid status: 1 L of fluid is equal to 1 kg of weight.*

NURSING CARE PLAN A person with heart failure

One year ago, Arthur Jackson, 67 years old, had a large anterior wall MI and underwent subsequent coronary artery bypass surgery. On discharge, he was started on a regimen of enalapril, digoxin, frusemide, warfarin and a potassium chloride supplement. He is now in the cardiac unit complaining of severe shortness of breath, haemoptysis and poor appetite for 1 week. He is diagnosed with acute heart failure.

ASSESSMENT

Mr Jackson refuses to settle in bed, preferring to sit in the bedside recliner in high-Fowler's position. He states, 'Lately, this is the only way I can breathe.' Mr Jackson states that he has not been able to work in his garden without getting short of breath. He complains of his shoes and belt being too tight.

Mr Jackson insists that he takes his medications regularly. He states that he normally works in his garden for light exercise. In his diet history, Mr Jackson admits fondness for bacon and takeaway food and sheepishly admits to snacking between meals 'even though I need to lose weight'.

Mr Jackson's vital signs are: BP 95/72 mmHg, HR 124 and irregular, R 28 and laboured, and T 36.5°C. The cardiac monitor shows atrial fibrillation. An S_3 is noted on auscultation; the cardiac impulse is left of the midclavicular line. He has crackles and diminished breath sounds in the bases of both lungs. Significant jugular venous distension, 3+ pitting oedema of feet and ankles and abdominal distension are noted. Liver size is within normal limits by percussion. His skin is cool and he is diaphoretic. Chest x-ray shows cardiomegaly and pulmonary infiltrates.

DIAGNOSES

- *Excess fluid volume* related to impaired cardiac pump and salt and water retention manifested by crackles and pitting oedema.
- *Risk of activity intolerance* related to impaired cardiac output manifested by inability to undertake activities of daily living.
- *Impaired health maintenance* related to lack of knowledge about diet restrictions manifested by frequent consumption of foods high in salt and fat.

PLANNING

- Hourly vital signs and haemodynamic pressure measurements.
- Monitor oxygen saturation continuously. Notify doctor if less than 94%.
- Administer and monitor effects of prescribed diuretics and vasodilators.
- Weigh daily; strict fluid balance monitoring.
- Enforce fluid restriction of 1500 mL/24 hours.
- Auscultate heart and breath sounds every 4 hours and as indicated.

Expected outcomes

- Demonstrate loss of excess fluid by weight loss and decreases in oedema, jugular venous distension and abdominal distension.
- Demonstrate improved activity tolerance.
- Verbalise understanding of diet restrictions.

IMPLEMENTATION

- Administer oxygen per nasal prongs at 2 L/min.
- High-Fowler's or position of comfort.
- Notify doctor of significant changes in laboratory values.
- Teach about all medications and how to take and record pulse. Provide information about anticoagulant therapy and signs of bleeding.
- Design an activity plan with Mr Jackson that incorporates preferred activities and scheduled rest periods.
- Instruct about sodium-restricted diet. Allow meal choices within allowed limits.
- Consult dietitian for planning and teaching Mr and Mrs Jackson about a low-sodium diet.

EVALUATION

Mr Jackson is discharged after 3 days in the cardiac unit. He has lost 3.5 kilograms during his stay and states it is much easier to breathe and his shoes fit better. He is able to sleep in semi-Fowler's position with only one pillow. His peripheral oedema has resolved. Mr and Mrs Jackson met with the dietitian, who helped them develop a realistic eating plan to limit sodium, sugar and fats. The dietitian also provided a list of high-sodium foods to avoid. The physiotherapist designed a progressive activity plan with Mr Jackson that he will continue at home. He remains in atrial fibrillation, a chronic condition. His knowledge of digoxin and warfarin has been assessed and reinforced. The nurse confirms that he is able to accurately check his pulse and can identify signs of digoxin toxicity and excessive bleeding.

CRITICAL THINKING IN THE NURSING PROCESS

1. Mr Jackson's medication regimen remains the same after discharge. What specific teaching does he need related to potential interactions of these drugs?
2. Mr Jackson exclaims, 'Talk to my wife about my medications—she's Tarzan and I'm Jane, now.' How would you respond?
3. Mr Jackson tells you, 'Sometimes I forget whether I have taken my aspirin, so I'll take another just to be sure. After all, they are only baby aspirin. One or two extra a day shouldn't hurt, right?' What is your response?
4. Mr Jackson is admitted to the neurology unit 6 months later with a cerebral vascular accident (CVA). What are the possible contributing factors of his stroke?

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Given that Mr Jackson has a cerebrovascular accident 6 months later, explore the influence of cardiovascular conditions on the development of other co-morbidities and conditions. What other conditions/co-morbidities are common in individuals who present with an ischaemic heart disease? Why? What specifically can a nurse do to influence a person with ischaemic heart disease to reduce the risk of developing these common co-morbidities and conditions in the future?

- Record abdominal girth every shift. Note complaints of a loss of appetite, abdominal discomfort or nausea. *Venous congestion can lead to ascites and may affect gastrointestinal function and nutritional status.*
- Monitor and record the person's haemodynamic measurements. Report significant changes and negative trends. *Haemodynamic measurements provide a means of monitoring condition and response to treatment.*
- Restrict fluids as ordered. Offer appropriate choices of fluid type and timing of intake, scheduling most fluid intake during morning and afternoon hours. Offer ice chips and frequent mouth care. *Providing choices increases the person's sense of control. Ice chips and frequent mouth care relieve dry mouth and thirst and promote comfort.*

Activity intolerance

People with heart failure have insufficient cardiac reserve to meet increased oxygen demands. As the disease progresses and cardiac function is further compromised, activity intolerance increases. The low cardiac output and inability to participate in activities may hinder self-care.

- Organise nursing care to allow rest periods. Grouping activities together allows adequate time to 'recharge'. However, care must be taken to avoid overtaxing the individual during the grouped activities. *Frequently gauge the person's capacity to continue and adjust the plans as required.*

CONSIDERATION FOR PRACTICE

Monitor vital signs and cardiac rhythm during and after activities. Tachycardia, arrhythmias, increasing dyspnoea, changes in blood pressure, diaphoresis, pallor, complaints of chest pain, excessive fatigue or palpitations indicate activity intolerance. Instruct to rest if manifestations are noted. The failing heart is unable to increase cardiac output to meet increased oxygen demands associated with activity. Assessing response to activities helps evaluate cardiac function. Decreasing activity tolerance may signal deterioration of cardiac function, not overexertion.

- Assist with ADLs as needed. Encourage independence within prescribed limits. *Assisting with ADLs helps ensure that care needs are met while reducing cardiac workload. Involving the individual promotes a sense of control and reduces helplessness.*
- Plan and implement progressive activities. Use passive and active range-of-motion (ROM) exercises as appropriate. Consult with physical therapist on activity plan. *Progressive activity slowly increases exercise capacity by strengthening and improving cardiac function without strain. Activity also helps prevent skeletal muscle atrophy. ROM exercises prevent complications of immobility in severely compromised individuals.*
- Provide written and verbal information about activity after discharge. *Written information provides a reference for important information. Verbal information allows clarification and validation of the material.*

Deficient knowledge: low-sodium diet

Diet is an important part of long-term management of heart failure to manage fluid retention.

- Discuss the rationale for sodium restrictions. *Understanding fosters compliance with the prescribed diet.*
- Consult with dietitian to plan and teach a low-sodium and, if necessary for weight control, low-kilojoule diet. Provide a list of high-sodium, high-fat, high-cholesterol foods to avoid. Provide National Heart Foundation materials. *Dietary planning and teaching increase the individual's sense of control and participation in disease management. Food lists are useful memory aids.*
- Assist the person to construct a 2-day meal plan choosing foods low in sodium. *This allows learning assessment, clarification of misunderstandings and reinforcement of teaching.*
- Encourage small, frequent meals rather than three heavy meals per day. *Small, frequent meals provide continuing energy resources and decrease the work required to digest a large meal.*

CONSIDERATION FOR PRACTICE

Teach a person with cardiovascular pathology how to read food labels for nutritional information. Many processed foods contain 'hidden' sodium, which can be identified by careful label reading.

Community-based care

Heart failure is a chronic condition requiring active participation by the individual and family for effective management. In teaching for home care, include the following topics:

- the disease process and its effects on the individual's life
- warning signals of cardiac decompensation requiring treatment
- desired and adverse effects of prescribed drugs; monitoring for effects; importance of compliance with drug regimen to prevent acute and long-term complications of heart failure
- prescribed diet and sodium restriction; practical suggestions for reducing salt intake; recommend National Heart Foundation materials and recipes
- exercise recommendations to strengthen the heart muscle and improve aerobic capacity (see Box 30.2)
- the importance of keeping scheduled follow-up appointments to monitor disease progression and effects of therapy.

Provide referrals for home healthcare and household assistance (shopping, transportation, personal needs and housekeeping) as indicated. Referrals to community agencies, such as local cardiac rehabilitation programs, heart support groups or the National Heart Foundation, can provide the person and their family with additional materials and psychosocial support.

BOX 30.2 Home activity guidelines for the person with heart failure

- Perform as many activities as independently as possible.
- Space meals and activities.
  a. Eat six small meals a day.
  b. Allow time during the day for periods of rest and relaxation.
- Perform all activities at a comfortable pace.
  a. If tired during activity, stop and rest for 15 minutes.
  b. Resume activity only when capable.
- Stop any activity that causes chest pain, shortness of breath, dizziness, faintness, excessive weakness or sweating. Rest. Notify the doctor if activity tolerance changes and if symptoms continue after rest.
- Avoid straining. Do not lift heavy objects. Eat a high-fibre diet and drink plenty of water to prevent constipation. Use laxatives or stool softeners, as approved by the doctor, to avoid constipation and straining during bowel movements.
- Begin a graded exercise program. Walking is good exercise that does not require any special equipment (except a good pair of walking shoes). The amount and quality of exercise should be determined by the NYHA functional class symptoms (see Table 30.5). Exercise programs should be initiated by a medical officer and supervised by appropriately trained exercise prescription professionals taking into account the clinical status of the individual. Isometric activity is not recommended as it increases left ventricular afterload.

TABLE 30.5 NYHA functional class symptoms

NYHA Class I and II	Progress gradually to at least 30 minutes of physical activity (continuously or in 10-minute bouts) up to moderate intensity on most, if not all, days of the week.
NYHA Class III and IV	Short intervals of low-intensity activity, with frequent rest days.
NYHA Class IV	Gentle mobilisation as symptoms allow.

Source: Modified from NHF Australia (2011). *Guidelines for the prevention, detection and management of chronic heart failure in Australia*, p. 19. © National Heart Foundation of Australia.

THE PERSON WITH PULMONARY OEDEMA

Pulmonary oedema is an abnormal accumulation of fluid in the interstitial tissue and alveoli of the lung. Both cardiac and non-cardiac disorders can cause pulmonary oedema. Cardiac causes include acute myocardial infarction, acute heart failure and valvular disease. *Cardiogenic pulmonary oedema*, the focus of this section, is a sign of severe cardiac decompensation. Non-cardiac causes of pulmonary oedema include primary pulmonary disorders, such as acute respiratory distress syndrome (ARDS), trauma, sepsis, drug overdose or neurological sequelae. Pulmonary oedema due to ARDS is discussed in Chapter 36.

Pulmonary oedema is a medical emergency: the person is literally drowning in the fluid in the alveolar and interstitial pulmonary spaces. Its onset may be acute or gradual, progressing to severe respiratory distress. Immediate treatment is necessary.

FAST FACTS

- Cardiogenic pulmonary oedema is a severe form of heart failure. Risk factors are those associated with heart failure and treatment focuses on maintaining oxygenation and improving cardiac function.
- Non-cardiogenic pulmonary oedema is a primary or secondary lung disorder. It usually occurs secondarily to a critical event such as major trauma, shock or disseminated intravascular coagulation (DIC). Treatment focuses on maintaining oxygenation and the primary, underlying disorder.

Pathophysiology

In cardiogenic pulmonary oedema, the contractility of the left ventricle is severely impaired. The ejection fraction falls because the ventricle is unable to eject the blood that enters it, causing a sharp rise in end-diastolic volume and pressure. Pulmonary hydrostatic pressures rise, ultimately exceeding the osmotic pressure of the blood. As a result, fluid leaking from the pulmonary capillaries congests interstitial tissues, decreasing lung compliance and interfering with gas exchange. As capillary and interstitial pressures increase further, the tight junctions of the alveolar walls are disrupted and the fluid enters the alveoli, along with large red blood cells (RBCs) and protein molecules. Ventilation and gas exchange are severely disrupted and hypoxia worsens.

Manifestations

The person with acute pulmonary oedema presents with classic manifestations (see box below). Dyspnoea and laboured respirations are acute and severe, accompanied by orthopnoea. Cyanosis is present and the skin is cool, clammy and diaphoretic. A productive cough with pink, frothy sputum develops due to fluid, RBCs and plasma proteins in the alveoli and airways. Crackles are heard throughout the lung fields on auscultation. As the condition worsens, lung sounds become harsher. The person often is restless and highly anxious, although severe hypoxia may cause confusion or lethargy.

As noted earlier, pulmonary oedema is a medical emergency. Without rapid and effective intervention, severe tissue hypoxia and acidosis will lead to organ system failure and death.

MANIFESTATIONS Acute pulmonary oedema

RESPIRATORY
- Tachypnoea
- Laboured respirations
- Dyspnoea
- Orthopnoea
- Paroxysmal nocturnal dyspnoea
- Cough productive of frothy, pink sputum
- Crackles, wheezes

CARDIOVASCULAR
- Tachycardia
- Hypotension
- Cyanosis
- Cool, clammy skin
- Hypoxaemia
- Ventricular gallop (S_3)

NEUROLOGICAL
- Restlessness
- Anxiety
- Sense of impending doom

INTERPROFESSIONAL CARE

Immediate treatment for acute pulmonary oedema focuses on restoring effective gas exchange and reducing fluid and pressure in the pulmonary vascular system. The individual is placed in an upright sitting position with the legs dangling to reduce venous return by trapping some excess fluid in the lower extremities. This position also facilitates breathing.

Diagnosis

Diagnostic testing is limited to assessment of the acute situation. *ABGs* are drawn to assess gas exchange and acid–base balance. Oxygen tension (PaO_2) is usually low. Initially, carbon dioxide levels ($PaCO_2$) may also be reduced because of rapid respirations. As the condition progresses, the $PaCO_2$ rises and respiratory acidosis develops (see Chapter 9). *Oxygen saturation* levels also are continuously monitored. The *chest x-ray* shows pulmonary vascular congestion and alveolar oedema. Provided the person's condition allows, *haemodynamic monitoring* is instituted. In cardiogenic pulmonary oedema, the pulmonary artery wedge pressure is elevated, usually over 25 mmHg. Cardiac output may be decreased.

Medications

Morphine is administered intravenously to relieve anxiety and improve the efficacy of breathing. It also is a vasodilator that reduces venous return and lowers left atrial pressure. Although morphine is very effective for individuals with cardiogenic pulmonary oedema, naloxone, its antidote, should be kept readily available in case respiratory depression occurs.

Oxygen is administered using a positive-pressure system that can achieve a 100% oxygen concentration. A continuous positive airway pressure (CPAP) mask system may be used or the person may be intubated and mechanical ventilation employed (see Chapter 36). Positive pressure increases alveolar pressures and gas exchange while decreasing fluid diffusion into the alveoli.

Potent loop diuretics such as frusemide are administered intravenously to promote rapid diuresis. Vasodilators such as intravenous glyceryl trinitrate may be given to improve cardiac output by reducing afterload. However, care must be taken to avoid profound hypotension. Positive inotropes may be administered to improve the myocardial contractility and cardiac output.

When the person's condition has stabilised, further diagnostic tests may be done to determine the underlying cause of pulmonary oedema and specific treatment measures directed at the cause instituted.

Nursing care

Nursing care of the person with acute pulmonary oedema focuses on relieving the pulmonary effects of the disorder. Interventions are directed towards improving oxygenation, reducing fluid volume and providing emotional support.

The nurse is often instrumental in recognising early manifestations of pulmonary oedema and initiating treatment. As with many critical conditions, emergent care is directed towards the ABCs: airway, breathing and circulation.

Nursing diagnoses and interventions

Promoting effective gas exchange and restoring an effective cardiac output are the priorities for nursing and interprofessional care of the person with cardiogenic pulmonary oedema. The experience of acute dyspnoea is terrifying for the individual; the nurse is instrumental in providing emotional support and reassurance.

Impaired gas exchange

Accumulated fluid in the alveoli and airways interferes with ventilation of the lungs. As a result, alveolar oxygen levels fall and carbon dioxide levels may rise. Reduced alveolar oxygen decreases diffusion of the gas into pulmonary capillaries. In addition, pulmonary oedema increases the distance over which gases must diffuse to cross the alveolar–capillary membrane, further reducing oxygen levels in the blood and oxygen delivery to the tissues.

- Ensure airway patency. *A patent airway is absolutely vital for pulmonary function, including ventilation and gas exchange.*

CONSIDERATION FOR PRACTICE

Assess the effectiveness of respiratory efforts and airway clearance. Pulmonary oedema increases the work of breathing. This increased effort can lead to fatigue and decreased respiratory effort.

- Assess respiratory status frequently, including rate, effort, use of accessory muscles, sputum characteristics, lung sounds and skin colour. *The status of a person in acute pulmonary oedema can change rapidly for the better or worse.*
- Place in high-Fowler's position with the legs dangling. *The upright position facilitates breathing and decreases venous return.*

- Administer oxygen as ordered by mask, CPAP mask or ventilator. *Supplemental oxygen promotes gas exchange; positive pressure increases the pressure within the alveoli, airways and thoracic cavity, decreasing venous return, pulmonary capillary pressure and fluid leak into the alveoli.*
- Encourage to cough up secretions; provide nasotracheal suctioning if necessary. *Coughing moves secretions from smaller airways into larger airways where they can be suctioned out or coughed up if necessary.*

CONSIDERATION FOR PRACTICE

Have emergency equipment readily available in case of respiratory arrest. Be prepared to assist with intubation and initiation of mechanical ventilation. Fatigue, impaired gas exchange and respiratory acidosis can lead to respiratory and cardiac arrest.

Decreased cardiac output

Cardiogenic pulmonary oedema usually is caused by either an acute decrease in myocardial contractility or increased workload that exceeds the ability of the left ventricle. The significant decrease in cardiac output increases pressure within the pulmonary vascular system and triggers compensatory mechanisms that increase the heart rate and blood volume. These compensatory mechanisms further increase the workload of the failing heart.

- Monitor vital signs, haemodynamic status and rhythm continuously. *Acute pulmonary oedema is a critical condition and cardiovascular status can change rapidly.*
- Assess heart sounds for possible S_3, S_4 or murmurs. *These abnormal heart sounds may be due to excess fluid.*
- Initiate an intravenous line for medication administration. Administer morphine, diuretics, vasodilators, bronchodilators and positive inotropic medications (e.g. digoxin, dopamine or dobutamine) as ordered. *These drugs reduce cardiac work and improve contractility.*
- Keep accurate intake and output records. Restrict fluids as ordered. *Fluids may be restricted to reduce vascular volume and cardiac work.*

Fear and anxiety

Acute pulmonary oedema is a very frightening experience for everyone (including the nurse).

- Provide emotional support for the person and their family members. *Fear and anxiety stimulate the sympathetic nervous system, which can lead to ineffective respiratory patterns and interfere with cooperation with care measures.*
- Explain all procedures and the reasons they are performed to the person and their family members. Keep information brief and to the point. Use short sentences and a reassuring tone. *Anxiety and fear interfere with the ability to assimilate information; brief, factual information and reassurance reduce anxiety and fear.*
- Maintain close contact and provide reassurance that recovery from acute pulmonary oedema is often as dramatic as its onset.

Answer questions and provide accurate information in a caring manner. *Knowledge reduces anxiety and psychological stress associated with this critical condition.*

CONSIDERATION FOR PRACTICE

Insert an indwelling catheter; record output hourly. Urine output of less than 30 mL/h indicates impaired renal perfusion due to severely impaired cardiac output and a risk of renal failure or other complications.

Community-based care

During the acute period, teaching is limited to immediate care measures. Once the acute episode of pulmonary oedema has resolved, teach the person and their family about its underlying cause and prevention of future episodes. If pulmonary oedema follows an acute MI, include information related to CHD and the acute AMI, as well as information related to heart failure. Review the teaching and home care needs for the person with these disorders.

THE PERSON WITH AN INFLAMMATORY OR INFECTIVE CONDITION OF THE HEART

Infective conditions include rheumatic heart disease and conditions that result in inflammation of any layer of the cardiac tissue. Manifestations of inflammatory heart disorders range from very mild to life threatening. This section discusses the causes and management of rheumatic heart disease, endocarditis, myocarditis and pericarditis.

THE PERSON WITH RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

Rheumatic fever is a systemic inflammatory disease caused by an abnormal immune response to pharyngeal infection by group A beta-haemolytic streptococci. Rheumatic fever usually is a self-limiting disorder, although it may become recurrent or chronic. The heart commonly is involved in the acute inflammatory process, and approximately 60% of people with rheumatic fever develop rheumatic heart disease (Wallace, 2014). Rheumatic heart disease frequently damages the heart valves and is a major cause of mitral and aortic valve disorders discussed in the next section of this chapter.

Incidence, prevalence and risk factors

In Australia, rheumatic fever is more common than in most other developed countries. As described earlier, rheumatic heart disease (RHD) remains a significant problem for northern and central Indigenous Australians, although RHD is relatively uncommon

in other Australian populations. Indigenous Australian females are 35 times and Indigenous males 30 times more likely to develop RHD than non-Indigenous Australians. The incidence of RHD is considered to be underreported as it is often difficult to diagnose (RHD Australia, 2012).

Rheumatic fever and rheumatic heart disease remain significant public health problems in many developing countries. Globally it is estimated that there are over 15.6 million people with rheumatic heart disease.

Risk factors for streptococcal infections of the pharynx include environmental and economic factors such as crowded living conditions, malnutrition, immunodeficiency and poor access to healthcare (RHD Australia, 2012).

Pathophysiology

The pathophysiology of rheumatic fever is not yet totally understood. It results from an abnormal immune response to M proteins on group A beta-haemolytic streptococcal bacteria. These antigens can bind to cells in the heart, muscles and brain. They also bind with receptors in synovial joints, provoking an autoimmune response (Wallace, 2014). The resulting immune response to the bacteria also leads to inflammation in tissues containing these M proteins. Inflammatory lesions develop in connective tissues on the heart, joints and skin. The antibodies may remain in the serum for up to 6 months following the initiating event. See Chapters 11 and 12 for more information about the immune system and inflammatory response.

FAST FACTS

- 98% of all cases of ARF recorded in the Northern Territory were experienced by Aboriginal and Torres Strait Islander people.
- Aboriginal and Torres Strait Islander people are 26 times more likely to develop RHD than non-Indigenous Australians.
- Aboriginal and Torres Strait Islander people are 6.7 times more likely to be hospitalised for ARF or RHD than non-Indigenous Australians.
- Aboriginal and Torres Strait Islander people are 1.75 times less likely to undergo a heart valve procedure for RHD than non-Indigenous Australians.
- Aboriginal and Torres Strait Islander people are 5 times more likely to die from RHD than non-Indigenous Australians.

Source: AIHW (2013).

Carditis, inflammation of the heart, develops in 30–60% of people with rheumatic fever. The inflammatory process usually involves all three layers of the heart—the pericardium, myocardium and endocardium. *Aschoff bodies*, localised areas of tissue necrosis surrounded by immune cells, develop in cardiac tissues. Pericardial and myocardial inflammation tends to be mild and self-limiting. Endocardial inflammation, however, causes swelling and erythema of valve structures and small vegetative lesions on valve leaflets. As the inflammatory process resolves, fibrous scarring occurs, causing deformity.

Rheumatic heart disease (RHD) is a slowly progressive valvular deformity that may follow acute or repeated attacks of rheumatic fever. Valve leaflets become rigid and deformed; commissures (openings) fuse and the chordae tendineae fibrose and shorten. This results in stenosis or regurgitation of the valve. In **stenosis**, a narrowed fused valve obstructs forward blood flow. **Regurgitation** occurs when the valve fails to close properly (an *incompetent* valve), allowing blood to flow back through it. Valves on the left side of the heart are usually affected; the mitral valve is most frequently involved.

Manifestations

Manifestations of acute rheumatic fever (ARF) typically follow the initial streptococcal infection by about 2 to 3 weeks. Fever and migratory joint pain are often initial manifestations. The knees, ankles, hips and elbows are common sites of swelling and inflammation. *Erythema marginatum* is a temporary non-pruritic skin rash characterised by red lesions with clear borders and blanched centres usually found on the trunk and proximal extremities. Neurological symptoms of rheumatic fever, although rare in adults, may range from irritability and an inability to concentrate to clumsiness and involuntary muscle spasms. See the 'Manifestations' box below.

Manifestations of carditis include chest pain, tachycardia, a pericardial friction rub or evidence of heart failure. On auscultation,

MANIFESTATIONS Acute rheumatic fever

CARDIAC
- Chest pain
- Friction rub
- Heart murmur

MUSCULOSKELETAL
- *Migratory polyarthritis*: redness, heat, swelling, pain and tenderness of more than one joint
- Usually affects large joints of extremities

SKIN
- *Erythema marginatum*: transitory pink, non-pruritic, macular lesions on trunk or inner aspect of upper arms or thighs
- *Subcutaneous nodules* over extensors of wrist, elbow, ankle and knee joints

NEUROLOGICAL
- *Sydenham's chorea:* irritability, behaviour changes; sudden, jerky, involuntary movements

BLOOD TESTS
- Serum antistreptolysin O (ASO) antibodies titres are measured to confirm the diagnosis. Serum ASO rises within 1–2 weeks of infection and reaches a maximum at about 3–6 weeks after infection.
- Serum antideoxyribonuclease B (anti-DNase B) antibodies are also commonly measured to increase the sensitivity of the testing. It is produced by all group A beta-haemolytic streptococci. Serum anti-DNase B peaks 4–8 weeks after infection, remains elevated for several months, and declines slowly.

TABLE 30.6 Major and minor manifestations of rheumatic fever

	HIGH-RISK GROUPS	ALL OTHER GROUPS
Major manifestations	Carditis (including subclinical evidence of rheumatic valve disease on echocardiogram) Polyarthritis, aseptic monoarthritis or polyarthralgia Chorea Erythema marginatum Subcutaneous nodules	Carditis (excluding subclinical evidence of rheumatic valvulitis on echocardiogram) Polyarthritis Chorea Erythema marginatum Subcutaneous nodules
Minor manifestations	Monoarthralgia Fever ESR ≥ 30 mm/h or CRP level ≥ 30 mg/L Prolonged PR interval on ECG	Fever Polyarthralgia or aseptic monoarthritis ESR ≥ 30 mm/h or CRP level ≥ 30 mg/L Prolonged PR interval on ECG

CRP = C-reactive protein, ECG = electrocardiogram, ESR = erythrocyte sedimentation rate.
Source: RHD Australia, National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand (2012). *Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease* (2nd ed.). Retrieved from www.rhdaustralia.org.au/sites/default/files/guideline_0.pdf.

an S_3, S_4 or heart murmur may be heard. Cardiomegaly or pericardial effusion may develop. See Table 30.6 for other manifestations.

INTERPROFESSIONAL CARE

Management of the person with rheumatic heart disease focuses on eradicating the streptococcal infection and managing the manifestations of the disease. Carditis and resulting heart failure are treated with measures to reduce the inflammatory process and manage the heart failure. Activities are limited, but bed rest is not generally ordered.

Diagnosis

As Australia has a significant incidence of rheumatic heart disease, RHD Australia, the National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand (2012) have modified the two previously used tools (Jones criteria and the WHO criteria) to develop the 2012 Australian guidelines for the diagnosis of ARF. In addition to the history and physical examination, a number of laboratory and diagnostic tests may be ordered for the person with suspected rheumatic fever. For diagnosis guidelines, see Tables 30.7 and 30.8.

TABLE 30.7 Australian diagnostic criteria for acute rheumatic fever, 2012

MANIFESTATION	HIGH RISK	LOW RISK
Carditis	Major	Major
Subclinical carditis	Major	n/a
Prolonged PR interval	Minor	Minor
Polyarthritis	Major	Major
Polyarthralgia	Major	Minor
Aseptic monoarthritis	Major	Minor
Monoarthralgia	Major	n/a
Subcutaneous nodules	Major	Major
Sydenham's chorea	Major	Major
Erythema marginatum	Major	Major
Fever	Minor	Minor
Inflammatory	Minor	Minor
Evidence of recent streptococcal infection	Required	Required

Source: RHD Australia, National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand (2012). *Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease* (2nd ed.). Retrieved from www.rhdaustralia.org.au/sites/default/files/guideline_0.pdf.

- *Full blood count (FBC)* and *erythrocyte sedimentation rate (ESR)* are indicators of the inflammatory process. The WBC count is elevated and the number of RBCs may be low due to the inflammatory inhibition of erythropoiesis. The ESR, a general indicator of inflammation, is elevated.

Medications

As soon as rheumatic fever is diagnosed, antibiotics are started to eliminate the streptococcal infection. Intramuscular benzathine penicillin G (BPG) is the antibiotic of choice to treat group A streptococci. A single dose of intramuscular BPG or a 10-day dose of oral penicillin V is administered. Erythromycin is used if the person is allergic to penicillin (RHD Australia, 2012).

Joint pain and fever are treated with salicylates (e.g. aspirin), ibuprofen or another non-steroidal anti-inflammatory drug (NSAID); corticosteroids may be used for severe pain due to inflammation. See Chapter 12 for information about the use of these anti-inflammatory medications.

Nursing care

Health promotion

Rheumatic fever is preventable. Prompt identification and treatment of a person's streptococcal throat infection helps to decrease spread of the pathogen and the risk of rheumatic fever. Characteristics of streptococcal pharyngitis include a sore throat,

TABLE 30.8 Diagnosis guidelines for rheumatic heart disease

	HIGH-RISK GROUPS	ALL OTHER GROUPS
Definite initial episode of ARF	2 major OR 1 major and 2 minor manifestations, PLUS evidence of a preceding group A streptococcal infection	As for high-risk groups
Definitive recurrent episode of ARF in a patient with known past ARF or RHD	2 major OR 1 major and 1 minor OR 3 minor manifestations, PLUS evidence of a preceding group A streptococcal infection	As for high-risk groups
Probable ARF (first episode or recurrence)	A clinical presentation that falls short by either one major or one minor manifestation, or the absence of streptococcal serology results, but one in which ARF is considered the most likely diagnosis. Such cases should be further categorised according to the level of confidence with which the diagnosis is made: • highly-suspected ARF • uncertain ARF	As for high-risk groups

Source: RHD Australia, National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand (2012). *Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease* (2nd ed.). Retrieved from www.rhdaustralia.org.au/sites/default/files/guideline_0.pdf.

odynophagia, headache, fever, tonsillopharyngeal erythema, swollen uvula and lymphadenopathy. The importance of finishing the complete course of medication to eradicate the pathogen must be emphasised. As the route of choice of antibiotic administration is intramuscular, methods to reduce pain at the injection site should be implemented. Simple interventions include: choosing a 23-gauge needle; ensuring the alcowipe is dry before inserting the needle; applying pressure on site (with thumb) for 10 seconds before administration; injecting slowly (over > 2–3 minutes); and using distraction techniques to assist with reducing pain.

Assessment

Assess the person at risk of rheumatic fever (prolonged, untreated or recurrent pharyngitis) for possible manifestations.

- *Health history:* complaints of recent sore throat with fever, difficulty swallowing and general malaise; treatment measures; previous history of strep throat or rheumatic fever; history of heart murmur or other cardiac problems; current medications.
- *Physical examination:* vital signs, including temperature; skin colour, presence of rash on trunk or proximal extremities; mental status; evidence of inflamed joints; heart and lung sounds.
- *Diagnostic tests:* full blood count with differential, ESR, CRP results, throat culture, ECG and echocardiogram.

Nursing diagnoses and interventions

The nursing care focus for the person with RHD is on providing supportive care and preventing complications. Teaching to prevent recurrence of rheumatic fever is extremely important. *Pain* and *Activity intolerance* are priority nursing diagnoses for the person with rheumatic fever and RHD.

Acute pain

Joint and chest pain due to acute inflammation is common in rheumatic fever. Pain and inflammation may interfere with rest and healing.

- Administer anti-inflammatory drugs as ordered. Promptly report manifestations of aspirin toxicity, including tinnitus, vomiting and gastrointestinal bleeding. Give aspirin and other NSAIDs with food, milk or antacids to minimise gastric irritation. *Joint pain and fever may be treated with anti-inflammatory agents such as aspirin and NSAIDs. When used for its anti-inflammatory effect, aspirin doses may be high and it is given around the clock (e.g. every 4 hours). Steroids may be prescribed for severe carditis.*
- Provide warm compresses for local pain relief of acutely inflamed joints. *Warmth helps relieve pain associated with inflamed joints by reducing inflammation.*
- Auscultate heart sounds as indicated (every shift or each home visit). Notify the doctor if a pericardial friction rub or a new murmur develops. *A friction rub is produced as inflamed pericardial surfaces rub against each other. This also stimulates pain receptors and may increase discomfort.*

Activity intolerance

The person with acute carditis or RHD may develop heart failure if the heart is unable to supply enough oxygen to meet the body's demand. Manifestations of fatigue, weakness and dyspnoea on exertion may result.

- Explain the importance of activity limitations and reinforce teaching as needed. *Activities are limited during the acute phase of carditis to reduce the workload of the heart. Understanding the rationale improves cooperation with the limitations.*
- Encourage social and diversional activities such as visits with friends and family, reading, playing cards or board games, watching television and listening to music or talking books. *Diversional activities provide a focus for the person whose physical activities must be limited.*
- Encourage gradual increases in activity, monitoring for evidence of intolerance or heart failure. Consult a cardiac rehabilitation specialist to help design an activity progression schedule. *Gradual activity progression is encouraged as the person's condition improves. Activity tolerance is monitored and activities modified as needed.*

Community-based care

Most people with rheumatic fever and carditis do not require hospitalisation. Teaching for home care focuses on both acute care and preventing recurrences and further tissue damage. Include the following topics:

- the importance of completing the full course of antibiotic therapy and continuing antibiotic prophylaxis as prescribed. For the individual with chronic RHD, include the importance of antibiotic prophylaxis for invasive procedures (e.g. dental care, endoscopy or surgery) to prevent bacterial endocarditis. Pamphlets on endocarditis prevention are helpful reminders and are available from the National Heart Foundation
- preventive dental care and good oral hygiene to maintain oral health and prevent gingival infections, which can lead to recurrence of the disease
- early recognition of streptococcal sore throat and appropriate treatment for both the individual and their family members
- early manifestations of heart failure to report to the doctor
- prescribed medications, including their dosage, route, intended and potential adverse effects, and manifestations to report to the doctor
- dietary sodium restriction if ordered or recommended. A high-carbohydrate, high-protein diet may be recommended to facilitate healing and combat fatigue.

Refer for home health services or household assistance as indicated.

THE PERSON WITH INFECTIVE ENDOCARDITIS

Endocarditis, inflammation of the endocardium, can involve any portion of the endothelial lining of the heart. The valves usually are affected. Endocarditis is usually infectious in nature, characterised by colonisation or invasion of the endocardium and heart valves by a pathogen.

Incidence and risk factors

Endocarditis is relatively uncommon in Australia. In 2013, endocarditis was listed as the cause of death for only 349 people, which equates to 0.24% of all deaths that year (ABS, 2015), and incidence statistics are difficult to locate. Nevertheless, a little over 2000 people were admitted to hospital with a primary diagnosis of endocarditis in 2012–2013 (AIHW, 2015).

The greatest risk factor for endocarditis is previous heart damage. Lesions develop on deformed valves, on valve prostheses or in areas of tissue damage due to congenital deformities or ischaemic disease. The left side of the heart—the mitral valve, in particular—is usually affected. Intravenous drug use also is a significant risk factor. The right side of the heart usually is affected in these individuals. Other risk factors include invasive catheters (e.g. a central venous catheter, haemodynamic monitoring or an indwelling urinary catheter), dental procedures or poor dental health, and recent heart surgery.

FAST FACTS

- Subacute bacterial endocarditis develops more slowly and usually occurs in people with previous heart valve damage.
- Acute bacterial endocarditis has an abrupt onset and typically affects people with no previous history of heart problems.

Prosthetic valve endocarditis (PVE) may occur in individuals with a mechanical or tissue valve replacement. This infection may develop in the early postoperative period (within 2 months after surgery) or late. Prosthetic valve endocarditis accounts for 10–20% of endocarditis cases (O'Connor & Kiernan, 2015). Early PVE occurs within 60 days of valve implantation and is usually due to prosthetic valve contamination during surgery or perioperative bacteraemia. Its course often is rapid and mortality is high. Late-onset PVE occurring after 60 days following surgery more closely resembles subacute endocarditis.

Pathophysiology

Entry of pathogens into the bloodstream is required for infective endocarditis to develop. Bacteria may enter through oral lesions, during dental work or invasive procedures such as intravenous catheter insertion, surgery or urinary catheterisation; during intravenous drug use; or as a result of infectious processes such as urinary tract or upper respiratory infection.

The initial lesion is a sterile platelet–fibrin vegetation formed on damaged endothelium (see Figure 30.4). In acute infective endocarditis, these lesions develop on healthy valve structures, although the mechanism is unknown. In subacute endocarditis, they usually develop on already damaged valves or in endocardial tissue that has been damaged by abnormal pressures or blood flow within the heart.

Organisms that have invaded the blood colonise these vegetations. The vegetation enlarges as more platelets and fibrin are attracted to the site and cover the infecting organism. This covering 'protects' the bacteria from quick removal by immune defences such as phagocytosis by neutrophils, antibodies and

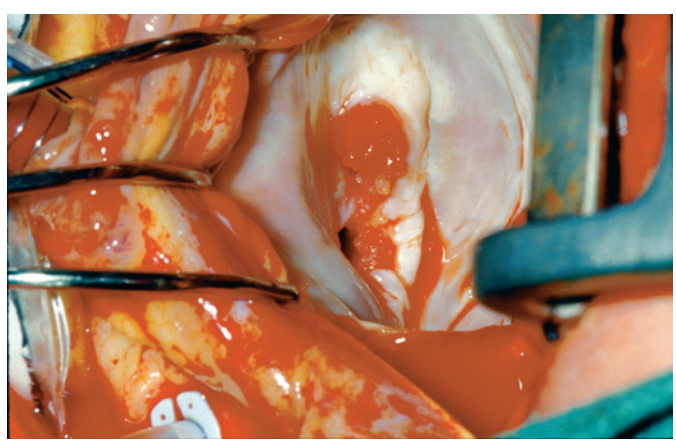


FIGURE 30.4 ■ A vegetative lesion of bacterial endocarditis

Source: M. English/Custom Medical Stock Photo, Inc.

complement. Vegetations may be singular or multiple. They expand while loosely attached to the edges of the valve. Friable vegetations can break or shear off, embolising and travelling through the bloodstream to other organ systems. When they lodge in small vessels, they may cause haemorrhages, infarcts or abscesses. Ultimately, the vegetations scar and deform the valves and cause turbulence of blood flowing through the heart. Heart valve function is affected, either obstructing forward blood flow or closing incompletely.

Endocarditis is classified by its acuity and disease course. *Acute infective endocarditis* (IE) has an abrupt onset and is a rapidly progressive, severe disease. Further classifications can be made as to whether the endocarditis has occurred on a person's native valve (NVE), on a prosthetic valve (PVE) or as a result of intravenous drug abuse (IVDA IE). Although almost any organism can cause infective endocarditis, virulent organisms such as *Staphylococcus aureus* cause a more abrupt onset and destructive course. *S. aureus* is commonly the infective organism in acute endocarditis. In contrast, *subacute infective endocarditis* has a more gradual onset, with predominant systemic manifestations. It is more likely to occur in people with pre-existing heart disease and is commonly caused by alpha-haemolytic streptococci or enterococci. In IVDA infective endocarditis, *S. aureus* is the most common organism with the majority of cases affecting the tricuspid valve (Brusch, 2014).

Manifestations

The manifestations of infective endocarditis often are non-specific (see the box below). A temperature above 39.4ºC and flu-like symptoms develop, accompanied by cough, shortness of breath and joint pain. The presentation of acute staphylococcal endocarditis is more severe, with a sudden onset, chills and a high fever. Heart murmurs are heard in 90% of individuals with infective endocarditis. An existing murmur may worsen or a new murmur may develop.

Splenomegaly is common in chronic disease. Peripheral manifestations of infective endocarditis result from microemboli or circulating immune complexes. These manifestations include:

- *Petechiae:* small, purplish-red haemorrhagic spots on the trunk, conjunctiva and mucous membranes.
- *Splinter haemorrhages:* haemorrhagic streaks under the fingernails or toenails.
- *Osler's nodes:* small, reddened, painful raised growths on finger and toe pads.
- *Janeway lesions:* small, non-tender, purplish-red macular lesions on the palms of the hands and soles of the feet.
- *Roth's spots:* small, whitish spots (cottonwool spots) seen on the retina.

Complications

Embolisation of vegetative fragments may affect any organ system, particularly the lungs, brain, kidneys and the skin and mucous membranes, with resulting organ infarction. Other common complications of infective endocarditis include heart failure, abscess and aneurysms due to infiltration of the arterial wall by organisms. Without treatment, endocarditis is almost universally fatal; fortunately, antibiotic therapy is usually effective to treat this disease.

MANIFESTATIONS Infective endocarditis

- Chills and fever
- General malaise, fatigue
- Arthralgias
- Cough, dyspnoea
- Heart murmur
- Anorexia, abdominal pain
- Petechiae, splinter haemorrhages
- Splenomegaly

INTERPROFESSIONAL CARE

Eradicating the infecting organism and minimising valve damage and other adverse consequences of infective endocarditis are the priorities of care.

Diagnosis

There are no definitive tests for infective endocarditis, but diagnostic tests help establish the diagnosis.

- *Blood cultures* usually are positive for bacteria or other pathogens. Blood cultures are considered positive when a typical infecting organism is identified from two or more separate blood cultures (drawn from different sites and/or at different times, e.g. 12-hour intervals).
- *Echocardiography* (either transthoracic or transoesophageal) to visualise vegetations can be diagnostic for infective endocarditis when combined with positive blood cultures. See Chapter 28 for more information about echocardiography.
- *Serological immune testing* for circulating antigens to typical infective organisms may be done.

Other diagnostic tests may include the FBC, ESR, serum creatinine, chest x-ray and an electrocardiogram.

Medications

Preventing endocarditis in individuals at high risk is important. Antibiotics are commonly prescribed for individuals with pre-existing valve damage or heart disease prior to high-risk procedures.

Antibiotic therapy effectively treats infective endocarditis in most cases. The goal of therapy is to eradicate the infecting organism from the blood and vegetative lesions in the heart. The fibrin covering that protects colonies of organisms from immune defences also protects them from antibiotic therapy. Therefore, an extended course of multiple intravenous antibiotics is required.

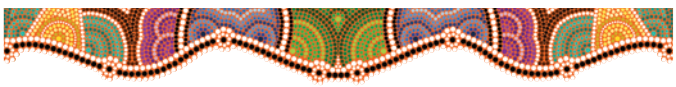
Following blood cultures, antibiotic therapy is initiated with drugs known to be effective against the most common infecting organisms: *Staphylococcus*, *Streptococcus* and *Enterococcus* species. The initial regimen will include an antibiotic to which the causative organism is sensitive. See Chapter 11 for the nursing implications for antibiotic therapy.

Surgery

Some individuals with infective endocarditis require surgery to:

- replace severely damaged valves
- remove large vegetations at risk of embolisation
- remove a valve that is a continuing source of infection that does not respond to antibiotic therapy.

The most common indication for surgery is valvular regurgitation that causes heart failure and does not respond to medical therapy. When the infection has not responded to antibiotic therapy within 7 to 10 days, the infected valve may be replaced to facilitate eradication of the organism. Individuals with fungal endocarditis usually require surgical intervention. More information on valve replacement surgery is provided in the section on valve disorders.



Nursing care

Health promotion

Prevention of endocarditis is vital in susceptible people. Education is a key part of prevention. Use every opportunity to educate individuals and the public about the risks of intravenous drug use, including endocarditis. Discuss preventive measures with all individuals with specific risk factors, such as a heart murmur or known heart disease.

Assessment

Assessment related to infective endocarditis includes identifying risk factors and manifestations of the disease.

- *Health history:* complaints of persistent flu-like symptoms, fatigue, shortness of breath and activity intolerance; history of recent dental work or other invasive procedures; known heart murmur, valve or other heart disorder; recent intravenous drug use.
- *Physical examination:* vital signs, including temperature; apical pulse and heart sounds; rate and ease of respirations, lung sounds; skin colour, temperature and presence of petechiae or splinter haemorrhages.
- *Diagnostic tests:* FBC and differential, ESR; blood culture and sensitivity results; echocardiogram reports.

Nursing diagnoses and interventions

Nursing care focuses on managing the manifestations of endocarditis, administering antibiotics and teaching the person and their family members about the disorder. In addition to the diagnoses identified below, nursing diagnoses and interventions for heart failure also may be appropriate for individuals with infective endocarditis.

Risk of imbalanced body temperature

Fever is common in individuals with infective endocarditis. It may be acutely elevated and accompanied by chills, particularly with acute infective endocarditis. The inflammatory process initiates a cycle of events that affects the regulation of temperature and causes discomfort.

- Record temperature every 2 to 4 hours. Report temperature above 38.5ºC. Assess for complaints of discomfort. *Fever is usually low grade (38ºC) in infective endocarditis; higher temperatures may cause discomfort. The temperature usually returns to normal within 1 week after initiation of antibiotic therapy. Continued fever may indicate a need to modify the treatment regimen.*
- Obtain blood cultures as ordered, before initial antibiotic dose. *Initial blood cultures are obtained before antibiotic therapy is started to obtain adequate organisms to culture and identify. Follow-up cultures are used to assess the effectiveness of therapy.*
- Provide anti-inflammatory or antipyretic agents as prescribed. *Fever may be treated with anti-inflammatory or antipyretic agents such as aspirin, ibuprofen or paracetamol.*
- Administer antibiotics as ordered; obtain peak and trough drug levels as indicated. Intravenous antibiotics are given to eradicate the pathogen. *Peak and trough levels are used to evaluate the dose effectiveness in maintaining a therapeutic blood level.*

Risk of ineffective tissue perfusion

Embolisation of vegetative lesions can threaten tissue and organ perfusion. Vegetations from the left heart may lodge in arterioles or capillaries of the brain, kidneys or peripheral tissues, causing infarction or abscess. A large embolism can cause manifestations of stroke or transient ischaemic attack, renal failure or tissue ischaemia. Emboli from the right side of the heart become entrapped in pulmonary vasculature, causing manifestations of pulmonary embolism.

- Assess for, document and report manifestations of decreased organ system perfusion:
  a. *Neurological:* changes in level of consciousness, numbness or tingling in extremities, hemiplegia, visual disturbances or manifestations of stroke.
  b. *Renal:* decreased urine output, haematuria, elevated urea or creatinine.
  c. *Pulmonary:* dyspnoea, haemoptysis, diminished breath sounds, restlessness, sudden chest or shoulder pain.
  d. *Cardiovascular:* chest pain radiating to jaw or arms, tachycardia, anxiety, tachypnoea, hypotension.

  All major organs and tissues and the microcirculation may be affected by emboli when vegetations break off due to turbulent blood flow. Emboli may cause manifestations of organ dysfunction. The most devastating effects of emboli are in the brain and the myocardium, with resulting infarctions. Intravenous drug users have a high risk of pulmonary emboli as a result of right-sided endocardial fragments.
- Assess and document skin colour and temperature, quality of peripheral pulses and capillary refill. *Peripheral emboli affect tissue perfusion, with a risk of tissue necrosis and possible extremity loss.*

Ineffective health maintenance

The person with endocarditis is often treated in the community. Teaching about disease management and prevention of possible recurrences of endocarditis is vital.

- Demonstrate intravenous catheter site care and intermittent antibiotic administration if the person and their family will manage therapy. Have the person and/or significant other redemonstrate appropriate techniques. *Intermittent antibiotic infusions may be managed by the person or family members, or the individual may go to an outpatient facility to receive the infusions. Appropriate site care is necessary to reduce the risk of trauma and infection.*
- Explain the actions, doses, administration and desired and adverse effects of prescribed drugs. Identify manifestations to be reported to the doctor. Provide practical information about measures to reduce the risk of superinfection (e.g. the concomitant use of antithrush preparations). *Careful compliance with prescribed drug therapy is vital to eradicate the infecting organism. Antibiotic therapy can, however, cause superinfections such as candidiasis due to elimination of normal body flora.*
- Teach the person about the function of heart valves and the effects of endocarditis on heart function. Include a simple definition of endocarditis and explain the risk of its recurrence. *Information helps the person and their family understand endocarditis, its treatment and its effects. Understanding increases compliance.*
- Describe the manifestations of heart failure to be reported to the doctor. *Evidence of heart failure may necessitate modification of the treatment regimen or replacement of infected valves.*
- Encourage good dental hygiene and mouth care and regular dental checkups. Teach the person how to prevent bleeding from the gums and avoid developing mouth ulcers (e.g. gentle tooth brushing, ensuring that dentures fit properly and avoiding toothpicks, dental floss and high-flow water devices). *The oropharynx harbours streptococci, which are common causes of endocarditis. Bleeding gums offer an opportunity for bacteria to enter the bloodstream.*
- Encourage the person to avoid people with upper respiratory infections. *Streptococci are normal pathogens in the upper respiratory tract; exposure to people with upper respiratory infections may increase the risk of infection.*
- If anticoagulant therapy is ordered, explain its actions, administration and major side effects. Identify manifestations of bleeding to be promptly reported to the doctor. *Individuals with valve disease or a prosthetic valve following infective endocarditis may require continued anticoagulant therapy to prevent thrombi and emboli. Knowledge is vital for appropriate management of anticoagulant therapy and prevention of complications.*

CONSIDERATION FOR PRACTICE

Stress the importance of notifying all care providers of valve disease, heart murmur or valve replacement before undergoing invasive procedures. Invasive procedures provide a portal of entry for bacteria. A history of valve disease increases the risk of the development or recurrence of endocarditis.

Community-based care

When preparing the person with infective endocarditis for home care, provide teaching as outlined for the nursing diagnosis of *Ineffective health maintenance*. In addition, discuss the following topics:

- Although serious and frightening, infective endocarditis can usually be treated effectively with intravenous antibiotics.
- The importance of promptly reporting any unusual manifestation, such as a change in vision, sudden pain or weakness, so that interventions to control complications can be promptly implemented.
- The rationale for all treatments and procedures.
- Preventing recurrences of infective endocarditis.
- The importance of maintaining contact with the doctor for follow-up care and monitoring for long-term effects such as progressive valve damage and dysfunction.
- If appropriate, explain the risks associated with intravenous drug use.

Provide educational materials on infective endocarditis from the National Heart Foundation. Refer as appropriate to home health or home intravenous therapy services. Refer the person and their family members or significant others as appropriate to a drug or substance abuse treatment program or facility. Provide follow-up care to ensure compliance with the referral and treatment plan.

THE PERSON WITH MYOCARDITIS

Myocarditis is inflammation of the heart muscle. It usually results from an infectious process, but also may occur as an immunological response or due to the effects of radiation, toxins or drugs.

Incidence and risk factors

Myocarditis may occur at any age and is more common in men than women. Factors that alter immune response (e.g. malnutrition, alcohol use, immunosuppressive drugs, exposure to radiation, stress and advanced age) increase the risk of myocarditis. It also is a common complication of rheumatic fever and pericarditis. Myocarditis can also be an adverse effect of a common antipsychotic, clozapine.

Pathophysiology

Myocardial cells are damaged by an inflammatory process that causes local or diffuse swelling and damage. Infectious

agents infiltrate interstitial tissues, forming abscesses. Autoimmune injury may occur when the immune system destroys not only the invading pathogen but also myocardial cells. The extent of damage to cardiac muscle ultimately determines the long-term outcome of the disease. Viral myocarditis usually is self-limiting; it may progress, however, to become chronic, leading to dilated cardiomyopathy. (See later in this chapter.) Severe myocarditis may lead to heart failure.

Manifestations

The manifestations of myocarditis depend on the degree of myocardial damage. The person may be asymptomatic. Non-specific manifestations of inflammation, such as fever, fatigue, general malaise, dyspnoea, palpitations and arthralgias, may be present. A non-specific febrile illness or upper respiratory infection often precedes the onset of myocarditis symptoms. Abnormal heart sounds such as muffled S_1, an S_3, murmur and pericardial friction rub may be heard. In some cases, manifestations of myocardial infarction, including chest pain, may occur.

INTERPROFESSIONAL CARE

Myocarditis treatment focuses on resolving the inflammatory process to prevent further damage to the myocardium.

Diagnosis

Diagnostic studies may be ordered to help diagnose myocarditis:

- *Electrocardiography* may show transient ST-segment and T-wave changes, as well as arrhythmias and possible heart block.
- *Cardiac markers*, such as creatinine kinase (CK), troponin T (cT_nT) and troponin I (cT_nI), may be elevated, indicating myocardial cell damage.
- *Endomyocardial biopsy* to examine myocardial cells is necessary to establish a definitive diagnosis; patchy cell necrosis and the inflammatory process can be identified.

Medications

If appropriate, antimicrobial therapy is used to eradicate the infecting organism. Antiviral therapy with interferon alpha may be instituted. Immunosuppressive therapy with corticosteroids or other immunosuppressive agents (see Chapter 12) may be used to minimise the inflammatory response. Heart failure is treated as needed, using ACE inhibitors and other cardiac drugs. Individuals with myocarditis often are particularly sensitive to the effects of digoxin, so it is used with caution. Other medications used in treating myocarditis include antiarrhythmic agents to control arrhythmias and anticoagulants to prevent emboli.

Bed rest and activity restrictions are ordered during the acute inflammatory process to reduce myocardial work and prevent myocardial damage. Activities may be limited for as long as 6 months to a year (Tang, 2014).

Nursing care

Nursing care is directed at decreasing myocardial work and maintaining cardiac output. Both physical and emotional rest are indicated because anxiety increases myocardial oxygen demand. Haemodynamic parameters and the ECG are monitored closely, especially during the acute phase of the illness. Activity tolerance, urine output and heart and breath sounds are frequently assessed for manifestations of heart failure. Consider the following nursing diagnoses for the person with myocarditis:

- *Risk of activity intolerance* related to impaired cardiac muscle function manifested by inability to undertake activities of daily living independently.
- *Risk of decreased cardiac output* related to myocardial inflammation manifested by hypotension, tachycardia, and shortness of breath.
- *Risk of fatigue* related to inflammation and impaired cardiac output manifested by inability to mobilise or perform self-care without resting frequently.
- *Risk of anxiety* related to possible long-term effects of the disorder manifested by verbal statements and behaviour consistent with anxiety.
- *Risk of excess fluid volume* related to compensatory mechanisms for decreased cardiac output manifested by orthopnoea, dyspnoea, oedema and weight gain.

Community-based care

Include the following topics when preparing the person with myocarditis for home care:

- activity restrictions and other prescribed measures to reduce cardiac workload
- early manifestations of heart failure to report to the doctor
- the importance of following the prescribed treatment regimen
- any recommended dietary modifications (such as a low-sodium diet for heart failure)
- prescribed medications, their purpose, doses and possible adverse effects
- the importance of adhering to the treatment plan and recommended follow-up appointments to reduce the risk of long-term consequences such as cardiomyopathy.

THE PERSON WITH PERICARDITIS

The pericardium is the outermost layer of the heart. It is a two-layered membranous sac with a thin layer of serous fluid (normally no more than 30 to 50 mL) separating the layers. It protects and cushions the heart and the great vessels, provides a barrier to infectious processes in adjacent structures, prevents displacement of the myocardium and blood vessels, and prevents sudden distension of the heart.

BOX 30.3 Selected causes of pericarditis

Infectious
- Viruses
- Bacteria
- Fungi
- Parasites

Non-infectious
- Myocardial and pericardial injury
- Uraemia
- Neoplasms
- Radiation
- Trauma or surgery
- Myxoedema
- Autoimmune disorders
- Rheumatic fever
- Connective tissue diseases
- Some drugs
- Post-cardiac injury

Pericarditis is the inflammation of the pericardium. Pericarditis may be a primary disorder or may develop secondarily to another cardiac or systemic disorder. Some possible causes of pericarditis are listed in Box 30.3. Viral infections are more common than bacterial and fungal pericarditis (Spangler, 2014). Other types of pericarditis can develop post myocardial infarction and post cardiotomy (following open-heart surgery).

Pathophysiology

Pericardial tissue damage triggers an inflammatory response. Inflammatory mediators released from the injured tissue cause vasodilation, hyperaemia and oedema. Capillary permeability increases, allowing plasma proteins, including fibrinogen, to escape into the pericardial space. White blood cells amass at the site of injury to destroy the causative agent. Exudate is formed, usually fibrinous or serofibrinous (a mixture of serous fluid and fibrinous exudate). In some cases, the exudate may contain red blood cells or, if infectious, purulent material. The inflammatory process may resolve without long-term effects or scar tissue and adhesions may form between the pericardial layers.

Fibrosis and scarring of the pericardium may restrict cardiac function. Pericardial effusions may develop as serous or purulent exudate (depending on the causative agent) collects in the pericardial sac. Pericardial effusion may be recurrent. Chronic inflammation causes the pericardium to become rigid.

Manifestations

Classic manifestations of acute pericarditis include chest pain, a pericardial friction rub and fever. Chest pain, the most common symptom, has an abrupt onset. It is caused by inflammation of nerve fibres in the lower parietal pericardium and pleura covering the diaphragm. The pain is usually sharp, may be steady or intermittent and may radiate to the back or neck. The pain can mimic myocardial ischaemia; careful assessment is important to rule out myocardial infarction. Pericardial pain is aggravated by respiratory movements (i.e. deep inspiration and/or coughing), changes in body position or swallowing. Sitting upright and leaning forward reduces the discomfort by moving the heart away from the diaphragmatic side of the lung pleura.

Although not always present, a pericardial friction rub is the characteristic sign of pericarditis. A pericardial friction rub is a leathery, grating sound produced by the inflamed pericardial layers rubbing against the chest wall or pleura. It is heard most clearly at the left lower sternal border with the person sitting up or leaning forward. The rub is usually heard on expiration and may be constant or intermittent.

The person may develop low-grade fever (below 38.4ºC) due to the inflammatory process. Dyspnoea and tachycardia are common.

Complications

Pericardial effusion, cardiac tamponade and constrictive pericarditis are possible complications of acute pericarditis.

Pericardial effusion

A *pericardial effusion* is an abnormal collection of fluid between the pericardial layers that threatens normal cardiac function. The fluid may consist of pus, blood, serum, lymph or a combination. The manifestations of a pericardial effusion depend on the rate at which the fluid collects. Although the pericardium normally contains about 30 to 50 mL of fluid, the sac can stretch to accommodate a gradual accumulation of fluid. Over time, the pericardial sac can accommodate up to 2 L of fluid without immediate adverse effects. Conversely, a rapid build-up of pericardial fluid (as little as 100 mL) does not allow the sac to stretch and can compress the heart, interfering with myocardial function. This compression of the heart is known as **cardiac tamponade**. Slowly developing pericardial effusion is often painless and has few manifestations. Heart sounds may be distant or muffled. The person may have a cough or mild dyspnoea.

Cardiac tamponade

Cardiac tamponade is a medical emergency that must be aggressively treated to preserve life. Cardiac tamponade may result from pericardial effusion, trauma, cardiac rupture or haemorrhage. Rapid collection of fluid in the pericardial sac interferes with ventricular filling and pumping, critically reducing cardiac output.

Classic manifestations of cardiac tamponade result from rising intracardiac pressures and decreased diastolic filling and cardiac output. A hallmark of cardiac tamponade is a paradoxical pulse, or *pulsus paradoxus*. A paradoxical pulse markedly decreases in amplitude during inspiration. Intrathoracic pressure normally drops during inspiration, enhancing venous return to the right heart. This draws more blood into the right side of the heart than the left, causing the interventricular septum to bulge slightly into the left ventricle. When ventricular filling is impaired by excess fluid in the pericardial sac, this bulging of the interventricular septum decreases cardiac output during inspiration (see Figure 30.5). On palpation of the carotid or femoral artery, the pulse is diminished or absent during inspiration. A drop in systolic blood pressure of more than 10 mmHg during inspiration also indicates pulsus paradoxus.

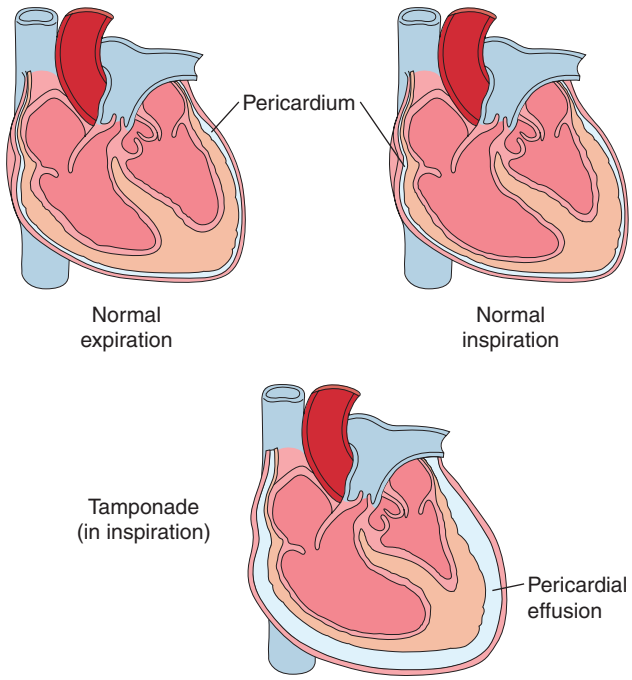


FIGURE 30.5 ■ Cardiac tamponade. Note increased volume in the right ventricle during inspiration in both the normal heart and the heart affected by a pericardial effusion. In tamponade, fluid in the pericardial sac and the distended right ventricle restrict filling of the left ventricle and, consequently, cardiac output

Other manifestations of cardiac tamponade include muffled heart sounds, dyspnoea and tachypnoea, tachycardia, a narrowed pulse pressure and distended neck veins (see the box below).

Chronic constrictive pericarditis

Chronic pericardial inflammation can lead to scar tissue formation between the pericardial layers. This scar tissue eventually contracts, restricting diastolic filling and elevating venous pressure. Constrictive pericarditis (see Figure 30.6) may follow viral infection, radiation therapy or heart surgery. Its manifestations include progressive dyspnoea, fatigue and weakness. Ascites is common; peripheral oedema may develop. Neck veins are distended and may be particularly noticeable during inspiration (*Kussmaul's sign*). This occurs because the right atrium is unable to dilate to accommodate increased venous return during inspiration.

MANIFESTATIONS Cardiac tamponade

- Paradoxical pulse
- Narrowed pulse pressure, hypotension
- Tachycardia
- Weak peripheral pulses
- Distant, muffled heart sounds
- Jugular venous distension
- High central venous pressure
- Decreased level of consciousness
- Low urine output
- Cool, mottled skin

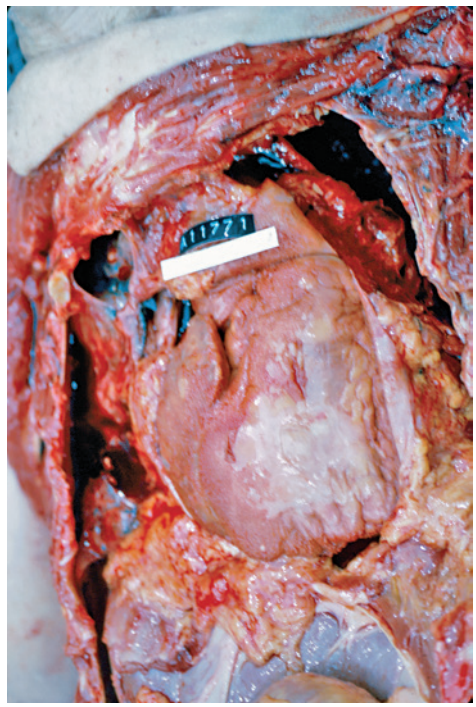


FIGURE 30.6 ■ Constrictive pericarditis

Source: Custom Medical Stock Photo, Inc.

INTERPROFESSIONAL CARE

Care for the person with pericarditis focuses on identifying its cause, if possible, reducing inflammation, relieving symptoms and preventing complications. The person is closely monitored for early manifestations of cardiac tamponade so that it can be treated promptly.

Diagnosis

There are no specific laboratory tests to diagnose pericarditis, but tests are often performed to differentiate pericarditis from myocardial infarction.

- *FBC* shows elevated WBCs and an ESR greater than 20 mm/h, indicating acute inflammation.
- *Cardiac enzymes* may be slightly elevated because the inflammatory process extends to involve the epicardial surface of the heart. Cardiac enzymes are typically much lower in pericarditis than in myocardial infarction.
- *Electrocardiography* shows typical changes associated with pericarditis, such as diffuse ST-segment elevation in all leads. This resolves more quickly than changes of acute MI and is not associated with the QRS-complex and T-wave changes typically seen in MI. With a large pericardial effusion, the QRS amplitude may be decreased. Atrial arrhythmias may occur in acute pericarditis.

- *Echocardiography* is used to assess heart motion, for pericardial effusion and the extent of restriction.
- *Haemodynamic monitoring* may be used in acute pericarditis or pericardial effusion to assess pressures and cardiac output. Elevated pulmonary artery pressures and venous pressures occur with impaired filling due to pericardial effusion or constrictive pericarditis.
- *Chest x-ray* may show cardiac enlargement if a pericardial effusion is present.
- *Computed tomography (CT) scan* or *magnetic resonance imaging (MRI)* may be used to identify pericardial effusion or constrictive pericarditis.

Medications

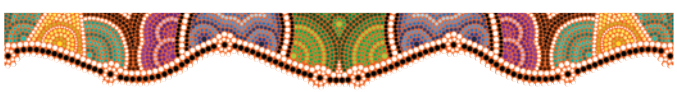
Drug treatment for pericarditis addresses its manifestations. Aspirin and paracetamol may be used to reduce fever. NSAIDs are used to reduce inflammation and promote comfort. In severe cases or with recurrent pericarditis, corticosteroids may be given to suppress the inflammatory response.

Pericardiocentesis

Pericardiocentesis may be done to remove fluid from the pericardial sac for diagnostic or therapeutic purposes. The doctor inserts a large (16- to 18-gauge) needle into the pericardial sac and withdraws excess fluid. The needle is attached to an ECG monitoring lead to help determine if the needle is touching the epicardial surface, which helps prevent piercing the myocardium. Pericardiocentesis may be an emergency procedure for the person with cardiac tamponade.

Surgery

For recurrent pericarditis or recurrent pericardial effusion, a rectangular piece of the pericardium, or 'window', may be excised to allow collected fluid to drain into the pleural space. Constrictive pericarditis may necessitate a partial or total *pericardiectomy*, removal of part or all of the pericardium, to relieve the ventricular compression and allow adequate filling.



Nursing care

Health promotion

While it may not yet be possible to identify many people at risk and to prevent acute pericarditis, early identification and treatment of the disorder can reduce the risk of complications. Promptly report a pericardial friction rub or other manifestations of pericarditis in individuals with recent acute AMI, cardiac surgery or systemic diseases associated with a risk of pericarditis.

Assessment

Assessment data to collect from the person with suspected pericarditis include:

- *Health history:* complaints of acute substernal or precordial chest pain, effect of movement and breathing on discomfort, pain radiation, associated symptoms; recent acute AMI, heart surgery or other cardiac disorder; current medications; chronic conditions such as renal failure or a connective tissue or autoimmune disorder.
- *Physical examination:* vital signs, including temperature, variation in systolic BP with respirations; strength of peripheral pulses, variations with respiratory movement; apical pulse, clarity, changes with respiratory movement, presence of a friction rub; neck vein distension; level of consciousness, skin colour and other indicators of cardiac output.
- *Diagnostic tests:* FBC and differential, ESR; cardiac enzyme levels; ECG and echocardiogram reports.

Nursing diagnoses and interventions

Nursing care for the person with pericarditis may occur in the acute or community setting. Closely observe for early manifestations of increasing effusion or cardiac tamponade. Priority nursing diagnoses relate to comfort, the risk of tamponade and effects of the acute inflammatory process.

Acute pain

Inflamed pericardial layers rubbing against each other and the lung pleura stimulate phrenic nerve pain fibres in the lower portion of the parietal pericardium. Pain is usually acute and may be severe until inflammation resolves.

- Assess chest pain using a standard pain scale and noting the quality and radiation of the pain. Note non-verbal cues of pain (grimacing, guarding behaviours) and validate with the person. *Careful assessment helps identify the cause of pain. The pain of pericarditis may radiate to the neck or back and is aggravated by movement, coughing or deep breathing. A pain scale allows evaluation of the effectiveness of interventions.*
- Auscultate heart sounds every 4 hours. *Presence of a pericardial friction rub often correlates with the location and severity of the pain.*
- Administer NSAIDs on a regular basis as prescribed with food. Document effectiveness. NSAIDs reduce fever, inflammation and pericardial pain. *They are most effective when administered around the clock on a consistent basis. Administering the medications with food helps decrease gastric distress.*
- Maintain a quiet, calm environment and position of comfort. Offer back rubs, heat/cold therapy, diversional activity and emotional support. *Supportive interventions enhance the effects of the medication, may decrease pain perception and convey a sense of caring.*

Ineffective breathing pattern

Respiratory movement intensifies pericardial pain. In an effort to decrease pain, the person often breathes shallowly, increasing the risk of pulmonary complications.

- Encourage deep breathing and use of the incentive spirometer. Provide pain medication before respiratory therapy, as needed. *Deep breathing and an incentive spirometer promote alveolar ventilation and prevent atelectasis. Administration of analgesia prior to respiratory treatments improves their effectiveness by decreasing guarding.*

- Administer oxygen as needed. *Supplementary oxygen promotes optimal gas exchange and tissue oxygenation.*
- Place the person in Fowler's or high-Fowler's position. Assist the person to a position of comfort. *Appropriate positioning reduces the work of breathing and decreases chest pain due to pericarditis.*

CONSIDERATION FOR PRACTICE

Document respiratory rate, effort and breath sounds every 2 to 4 hours. Report adventitious or diminished breath sounds. Shallow, guarded respirations may lead to increased respiratory rate and effort. Poor ventilation of peripheral alveoli may lead to congestion or atelectasis.

Risk of decreased cardiac output

The acute inflammatory process of pericarditis can lead to significant pericardial effusion and cardiac tamponade. This potentially fatal complication can also occur with chronic pericardial effusion if the amount of fluid exceeds the ability of the pericardial sac to expand. Constrictive pericarditis increases the risk of decreased cardiac output because of restricted cardiac filling.

- Document vital signs hourly during the acute inflammatory processes. *Frequent assessment allows early recognition of manifestations of decreased cardiac output, such as tachycardia, hypotension or changes in pulse pressure.*
- Report significant changes or trends in haemodynamic parameters and arrhythmias. *Compression of the heart interferes with venous return, increasing CVP and right atrial pressures; arrhythmias may also occur.*
- Promptly report other signs of decreased cardiac output: decreased level of consciousness; decreased urine output; cold, clammy, mottled skin; delayed capillary refill; and weak peripheral pulses. *These signs of decreased organ and tissue perfusion indicate a significant drop in cardiac output.*
- Maintain at least one patent intravenous access site. *The person in cardiac tamponade may require rapid intravenous fluid infusion to restore blood volume and administration of emergency drugs to support the circulation.*
- Prepare for emergency pericardiocentesis and/or surgery as necessary. Provide appropriate explanations and reassurance. Observe for adverse responses during pericardiocentesis. *Excess pericardial fluid must be rapidly evacuated to prevent further compromise of cardiac output and death. Emotional support and explanations reduce the person's and their family's anxiety and promote a caring atmosphere.*

Activity intolerance

In chronic constrictive pericarditis, pericardial adhesions and scarring restrict pericardial compliance, restricting heart filling and movement. Restricted filling and ineffective cardiac contraction decrease the cardiac output. The heart cannot compensate for increased metabolic demands by increasing cardiac output and cardiac reserve falls significantly.

- Work with the person and physiotherapist to develop a realistic, progressive activity plan. Monitor response. Encourage independence, but provide assistance as needed. *Involvement in their own planning increases the likelihood of success, as well as the person's self-esteem and sense of control. Promoting self-care provides additional control and independence and enhances self-image. Activity that significantly increases the heart rate (more than 20 bpm over resting) should be stopped and reassessed for intensity.*
- Plan interventions and care activities to allow uninterrupted rest and sleep. *This supports healing and restoration of physical and emotional health.*

Community-based care

Include the following topics when teaching the person and their family in preparation for home care:

- The importance of continuing anti-inflammatory medications as ordered. Advise to take NSAIDs with food, milk or antacids to minimise gastric distress and to notify the doctor if unable to tolerate the drug.
- Prescribed medications, including dose, desired and possible adverse effects, and interactions with other drugs or food.
- Monitoring weight twice weekly because NSAIDs may cause fluid retention.
- Maintaining fluid intake of at least 2500 mL/day to minimise the risk of renal toxicity due to NSAID use.
- Measures to maintain activity restriction if ordered. Activity will be gradually increased once the inflammatory process has resolved.

DISORDERS OF CARDIAC STRUCTURE

THE PERSON WITH VALVULAR HEART DISEASE

Proper heart valve function ensures one-way blood flow through the heart and vascular system. **Valvular heart disease** interferes with blood flow to and from the heart. Acquired valvular disorders can result from acute conditions, such as infective endocarditis, or from chronic conditions, such as rheumatic heart disease. Rheumatic heart disease is the most common cause of valvular disease (Papadakis, McPhee & Rabow, 2013). Acute myocardial infarction also can damage heart valves, causing tearing, ischaemia or damage to the papillary muscles that affects valve leaflet function. Congenital heart defects may affect the heart valves, often with no manifestations until adulthood.

Physiology review

The heart valves direct blood flow within and out of the heart. The valves are fibroelastic tissue supported by a ring of fibrous tissue (the annulus) that provides support.

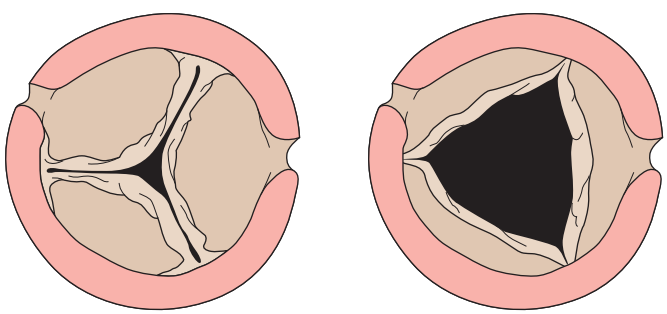
The AV valves, the **mitral** (or *bicuspid*) **valve** on the left and the **tricuspid valve** on the right, separate the atria from the ventricles. These valves normally are fully open during diastole, allowing blood to flow freely from the atria into the ventricles. Rising pressure within the ventricles at the onset of systole (contraction) closes the AV valves, creating the S_1 heart sound ('lub'). The leaflets of the AV valves are connected to ventricular papillary muscles by fibrous *chordae tendineae*. The chordae tendineae prevent the valve leaflets from bulging back into the atria during systole.

The semilunar valves, the **aortic** and **pulmonic valves**, separate the ventricles from the great vessels. They open during systole, allowing blood to flow out of the heart with ventricular contraction. As the ventricle relaxes and intraventricular pressure falls at the beginning of diastole, the higher pressure within the great vessels (the aorta and pulmonary artery) closes these valves, creating the S_2 heart sound ('dub').

Pathophysiology

Valvular heart disease occurs as two main types of disorders: stenosis and regurgitation. Stenosis occurs when valve leaflets fuse together and cannot fully open or close. The valve opening narrows and becomes rigid (see Figure 30.7A). Scarring of the valves from endocarditis or infarction and calcium deposits can lead to stenosis. Stenotic valves impede the forward flow of blood, decreasing cardiac output because of impaired ventricular filling or ejection, and stroke volume.

Regurgitant valves (also called *insufficient* or *incompetent* valves) do not close completely (see Figure 30.7B). Regurgitation can result from deformity or erosion of valve cusps caused by the vegetative lesions of bacterial endocarditis, by scarring or tearing from myocardial infarction, or by cardiac dilation. As the heart enlarges, the *valve annulus* (supporting ring of the valve) is stretched and the valve edges no longer meet to allow complete closure.



A Thickened and stenotic valve leaflets

B Retracted fibrosed valve openings

FIGURE 30.7 ■ Valvular heart disorders. *A*, Stenosis of a heart valve. *B*, An incompetent or regurgitant heart valve

Valvular disease causes haemodynamic changes both in front of and behind the affected valve. Blood volume and pressures are reduced in front of the valve. By contrast, volumes and pressures characteristically increase behind the diseased valve. These haemodynamic changes may lead to pulmonary complications, cardiac remodelling, hypertrophy or heart failure.

Stenosis increases the work of the chamber behind the affected valve as the heart attempts to move blood through the narrowed opening. Excess blood volume behind regurgitant valves causes dilation of the chamber. In mitral stenosis, for example, the left atrium hypertrophies to generate enough pressure to open and deliver its blood through the narrowed mitral valve. Not all of the blood is delivered before the valve closes, leaving blood to accumulate in the left atrium. This chamber dilates to accommodate the excess volume.

Eventually, cardiac output falls as compensatory mechanisms become less effective. The normal balance of oxygen supply and demand is upset and the heart begins to fail. Increased muscle mass and size increase myocardial oxygen consumption. The size and workload of the heart exceed its blood supply, causing ischaemia and chest pain. Eventually, necrosis occurs and functional muscle is lost. Contractile force, stroke volume and cardiac output decrease. High pressures on the left side of the heart are reflected backward into the pulmonary system, causing pulmonary oedema, pulmonary hypertension and, eventually, right ventricular failure.

Valvular disorders interfere with the smooth flow of blood through the heart. The flow becomes turbulent, causing a **murmur**, a characteristic manifestation of valvular disease. Table 30.9 describes the murmurs associated with various types of valvular disorders.

Blood forced through the narrowed opening of a stenotic valve or regurgitated from a higher pressure chamber through an incompetent valve creates a jet stream effect (much like water spurting out of a partially occluded hose opening). The physical force of this jet stream damages the endocardium of the receiving chamber, increasing the risk of infective endocarditis.

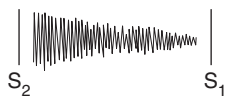
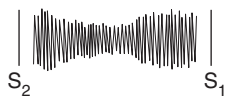
The higher pressures on the left side of the heart subject its valves (the mitral and aortic valves) to more stress and damage than those on the right side of the heart (the tricuspid and pulmonic). Pulmonic valve disease is the least common of the valvular disorders.

Mitral stenosis

Mitral stenosis narrows the mitral valve, obstructing blood flow from the left atrium into the left ventricle during diastole. It is usually caused by rheumatic heart disease or bacterial endocarditis; it rarely results from congenital defects. In Australia, the mean age for mitral stenosis is 33 years. However, in the Northern Territory, 30% of children aged 10–19 years with rheumatic heart disease have mitral stenosis (RHD Australia, 2012). Mitral stenosis is chronic and progressive.

In mitral valve stenosis, fibrous tissue replaces normal valve tissue, causing valve leaflets to stiffen and fuse. Resulting changes in blood flow through the valve lead to calcification of

TABLE 30.9 Heart murmur timing and characteristics

MURMUR	CARDIAC CYCLE TIMING	AUSCULTATION SITE	CONFIGURATION OF SOUND	CONTINUITY
Mitral stenosis	Diastole	Apical		Rumble that increases in sound towards the end, continuous
Mitral regurgitation	Systole	Apex		Holosystolic (occurs throughout systole), continuous
Aortic stenosis	Midsystolic	Right sternal border (RSB) 2nd intercostal space (ICS)		Crescendo–decrescendo, continuous
Aortic regurgitation	Diastole (early)	3rd ICS, left sternal border (LSB)		Decrescendo, continuous
Tricuspid stenosis	Diastole	Lower LSB		Rumble that increases in sound towards the end, continuous
Tricuspid regurgitation	Systole	4th ICS, LSB		Holosystolic, continuous

the valve leaflets. As calcium is deposited in and on the valve, the leaflets become more rigid and narrow the opening further (see Figure 30.8). As the valve leaflets become less mobile, the chordae tendineae fuse, thicken and shorten. Thromboemboli may form on the calcified leaflets.

The narrowed mitral opening impairs blood flow into the left ventricle, reducing end-diastolic volume and pressure, and decreasing stroke volume. The narrowed opening also forces the left atrium to generate higher pressure to deliver blood to the left ventricle. This leads to left atrial hypertrophy. The left atrium also dilates as obstructed blood flow increases its volume. As the resistance to blood flow increases, high atrial pressures are reflected back into the pulmonary vessels, increasing pulmonary pressures (see Figure 30.8). Pulmonary hypertension increases the workload of the right ventricle, causing it to dilate and hypertrophy. Eventually, heart failure occurs.

MANIFESTATIONS Mitral stenosis may be asymptomatic or cause severe impairment. Its manifestations depend on cardiac output and pulmonary vascular pressures. Dyspnoea on exertion is typically the earliest manifestation. Others include cough, haemoptysis, frequent pulmonary infections such as bronchitis and pneumonia, paroxysmal nocturnal dyspnoea, orthopnoea, weakness, fatigue and palpitations. As the stenosis worsens, manifestations of right heart failure, including jugular venous distension, hepatomegaly, ascites and peripheral oedema, develop. Crackles may be heard in the lung bases. In severe mitral stenosis, cyanosis of the face and extremities may be noted. Chest pain is rare but may occur.

On auscultation, a loud S_1, a split S_2 and a mitral opening snap may be heard. The opening snap reflects high left atrial pressure. The murmur of mitral stenosis occurs during diastole and is typically low pitched, rumbling, crescendo–decrescendo. It is heard best with the bell of the stethoscope in the apical region. It may be accompanied by a palpable thrill (vibration).

COMPLICATIONS Atrial arrhythmias, particularly atrial fibrillation, are common due to chronic atrial distension. Thrombi may form and subsequently embolise to the brain, coronary arteries, kidneys, spleen and extremities—potentially devastating complications.

Women with mitral stenosis may be asymptomatic until pregnancy. As the heart tries to compensate for increased circulating volume (30% more in pregnancy) by increasing cardiac output, left atrial pressures rise, tachycardia reduces ventricular filling, and stroke volume and pulmonary pressures increase. Sudden pulmonary oedema and heart failure may threaten the lives of the mother and foetus.

Mitral regurgitation

Mitral regurgitation or *insufficiency* allows blood to flow back into the left atrium during systole because the valve does not close fully. Rheumatic heart disease is a common cause of mitral regurgitation. Processes that dilate the mitral annulus or affect the supporting structures, papillary muscles or the chordae tendineae may cause mitral regurgitation (e.g. left ventricular hypertrophy and MI). Congenital defects also may cause mitral regurgitation.

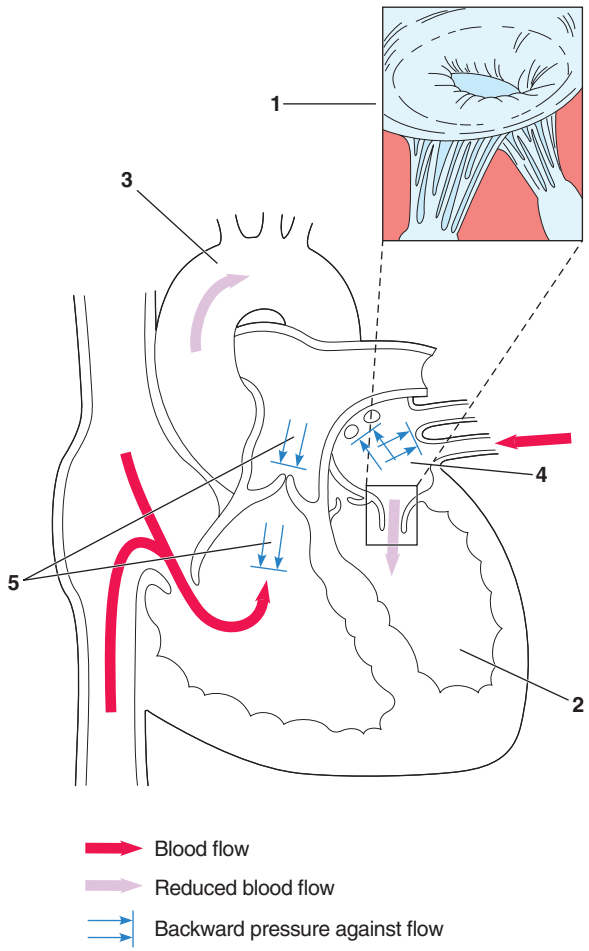


FIGURE 30.8 ■ Mitral stenosis. Narrowing of the mitral valve orifice (1) reduces blood volume to left ventricle (2) reducing cardiac output (3). Rising pressure in the left atrium (4) causes left atrial hypertrophy and pulmonary congestion. Increased pressure in pulmonary vessels (5) causes hypertrophy of the right ventricle and right atrium

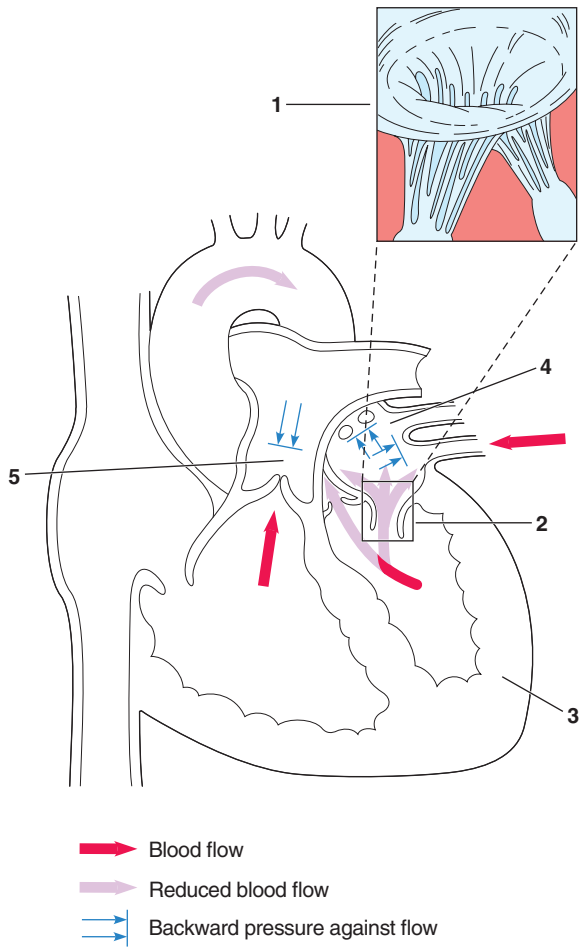


FIGURE 30.9 ■ Mitral regurgitation. The mitral valve closes incompletely (1), allowing blood to regurgitate during systole from the left ventricle to the left atrium (2). Cardiac output falls; to compensate, the left ventricle hypertrophies (3). Rising left atrial pressure (4) causes left atrial hypertrophy and pulmonary congestion. Elevated pulmonary artery pressure (5) causes slight enlargement of the right ventricle

In mitral regurgitation, blood flows into both the systemic circulation and back into the left atrium through the deformed valve during systole. This increases left atrial volume (see Figure 30.9). The left atrium dilates to accommodate its extra volume, pulling the posterior valve leaflet further away from the valve opening and worsening the defect. The left ventricle dilates to accommodate its increased preload and low cardiac output, further aggravating the problem.

MANIFESTATIONS Mitral regurgitation may be asymptomatic or cause symptoms such as fatigue, weakness, exertional dyspnoea and orthopnoea. In severe or acute regurgitation, manifestations of left-sided heart failure develop, including pulmonary congestion and oedema. High pulmonary pressures may lead to manifestations of right-sided heart failure.

The murmur of mitral regurgitation is usually loud, high pitched, rumbling and holosystolic (occurring throughout systole). It is often accompanied by a palpable thrill and is heard most clearly at the cardiac apex.

Mitral valve prolapse

Mitral valve prolapse (MVP) is a type of mitral insufficiency that occurs when one or both mitral valve cusps billow into the atrium during ventricular systole. Its cause often is unclear. However, it can also result from acute or chronic rheumatic damage, ischaemic heart disease or other cardiac disorders. It commonly affects people with inherited connective tissue disorders such as Marfan syndrome (see the 'Genetic considerations' box below).

Excess collagen tissue in the valve leaflets and elongated chordae tendineae impair closure of the mitral valve, allowing the leaflets to billow into the left atrium during systole. Some ventricular blood volume regurgitates into the left atrium (see Figure 30.10).

MANIFESTATIONS AND COMPLICATIONS Mitral valve prolapsed is usually asymptomatic. A midsystolic ejection click or murmur may be audible. A high-pitched late systolic

GENETIC CONSIDERATIONS
The person with Marfan syndrome

Marfan syndrome is a genetic (autosomal dominant) connective tissue disorder that affects the skeleton, eyes and cardiovascular system. Skeletal characteristics include a long, thin body, with long extremities and long, tapering fingers, sometimes called *arachnodactyly* (spider fingers). Joints are hyperextensible, and skeletal deformities such as kyphosis, scoliosis, pigeon chest or pectus excavatum are common (Papadakis et al., 2013). The potentially life-threatening cardiovascular effects of Marfan syndrome include mitral valve prolapse, progressive dilation of the aortic valve ring and weakness of arterial walls. People with Marfan syndrome frequently die young, between 30 and 40 years, often due to dissection and rupture of the aorta (Chen, 2014; Prashanth, 2014).

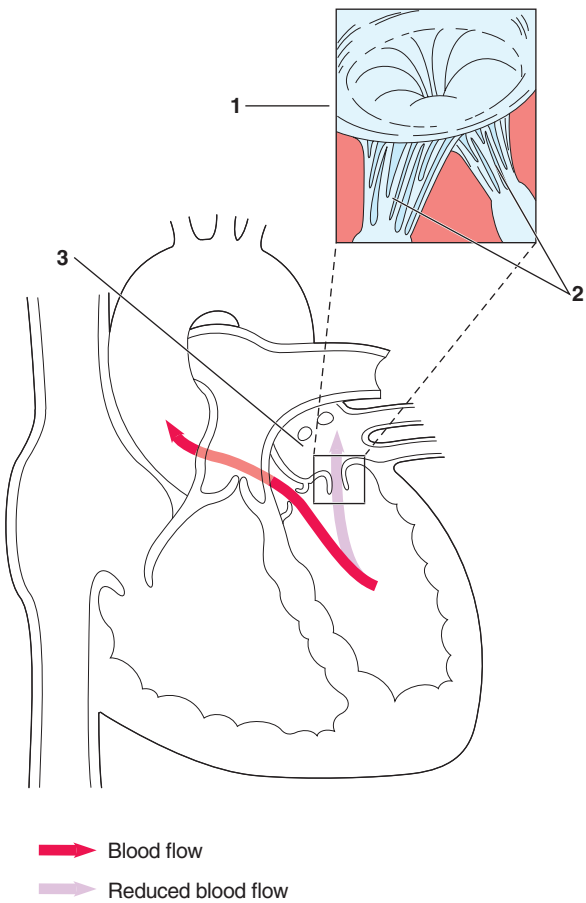


FIGURE 30.10 ■ Mitral valve prolapse. Excess tissue in the valve leaflets (1) and elongated chordae tendineae (2) impair mitral valve closure during systole. Some ventricular blood regurgitates into the left atrium (3)

murmur, sometimes described as a 'whoop' or 'honk', due to the regurgitation of blood through the valve, may develop in MVP. Atypical chest pain is the most common symptom of MVP. It may be left sided or substernal and is frequently related to fatigue, not exertion (Papadakis et al., 2013). Tachyarrhythmias may develop with MVP, causing palpitations, light headedness and syncope. Increased sympathetic nervous system tone may cause a sense of anxiety.

Mitral valve prolapse increases the risk of bacterial endocarditis. Progressive worsening of regurgitation can lead to heart failure. Thrombi may form on prolapsed valve leaflets; embolisation may cause transient ischaemic attacks (TIAs).

Aortic stenosis

Aortic stenosis obstructs blood flow from the left ventricle into the aorta during systole. Aortic stenosis may be idiopathic or due to a congenital defect, rheumatic damage or degenerative changes. When rheumatic heart disease is the cause, mitral valve deformity is also often present. RHD destroys aortic valve leaflets, with fibrosis and calcification causing rigidity and scarring. In the older adult, calcific aortic stenosis may result from degenerative changes associated with ageing. Constant wear and tear on this valve can lead to fibrosis and calcification. Idiopathic calcific stenosis generally is mild and does not impair cardiac output.

As aortic stenosis progresses, the valve annulus decreases in size, increasing the work of the left ventricle to eject its volume through the narrowed opening into the aorta. To compensate, the ventricle hypertrophies to maintain an adequate stroke volume and cardiac output (see Figure 30.11). Left ventricular compliance also decreases. The additional workload increases myocardial oxygen consumption, which can precipitate myocardial ischaemia. Coronary blood flow may also decrease in aortic stenosis. As left ventricular end-diastolic pressure increases because of reduced stroke volume, left atrial pressures increase. These pressures also affect the pulmonary vascular system; pulmonary vascular congestion and pulmonary oedema may result.

COURSE AND MANIFESTATIONS Aortic stenosis may be asymptomatic for many years. As the disease progresses and compensation fails, usually between age 50 and 70 years, obstructed cardiac output causes manifestations of left ventricular failure. Dyspnoea on exertion, angina pectoris and exertional syncope are classic manifestations of aortic stenosis. Pulse pressure, an indicator of stroke volume, narrows to 30 mmHg or less.

Aortic stenosis produces a harsh systolic murmur best heard in the second intercostal space to the right of the sternum. This crescendo–decrescendo murmur is produced by turbulence of blood entering the aorta through the stenotic valve. A palpable thrill is often felt. The murmur may radiate to the carotid arteries. Ventricular hypertrophy displaces the cardiac impulse to the left of the midclavicular line. As aortic stenosis progresses, S_3 and S_4 heart sounds may be heard, indicating heart failure and reduced left ventricular compliance.

As cardiac output falls, tissue perfusion decreases. Late in the disease, pulmonary hypertension and right ventricular failure develop. Untreated, symptomatic aortic stenosis has a poor prognosis as at 3 years after diagnosis only approximately 50% of people are still alive (Papadakis et al., 2013).

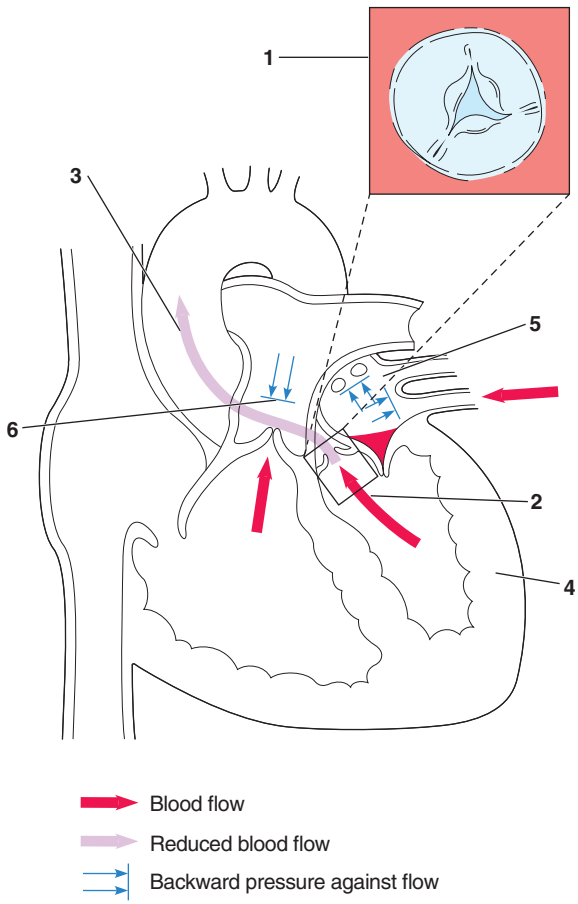


FIGURE 30.11 ■ Aortic stenosis. The narrowed aortic valve orifice (1) decreases the left ventricular ejection fraction during systole (2) and cardiac output (3). The left ventricle hypertrophies (4). Incomplete emptying of the left atrium (5) causes backward pressure through pulmonary veins and pulmonary hypertension. Elevated pulmonary artery pressure (6) causes right ventricular strain

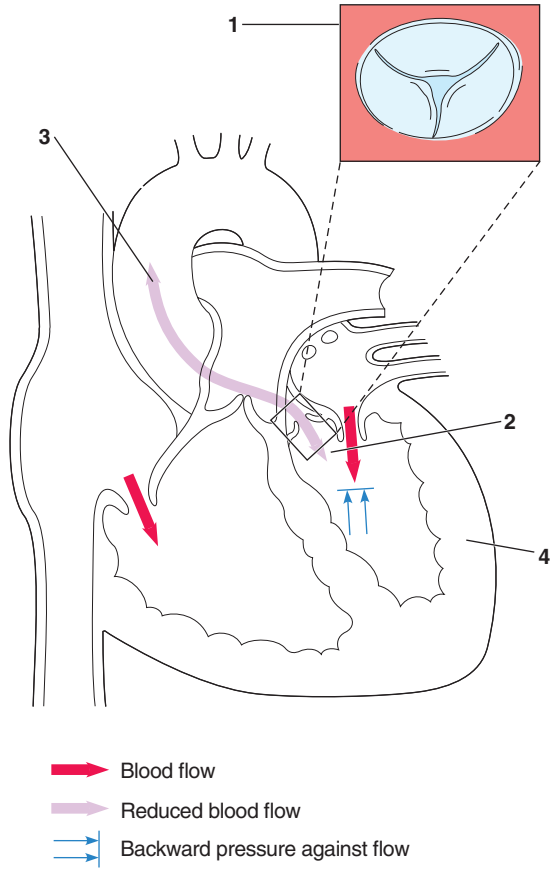


FIGURE 30.12 ■ Aortic regurgitation. The cusps of the aortic valve widen and fail to close during diastole (1). Blood regurgitates from the aorta into the left ventricle (2), increasing left ventricular volume and decreasing cardiac output (3). The left ventricle dilates and hypertrophies (4) in response to the increase in blood volume and workload

Aortic regurgitation

Aortic regurgitation, also called *aortic insufficiency*, allows blood to flow back into the left ventricle from the aorta during diastole. Most aortic regurgitation results from rheumatic heart disease. Other causes include congenital disorders, infective endocarditis, blunt chest trauma, aortic aneurysm, syphilis, Marfan syndrome and chronic hypertension.

In aortic regurgitation, thickened and contracted valve cusps, scarring, fibrosis and calcification impede complete valve closure. Chronic hypertension and aortic aneurysm may dilate and stretch the aortic valve opening, increasing the degree of regurgitation.

In aortic regurgitation, volume overload affects the left ventricle as blood from the aorta adds to blood received from the atrium during diastole. This increases diastolic left ventricular pressure. Increased preload causes more forceful contractions and a high stroke volume (see Figure 30.12). With time, muscle cells hypertrophy to compensate for increased cardiac work and afterload; eventually this hypertrophy compromises cardiac output and increases regurgitation.

High left-ventricular pressures increase left atrial workload and pressure. This pressure is transmitted to the pulmonary vessels, causing pulmonary congestion. The workload of the right ventricle increases as a result and right-sided heart failure may develop. Acute aortic regurgitation from traumatic injury or infective endocarditis causes a rapid decline in haemodynamic status from acute heart failure and pulmonary oedema, because compensatory mechanisms do not have time to develop.

MANIFESTATIONS Aortic regurgitation may be asymptomatic for many years, even when severe. The increased stroke volume may cause complaints of persistent palpitations, especially when recumbent. A throbbing pulse may be visible in arteries of the neck; the force of contraction may cause a characteristic head bob (Musset's sign) and shake the whole body. Other symptoms include dizziness and exercise intolerance.

Fatigue, exertional dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea are common in aortic regurgitation.

Anginal pain may result from excessive cardiac work and decreased coronary perfusion. Unlike CHD, angina often occurs at night and may not respond to conventional therapy.

The murmur of aortic regurgitation is heard during diastole as blood flows back into the left ventricle from the aorta. It is a 'blowing', high-pitched sound heard most clearly at the third left intercostal space. A palpable thrill and ventricular heave may be noted. An S_3 and S_4 may be heard as the heart fails and ventricular compliance diminishes. The apical impulse is displaced to the left.

High systolic and low diastolic pressures cause a widened pulse pressure. The arterial pressure waveform has a rapid upstroke and quickly collapsing downstroke, known as a *water-hammer pulse*. It is caused by the force of rapid and early delivery of the stroke volume into the aorta.

Tricuspid valve disorders

Tricuspid stenosis obstructs blood flow from the right atrium to the right ventricle. It usually results from rheumatic heart disease; mitral stenosis often occurs concurrently with tricuspid stenosis.

Fibrosed, retracted tricuspid valve cusps and fused leaflets narrow the valve orifice and prevent complete closure. Right ventricular filling is impaired during diastole, and during systole some blood regurgitates back into the right atrium. Pressure in the right atrium increases and enlarges in response to the increased pressure and workload. This increased right atrial pressure is reflected backward into the systemic circulation. Right ventricular stroke volume decreases, reducing the volume delivered to the pulmonary system and left heart. Stroke volume, cardiac output and tissue perfusion fall.

Manifestations of tricuspid stenosis relate to systemic congestion and right-sided heart failure. They include increased CVP, jugular venous distension, ascites, hepatomegaly and peripheral oedema. Low cardiac output causes fatigue and weakness. The low-pitched, rumbling diastolic murmur of tricuspid stenosis is most clearly heard in the fourth intercostal space at the left sternal border or over the xiphoid process.

Tricuspid regurgitation usually occurs secondarily to right ventricular dilation. Stretching distorts the valve and its supporting structures, preventing complete valve closure. Left ventricular failure is the usual cause of right ventricular overload; pulmonary hypertension is another cause. The valve may also be damaged by rheumatic heart disease, infective endocarditis, inferior MI, trauma or other conditions.

Tricuspid regurgitation allows blood to flow back into the right atrium during systole, increasing right atrial pressures. Increased right atrial pressure causes manifestations of right-sided heart failure, including systemic venous congestion and low cardiac output. Atrial fibrillation due to atrial distension is common.

Pulmonic valve disorders

Pulmonic stenosis obstructs blood flow from the right ventricle into the pulmonary system. It usually is a congenital disorder, although rheumatic heart disease or cancer also may cause pulmonic stenosis. Both the right atrium and ventricle hypertrophy to overcome high pressures. Right-sided heart failure occurs when the ventricle can no longer generate adequate pressure to force blood past the narrowed valve opening.

Pulmonic stenosis typically is asymptomatic unless severe. Dyspnoea on exertion and fatigue are early signs. As the condition progresses, right-sided heart failure develops, with peripheral oedema, ascites, hepatomegaly and increased venous pressures. Turbulent blood flow caused by the narrowed valve generates a harsh, systolic crescendo–decrescendo murmur heard in the pulmonic area, the second left intercostal space.

Pulmonic regurgitation is more common than pulmonary stenosis. It is a complication of pulmonary hypertension, which stretches and dilates the pulmonary orifice, causing incomplete valve closure. Infective endocarditis, pulmonary artery aneurysm and syphilis also may cause pulmonic regurgitation.

Incomplete valve closure allows blood to flow back into the right ventricle during diastole, decreasing blood flow to the pulmonary circuit increasing end-diastolic pressure. When the ventricle can no longer compensate for the increased volume, right-sided heart failure develops.

INTERPROFESSIONAL CARE

A heart murmur identified during routine physical examination often is the initial indication of valvular disease. If no symptoms are present, close observation for disease progression and prophylactic therapy to prevent infection of the diseased heart may be the only treatment. When medical management is no longer effective, surgery is considered.

Diagnosis

The following diagnostic tests help to identify and diagnose valvular disease. See Chapter 28 for more information about these tests and related nursing care.

- *Echocardiography* is used routinely to diagnose valvular disease. Thickened valve leaflets, vegetations or growths on valve leaflets, myocardial function and chamber size can be determined, and pressure gradients across valves and pulmonary artery pressures can be estimated. Either transthoracic or transoesophageal echocardiography may be used.
- *Chest x-ray* can identify cardiac hypertrophy, chamber and great vessel enlargement, and dilation of the pulmonary vasculature. Calcification of the valve leaflets and annular openings may also be visible.
- *Electrocardiography* can demonstrate atrial and ventricular hypertrophy, conduction defects and arrhythmias associated with valvular disease.
- *Cardiac catheterisation* may be used to assess contractility and to determine the pressure gradients across the heart valves, in the heart chambers and in the pulmonary system.

Medications

Heart failure resulting from valvular disease is treated with diuretics, ACE inhibitors, vasodilators and possibly digoxin. Digoxin increases the force of myocardial contraction to maintain cardiac output. Diuretics, ACE inhibitors and vasodilators reduce preload and afterload. (See the 'Medication administration' box on pages 1053–1054.)

In individuals with valvular disorders, atrial distension often causes atrial fibrillation. Digoxin or small doses of beta-blockers are given to slow the ventricular response. (See Chapter 29 for more information about atrial fibrillation and its treatment.) Anticoagulant therapy is added to prevent clot and embolus formation, a common complication of atrial fibrillation as blood pools in the non-contracting atria. Anticoagulant therapy also is required following insertion of a mechanical heart valve. See Chapter 31 for more information about anticoagulant therapy.

Valvular damage increases the risk of infective endocarditis as altered blood flow allows bacterial colonisation. Antibiotics are prescribed prophylactically prior to any dental work, invasive procedures or surgery to minimise the risk of bacteraemia (bacteria in the blood) and subsequent endocarditis.

Percutaneous balloon valvuloplasty

Percutaneous balloon valvuloplasty is an invasive procedure performed in the cardiac catheterisation laboratory. A balloon catheter similar to that used in coronary angioplasty procedures is inserted into the femoral vein or artery. Guided by fluoroscopy, the catheter is advanced into the heart and positioned with the balloon straddling the stenotic valve. The balloon is then inflated for approximately 90 seconds to divide the fused leaflets and enlarge the valve orifice (see Figure 30.13). Balloon valvuloplasty is the treatment of choice for symptomatic mitral valve stenosis. It is used to treat children and young adults with aortic stenosis. Nursing care of the individual with a balloon valvuloplasty is similar to that of the person having percutaneous coronary revascularisation (see Chapter 29).

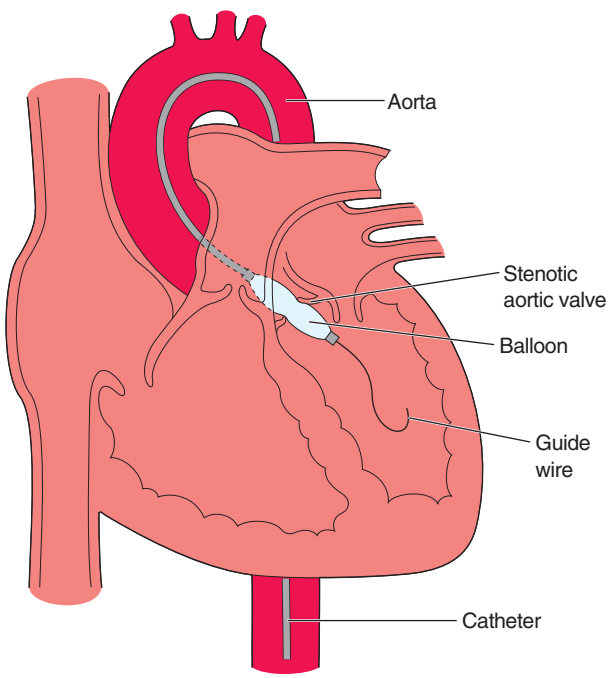


FIGURE 30.13 ■ Balloon valvuloplasty. The balloon catheter is guided into position straddling the stenosed valve. The balloon is then inflated to increase the size of the valve opening

Surgery

Surgery to repair or replace the diseased valve may be done to restore valve function, alleviate symptoms and prevent complications and death. The diseased valve is repaired when possible, because the risk of surgical mortality and complications is lower than with valve replacement.

RECONSTRUCTIVE SURGERY *Valvuloplasty* is a general term for reconstruction or repair of a heart valve. Methods include 'patching' the perforated portion of the leaflet, resecting excess tissue, debriding vegetations or calcification, and other techniques. Valvuloplasty may be used for stenotic or regurgitant mitral and tricuspid valves, mitral valve prolapse and aortic stenosis. Common valvuloplasty procedures include the following:

- *open commissurotomy*, surgical division of fused valve leaflets, is done to open stenotic valves
- *annuloplasty,* which repairs a narrowed or an enlarged or dilated valve annulus, the supporting ring of the valve.

VALVE REPLACEMENT Valve replacement is indicated when manifestations of valve dysfunction develop, preferably before left heart function is seriously impaired. In general, three factors determine the outcome of valve replacement surgery: (1) heart function at the time of surgery, (2) intraoperative and postoperative care, and (3) characteristics and durability of the replacement valve.

Many different prosthetic heart valves are available, including mechanical and biological tissue valves. Selection depends on numerous variables, including the valve haemodynamics, resistance to clot formation, ease of insertion, anatomical suitability and the person's acceptance of the procedure. The individual's age, underlying condition and contraindications to anticoagulation (such as a desire to become pregnant) also are considered in selecting the appropriate prosthesis. Table 30.10 lists the advantages and disadvantages of biological and mechanical valves.

Biological tissue valves may be *heterografts*, excised from a pig (see Figure 30.14A), or made of calf pericardium or *homografts* from a human (obtained from a cadaver or during heart transplant). Biological valves allow more normal blood flow and have a low risk of thrombus formation. As a result, long-term anticoagulation rarely is necessary. They are less durable, however, than mechanical valves.

Mechanical prosthetic valves have the major advantage of long-term durability. These valves are frequently used when life expectancy exceeds 10 years. Their major disadvantage is the need for lifetime anticoagulation to prevent the development of clots on the valve.

Most mechanical valves are either a tilting-disk (see Figure 30.14B) or a ball-and-cage design. Both biological and mechanical valves increase the risk of endocarditis, although its incidence is fairly low.

TABLE 30.10 Advantages and disadvantages of prosthetic heart valves

CATEGORY	TYPES	ADVANTAGES	DISADVANTAGES
Mechanical valves	Ball-and-cage Tilting disk	Long-term durability Good haemodynamics	Lifetime anticoagulation Audible click Risk of thromboembolism Infections are harder to treat
Biological tissue valves	Porcine heterograft Bovine heterograft Human aortic homograft	Low incidence of thromboembolism No long-term anticoagulation Good haemodynamics Quiet Infections are easier to treat	Prone to deterioration Frequent replacement is required

A

B

FIGURE 30.14 ■ Prosthetic heart valves. *A*, Medtronic Mosaic® bioprosthetic valve. *B*, St Jude Medical valve

Sources: A, © BURGER/phanie/Phanie Sarl/Corbis; *B*, Layne Kennedy/Corbis.

Nursing care

Health promotion

Preventing rheumatic heart disease is a key element in preventing heart valve disorders. Rheumatic heart disease is a consequence of rheumatic fever (see the previous section of this chapter), an immune process that may be a sequela to beta-haemolytic streptococcal infection of the pharynx (strep throat). Early treatment of strep throat prevents rheumatic fever. Teach individuals, families and communities about the importance of timely and effective treatment of strep throat. Emphasise the importance of completing the full prescription of antibiotics to prevent development of resistant bacteria. Prophylactic antibiotic therapy before invasive procedures to prevent infectious endocarditis is an important health promotion measure for individuals with pre-existing heart disease.

Assessment

Assessment data related to valvular heart disease include the following:

- *Health history:* complaints of decreasing exercise tolerance, dyspnoea on exertion, palpitations; history of frequent respiratory infections; previous history of rheumatic heart disease, endocarditis or a heart murmur.
- *Physical examination:* vital signs; skin colour and temperature, evidence of clubbing or peripheral oedema; neck vein distension; breath sounds; heart sounds and presence of S_3, S_4 or murmur; timing, grade and characteristics of any murmur; palpate for cardiac heave and thrills; abdominal contour, liver and spleen size.
- *Diagnostic tests:* echocardiogram and cardiac catheterisation reports; cardiac index and cardiac output.

Nursing diagnoses and interventions

Nursing priorities include maintaining cardiac output, managing manifestations of the disorder, teaching about the disease process and its management, and preventing complications.

Nursing care of the person undergoing valve surgery is similar to that of the person having other types of open-heart surgery, with increased attention to anticoagulation and preventing endocarditis.

Decreased cardiac output

Nearly all valve disorders affect ventricular filling and/or emptying, reducing cardiac output. Stenosis of the AV valves impairs ventricular filling and increases atrial pressures. Regurgitation of these valves reduces cardiac output as a portion of the blood in the ventricle regurgitates into the atria during systole. Stenosis of the semilunar valves obstructs ventricular outflow to the great vessels; regurgitation allows blood to flow back into the ventricles, creating higher filling pressures. When compensatory measures fail, heart failure develops.

- Monitor vital signs and haemodynamic parameters, reporting changes from the baseline. *A fall in systolic blood pressure and tachycardia may indicate decreased cardiac output.*

CONSIDERATION FOR PRACTICE

Promptly report changes in level of consciousness; distended neck veins; dyspnoea or respiratory crackles; urine output less than 30 mL/h (or 0.5 mL/h in children); cool, clammy or cyanotic skin; diminished peripheral pulses; or slow capillary refill. These findings indicate decreased cardiac output and impaired tissue and organ perfusion.

- Monitor intake and output; weigh daily. Report weight gain of 1.5 kg within 24 hours. *Fluid retention is a compensatory mechanism that occurs when cardiac output decreases; 1 kg of weight equals 1 L of fluid.*
- Restrict fluids as ordered. *Fluid intake may be restricted to reduce cardiac workload and pressures within the heart and pulmonary circuit.*
- Monitor oxygen saturation continuously and arterial blood gases as ordered. Report oxygen saturation less than 95% (or as specified) and abnormal ABG results. *Oxygen saturation levels and ABGs allow assessment of oxygenation.*
- Elevate the head of the bed. Administer supplemental oxygen as ordered. *These measures improve alveolar ventilation and oxygenation.*
- Provide for physical and emotional rest. *Physical and psychological rest decrease the cardiac workload.*
- Administer prescribed medications as ordered to reduce cardiac workload. *Diuretics, ACE inhibitors and direct vasodilators may be prescribed to reduce fluid volume and afterload, reducing cardiac work.*

Activity intolerance

Altered blood flow through the heart impairs delivery of oxygen and nutrients to the tissues. As the heart muscle fails and is unable to compensate for altered blood flow, tissue perfusion is further compromised. Dyspnoea on exertion is often an early symptom of valvular disease.

- Monitor vital signs before and during activities. *A change in heart rate of more than 20 bpm, a change of 20 mmHg or more in systolic BP and complaints of dyspnoea, excessive fatigue, chest pain, diaphoresis, dizziness or syncope may indicate activity intolerance.*
- Encourage self-care and gradually increasing activities as allowed and tolerated. Provide for rest periods, uninterrupted sleep and adequate nutritional intake. *Gradual progression of activities avoids excessive cardiac stress. Encouraging self-care increases the person's self-esteem and sense of power. Adequate rest and nutrition facilitate healing, decrease fatigue and increase energy reserves.*
- Provide assistance as needed. Suggest the person uses a shower chair, sitting while brushing hair or teeth, and other energy-saving measures. *Reducing energy expenditure helps maintain a balance of oxygen supply and demand.*
- Consult with cardiac rehabilitation nurse or physiotherapist for in-bed exercises and an activity plan. *In-bed exercises may help improve strength.*
- Discuss ways to conserve energy at home. *Information provides practical ways to deal with activity limitations and empowers the person to manage these limitations.*

Risk of infection

Damaged and deformed valve leaflets and turbulent blood flow through the heart significantly increase the risk of infective endocarditis. Invasive diagnostic and monitoring lines (e.g. cardiac catheterisation, haemodynamic monitoring) and disrupted skin with surgery also increase the risk of infection.

- Use aseptic technique for all invasive procedures. *Invasive procedures breach the body's protective mechanisms, potentially allowing bacteria to enter. Aseptic technique reduces this risk.*
- Assess wounds and catheter sites for redness, swelling, warmth, pain or evidence of drainage. *These signs of inflammation may signal infection.*
- Administer antibiotics as ordered. Ensure completion of the full course. *Antibiotics are used to prevent and treat infection. Completion of the full course of therapy prevents drug-resistant organisms from multiplying.*
- Monitor FBC and differential. Notify doctor of leucocytosis or leucopenia. *A high WBC count and increased percentage of immature WBCs (bands) may indicate bacterial infection; a low WBC count may indicate an impaired immune response and increased susceptibility to infection.*

Ineffective protection

Anticoagulant therapy commonly is prescribed for people with chronic atrial fibrillation, a history of emboli and following valve replacement surgery. Although chronic anticoagulant therapy decreases the risk of clots and emboli, it increases the risk of bleeding and haemorrhage.

- Test stools and vomitus for occult blood. *Bleeding due to excessive anticoagulation may not be apparent.*
- Instruct to avoid using aspirin or other NSAIDs. Encourage the person to read ingredient labels on over-the-counter drugs; many contain aspirin. *Aspirin and other NSAIDs*

interfere with clotting and may potentiate the effects of the anticoagulant therapy.
- Advise using a soft-bristled toothbrush, electric razor and gentle touch when cleaning fragile skin. *These measures decrease the risk of skin or gum trauma and bleeding.*

CONSIDERATION FOR PRACTICE

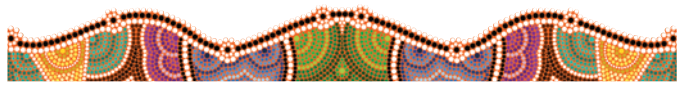
Monitor the International Normalized Ratio (INR) or prothrombin time (PT). For anticoagulation in people:
- with mechanical prosthetic heart valves: INR 2.5–3.5
- all other indications such as AF, DVT, PE: INR 2.0–3.0

Report an INR > 3.5 or a PT > 2.5 times the normal to the doctor. An excessively high INR or PT indicates excessive anticoagulation and an increased risk of bleeding.

Community-based care

For most individuals, valvular disease is a chronic condition. The person has the primary responsibility for managing effects of the disorder. To prepare the individual and their family for home care, discuss the following topics:
- management of symptoms, including any necessary activity restrictions or lifestyle changes
- the importance of adequate rest to prevent fatigue
- diet restrictions to reduce fluid retention and symptoms of heart failure
- information about prescribed medications, including purpose, desired and possible adverse effects, scheduling, and possible interactions with other drugs
- the importance of keeping follow-up appointments to monitor the disease and its treatment
- notifying all healthcare providers about valve disease or surgery to facilitate prescription of prophylactic antibiotics before invasive procedures or dental work
- reporting increasing severity of symptoms, especially of worsening heart failure or pulmonary oedema; signs of transient ischaemic attacks or other embolic events; evidence of bleeding, such as joint pain, easy bruising, black and tarry stools, bleeding gums or blood in the urine or sputum.

Provide referrals to community resources such as cardiac rehabilitation programs and community support services. Refer the person and their family (especially the primary food preparer) to a dietician or nutritionist for teaching and assistance with menu planning. See the accompanying nursing care plan for additional nursing care and teaching for an individual with mitral valve prolapse.



THE PERSON WITH CARDIOMYOPATHY

The **cardiomyopathies** are disorders that affect the heart muscle itself. They are a diverse group of disorders that affect both systolic and diastolic functions. Cardiomyopathies may be either primary or secondary in origin. Primary cardiomyopathies are idiopathic; their cause is unknown. Secondary cardiomyopathies occur as a result of other processes, such as ischaemia, infectious disease, exposure to toxins, connective tissue disorders, metabolic disorders or nutritional deficiencies. In many cases, the cause of cardiomyopathy is unknown. In 2013 in Australia, cardiomyopathy was listed as the cause of death in 993 people, which equates to 0.67% of all deaths (ABS, 2015).

Pathophysiology

The cardiomyopathies are categorised by their pathophysiology and presentation into three groups: dilated, hypertrophic

NURSING CARE PLAN A person with mitral valve prolapse

Julie Snow, a 22-year-old university student, sees the local medical officer for a physical examination after experiencing palpitations, fatigue and a headache during midterm examinations. Ms Snow stated, 'I'm scared that something is wrong with me.'

Over the past few months, Ms Snow has had occasional palpitations that she describes as 'feeling like my heart is doing flip-flops'. Rarely, these palpitations have been accompanied by a sharp, stabbing pain in her chest that lasts only a few seconds. She initially attributed her symptoms to stress, but she is increasingly concerned because the 'attacks' are becoming more frequent. Ms Snow states that she has 'always been healthy', does not smoke, uses alcohol socially and exercises, albeit intermittently. Ms Snow admits that she has been drinking a lot of coffee and cola and eating a lot of 'junk food' lately.

ASSESSMENT

Ms Snow's assessment revealed the following: height 168 cm, weight 63.6 kg, T 37°C, BP 118/64, P 82 and R 18. She was slightly anxious but in no acute distress. Systolic click and soft crescendo murmur grade II/VI was noted on auscultation. Apical impulse at fifth intercostal space left midclavicular line. Her lungs are clear to auscultation. A review of the remaining systems reveals no apparent abnormalities. An ECG shows sinus rhythm with occasional premature atrial contractions (PACs). Based on the admission history, manifestations and physical assessment, a mitral valve prolapse (MVP) is suspected.

DIAGNOSES

- *Anxiety* related to fear of heart disease and implications for lifestyle manifested by facial expressions and statements consistent with anxiety.
- *Feelings of powerlessness* related to unpredictability of symptoms manifested by anxiety.
- *Risk of infection* (endocarditis) related to altered valve function manifested by chest pain, cardiac dysfunction and fever.

NURSING CARE PLAN A person with mitral valve prolapse (continued)

PLANNING

- Consult with and refer the person to a cardiologist for continued monitoring and follow up.
- Discuss symptoms of progressive mitral regurgitation and the need to report these to the cardiologist.
- Discuss recommended follow-up care and its rationale.
- Discuss ways to decrease or relieve MVP symptoms.
- Acknowledge the risk of endocarditis and identify precautions to prevent it.
- Discuss the prognosis for MVP, emphasising that most people live normal lives using diet and lifestyle management.
- Discuss lifestyle changes to manage symptoms: aerobic exercise with warm-up and cool-down periods; maintaining adequate fluid intake, especially during hot weather or exercise; relaxation techniques (e.g. meditation, deep-breathing exercises, music therapy, yoga, guided imagery, heat therapy or progressive muscle relaxation) to perform daily; avoiding caffeine and crash diets; forming healthy eating habits.

Expected outcomes

- Verbalises an understanding of MVP and its management.
- Is able to identify signs of deterioration of the condition and the methods by which they will report it.
- Attends follow-up appointments.
- Verbalises methods to prevent endocarditis.
- Demonstrates hopes for future health and management.
- Undertakes lifestyle changes that result in weight loss, increased circulation and improved cardiovascular and muskuloskeletal condition.

IMPLEMENTATION

- Teach the person about MVP, including heart valve anatomy, physiology and function, common manifestations of MVP and treatment rationale.
- Provide opportunity for the person to verbalise feelings and share concerns about MVP. Encourage to attend an MVP support group meeting.
- Instruct the person to keep a weekly record of symptoms and their frequency for 1 month.
- Teach the person about infective endocarditis risk and discuss the protocol for prevention with prophylactic antibiotics with the doctor. Encourage notifying dentist and other healthcare providers of MVP before dental or any invasive procedure.

EVALUATION

After several educational sessions Ms Snow verbalises an understanding of MVP by explaining heart valve function, listing common manifestations of MVP and describing indications of deteriorating heart function. She states she will report these manifestations to her cardiologist if they occur. She is given a booklet on MVP for additional reading. She also verbalises understanding of the risk of endocarditis and states that she will notify her doctors of her MVP and the need for antibiotics before invasive procedures. Ms Snow is attending a monthly MVP support group (led by a cardiac rehabilitation nurse) and states, 'I am so glad to know I'm not alone! It really helps to know that others are living well with MVP.' Her weekly symptom log shows her symptoms are associated with late-night studying and drinking large amounts of coffee and cola. Ms Snow has moderated her caffeine intake and increased her fluids, relieving her symptoms. Ms Snow states that she realises that she has 'the ability to control my life through the choices I make'.

CRITICAL THINKING IN THE NURSING PROCESS

1. Develop an action plan for Ms Snow that outlines specific activities she can use to manage symptoms of MVP.
2. Why are people with symptomatic MVP encouraged to include regular exercise in their health habits?
3. How does the support of family, friends and other people with MVP assist individuals in managing their condition?
4. What manifestations would indicate a progressive worsening of Ms Snow's mitral regurgitation?

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. What techniques would assist you to facilitate communication about lifestyle change principles in an individual with mitral valve prolapse?

and restrictive. Table 30.11 compares the causes, pathophysiology, manifestations and management of the cardiomyopathies.

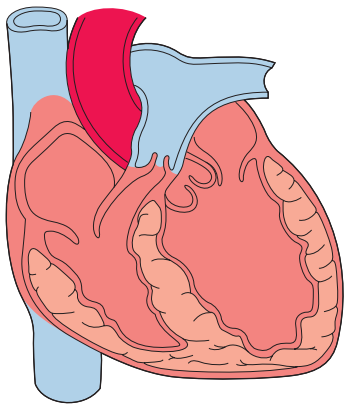
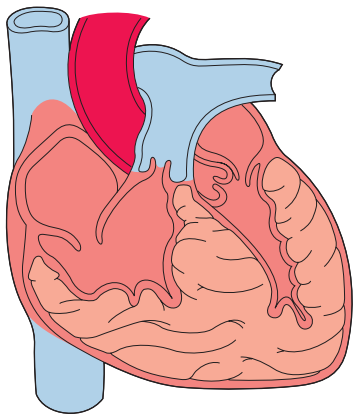
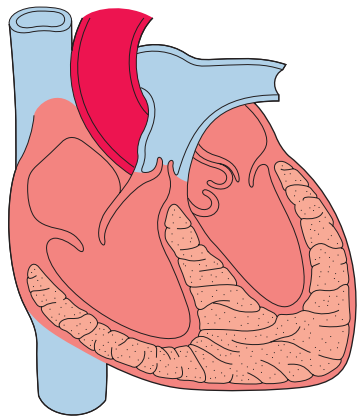
Dilated cardiomyopathy

Dilated cardiomyopathy is the most common type of cardiomyopathy.

The cause of dilated cardiomyopathy is unknown, although it appears to frequently result from toxins, metabolic conditions or infection. Reversible dilated cardiomyopathy may develop due to alcohol and cocaine abuse, chemotherapeutic drugs, pregnancy and systemic hypertension. In dilated cardiomyopathy, heart chambers dilate and ventricular contraction is impaired. Both end-diastolic and end-systolic volumes increase and the left ventricular ejection fraction is substantially reduced, decreasing cardiac output. Left ventricular dilation is prominent; left ventricular hypertrophy is usually minimal. The right ventricle may also be enlarged and extensive interstitial fibrosis (scarring) may be evident (Goswami, 2014).

MANIFESTATIONS AND COURSE Manifestations of dilated cardiomyopathy develop gradually. Heart failure often presents years after the onset of dilation and pump failure. Both right- and left-sided failure occur, with dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, weakness, fatigue, peripheral oedema and ascites. Both S_3 and S_4 heart sounds are commonly heard, as well as an AV regurgitation murmur.

TABLE 30.11 Classifications of cardiomyopathy

	DILATED	HYPERTROPHIC	RESTRICTIVE
			
Causes	Usually idiopathic; may be secondary to chronic alcoholism or myocarditis	Hereditary; may be secondary to chronic hypertension	Usually secondary to amyloidosis, radiation or myocardial fibrosis
Pathophysiology	Scarring and atrophy of myocardial cells Thickening of ventricular wall Dilation of heart chambers Impaired ventricular pumping Increased end-diastolic and end-systolic volumes Mural thrombi are common	Hypertrophy of ventricular muscle mass Small left ventricular volume Septal hypertrophy may obstruct left ventricular outflow Left atrial dilation	Excess rigidity of ventricular walls restricts filling Myocardial contractility remains relatively normal
Manifestations	Heart failure Cardiomegaly Arrhythmias S_3 and S_4 gallop; murmur of mitral regurgitation	Dyspnoea, anginal pain, syncope Left ventricular hypertrophy Arrhythmias Loud S_4 Sudden death	Dyspnoea, fatigue Right-sided heart failure Mild to moderate cardiomegaly S_3 and S_4 Mitral regurgitation murmur
Management	Management of heart failure Implantable cardioverter-defibrillator (ICD) as needed Cardiac transplantation	Beta-blockers Antiarrhythmic agents Calcium channel blockers ICD, dual-chamber pacing Surgical excision of part of the ventricular septum	Management of heart failure Exercise restriction

Arrhythmias are common, including supraventricular tachycardias, atrial fibrillation and complex ventricular tachycardias. Untreated arrhythmias and thromboembolism can lead to sudden death (Goswami, 2014).

The prognosis of dilated cardiomyopathy is grim; most individuals get progressively worse.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy is characterised by decreased compliance of the left ventricle and hypertrophy of the ventricular muscle mass. This impairs ventricular filling, leading to small end-diastolic volumes and low cardiac output. Several people with hypertrophic cardiomyopathy have a family history of the disease. It is genetically transmitted in an autosomal dominant pattern (Shah, 2014).

The pattern of left ventricular hypertrophy is unique in that the muscle may not hypertrophy 'equally'. In a majority of people, the interventricular septal mass, especially the upper portion, increases to a greater extent than the free wall of the ventricle. The enlarged upper septum narrows the passageway of blood into the aorta, impairing ventricular outflow. For this reason, this disorder is also known as *idiopathic hypertrophic subaortic stenosis (IHSS)* or *hypertrophic obstructive cardiomyopathy (HOCM)*.

MANIFESTATIONS AND COURSE Hypertrophic cardiomyopathy may be asymptomatic for many years. Symptoms typically occur when increased oxygen demand causes increased ventricular contractility. They may develop suddenly during or after physical activity; in children and young adults, sudden cardiac death may be the first sign of the disorder. Hypertrophic cardiomyopathy is the probable cause of death in young athletes who die suddenly.

The usual manifestations of hypertrophic cardiomyopathy are dyspnoea, angina and syncope. Angina may result

from ischaemia due to overgrowth of the ventricular muscle, coronary artery abnormalities or decreased coronary artery perfusion. Syncope may occur when the outflow tract obstruction severely decreases cardiac output and blood flow to the brain. Ventricular arrhythmias are common; atrial fibrillation also may develop. Other manifestations of hypertrophic cardiomyopathy include fatigue, dizziness and palpitations. A harsh, crescendo–decrescendo systolic murmur of variable intensity heard best at the lower left sternal border and apex is characteristic in hypertrophic cardiomyopathy. An S_4 may also be noted on auscultation.

Restrictive cardiomyopathy

The least common form of cardiomyopathy, *restrictive cardiomyopathy*, is characterised by rigid ventricular walls that impair diastolic filling. Causes of restrictive cardiomyopathy include myocardial fibrosis and infiltrative processes, such as amyloidosis. Fibrosis of the myocardium and endocardium causes excessive stiffness and rigidity of the ventricles. Decreased ventricular compliance impairs filling, with decreased ventricular size, elevated end-diastolic pressures and decreased cardiac output. Contractility is unaffected and the ejection fraction is normal.

MANIFESTATIONS AND COURSE The manifestations of restrictive cardiomyopathy are those of heart failure and decreased tissue perfusion. Dyspnoea on exertion and exercise intolerance are common. Jugular venous pressure is elevated and S_3 and S_4 are common. The prognosis for restrictive cardiomyopathy is poor. Many people die within 3 years as the systemic nature of the underlying disease process precludes effective treatment.

INTERPROFESSIONAL CARE

With the exception of treating an underlying cause, little can be done to treat either dilated or restrictive cardiomyopathies. For these disorders, treatment focuses on managing heart failure and treating arrhythmias. Refer to the section of this chapter on heart failure and Chapter 29 for specific treatment strategies. Treatment of hypertrophic cardiomyopathy focuses on reducing contractility and preventing sudden cardiac death. Strenuous physical exertion is restricted, because it may precipitate arrhythmias or sudden cardiac death. Dietary and sodium restrictions may help diminish the manifestations.

Diagnosis

Diagnosis begins with a history and physical assessment to rule out known causes of heart failure. Other tests may include the following:

- *Echocardiography* is done to assess chamber size and thickness, ventricular wall motion, valvular function and systolic and diastolic function of the heart.
- *Electrocardiography* and *ambulatory ECG monitoring* demonstrate cardiac enlargement and detect arrhythmias.
- *Chest x-ray* shows cardiomegaly, enlargement of the heart and any pulmonary congestion or oedema.
- *Haemodynamic studies* are used to assess cardiac output and pressures in the cardiac chambers and pulmonary vascular system.
- *Radionuclear scans* help identify changes in ventricular volume and mass, as well as perfusion deficits.
- *Cardiac catheterisation* and *coronary angiography* may be done to evaluate coronary perfusion, the cardiac chambers, valves and great vessels for function and structure, pressure relationships and cardiac output.
- *Myocardial biopsy* uses the transvenous route to obtain myocardial tissue for biopsy. The cells are examined for infiltration, fibrosis or inflammation.

Medications

The drug regimen used to treat heart failure also is used for dilated or restrictive cardiomyopathy. This includes ACE inhibitors, vasodilators and digoxin (see the earlier section of this chapter on medications for heart failure). Beta-blockers also may be used with caution in individuals with dilated cardiomyopathy. Anticoagulants are given to reduce the risk of thrombus formation and embolisation. Antiarrhythmic drugs are avoided if possible due to their tendency to precipitate further arrhythmias (Dumitru, 2015; Papadakis et al., 2013).

Beta-blockers are the drugs of choice to reduce anginal symptoms episodes associated with hypertrophic cardiomyopathy. The negative inotropic effects of beta-blockers and calcium channel blockers decrease the myocardial contractility, decreasing obstruction of the outflow tract. Beta-blockers also decrease heart rate and increase ventricular compliance, increasing diastolic filling time and cardiac output. Vasodilators, digoxin, nitrates and diuretics are contraindicated. Amiodarone may be used to treat ventricular arrhythmias (Dumitru, 2015; Papadakis et al., 2013).

Surgery

Without definitive treatment, individuals with cardiomyopathy develop end-stage heart failure. Cardiac transplant is the definitive treatment for dilated cardiomyopathy. Ventricular assist devices may be used to support cardiac output until a donor heart is available. Transplantation is not a viable option for restrictive cardiomyopathy because transplantation does not eliminate the underlying process causing infiltration or fibrosis and eventually the transplanted organ is affected as well. See the section on heart failure for more information about cardiac transplantation.

An implantable cardioverter-defibrillator (ICD) often is inserted to treat potentially lethal arrhythmias. A dual-chamber pacemaker also may be used to treat hypertrophic cardiomyopathy.

Nursing care

Nursing assessment and care for individuals with dilated and restrictive cardiomyopathy are similar to those for individuals with heart failure. Teaching about the disease process and its management is vital. Some degree of activity restriction is often

TRANSLATION TO PRACTICE

Outcomes and opportunities: a nurse-led model of chronic disease management in Australian general practice

A NURSE-LED MODEL OF CHRONIC DISEASE MANAGEMENT

In a study by Eley et al. (2012), mixed-method research trialled the feasibility of a new nurse-led model of disease management in people with chronic cardiovascular disease. Individuals were randomised to either a practice-nurse-managed group or general-practitioner-managed group. The results indicated no statistical difference between self-reported quality of life and perceptions of the models' acceptability over a 12-month period. At the conclusion of the study, all GPs elected to continue the practice-nurse-led model as they saw significant advantages.

IMPLICATIONS FOR NURSING

A practice nurse may be suitable to lead the routine community management of people with stable chronic cardiovascular disease. An appropriately designed and supported nurse-led model would provide appropriate and feasible primary health opportunity to individuals with chronic conditions. Other benefits of such a model could include an increase in nurses' scope of practice and also an economic benefit for the already overburdened health-care system.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. In this study, the findings indicate that a practice nurse is capable of providing to people with cardiovascular conditions selected services that are sufficiently similar to those of GPs. Given your knowledge of what is required to assist an individual to maintain their cardiovascular health in the context of heart failure, what components would a nurse-led primary health model contain? Consider aspects of education, intervention and monitoring. Make a table outlining these three components with statements to justify each component identified.
2. How will the findings of this study impact on the scope of practice of a practice nurse? What further education requirements would be needed to adequately and safely support a practice-nurse-led model?

Source: D. Eley, E. Patterson, J. Young et al. (2012). Outcomes and opportunities: A nurse-led model of chronic disease management in Australian general practice. *Australian Journal of Primary Health*, 8 May (online).

necessary; assist to conserve energy while encouraging self-care. Support coping skills and adaptation to required lifestyle changes. Provide information and support for decision making about cardiac transplantation if that is an option. Discuss the toxic and vasodilator effects of alcohol and encourage abstinence. For nursing diagnoses and suggested interventions see the nursing care section for heart failure.

The person with hypertrophic cardiomyopathy requires care similar to that provided for myocardial ischaemia; nitrates and other vasodilators, however, are avoided. If surgery is performed, nursing care is similar to that for any person undergoing open-heart surgery. Discuss the genetic transmission of hypertrophic cardiomyopathy and suggest screening of close relatives (parents and siblings).

Provide pre- and postoperative care and teaching as appropriate for individuals undergoing invasive procedures or surgery for cardiomyopathy.

Nursing diagnoses that may be appropriate for people with cardiomyopathy include:

- *Risk of decreased cardiac output* related to impaired left ventricular filling, contractility or outflow obstruction.
- *Risk of fatigue* related to decreased cardiac output.
- *Risk of ineffective breathing pattern* related to heart failure.
- *Risk of fear* related to risk of sudden cardiac death.
- *Risk of ineffective role performance* related to decreasing cardiac function and activity restrictions.
- *Risk of anticipatory grieving* related to poor prognosis.

Community-based care

Cardiomyopathies are chronic, progressive disorders generally managed in home and community care settings unless surgery or transplant is planned or end-stage heart failure develops. When teaching the person and family for home care, include the following topics:

- activity restrictions and dietary changes to reduce manifestations and prevent complications
- prescribed drug regimen, its rationale, intended and possible adverse effects
- the disease process, its expected ultimate outcome and treatment options
- cardiac transplantation, including the procedure, the need for lifetime immunosuppression to prevent transplant rejection and the risks of postoperative infection and long-term immunosuppression
- symptoms to report to the doctor or for which immediate care is needed
- cardiopulmonary resuscitation procedures and available training sites.

Refer the person and their family for home and social services and counselling as indicated. Provide community resources such as support groups or the National Heart Foundation.

BOX 30.4 Comparison of Australian hospital separations for selected cardiovascular conditions, 2012–2013 by gender

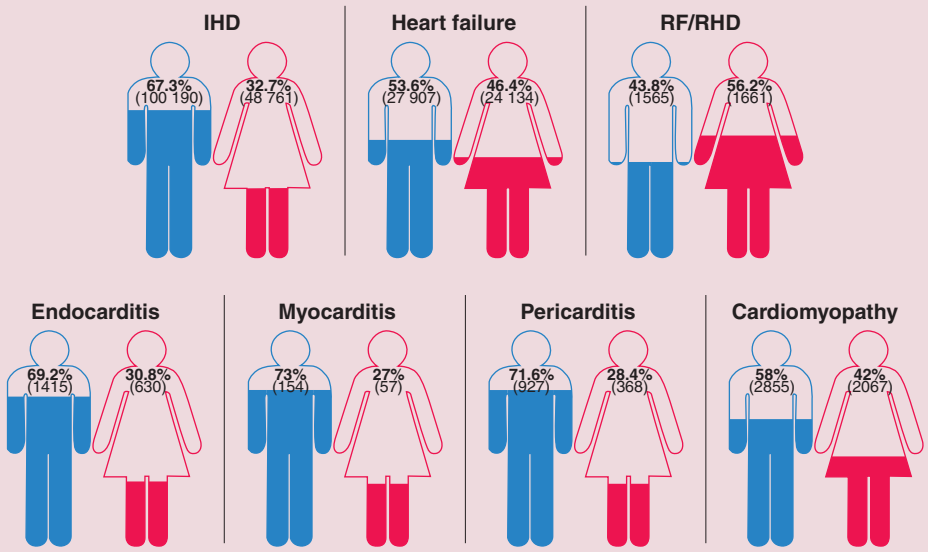


FIGURE 30.15 ■ Top value—percentage admitted with selected condition comparing gender; bracketed value—actual number admitted in 2012–2013 by gender.

IHD = ischaemia heart disease; RF = rheumatic fever; RHD = rheumatic heart disease.

Source: Generated using data from Australian Institute of Health and Welfare (AIHW) (2015). *Separation statistics by principal diagnosis in ICD-10-AM, Australia, 2011–12 to 2012–13.* Retrieved from www.aihw.gov.au/hospitals-data/principal-diagnosis-data-cubes/.

CHAPTER HIGHLIGHTS

- All of the disorders discussed in this chapter can lead to heart failure, a condition in which the heart is unable to pump effectively to meet the body's needs for blood and oxygen to the tissues.
- Coronary heart disease (myocardial ischaemia) and cardiomyopathies are the leading causes of heart failure.
- When the heart starts to fail, compensatory mechanisms are activated to help maintain tissue perfusion. While these mechanisms, including increased contractile force, vasoconstriction, sodium and water retention and remodelling of the heart, effectively maintain cardiac output in the short term, in the long term they hasten deterioration of heart function.
- The goals of heart failure management are to reduce the workload of the heart and improve its function. Medical management includes administration of drugs such as ACE inhibitors, beta-blockers, diuretics and vasodilators to reduce the workload of the heart, and inotropic medications such as digoxin to improve the strength of cardiac muscle contraction.
- Nursing care of the person with heart failure is primarily supportive and educative, providing the individual and their family with necessary knowledge and resources to manage this chronic condition.
- Cardiogenic pulmonary oedema, a manifestation of severe cardiac decompensation, is a medical emergency, requiring immediate and effective treatment to preserve life. The nurse's role in managing pulmonary oedema focuses on supporting respiratory and cardiac function, administering prescribed medications and providing reassurance to the person and their family.
- Inflammatory and infectious processes, such as rheumatic fever, endocarditis, myocarditis and pericarditis, can affect any layer of the heart. While some processes, such as myocarditis and pericarditis, typically are mild and self-limiting, others can have long-term effects on cardiac structure and function.
- Processes such as rheumatic heart disease, endocarditis and congenital conditions can affect the structure and function of the heart valves, resulting in either stenosis (narrowing) of the valve and restricted flow through it, or regurgitation (backflow of blood through a valve that does not fully close). The mitral and aortic valves are commonly affected due to the higher pressures and increased workload of the left side of the heart.
- Valve disorders may be mild, producing a heart murmur but no functional impairment for the individual, or severe, causing symptoms of heart failure even at rest. Repair or replacement of the valve may ultimately be required.
- Cardiomyopathies affect the heart muscle and its ability to stretch during filling and to contract effectively. Dilated cardiomyopathy, the most common type, is progressive,

ultimately necessitating heart transplant. Hypertrophic cardiomyopathy affects both ventricular filling and outflow through the aortic valve. Surgical resection of excess tissue may relieve its manifestations.

CONCEPT CHECK

1 In reviewing the doctor's admitting notes for a person with heart failure, the nurse notes that the person has an ejection fraction of 25%. The nurse recognises this as meaning:
   1 ventricular function is severely impaired
   2 the amount of blood being ejected from the ventricles is within normal limits
   3 25% of the blood entering the ventricle remains in the ventricle after systole
   4 cardiac output is greater than normal, overtaxing the heart

2 In assessing a person admitted 24 hours previously with heart failure, the nurse notes that the person has lost 1 kg of weight, his heart rate is 88 (HR was 105 on admission) and he now has crackles in the bases of his lung fields only. The nurse correctly interprets these data as indicating:
   1 the person's condition is unchanged from admission
   2 a need for more aggressive treatment
   3 the treatment regimen is achieving the desired effect
   4 no further treatment is required at this time as the failure has resolved

3 The nurse assessing a person admitted with left ventricular failure would recognise which of the following findings as consistent with the diagnosis? (Select all that apply.)
   1 5 cm jugular vein distension at 30 degrees
   2 complaints of shortness of breath with minimal exertion
   3 substernal chest pain during exercise
   4 bilateral inspiratory crackles to midscapular
   5 fatigue

4 Morphine 2–5 mg IV as needed for pain and dyspnoea is ordered for a person in acute pulmonary oedema. The nurse appropriately:
   1 questions this order because no time intervals have been specified
   2 administers the drug as ordered, monitoring respiratory status
   3 withholds the drug until the person's respiratory status improves
   4 administers the drug only when the person complains of chest pain

5 An expected assessment finding in a person with mitral stenosis being admitted for a valve replacement would be:
   1 muffled heart sounds
   2 S_3 and S_4 heart sounds
   3 diastolic murmur heard at the apex
   4 cardiac heave

6 A person facing heart valve replacement asks the nurse which type of valve is the best, biological or mechanical. Which of the following would be an appropriate response?
   1 The need to take drugs to prevent rejection of biological tissue is a major consideration.
   2 Clotting is a risk with mechanical valves, necessitating anticoagulant drug therapy after insertion.
   3 Biological valves tend to be more durable than mechanical valves.
   4 Endocarditis is a risk following valve replacement; it is more easily treated with mechanical valves.

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CHAPTER 31

NURSING CARE OF PEOPLE WITH VASCULAR AND LYMPHATIC DISORDERS

MAJELLA HALES

KEY TERMS

LEARNING OUTCOMES

- Discuss the course, implications and health management options for a person with a hypertensive condition.
- Differentiate between various types, implications and management options for an individual with an aneurysm.
- Relate the implications and interventions for a person with peripheral vascular disease to its pathophysiology.
- Compare and contrast the types of arterial occlusive conditions a person may develop.
- Discuss the risks, implications and health management options for a person who has developed a venous thrombosis.
- Differentiate between implications and management options for a person with venous insufficiency or varicose veins.
- Describe the aetiology, pathophysiology and manifestations of lymphadenopathy and lymphoedema.

CLINICAL COMPETENCIES

- Perform a comprehensive vascular assessment on people with vascular disorders, using data to select and prioritise appropriate nursing diagnoses, implementation of care and identify desired outcomes of care.
- Identify the effects on the functional health status of people with vascular disorders.
- Use research-informed practice and evidence-based plans to provide individualised care for people with vascular disorders.
- Collaborate with the interprofessional care team in planning and providing care for people with vascular disorders.
- Safely and knowledgeably administer medications and prescribed treatments for people with vascular disorders.
- Provide person and family teaching to promote, maintain and restore health in people with common vascular disorders.

The main processes that interfere with peripheral blood flow and that of lymphatic fluid include constriction, obstruction, inflammation and vasospasm. These conditions lead to disorders of blood pressure regulation, peripheral artery function, aortic structure, venous circulation and lymphatic circulation.

A holistic approach is important when caring for people with disorders of the vascular and lymphatic systems. The focus of care is on teaching long-term care measures, pain relief, improving peripheral blood and lymphatic circulation, preventing tissue damage and promoting healing. The prescribed treatment may have emotional, social and economic effects on the person and family.

Prevalence of cardiovascular conditions such as angina, myocardial infarction, stroke, heart failure, oedema and hypertension are so high that the Australian government considers them to be a National Health Priority. These conditions are not only causing a financial burden, but also a physical and emotional cost for many individuals. See Figure 31.1 for information regarding prevalence of common cardiovascular conditions.

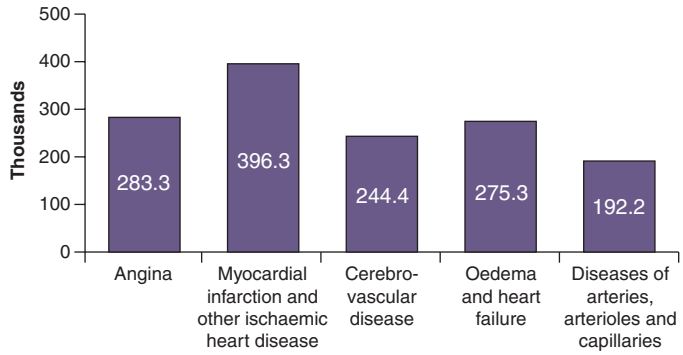


FIGURE 31.1 ■ Prevalence of heart, stroke and vascular conditions in Australia, 2011–2012

Source: Generated using data from Australian Bureau of Statistics (ABS) (2013b). *Australian Health Survey: First Results—Table 3: Long-term Conditions by Age then Sex*. (Cat. no. 4364.0.55.001.) Retrieved from www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/4364.0.55.0012011-12?OpenDocument. © Commonwealth of Australia.

DISORDERS OF BLOOD PRESSURE REGULATION

Blood flows through the circulatory system from areas of higher pressure to areas of lower pressure. The amount of pressure in any portion of the vascular system is affected by a number of factors, including blood volume, vascular resistance and cardiac output. The **blood pressure (BP)** is the tension or pressure exerted by blood against arterial walls. A certain amount of pressure within the system is necessary to maintain open vessels, capillary perfusion and oxygenation of all body tissues. Excess pressure, however, has harmful effects, increasing the workload of the heart, altering the structure of the vessels and affecting sensitive body tissues such as the kidneys, eyes and central nervous system (CNS).

This section focuses on **hypertension**, or excess pressure in the arterial portion of systemic circulation. Excessively low blood pressure, *hypotension*, is discussed in the shock section of Chapter 10. Altered pulmonary vascular pressures are discussed in Chapter 36.

Physiology review

Blood flow through the circulatory system requires *sufficient blood volume* to fill the blood vessels and *pressure differences* within the system that allow blood to move forward. The arterial, or supply, side of the circulation has relatively high pressures created by the thick elastic walls of the arteries and arterioles. The venous, or return, side of the system, on the other hand, is a low-pressure system of thin-walled, distensible veins. Blood flows through the capillaries linking these two systems from the higher-pressure arterial side to the lower-pressure venous side.

The arterial blood pressure is created by the ejection of blood from the heart during systole (*cardiac output*, or *CO*) and the tension or resistance to blood flow created by the elastic arterial walls (*systemic vascular resistance*, or *SVR*). The blood pressure rises as the heart contracts during systole, ejecting its blood. This pressure wave, or the **systolic blood pressure**, is felt as the peripheral pulse and heard as the Korotkoff's sounds during blood pressure measurement. In healthy adults the average systolic pressure is less than 120 mmHg. During diastole or cardiac relaxation and filling, elastic arterial walls maintain a minimum pressure, the **diastolic blood pressure**, to maintain blood flow through the capillary beds. The average diastolic pressure in a healthy adult is less than 80 mmHg. The difference between the systolic and diastolic pressure, normally about 40 mmHg, is known as the **pulse pressure**. The **mean arterial pressure (MAP)** is the average pressure in the arterial circulation throughout the cardiac cycle. It can be calculated using the formula (systolic BP + diastolic BP + diastolic BP)/3. Diastole counts twice as much as the systole because two-thirds of the cardiac cycle is spent in diastole.

CONSIDERATION FOR PRACTICE

- Cardiac output and systemic (or peripheral) vascular resistance are the primary factors that determine blood pressure.
- A decrease in cardiac output (e.g. due to haemorrhage) or decreased peripheral vascular resistance (e.g. systemic vasodilation) causes the blood pressure to fall.
- Increased cardiac output (e.g. during exercise) or increased peripheral vascular resistance (e.g. vasoconstriction due to drug administration) causes the blood pressure to rise.

Cardiac output is determined by the blood volume and the ability of the ventricles to fill and effectively pump that blood. A number of factors contribute to systemic vascular resistance, including vessel length, blood viscosity and vessel diameter

and distensibility (compliance). While vessel length and blood viscosity remain relatively constant, vessel diameter and compliance are subject to normal regulatory activities and disease.

The arterioles normally determine the SVR as their diameter changes in response to a variety of stimuli:

- *Sympathetic nervous system (SNS) stimulation*. Baroreceptors in the aortic arch and carotid sinus signal the SNS via the cardiovascular control centre in the medulla when the MAP changes. A drop in MAP stimulates the SNS, increasing the heart rate and cardiac output and constricting arterioles (except in skeletal muscle). As a result, BP rises. A rise in MAP has the opposite effect, decreasing the heart rate and cardiac output causing arteriolar vasodilation.
- *Circulating adrenaline and noradrenaline* from the adrenal cortex (e.g. the fight-or-flight response) have the same effect as SNS stimulation.
- *Renin–angiotensin–aldosterone system* responds to renal perfusion. A drop in renal perfusion stimulates renin release. Renin converts angiotensinogen to angiotensin I, which is subsequently converted to angiotensin II in the lungs by angiotensin-converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor. It also promotes sodium and water retention both directly and by stimulating the adrenal medulla to release aldosterone. Both SVR and CO increase, resulting in a rise in BP.
- *Atrial natriuretic peptide (ANP)* and *brain natriuretic peptide (BNP)* are released from atrial cells in response to stretching by excess blood volume. These hormones promote vasodilation along with sodium and water excretion, resulting in lowering of BP.
- *Adrenomedullin* is a peptide synthesised and released by endothelial and smooth muscle cells in blood vessels. It is a potent vasodilator.
- *Vasopressin* or *antidiuretic hormone* (from the posterior pituitary gland) promotes water retention and vasoconstriction, resulting in a rise in BP.
- *Local factors* such as inflammatory mediators and various metabolites can promote vasodilation, causing changes in BP.

In addition to the preceding, the primary factor affecting vessel compliance is the extent of arteriosclerosis (hardening of the arteries) and atherosclerosis (plaque accumulation). Figure 31.2 summarises the interrelationships of major factors regulating blood pressure.

CONSIDERATION FOR PRACTICE

- Sympathetic nervous system stimulation, adrenaline and noradrenaline, and the hormones angiotensin II and vasopressin (antidiuretic hormone or ADH) are *vasoconstrictors*, increasing BP.
- Parasympathetic nervous system stimulation and the hormones ANP, BNP and adrenomedullin are *vasodilators*, decreasing BP.
- The hormones aldosterone and ADH promote sodium and water retention, increasing BP.

PRIMARY HYPERTENSION

Primary hypertension, also known as *essential hypertension*, is a persistently elevated systemic blood pressure. Hypertension is defined as systolic blood pressure of 140 mmHg or higher, or diastolic pressure of 90 mmHg or higher, based on the average of three or more readings taken on separate occasions (Australian Institute of Health and Welfare (AIHW), 2014a). People who are currently normotensive but are taking antihypertensive medication are also considered to have hypertension for the sake of statistics.

Hypertension is an important and very common public health issue. While it rarely causes symptoms or noticeably limits the person's functional health patterns, hypertension is a major risk factor for coronary heart disease, heart failure, stroke and kidney failure.

Despite steady improvement over the past three decades, cardiovascular disease remains one of the biggest causes of death in Australia and continues to generate a considerable burden on the population in terms of illness and disability. The treatment options for cardiovascular disease prolong life, but as individuals develop more risk factors such as hypertension, obesity and diabetes, the burden on the health dollar will only increase (Department of Health (DOH), 2015).

Incidence and risk factors

In Australia, almost 8500 people were admitted for primary hypertension in 2012–2013. Hypertension is the third most common chronic disease behind arthritis and back pain and has been diagnosed in 10.2% (2.2 million) of Australians (AIHW, 2014a). However, in the 2011–2012 *Australian Health Survey*, 32% of all adults who had their blood pressure measured were hypertensive, with a slightly higher incidence in males (34%) than females (29%) (ABS 2013a; AIHW 2014a). Nevertheless, interestingly, in the same period of time, 62% of all admissions to Australian hospitals with a primary diagnosis of hypertension were women (ABS, 2013b).

Prevalence of hypertension in Aboriginal and Torres Strait Islander people is also more complex than one would initially consider. Government statistics examining the prevalence of hypertension in Aboriginal and Torres Strait Islander people report a figure 1.2 times higher than in non-Indigenous Australians (ABS, 2014). When both cultures are analysed separately, a recently published research article suggested that Aboriginal people were twice as likely to develop hypertension than Torres Strait Islander people (Li & McDermott, 2015) (see the 'Focus on cultural diversity' box below). Another variable that appears important in determining the prevalence of hypertension in Australia is location and degree of remoteness. Australians living in rural and remote areas are 1.15 to 3 times more likely to develop hypertension (ABS, 2011; National Heart Foundation of Australia (NHF), 2010).

Hypertension and its consequences are not unique to Australia. The World Health Organization identified that 40% of adults have hypertension worldwide (WHO, 2013). However, prevalence differs between countries and within regions; for example, the Republic of Korea (13.2%) is reported to have the lowest prevalence and Estonia (38.3%) the highest (WHO, 2014). See Table 31.1 for prevalence of hypertension in the Western Pacific region.

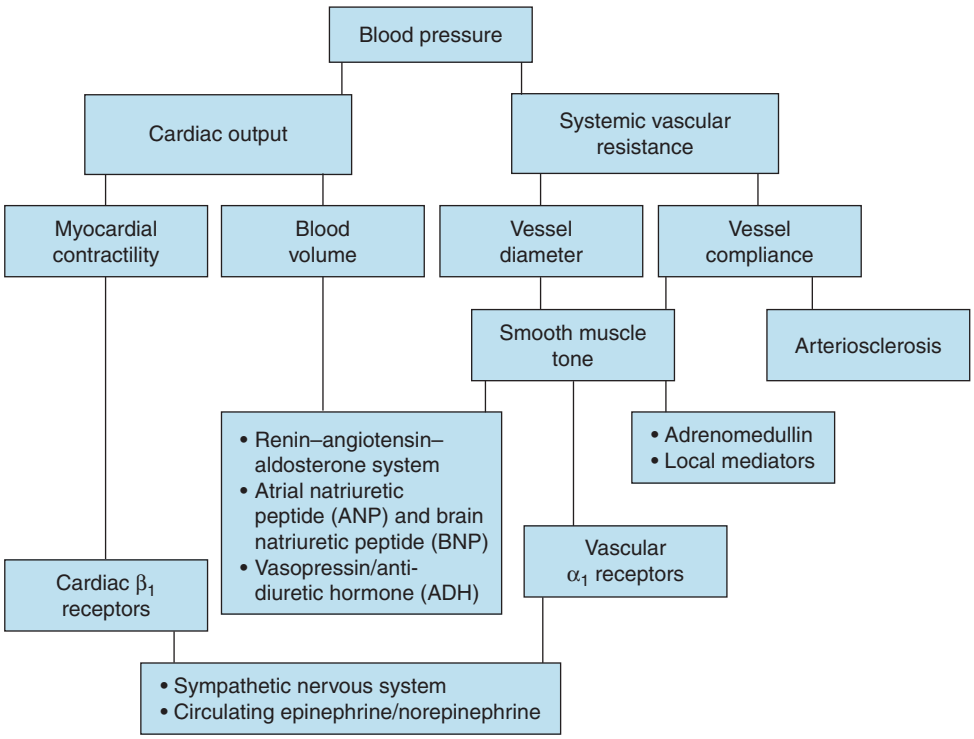


FIGURE 31.2 ■ Factors affecting blood pressure

TABLE 31.1 Age-standardised estimated prevalence of hypertension statistics for selected countries in the Pacific region

COUNTRY	ESTIMATED PREVALENCE REPORTED BY WHO
Australia	18.4%
New Zealand	19.1%
Solomon Islands	22.9%
Papua New Guinea	23.2%
Philippines	23.5%
Malaysia	24.5%
Cook Islands	25.3%
Fiji	25.5%
Nauru	25.9%
Mongolia	34.3%

Source: Generated using data from WHO (2014).

A number of risk factors have been identified for primary hypertension (see Box 31.1). Genetics play a role, as do environmental factors.

- *Family history*. Studies show a genetic link in about 31–68% of people with primary hypertension with over 40 potential loci identified for genes which may play a role in blood pressure regulation (Padmanabhan, Caulfield & Dominiczak, 2015). These include genes involved in the renin–angiotensin–aldosterone system, vascular tone, sodium transport, renal function, steroid synthesis, sympathetic nervous system function, endothelial function, obesity and insulin resistance (Padmanabhan, Newton-Cheh & Dominiczak, 2012).
- *Age*. The incidence of hypertension rises with increasing age. Ageing affects baroreceptors involved in blood pressure regulation as well as arterial compliance. As the arteries become less compliant, pressure within the vessels increases. This is often most apparent as a gradual increase in the systolic pressure with ageing.
- *Race*. The prevalence of hypertension in Aboriginal and Torres Strait Islander people is 25% compared to 20% for non-Indigenous Australians (NHF, 2014).

FOCUS ON CULTURAL DIVERSITY

- Aboriginal and Torres Strait Island people are 1.2 times more likely to develop hypertension than non-Indigenous Australians.
- Aboriginal Australians are twice as likely to develop hypertension than Torres Strait Islander Australians.
- The prevalence of hypertension in New Zealand in 2012–2013 was 15.9%, with Māori people 1.4 times and Pacific Island people 1.3 times more likely to have high blood pressure than non-Māori people.

Source: ABS (2014); Li & McDermott (2015); Ministry of Health (MOH) 2013).

BOX 31.1 Factors contributing to hypertension

Modifiable factors
- High sodium intake
- Low potassium, calcium and magnesium intake
- Obesity
- Excess alcohol consumption
- Insulin resistance

Non-modifiable factors
- Genetic factors
- Age
- Family history
- Race

- *Mineral intake*. High sodium intake often is associated with fluid retention. Hypertension related to sodium intake involves a number of different physiological mechanisms, including the renin–angiotensin–aldosterone system, nitric oxide, catecholamines, endothelin and atrial natriuretic peptide. Low potassium, calcium and magnesium intake can contribute to the development of hypertension, as it results in renin–angiotensin–aldosterone system activity (Adebamowo et al., 2015). The ratio of sodium to potassium intake appears to play a role, with a sodium-to-potassium ratio of < 1 being protective. Potassium promotes vasodilation by reducing responses to catecholamines and angiotensin II, modulating baroreflex sensitivity and reducing intracellular sodium. Magnesium has antihypertensive effects through numerous mechanisms including an increase in nitric-oxide-mediated vasodilation, reduction of noradrenaline and inhibition of platelet aggregation (Houston, 2014).
- *Obesity*. Central obesity (fat cell deposits in the abdomen), determined by an increased waist-to-hip ratio, has a stronger correlation with hypertension than body mass index or skinfold thickness. Weight reduction by as little as 4.5 kg reduces BP and/or prevents hypertension in a large proportion of overweight people (Iqubal et al., 2015). Weight loss of 10 kg can reduce systolic BP by 5–20 mmHg (Williams, 2015). Although a clear correlation exists between obesity and hypertension, the relationship may be one of common cause: genetic factors appear to play a role in the common triad of obesity, hypertension and insulin resistance. (See the 'Focus on cultural diversity' box below.)
- *Insulin resistance*. Insulin resistance with resulting hyperinsulinaemia is linked with hypertension by its effects of excess circulating insulin on the sympathetic nervous system, vascular smooth muscle, renal regulation of sodium and water and ion transport across cell membranes. Insulin resistance may be a genetic or an acquired trait. Although it is more commonly seen in obese individuals, insulin resistance also has been found in people of normal weight.
- *Excess alcohol consumption*. Regular consumption of three or more drinks a day increases the risk of hypertension. Decreasing or discontinuing alcohol consumption reduces the blood pressure, particularly systolic readings. Lifestyle factors associated with excessive alcohol intake (obesity and lack of exercise) may contribute to hypertension as well.
- *Stress*. Physical and emotional stress cause transient elevations of blood pressure, but the role of stress in primary hypertension is less clear. Blood pressure normally fluctuates throughout the day, increasing with activity, discomfort or emotional responses such as anger. Sympathetic nervous system hyperactivity, renin–angiotensin–aldosterone system activation and inflammation-mediated endovascular dysfunction have all been implicated (De Ciuceis et al., 2014).

FAST FACTS

- Major preventable risk factors for cardiovascular disease include tobacco smoking, hypertensive disease (high blood pressure), high blood cholesterol, inadequate physical activity, overweight and obesity, poor nutrition and diabetes.
- Data from the *Australian Health Disease Statistics* showed that prevalence of hypertensive disease increases with age, with less than 10% of those aged 25–34 years reporting the disease compared to almost 50% for those aged 75 years and over.
- The proportion of overweight and obese Australians is increasing. In 1995, 56.3% of people aged 18 years and over were classified as overweight or obese. This increased to 62.8% by 2012 (age adjusted).

Source: National Heart Foundation of Australia (2014). *Australian Heart Disease Statistics*. Retrieved from www.heartfoundation.org.au/SiteCollectionDocuments/HeartStats_2014_web.pdf.

Pathophysiology

Primary hypertension is thought to develop from complex interactions among factors that regulate cardiac output and systemic vascular resistance. These interactions may include:
- Excess sympathetic nervous system with overstimulation of α- and β-adrenergic receptors, resulting in vasoconstriction and increased cardiac output.
- Altered function of the renin–angiotensin–aldosterone system and its responsiveness to factors such as sodium intake and overall fluid volume. The renin–angiotensin–aldosterone system affects vasomotor tone and salt and water excretion. Chronically high levels of angiotensin II lead to arteriolar remodelling, which permanently increases SVR. In approximately 20% of people with primary hypertension, renin levels are lower than normal. Increased sodium intake increases the blood pressure in these people. Another 15% of people with hypertension

FOCUS ON CULTURAL DIVERSITY

Australia is rich with people who were born overseas, but choose to live here. See Figure 31.3 for comparisons between obesity values in Australians and other ethnic groups.

FIGURE 31.3 ■ Obesity prevalence (reported as a percentage) for people living in Australia by country of birth, 2012

Source: Generated using data from Australian Bureau of Statistics (2013c). *Australian Health Survey: Updated Results 2011–2012—Table 7: Body Mass Index by Selected Population Characteristics—Australia.* (Cat. no. 4364.0.55.001.) Retrieved from www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/4364.0.55.0032011-2012?OpenDocument. © Commonwealth of Australia.

have higher than normal plasma renin levels. For these people, salt intake has less of an effect on blood pressure (Kasper et al., 2015). Most people with hypertension have normal levels of renin activity.

- Other chemical mediators of vasomotor tone and blood volume, such as atrial natriuretic peptide, also play a role by affecting vasomotor tone and sodium and water excretion. Vascular endothelium itself produces hormones (*endothelins*) that also affect vasomotor tone. Endothelin-1 is a potent vasoconstrictor (Kasper et al., 2015).
- The interaction between insulin resistance, hyperinsulinaemia and endothelial function may be a primary cause of hypertension. Excess insulin has several effects that potentially contribute to hypertension: (1) sodium retention by the kidneys, (2) increased SNS activity, (3) hypertrophy of vascular smooth muscle, and (4) changes in ion transport across cell membranes (Kasper et al., 2015).

The result is sustained increases in blood volume and peripheral resistance. The cardiovascular system adapts to increased blood volume by increasing cardiac output. Autoregulatory mechanisms in the systemic arteries react to the increased volume, causing vasoconstriction. The increased systemic vascular resistance causes hypertension.

It appears unlikely that one single cause and pathological process will be found to account for essential hypertension. Increasingly, evidence points to hypertension as a diverse group of pathophysiological mechanisms resulting in the common manifestation of elevated blood pressure.

Manifestations

The early stages of primary hypertension typically are asymptomatic, marked only by elevated blood pressure. Blood pressure elevations are initially transient but eventually become permanent. When symptoms do appear, they are usually vague. Headache, usually in the back of the head and neck, may be present on awakening, subsiding during the day. See Box 31.2 for common errors that lead to the undertreatment of hypertension.

Other symptoms result from target-organ damage and may include nocturia, confusion, nausea and vomiting, and visual disturbances. Examination of the retina of the eye may reveal narrowed arterioles, haemorrhages, exudates and *papilloedema* (swelling of the optic nerve).

Complications

Sustained hypertension affects the cardiovascular, neurological and renal systems. The rate of atherosclerosis accelerates,

BOX 31.2 Common errors in assessing hypertension

The following errors can contribute to undertreatment of hypertension:

- Cuff placed over clothing
- Incorrect cuff size
- Worn cuff
- Inaccurate sphygmomanometer (e.g. not serviced regularly, not validated correctly)
- Arm elevated above heart
- Failure to check that both arms give comparable readings (e.g. at initial visit)
- Person not rested before measurement
- Person talking during measurement
- Failure to palpate radial pulse before auscultatory measurements (results in failure to detect auscultatory gap)
- Deflating the cuff too quickly (> 2–3 mmHg/beat, whether using a mercury or digital sphygmomanometer)
- Re-inflating the cuff to repeat measurement before it has fully deflated
- Rounding off actual reading by more than 2 mmHg when recording measurement
- Taking a single measurement.

Source: National Heart Foundation (2010). *Guide to management of hypertension 2008: Updated December 2010.* Retrieved from www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008to2010Update.pdf.

increasing the risk of coronary heart disease and stroke. The workload of the left ventricle increases, leading to ventricular hypertrophy, which then increases the risk of coronary heart disease, arrhythmias and heart failure. The diastolic blood pressure is a significant cardiovascular risk factor until age 50; the systolic pressure then becomes the more important factor contributing to cardiovascular risk. The National Heart Foundation (2010) recommends a cardiovascular risk assessment be performed by all Australians aged 45–74 who are not already known to be at high risk. Most deaths due to hypertension result from coronary heart disease and acute myocardial infarction or heart failure (Kasper et al., 2015).

Accelerated atherosclerosis associated with hypertension increases the risk of cerebral infarction (stroke). Increased pressure in the cerebral vessels can lead to development of microaneurysms and an increased risk of cerebral haemorrhage. *Hypertensive encephalopathy*, a syndrome characterised by extremely high blood pressure, altered level of consciousness, increased intracranial pressure, papilloedema and seizures, may develop. Its aetiology is unclear.

Hypertension also can lead to nephrosclerosis and renal insufficiency. Proteinuria and microscopic haematuria develop, as well as signs of chronic kidney failure. In 2013, after adjusting for differences in the age structure of the populations, Indigenous Australians were 3.3 times more likely than non-Indigenous Australians to have diabetes and more than 2.1 times as likely to have kidney disease. However, the gaps in remote areas were more remarkable, with Aboriginal and Torres Strait Australians being 5.4 times more likely to have diabetes and almost 4 times more likely to have kidney disease than non-Indigenous Australians.

INTERPROFESSIONAL CARE

Hypertension management focuses on reducing the blood pressure to less than 140 mmHg systolic and 90 mmHg diastolic. The ultimate goal of hypertension management is to reduce cardiovascular and renal morbidity and mortality. The risk of cardiovascular complications (coronary heart disease, heart failure, stroke) decreases when the average blood pressure is less than 140/90; when the person also has diabetes or renal disease, the treatment goal is a blood pressure of less than 130/80. The decision to intervene and the development of a comprehensive management plan (including lifestyle advice and drug treatment) should be based on a thorough clinical investigation to identify associated clinical conditions and/or end-organ damage and assessment of cardiovascular risk (NHF, 2010). Although there is no cure for hypertension, it can be controlled. Figure 31.4 shows the recommended algorithm for hypertension management.

Diagnosis

The person with hypertension is evaluated for the presence of identifiable causes of hypertension, cardiovascular risk factors and the presence or absence of target-organ damage (heart, brain, kidneys, peripheral vascular systems and retina of the eye). Before treatment is started, the following diagnostic tests are performed: electrocardiogram (ECG); urinalysis; blood glucose level; haematocrit; serum potassium, creatinine and calcium; and cholesterol and lipoprotein profile, including HDL, LDL and triglycerides.

Additional tests that may be done include urinary albumin excretion, evaluation of the glomerular filtration rate (such as the creatinine clearance) and tests for emerging cardiovascular risk factors such as C-reactive protein and homocystine levels.

Lifestyle modifications

Lifestyle modifications are recommended for all people whose blood pressure falls within the prehypertension range (120–139/80–89 mmHg) and everyone with intermittent or sustained hypertension. These modifications include weight loss, dietary changes, restricted alcohol use and cigarette smoking, increased physical activity and stress reduction (see Box 31.3).

DIET Dietary approaches to managing hypertension focus on reducing sodium intake, maintaining adequate potassium and calcium intakes, and reducing total and saturated fat intake. A mild to moderate sodium restriction (no added salt) lowers blood pressure and potentiates the effect of antihypertensive drugs for most people with hypertensive. A diet that focuses on whole foods rather than individual nutrients has proven to have beneficial effects in lowering blood pressure (see Box 31.4).

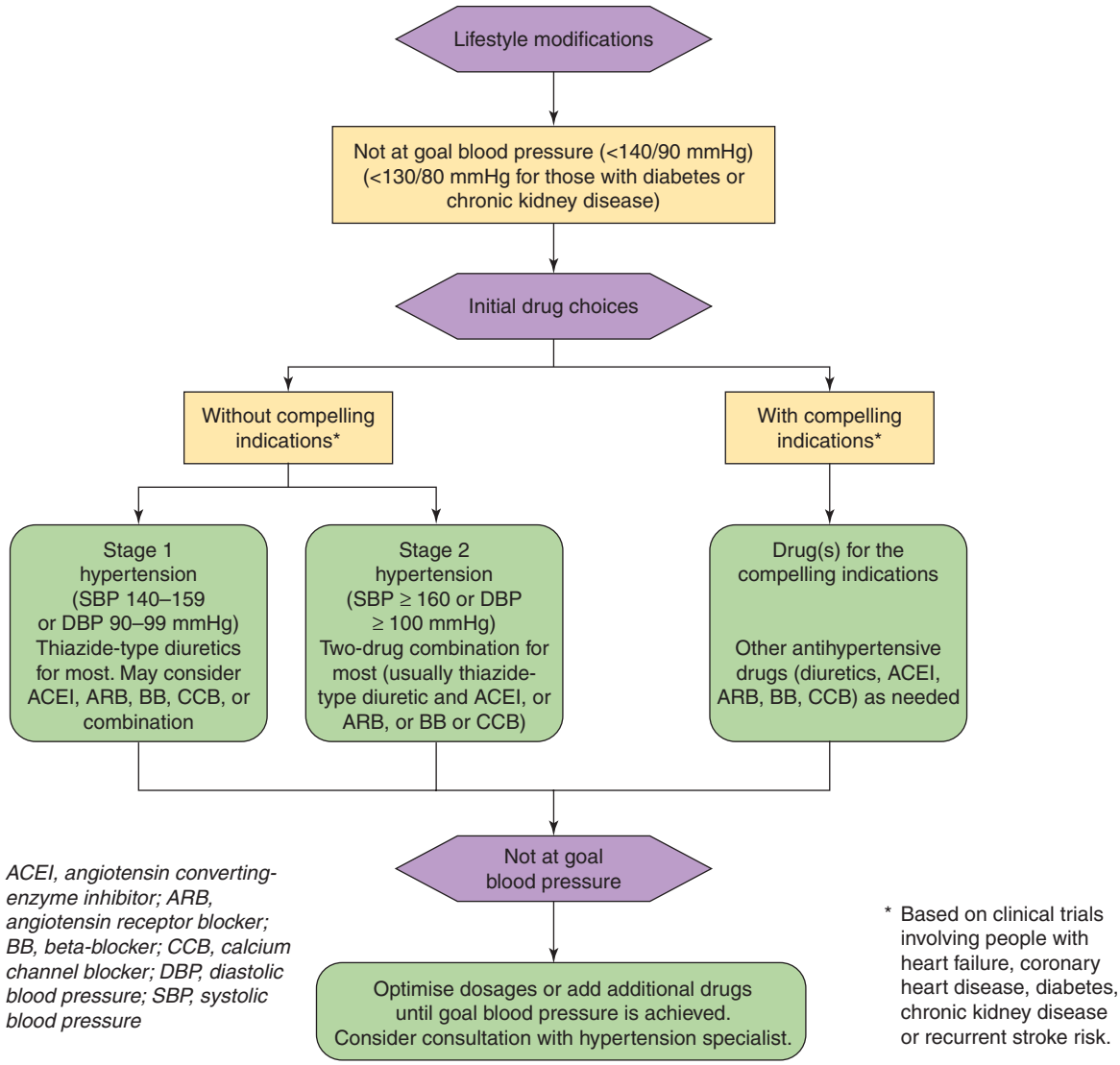


FIGURE 31.4 ■ Algorithm for treating hypertension

Source: Adapted from *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure* (2004). National Heart, Lung and Blood Institute, Bethesda, MD: National Institutes of Health.

It is rich in fruit and vegetables (up to 10 servings per day) and low in total and saturated fats.

Managing identifiable lifestyle risk factors is recommended for people with or without hypertension. Waist measurements of < 94 cm for men and < 80 cm for women along with a body mass index (BMI) of < 25 kg/m^2 should be encouraged. When recommending weight loss, advise on reducing kilojoule intake as well as increasing physical activity (NHF, 2010).

PHYSICAL ACTIVITY Regular exercise (such as walking, cycling, jogging or swimming) reduces blood pressure and contributes to weight loss, stress reduction and feelings of overall wellbeing. Advise all people to become physically active as part of a comprehensive plan to control hypertension, regardless of drug treatment. Aim for 30 minutes of moderate-intensity physical activity on most, if not all, days of the week (NHF, 2010).

ALCOHOL Moderate drinking may increase BP by 38–40 mmHg and binge drinking may increase the risk of hypertension. Reducing alcohol consumption can substantially lower BP in some people. Although alcohol withdrawal may increase blood pressure, this is usually temporary and diminishes as abstinence or restricted intake continues. Advise people with hypertension to limit their intake to a maximum of two standard drinks (a standard drink equals 10 g of alcohol) per day for men, and a maximum of one standard drink per day for women and lighter-weight men. Finally, people should be advised to observe at least two alcohol-free days per week.

SMOKING CESSATION Smoking cessation may not directly reduce BP, but it markedly reduces overall cardiovascular risk. The risk of myocardial infarction is two to six times higher, and the risk of stroke is three times higher, in people who smoke than in non-smokers. Advice from health professionals is effective in increasing quit rates. Even 3–5 minutes taken to

BOX 31.3 Lifestyle modifications for hypertension

The following guidelines need to take into account a person's cultural background and the resources available to them. Further, nurses need to assess a person's readiness for change and tailor recommendations accordingly.

Advise person to aim for healthy targets:

- At least 30 minutes of moderate-intensity physical activity on most, if not all, days of the week (daily total can be accumulated, e.g. three 10-minute sessions). Advise people of all ages to become more active.
- Smoking cessation. Refer the person to Quit Helpline. Consider recommending nicotine replacement therapy and/or prescribing oral therapy (bupropion or varenicline) in people who smoke more than 10 cigarettes per day and have no contraindications.
- Waist measurement < 94 cm for men and < 80 cm for women, and body mass index (BMI) < 25 kg/m^2. When recommending weight loss, advise the person on reducing kilojoule intake as well as increasing physical activity.
- Dietary salt restriction: ≤ 4 g/day (65 mmol/day sodium). Recommend low-salt and reduced-salt foods as part of a healthy eating pattern.
- Limited alcohol intake: maximum of two standard drinks per day for men or one standard drink per day for women.

Source: NHF (2010). *Guide to the management of hypertension 2008: Updated December 2010.* © National Heart Foundation of Australia.

BOX 31.4 Dietary recommendations

A healthy eating pattern includes mainly plant-based foods (e.g. fruit, vegetables, pulses); a wide selection of wholegrain foods; moderate amounts of low-fat or reduced-fat dairy products; moderate amounts of lean unprocessed meats, poultry and fish; and moderate amounts of polyunsaturated and monounsaturated fats (e.g. olive oil, canola oil, reduced-salt margarines).

Source: NHF (2010). *Guide to the management of hypertension 2008: Updated December 2010.* © National Heart Foundation of Australia.

encourage smokers to attempt to quit can increase success rates (NHF, 2010).

STRESS REDUCTION Stress stimulates the sympathetic nervous system, increasing vasoconstriction, systemic vascular resistance, cardiac output and blood pressure. Regular, moderate exercise is the treatment of choice for reducing stress in people with hypertension. Relaxation techniques such as biofeedback, therapeutic touch, yoga and meditation to relax both mind and body may also lower blood pressure, although their effect has not been proven in hypertension management (Oza & Garcellano, 2015).

Medications

Current pharmacological treatment of hypertension involves using one or more of the following drug classes: diuretics, beta-adrenergic blockers, centrally acting sympatholytics, vasodilators, ACE inhibitors, angiotensin II receptor blockers (ARBs) and calcium channel blockers. For most people, two or more antihypertensive drugs selected from different drug classes are necessary to achieve effective control. These drug classes have different sites of action (see Figure 31.5). See the 'Medication administration' box below.

DRUG CLASSES For all major antihypertensive drug classes, the beneficial effect is mainly due to BP lowering, irrespective of their mechanism of action. In uncomplicated hypertension, the following classes of antihypertensive agents are equally effective for first-line use, both in initial and maintenance therapy:

- ACE inhibitors (or angiotensin II receptor antagonists)
- calcium channel blockers
- low-dose thiazide diuretics (for people aged 65 years and older).

Thiazide diuretics have been associated with increased risk of new-onset diabetes and should be used with caution in people with glucose intolerance and/or metabolic syndrome. The use of thiazide diuretics as first-line therapy should be limited to older people, in whom the benefits of managing isolated systolic hypertension and preventing stroke with these agents are likely to outweigh the risk of diabetes onset. Beta-blockers are no longer recommended as first-line therapy in uncomplicated hypertension because of the increased risk of developing diabetes and the recently described trend towards worse outcomes in people treated with beta-blockers compared to those treated with other classes of antihypertensive drugs.

For people with stable, well-controlled hypertension who are already taking a beta-blocker, it is reasonable to continue the regimen unchanged.

The initial drug choice should be based on:

- age
- presence of associated clinical conditions or end-organ damage
- presence of other coexisting conditions that either favour or limit the use of particular drug classes
- potential interactions with other drugs
- implications for adherence
- cost.

Most classes of antihypertensive agents used as monotherapy lower BP by a similar average amount. However, the individual response to each agent is unpredictable.

People with heart failure, coronary heart disease (CHD) or diabetes may initially be treated with a beta-blocker. These drugs lower blood pressure, apparently by reducing peripheral vascular resistance. They may also reduce the amount of renin released by the kidneys by blocking beta$_1$-receptors in the kidney. Beta-blockers reduce the risk of complications such as heart failure and stroke. They are, however, relatively contraindicated for people with asthma or chronic obstructive pulmonary disease, because they promote bronchial constriction.

ACE inhibitors and ARBs also are commonly used in initial treatment of hypertension, particularly for people who are

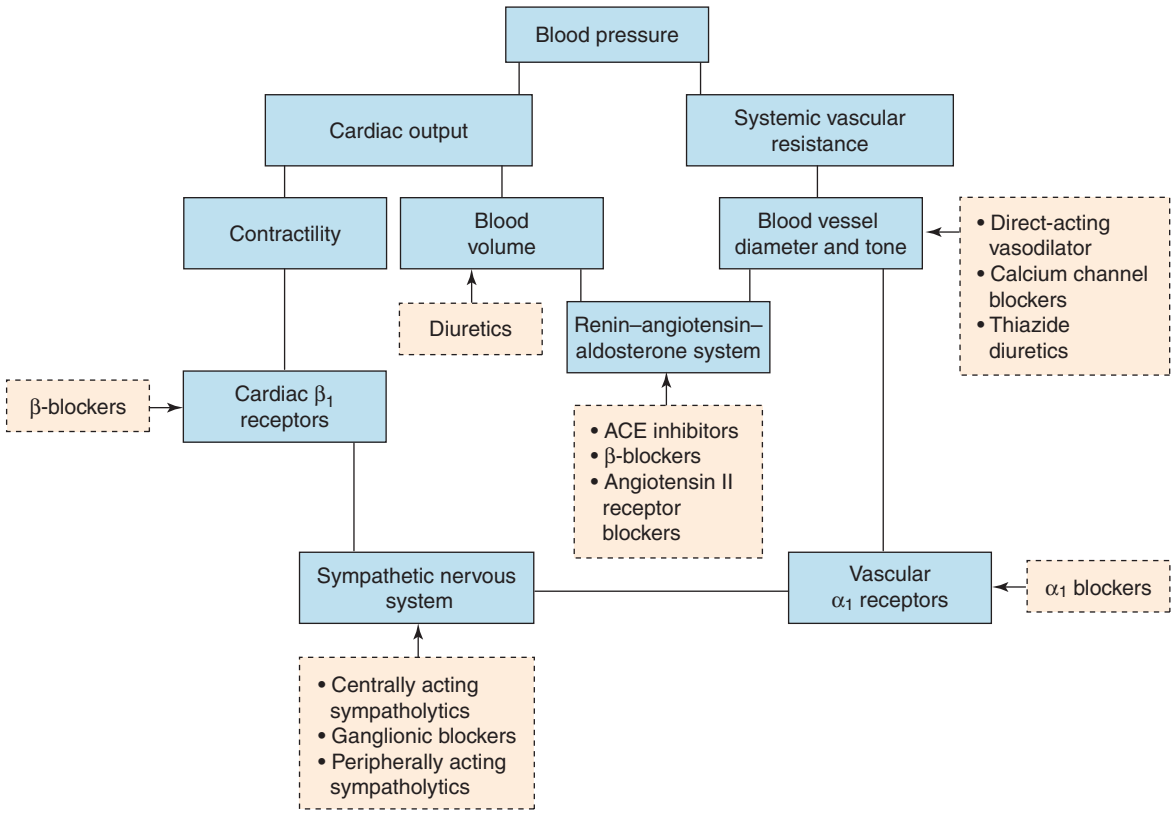


FIGURE 31.5 ■ Sites of antihypertensive drug action

diabetic or have heart failure, a history of myocardial infarction (MI) or chronic kidney disease. ACE inhibitors block formation of angiotensin II by inhibiting the action of angiotensin-converting enzyme. Angiotensin II is a potent vasoconstrictor that also stimulates aldosterone release from the adrenal gland; blocking its action prevents vasoconstriction and sodium and water retention resulting from aldosterone release. ARBs have a very similar effect, although their action is to block angiotensin II receptors, thus preventing its vasoconstrictive and volume expansion effects.

Several drug classes work through their ability to promote vasodilation and reduce peripheral vascular resistance. Alpha-blockers such as prazosin and terazosin block stimulation of alpha$_1$-receptors on arterioles and veins, preventing vasoconstriction. Because of their ability to dilate both arterioles and veins, alpha-blockers can cause significant orthostatic hypotension, particularly following the initial dose. Calcium channel blockers promote dilation of arterioles, the primary regulators of peripheral vascular resistance. These drugs can cause reflex tachycardia. Some calcium channel blockers, verapamil and diltiazem in particular, also suppress heart function, reducing stroke volume and cardiac output. Reflex tachycardia is minimal with these calcium channel blockers. Direct-acting vasodilators such as hydralazine and minoxidil also directly affect the arterioles, reducing peripheral vascular resistance. These drugs have little effect on veins, so the risk of orthostatic hypotension is minimal. They are, however, associated with reflex tachycardia and fluid retention, so rarely are administered as in single-drug treatment regimens.

Other factors considered in selecting drugs for treating hypertension include demographic characteristics of the person, concurrent conditions, quality of life, cost and possible interactions among prescribed drugs. In general, diuretics and calcium channel blockers are more effective for treating hypertension than beta-blockers or ACE inhibitors. Beta-blockers are preferred to treat hypertension with concurrent coronary heart disease and angina, but are contraindicated for those who have asthma or depression. Beta-blockers also reduce exercise tolerance and may adversely affect lifestyle for some people.

DRUG REGIMENS Treatment usually is initiated using a single antihypertensive drug at a low dose. Unless otherwise indicated, a diuretic is recommended as the initial drug of choice. The dose is slowly increased until optimal blood pressure control is achieved. If the drug does not effectively lower the blood pressure or has troubling side effects, a different drug from another class of antihypertensive medications is substituted. If, on the other hand, the drug is tolerated well but has not lowered blood pressure to the desired level, a second drug from another class may be added to the treatment regimen.

Treatment of people with stage 2 hypertension generally is more aggressive to minimise the risk of MI, heart failure or stroke. When the average blood pressure is greater than 200/120 mmHg, immediate therapy and possible hospitalisation is vital.

After a year of effective hypertension control, an effort may be made to reduce the dosage and number of drugs. This is known as step-down therapy. It is more successful in people who

MEDICATION ADMINISTRATION Antihypertensives

ALPHA-ADRENERGIC BLOCKERS

Alpha-adrenergic blocking agents block alpha-receptors in vascular smooth muscle, decreasing vasomotor tone and vasoconstriction. They also reduce serum levels of low-density lipoproteins (LDLs) and very-low-density lipoproteins (VLDLs). However, vasodilation may cause orthostatic hypotension and reflex stimulation of the heart, resulting in tachycardia and palpitations. A beta-blocker may be ordered to minimise this effect.

Nursing responsibilities

- Give the first dose at bedtime to minimise risk of fainting (called 'first-dose syncope'). If the first dose is given in the daytime (or if the dose is increased), instruct to remain in bed for 3 to 4 hours.
- Assess blood pressure and apical pulse before each dose and as indicated thereafter.

Health education for the person and family

- There is a risk of fainting after taking the first dose of this drug. Take the drug at bedtime to reduce this risk and do not drive or engage in other hazardous activities for 12 to 24 hours after the first dose.
- This drug may cause dizziness or light headedness. Change positions slowly and sit down if you become dizzy or light headed.
- Notify your primary care provider if you develop nasal congestion or impotence while taking this drug.
- Notify your doctor before discontinuing this medication.

ACE INHIBITORS AND ANGIOTENSIN II RECEPTOR ANTAGONISTS

Angiotensin-converting-enzyme (ACE) inhibitors lower blood pressure by preventing conversion of angiotensin I to angiotensin II. This in turn prevents vasoconstriction and sodium and water retention. Angiotensin II receptor blockers (ARBs) have the same effect, but they act by blocking the effect of angiotensin II on receptors. ACE inhibitors have been shown to reduce mortality in an Aboriginal community with a high prevalence of end-stage kidney failure (O'Dea, Rowley & Brown, 2007). Their primary adverse effects are persistent cough, first-dose hypotension and hyperkalaemia.

Nursing responsibilities

- Assess blood pressure and WBC before giving the first dose. Monitor blood pressure for 2 hours after the first dose and regularly thereafter.
- Administer PO 1 hour before meals; tablets may be crushed.
- Report changes in WBC or differential, hyperkalaemia or changes in serum urea or creatinine to the doctor.
- Do not administer to people with renal artery stenosis or who are pregnant.
- Immediately report and treat manifestations of angio-oedema (giant wheals and oedema of the tongue, glottis and pharynx). Initiate resuscitation measures as needed. Discontinue drug immediately and do not use in the future.

Health education for the person and family

- Report peripheral oedema, signs of infection or difficulty breathing to your primary care provider.
- Change position (lying to sitting and sitting to standing) slowly to prevent dizziness; sit down if dizziness or light headedness develops.
- Do not take a potassium supplement or use a potassium-based salt substitute while taking this drug unless prescribed by your doctor.
- Notify your doctor if you become pregnant while taking this drug. Although it is safe early in pregnancy, taking the drug during the second and third trimesters may harm the foetus.

BETA-ADRENERGIC BLOCKERS (BETA-BLOCKERS)

Beta-blockers are commonly used to control hypertension. They reduce blood pressure by preventing beta-receptor stimulation in the heart, thereby decreasing heart rate and cardiac output. Beta-blockers also interfere with renin release by the kidneys, decreasing the effects of angiotensin and aldosterone. Potential adverse effects of beta-blockers include bronchospasm, fatigue, sleep disturbances, nightmares, bradycardia, heart block, worsening of heart failure, gastrointestinal disturbances, impotence and increased triglyceride levels.

Nursing responsibilities

- Before giving initial dose, assess for contraindications to beta-blockers, such as asthma, chronic lung disease, bradycardia or heart block.
- Assess blood pressure and apical pulse before giving; notify primary care provider if vital signs are outside established parameters.
- Report adverse effects such as bradycardia, decreased cardiac output (fatigue, dyspnoea with exertion, hypotension, decreased level of consciousness), heart failure, heart block, bronchoconstriction (wheezing, dyspnoea) or altered blood glucose levels (in diabetic people).
- Carefully monitor responses of the older person.

Health education for the person and family

- Monitor blood pressure and pulse daily as instructed.
- Change position (lying to sitting and sitting to standing) slowly to prevent dizziness and possible falls.
- Report effects such as fatigue, lethargy and impotence to your primary care provider.
- Notify your doctor if you become short of breath or develop a cough or swelling of your extremities.
- If you have diabetes, check blood glucose levels more frequently because hypoglycaemia may develop with few symptoms.
- Talk to your primary care provider before taking any over-the-counter medications.
- Carry an adequate supply of the drug when travelling. Do not stop taking this drug without notifying your primary care provider.

CALCIUM CHANNEL BLOCKERS

Calcium channel blockers inhibit the flow of calcium ions across the cell membrane of vascular tissue and cardiac cells. In doing so, they relax arterial smooth muscle, lowering

MEDICATION ADMINISTRATION Antihypertensives (continued)

peripheral resistance through vasodilation. Calcium channel blockers can cause reflex tachycardia and some (e.g. verapamil and diltiazem) may impair cardiac function, worsening heart failure.

Nursing responsibilities

- Assess blood pressure, apical pulse and liver and renal function tests prior to giving these drugs.
- Calcium channel blockers may be given orally or intravenously.
- Do not administer verapamil or diltiazem to people with severe hypotension, sinus or atrioventricular blocks. Administer with caution to people also taking digoxin or a beta-blocker.
- Periodically monitor blood pressure and apical pulse during therapy. Promptly report signs of bradycardia, AV block or heart failure to the doctor.

Health education for the person and family

- Take blood pressure and pulse daily as taught. Notify your doctor if your pulse is less than 60 bpm or your blood pressure is not within the specified range.
- This drug may cause constipation. Drink six to eight glasses of water each day and increase fibre in diet.
- Report shortness of breath, weight gain or swelling in feet or ankles to your primary care provider.

THIAZIDE DIURETICS

These drugs inhibit reabsorption of sodium and chloride in the proximal (diluting) segment of the distal convoluted tubules. They also increase urinary secretion of water, chloride, potassium, magnesium, whereas excretion of uric acid and calcium is decreased. Indications include the treatment of mild to moderate hypertension and oedema associated with heart failure. They should be used with caution for people who have type 1 diabetes, gout, renal or hepatic impairment and in the elderly. These drugs are contraindicated in severe renal impairment, anuria, Addison's disease and in people with thiazide or sulfonamide hypersensitivity (Bryant & Knights, 2011, p. 515). Loop diuretics (e.g. frusemide) are not recommended as antihypertensive agents unless volume overload is present.

Nursing responsibilities

- Monitor closely if concurrent potassium supplementation or a potassium chloride salt substitute is ordered for people receiving a potassium-sparing diuretic. Hyperkalaemia and death have been reported with this combination.
- Use extreme caution in elderly people. Start with lowest dose possible, titrate slowly to achieve the desired effect.
- Monitor for diuresis and subsequent incontinence.
- Be alert to signs and symptoms of diuretic toxicity, such as anorexia, nausea, vomiting, confusion, increased weakness and paraesthesia of the extremities.
- When diuretic is to be discontinued, reduce the drug gradually to avoid the development of fluid retention and oedema.
- If plasma potassium concentration drops below the laboratory reference range during thiazide diuretic therapy, a potassium-sparing diuretic may be prescribed in combination with the thiazide.

Health education for the person and family

- Fluid intake should be discussed with the individual as many people refer to diuretics as 'water pills' and mistakenly believe they should restrict their fluid intake.
- Report any anorexia, nausea, vomiting, confusion or increased weakness of arms and legs to your health care provider.
- Do not decrease or discontinue medication without talking to your healthcare provider.
- Advise person their urinary output will be increased and they may experience incontinence. Provide education on managing same.

OTHER

Most of the drugs in this category are adjunctive therapies to those listed in the above categories. Please refer to pharmacology texts for guidance on the use of these additional drugs.

Source: Selected data in this box is adapted from National Heart Foundation (2010). *Guide to the management of hypertension 2008: Updated December 2010.* Retrieved from www.heartfoundation.org.au/SiteCollectionDocuments/HypertensionGuidelines2008QRG2010Update.pdf.

have made lifestyle modifications. Careful blood pressure monitoring is necessary during and after step-down therapy because the blood pressure often rises again to hypertensive levels.

Complementary therapies

Behavioural and mind–body therapies may be helpful for some people in lowering blood pressure. Blood pressure increases in response to physiological and psychological stress and anxiety. Mind–body therapies such as yoga and tai chi, meditation and guided imagery are designed to modify both physiological and cognitive aspects of the stress response. However, it is critical to understand that alternative medicines and foods can negatively influence conventional treatments. See the 'Translation to practice' box identifying the effects of green tea on antihypertensive medications.

Nursing care

Health promotion

Health promotion teaching and activities focus on the modifiable risk factors for hypertension. Advise and support all people (including children and adolescents) to stop or never start smoking. Discuss the risks of obesity, excess alcohol intake and a sedentary lifestyle. Encourage everyone to eat a diet rich in fruit and vegetables and low in total and saturated fat. Discuss the potential benefits of following a healthy diet. Advise all people to remain active and engage in aerobic exercise 5 or

TRANSLATION TO PRACTICE Evidence-based practice: green tea interferes with antihypertensive medication function

In a randomised, crossover study by Misaka et al. (2014), subjects were given a beta-blocker every day for 14 days. The subjects consumed green tea for a week, followed by water for the next week, concomitantly with administration and within 30 minutes of the dose to a volume of 700 mL. The effects of the green tea were significant and worrying. A reduction in plasma concentrations of 85% were demonstrated without changes in renal clearance, suggesting that the green tea probably reduced gastrointestinal absorption of the drug. Subsequently, desired antihypertensive and rate-slowing effects of the beta-blocker were markedly reduced.

IMPLICATIONS FOR NURSING

Understanding that the consumption of certain foods and beverages may significantly influence the effectiveness of prescribed drugs is critical. Nurses not only are responsible for administration of medications in both acute and community care situations, they are often the healthcare professional tasked with ensuring that individuals understand medication that they are prescribed.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Review the four concepts of pharmacokinetics: absorption, distribution, metabolism and excretion. Consider how each of these parameters may be influenced and what result this would have on an antihypertensive medication.
2. Grapefruit is known to interfere with over 80 medications, with more than half of these interactions resulting in potentially serious consequences (NPS Medicinewise, 2014). Research other foods and beverages and their influence on the effects of cardiovascular medication. Make a report that includes the food or beverage name and its effects.
3. How will this research influence your future practice?

more days a week. Discuss the stress-reducing benefits of exercise.

Offer blood pressure screening and refer individuals for follow-up as indicated.

Assessment

Absolute cardiovascular risk

The management plan for a person with hypertension should take into consideration the individual's absolute risk of cardiovascular disease. Absolute cardiovascular risk is the probability (expressed as a percentage) of an individual experiencing a cardiovascular event (e.g. myocardial infarction or stroke) during a predefined period of time (e.g. the next 5 years). Blood pressure is a major determinant of absolute cardiovascular risk. People at highest absolute risk include those with existing cardiovascular disease or those with multiple risk factors (e.g. diabetes, older age, overweight/obesity and dyslipidaemia).

The purposes of assessing absolute cardiovascular risk are to:

- identify other modifiable risk factors that require management
- predict who will benefit most from intervention and determine the appropriate management plan to reduce BP
- enable the person to understand the degree of urgency for reducing BP and correcting other risk factors.

The National Vascular Disease Prevention Alliance (NVPDA) (2012) has produced guidelines for the assessment and management of cardiovascular disease in Australia. The ultimate goal is to reduce a person's absolute risk. Risk management decisions can be made once these factors are considered.

Risk analysis in Aboriginal and Torres Strait Islander people is complex for numerous reasons including the increased prevalence of co-morbidity at a younger age than non-Indigenous Australians. Indigenous Australians also have an exceedingly high mortality, which has not reduced in the past 40 years despite targeted actions. Therefore, NVDPA recommends screening of Aboriginal and Torres Strait Islander people from 35 years of age (instead of 45 years as for non-Indigenous Australians), and the use of specifically designed absolute risk charts.

Absolute cardiovascular risk assessment is now recommended for all Australians aged 45–74 who are not already known to be at high risk, whether or not they have hypertension.

The management of hypertension should be based on a thorough clinical assessment that includes an estimate of the person's absolute risk of cardiovascular disease, as well as BP levels and other clinical investigations.

Assessment of absolute cardiovascular risk helps both the healthcare practitioners and the person understand the individual's overall risk profile and the potential benefit of preventive interventions. Figure 31.6 demonstrates when management of hypertension should be undertaken.

People who need immediate antihypertensive drug treatment include (but are not restricted to) those at high absolute cardiovascular risk (> 15% probability of a cardiovascular event within the next 5 years).

High cardiovascular risk can be assumed for the following groups of people without using a risk calculator:

- *Group A. People aged 75 years and over*. For almost all individuals aged ≥ 75 years, the absolute risk of a cardiovascular event in the next 5 years is > 15%.
- *Group B. People with existing cardiovascular disease*. Assume risk of a cardiovascular event > 15% in the next 5 years if either of the following present:
  - symptomatic cardiovascular disease (e.g. angina, myocardial infarction, chronic heart failure, stroke)
  - transient ischaemic attack, peripheral arterial disease
  - left ventricular hypertrophy diagnosed with electrocardiography or echocardiography.

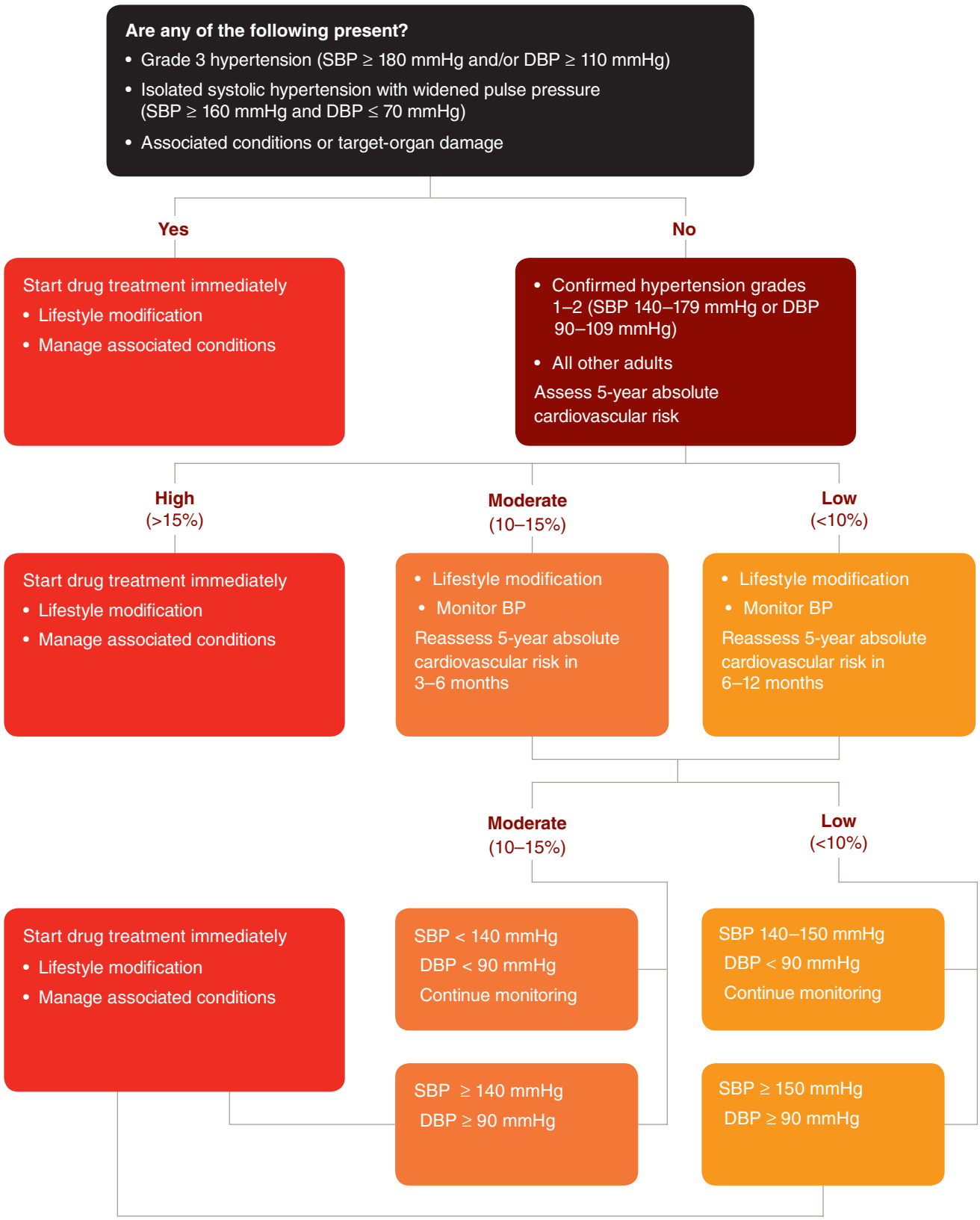


FIGURE 31.6 ■ When to initiate blood-pressure lowering medications

Source: NHF (2010). *Guide to the management of hypertension 2008: Updated December 2010.* © National Heart Foundation of Australia.

- *Group C. People with associated clinical conditions and end-organ disease*. For this group, assume risk of a cardiovascular event > 15% in the next 5 years. Antihypertensive drug treatment is required (e.g. to preserve renal function). For all other people, estimate absolute risk using Figure 31.6 (NHF, 2010).

Focused assessment of the person with hypertension includes:

- *Health history:* complaints of morning headache, cervical pain; cardiovascular or central nervous system manifestations; history of hypertension, renal disease, diabetes; family history of high blood pressure, heart failure or kidney disease; current medications.
- *Physical examination:* pulse rate, rhythm and character, jugular venous pulse and pressure, evidence of cardiac enlargement (displaced apex, extra heart sounds) or evidence of decompensation (basal crackles or wheeze on lung auscultation, peripheral oedema, abdominal signs, e.g. pulsatile liver).
- *Diagnostic tests:* blood analysis (sodium, potassium, chloride, bicarbonate, urea, creatinine, uric acid, haemoglobin, fasting glucose, total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides, liver function tests).
  - *Electrocardiogram (ECG)* to detect conduction disturbances, arrhythmias, coronary heart disease or left ventricular hypertrophy. The presence of strain pattern (ST depression and T-wave inversion) is associated with increased cardiovascular risk in people with hypertension.
  - *Dipstick testing of urine* for blood and protein. If abnormal, proceed to urine microscopy. If proteinuria detected (≥ 1+ on dipstick), measure 24-hour urinary protein excretion.

Nursing diagnoses and interventions

All people with primary hypertension and their families need significant teaching to manage this chronic condition. Health maintenance is a high-priority problem. Depending on the stage of hypertension and concurrent illnesses, other appropriate nursing diagnoses may include *Imbalanced nutrition*, *Excess fluid volume* and *Risk of non-compliance*.

Ineffective health maintenance

Unhealthy lifestyle and behaviours can contribute to health problems such as hypertension. When hypertension has been identified, knowledge of the disease and its management is vital for the person. Willingness to take responsibility for hypertension management is central to effective blood pressure control. Adopting healthy lifestyle changes enhances drug therapy; in some cases, the need for medications may be eliminated or reduced. Because hypertension is often an asymptomatic disease and many antihypertensive drugs have unpleasant side effects, it is vital that the person understand the chronic progressive nature of the disease and its long-term consequences.

- Assist with identifying current behaviours that contribute to hypertension. The person must first identify contributory behaviours before they can change them. *Using knowledge of hypertension risk factors, the nurse can help identify behaviours and factors contributing to hypertension that can be changed. Including the family in this process is important to reduce potential sabotage of the person's efforts to adopt healthier behaviours.*
- Assist in developing a realistic health maintenance plan. *Preparing a health maintenance plan for the person does little to encourage personal responsibility for health. However, nurses can guide people in developing realistic goals and expectations for the treatment plan and modifying risk factors such as smoking, exercise, diet and stress.*
- Help the person and family identify strengths and weaknesses in maintaining health. *Discussing areas of the health maintenance plan that are working well and those that present difficulties can help to identify necessary changes in the plan and additional strategies for implementing it.*

Risk of non-compliance

Non-compliance, or failure to follow the identified treatment plan, is a continuing risk of any person with a chronic disease. Recommended lifestyle changes such as diet, exercise, restricted alcohol intake, stress reduction and smoking cessation often are difficult to maintain on a continuing basis. In addition, prescribed medications may have undesirable effects, whereas hypertension itself often has no symptoms or noticeable effects.

- Inquire about reasons for non-compliance with recommended treatment plan. Listen openly and without judging. *Non-threatening discussion of factors contributing to non-compliance validates the person's self-esteem and partnership in the treatment plan.*

CONSIDERATION FOR PRACTICE

Assess factors contributing to non-compliance, such as adverse drug effects. Suggest measures to manage adverse effects or, if indicated, contact the primary care provider about possible alternative drugs. Some adverse effects of antihypertensive drugs, such as gastric upset, light headedness or nocturia, may be easily managed by changing the timing of the drug dose. Others, such as fatigue, decreased exercise tolerance or impotence, may interfere with lifestyle and life roles to the extent that the person finds them intolerable.

- Evaluate knowledge of hypertension, its long-term effects and treatment. *Provide additional information and reinforce teaching as needed. Knowledge increases the sense of control, which also increases the likelihood of compliance with treatment.*
- Assist to develop realistic short-term goals for lifestyle changes. *Attempting to lose weight, exercise daily, stop smoking and dramatically change the diet all at the same time may be overwhelming, leading to a sense of failure. Smaller, gradual changes are more easily*

incorporated into lifestyle and daily activities, improving compliance.

CONSIDERATION FOR PRACTICE

Work with the person to develop mutual outcomes for the treatment plan. Discuss measures to improve compliance. The person has absolute control over compliance with the treatment plan. Demonstrating respect and involving the person in decision making and planning can improve compliance.

- Help the person identify cues and develop reminders (e.g. written notes, a medication box filled weekly) to assist with maintaining a schedule for exercise and medications. *Cues and other devices provide helpful reminders of activities and schedules until they are incorporated into habits.*
- Reassure the person that relapse into old habits and behaviours is common. Encourage avoiding feelings of guilt associated with relapse and use the circumstance to renew efforts to comply with treatment. *Guilt and feelings of failure can lead to further non-compliance unless the event is used to identify reasons for non-compliance and ways to prevent it from recurring in the future.*

Imbalanced nutrition: more than body requirements

The relationship between obesity, excess alcohol intake and hypertension is well documented. Hypertension is particularly associated with central obesity, identified by waist circumference greater than hip circumference. Although weight loss is difficult and takes commitment to changing eating and exercise habits, it is possible for most people to achieve.

- Assess usual daily food intake and discuss possible contributing factors to excess weight, such as sedentary lifestyle or using food as a reward or stress reliever. Inquire about diversional activities, exercise patterns and previous weight reduction efforts (e.g. participation in weight reduction programs or using fad or crash diets). *Assessment data provide clues about contributing factors to obesity, the person's knowledge base about the relationship between eating and exercise habits and weight, and safe weight loss strategies. This provides direction for further teaching and for developing a realistic weight reduction plan.*
- Mutually determine with the person a realistic target weight (e.g. loss of 10% of current body weight over a 6-month period). Regularly monitor weight. Encourage a system of non-food rewards for achieving small, incremental goals. *Setting weight loss goals helps formalise the process and provides motivation for continued progress. Developing realistic goals may be difficult; unrealistic goals, however, set the person up for failure. Continuous incremental weight loss provides reassurance that it can be achieved and promotes permanent weight reduction.*
- Refer to a dietitian for information about low-fat, low-kilojoule foods and eating plans. Focus on changing eating habits as opposed to 'following a diet'. *Focusing on changing eating habits promotes the sense that low-fat, low-kilojoule eating patterns should become a part of lifestyle, rather than a short-term measure to be endured until the weight loss goal is achieved.*
- Recommend participating in an approved weight loss program such as Weight Watchers. *Organised weight loss programs provide structure for a balanced weight reduction program, as well as mutual support from others trying to lose weight.*

Excess fluid volume

Excess fluid volume often contributes to hypertension by increasing the cardiac output. A number of factors associated with hypertension can cause excess fluid volume, including sodium retention and disruption of the renin–angiotensin–aldosterone system. In addition, some antihypertensive drugs, such as calcium channel blockers and vasodilators, can contribute to excess fluid in the interstitial spaces and peripheral oedema.

- Monitor intake and output and weigh daily (if in an acute or long-term care facility) or weekly (in the community). *Rapid weight changes (over days) more accurately reflect fluid balance than intake and output records. 1 L of fluid weighs 1 kg. Weight changes and intake and output records help monitor the effects of therapy.*
- Monitor for peripheral oedema (sacral oedema in the bedridden person). *Drugs such as vasodilators can cause fluid accumulation in interstitial tissues, leading to peripheral or dependent oedema. Adding a diuretic to the treatment plan may be necessary.*

CONSIDERATION FOR PRACTICE

Monitor blood pressure and other vital signs as indicated: every 1 to 2 hours or more frequently during acute hypertensive states; once a week or more frequently during initial treatment in the community. Vital signs are an indicator of fluid balance and the effectiveness of treatment. An elevated blood pressure, pulse and respiratory rate may indicate fluid retention, whereas orthostatic hypotension and tachycardia may indicate fluid volume deficit.

- Refer to a dietitian for teaching about a restricted sodium diet. Discuss the relationship between sodium intake and fluid retention. Provide opportunities to choose low-sodium foods from simulated menus. Support efforts and reassure that lifestyle changes such as consuming less sodium take time. *Knowledge provides the power to take control of sodium intake. Patience and perseverance are needed to succeed; positive reinforcement of efforts to change long-standing dietary patterns is important.*

CONSIDERATION FOR PRACTICE

Monitor laboratory values, such as urine-specific gravity, urea and creatinine, electrolytes and haematocrit and haemoglobin. Hypertension can alter renal perfusion and function, leading to fluid retention and altered laboratory values. Changes in urea and creatinine indicate impaired renal function, whereas changes in haematocrit and haemoglobin often reflect changes in fluid volume.

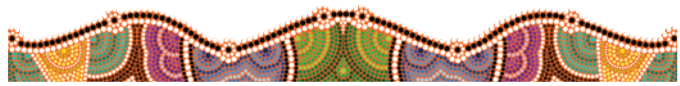
- Discuss the importance of adhering to treatment plans such as dietary restrictions and medication schedules. *Understanding the rationale for treatment measures promotes the person's sense of control and encourages compliance with the treatment regimen.*

Community-based care

Effective control of hypertension requires the person not only to participate in the plan of care, but also to take an active role in managing the disease. Treatment is managed in community settings, with regular visits to a clinic or office to monitor blood pressure and effects of treatment measures. Include the following topics in teachings about hypertension:

- Specific lifestyle changes recommended for the person and suggestions for implementing them. For example:
  - Increase activity gradually. Develop a realistic exercise program that is enjoyable and fits into lifestyle. Identify an exercise buddy for additional motivation. Activity and exercise, through a gradual conditioning of muscles and blood vessels, lower blood pressure by reducing peripheral vascular resistance. As the heart becomes conditioned and pumps more efficiently, kidney perfusion improves and intravascular volume falls, further reducing blood pressure. Exercise also reduces stress and contributes to weight loss and maintenance. Aerobic exercise, such as walking, jogging, swimming and cycling, are appropriate; isometric activities (such as weight lifting) should be avoided without doctor's approval.
  - Adopt healthy eating patterns, following a low-fat, low-cholesterol, moderate-sodium diet that also is rich in fruit and vegetables and includes at least two servings of low-fat milk or milk products daily. Do not give up if you slip into old eating habits on occasion; use such occasions to identify ways to avoid future lapses.
  - Stop smoking. Participating in organised smoking cessation programs or using aids such as nicotine patches can help.
  - Use alcohol in moderation if at all, consuming no more than 10–20 g of alcohol per day.
  - Use stress-reducing techniques such as meditation, relaxation, deep breathing and exercise to manage stress. Anger and hostility intensify vasoconstriction; channelling these emotions into more positive responses such as using a change process to modify factors that provoke these emotions can reduce their harmful effects on blood pressure.
- Prescribed medications, their intended effect, dose and timing, interactions and possible adverse effects. Discuss effects that should be reported to the doctor and those that can be managed by the person or that will diminish over time.
- The importance of monitoring blood pressure and regular visits to the primary care provider or hypertension clinic to monitor treatment. During follow-up visits, assess the blood pressure and specific laboratory work (such as serum urea, creatinine and electrolytes) to evaluate the disease and the effects of antihypertensive medications.

Refer the person to community blood pressure clinics and to home health services as needed for regular follow-up and reinforcement of teaching. Refer to a dietitian or to an organised weight loss program as indicated for further teaching and weight loss support. The 'Nursing care plan' below provides additional information about community-based care for the person with high blood pressure.



SECONDARY HYPERTENSION

Secondary hypertension is elevated blood pressure resulting from an identifiable underlying process. Prevalence is poorly documented but is often reported as between 5% and 10% of identified cases of hypertension (Sperati & Whaley-Connell, 2015). Kidney disease is the most common identifiable cause of high blood pressure in both adults and children. Other common identifiable causes of hypertension in adults include renovascular disease (reduced blood flow to the kidneys), disorders of the adrenal cortex, pheochromocytoma, coarctation of the aorta and sleep apnoea (O'Callaghan, Goh & Rong, 2013). The pathophysiology of selected causes of high blood pressure are summarised here:

- *Kidney disease*. Any disease that affects renal blood flow (e.g. renal artery stenosis) or renal function (e.g. glomerulonephritis, renal failure) can lead to hypertension. Disruption of the blood supply stimulates the renin–angiotensin–aldosterone system, with resulting vasoconstriction and sodium and water retention. Altered kidney function affects the elimination of water and electrolytes, leading to hypertension.
- *Coarctation of the aorta*. Coarctation of the aorta is narrowing of the aorta, usually just distal to the subclavian arteries. Reduced renal and peripheral blood flow stimulates the renin–angiotensin–aldosterone system and local vasoconstrictive responses, raising the blood pressure. A marked difference between pressures in the upper and lower extremities is common, with weak pulses and poor capillary refill in the lower extremities.
- *Endocrine disorders*. Adrenal gland disorders such as Cushing's syndrome and primary aldosteronism can cause hypertension. A rare tumour of the adrenal medulla, *pheochromocytoma*, causes persistent or intermittent hypertension. Other endocrine disorders such as hyperthyroidism and pituitary disorders also can lead to hypertension.
- *Neurological disorders*. Increased intracranial pressure causes an elevated blood pressure as the body attempts to maintain cerebral blood flow. Disorders that interfere with autonomic nervous system regulation (such as high spinal cord injury) may allow the sympathetic nervous system to predominate, increasing systemic vascular resistance and blood pressure.
- *Drug use*. Oestrogen and oral contraceptive use may lead to hypertension, possibly by prompting sodium and water retention and affecting the renin–angiotensin–aldosterone

NURSING CARE PLAN A person with hypertension

Margaret Simpson is a married, 49-year-old Anglo-Australian with six children whose ages range from 6 to 16 years. For the past 2 months, Mrs Simpson has had frequent morning headaches and occasional dizziness and blurred vision. At her annual health check 1 month ago, her blood pressure was 168/104 mmHg and 156/94 mmHg. She was instructed to reduce her fat and cholesterol intake, to avoid using salt at the table and to start walking for 30 to 45 minutes daily. Mrs Simpson returns to the clinic for follow up.

ASSESSMENT

While escorting Mrs Simpson to the exam room and obtaining her weight, blood pressure and history, Lisa Christos, RN, notices that Mrs Simpson seems restless and upset. Ms Christos says, 'You look upset about something. Is everything OK?' Mrs Simpson responds, 'Well, my head is throbbing and I'm sort of dizzy. I think I'm just overdoing it and not getting enough rest. You know, raising six children is a lot of work and expense. I just started working part time so we wouldn't get behind in our bills. I thought the extra money might relieve some of my stress, but I'm not so sure that's really happening. I'm not getting any better and I'm worried that I'll lose my job or become disabled and that my husband won't be able to manage the children by himself. I really need to go home, but first, I want to get rid of this awful headache. Would you please get me a couple of aspirin or something?'

Mrs Simpson's history shows a steady weight gain during the past 18 years. She has no known family history of hypertension. Physical findings include height 160 cm, weight 102 kg, T 37.2°C, P 100 regular, R 16, BP 180/115 mmHg (lying), 170/110 mmHg (sitting), 165/105mmHg (standing), average 10 point difference in readings between right and left arm (lower on left). Skin cool and dry, capillary refill 4 seconds right hand, 3 seconds left hand. Mrs Simpson's total serum cholesterol is 6.33 mmol/L (normal < 5.2 mmol/L). All other blood and urine studies are within normal limits. Based on analysis of the data, Mrs Simpson is started on enalapril 5 mg and placed on a low-fat, low-cholesterol, no-added-salt diet.

DIAGNOSES

- *Fatigue* related to effects of hypertension and stresses of daily life.
- *Imbalanced nutrition: more than body requirements* related to excessive food intake.
- *Ineffective health maintenance* related to inability to modify lifestyle.
- *Deficient knowledge* related to effects of prescribed treatment.

PLANNING

- Discuss strategies for achieving a realistic weight loss goal.
- Refer for a dietary consultation for further teaching about fat and sodium restrictions.
- Discuss stress-reducing techniques, helping identify possible choices.

Expected outcomes

- Reduce blood pressure readings to less than 150 systolic and 90 diastolic by return visit next week.
- Incorporate low-sodium and low-fat foods from a list provided into her diet.
- Develop a plan for regular exercise.
- Verbalise understanding of the effects of prescribed drug, dietary restrictions, exercise and follow-up visits to help control hypertension.

IMPLEMENTATION

- Teach to take own blood pressure daily and record it, bringing the record to scheduled clinic visits.
- Teach name, dose, action and side effects of antihypertensive medication.
- Instruct to walk for 15 minutes each day this week and to investigate swimming classes at the local pool.

EVALUATION

Mrs Simpson returns to the clinic 1 week later. Her average blood pressure is now 148/88 mmHg. She has lost 1 kg and states that her oldest daughter has suggested that they join a weight reduction program together. Mrs Simpson is walking for an average of 20 minutes each day. She verbalises an understanding of her medication and is taking it in the morning and before dinner each day. She met with the dietitian and discussed ways to reduce the sodium and fat in her diet. The dietitian provided a list of low-fat, low-sodium foods and recommended cookbooks to help Mrs Simpson modify her cooking. Mrs Simpson tells Ms Christos, 'I just can't believe how much better I feel already. My headaches are gone and I've actually lost some weight—and I feel motivated to keep going. If I had only known how much better I could feel! I don't expect I'll ever go back to my old habits again; it's just not worth it!'

CRITICAL THINKING IN THE NURSING PROCESS

1. Identify the factors that contributed to Mrs Simpson's hypertension. Which were modifiable and which were not?
2. What is the rationale for reducing sodium and fat in Mrs Simpson's diet?
3. Suppose that a hypertensive person is homeless and has no source of income. How could you help ensure that this person would follow the treatment plan? What would you do if the person did not follow it?
4. Discuss the role of stress in hypertension. What factors in Mrs Simpson's life contribute to her stress level?
5. Develop a plan of care for the nursing diagnosis *Low self-esteem* related to obesity.

REFLECTION ON THE NURSING PROCESS

1. Listening to people is an essential quality and skill. Discuss which essential cues in Mrs Simpson's presentation lead the nurse to question her further.
2. What health promotion strategies/programs could be advised for Mrs Simpson to engage in and how are they relevant to her presenting condition?

system. Stimulant drugs, such as cocaine and methamphetamines, increase systemic vascular resistance and cardiac output, resulting in hypertension.

- *Pregnancy*. About 10% of all pregnant women are hypertensive. Hypertension may pre-date pregnancy or occur as a direct response to the pregnancy. The mechanism of pregnancy-induced hypertension (PIH) is unclear. It is a significant cause of maternal and foetal morbidity and mortality and requires careful perinatal management.

The pattern of secondary hypertension varies, depending on its cause. Pheochromocytoma may cause attacks of hypertension that last for minutes to hours, accompanied by anxiety, palpitations, diaphoresis, pallor and nausea and vomiting. Primary aldosteronism may cause hypertension, weakness, paraesthesias, polyuria and nocturia (see Chapter 17). Symptoms of kidney disease accompany hypertension when a renal disorder is the cause.

The following diagnostic tests may be ordered to differentiate primary from secondary hypertension:

- *Renal function studies* and *urinalysis* to identify renal causes of hypertension. Elevated serum urea and creatinine, haematuria, proteinuria and casts often indicate kidney disease.
- *Serum potassium* is decreased in hyperaldosteronism.
- *Blood chemistries*, including serum electrolytes, glucose and lipid studies, are done to detect abnormalities indicative of endocrine or cardiovascular disease.
- *Intravenous pyelography (IVP), renal ultrasonography*, *renal arteriography* and *CT* or *MRI* may be done when secondary hypertension is suspected.

Interprofessional and nursing care for the person with secondary hypertension is the same as that for primary hypertension, discussed in the previous section. In addition, the underlying process is treated. See chapters covering specific disorders for more information about treatment measures.

HYPERTENSIVE CRISIS

Some people with hypertension may, for reasons not clearly understood, develop rapid, significant elevations in systolic and/or diastolic pressures. In a *hypertensive emergency* (or *malignant hypertension*), the systolic pressure is greater than 180 mmHg and the diastolic pressure is higher than 120 mmHg. Immediate treatment (within 1 hour) is vital to prevent cardiac, renal and vascular damage, and reduce morbidity and mortality. Intense cerebral artery spasms help protect the brain from excess pressure; however, cerebral oedema often develops. Prolonged severe hypertension damages the walls of the arterioles and renal blood vessels and may lead to intravascular coagulation and acute renal failure.

People presenting with a hypertensive emergency may have manifestations such as headache, confusion, swelling of the optic nerve (papilloedema), blurred vision, restlessness and motor and sensory deficits. Manifestations of hypertensive emergencies are listed in the box below.

Most hypertensive emergencies occur when people suddenly stop taking their medications or their hypertension is poorly controlled. Younger people (30 to 50 years old), Indigenous Australian men, pregnant women with pre-eclampsia and people with collagen and/or renal disease also are at higher risk of a hypertensive emergency (NHF, 2010).

The goal of care in hypertensive emergencies is to reduce the blood pressure by no more than 25% within minutes to 1 hour, then towards 160/100 within 2 to 6 hours. It is important to avoid rapid or excessive blood pressure decreases that may lead to renal, cerebral or cardiac ischaemia (Ventura & Reddy, 2014). Blood pressure is monitored frequently (every 5 to 30 minutes) during a hypertensive emergency. The serum urea, creatinine, calcium and total protein levels are carefully

MANIFESTATIONS Hypertensive emergencies

- Rapid onset
- Blurred vision, papilloedema
- Systolic pressure > 180 mmHg
- Diastolic pressure > 120 mmHg
- Headache
- Confusion
- Motor and sensory deficits

TABLE 31.2 Intravenous drugs used to treat hypertensive emergencies

DRUG	ONSET	DURATION	NURSING IMPLICATIONS
Sodium nitroprusside dihydrate	Seconds	1 to 2 min	• Effective, easy to titrate • May cause nausea, vomiting, muscle twitching, sweating • Use with caution in increased intracranial pressure
Glyceryl trinitrate	2 to 5 min	5 to 10 min	• Used when coronary ischaemia accompanies hypertension • May cause headache, vomiting • Tolerance may develop with prolonged use
Diazoxide	1 to 2 min	4 to 24 h	• Avoided in people with coronary artery disease • Used with beta-blockers and diuretics • Painful if it enters tissues
Hydralazine (Apresoline)	10 to 30 min	2 to 6 h	• May be used for hypertension associated with eclampsia • Avoided in people with CHD • May cause tachycardia, flushing, headache, vomiting, angina

monitored to help determine the prognosis for recovery. Drug treatment for malignant hypertension includes parenteral administration of a rapidly acting antihypertensive, such as the potent vasodilator sodium nitroprusside. Other vasodilating agents that may be used are outlined in Table 31.2. Management also focuses on treating any underlying or coexisting heart, kidney and CNS disorders.

Nursing care for people with a hypertensive emergency focuses on continuous monitoring of the blood pressure and titrating drugs (administered by intravenous bolus or infusion) as ordered to achieve desired blood pressure. Avoiding excessive or very rapid blood pressure reductions is as important as achieving the desired blood pressure readings. Reassure the person and family of the rapid effect of prescribed drugs. Provide psychological and emotional support as needed. Maintain an attitude of confidence that the treatment will achieve the desired effect. Following resolution of the hypertensive crisis, review causes of the crisis. Teach the person and family measures to effectively manage hypertension and prevent future hypertensive emergencies.

DISORDERS OF THE AORTA AND ITS BRANCHES

The aorta and its branches may be affected by occlusions, aneurysms and inflammations. These disorders may be chronic or acute and life threatening (e.g. a thoracic dissection). This section focuses on aneurysms of the aorta and its branches.

THE PERSON WITH AN ANEURYSM

An **aneurysm** is an abnormal dilation of a blood vessel, commonly at a site of a weakness or tear in the vessel wall. Aneurysms commonly affect the aorta and peripheral arteries because of the high pressure in these vessels. An aneurysm also may develop in the ventricular wall, usually affecting the left ventricle. Most arterial aneurysms are caused by arteriosclerosis or atherosclerosis; trauma also may lead to aneurysm formation.

Arterial aneurysms are most common in men over age 50, most of whom are asymptomatic at the time of diagnosis. Hypertension is a major contributing factor in the development of some types of aortic aneurysms.

FAST FACTS

- The incidence of aortic aneurysm-related deaths for males in Australia in 2013 was 573.
- The incidence of aortic aneurysm-related deaths for females in Australia in 2013 was 388.
- Aortic aneurysm and dissection in 2013 accounted for 0.65% of all deaths in Australia.

Source: ABS, 2015.

Pathophysiology and manifestations

Aneurysms form due to weakness of the arterial wall. The main structural proteins of the aorta are collagen and elastin. Collagen provides tensile strength of the vessel, preventing excessive dilation. Elastin allows vessel recoil, during which the vessel returns to its original size following systole. This recoil provides continued propulsion of the bolus of blood expelled from the ventricle. Elastin is a primary component of internal elastic lamina, which separates the intimal and medial layers of the aorta and of the media, the smooth muscle layer of the aorta. Destruction of elastin can lead to abnormal dilation of the vessel; collagen destruction can allow the vessel to rupture (Tseng, 2014).

True aneurysms are caused by slow weakening of the arterial wall due to the long-term, eroding effects of atherosclerosis and hypertension. True aneurysms affect all three layers of the vessel wall and most are fusiform and circumferential. *Fusiform aneurysms* are spindle shaped and taper at both ends. Circumferential aneurysms involve the entire diameter of the vessel (see Figure 31.7). They generally grow slowly but progressively. Their length and diameter vary considerably among people. A large fusiform aneurysm may affect most of the ascending aorta as well as a large portion of the abdominal aorta.

False aneurysms, also known as traumatic aneurysms, are caused by a traumatic break in the vessel wall rather than weakening of the vessel. They often are *saccular*, shaped like small outpouchings (sacs) on a portion of the vessel wall (see

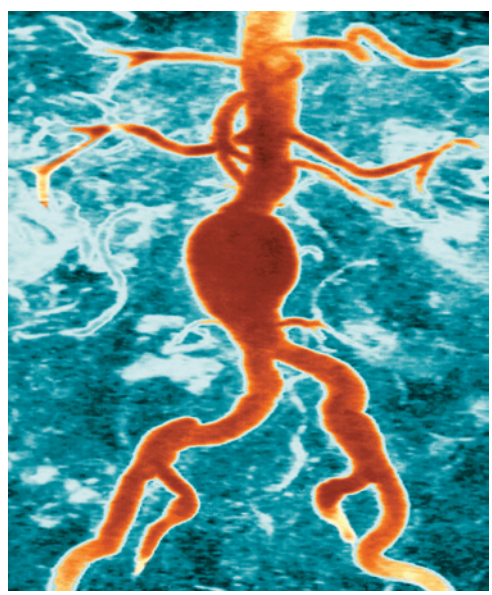


FIGURE 31.7 ■ A magnetic resonance angiogram (MRA) showing a circumferential aneurysm of the lower abdominal aorta

Source: Zephyr/Science Source.

Figure 31.8). A *berry aneurysm* is a type of saccular aneurysm. They are often small (less than 2 cm in diameter), caused by congenital weakness in the tunica media of the artery. Berry aneurysms are commonly found in the circle of Willis in the brain.

Dissecting aneurysms are unique, developing when a break or tear in the tunica intima and media allows blood to invade or *dissect* the layers of the vessel wall. The blood usually is contained by the adventitia, forming a saccular or longitudinal aneurysm.

Aneurysms affect different segments of the aorta and its branches. Their manifestations generally are due to pressure of the aneurysm on adjacent structures. Table 31.3 summarises the manifestations and complications of various types of aortic aneurysms.

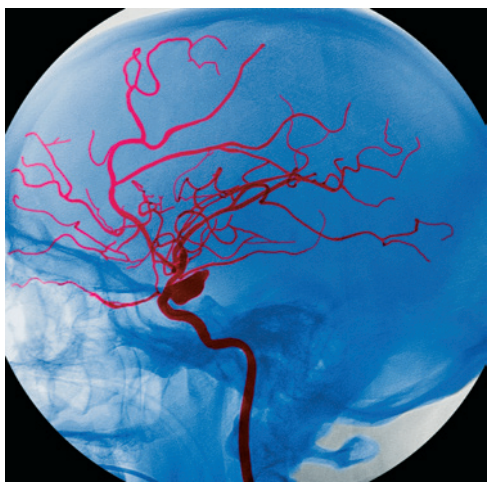


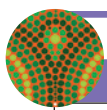
FIGURE 31.8 ■ An angiogram showing a saccular (berry) aneurysm in the carotid artery of a 50-year-old man

Source: Simon Fraser/RNC, Newcastle upon Tyne/Science Source.

Thoracic aortic aneurysms

*Thoracic aortic aneurysm*s (see Figure 31.9) account for about 10% of aortic aneurysms, with an annual incidence of about 6 per 100 000 people. They usually result from weakening of the aortic wall by arteriosclerosis and hypertension. Other causes include trauma, coarctation of the aorta, tertiary syphilis, fungal infections and Marfan syndrome. The syphilis spirochete can invade and weaken aortic smooth muscle, causing an aneurysm to develop as long as 20 years after the primary infection. Marfan syndrome fragments elastic fibres of the aortic media, weakening the vessel wall. The box below discusses genetic links associated with thoracic aortic aneurysms.

Thoracic aneurysms frequently are asymptomatic. When present, manifestations are caused by the effects of the aneurysm on blood flow (to the coronary arteries and great vessels of the head and upper body) and pressure placed by distended aorta on surrounding structures. Consequently, manifestations vary by the location, size and growth rate of the aneurysm. Substernal, neck or back pain may occur. Pressure on the



GENETIC CONSIDERATIONS
Thoracic aortic aneurysms

About 15–25% of people with aortic aneurysms have a family history of the disorder (Gong et al., 2015; Rahimi, 2014). A condition known as *cystic medial necrosis* is prevalent in people with Marfan syndrome and Ehlers–Danlos syndrome, inherited disorders involving connective tissues. In cystic medial necrosis, collagen and elastic fibres of the tunica media of the aorta degenerate. This loss of collagen and elastic tissues weakens the wall of the proximal aorta, leading to circumferential dilation of the ascending aorta and development of a fusiform aneurysm.

TABLE 31.3 Manifestations and complications of aortic aneurysms

TYPE OR LOCATION	MANIFESTATIONS	COMPLICATIONS
Thoracic	• May be asymptomatic • Back, neck or substernal pain • Dyspnoea, stridor or brassy cough if pressing on trachea • Hoarseness and dysphagia if pressing on oesophagus or laryngeal nerve • Oedema of the face and neck • Distended neck veins	• Rupture and haemorrhage
Abdominal	• Pulsating abdominal mass • Aortic calcification noted on x-ray • Mild to severe midabdominal or lumbar back pain • Cool, cyanotic extremities if iliac arteries are involved • Claudication (ischaemic pain with exercise, relieved by rest)	• Peripheral emboli to lower extremities • Rupture and haemorrhage
Aortic dissection	• Abrupt, severe, ripping or tearing pain in area of aneurysm • Mild or marked hypertension early • Weak or absent pulses and blood pressure in upper extremities • Syncope	• Haemorrhage • Kidney failure • MI, heart failure, cardiac tamponade • Sepsis • Weakness or paralysis of lower extremities

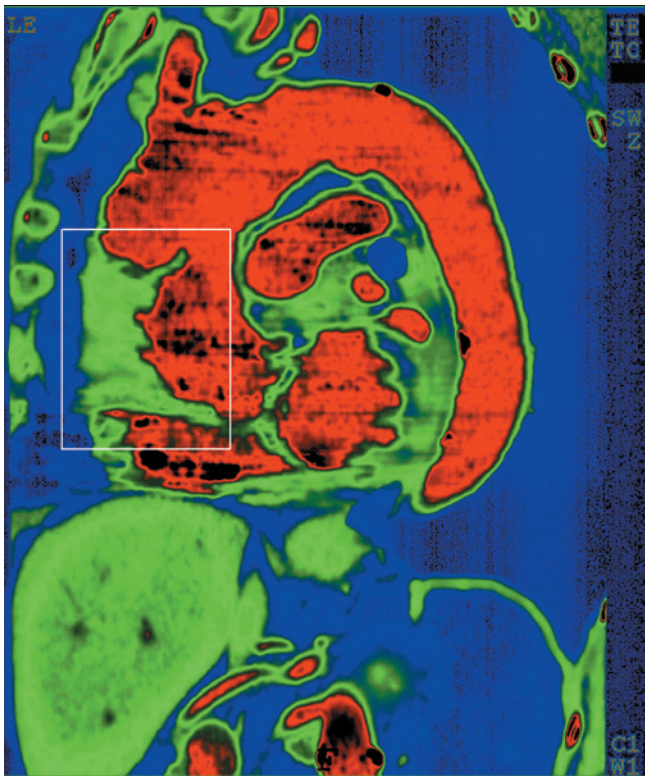


FIGURE 31.9 ■ In an aortic dissection, a tear in the intima and media of the artery allows blood to invade and dissect the wall of the aorta

Source: J. Cavallini/Custom Medical Stock Photo.

trachea, oesophagus, laryngeal nerve or superior vena cava may cause dyspnoea, stridor, cough, difficult or painful swallowing, hoarseness, oedema of the face and neck, and distended neck veins.

Aneurysms of the ascending aorta typically cause angina due to disruption of blood flow into the coronary arteries. Heart failure may develop as a result of disruption of the aortic valve and regurgitation of blood back into the left ventricle. Aneurysms of the aortic arch often cause dysphagia, dyspnoea, hoarseness, confusion and dizziness (due to disrupted cerebral blood flow). Thrombi that form within a thoracic aneurysm can embolise, causing a stroke, renal or mesenteric ischaemia, or ischaemia of the lower extremities. Aneurysms of the thoracic aorta tend to enlarge progressively and may rupture, causing death (Tseng, 2014).

Abdominal aortic aneurysms

Abdominal aortic aneurysms are associated with arteriosclerosis and hypertension. Increasing age and smoking are believed to contribute as well. Most abdominal aortic aneurysms are found in adults over age 70. The vast majority (over 90%) develop below the renal arteries, usually where the abdominal aorta branches to form the iliac arteries (Rahimi, 2014).

Most abdominal aneurysms are asymptomatic, but a pulsating mass in the mid and upper abdomen and a bruit over the mass are found on exam. When pain is present, it may be constant or intermittent, usually felt in the midabdominal region or lower back. Its intensity may range from mild discomfort to severe pain. Pain intensity often correlates with the size and severity of the aneurysm. Severe pain may indicate impending rupture.

Sluggish blood flow within the aneurysm may cause thrombi (blood clots) to form. These can become emboli (circulating clots), travelling to the lower extremities and occluding peripheral arteries. Aneurysms generally enlarge by approximately 0.2–0.8 mm/year; eventually they may rupture, resulting in haemorrhage and hypovolaemic shock. After acute rupture, the mortality rate is greater than 65%, even when emergency surgery is performed (Rahimi, 2014).

Popliteal and femoral aneurysms

Most popliteal and femoral aneurysms are due to arteriosclerosis. They are often bilateral and usually affect men.

Popliteal aneurysms (see Figure 31.10) may be asymptomatic. Manifestations, if any, are due to decreased blood flow to the lower extremity and include intermittent **claudication** (cramping or pain in the leg muscles brought on by exercise and relieved by rest), rest pain and numbness. A pulsating mass may be palpable in the popliteal fossa (behind the knee). Thrombosis and embolism are complications; gangrene may result, often necessitating amputation.

A *femoral aneurysm* usually is detected as a pulsating mass in the femoral area. The manifestations are similar to those of popliteal aneurysms, resulting from impaired blood flow. Femoral aneurysms may rupture.

Aortic dissections

Dissection is a life-threatening emergency caused by a tear in the intima of the aorta with haemorrhage into the media.

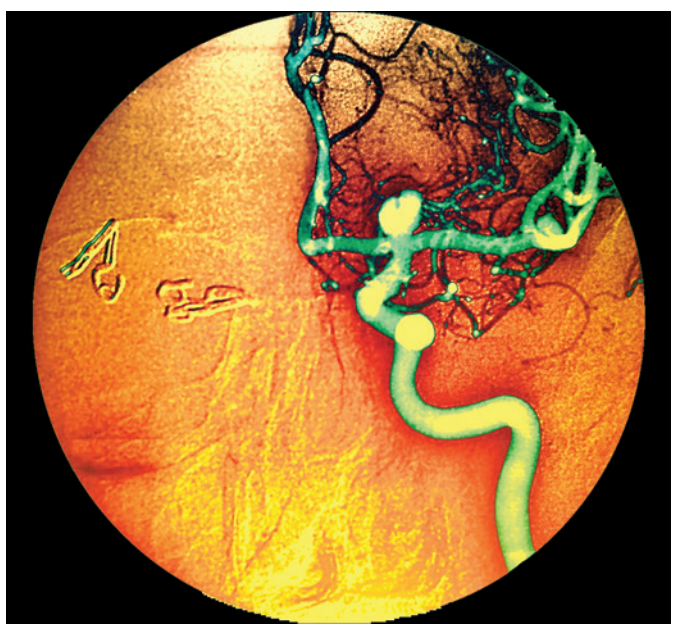


FIGURE 31.10 ■ An angiogram showing several popliteal aneurysms

Source: Zephyr/Science Source.

The haemorrhage dissects or splits the vessel wall, forming a blood-filled channel between its layers. Dissection can occur anywhere along the aorta. *Type A dissection* (also called *proximal dissection*) affects the ascending aorta; *type B dissection* (*distal dissection*) is limited to the descending aorta.

Hypertension is a major predisposing factor for aortic dissection, accounting for 70% of aortic dissections. Cystic medial necrosis (see the 'Genetic considerations' box above) also is a major risk factor. Other risk factors include male gender, advancing age, pregnancy, congenital defects of the aortic valve, coarctation of the aorta and inflammatory aortitis (Mancini, 2014).

Dissection of the thoracic aortic walls progresses along the length of the vessel, moving both proximally and distally. As the aneurysm expands, pressure may prevent the aortic valve from closing or may occlude the branches of the aorta. Descending aortic dissection may extend into the renal, iliac or femoral arteries.

The primary symptom of an aortic dissection is sudden, excruciating pain. The pain, often described as a ripping or tearing sensation, is usually over the area of dissection. Thoracic dissections cause chest or back pain. Other symptoms may include syncope, dyspnoea and weakness. The blood pressure may initially be increased, but rapidly falls and is often inaudible as the dissection occludes blood flow. Peripheral pulses are absent for the same reason.

Complications develop if major arteries are affected. Obstruction of the carotid artery causes neurological symptoms such as weakness or paralysis. The myocardium, kidneys or bowel may become ischaemic or infarct if blood flow to the coronary arteries, renal arteries or mesenteric artery is affected. Acute aortic regurgitation may develop with dissection of the ascending aorta. With treatment, the long-term prognosis is generally good, although the in-hospital mortality rate with medical management is approximately 60% and following surgery remains quite high at 30% (Mancini, 2014).

INTERPROFESSIONAL CARE

Most aneurysms are asymptomatic, detected through a routine physical examination. Treatment depends on the size of the aneurysm. Small, asymptomatic aneurysms may not be treated or are medically managed; large aneurysms (> 5 cm) at risk of rupture require surgery.

Diagnosis

Diagnostic studies done to establish the diagnosis and determine the size and location of the aneurysm may include:

- *chest x-ray* to visualise thoracic aortic aneurysms
- *abdominal ultrasonography* to diagnose abdominal aortic aneurysms
- *transoesophageal echocardiography* to identify the specific location and extent of a thoracic aneurysm and to visualise a dissecting aneurysm
- *contrast-enhanced CT* or *MRI* allows precise measurements of aneurysm size
- *angiography* uses contrast solution injected into the aorta or involved vessel to visualise the precise size and location of the aneurysm.

Medications

Thoracic aortic aneurysms may be treated with long-term beta-blocker therapy and additional antihypertensive drugs as needed to control heart rate and blood pressure.

People with aortic dissection are initially treated with intravenous beta-blockers to reduce the heart rate to about 60 bpm. Sodium nitroprusside infusion is started concurrently to reduce the systolic pressure to 120 mmHg or less. Calcium channel blockers also may be used. Constant monitoring of vital signs, haemodynamic pressures (via Swan–Ganz catheter; see Chapter 30 for more information about haemodynamic pressure monitoring) and urine output is vital to ensure adequate perfusion of vital organs.

Following surgical correction of an aneurysm, anticoagulant therapy may be initiated. Heparin therapy is used initially, with conversion to oral anticoagulation prior to discharge. Many people are maintained indefinitely on anticoagulant therapy; others may use lifelong, low-dose aspirin therapy to reduce the risk of clot formation.

Surgery

Operative repair of aortic aneurysms is indicated when the aneurysm is symptomatic or expanding rapidly. Ascending thoracic aneurysms of more than 5.5 cm and descending thoracic aneurysms of 6.5cm or more should be surgically repaired; asymptomatic abdominal aneurysms greater than 5 cm in diameter may be repaired, depending on the person's operative risk factors. It is recommended that individuals with Marfan syndrome or familial aneurysms should be repaired at 0.5 cm or sooner (Tseng 2014).

Endovascular aneurysm repairs (EVAR) are increasingly used to treat abdominal and thoracic aortic aneurysms. The use of EVAR to treat aortic dissections is in investigational stages. The stent, which consists of a metal sheath covered with polyester fabric or a woven polyester tube, usually is placed percutaneously via the femoral artery. Fluoroscopy is used to guide its placement. Both straight and bifurcated grafts are available. Endovascular stent placement results in a shorter hospital stay and lower treatment cost. EVAR is associated with fewer pulmonary, renal and cardiovascular complications than open surgical aneurysm repair in individuals as a planned treatment option prior to rupture. However, a recent Cochrane Review identified that there is currently no reliable evidence to suggest improvement in survival rate or reduction of late complications when using the endoscopic approach for individuals once the aorta is ruptured (Badger et al., 2014). The most common complication of endovascular aneurysm repair is persistent perfusion of the aneurysm (*endoleak*) caused by an ineffective seal at the proximal or distal end of the graft. Regular follow up with abdominal CT scans is necessary to detect this complication, which can develop at any time postoperatively. Numerous techniques exist to successfully manage endoleak, although there is no current evidence of any particular technique's superiority (Millen et al., 2015). On rare

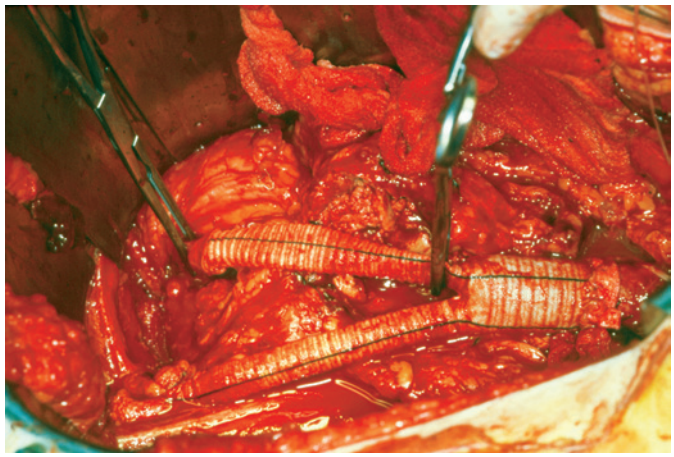


FIGURE 31.11 ■ Repair of an abdominal aortic aneurysm. The aorta is exposed and clamped between the renal and iliac arteries. Atherosclerotic plaque and thrombotic material are removed. A synthetic graft is used to replace the aneurysm. The aneurysm walls are then sutured around the graft

Source: Stevie Grand/Science Source.

occasions, the graft may be malpositioned or may migrate from the desired location.

An open surgical procedure in which the aneurysm is excised and replaced with a synthetic fabric graft is the standard treatment for expanding abdominal aortic aneurysms (see Figure 31.11). Although the aneurysm walls may be excised, they usually are left intact and used to cover the graft. Surgical repair of thoracic aneurysms is similar but more complex due to major vessels exiting at the aortic arch. Cardiopulmonary bypass is required if the ascending aorta is involved. The aortic valve also may be replaced during surgery. See the following box for nursing care of the person having surgery of the aorta.

Nursing care

Assessment

Focused assessment for the person with a suspected aortic aneurysm includes:

- *Health history:* complaints of chest, back or abdominal pain; extremity weakness; shortness of breath, cough, difficult or painful swallowing, hoarseness; history of hypertension, coronary heart disease, heart failure or peripheral vascular disease.
- *Physical examination:* vital signs, including blood pressure in upper and lower extremities; peripheral pulses; skin colour and temperature; neck veins; abdominal exam, including gentle palpation for masses and auscultation for bruits; neurological exam, including level of consciousness (LOC), sensation and movement of extremities.

Nursing diagnoses and interventions

Nursing care for people with an aneurysm of the aorta or its branches focuses on monitoring and maintaining tissue perfusion, relieving pain and reducing anxiety. Nursing care usually is acute, precipitated by a complication or surgical repair of the aneurysm.

Risk of ineffective tissue perfusion

People with aortic aneurysms are at risk of impaired tissue perfusion due to aneurysm rupture with resulting haemorrhage and lack of blood flow to tissues distal to the rupture. In addition, thrombi often form within the aneurysm and may become emboli, obstructing distal arterial blood flow.

CONSIDERATION FOR PRACTICE

Immediately report manifestations of impending rupture, expansion or dissection of the aneurysm: increased pain; discrepancy between upper and lower extremity blood pressures and peripheral pulses; increased mass size; change in LOC or motor or sensory function; laboratory results. Rapid expansion may indicate increased risk of rupture, with resulting haemorrhage, shock and possible death. Elective or planned surgery may rapidly become emergency surgery to prevent complications.

- Implement interventions to reduce the risk of aneurysm rupture:
  a. Maintain bed rest with legs flat.
  b. Maintain a calm environment, implementing measures to reduce psychological stress.
  c. Prevent straining during defecation and instruct to avoid holding the breath while moving.
  d. Administer beta-blockers and antihypertensives as prescribed.

  Activity, stress and the Valsalva manoeuvre increase blood pressure, increasing the risk of rupture. Elevating or crossing the legs restricts peripheral blood flow and increases pressure in the aorta or iliac arteries. Beta-blockers and antihypertensives often are ordered to reduce pressure in the dilated vessel.

CONSIDERATION FOR PRACTICE

Report manifestations of arterial thrombosis or embolism: absent peripheral pulses; a pale or cyanotic, cool extremity; severe, diffuse abdominal pain with guarding; or increased groin, lumbar or lower extremity pain. Sluggish blood flow within the aneurysm often causes thrombi to form. These thrombi can break loose, becoming emboli that can occlude peripheral arteries or arteries to the kidneys or mesentery. Arterial occlusion may necessitate emergency surgery to restore blood flow and prevent tissue infarct or gangrene.

- Continuously monitor cardiac rhythm. Report complaints of chest pain or changes in ECG tracing. Administer oxygen as indicated. *Aortic dissection and repair place the person at significant risk of MI, a major cause of postoperative mortality and morbidity (Kasper et al., 2015).*

NURSING CARE OF THE PERSON having surgery of the aorta

PREOPERATIVE CARE

- As time permits, provide routine preoperative care and teaching as outlined in Chapter 3. *People having vascular surgery have similar preoperative nursing care needs to other people having major abdominal or thoracic surgery. If emergency surgery is required, time for preoperative care and teaching may be limited.*
- Implement measures to reduce fear and anxiety:
  a. Orient to the intensive care unit, if appropriate.
  b. Describe and explain the reason for all equipment and tubes, such as cardiac monitors, ventilators, nasogastric tubes, urinary catheters, intravenous lines and fluids, and intra-arterial lines.
  c. Explain what to expect following surgery (sights, sounds, frequency of taking vital signs, dressings, pain relief measures, communication strategies).
  d. Allow time for questions and expression of fears and concerns.

  These explanations provide a sense of control for the person and family.
- Monitor for and implement care to reduce the risk of aneurysm rupture (see the following section). *People with a rapidly expanding or symptomatic aneurysm are at risk of rupture prior to surgical repair.*

POSTOPERATIVE CARE

- Provide routine postoperative care and specific measures as ordered by the doctor. *People undergoing aneurysm repair require nursing care similar to that provided to all people with major thoracic or abdominal surgery, in addition to specific measures related to vascular surgery.*
- Maintain fluid replacement and blood or volume expanders as ordered. Promptly report changes in vital signs, level of consciousness and urine output. *Hypovolaemic shock may develop due to blood loss during surgery, third spacing, inadequate fluid replacement, and/or haemorrhage if graft separation or leakage occurs.*

CONSIDERATION FOR PRACTICE

Monitor for and report manifestations of graft leakage:

a. ecchymoses of the scrotum, perineum or penis; a new or expanding haematoma
b. increased abdominal girth
c. weak or absent peripheral pulses; tachycardia; hypotension
d. decreased motor function or sensation in the extremities
e. fall in haemoglobin and haematocrit
f. increasing abdominal, pelvic, back or groin pain
g. decreasing urinary output (less than 30 mL/h)
h. decreasing CVP, pulmonary artery pressure or pulmonary artery wedge pressure.

These manifestations may signal graft leakage and possible haemorrhage. Pain may be due to pressure from an expanding haematoma or bowel ischaemia. Decreased renal perfusion causes the glomerular filtration rate and urine output to fall.

- Report manifestations of lower extremity embolism: pain and numbness in lower extremities, decreasing pulses and pale, cool or cyanotic skin. *Pulses may be absent for 4 to 12 hours postoperatively due to vasospasm; however, absent pulses with pain, changes in sensation and a pale, cool extremity are indicative of arterial occlusion.*
- Report manifestations of bowel ischaemia or gangrene: abdominal pain and distension, occult or fresh blood in stools, and diarrhoea. *Bowel ischaemia may result from an embolism or occur as a complication of surgery.*
- Report manifestations of impaired renal function: urine output less than 30 mL/h, fixed specific gravity, increasing serum urea and creatinine levels. *Hypovolaemia or clamping of the aorta during surgery may impair renal perfusion, leading to acute renal failure.*
- Report manifestations of spinal cord ischaemia: lower extremity weakness or paraplegia. *Impaired spinal cord perfusion may lead to ischaemia and impaired function.*

Rapid identification and treatment of this complication can reduce the risk of death or long-term adverse effects of MI.

CONSIDERATION FOR PRACTICE

Immediately report changes in mental status or symptoms of peripheral neurological impairment (weakness, paraesthesias, paralysis). The expanding aneurysm or dissection can affect carotid and cerebral blood flow or spinal cord perfusion, leading to neurological symptoms. Immediate restoration of blood flow is vital to prevent permanent neurological deficits.

Risk of injury

Potent antihypertensive drugs often are given intravenously to reduce the pressure on an expanding or dissecting aneurysm. Continuous monitoring of infusions and haemodynamic parameters such as arterial pressure, pulmonary pressures and cardiac output is vital to ensure that adequate tissue perfusion is maintained during infusions of these potent drugs.

- Continuously monitor arterial pressure and haemodynamic parameters as indicated. Promptly report results outside the specified parameters to the doctor. *Many of the drugs used are effective within minutes. Responses vary among individuals, particularly in the older adult, necessitating continuous monitoring.*
- Monitor urine output hourly. Report output less than 30 mL/h. *The kidneys are very sensitive to reduced perfusion pressure; inadequate renal blood flow can lead to acute kidney failure.*

CONSIDERATION FOR PRACTICE

Use an infusion control device for all drug infusions. These devices prevent accidental or inadvertent changes in the rate of the infusion and dose of the drug.

Anxiety

People with aortic aneurysms often are highly anxious because of the urgent nature of the disorder. The nurse must manage the anxiety levels of both the person and family members to effectively address physiological care needs. Stress reduction also is necessary to help maintain the blood pressure within desired limits.

- Explain all procedures and treatments, using simple and understandable terms. *Simplified explanations are necessary when anxiety levels interfere with learning and understanding.*
- Respond to all questions honestly, using a calm, empathetic but matter-of-fact manner. *Honesty with the person and family promotes trust and provides reassurance that the true nature of the situation is not being 'hidden' from them.*
- Provide care in a calm, efficient manner. *Using a calm manner, even during preparations for emergency surgery, reassures the person and family that although the situation is critical, the staff is prepared to handle things effectively.*
- Spend as much time as possible with the person. Allow supportive family members to remain with the person when possible. *The presence of a health professional and supportive family member reassures the person that they are not alone in facing this crisis.*

Community-based care

Topics to discuss when preparing people and their families for home care or care in a community-based setting depend on the treatment plan. Discuss the following topics when surgical repair is not immediately planned and the aneurysm will be monitored:

- measures to control hypertension, including lifestyle and prescribed drugs
- the benefits of smoking cessation
- manifestations of increasing aneurysm size or complications to report to the doctor.

Following surgery, discuss the following topics in preparing the person and family for home care:

- wound care and preventing infection; manifestations of impaired healing or infection to be reported
- prescribed antihypertensive and anticoagulant medications and their expected and unintended effects
- the importance of adequate rest and nutrition for healing
- measures to prevent constipation and straining at stool (such as increasing fluid and fibre in the diet)
- the importance of avoiding prolonged sitting, lifting heavy objects, engaging in strenuous exercise and having sexual intercourse until approved by the doctor (usually 6 to 12 weeks)
- signs and symptoms of complications to report to the doctor.

Provide referrals to a home health agency or community health service as necessary. Referrals are especially important for older adults and their caregivers, who may require additional assistance with the complex care needs.

DISORDERS OF THE PERIPHERAL ARTERIES

Disorders that impair peripheral arterial blood flow may be acute (e.g. arterial thrombosis) or chronic (e.g. peripheral arteriosclerosis). Chronic occlusive disorders may be due to structural defects of the arterial walls or spasm of affected arteries. Impaired peripheral arterial circulation limits the availability of oxygen and nutrients to the tissues and can have significant adverse effects. This section focuses on acute and chronic disorders affecting peripheral arteries. The nurse's role in caring for people with peripheral arterial disorders focuses on maintaining tissue perfusion and educating the person and family about the disorder and its management.

Physiology review

Peripheral arteries are the part of the systemic circulation that delivers oxygen and nutrients to the skin and the extremities. Arterial walls have three layers: the intima, which includes the endothelium and a layer of connective tissue and the basement membrane; the media, composed of smooth muscle and elastic fibres; and the adventitia, a thin layer of connective tissue that contains collagen and elastic fibres. The smooth muscle of peripheral arteries controls blood flow as it contracts and relaxes. Contraction narrows the vessel lumen (**vasoconstriction**), whereas smooth muscle relaxation expands the vessel (**vasodilation**). Peripheral arteries become progressively smaller; arterioles are less than 0.5 mm in diameter and are composed primarily of smooth muscle. The arterioles control blood flow through the capillary beds where gas, nutrient and waste product exchange occurs. Capillary walls are very thin, consisting of a single layer of endothelial cells surrounded by a thin basement membrane.

Blood flows from an area of higher pressure to an area of lower pressure. *Resistance* opposes blood flow. Resistance is created by friction of the blood itself, although the primary determinants of vascular resistance are the diameter and length of the blood vessel. See the physiology review section earlier in this chapter under 'Disorders of blood pressure regulation' for more information about factors that determine vessel resistance.

THE PERSON WITH PERIPHERAL VASCULAR DISEASE

Arteriosclerosis is the most common chronic arterial disorder, characterised by thickening, loss of elasticity and calcification of arterial walls. **Atherosclerosis** is a form of arteriosclerosis in which deposits of fat and fibrin obstruct and harden the arteries. In the peripheral circulation, these

pathological changes impair the blood supply to peripheral tissues, particularly the lower extremities. This is known as **peripheral vascular disease (PVD)** or peripheral artery disease (PAD).

Incidence and risk factors

PVD usually affects people over 65; men are more often affected than women. Rates of PVD are 20% higher among Indigenous Australians than among non-Indigenous Australians (ABS, 2014).

Risk factors for PVD are similar to those for atherosclerosis and CHD (see Chapter 29). The main preventable risk factors for peripheral vascular disease are diabetes, cigarette smoking, hypertension, hyperviscosity, hypercholesterolaemia and obesity (Stephens, 2014).

FAST FACTS

- PVD is a common manifestation of atherosclerosis, particularly in older men.
- Peripheral vascular disease (also known as peripheral artery disease) refers to diseases of arteries outside the heart and brain. It occurs when fatty deposits build up in the inner walls of these arteries and affect blood circulation, mainly in the arteries leading to the legs and feet. It ranges from asymptomatic disease, through to pain on walking, to pain at rest and limb-threatening reduced blood supply that can lead to amputation.
- Regular daily exercise is a primary intervention for all types of peripheral arterial disease to promote development of collateral circulation and maintain tissue perfusion.

Pathophysiology

The pathophysiology of atherosclerosis is detailed in Chapter 29. Atherosclerotic lesions involve both the intima and the media of the involved arteries. Lesions typically develop in large and midsized arteries, particularly the abdominal aorta, iliac, femoral popliteal, tibial peroneal arteries.

Plaque tends to form at arterial bifurcations. The vessel lumen is progressively obstructed, decreasing blood flow to the lower extremities. Tissue hypoxia or anoxia results. With gradual obstruction of the vessel, collateral circulation often develops. However, it is usually not adequate to supply tissue needs, especially when metabolic demand increases (e.g. during exercise). Manifestations typically develop only when the vessel is occluded by 60% or more.

Manifestations and complications

Pain is the primary symptom of peripheral atherosclerosis. Intermittent claudication—a cramping or aching pain in the calves of the legs, the thighs and the buttocks that occurs with a predictable level of activity—is characteristic of PVD. The pain is often accompanied by weakness and is relieved by rest.

Rest pain, in contrast, occurs during periods of inactivity. It is often described as a burning sensation in the lower legs. Rest pain increases when the legs are elevated and decreases when the legs are dependent (e.g. hanging over the side of the bed). The legs also may feel cold or numb along with the pain. Sensation is diminished and the muscles may atrophy.

Peripheral pulses may be decreased or absent. A bruit may be heard over large affected arteries, such as the femoral artery and the abdominal aorta. The legs are pale when elevated, but often are dark red when dependent (*dependent rubor*). The skin often is thin, shiny and hairless, with discoloured areas. Toenails may be thickened. Areas of skin breakdown and ulceration may be evident. Oedema may develop with severe PVD. See the box below for manifestations of peripheral atherosclerosis.

MANIFESTATIONS Peripheral atherosclerosis

- Intermittent claudication
- Rest pain
- Paraesthesias (numbness, decreased sensation)
- Diminished or absent peripheral pulses
- Pallor with extremity elevation, dependent rubor when dependent
- Thin, shiny, hairless skin; thickened toenails
- Areas of discolouration or skin breakdown

Complications of peripheral atherosclerosis include gangrene and extremity amputation, rupture of abdominal aortic aneurysms and possible infection and sepsis.

INTERPROFESSIONAL CARE

Management of peripheral vascular disease focuses on slowing the atherosclerotic process and maintaining tissue perfusion.

Diagnosis

Although PVD often can be diagnosed by the history and physical examination, diagnostic tests may be ordered to evaluate its extent. Non-invasive studies are often sufficient.

- *Segmental pressure measurements* use sphygmomanometer cuffs and a Doppler device to compare blood pressures between the upper and lower extremities (normally similar) and within different segments of the affected extremity. In PVD, the BP may be lower in the legs than in the arms.
- *Stress testing* using a treadmill provides functional assessment of limitations. In PVD, pressure at the ankle may decline even further with exercise, confirming the diagnosis. Evaluation for coronary heart disease may be done simultaneously during exercise testing (Kasper et al., 2015).
- *Doppler ultrasound* uses sound waves reflected off moving red blood cells within a vessel to evaluate blood flow. The impulses may be translated into an audible signal or a graphic waveform. With significant PVD, the waveform becomes progressively flatter as the transducer is moved distally along the affected vessel. Segmental pressures may be used to locate the site of obstruction.

- *Duplex Doppler ultrasound* combines the audible or graphic Doppler ultrasound with ultrasound imaging to identify arterial or venous abnormalities. Ultrasonic imaging provides views of the affected vessel, while Doppler ultrasound evaluates blood flow. *Colour-flow Doppler ultrasound (CDU)* provides colour images of the vessel and blood flow.
- *Transcutaneous oximetry* evaluates oxygenation of tissues.
- *Angiography* or *magnetic resonance angiography (MRA)* is done before revascularisation procedures to locate and evaluate the extent of arterial obstruction. For angiography, a contrast medium is injected and vessels are visualised using fluoroscopy and x-rays. MRA does not require injection of a contrast medium and may replace angiography.

See Chapter 28 for more information on diagnostic testing for PVD.

Medications

Drug treatment of peripheral atherosclerosis is less effective than it is for coronary heart disease. Medications to inhibit platelet aggregation, such as aspirin or clopidogrel, are ordered to reduce the risk of arterial thrombosis. Cilostazol, a platelet inhibitor with vasodilator properties, improves claudication. Pentoxifylline decreases blood viscosity and increases red blood cell flexibility, increasing blood flow to the microcirculation and tissues of the extremities. Parenteral vasodilator prostaglandins may be given on a long-term basis to decrease pain and facilitate healing in people with severe limb ischaemia (Kasper et al., 2015).

Treatments

Smoking cessation is vital. Nicotine not only promotes atherosclerosis but also causes vasospasm, further reducing blood flow to the extremities.

Meticulous foot care is vital to prevent ulceration and infection (see Box 31.5). Elastic compression stockings, which reduce circulation to the skin, are avoided. Elevating the head of the bed on blocks may help relieve rest pain. Regular, progressively strenuous exercise, such as 30 to 45 minutes of walking daily, is important. The person is taught to rest at the onset of claudication, resuming activity when the pain resolves.

Other measures to slow the process of atherosclerosis, such as controlling diabetes and hypertension, lowering cholesterol levels and weight loss, also are recommended. See the box below for care of the older adult.

Revascularisation

Revascularisation may be performed if symptoms are progressive, severe or disabling. Other indications for surgery include symptoms that significantly interfere with activities of daily living (ADLs), rest pain and pregangrenous or gangrenous lesions. Either non-surgical revascularisation procedures or surgery may be performed.

Non-surgical procedures include percutaneous transluminal angioplasty (PTA), stent placement or atherectomy. Techniques may include balloon angioplasty to dilate the narrowed lumen, mechanical atherectomy to remove plaque or laser or thermal angioplasty to vaporise the occluding material. In any case, a stent typically is placed at the time of PTA to maintain vessel patency. Iliac and femoral–popliteal PTA initially re-establish good blood flow and relieve symptoms in more than 80% of people. While the 3-year success rate is lower, stent placement improves the duration of symptom relief (Kasper et al., 2015). See Chapter 29 for more information about revascularisation procedures.

Surgical options include endarterectomy to remove occlusive plaque from the artery and bypass grafts. Knitted Dacron bypass grafts are commonly used. Both immediate and long-term graft patency is better with bypass grafting than with non-surgical revascularisation procedures, but the risk of operative complications such as myocardial infarction, stroke, infection and peripheral embolisation is higher (Kasper et al., 2015). Nursing care for the person having revascularisation surgery is similar to that provided for people having an aortic aneurysm repair (see the previous box).

Complementary therapies

Complementary therapies for peripheral vascular disease include interventions to improve circulation and to reduce

BOX 31.5 Foot care for the person with peripheral atherosclerosis

1. Keep legs and feet clean, dry and comfortable.
   - Wash legs and feet daily in warm water, using mild soap.
   - Pat dry using a soft towel; be sure to dry between the toes.
   - Apply moisturising cream to prevent drying.
   - Use powder on the feet and between the toes.
   - Buy shoes in the afternoon (when feet are largest); never buy shoes that are uncomfortable. Be sure toes have adequate room.
   - Wear a clean pair of cotton socks each day.
2. Prevent accidents and injuries to the feet.
   - Always wear shoes or slippers when getting out of bed.
   - Walk on level ground and avoid crowds, if possible.
   - Do not go barefoot.
   - Inspect legs and feet daily; use a mirror to examine backs of legs and bottoms of feet.
   - Have a professional foot care provider trim toenails and care for corns, calluses, ingrown toenails or athlete's foot.
   - Always check the temperature of the water before stepping into the tub.
   - Do not get the legs or tops of the feet sunburned.
   - Report leg or foot problems (increased pain, cuts, bruises, blistering, redness or open areas) to your healthcare provider.
3. Improve blood supply to the legs and feet.
   - Do not cross legs.
   - Do not wear garters or knee stockings.
   - Do not swim or wade in cold water.

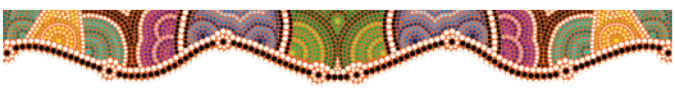
NURSING CARE OF THE OLDER ADULT Peripheral vascular disease

With ageing, blood vessels thicken and become less compliant. These changes reduce oxygen delivery to the tissues and impair carbon dioxide and waste product removal from the tissues. When normal effects of ageing combine with an increased risk of atherosclerosis, the risk of peripheral vascular disease is high.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

The older adult with peripheral vascular disease requires the same care and teaching as other people. However, visual deficits and osteoarthritis may make foot care more difficult. Long-standing smoking habits are difficult to break. Mobility may be impaired by arthritis or the effects of neurological disorders. The person who lives alone may resist walking. Periodic visits by a community or home health nurse may be helpful, as may be encouraging the person to join a support group for stopping smoking, changing eating habits and taking part in regular activity.

stress. A number of complementary therapies may improve peripheral circulation, including aromatherapy with rosemary or vetiver; biofeedback; healing or therapeutic touch and massage; herbals such as ginkgo, garlic, cayenne, hawthorn and bilberry; and exercise, including yoga. Aromatherapy and yoga also may reduce stress, as can breathing exercises, meditation and counselling. In addition, complementary therapies to reduce atherosclerosis and lower cholesterol levels may slow the progress of PVD. Measures such as a very low-fat or vegetarian diet, including antioxidant nutrients or using vitamin C, vitamin E or garlic supplements, and traditional Chinese medicine may be useful.



Nursing care

Health promotion

Discuss healthy lifestyle habits with community and religious groups, schoolchildren and through the print media to reduce the incidence and slow the progression of atherosclerosis.

Strongly encourage all people to avoid smoking in the first place and to stop all forms of tobacco use. Discuss the adverse effects of smoking and the benefits of quitting. Provide information about dietary recommendations to maintain a healthy weight and optimal cholesterol levels. Discuss the benefits and importance of regular exercise. Finally, encourage people with cardiovascular risk factors to undergo regular screening for hypertension, diabetes and hyperlipidaemia.

Assessment

Focused assessment related to peripheral atherosclerosis includes the following:

- *Health history:* complaints of pain, its relationship to exercise or rest, timing, associated symptoms and relief measures; history of coronary heart disease, peripheral vascular disease, hyperlipidaemia, hypertension or diabetes; current medications; smoking history; usual diet and activity patterns.
- *Physical examination:* vital signs; strength and equality of peripheral pulses of all extremities; capillary refill; skin colour, temperature, hair distribution, presence of any discolourations or lesions; movement and sensation of lower extremities.

Nursing diagnoses and interventions

Impaired tissue perfusion is an obvious problem in peripheral atherosclerosis. Acute and chronic pain may interfere with ADLs, and ambulation may be limited. The possibility of losing a lower extremity is frightening.

Ineffective tissue perfusion: peripheral

Impaired blood flow to the lower extremities affects gas, nutrient and waste product exchange between the capillaries and cells. Oxygen and nutrient deprivation impairs cell function and tissue integrity, causing pain and impaired healing. Pain develops with exercise and when extremities are elevated.

- Assess peripheral pulses, pain, colour, temperature and capillary refill every 4 hours and as needed. Use a Doppler device if pulses are not palpable. Mark pulse locations with an indelible marker. *Assessment data provide a baseline for evaluating the effectiveness of interventions and identifies changes in arterial blood flow.*
- Position with extremities dependent. *Gravity promotes arterial flow to the dependent extremity, increasing tissue perfusion and relieving pain.*
- Discuss the benefits of regular exercise. *Exercise promotes development of collateral circulation to ischaemic tissues and slows the process of atherosclerosis.*
- Use a foot cradle and lightweight blankets, socks and slippers to keep extremities warm. Avoid electric heating pads or hot water bottles. *Keeping extremities warm conserves heat, prevents vasospasm and promotes arterial flow. External heating devices are avoided to reduce the risk of burns in the person with impaired sensation. A foot cradle protects tissues from compression by bed linen.*
- Encourage frequent position changes. Instruct to avoid crossing legs or using a pillow under the knees. *Position changes promote blood flow and reduce damage caused by pressure. Leg crossing and excessive flexion of the hip or knee joints can compress partially obstructed arteries and impair blood flow to distal tissues.*

CONSIDERATION FOR PRACTICE

Instruct to avoid smoking. If necessary, obtain an order for a nicotine patch or gum from the doctor. Refer to Quit Helpline for practical and ongoing support. Nicotine is a potent vasoconstrictor that further impairs arterial blood flow. Smoking cessation is a vital component of care. Nicotine patches and gum contain less nicotine than cigarettes and can help reduce the stress of smoking cessation.

Pain

Impaired blood flow results in tissue ischaemia. Metabolism shifts from an efficient aerobic process to an anaerobic process. Lactic acid and metabolic waste products accumulate in tissues, causing pain. Severe and cramping pain generally occurs with exercise early in the disease. Rest initially produces relief, similar to the process used to treat angina (see Chapter 29). As the disease progresses, pain develops with less exercise and often occurs even at rest. Rest pain disrupts sleep, the sense of wellbeing and has significant disruptive effects on life roles.

- Assess pain at least every 4 hours, using a standard pain scale. *Pain is a subjective experience. Using a standard pain scale allows evaluation of treatment measures in relieving pain and restoring blood flow.*
- Keep extremities warm. *Cooling leads to vasoconstriction, increasing pain. Warming the extremities promotes vasodilation and improves arterial flow, reducing pain.*
- Teach pain relief and stress reduction techniques such as relaxation, meditation and guided imagery. *Pain increases stress. The stress response leads to vasoconstriction, increasing pain. Stress reduction techniques, when combined with other measures to promote blood flow, can help reduce pain.*

Impaired skin integrity

People with PVD are at risk of impaired skin integrity as a result of oxygen and nutrient deprivation. Chronic tissue ischaemia leads to dry, scaly and atrophied skin. Pruritus can lead to scratching; minor injuries may go unnoticed due to impaired sensation. Impaired tissue healing can lead to ulceration, infection and potential gangrene.

- Provide meticulous daily skin care, keeping the skin clean and dry. Apply a moisturising cream to dry or scaly areas. *Intact skin is the body's first defence against bacterial invasion. Ischaemic tissues of the injured extremity provide an excellent medium for microorganism growth. Clean, dry, supple skin decreases the risk of breakdown.*
- Apply a bed cradle. *The bed cradle suspends bed linen over the legs, preventing them from placing pressure on extremities and injured tissues. Minimising pressure on the tissues promotes capillary blood flow.*
- Provide an egg-crate mattress, flotation pad, sheepskin or heel protectors. *Ischaemic tissues may be damaged by minor trauma such as that created by the shearing forces of skin against bed linen.*

CONSIDERATION FOR PRACTICE

Assess and document skin condition at least every 8 hours or with each home visit; or more frequently as indicated. Tissue ischaemia increases the risk of damage, even with minor trauma such as pressure from poorly fitting shoes or bed linens. Frequent inspection and documentation of skin condition is vital to identify early indicators of impaired skin integrity and reduce the risk of complications such as infection.

Activity intolerance

Pain and impaired perfusion of peripheral tissues may limit the person's ability to engage in desired activities, even impairing self-care.

- Assist with care activities as needed. *Severe claudication or rest pain may limit activities. Muscle atrophy of affected extremities is common, leading to fatigue and weakness.*
- Unless contraindicated, encourage gradual increases in duration and intensity of exercise. Teach to rest with extremities dependent when claudication develops, resuming activity after pain has abated. *Gradual increases in the duration and intensity of exercise promote development of collateral circulation, improve exercise tolerance, provide a sense of wellbeing and support self-esteem.*
- Provide diversional activities during periods of prescribed bed rest. Encourage relaxation techniques to reduce muscle tension. *Diversional activities help prevent boredom and stress associated with enforced rest. Relaxation techniques reduce vasoconstriction induced by stress, improving peripheral circulation.*
- Encourage frequent position changes and active range-of-motion exercises. Encourage self-care to the extent possible. *Position changes relieve pressure on tissues, improving capillary circulation and reducing tissue ischaemia. Range-of-motion exercises help prevent muscle atrophy and joint contractures. Self-care supports self-esteem.*

Community-based care

Discuss the following topics when preparing the person and family for home and community-based care (see the accompanying nursing care plan for additional community-based nursing interventions):

- smoking cessation strategies and ways to avoid second-hand smoke
- prescribed medications and anticoagulants, their purpose, doses, desired and adverse effects
- signs of excess bleeding to report to the doctor
- skin surveillance and foot care (see Box 31.5)
- recommended diet and exercise
- weight loss strategies if appropriate.

If revascularisation or surgery has been performed, include the following topics as appropriate:

- incision care

NURSING CARE PLAN A person with peripheral vascular disease

William Duffy, aged 69, is retired. His wife convinces him to see his doctor about his increasing leg pain with walking and other exercise.

ASSESSMENT

Katie Kotson, RN, obtains Mr Duffy's history before he sees his doctor. He states that he can only walk about a block before the pain in his calves gets so bad that he has to stop and rest. As a result, he has been less and less active, spending most of his time the past few months watching sports on television. He denies rest pain. He was diagnosed with type 2 diabetes about 15 years ago, which he manages with daily glibenclamide, an oral hypoglycaemic. He also has stable angina, for which he takes atenolol and an occasional glyceryl trinitrate tablet. His alcohol intake is moderate, averaging one to two beers per day, and he smokes about a packet of cigarettes per day. He states he tried to quit smoking after developing angina, but 'after nearly 50 years of smoking, I think that's impossible!'.

Physical exam findings include height 173 cm, weight 107 kg, BP 168/78, P 66, R 16, T 36.5°C; upper extremities warm and pink, normal hair distribution, pulses strong and equal; lower extremities below knees cool and ruddy when dependent, pale to pink when elevated, skin shiny, scant hair; posterior tibial pulses weak bilaterally; weak pedal pulse on R, unable to palpate on L; 1+ to 2+ oedema both feet and ankles.

The doctor finds that Mr Duffy's systolic blood pressure in his legs is an average of 28 mmHg lower than in his arms. He makes the diagnosis of peripheral atherosclerosis and schedules Mr Duffy for an exercise stress test with ankle pressure measurements before and after exercise and a colour-flow Doppler ultrasound. Mr Duffy is to return in 3 weeks after these studies have been completed.

DIAGNOSES

- *Activity intolerance* related to poor blood flow to lower extremities.
- *Ineffective health maintenance* related to smoking and lack of information about disease management.
- *Risk of impaired skin integrity* related to ischaemic tissues of legs and feet.
- *Risk of peripheral neurovascular dysfunction* related to impaired peripheral blood flow to lower extremities.

PLANNING

- With Mr and Mrs Duffy, plan strategies to start and maintain a program of regular exercise.
- Schedule an appointment with the dietitian to develop a low-kilojoule, low-fat and low-cholesterol diet that includes preferred foods and considers usual eating patterns.
- Plan meeting times with Mr Duffy to discuss prevention and risk factors including modifiable factors.

Expected outcomes

- Walk for at least 15 minutes three to four times per day, gradually increasing his pace and duration of exercise.
- Relate the benefits of smoking cessation.
- Identify strategies to improve chances for success in stopping smoking.
- Meet with dietitian before next visit to discuss dietary measures to promote weight loss and slow atherosclerosis.
- Verbalise an understanding of appropriate foot care measures.
- Identify measures to prevent inadvertent injury of feet and legs.

IMPLEMENTATION

- Teach about peripheral atherosclerosis and its relationship to Mr Duffy's symptoms.
- Instruct to warm up slowly and to stop exercise and rest for 3 minutes (or until pain is relieved) when claudication develops, then resume exercising.
- Discuss the effects of smoking on blood vessels.
- Help Mr Duffy identify smoking cessation strategies such as support groups, clinics and nicotine patches.
- Reinforce and supplement previous foot care teaching.
- Discuss effects of impaired circulation on sensation in feet and legs and measures to prevent injury.

EVALUATION

When Mr Duffy returns 3 weeks later, his diagnosis has been confirmed by the diagnostic studies. The doctor decides to continue conservative therapy, now prescribing atorvastatin to lower Mr Duffy's serum cholesterol level and cilostazol to reduce the risk of thrombosis and improve symptoms of claudication. Mr Duffy also asks his doctor for a prescription for nicotine patches, saying he is ready to quit smoking, but thinks he needs help to be successful. Mr and Mrs Duffy tell Nurse Kotson that they are walking before every meal and really enjoying being outside more. They plan to walk in the local shopping centre whenever the weather is poor. Mrs Duffy has bought an Australian Heart Foundation cookbook (*The Deliciously Healthy Cookbook*) and is carefully planning their meals. Mr and Mrs Duffy have both lost between 0.5 and 1 kg a week since the previous visit. Mr Duffy's skin on his legs and feet remains intact and he identifies the measures he is using to protect his lower extremities from injury.

CRITICAL THINKING IN THE NURSING PROCESS

1. What additional lifestyle changes related to peripheral atherosclerosis might be appropriate to suggest to Mr Duffy at this time? Why?
2. Explain the relationship between physical exercise and pain in the person with peripheral atherosclerosis. Compare this relationship to that between exercise and angina.
3. Mr Duffy uses a beta-blocker, atenolol, to prevent angina. Why is this drug not effective in preventing claudication?
4. Develop a nursing care plan for the nursing diagnosis *Imbalanced nutrition: more than body requirements*.

REFLECTION ON THE NURSING PROCESS

1. What strategies can be initiated to ensure Mr Duffy remains on track with improving his health?
2. Mr Duffy stated he tried to quit smoking after developing angina, but 'after nearly 50 years of smoking, I think that's impossible!'. What would the nurse assess to ensure he is compliant with improving his health?

- manifestations of complications (e.g. infection, graft leakage or thrombosis) to be reported to the doctor
- activity limitations.

Provide referrals to home health services, physical or occupational therapy and home maintenance assistance services as indicated. Consider resources such as Meals on Wheels for people who are severely limited by their disease.

THE PERSON WITH SELECTED PERIPHERAL ARTERIAL CONDITIONS

ACUTE ARTERIAL OCCLUSION

A peripheral artery may be acutely occluded by development of a thrombus (blood clot) or by an embolism. Blood flow to tissues supplied by the artery is impaired, resulting in acute tissue ischaemia and a risk of necrosis and gangrene.

Pathophysiology

Arterial thrombosis

A **thrombus** is a blood clot that adheres to the vessel wall. Thrombi tend to develop in areas where intravascular factors stimulate coagulation (e.g. where a vessel lumen is partially obstructed and its wall is damaged and roughened by atherosclerosis). Other disorders, such as infection or inflammation of the vessel wall or pooling of blood (e.g. in an aneurysm), also can prompt coagulation and thrombus formation (Piechota-Polanczyk et al., 2015). A developing thrombus can occlude arterial blood flow through the vessel, leading to ischaemia of tissues supplied by that artery. The extent of ischaemia depends on the size of the affected artery and the degree of collateral circulation. In gradual processes of arterial occlusion such as atherosclerosis, collateral vessels often develop to compensate for impaired arterial flow. The extent of collateral circulation affects the degree of tissue ischaemia distal to the thrombus.

Arterial embolism

An **embolism** is the sudden obstruction of a blood vessel by debris. A thrombus can break loose from the arterial wall to become a **thromboembolus**. Other substances also can become emboli: atherosclerotic plaque, masses of bacteria, cancer cells, amniotic fluid, bone marrow fat and foreign objects such as air bubbles or broken intravenous catheters. Regardless of cause, an embolus eventually lodges in a vessel that is too small to allow it to pass.

Arterial emboli often originate in the left side of the heart. They are associated with myocardial infarction, valvular heart disease, left-sided heart failure, atrial fibrillation or infectious heart diseases. Emboli from the left heart often enter the carotid arteries and become trapped in the cerebral circulation, causing neurological deficits. Thromboemboli that develop in the aorta or peripheral arterial circulation tend to lodge in areas where the arterial lumen is narrowed by atherosclerotic plaque and at arterial bifurcations.

Manifestations

The manifestations of arterial thrombosis and embolism are those of tissue ischaemia. Ischaemic tissues are painful, pale and cool or cold. Distal pulses are absent. Paraesthesias (numbness and tingling) develop in the extremity. Cyanosis and mottling are common. Paralysis and muscle spasms may develop in the affected extremity. A line of demarcation between normal and ischaemic tissue may be seen, particularly with embolism. Tissue below the line is cool or cold and pale, cyanotic or mottled. See the 'Manifestations' box below.

MANIFESTATIONS Acute arterial occlusion

- Pain
- Pallor or mottling
- Paraesthesias (numbness and tingling)
- Cool or cold skin
- Pulselessness distal to the blockage
- Possible paralysis, weakness or muscle spasms
- Possible line of demarcation; with pallor, cyanosis and cooler skin distal to the blockage (especially with arterial embolism)

Arterial occlusion can result in permanent vessel and limb damage. Complete arterial occlusion leads to tissue necrosis and gangrene unless blood flow is promptly restored.

INTERPROFESSIONAL CARE

Acute arterial occlusions may require emergency treatment to preserve the limb if the obstructed vessel is large or collateral circulation is minimal. If the limb is not in jeopardy, more conservative management may be initiated.

Diagnosis

The diagnosis of acute arterial occlusion often is apparent by the signs and symptoms. *Arteriography* is used to confirm the diagnosis, locate the occlusion and determine its extent.

Medications

Anticoagulation with intravenous heparin is initiated to prevent further clot propagation and recurrent embolism. Anticoagulation is continued with oral anticoagulants after discharge. See the section on venous thrombosis later in this chapter for more information about anticoagulant therapy.

Arterial thrombosis may be treated with intra-arterial fibrinolytic therapy using streptokinase, urokinase or tissue plasminogen activator (t-PA) (see Chapter 29). Lysis of the

thrombus or embolus is achieved in 50–80% of the cases (Papadakis, McPhee & Rabow, 2013).

Local intra-arterial injection of the fibrinolytic drug allows use of lower doses and reduces the bleeding risk associated with fibrinolytic drugs.

Surgery

Immediate embolectomy (within 4 to 6 hours) is the treatment of choice for acute arterial occlusion by an embolus to prevent tissue necrosis and gangrene. When the involved vessel is in an extremity, local anaesthesia and a special balloon-tipped catheter known as a Fogarty catheter may be used for people with a high surgical risk (Papadakis et al., 2013).

An embolus in the mesenteric circulation necessitates emergency laparotomy. The risk of complications and limb loss increases significantly if surgery is delayed by 12 or more hours. Potential major complications include compartment syndrome (see Chapter 38), acute respiratory distress syndrome (Chapter 36) or acute kidney failure (Chapter 27).

Arterial thrombosis also may be treated surgically, although the required surgery may be more extensive due to the length of the vessel involved. *Thromboendarterectomy* is done to remove the thrombus and plaque in the artery. An arterial graft may be required. Nursing care for people who have undergone embolectomy or thrombus removal is discussed in the nursing care section that follows.

Nursing care

Assessment

Nursing assessment for the person with an acute arterial occlusion is highly focused due to the emergency nature of the problem.

- *Health history:* complaints of pain, numbness, tingling or weakness in the involved extremity; history of atherosclerotic vessel disease, heart disease or recent invasive procedure (e.g. angiography, percutaneous revascularisation procedure).
- *Physical examination:* vital signs; peripheral pulses in both extremities; colour, temperature, sensation and movement of involved extremity; skin condition; presence of a line of demarcation.

Nursing diagnoses and interventions

Nursing care related to acute arterial occlusion focuses on protecting the affected extremity, managing anxiety and reducing the risk of complications related to anticoagulant therapy.

Ineffective tissue perfusion: peripheral

Protecting ischaemic tissue from injury prior to surgery or medical thrombolysis is vital. Following surgery, there is a risk of thrombosis at the graft site or impaired perfusion due to oedema of the surgical site.

CONSIDERATION FOR PRACTICE

Monitor extremity perfusion, comparing affected and unaffected extremities. Assess peripheral pulses (using the Doppler stethoscope as needed), skin temperature and colour, capillary refill, movement and sensation every 1 to 4 hours. Promptly report changes or complaints of increased or unrelieved pain. Propagation of a thrombus can further obstruct arterial flow, increasing tissue ischaemia. Following surgery, arterial spasms may cause a cyanotic, pulseless extremity; normal colour and pulses should return within 12 hours. A thrombus may form at the surgical site or within a graft, causing tissue ischaemia with pain and other manifestations of arterial occlusion. Further measures to restore circulation may be necessary.

- Maintain intravenous fluids as ordered. *Adequate circulating blood volume is necessary to maintain cardiac output and tissue perfusion.*
- Protect the extremity, keeping it horizontal or lower than the heart. Use a cradle to keep bedclothes off the extremity and a sheepskin or foam pad to protect it from hard or abrasive surfaces. Do not apply heat or cold. *Keeping the extremity lower than the heart promotes collateral blood flow. Ischaemic tissue is easily damaged by minimal trauma such as shearing by bed linen or heat or cold application.*
- Following surgery, avoid raising the knees, placing pillows under the knees or sitting with 90-degree hip flexion. *These activities may impair blood flow through the affected vessel.*

Anxiety

People with an acute arterial occlusion often are very anxious. The rapid and intense nature of preoperative activities can be overwhelming, increasing anxiety about the disorder and its outcome. Manifestations of anxiety may include trembling, palpitations, restlessness, dry mouth, helplessness, inability to relax, irritability, forgetfulness and lack of awareness of surroundings. Nursing measures focus on establishing trust and minimising the effects of anxiety to decrease surgical risk and improve recovery.

- Spend as much time as possible with the person. Provide opportunities to verbalise anxiety; offer reassurance and support. Support adaptive coping mechanisms. *The presence of a caring nurse provides a safe environment for expressing fears and anxieties. Coping mechanisms reduce the immediate perceived threat and increase the ability to deal with the situational crisis.*
- Perform required measures in an expedient but calm manner. *Calm, confident performance of treatment measures reassures the person and family that appropriate care is being given to treat the problem at hand.*
- Assess anxiety level at least every 8 hours; more often as needed. Intervene as indicated to reduce anxiety. *Assessment helps determine the intensity of anxiety, the person's ability to control it and directs interventions to reduce it.*
- Decrease sensory stimuli as much as possible. *Reducing environmental stimuli provides the person with a degree of control over anxiety.*

- Speak slowly and clearly and avoid unnecessary interruptions when listening. Give concise directions, focusing on the present. Involve the person in simple tasks and decisions to the extent possible. *High levels of anxiety interfere with learning. Keeping interactions focused on the present situation directs the person's focus and provides reassurance that it is the most important focus for the nurse as well. Providing opportunities for self-care and decision making reinforces the person's importance and power to control the situation.*

Altered protection

Fibrinolytic and/or anticoagulant therapy used to dissolve existing clots and prevent further clot formation increases the risk of bleeding. Close monitoring of physical status and laboratory data is vital, as are measures to reduce the risk of injury and bleeding.

- Monitor activated partial thromboplastin time (APTT) during heparin therapy and prothrombin time (PT) or International Normalized Ratio (INR) during oral anticoagulant therapy. Report values outside desired range. *The APTT, PT and INR are prolonged by anticoagulant therapy. Values higher than the desired range may indicate an increased risk of bleeding; values below the target may indicate inadequate anticoagulation.*
- Protect from injury: use side rails or other measures as needed to prevent falls; avoid parenteral injections and other invasive procedures as much as possible; hold firm pressure over injection and intravenous sites for 5 minutes and over arterial punctures for 20 minutes; use a soft toothbrush or sponge for oral care; use an electric razor for shaving. *Minor trauma can lead to extensive bleeding, particularly in the person who has received a fibrinolytic drug.*

CONSIDERATION FOR PRACTICE

Assess for and report manifestations of impaired clotting, including excessive incisional bleeding; prolonged oozing from injection sites; bleeding gums, nosebleed or haematuria; petechiae, bruising or purpura. Anticoagulants and fibrinbolytics interfere with the clotting cascade and may cause abnormal bleeding.

Community-based care

When preparing the person and family for home or community-based care related to an acute arterial occlusion, discuss the following topics as indicated:

- care of the incision
- manifestations of complications to be reported, including symptoms of infection or occlusion of the graft or artery
- long-term anticoagulant therapy, including the reason, prescribed dose, follow-up laboratory testing and appointments, interactions with other drugs and manifestations of excessive bleeding
- any activity restrictions or dietary modifications
- lifestyle modifications to slow atherosclerosis and control hypertension
- measures to promote peripheral circulation and maintain tissue integrity (see the discussion of peripheral atherosclerosis earlier in the chapter).

Refer for home care services (nursing care, physical therapy, housekeeping services) as indicated.

THE PERSON WITH THROMBOANGIITIS OBLITERANS

Thromboangiitis obliterans (also called *Buerger's disease*) is an occlusive vascular disease in which small and midsized peripheral arteries become inflamed and spastic, causing clots to form. This disease may affect either the upper or lower extremities; it often affects a leg or foot. Its exact aetiology is unknown.

Incidence and risk factors

Thromboangiitis obliterans primarily affects men under age 40 who smoke. Cigarette smoking is the single most significant cause of the disease. The disease is more prevalent in Asians and people of Eastern European descent. The incidence of HLA-B5 and 2A9 antigens is higher in people with thromboangiitis obliterans, suggesting a genetic link.

Pathophysiology and course

Inflammatory cells infiltrate the wall of small and midsized arteries in the feet and possibly the hands. This inflammatory process is accompanied by thrombus formation and vasospasms of arterial segments that impair blood flow. Adjacent veins and nerves also may be affected. As the disease progresses, affected vessels become scarred and fibrotic.

The course of the disease is intermittent with dramatic exacerbations and marked remissions. The disease may remain dormant for periods of weeks, months or years. As the disease progresses, collateral vessels are more extensively involved. Consequently, subsequent episodes are more intense and prolonged. Prolonged periods of tissue hypoxia increase the risk of tissue ulceration and gangrene.

Manifestations and complications

Pain in the affected extremities is the primary manifestation of thromboangiitis obliterans. Both claudication, cramping pain in the calves and feet or the forearms and hands, and rest pain in the fingers and toes may occur. Sensation is diminished. Eventually, the skin becomes thin and shiny and the nails are thickened and malformed. On examination, the involved digits and/or extremities are pale, cyanotic or ruddy, and cool or cold to touch. Distal pulses (e.g. the dorsalis pedis, posterior tibial, ulnar or radial) are either difficult to locate or absent, even with a Doppler device.

Painful ulcers and gangrene may develop in the fingers and toes as a result of severely impaired blood flow. Amputation may be necessary to remove necrotic tissue.

INTERPROFESSIONAL CARE

Diagnosis

Thromboangiitis obliterans usually is diagnosed by the history and physical examination. Doppler studies may be used to locate and determine the extent of the disease. Angiography and magnetic resonance imaging may also be used to evaluate the extent of the disease, but usually are unnecessary.

Lifestyle modifications

The one most important component in managing this disease is smoking cessation. While stopping smoking does not cure the disease, it may slow its extension to other vessels. With continued smoking, attacks become increasingly intense and last much longer, significantly increasing the risk of ulcerations and gangrene.

Additional conservative measures are used to prevent vasoconstriction, improve peripheral blood flow and prevent complications of chronic ischaemia. These measures include keeping extremities warm, managing stress, keeping affected extremities in a dependent position, preventing injury to affected tissues and regular exercise. Walking for 20 or more minutes several times a day is recommended.

Medications

There are no specific drugs for thromboangiitis obliterans. Calcium channel blockers such as diltiazem or verapamil, and/or oxpentifylline, which decrease blood viscosity and increase red blood cell flexibility to improve peripheral blood flow, may provide some symptom relief.

Surgery

Surgical approaches for thromboangiitis obliterans include sympathectomy or arterial bypass graft. Sympathectomy interrupts sympathetic nervous system input to affected vessels, reducing vasoconstriction and spasm. Arterial bypass grafts may be useful when larger vessels are affected by the disease. Amputation of an affected digit or extremity may be necessary if gangrene develops (see Chapter 38 for more information about amputation). Only portions of digits or of limbs (e.g. below the knee) are usually amputated, to preserve as much healthy tissue as possible.

The prognosis for thromboangiitis obliterans depends significantly on the person's ability and willingness to stop smoking. With smoking cessation and good foot care, the prognosis for saving the extremities is good, even though no cure is available.

Nursing care

Health promotion

Health promotion activities to prevent thromboangiitis obliterans focus on preventing smoking, especially in high-risk populations.

Assessment

Nursing assessment and care for people with this disease are similar to those provided for people with other arterial occlusive diseases. Nursing care focuses on promoting arterial circulation and preventing prolonged tissue hypoxia. Due to inflammation, spastic episodes may be unpredictable; care focuses on smoking cessation and relieving acute manifestations. In addition, postsurgical care is necessary if surgery has been performed. See the nursing care section for peripheral atherosclerosis, as well as nursing care of the postsurgical person (Chapter 3) and following amputation (Chapter 38).

Community-based care

Discuss the following topics when preparing people with thromboangiitis obliterans and their families for home or community-based care:

- absolute necessity of smoking cessation
- foot care
- protecting affected extremities from injury
- purpose, dose, desired and adverse effects, interactions and any precautions associated with prescribed medications
- signs and symptoms to report to the doctor.

THE PERSON WITH RAYNAUD'S DISEASE

Raynaud's disease and **Raynaud's phenomenon** are characterised by episodes of intense vasospasm in the small arteries and arterioles of the fingers and sometimes the toes (Hansen-Dispenza, 2014). Raynaud's disease and Raynaud's phenomenon differ only in terms of cause. Raynaud's disease has no identifiable cause; Raynaud's phenomenon occurs secondarily to another disease (such as collagen vascular diseases such as scleroderma and rheumatoid arthritis), other known causes of vasospasm or long-term exposure to cold or machinery (Hansen-Dispenza, 2014).

Raynaud's disease primarily affects young women between the ages of 20 and 40. Genetic predisposition may play a role in its development, although the actual cause is unknown. Table 31.4 compares thromboangiitis obliterans and Raynaud's disease.

Pathophysiology and manifestations

Raynaud's disease and Raynaud's phenomenon are characterised by spasms of the small arteries in the digits. The arterial spasms limit arterial blood flow to the fingers and possibly the toes. Initial attacks may involve only the tips of one or two fingers; with disease progression, the entire finger and all fingers may be affected.

The manifestations of Raynaud's occur intermittently when spasms develop. Raynaud's disease has been called the 'blue-white-red disease', because affected digits initially turn blue as blood flow is reduced due to vasospasm, then white as

TABLE 31.4 Comparison of Raynaud's disease and thromboangiitis obliterans

TOPIC	RAYNAUD'S DISEASE	THROMBOANGIITIS OBLITERANS
Aetiology	• Unknown • Possible genetic predisposition	• Cigarette smoking most probable single cause • Possible autoimmune response
Incidence/course of the disease	• Onset commonly between 15 and 45 years of age • Usually affects young women • Becomes progressively worse over time	• Occurs predominantly in men under age 40 • More common in people of Asian or European heritage • Intermittent course with exacerbations and remissions • Increased severity and duration of attacks over time
Triggering stimuli	• Emotional stress • Exposure to cold	• Cigarette smoking
Assessment findings	• Usually affects hands, sometimes toes • Pain becomes more severe and prolonged as disease progresses • 'Blue-white-red' changes in colour of hands with accompanying changes in skin temperature	• Claudication and pain • Numbness or diminished sensation • Cool, pale or cyanotic skin • Shiny, thin skin and white, malformed nails in affected extremities • Distal pulses difficult to find or absent • Trophic changes to nail beds • Ulceration and gangrene in later stages • Small, red, tender vascular cords in affected extremities
Management	• Avoid unnecessary cold exposure • Emphasise smoking cessation • Medications such as calcium channel or alpha-adrenergic blockers as indicated • Teach stress management	• Stop smoking (crucial) • Regular exercise • Protect extremities from cold injury • Teach stress management

circulation is more severely limited and, finally, very red as the fingers are warmed and the spasm resolves (see Figure 31.12). Sensory changes may occur during attacks, including numbness, stiffness, decreased sensation and aching pain.

The attacks tend to become more frequent and prolonged over time. With repeated attacks (and resultant decrease in oxygenation), the fingertips thicken and the nails become brittle. Ulceration and gangrene are serious complications that rarely occur.

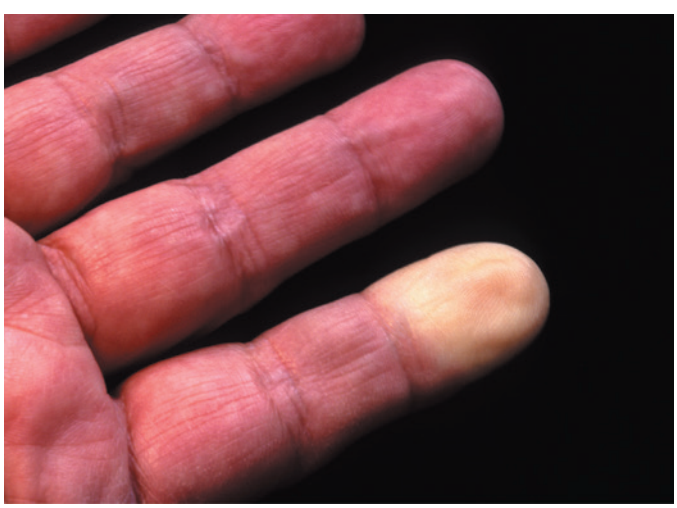


FIGURE 31.12 ■ Fingers of a patient with Raynaud's phenomenon. Note the extreme pallor of the fifth digit in response to exposure to cold

Source: P. Stocklein/Custom Medical Stock Photo.

INTERPROFESSIONAL CARE

Diagnosis

Raynaud's disease and Raynaud's phenomenon are primarily diagnosed by the history and physical examination. There are no specific diagnostic tests for these disorders.

Medications

Vasodilators may be prescribed to provide symptomatic relief. Low doses of a sustained-release calcium channel blocker such as nifedipine or diltiazem may be prescribed. The alpha-adrenergic blocker prazosin also may reduce the frequency and severity of attacks. Transdermal glyceryl trinitrate (or longer-acting oral nitrates) helps some people by decreasing the amount of time necessary for the hands to return to normal following an attack (Papadakis et al., 2013).

Lifestyle modifications

Conservative measures are a mainstay of treatment. People are instructed to keep their hands warm, wearing gloves when outside in cold weather and kitchen gloves when handling cold items (e.g. when preparing and serving cold foods and cleaning

the refrigerator). Measures to avoid injury to the hands are taught. Sometimes attacks can be stopped by swinging the arms back and forth, increasing perfusion pressure in the small arteries by centrifugal force.

Smoking cessation is important. Stress reduction measures such as exercise, relaxation techniques, massage therapy, hobbies, aromatherapy and counselling are taught or suggested. Additional lifestyle habits that contribute to vascular health are encouraged, such as reducing dietary fat, increasing activity level and maintaining normal body weight.

Nursing care

Nursing care for the person with Raynaud's disease or Raynaud's phenomenon is primarily educative and supportive. Protecting the hands and feet from exposure to cold and trauma is the main teaching topic. Nursing diagnoses and interventions previously outlined for peripheral atherosclerosis also are appropriate for people with Raynaud's disease.

Community-based care

Reassure people with Raynaud's phenomenon that most people with the disorder experience only mild, infrequent episodes. Discuss the following topics in preparing the person for managing the disorder:

- Dress warmly, keeping the trunk and hands warm.
- Avoid unnecessary exposure to cold.
- Stop smoking or do not start.
- The use, purpose, desired and potential adverse effects of prescribed medications, if any.

DISORDERS OF VENOUS CIRCULATION

The two primary categories of venous system disorders are occlusive disorders and those related to ineffective venous blood flow. Impaired venous blood flow can lead to stasis and clotting, as well as tissue changes associated with venous congestion.

Physiology review

The venous system is a low-pressure system in comparison with the arterial circulation. Veins and venules are thin-walled, distensible vessels. While they contain smooth muscle that allows them to contract or expand, the media (muscle layer) of veins is significantly thinner than that of arteries. The low pressures in the venous system allow it to serve as a reservoir for blood. Stimulation by the sympathetic nervous system causes veins to contract, helping maintain vascular volume. The low-pressure venous system relies on skeletal muscle contractions and pressure changes in the abdomen and thorax to facilitate blood return to the heart. Unlike arteries, veins of the extremities contain valves to prevent retrograde blood flow.

THE PERSON WITH VENOUS THROMBOSIS

Venous thrombosis (also known as *thrombophlebitis*) is a condition in which a blood clot (thrombus) forms on the wall of a vein, accompanied by inflammation of the vein wall and some degree of obstructed venous blood flow.

Venous thrombi are more common than arterial thrombi because of lower pressures and flow within the venous system (Patel, 2014). Thrombi can form in either superficial or deep veins. **Deep venous thrombosis (DVT)** is a common complication of hospitalisation, surgery and immobilisation. Obstetric and orthopaedic procedures carry a higher risk of venous thrombosis; it may develop in more than 50% of people having orthopaedic surgery, particularly surgeries involving the hip or knee (Patel, 2014). Other significant risk factors for venous thrombosis include abdominal or thoracic surgery, certain cancers, trauma, pregnancy and use of oral contraceptives or hormone replacement therapy (see Box 31.6).

Pathophysiology

Three pathological factors, called *Virchow's triad*, are associated with thrombophlebitis: stasis of blood, vessel damage and increased blood coagulability. Vessel trauma stimulates the clotting cascade. Platelets aggregate at the site, particularly

BOX 31.6 Factors associated with venous thrombosis

- Immobilisation: myocardial infarction, heart failure, stroke, postoperative
- Surgery: orthopaedic, thoracic, abdominal, genitourinary
- Cancer: pancreatic, lung, ovary, testes, urinary tract, breast, stomach
- Trauma: fractures of the spine, pelvis, femur, tibia; spinal cord injury
- Pregnancy and delivery
- Hormone therapy: oral contraceptives, hormone replacement therapy
- Coagulation disorders

when venous stasis is present. Platelets and fibrin form the initial clot. Red blood cells are trapped in the fibrin meshwork and the thrombus propagates (grows) in the direction of blood flow. The inflammatory response is triggered, causing tenderness, swelling and erythema in the area of the thrombus. Initially the thrombus floats within the vein. Pieces of the thrombus may break loose and travel through the circulation as emboli. Fibroblasts eventually invade the thrombus, scarring the vein wall and destroying venous valves. Although patency of the vein may be restored, valve damage is permanent, affecting directional flow (Papadakis et al., 2013).

FAST FACTS

- DVT is a common complication of surgery and immobility. It usually develops in the deep veins of the calf (80%).
- Venous stasis (sluggish blood flow), altered blood coagulation and damage (e.g. inflammation) to blood vessels are precipitating factors for DVT.
- A thrombus or a portion of a thrombus may break loose, travelling through the venous system to the right side of the heart and into the pulmonary circulation, where it ultimately becomes lodged (pulmonary embolus).

Deep venous thrombosis

The deep veins of the legs, primarily in the calf and the pelvis, provide the most hospitable environment for venous thrombosis. Approximately 80% of deep venous thromboses begin in the deep veins of the calf, often propagating into the popliteal and femoral veins (see Figure 31.13) (Papadakis et al., 2013). DVT usually is asymptomatic; in some people, a pulmonary embolism may be the first indication.

MANIFESTATIONS When present, the manifestations of DVT are primarily due to the inflammatory process accompanying the thrombus. Calf pain, which may be described as tightness or a dull, aching pain in the affected extremity, particularly upon walking, is the most common symptom. Tenderness, swelling, warmth and erythema may be noted along the course of involved veins. The affected extremity may be cyanotic and often is oedematous. Rarely, a cord may be palpated over the affected vein. A positive Homan's sign (pain in the calf when the foot is dorsiflexed) is an unreliable indicator of DVT. See the 'Manifestations' box for a summary of the manifestations of deep and superficial venous thrombosis.

COMPLICATIONS The major complications of DVT are chronic venous insufficiency (see the next section of this chapter) and pulmonary embolism. Pulmonary embolism occurs when the clot fragments or breaks loose from the vein wall. As the clot travels, it moves through progressively larger veins and into the right side of the heart. From there it enters the pulmonary circulation, where it eventually occludes arterial flow to a portion of the lungs. The result is a mismatch between ventilation (air flow) and perfusion (blood flow) in a portion of the lungs. The effect on gas exchange depends on the size of the embolism and the vessel it occludes. See Chapter 36 for more information about pulmonary emboli.

MANIFESTATIONS Venous thrombosis

DEEP VENOUS THROMBOSIS

- Usually asymptomatic
- Dull, aching pain in affected extremity, especially when walking
- Possible tenderness, warmth, erythema along affected vein
- Cyanosis of affected extremity
- Oedema of affected extremity

SUPERFICIAL VENOUS THROMBOSIS

- Localised pain and tenderness over the affected vein
- Redness and warmth along the course of the vein
- Palpable cordlike structure along the affected vein
- Swelling and redness of surrounding tissue

Superficial venous thrombosis

Venous catheters and infusions are the primary risk factors for superficial venous thrombosis. Superficial venous thrombosis also may develop in conjunction with thromboangiitis obliterans, varicose veins or DVT. It may develop spontaneously in pregnant women or following delivery. In some cases, superficial venous thrombosis of the long saphenous vein is the earliest sign of an abdominal cancer such as pancreatic cancer (Papadakis et al., 2013).

Superficial venous thrombosis is marked by pain and tenderness at the site of the thrombus. A reddened, warm, tender cord extending along the affected vein can be palpated. The area surrounding the vein may be swollen and red (see the 'Manifestations' box).

INTERPROFESSIONAL CARE

It is important to differentiate venous thrombosis from other causes of extremity pain, such as cellulitis, muscle strain, contusion and lymphoedema. The history, physical examination and diagnostic tests are used to establish the diagnosis. Treatment focuses on preventing further clotting or extension of the clot and addressing underlying causes.

Diagnosis

- *Duplex venous ultrasonography* is a non-invasive test used to visualise the vein and measure the velocity of blood flow in the veins. Although the clot often cannot be visualised directly, its presence can be inferred by an inability to compress the vein during the examination.
- *Plethysmography* is a non-invasive test that measures changes in blood flow through the veins. It is often used in conjunction with Doppler ultrasonography.

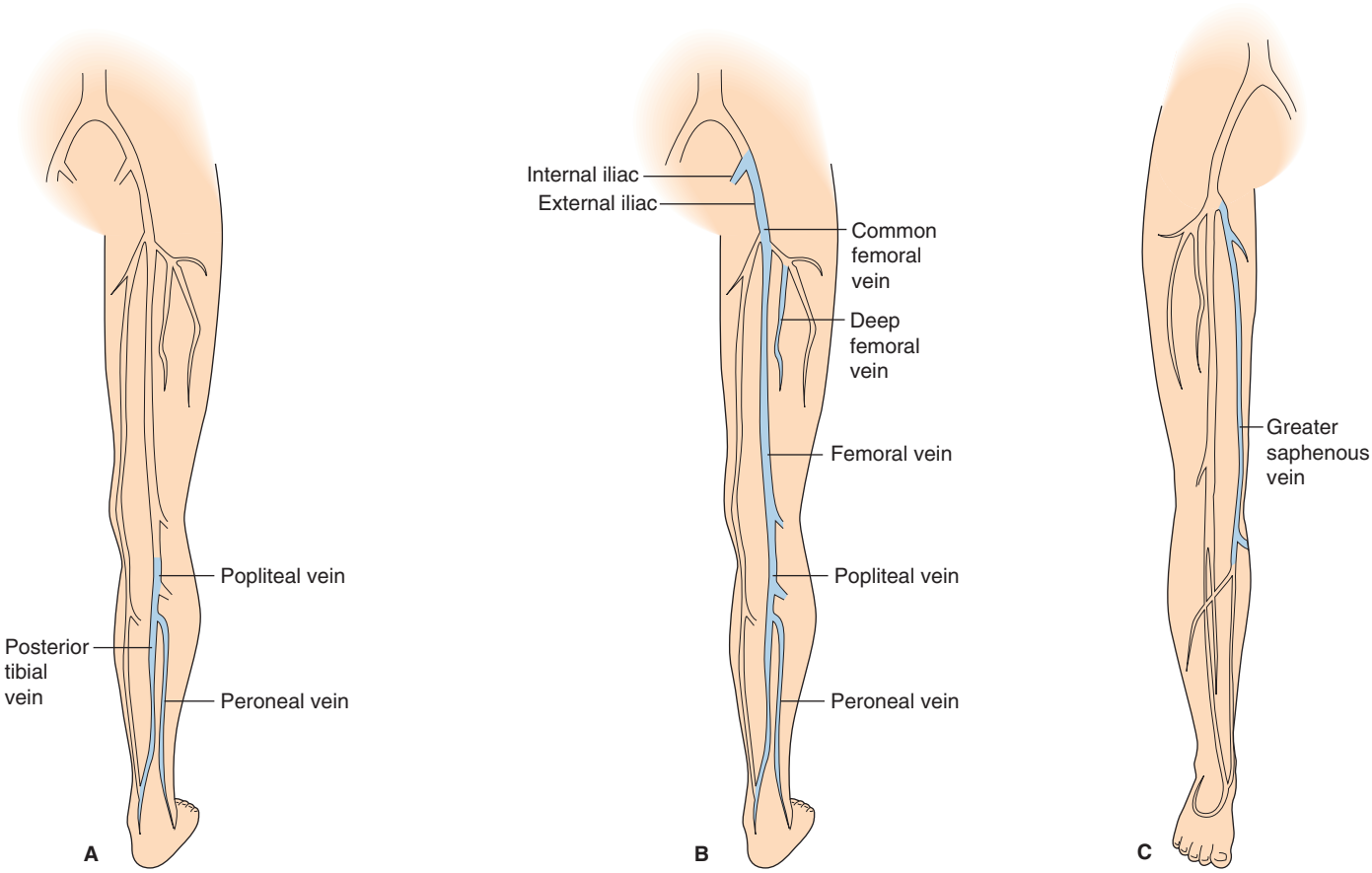


FIGURE 31.13 ■ Common locations of venous thrombosis. *A*, The most common sites of DVT. *B*, DVT extending from the calf to the iliac veins. *C*, Superficial venous thrombosis

Plethysmography is most valuable in diagnosing thromboses of larger or more superficial veins.

- *Magnetic resonance imaging (MRI)* is another non-invasive means of detecting DVT. It is particularly useful when thrombosis of the venae cavae or pelvic veins is suspected.
- *Ascending contrast venography* uses an injected contrast medium to assess the location and extent of venous thrombosis. Although invasive, expensive and uncomfortable, contrast venography is the most accurate diagnostic tool for venous thrombosis. It is used when the results of less invasive tests leave the diagnosis unclear (Papadakis et al., 2013).

Prophylaxis

Medications and other measures are used to prevent venous thrombosis when the risk is high. Low-molecular-weight (LMW) heparins prevent DVT in people who are undergoing general or orthopaedic surgery, experiencing acute medical illness or are on prolonged bed rest. Oral anticoagulation also may be used as a prophylactic measure in people with fractures or who are undergoing orthopaedic surgery.

Elevating the foot of the bed with the knees slightly flexed promotes venous return. Early mobilisation and leg exercises such as ankle flexion and extension assist venous flow by muscle compression. Intermittent pneumatic compression devices applied to the legs are effective to prevent DVT. They also are used when anticoagulation is contraindicated due to the increased risk of bleeding (Kasper et al., 2015). Elastic stockings are also used to prevent venous thrombosis in people at risk.

Medications

Anticoagulants to prevent clot propagation and enable the body's own lytic system to dissolve the clot are the mainstay of treatment for venous thrombosis. Fibrinolytic drugs such as streptokinase or tissue plasminogen activator (t-PA) may accelerate the process of clot lysis and prevent damage to venous valves. There is, however, no evidence that fibrinolytic therapy is more effective in preventing pulmonary embolism in people with existing DVT than anticoagulants (Kasper et al., 2015). It also significantly increases the risk of bleeding and haemorrhage.

Non-steroidal anti-inflammatory agents (NSAIDs) such as indomethacin or naproxen may be ordered to reduce inflammation in the veins and provide symptomatic relief, particularly for people with superficial venous thrombosis.

ANTICOAGULANTS Anticoagulants are given to prevent clot extension and reduce the risk of subsequent pulmonary embolism. See the 'Medication administration' box below for the nursing implications for anticoagulant therapy.

MEDICATION ADMINISTRATION Anticoagulant therapy

HEPARIN

Heparin interferes with the clotting cascade by inhibiting the effects of thrombin and preventing the conversion of fibrinogen to fibrin. This prevents the formation of a stable fibrin clot. At therapeutic levels, heparin prolongs the thrombin time, clotting time and activated partial thromboplastin time. When given intravenously, its effect is immediate. Given subcutaneously, its onset of action is within 1 hour. *Heparin-induced thrombocytopenia (HIT)* is a potential complication of therapy with unfractionated heparin. See Chapter 32 for more information about HIT and nursing responsibilities in monitoring for this dangerous potential complication.

Nursing responsibilities

- Assess for history of unexplained or active bleeding. Assess laboratory results for abnormal clotting profile or evidence of active bleeding.
- Give a test dose as indicated to people with a history of multiple allergies or a history of asthma.
- Administer by deep subcutaneous injection; abdominal sites are preferred. Avoid injecting within 5 cm of the umbilicus. Rotate sites. Do not aspirate prior to injecting or massage after the injection.
- Intravenous solutions may be diluted with dextrose, normal saline or Ringer's solution. Use an infusion pump.
- Keep protamine sulfate, a heparin antagonist, available to treat excessive bleeding.
- Monitor and report abnormal laboratory results and APTT values outside the desired range.
- Promptly report evidence of bleeding such as haematemesis, haematuria, bleeding gums or unexplained abdominal or back pain.

Health education for the person and family

- Report unusual bleeding or excessive menstrual flow.
- Use an electric razor and a soft-bristle toothbrush; prevent injury by clearing pathways, using a night light and other measures. Do not consume alcohol.
- Avoid contact sports while on anticoagulant therapy.
- Do not consume large amounts of food rich in vitamin K (yellow and dark green vegetables).
- Do not use aspirin or NSAIDs while on heparin therapy unless advised to do so by your doctor.
- Wear a MedicAlert® tag and advise all healthcare providers (including dentists and podiatrists) of therapy.

LOW-MOLECULAR-WEIGHT HEPARINS

Enoxaparin
Dalteparin
Sapropterin
Danaparoid

LMW heparins are the most bioavailable fraction of heparin. They provide a more precise and predictable anticoagulant effect than unfractionated heparins. Like unfractionated heparin, LMW heparin prevents conversion of prothrombin to thrombin, liberation of thromboplastin from platelets and formation of a stable clot. LMW heparins cannot be used interchangeably with each other or with unfractionated heparin. Although the risk of heparin-induced thrombocytopenia is significantly lower with LMW heparin, people who were previously treated with unfractionated heparin may develop HIT when treated with LMW heparin.

Nursing responsibilities

- Assess for evidence of active bleeding, a history of bleeding disorders or thrombocytopenia, or sensitivity to heparin, sulfites or pork products.
- Monitor for unusual or masked bleeding. PT and APTT levels may be within normal levels even in the presence of haemorrhage.
- Administer by deep subcutaneous injection into abdominal wall, thigh or buttocks. Rotate sites. Do not aspirate or massage.

Health education for the person and family

- Subcutaneous self-administration technique, timing of doses and site rotation. To minimise bruising, do not rub site after administering.
- Do not take aspirin, NSAIDs or other over-the-counter drugs unless recommended by your doctor.
- Promptly report excessive bruising or bleeding, chest pain, difficulty breathing, itching, rash or swelling to your healthcare provider.
- Keep follow-up appointments as scheduled.

ORAL ANTICOAGULANT

Warfarin

Warfarin interferes with synthesis of vitamin-K-dependent clotting factors by the liver, leading to depletion of these factors. It has no effect on already circulating clotting factors or on existing clots. Warfarin inhibits extension of existing thrombi and the formation of new clots. Its action is cumulative and more prolonged than that of heparin.

Nursing responsibilities

- Assess laboratory results and history for evidence of abnormal bleeding.
- Multiple drugs affect the metabolism and protein binding of warfarin; note all medications and assess for interactions with warfarin.
- Do not give during pregnancy because warfarin may cause congenital malformations.
- Oral tablets may be crushed and given without regard to meals.
- Dilute intravenous warfarin with supplied dilutant; administer within 4 hours by direct intravenous injection at a rate of 25 mg/min.
- Keep vitamin K available to reverse effects of warfarin in the event of excessive bleeding or haemorrhage.
- Monitor PT or INR; report values outside the desired range.

Health education for the person and family

- If bleeding occurs (haematemesis, bright red or black tarry faeces, haematuria, bleeding gums, excessive bruising, etc.), do not take your prescribed dose and

(continued)

MEDICATION ADMINISTRATION Anticoagulant therapy (continued)

notify your doctor immediately. Report rash or manifestations of hepatitis (dark urine, malaise, yellow skin or sclera).
- Take your warfarin at the same time every day; do not change brands because their effects may differ.
- Menstrual bleeding may be slightly increased; contact your healthcare provider if it increases significantly. Use reliable birth control to prevent pregnancy while taking warfarin. Immediately contact your doctor if you think you may be pregnant.
- Take precautions to prevent injury and bleeding: use a soft-bristle toothbrush and electric razor, wear shoes and use a night light. Avoid participating in contact sports.
- Do not smoke, use alcohol or take any over-the-counter drugs unless specifically recommended by your doctor. Notify all healthcare providers, including dentists and podiatrists, of therapy. Wear a MedicAlert® tag.
- Obtain lab tests as scheduled and keep all scheduled follow-up appointments.

Anticoagulation is initiated with unfractionated heparin or low-molecular-weight (LMW) heparin. Following an initial intravenous bolus of 7500 to 10 000 units of unfractionated heparin, a continuous heparin infusion of 1000 to 1500 international units per hour (IU/h) is started. The dosage is calculated to maintain the activated partial thromboplastin time (APTT) at approximately twice the control or normal value. An infusion pump is used to deliver the prescribed dosage. Frequent monitoring of the infusion is an important nursing responsibility. Subcutaneous heparin injections may be used as an alternative to intravenous infusion in some instances.

LMW heparins are increasingly used to prevent and treat venous thrombosis. They do not require the close laboratory monitoring of unfractionated heparins. LMW heparin is administered subcutaneously in fixed doses once or twice daily, allowing for the option of community-based treatment. LMW heparins have additional advantages, in that they are more effective and carry lower risks for bleeding and thrombocytopenia than conventional, unfractionated heparins.

Oral anticoagulation with warfarin may be initiated concurrently with heparin therapy. Overlapping heparin and warfarin therapy for 4 to 5 days is important because the full anticoagulant effect of warfarin is delayed and it may actually promote clotting during the first few days of therapy (Papadakis et al., 2013). Warfarin doses are adjusted to maintain the INR at 2.0 to 3.0 (Kasper et al., 2015). Once this level is achieved, the heparin is discontinued and a maintenance dose of warfarin is prescribed to prevent recurrent thrombosis.

Anticoagulation generally is continued for at least 3 months. When DVT is recurrent or risk factors such as altered coagulability or cancer are present, anticoagulant therapy may be prolonged. Regular follow up is necessary to be sure prothrombin times (INR) remain within the desirable range for anticoagulation.

Treatments

Treatment of venous thrombosis also includes measures to relieve symptoms and reduce inflammation. With superficial venous thrombosis, applying warm, moist compresses over the affected vein, extremity rest and anti-inflammatory agents usually provide relief of symptoms.

Bed rest may be ordered for deep venous thrombosis. The duration of bed rest typically is determined by the extent of leg oedema. The legs are elevated 15 to 20 degrees, with the knees slightly flexed, above the level of the heart to promote venous return and discourage venous pooling. Elastic anti-embolism stockings (TEDS) or pneumatic compression devices are also frequently ordered to stimulate the muscle-pumping mechanism that promotes the return of blood to the heart. When permitted, walking is encouraged while avoiding prolonged standing or sitting. Crossing the legs also is avoided, as are tight-fitting garments or stockings that bind.

Surgery

Venous thrombosis usually is effectively treated with conservative measures and anticoagulation. In some cases, however, surgery is required to remove the thrombus, prevent its extension into deep veins or prevent the effects of embolisation.

Venous thrombectomy is done when thrombi lodge in the femoral vein and their removal is necessary to prevent pulmonary embolism or gangrene. Successful thrombus removal rapidly improves venous circulation. The duration of this effect varies.

When venous thrombosis is recurrent and anticoagulant therapy is contraindicated, a filter may be inserted into the vena cava to capture emboli from the pelvis and lower extremities, preventing pulmonary embolism. Several different filters are available (see Figure 31.14). The Greenfield filter is widely used for its ability to trap emboli within its apex while

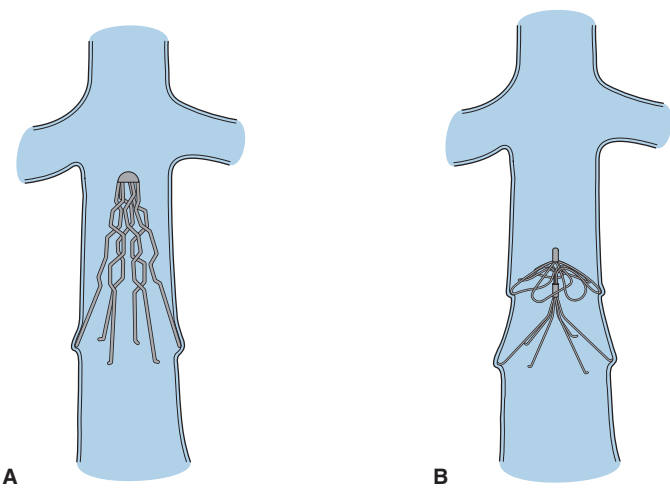


FIGURE 31.14 ■ Venal caval filters. *A*, Greenfield filter. *B*, Nitinol filter

maintaining patency of the vena cava. The filter can be inserted under fluoroscopy with local anaesthesia. Mortality and morbidity associated with the filter are very low.

Extensive thrombosis of the saphenous vein may necessitate ligation and division of the saphenous vein where it joins the femoral vein to prevent clot extension into the deep venous system. A vein affected by septic venous thrombosis is excised to control the infection. Antibiotic therapy also is initiated.

Nursing care

Health promotion

Prevention of venous thrombosis is an important component of nursing care for all at-risk people. Position people to promote venous blood flow from the lower extremities, with the feet elevated and the knees slightly bent. Avoid placing pillows under the knees and positions in which the hips and knees are sharply flexed. Use a recliner chair or footstool when sitting. Ambulation should be encouraged as soon as possible and a regular schedule of ambulation maintained throughout the day. Teach ankle flexion and extension exercises, and remind people to frequently perform them. Apply elastic compression stockings and pneumatic compression devices when appropriate. Instruct to avoid crossing legs when in bed or sitting. Inquire about possible prophylactic heparin or warfarin therapy for people undergoing orthopaedic surgery or other high-risk procedures. Frequently assess intravenous sites. Change the site and catheter as dictated by agency protocol and if evidence of local inflammation is noted.

Assessment

Assess people at risk of venous thrombosis for manifestations and risk factors.

- *Health history:* complaints of leg or calf pain, its duration and characteristics, and the effect of walking on the pain; history of venous thrombosis or other clotting disorders; current medications.
- *Physical examination:* inspect affected extremity for redness, oedema; palpate for tenderness, warmth, cordlike structures; body temperature.
- *Diagnostic tests:* clotting studies (activated partial thromboplastin time (APTT), prothrombin time (PT), International Normalized Ratio (INR)).

See the accompanying nursing care plan for an example of an assessment of a person with deep venous thrombosis.

Nursing diagnoses and interventions

In addition to the preventive measures identified earlier, priority nursing diagnoses for the person with venous thrombosis relate to pain, maintenance of tissue perfusion and integrity, and the potential adverse effects of prescribed treatments.

Pain

The pain associated with venous thrombosis results from inflammation of the involved vein. It may be aggravated by use of the involved extremity. Associated oedema and swelling may contribute to discomfort. Measures to reduce the inflammation often help relieve the pain.

- Regularly assess pain location, characteristics and level using a standardised pain scale. Report increasing pain or changes in its location or characteristics. *Tissue substances released during the inflammatory process can stimulate pain receptors. In addition, localised swelling presses on pain-sensitive structures in the area of the inflammation, contributing to discomfort. As inflammation and swelling are reduced, pain should abate. Continued or increasing pain may indicate extension of the thrombosis. Sudden chest pain may indicate a pulmonary embolism, necessitating immediate intervention.*
- Measure calf and thigh diameter of the affected extremity on admission and daily thereafter. Report increases promptly. *The inflammatory process causes vasodilation and increases vessel permeability, causing oedema of the affected extremity. Baseline and subsequent measurements provide a measure of treatment effectiveness.*
- Apply warm, moist heat to affected extremity at least four times daily, using warm, moist compresses or an aqua-K pad. *Moist heat penetrates tissues to a greater depth. Warmth promotes vasodilation, allowing reabsorption of excess fluid into the circulation. Vasodilation also reduces resistance within the affected vessel, reducing pain. As oedema subsides, pressure on surrounding tissues is relieved, thereby reducing pain.*
- Maintain bed rest as ordered. *Using leg muscles during walking exacerbates the inflammatory process and increases oedema. This, in turn, increases venous compression and pain.*

Ineffective tissue perfusion: peripheral

As thrombi develop, they occlude the lumen of the vein and obstruct blood flow. In addition, the accompanying inflammatory response may precipitate vessel spasms, further impairing arterial and venous blood flow and tissue perfusion. Impaired tissue perfusion, in turn, deprives tissues of nutrients and oxygen. As a result, distal tissues of the affected extremity are at risk of ulceration and infection.

CONSIDERATION FOR PRACTICE

Assess peripheral pulses, skin integrity, capillary refill times and colour of extremities at least every 8 hours. Report changes promptly. Assessment of both extremities allows comparison of the affected and unaffected limbs. Weak or absent pulses, impaired capillary refill or significant colour changes in the affected extremity may indicate extension of the thrombus or a possible complication.

- Assess skin of the affected lower leg and foot at least every 8 hours; more often as indicated. *Frequent assessment is important to rapidly detect early signs of tissue breakdown*

NURSING CARE PLAN A person with deep venous thrombosis

Mrs Opal Hipps, aged 75, lives alone with her dog, Roxy, in her family home in the western suburbs of Melbourne. She retired from her job at the post office 10 years ago and now spends a lot of time reading and watching television. Over the past week she has developed a vague aching pain in her right leg. She ignored the pain until last night when it developed into a much more severe pain in her right calf. She noticed that her right lower leg seemed larger than the left and it was very tender to the touch. After seeing her doctor and undergoing Doppler ultrasound studies, Mrs Hipps is admitted to the hospital with the diagnosis of deep venous thrombosis in the right leg. She is placed on bed rest and intravenous heparin. Michael Cookson, RN, is assigned to admit and care for Mrs Hipps.

ASSESSMENT

RN Cookson notices that Mrs Hipps was admitted 14 months ago for repair of a fractured femur. Mrs Hipps says, 'This business about a blood clot really has me worried.' She also tells Mr Cookson that she is worried about who will care for her dog while she is in the hospital. Physical findings include height 157 cm, weight 68 kg, T 37.3°C; vital signs within normal limits otherwise. Her left leg is warm and pink, with strong peripheral pulses and good capillary refill. Her right calf is dark red, very warm and dry to touch. It is tender to palpation. The right femoral and popliteal pulses are strong, but the pedal and posterior tibial pulses are difficult to locate. The right calf diameter is 1.27 cm larger than the left.

DIAGNOSES

- *Pain* related to inflammatory response in affected vein.
- *Anxiety* related to unexpected hospitalisation and uncertainty about the seriousness of her illness.
- *Ineffective tissue perfusion: peripheral* related to decreased venous circulation in the right leg.
- *Risk of impaired skin integrity* related to pooling of venous blood in the right leg.

PLANNING

- Plan time with Mrs Hipps to explain venous thrombosis and its treatment.
- Discuss arrangements for a friend or neighbour to care for Mrs Hipps's dog.
- Plan time to liaise with interprofessional team for assessment and interventions.

Expected outcomes

- Verbalise relief of right leg pain by day of discharge.
- Verbalise reduced anxiety by the second day of her hospitalisation.
- Demonstrate reduced right leg diameter by 0.65 cm by the fifth day of hospitalisation.
- Maintain intact skin in the right foot throughout the hospital stay.

IMPLEMENTATION

- Elevate legs, maintaining slight knee flexion, while in bed.
- Apply warm, moist compresses to right leg using a 2-hour-on, 2-hour-off schedule around the clock.
- Administer prescribed analgesics and evaluate effectiveness.
- Spend time with Mrs Hipps to explain venous thrombosis and its treatment.
- Arrange for a friend or neighbour to care for Mrs Hipps's dog.
- Apply anti-embolism stockings as ordered; remove for 30 minutes every 8 hours.
- Monitor laboratory values to assess effect of anticoagulant therapy; report values outside desired range.
- Assist with progressive ambulation when allowed.
- Inspect legs and feet and record findings every 8 hours.

EVALUATION

Seven days after admission, the pain in Mrs Hipps's right leg has subsided and the diameter of her right calf is equal to that of her left calf. Mrs Hipps admits to RN Cookson that her fears really relate to a cousin who was hospitalised for a similar problem and had his leg amputated. After talking about her condition and the steps she can take to prevent its recurrence, she is much less anxious. Before discharge, Mr Cookson reviews instructions for anti-embolism stockings, daily walking, warfarin schedule and scheduled follow-up appointment. Mrs Hipps's neighbour, Kate, has come to pick her up. As Mr Cookson is helping Mrs Hipps into the car, Kate hands her a small brown dog and says, 'I took good care of Roxy for you, but he's missed you.' Mrs Hipps smiles and assures Mr Cookson that she will call the number he provided if she has any questions.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the pathophysiological reasons for the pain in Mrs Hipps's right leg.
2. How would you respond if Mrs Hipps tells you she does not have the money to buy the prescribed anticoagulant when she goes home?
3. How would you change your teaching and discharge planning if Mrs Hipps had difficulty caring for herself?
4. Design a plan of care for Mrs Hipps for the nursing diagnosis *Activity intolerance*.

REFLECTION ON THE NURSING PROCESS

1. Focusing on the elements of listening to the person, what in Mrs Hipps's case would be important when obtaining a patient history?
2. Discuss four priorities of education pertinent to Mrs Hipps's presentation.

and implementation of measures to protect vulnerable tissues. Early intervention allows healing and restoration of tissue integrity; allowed to continue, the process can lead to necrosis and potential gangrene.

- Elevate extremities at all times, keeping knees slightly flexed and legs above the level of the heart. *Elevation of the extremities promotes venous return and reduces peripheral oedema. Knee flexion promotes muscle relaxation.*
- Use mild soaps, solutions and lotions to clean the affected leg and foot daily. Pat dry after washing and apply a non-alcohol-based lotion or moisturising cream. *Daily*

CONSIDERATION FOR PRACTICE

Remove anti-embolic stockings or pneumatic compression device for 30 to 60 minutes during daily hygiene. Anti-embolic stockings and pneumatic compression devices exert pressure on the extremity and promote venous return. They can, however, impair perfusion of the dermis. Removing them periodically allows assessment of the underlying tissue and restores perfusion of the dermis, reducing the risk of skin breakdown. Their use may be continued following discharge to reduce the risk of recurrent venous thrombosis.

hygiene with non-drying soaps and solutions removes potential pathogens from the skin surface and maintains skin integrity and the first line of defence against infection. Caustic or harsh soaps or solutions can dry and crack the skin. Dry, cracked skin permits bacteria and other microorganisms to enter and infect the tissue, potentially leading to ulceration and venous gangrene.

- Use an egg-crate mattress or sheepskin on the bed as needed. *Egg-crate mattresses and sheepskins distribute weight more evenly, preventing excess pressure on affected tissues.*
- Encourage frequent position changes, at least every 2 hours while awake. *Frequent position changes reduce pressure on bony prominences and oedematous tissue, reducing the risk of tissue breakdown.*

Ineffective protection

Anticoagulant therapy interferes with the body's normal clotting mechanisms, increasing the risk of bleeding and haemorrhage.

- Monitor laboratory results, including the INR (prothrombin time), APTT, haemoglobin and haematocrit, as indicated. Report values outside the normal or desired range. *Coagulation studies are used to monitor the effect of anticoagulant medications. Values within the desired range prevent further clot development while carrying a low risk of bleeding and haemorrhage. A fall in the haemoglobin and haematocrit may indicate undetected bleeding.*

CONSIDERATION FOR PRACTICE

Assess for and promptly report evidence of bleeding, such as petechiae, bruising, bleeding gums, obvious or occult blood in vomitus, stool or urine, unexplained back or abdominal pain. Anticoagulants interfere with the ability to form a stable clot and prevent excessive bleeding. Even minor trauma such as tooth brushing or bumping into furniture can result in bleeding.

Impaired physical mobility

Although prolonged bed rest rarely is required, it is associated with many problems, including constipation, joint contractures, muscle atrophy and boredom. Nursing care goals include maintaining joint range of motion, minimising muscle atrophy and reducing boredom.

- Encourage active range-of-motion (ROM) exercises at least every 8 hours. Provide passive range of motion as needed. *ROM exercises maintain joint mobility and prevent contractures. Active range of motion (performed by the person) also helps prevent muscle atrophy and preserve function. While passive ROM exercises do not prevent muscle atrophy, they do maintain joint mobility.*
- Encourage frequent position changes, deep breathing and coughing. *Prolonged immobility can lead to impaired airway clearance and respiratory complications, such as atelectasis or pneumonia. Turning, coughing and deep breathing facilitate expulsion of secretions from the respiratory tract, airway clearance and alveolar ventilation.*
- Encourage increased fluid and dietary fibre intake. *Constipation is a frequent complication of immobility due to decreased gastrointestinal motility and loss of abdominal muscle strength. Increasing fluid and fibre intake helps maintain soft, easily expelled stools.*
- Assist with and encourage ambulation as allowed. *Ambulation promotes venous blood flow, helps maintain muscle tone and joint mobility, and increases the sense of wellbeing.*
- Encourage diversional activities such as reading, handiwork or other hobbies, television or video games, and socialising. *Boredom may lead to dozing and inertia, with little physical movement or mental stimulation, increasing the risk of complications of immobility.*

Risk of ineffective tissue perfusion: cardiopulmonary

A thrombus that forms in the deep veins of the legs or pelvis may break loose or fragment, becoming an embolism. Emboli that originate in the venous system usually become trapped in the pulmonary circulation (pulmonary embolism). Gas exchange in the affected area is impaired as blood flow ceases or is reduced to an area of the lungs that is well ventilated (see Chapter 35).

- Frequently assess respiratory status, including rate, depth, ease and oxygen saturation levels. *A mismatch of ventilation and perfusion can significantly affect gas exchange, leading to rapid, shallow respirations, dyspnoea and air hunger, and a fall in oxygen saturation levels.*
- Initiate oxygen therapy, elevate the head of the bed and reassure the person who is experiencing manifestations of pulmonary embolism. *Oxygen therapy and elevating the head of the bed promote ventilation and gas exchange in those alveoli that are well perfused, helping maintain tissue oxygenation. Reassurance helps reduce anxiety and slow the respiratory rate, promoting greater respiratory depth and alveolar ventilation.*

CONSIDERATION FOR PRACTICE

Immediately report complaints of chest pain and shortness of breath, anxiety or a sense of impending doom. The manifestations of pulmonary embolism are similar to those of myocardial infarction. Prompt intervention to restore pulmonary blood flow can reduce the risk of significant adverse effects.

Community-based care

Treatment measures for venous thrombosis may be initiated and carried out on an outpatient basis or continued for an extended period of time following hospital discharge. Include the following topics when teaching for home care:

- explanation of the disease process
- treatment measures, including laboratory tests and their purposes, medications and adverse effects that should be reported
- appropriate methods of heat application
- prescribed activity restrictions
- measures to prevent future episodes of venous thrombosis
- the importance of follow-up visits and laboratory tests as scheduled.

Refer people for community nursing services for continued assessment and reinforcement of teaching. Provide referrals for assistance with ADLs and home maintenance services as indicated. Consider referral for physical therapy if needed.

SELECTED PERIPHERAL VENOUS CONDITIONS

THE PERSON WITH CHRONIC VENOUS INSUFFICIENCY

Chronic venous insufficiency is a disorder of inadequate venous return over a prolonged period. Deep venous thrombosis (DVT) is the most frequent cause of chronic venous insufficiency. Other conditions, such as varicose veins or leg trauma, may contribute; in some instances, it develops without an identified precipitating cause (Kasper et al., 2015; Papadakis et al., 2013).

Pathophysiology

Following DVT, large veins may remain occluded, increasing the pressure in other veins of the extremity. This increased pressure distends the veins, separating valve leaflets and impairing their ability to close. DVT also damages valve leaflets, causing them to thicken and contract. The result is impaired unidirectional blood flow and deep vein emptying (Patel, 2014).

When venous valves are incompetent, the muscle-pumping action produced during activity cannot propel blood back to the heart. Venous blood collects and stagnates in the lower leg (**venous stasis**). Venous pressures in the calf and lower leg increase, particularly during ambulation. This increased pressure impairs arterial circulation to the lower extremities as well. The body's ability to provide sufficient oxygen and nutrients to the cells and remove metabolic waste products diminishes. Eventually, there is so little oxygen and nutrients that cells begin to die. The skin atrophies and subcutaneous fat deposits necrose. Breakdown of red blood cells in the congested tissues causes brown skin pigmentation (Flugman, 2014). Venous stasis ulcers develop. Congested tissues impair the body's ability to increase the supply of oxygen, nutrients and metabolic energy to heal the ulcer. As a result, the condition worsens and, over time, the ulcers enlarge. The congested venous circulation also prevents the blood from mounting effective inflammatory and immune responses, significantly increasing the risk of infection in the ulcerated tissue (Flugman, 2014).

Manifestations

Manifestations of chronic venous insufficiency include lower leg oedema, itching and discomfort of the affected extremity that increase with prolonged standing. The extremity is cyanotic. Recurrent stasis ulcers develop (see Figure 31.15), usually forming just above the ankle, on the medial or anterior aspect of the leg. They heal poorly, forming scar tissue that breaks down easily. Tissue surrounding the ulcer is shiny, atrophic and cyanotic, and there is a brownish pigmentation to the skin. Other skin changes may develop as well, such as

MANIFESTATIONS Chronic venous insufficiency

- Lower extremity oedema that worsens with standing
- Itching, dull leg discomfort or pain that increases with standing
- Thin, shiny, atrophic skin
- Cyanosis and brown skin pigmentation of lower leg and foot
- Possible weeping dermatitis
- Thick, fibrous (hard) subcutaneous tissue
- Recurrent ulcerations of medial or anterior ankle

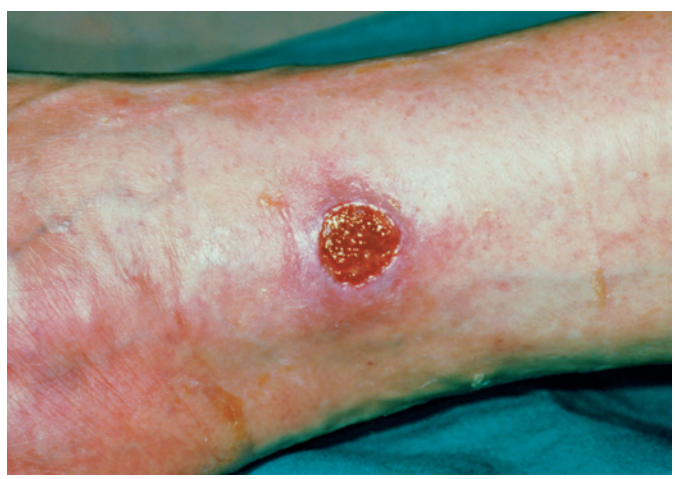


FIGURE 31.15 ■ Chronic venous insufficiency. Note the discolouration of the ankle and the stasis ulcer

Source: Dr P. Marazzi/Science Source.

TABLE 31.5 Comparison of arterial and venous leg ulcers

FACTOR	ARTERIAL ULCERS	VENOUS ULCERS
Location	Toes, feet, shin	Over medial or anterior ankle
Ulcer appearance	Deep, pale	Superficial, pink
Skin appearance	Normal to atrophic Pallor on elevation Rubor on dependency	Brown discolouration Stasis dermatitis Cyanosis on dependency
Skin temperature	Cool	Normal
Oedema	Absent or mild	May be significant
Pain	Usually severe Intermittent claudication Rest pain	Usually mild Aching pain
Gangrene	May occur	Does not occur
Pulses	Decreased or absent	Normal

eczema or stasis dermatitis. Necrosis and fibrosis of subcutaneous tissue cause the affected area of the leg to feel hard and somewhat leathery to the touch, but even the slightest trauma to the area can produce serious tissue breakdown. See the box above for the manifestations of chronic venous insufficiency. Table 31.5 compares venous and arterial ulcers.

INTERPROFESSIONAL CARE

Collaborative care for the person with venous insufficiency focuses on relieving symptoms, promoting adequate circulation and healing and preventing tissue damage.

Diagnosis

The history and physical examination often establish the diagnosis of chronic venous insufficiency. Because a history of DVT is a major risk factor, careful evaluation of the past medical history and questioning of the person is important. There are no specific diagnostic tests to confirm the diagnosis of chronic venous insufficiency.

Lifestyle modifications

Conservative management of venous insufficiency focuses on reducing oedema and treating ulcerations. Prolonged standing or sitting is discouraged. Graduated compression hosiery is ordered for daytime use and frequent elevation of the legs and feet during the day is recommended. At night, the legs and feet should be elevated above the level of the heart by raising the foot of the mattress.

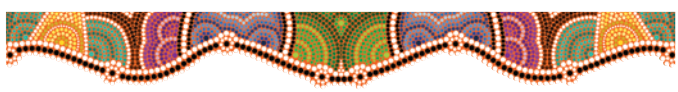
Treatment

Treatment of associated stasis dermatitis varies, based on the duration of the condition. Wet compresses of boric acid, buffered aluminium acetate (Burow's solution) or isotonic saline solution are applied to acute weeping dermatitis four times a day for 1-hour periods. Following the compress, a topical corticosteroid (such as 0.5% hydrocortisone cream) is applied. Bed rest is prescribed during the acute period. Stasis dermatitis that is subsiding or chronic may be treated with a topical corticosteroid, zinc oxide ointment or a topical broad-spectrum antifungal cream such as clotrimazole cream or miconazole cream (Papadakis et al., 2013).

Isotonic saline compresses or wet-to-dry dressings are applied to stasis ulcers to promote healing. A dilute topical antibiotic solution also may be used (Kasper et al., 2015). The ulcer may be treated by using a semi-rigid boot applied to the foot and lower leg. This device may be made of Unna's paste or Gauzetex bandage. Bony prominences must be well padded. The boot must be changed every 1 to 2 weeks, depending on the amount of drainage from the ulcer. This device often allows ambulatory treatment.

Surgery

A very large, chronic ulcer may require surgery. In this case, the incompetent veins are ligated, the ulcer is excised and the area is covered with a skin graft (see Chapter 16).



Nursing care

Education

Nursing care for the person with chronic venous insufficiency is primarily educative and supportive. Teaching includes the following recommendations:

- Elevate the legs while resting and during sleep. See the 'Translation to practice' box below for a nursing research study that suggests the supine position for resting.
- Walk as much as possible, but avoid sitting or standing for long periods of time.
- When sitting, do not cross your legs or allow pressure on the back of the knees (such as sitting on the side of the bed).
- Do not wear anything that pinches your legs (such as knee-high stockings, garters or girdles).

TRANSLATION TO PRACTICE Evidence-based practice for the person with venous leg ulcers

A study by Finlayson, Wu and Edwards (2015) analysed data from 250 adults recruited from two hospital and three community-based wound clinics. The study participants had healed leg ulcers of mostly venous aetiology. Various parameters were recorded and analysed for risk factors which may have contributed to recurrence of a leg ulcer and for protective factors which may have reduced the likelihood of leg ulcer recurrence.

The group discovered that the four key risk factors for recurrence of venous ulcers were age, history of deep venous thrombosis, history of multiple previous leg ulcers and total duration of the previous ulcer. The data also suggested that the protective factors preventing leg ulcer recurrence were leg elevation, walking and self-efficacy (*having control over one's ability to succeed*).

IMPLICATIONS FOR NURSING

The results of this study are important for nurses to understand factors that may promote or reduce the risk of a person developing recurrence of a leg ulcer. This knowledge can be used in education sessions and primary and community healthcare situations to reduce a person's morbidity or to predict the individuals who would most benefit from further support and information.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. When designing an education session on venous leg ulcer recurrence, which identified factors are modifiable and which are non-modifiable? How will these considerations influence the information provided?
2. When designing an education session on venous leg ulcer recurrence, what specific information should be provided regarding the method and duration of leg elevation? What specific information should be provided regarding the distance, type and duration of walking? Can a nurse influence a person's self-efficacy? Explain.
3. How will this information influence your future practice when caring for individuals with current or previous venous ulcers?

- Wear elastic compression stockings as prescribed. They should be tighter over the feet than at the top of the leg. Be sure the tops of the stockings do not cut into your legs. Put them on after your legs have been elevated.
- Keep the skin on your feet and legs clean, soft and dry. Follow guidelines in Box 31.5 for care of the legs and feet.

The following nursing diagnoses may apply to the person with chronic venous insufficiency.

- *Disturbed body image* related to oedema and stasis ulcers on lower leg.
- *Ineffective health maintenance* related to lack of knowledge about disorder and prescribed treatments.
- *Risk of infection* related to ulcerations.
- *Impaired physical mobility* related to pain and oedema in lower legs.
- *Impaired skin integrity* related to presence of stasis ulcers.
- *Ineffective tissue perfusion: peripheral* related to incompetent venous valves.

See other sections of this chapter for specific nursing interventions related to many of these diagnoses. See the box below for nursing care for the older adult with chronic venous stasis.

THE PERSON WITH VARICOSE VEINS

Varicose veins are irregular, tortuous veins with incompetent valves. Varicosities may develop in any vein and may be called by other names, such as haemorrhoids in the rectum and varices in the oesophagus. Varicosities usually affect the veins of the lower extremities; the long saphenous vein is often affected and they also may develop in the short saphenous vein.

Incidence and risk factors

Varicose veins affect about 10–15% of men and 20–25% of women in industrialised nations (Sheehan, 2015). They are more common in women over age 35. Studies also suggest that the increased risk of varicose veins in women may relate to venous stasis during pregnancy. Ageing is a risk factor, possibly related to decreased exercise and other factors that contribute to venous stasis. People in occupations that involve prolonged standing (such as beauticians, salespeople and nurses) also have an increased incidence of varicose veins. Race is a risk factor: white-skinned people are more frequently affected than dark. The majority of people with primary varicose veins (those affecting superficial veins) have a family history of the disorder, suggesting a genetic link (Kasper et al., 2015). A person is at approximately 90% risk of developing varicose veins if both parents are affected, and approximately 62% risk if one parent is affected (Sheehan, 2015).

Most varicosities occur in the deep veins of the legs. Contributing causes include obesity, venous thrombosis, congenital arteriovenous malformations or sustained pressure on abdominal veins (as in pregnancy and/or the presence of abdominal tumours). The effects of gravity, produced by long periods of standing, are a major causative factor.

Pathophysiology

Varicose veins are classified as primary (with no involvement of deep veins) or secondary (caused by the obstruction of deep veins). In both cases, long-standing increased venous pressure stretches the vessel wall. This sustained stretching impairs the ability of the venous valves to close, causing them to become incompetent.

The erect position produces a twofold negative effect on the veins. When standing, the leg veins resemble vertical columns

NURSING CARE OF THE OLDER ADULT Chronic venous stasis

Disorders of venous stasis are common after the fifth decade of life. Ageing affects vessels and tissues, increasing the risk of venous insufficiency and varicose veins. In addition, mobility frequently declines with ageing, reducing the effect of the muscle pump in promoting venous return.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

Regular exercise—walking, in particular—is an important part of the treatment plan. Safety when walking is an important issue for older people. Assess the person's mobility and stability during ambulation. If appropriate, suggest using a walker and quad-cane as needed. Assist older people holding jobs that require prolonged standing to identify strategies to minimise standing and incorporate periods of activity into their work.

HOME CARE

Following surgery or during treatment for stasis ulcers, older people may need additional assistance with home care and maintenance. Initiate referral to social services as needed to arrange for home nursing care, meals, assistance with ADLs and home maintenance services as indicated. In some instances, temporary placement in an extended care facility is necessary until the person and family can assume care.

and must withstand the full force of venous blood pressure. Prolonged standing, the force of gravity, lack of leg exercise and incompetent venous valves all weaken the muscle-pumping mechanism, reducing venous blood return to the heart. As standing continues, the amount of blood pooled in the veins increases, further stretching the vessel wall. The venous valves become increasingly incompetent.

Manifestations

Although varicose veins may be asymptomatic, most cause manifestations such as severe aching leg pain, leg fatigue, leg heaviness, itching or feelings of heat in the legs. The degree of valvular incompetence does not seem to correlate well with the extent of symptoms. The menstrual cycle tends to worsen symptoms, suggesting a possible correlation with hormonal factors in women. Assessment reveals obvious dilated, tortuous veins beneath the skin of the upper and lower leg. If varicose veins are long standing, the skin above the ankles may be thin and discoloured, with a brown pigmentation. (See the 'Manifestations' box below.)

Complications

Complications of varicose veins include venous insufficiency and stasis ulcers. Chronic stasis dermatitis may also develop. Superficial venous thrombosis may develop in varicose veins, especially during and after pregnancy, following surgery and in people on oestrogen therapy (oral contraceptives or hormone replacement therapy).

MANIFESTATIONS Varicose veins

- Severe, aching pain in the leg
- Leg fatigue, heaviness
- Itching of the affected leg (stasis dermatitis)
- Feelings of warmth in the leg
- Visibly dilated veins
- Thin, discoloured skin above the ankles
- Stasis ulcers

INTERPROFESSIONAL CARE

Varicose veins usually can be managed using conservative measures, although surgery may be required if symptoms are severe, when complications develop or for cosmetic reasons.

Diagnosis

While varicose veins often are diagnosed by the history and physical examination, diagnostic tests may be ordered.

- *Doppler ultrasonography* or *duplex Doppler ultrasound* may be performed to identify specific locations of incompetent valves. This test is particularly useful before surgery to identify valves that allow reflux of blood from the femoral, popliteal or peripheral deep veins into the superficial veins (Papadakis et al., 2013).
- *Trendelenburg test* may be performed to determine the underlying cause of superficial venous insufficiency. The leg is elevated, then an elastic tourniquet is placed around the distal thigh. The varicosities then are observed as the person stands. When valves of the deep veins are incompetent, the veins remain flat on standing; they rapidly distend when the superficial venous valves are the underlying cause.

Treatments

Although there is no real cure, conservative measures are the core of treatment for most people with uncomplicated varicose veins. These measures often relieve symptoms and prevent complications by improving venous circulation and relieving pressure on venous tissues. Properly fitted graduated compression stockings are commonly prescribed. They compress the veins, propelling blood back to the heart. Compression stockings augment the muscle pumping action of the legs. When worn during times of prolonged standing and in combination with frequent leg elevation, compression stockings often prevent progression of the condition and development of complications.

Regular, daily walking also is important. Prolonged sitting and standing are discouraged, although elevating the legs for specified periods during the day is beneficial. Leg elevation

promotes venous return, prevents venous stasis and decreases leg heaviness and fatigue.

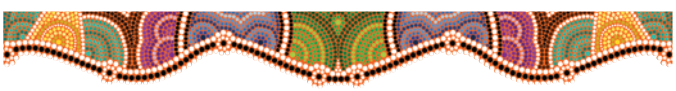
COMPRESSION SCLEROTHERAPY In compression sclerotherapy, a sclerosing solution is injected into the varicose vein and a compression bandage is applied for a period of time. This obliterates the vein. Venous blood is rerouted through healthy vessels whose valves are not compromised. Compression sclerotherapy may be used to treat small, symptomatic varicosities. It may be the primary treatment or it may be used in conjunction with varicose vein surgery. While compression sclerotherapy may be done for cosmetic reasons, complications such as phlebitis, tissue necrosis or infection may occur and need to be considered prior to the procedures.

Surgery

Surgical treatment of varicose veins generally is reserved for people who are very symptomatic, experience recurrent superficial venous thrombosis, and/or develop stasis ulcers. The objective of surgery is to remove the diseased veins. It may be considered for cosmetic reasons.

Surgery usually involves extensive ligation and stripping of the greater and lesser saphenous veins (Kasper et al., 2015). The evening before surgery, the surgeon marks all incompetent superficial and perforating varicose veins with a permanent ink marker. Under either regional or general anaesthesia, the greater saphenous vein is removed and the connected smaller tributaries that have not naturally clotted off are tied off. Multiple small incisions may be made over the varicosities, allowing removal of the affected segments of the vein. Incompetent tributaries that communicate with larger vessels also are ligated. For people with less extensive disease or people seeking cosmetic improvement, surgery may involve only the removal of the lesser saphenous vein through an incision in the popliteal fossa.

Postoperative care includes applying pressure bandages for a minimum of 6 weeks, elevating the extremities to minimise postoperative oedema and gradually increasing amounts of ambulation. Sitting and standing are prohibited during the initial recovery period and are gradually reintroduced as deemed appropriate by the surgeon.



Nursing care

Health promotion

Health promotion activities to reduce the incidence of varicose veins include teaching all people, particularly young women, the benefits of regular exercise continued over the lifetime. Discuss the effect of prolonged sitting or standing on the legs and encourage the person whose occupation involves these activities to periodically get up and move or to sit with the legs elevated. Encourage everyone to maintain normal weight for his or her height.

Assessment

Focused assessment of the person with varicose veins includes the following:

- *Health history:* complaints of leg pain, aching, heaviness or fatigue; ankle swelling; history of venous thrombosis.
- *Physical examination:* visible, dilated, tortuous superficial veins in lower extremities.

Nursing diagnoses and interventions

In planning and providing nursing care for people with varicose veins, emphasis is placed on the importance of health teaching to manage the symptoms of varicose veins, particularly because there is no cure for the disease. Nursing care for people who have undergone surgical treatment for varicose veins focuses on assessing and promoting wound healing and preventing infection. Nursing diagnoses may include those related to pain, impaired tissue perfusion and skin integrity, and a risk of impaired neurovascular function.

Chronic pain

Varicose veins can lead to pooling of venous blood in the lower extremities. Venous congestion can cause a dull ache or feeling of pressure in the legs, particularly after prolonged standing. As venous pressure rises, arterial circulation and delivery of oxygen and nutrients to tissues is impaired. Tissue ischaemia contributes to the pain. The pain associated with varicose veins tends to be chronic, developing and progressing gradually over a long period of time.

- Assess pain, including its intensity, duration and aggravating and relieving factors. *Pain assessment allows collaborative planning with the person to identify appropriate interventions.*
- Inquire about current measures being used by the person to manage pain and its effects. Ask about the effectiveness of current management strategies and the desire to change. *Chronic pain management ultimately falls to the person. Strategies to address the pain must meet the person's needs.*

CONSIDERATION FOR PRACTICE

Suggest keeping a diary of pain intensity, timing, precipitating events and effectiveness of relief measures. Systematic tracking of pain is an important measure in improving its management.

- Teach and reinforce non-pharmacological pain management strategies such as progressive relaxation, imagery, deep breathing, distraction and meditation. *The effectiveness of such strategies is well documented. Non-pharmacological measures provide a variety of options for controlling pain while maintaining independence. These measures also can reduce reliance on analgesics.*
- Collaborate with the person to establish a pain control plan. *Collaborative planning for pain management increases the person's sense of control and reduces powerlessness. This, in turn, enhances the ability to cope with pain and its effects.*
- Regularly evaluate the effectiveness of planned interventions and pain management strategies. *Regular evaluation allows*

modification of the care plan as needed, as well as providing a measure of disease progression. Increasing or poorly controlled pain may necessitate additional collaborative interventions to manage the disorder.

Ineffective tissue perfusion: peripheral

Varicose veins and venous stasis impair delivery of nutrients and oxygen to peripheral tissues as elevated venous pressures interfere with blood flow through the capillary beds. Improving venous blood flow reduces venous pressures and promotes arterial flow to peripheral tissues.

- Assess peripheral pulses, capillary refill, skin colour and temperature, and extent of oedema. *Assessment of arterial flow and tissue perfusion provides baseline and continuing data for evaluating the effectiveness of interventions.*
- Teach application and use of properly fitted elastic graduated compression stockings. *Elastic compression stockings compress the veins, promoting venous return from the lower extremities. During ambulation, the stockings enhance the blood-pumping action of the muscles. Because elastic stockings inhibit blood flow through small superficial vessels, they should be removed at least once each day for at least 30 minutes.*

CONSIDERATION FOR PRACTICE

Instruct to maintain a program of regular exercise, such as walking for 20 to 30 minutes several times a day. Exercise stimulates circulation and promotes blood flow through the vascular system. When ambulation is restricted, active ROM exercises help maintain muscle tone, joint mobility and venous return.

- Advise to elevate the legs for 15 to 20 minutes several times a day and to sleep with the legs elevated above the level of the heart. *Elevating the legs promotes venous return, reducing tissue congestion and improving arterial circulation. Improved venous return also increases the cardiac output and renal perfusion, promoting elimination of excess fluid and decreasing peripheral oedema.*

Risk of impaired skin integrity

Ineffective venous valve function impairs venous return and increases venous pressures. These increased pressures oppose arterial blood flow and the delivery of oxygen and nutrients to the cells. As a result, tissues are vulnerable to any additional insult and may break down.

- Assess lower extremity colour, temperature and moisture, and for evidence of pressure or breakdown, on admission and at each visit. *Initial and continuing assessment allows timely detection of early signs of skin and tissue breakdown. This, in turn, allows early institution of measures to prevent further tissue damage and promote healing.*
- Teach foot and skin care measures such as daily cleansing with non-drying soap, gentle drying and lotions to prevent skin dryness and cracking. *Cleansing removes potentially harmful microorganisms and stimulates circulation. Care is taken to keep the skin moist and supple, promoting its function as the first line of defence against infection.*
- Discuss the importance of adequate nutrition and fluid intake. *Adequate nutrients are necessary to maintain tissue integrity and promote healing. A diet high in protein, carbohydrates and vitamins and minerals promotes growth and maintenance of skin cells, provides energy and helps prevent skin breakdown. Adequate hydration helps maintain the moisture and turgor of skin, reducing the risk of drying and breakdown.*

Risk of peripheral neurovascular dysfunction

Severe varicose veins can lead to chronic venous insufficiency, impaired arterial circulation and, ultimately, disrupted sensation in the affected extremity. Impaired neurological function increases the person's risk of injury and infection of the extremity, because minor trauma may go unnoticed.

- Assess circulation, sensation and movement of the lower extremities. *Disrupted circulation and venous congestion may interfere with sensory and motor function of the affected extremity. The potential for nerve and muscle involvement is especially high in people with venous stasis ulcers.*
- Teach measures to protect the extremities from injury, such as always wearing shoes or firm slippers, cotton socks to absorb moisture and testing the temperature of bath water with a thermometer or the upper extremities before stepping in. *Sensation in the lower extremities may be affected by poor circulation, necessitating additional measures to protect the legs and feet from injury.*

CONSIDERATION FOR PRACTICE

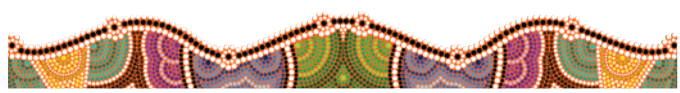
Instruct to report signs of neurovascular dysfunction, such as numbness, coldness, pain or tingling of an extremity. Early recognition of neurovascular dysfunction facilitates institution of interventions to prevent complications. Because the postoperative hospital stay following varicose vein surgery or venous stasis ulcer repair is brief, manifestations of neurovascular dysfunction may initially be detected by the person. Careful assessment and prompt reporting help prevent potential complications such as skin breakdown, infection and nerve damage.

Community-based care

Most people with varicose veins provide self-care at home. Include the following topics when preparing the person and family for home care:

- leg elevation and exercise program
- application and use of graduated elastic compression stockings
- foot and leg care (see Box 31.5 earlier in this chapter)
- measures to avoid injury and skin breakdown
- symptoms or potential complications to report to the doctor.

Provide information about suppliers of elastic stockings and any other required supplies. If venous stasis ulcers have developed, consider referral to home health services for regular assessment of healing and additional teaching.



DISORDERS OF THE LYMPHATIC SYSTEM

The lymphatic system, which includes the lymphatic vessels and the lymph nodes, is a unique part of the circulatory system. The lymphatic system returns plasma and plasma proteins filtered out of the capillaries from interstitial tissues to the bloodstream. This fluid is called *lymph*. The lymphatic system consists of closed capillaries leading to larger lymphatic venules and lymphatic veins. These vessels contain smooth muscle and one-way valves that help move fluid towards the heart. Lymphatic vessels share the same sheath as arteries and veins; arterial pulsations and skeletal muscle contractions compress the lymphatic vessels to assist in maintaining lymph flow. As lymph moves through the lymphatic system, it is filtered through thousands of bean-shaped lymph nodes clustered along the vessels. Within these nodes, phagocytes remove foreign material from the lymph, preventing it from entering the bloodstream.

GENETIC CONSIDERATIONS
Primary lymphoedema

- Primary lymphoedema develops as a result of agenesis, hypoplasia or obstruction of lymphatic vessels.
- *Congenital lymphoedema* appears shortly after birth; two other forms of lymphoedema develop later, one at the time of puberty (*lymphoedema praecox*), the other usually after age 35 (*lymphoedema tarda*).
- Congenital lymphoedema and lymphoedema praecox may be inherited as an autosomal dominant trait with variable penetrance.
- Lymphoedema also may be inherited (although less commonly) as an autosomal or sex-linked recessive disorder (Rossy, 2015).

THE PERSON WITH LYMPHATIC SYSTEM COMPROMISE

Lymphadenopathy

Lymphadenopathy, enlarged lymph nodes, may be localised or generalised. Localised lymphadenopathy usually results from an inflammatory process (e.g. streptococcal pharyngitis or an infected wound). The node enlarges as lymphocytes and monocytes proliferate within the node to destroy infectious material. Palpable lymph nodes often develop in response to minor trauma or a localised infection. Generalised lymphadenopathy usually is associated with malignancy or disease. Malignant cells or other abnormal cells invade the node, causing it to enlarge.

Lymphangitis, inflammation of the lymph vessels draining an infected area of the body, is characterised by a red streak along the inflamed vessels, pain, heat and swelling. Fever and chills also may be present. Local lymph nodes are swollen and tender.

Treatment for lymphadenopathy and lymphangitis focuses on identifying and treating the underlying condition. Elevating the body part and applying heat to inflamed lymphatic vessels help reduce swelling and promote blood flow to the affected area.

Lymphoedema

Lymphoedema may be a primary or a secondary disorder resulting from inflammation, obstruction or removal of lymphatic vessels. It is characterised by extremity oedema due to accumulation of lymph. *Primary lymphoedema* is uncommon, affecting about 1 in 10 000 people. It affects females more frequently than males and may be associated with a genetic disorder such as Turner's syndrome or Klinefelter's syndrome. (See the accompanying 'Genetic considerations' box.)

Secondary lymphoedema is an acquired condition, resulting from damage, obstruction or removal of lymphatic vessels. The most common worldwide cause of secondary lymphoedema is *filariasis*, infestation of the lymphatic vessels by filaria, a nematode worm. Other important causes of secondary lymphoedema include recurrent episodes of bacterial lymphangitis, obstruction of lymph vessels by tumours and surgical or radiation treatment for breast cancer (Kasper et al., 2015).

Pathophysiology and manifestations

Obstruction of lymph drainage prevents fluid and protein molecules from interstitial tissues from returning to the circulation. The protein molecules increase the osmotic pressure in interstitial tissues, drawing in additional fluid that causes oedema in the soft tissues. One or both extremities may be affected.

The oedema begins distally, progressing up the limb to involve the entire extremity. Initial oedema is soft and pitting; with chronic congestion, subcutaneous tissues become fibrotic, causing thick, rough skin and a woody texture of the limb (*brawny oedema*). In contrast, the oedema associated with venous disorders is softer and the skin often is hyperpigmented with evidence of stasis dermatitis. Lymphoedema generally is painless, although the limb may feel heavy.

INTERPROFESSIONAL CARE

Interprofessional care for the person with lymphoedema focuses on relieving oedema and preventing or treating infection. The disorder may be difficult to treat effectively and can lead to progressive disability due to the weight and awkwardness of the affected extremity.

Diagnosis

Abdominal or pelvic ultrasound and CT scans are used to detect obstructing lesions. MRI can show oedema and identify lymph nodes and enlarged lymphatic vessels. More invasive procedures such as lymphangiography and radioactive isotope

studies may occasionally be necessary to identify the lymphatic defect causing lymphoedema.

- *Lymphangiography* uses injected contrast media to illustrate lymphatic vessels on x-rays. Organic dyes are used to identify a distal lymphatic vessel and then a contrast medium is injected into the vessel for visualisation of the lymphatic system of the limb. In primary lymphoedema, lymph vessels are absent or hypoplastic (underdeveloped). In secondary lymphoedema, lymph channels often are dilated; it may be possible to determine the level of obstruction (Rossy, 2015).
- *Lymphoscintigraphy* involves injecting a radioactively tagged substance into distal subcutaneous tissues of the extremity, then mapping its flow through the lymphatic system. The pattern of lymph fluid distribution and transport is abnormal in people with lymphoedema.

Treatments

Meticulous skin and foot care is vital to prevent infection in the affected extremity. Shoes should always be worn to reduce the risk of injury. Careful cleansing and use of emollient lotions are recommended to prevent drying of the skin. Exercise is encouraged, as are frequent periods of leg elevation. The foot of the bed is raised by 15 to 20 degrees at night to promote lymph flow. Elastic graduated compression stockings may be ordered for use during the day. In some cases, an intermittent pneumatic compression device to reduce oedema may be prescribed for home use.

Antibiotics are given to prevent and treat infection, which can be recurrent and difficult to eradicate. Diuretic therapy may be used intermittently, particularly when primary lymphoedema is exacerbated by the menstrual cycle or seasonal variability (Papadakis et al., 2013).

People who do not respond to conservative treatment measures or who experience recurrent episodes of cellulitis and lymphangitis may require surgical treatment. Microvascular techniques may be used to create anastomoses between obstructed lymphatic vessels and adjacent veins, providing channels to redirect lymph into the venous system. Successful surgery may improve both extremity function and its cosmetic appearance (Papadakis et al., 2013).

Nursing care

Nursing care for people with lymphatic disorders focuses on reducing oedema, preventing tissue damage related to the oedema and promoting effective coping with the effect of the disorder on body image and function.

Nursing diagnoses and interventions

Nursing diagnoses for the person with lymphoedema may include *Impaired tissue integrity*, *Excess fluid volume* and *Disturbed body image*.

Impaired tissue integrity

Obstructed lymphatic flow leads to fluid congestion of the interstitial spaces of subcutaneous tissue. The resulting oedema compresses and damages tissues of the affected extremity. Subcutaneous tissues become fibrotic, reducing their protective functions of shock absorption and insulation. In addition, obstructed lymphatic flow reduces the effectiveness of lymph nodes in filtering and removing foreign material and pathogens from the body. This increases the risk of local tissue infection such as *cellulitis*, a diffuse bacterial infection of the skin. Cellulitis increases the risk of skin and tissue breakdown and, if not effectively treated, can lead to sepsis.

CONSIDERATION FOR PRACTICE

Frequently inspect the skin of the affected extremity, documenting condition with each assessment. Promptly report areas of pallor, redness or apparent inflammation. Breaks in the skin surface allow microbial invasion and increase the risk of infection. Prompt identification and treatment of any lesions is vital to prevent further tissue breakdown and infection.

- Apply well-fitting elastic graduated compression stockings or intermittent pneumatic pressure devices as ordered. *Elastic stockings and/or pneumatic pressure devices oppose the movement of fluid out of capillaries and improve its reabsorption into vascular spaces for transportation back to the heart.*
- Instruct to elevate the extremities while seated and during sleep. *Elevation of the extremities diminishes venous congestion, promotes venous return, facilitates arterial circulation and tissue perfusion and helps reduce the accumulation of excess fluids in interstitial spaces of the affected extremity.*

CONSIDERATION FOR PRACTICE

Remove elastic stockings and intermittent pressure devices every 8 hours or at each home visit to inspect the underlying skin for evidence of redness, irritation, dryness or breakdown. Elastic graduated compression stockings, anti-embolic stockings and pneumatic compression devices compress small vessels nourishing the skin and subcutaneous tissue. Periodic removal not only allows inspection of the underlying skin, but also allows restoration of blood flow to these small vessels and the tissues.

CONSIDERATION FOR PRACTICE

Use preventive skin care devices as indicated. Collected fluid in the affected extremity increases its weight and interferes with regular movement. The increased weight places greater pressure on surfaces of the limb that come in contact with furniture. Protective devices such as egg-crate foam, sheepskin, pillows or padding help prevent tissue compression, promoting circulation and reducing the risk of skin and tissue breakdown.

- Keep skin clean and dry, especially in interdigital spaces. Teach skin and foot care to the person and family. *Clean, dry skin provides the first line of defence against infection. Significant limb oedema can interfere with reaching the*

distal extremity and cleaning interdigital spaces. The dark, moist spaces between the toes are an excellent environment for bacterial growth. Teaching fosters self-care and independence, as well as preparing the person and family to manage this often chronic condition.

- Discuss the importance of adhering to the therapeutic regimen. *Lymphoedema generally is a chronic condition; effective management requires active person participation in planning and implementing care to reduce oedema and maintain tissue integrity.*

Excess fluid volume

In lymphoedema, obstruction, destruction or congenital malformation of lymphatic vessels interferes with the normal circulation of lymphatic fluid. As a result, lymph collects in the subcutaneous tissues of the affected extremity, causing excess fluid volume of that extremity. Some people may benefit from intermittent diuretic therapy and dietary sodium restriction.

- Discuss the rationale for restricted sodium intake if ordered. Teach ways to maintain the recommended sodium restriction and assist to choose foods that are low in sodium. *Sodium causes retention of extracellular water; restricting dietary sodium may help prevent additional fluid accumulation in interstitial spaces.*
- During acute periods, assess the affected extremity daily for increased oedema; measure girth of the extremity using consistent technique. *The size of the affected extremity provides a measure of the effectiveness of ordered interventions and progression of the disorder.*

CONSIDERATION FOR PRACTICE

Monitor intake and output and/or weight (daily or weekly). Use consistent scales, timing and clothing for accurate weight measurements. Intake and output records and short-term changes in weight reflect fluid balance. Measures of fluid balance permit evaluation of the effectiveness of interventions such as restricted sodium intake and diuretic therapy.

Disturbed body image

The disproportionate size of an extremity or extremities due to lymphoedema can profoundly affect body image. During early stages of the disease, conservative measures may effectively reduce the oedema and size of the affected limb. However, as the disease progresses, conservative measures may become less effective, leading to more permanent disfigurement. Mobility may be impaired and the person may develop an increasingly negative self-perception.

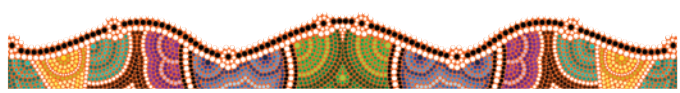
- Encourage discussions about usual coping patterns and perception of self. *Knowledge of existing coping patterns and behaviours helps the nurse assess the person's ability to cope with the current situation. This knowledge is then used to reinforce effective coping mechanisms and help develop more effective coping strategies. This exchange also allows the person to voice feelings related to actual or perceived changes in body image.*
- Accept the person's perception of self and of the impact of the changes in appearance. *Non-judgmental acceptance of the person's view of self and of the effects of changes in appearance builds trust and promotes rapport. A trusting relationship promotes the person's ability to take an active role in managing the disorder, participate in healthcare decisions and adhere to the plan of care. Non-judgmental listening also promotes mutual respect and demonstrates caring and compassion.*
- Encourage active participation in self-care. Assist with identifying alternative self-care strategies when the extent of oedema interferes with performing some aspects of self-care such as trimming toenails or washing feet. *The person initially may have difficulty viewing or touching the affected body part. Gentle encouragement and support from the nurse helps the person assume self-care and accept the affected body part. Brainstorming to identify alternative care strategies promotes the person's independence even when total self-care is not feasible.*

Community-based care

When preparing the person with chronic lymphoedema and family to manage the disorder, include the following teaching topics:

- recommended program of exercise and elevation of the extremity
- foot and skin care
- use of elastic graduated compression stockings and/or intermittent pressure devices
- importance of wearing elastic stockings during the majority of waking hours, removing them once during the daytime and while sleeping
- measures to prevent infection in the affected extremity, such as wearing gloves while gardening
- signs and symptoms to report to the healthcare provider (e.g. manifestations of tissue breakdown or infection, increasing oedema or evidence of compromised circulation)
- use and precautions associated with any prescribed medications
- sodium-restricted diet if ordered.

Provide information about contacts for questions and make referrals as needed. Evaluate the need for home health, home maintenance assistance and other services such as physical or occupational therapy.



CHAPTER HIGHLIGHTS

- Essential hypertension, blood pressure of 140/90 mmHg or higher with no clearly identified cause, rarely causes symptoms but is a major risk factor for coronary heart disease, heart failure, stroke and renal insufficiency.
- Prehypertension, a newly identified category, is an average blood pressure of 120–139/80–89 mmHg. People with prehypertension are advised to make lifestyle changes indicated for hypertension (weight loss, exercise, dietary changes, limited alcohol intake and stress reduction), but generally are not treated with medications unless other risk factors such as diabetes or kidney disease are present.
- Systolic hypertension, an elevated systolic blood pressure without elevation of the diastolic pressure, is common in older adults and contributes to complications such as coronary heart disease and stroke.
- Cardiovascular disease in Indigenous Australians is 30% more common than in non-Indigenous Australians.
- Risk factors for cardiovascular disease are more common in Indigenous Australians than in non-Indigenous Australians: diabetes is four times as common, smoking daily and obesity are twice as common.
- Medications to treat hypertension include diuretics, alpha- and beta-adrenergic blockers, ACE inhibitors and angiotensin II blockers, calcium channel blockers and vasodilators. A combination of two or more drugs often is required for effective blood pressure control.
- Aneurysms, abnormal dilation of a blood vessel, commonly affect the aorta and the iliac arteries, particularly in older men. A slowly expanding abdominal aortic aneurysm that does not produce symptoms or impair flow through the renal arteries may not be repaired, particularly in an older person. Percutaneously inserted endovascular splints provide an alternative to surgery for abdominal aortic aneurysms.
- Peripheral vascular disease, obstruction or occlusion of peripheral arteries by atherosclerotic plaque, is common and a leading cause of disability and amputation.
- Smoking cessation and regular daily exercise are key components of treatment for peripheral vascular disorders such as atherosclerosis, thromboangiitis obliterans and Raynaud's disease.
- Venous thrombosis, particularly of the deep veins of the legs and pelvis, develops as a result of venous stasis, blood vessel damage and increased coagulability of the blood. The developing clot may fragment or break loose, becoming an embolus that typically lodges in the pulmonary circulation (pulmonary embolus). Chronic venous insufficiency and venous stasis may develop as a result of deep venous thrombosis.
- Prophylactic anticoagulation and mobilisation of the person are the primary preventive measures for venous thrombosis. Monitoring coagulation studies and assessing for evidence of bleeding (overt or covert) are important nursing measures for the person on anticoagulant therapy.
- Lymphadenopathy (enlarged lymph nodes), lymphangitis (inflammation of the lymph vessels) and lymphoedema are the most common disorders affecting the lymph system.

CONCEPT CHECK

1 A potential blood donor whose blood pressure is found to average 180/106 mmHg on two different readings tells the nurse, 'I don't understand how it could be so high—I feel just fine.' The appropriate response by the nurse is:

1 'This is probably just a false reading due to "white coat syndrome". Don't worry about it.'
2 'It is unusual that you are not having some symptoms such as severe headaches and nosebleeds.'
3 'High blood pressure often has few or no symptoms; that's why it is called the "silent killer".'
4 'You probably should have your blood pressure rechecked in 3 months or so and then follow up with your primary care provider if it is still high.'

2 The nurse teaching a person about a healthy diet determines that additional teaching is necessary when the person states:

1 'I'm glad I can still eat as much pasta as usual; I was afraid I would have to give up my weekly lasagna.'
2 'It will be a challenge to incorporate all those servings of fruit and vegetables into my diet.'
3 'Having a handful of nuts when the pre-dinner "munchies" hit is a good idea.'
4 'I will enjoy having frozen yogurt as my bedtime snack on occasion.'

3 The nurse teaching a person about his new prescription for Co-Diovan, a combination angiotensin II receptor blocker and thiazide diuretic, includes which of the following in his instructions? (Select all that apply.)

1 Use a potassium-based salt substitute to prevent hypokalaemia while taking this drug.
2 Use caution when rising from bed or a chair to prevent dizziness.
3 Take the drug at bedtime to reduce the risk of falling due to light headedness.
4 Report a persistent disruptive cough to your doctor.
5 You may stop taking this drug once your blood pressure is within the normal range for 2 months.

4 A person is complaining of new-onset calf and foot pain. The nurse notes that the leg below the knee is cool and pale and that dorsalis pedis and posterior tibial pulses are absent. The priority nursing intervention is to:

1 notify the doctor
2 place a cradle over the leg to prevent pressure from bedding
3 position the leg flat, supported in anatomical position
4 prepare to initiate heparin therapy

5 An 86-year-old person with a newly diagnosed abdominal aortic aneurysm wonders if he will need surgery to repair the aneurysm, even though he feels fine. The nurse's response is based on the knowledge that:

1 the risk of surgical repair is lower than the risk that the aneurysm will rupture
2 opening the abdomen for the surgical procedure greatly increases the risk of rupture

3 surgery is indicated for type A aneurysms
4 a percutaneously inserted endovascular stent may be considered because of his age

6 An expected assessment finding in a person with peripheral atherosclerosis would be:
1 pallor of the legs and feet when dependent
2 increased hair growth on the affected extremity
3 higher blood pressure readings in the affected extremity
4 impaired sensation in the affected extremity

7 All of the following are appropriate home care measures for the person with peripheral vascular disease. Place them in order of priority.
1 foot and leg care
2 smoking cessation
3 daily inspection of feet and legs
4 regular daily exercise
5 weight loss strategies

8 The nurse evaluates her teaching of a person admitted with deep venous thrombosis as effective when the person states:
1 'I'll use a hard-backed, upright chair when sitting instead of my recliner.'
2 'I'll get my blood drawn as scheduled and notify the doctor if I have any unusual bleeding or bruising.'
3 'I understand why I am not allowed to exercise for the next 6 weeks and will take it easy.'
4 'I'll have my wife buy a low-cholesterol cookbook and we'll make an appointment with the dietitian to learn about a low-fat, low-cholesterol diet.'

9 A person with visible varicose veins tells the nurse that she wants to have surgery to remove them, because 'my legs ache every evening and they are really ugly!'. The most appropriate response would be:
1 'Often measures such as elevating your legs and elastic stockings can relieve the discomfort associated with varicose veins.'
2 'Surgery will have a good cosmetic effect, but will not relieve the discomfort associated with varicose veins.'
3 'All varicose veins should be surgically removed to restore adequate blood flow to your legs and prevent gangrene.'
4 'Surgery is never indicated unless the varicose veins are interfering with circulation. Have you tried cosmetic measures to cover them up?'

10 Which of the following nursing interventions is of highest priority for the person with lymphoedema?
1 Elevate affected extremities at night.
2 Assist to don elastic compression stockings during the day.
3 Carefully dry and apply emollient lotion to affected extremities after bathing.
4 Reinforce the importance of taking prescribed diuretics.

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CHAPTER 32

NURSING CARE OF PEOPLE WITH HAEMATOLOGICAL DISORDERS

LIZ RYAN

KEY TERMS

LEARNING OUTCOMES

- Relate changes in erythrocyte morphology to the pathophysiological effects resulting in red blood cell disorders.
- Identify the care and health management principles for individuals experiencing red blood cell disorders.
- Describe the main types of leukaemia, and examine their major health management principles.
- Discuss indications, complications and management practices for a person receiving bone marrow and stem cell transplant.
- Differentiate between Hodgkin's disease and non-Hodgkin's lymphoma.
- Compare and contrast the pathophysiology, manifestations and management for people experiencing bleeding disorders.

CLINICAL COMPETENCIES

- Assess effects of haematological disorders and prescribed treatments on a person's functional health status.
- Monitor and document continuing assessment data, including laboratory test results, subjective and objective information, and reporting data outside the normal or expected range.
- Based on knowledge of pathophysiology, prescribed treatment and assessed data, identify and prioritise nursing diagnoses for people with haematological disorders.
- Use nursing research and evidence-based practice to identify and implement individualised nursing interventions for the person with a haematological disorder.
- Safely and knowledgeably administer prescribed medications and treatments for people with haematological disorders.
- Collaborate with the interprofessional care team to plan and provide coordinated, effective care for people with haematological disorders.
- Provide appropriate teaching for people with haematological disorders, evaluating learning and the need for continued reinforcement of information.
- Use continuing assessment data to revise the plan of care as needed to restore, maintain or promote functional health in the person with a haematological disorder.

Disorders affecting blood and blood-forming organs have effects that range from minor disruptions in daily activities to major life-threatening crises. People with haematological disorders need holistic nursing care, including emotional support and care for problems involving major body systems.

This chapter focuses on health changes resulting from changes in red cells, white cells, platelets and clotting factors. Before proceeding with this chapter, read Chapter 28 which provides a review of the physiology of blood and its formation as well as important information about assessing those with haematological disorders.

RED BLOOD CELL DISORDERS

Red blood cells (RBCs) transport oxygen to body tissues and help return carbon dioxide to the lungs for excretion. Alterations in the number, size, shape or composition of RBCs affect their ability to effectively carry out these functions. Anaemia, the most common RBC disorder, is an abnormally low RBC count or reduced haemoglobin content. Polycythaemia is an abnormally high RBC count.

THE PERSON WITH ANAEMIA

Anaemia is an abnormally low number of circulating RBCs, low haemoglobin concentration, or both. Decreased numbers of circulating RBCs is the usual cause of anaemia. This may result from blood loss, inadequate RBC production or increased RBC destruction. Insufficient or defective haemoglobin within RBCs contributes to anaemia. Depending on its severity, anaemia may affect all major organ systems.

FAST FACTS

- Iron deficiency anaemia, a nutritional anaemia, is the most common type of anaemia.
- Blood loss anaemia may be acute, resulting from haemorrhage, or chronic, resulting from chronic blood loss (e.g. menstrual flow, slow GI bleeding).

Physiology review

As blood flows through the pulmonary vascular system, oxygen diffuses from alveoli into capillary blood. The majority of the oxygen binds reversibly with the haemoglobin in red blood cells; only about 3% of the oxygen remains in solution in the blood. When the blood reaches the capillaries serving body tissues, oxygen is released from the haemoglobin molecule and diffuses out of the capillary to reach the cells. The amount of oxygen that reaches the tissues depends on a number of factors, including:

- available oxygen in the alveoli
- diffusing surface and capacity of the lungs
- number of red blood cells and the amount and type of haemoglobin they contain
- ability of the cardiovascular system to transport blood and oxygen to the tissues.

For more information about red blood cells and haemoglobin, and their production and function, see Chapter 28.

Pathophysiology and manifestations

A number of different pathological mechanisms can lead to anaemia (see Box 32.1). Regardless of the cause, every type of anaemia reduces the oxygen-carrying capacity of the blood due to a deficiency of RBCs or haemoglobin, leading to tissue hypoxia. The resulting effects on a person depend on the severity of the anaemia, how quickly it develops and other factors such as age and health status.

When anaemia develops gradually and the RBC reduction is moderate, successful compensatory mechanisms may result in few symptoms except when the oxygen needs of the body increase due to exercise or infection. Symptoms develop as

BOX 32.1 Pathophysiological mechanisms of anaemia

Decreased RBC production

- Altered haemoglobin synthesis
- Iron deficiency
- Thalassaemia
- Chronic inflammation
- Altered DNA synthesis
- Vitamin B_{12} or folic acid malabsorption or deficiency
- Bone marrow failure
- Aplastic anaemia (stem cell dysfunction)
- Red cell aplasia
- Myeloproliferative leukaemia
- Cancer metastasis, lymphoma
- Chronic infection or inflammation, physical and emotional fatigue

Increased RBC loss or destruction

- Acute or chronic blood loss
- Haemorrhage or trauma
- Chronic gastrointestinal bleeding, menorrhagia
- Increased haemolysis
- Hereditary cell membrane disorders
- Defective haemoglobin—sickle cell anaemia or trait
- Pyruvate kinase (PK) or glucose-6-phosphate dehydrogenase (G6PD) deficiency affecting glycolysis or cell oxidation
- Immune mechanisms and disorders (e.g. blood reaction, hypersensitivity responses, autoimmune disorders)
- Splenomegaly and hypersplenism
- Infection
- Erythrocyte trauma (e.g. due to cardiopulmonary bypass, haemolytic uraemic syndrome)

RBCs and haemoglobin levels are further reduced. Pallor of the skin, mucous membranes, conjunctiva and nail beds develops as a result of blood redistribution to vital organs and lack of haemoglobin (see Figure 32.1). As tissue oxygenation decreases, the heart and respiratory rates rise in an attempt to increase cardiac output and tissue perfusion. Tissue hypoxia may cause angina, fatigue, dyspnoea on exertion and night cramps. It also stimulates erythropoietin release; increased erythropoietin activity stimulates RBC production in the bone marrow and may lead to bone pain. Cerebral hypoxia can lead to headache, dizziness and dim vision. Heart failure may develop in severe anaemia.

With rapid blood loss, blood volume is decreased as well as the oxygen-carrying capacity of the blood. Initial manifestations include tachycardia and tachypnoea; the skin may be pale, cool and clammy as peripheral vessels constrict to maintain blood flow to the heart and brain. With significant blood loss, signs of circulatory shock may occur, including hypotension, tachycardia, decreased level of consciousness and oliguria. With chronic bleeding, fluid shifts from the interstitial spaces into the vessels, in an effort to maintain blood volume. Blood viscosity is reduced, which may result in a systolic heart murmur. See the following multisystem effects of anaemia.

Anaemia is categorised by cause: blood loss, nutritional, haemolytic and bone marrow suppression. The pathophysiology and specific manifestations of these types of anaemias follow.

Blood loss anaemia

When anaemia results from acute or chronic bleeding, RBCs and other blood components (such as iron) are lost from the body. With acute blood loss, circulating volume decreases. As a result, cardiac output falls. Compensatory mechanisms are activated to maintain the cardiac output: the heart rate increases and peripheral blood vessels constrict. Vessels in the liver, a blood storage organ, also constrict, increasing circulating volume. Fluid shifts from the interstitial spaces into the vascular compartment to maintain blood volume, diluting the cellular components of the blood and reducing its viscosity. If haemorrhage continues, compensatory mechanisms become less effective, increasing the risk of shock and circulatory failure (see Chapter 10).

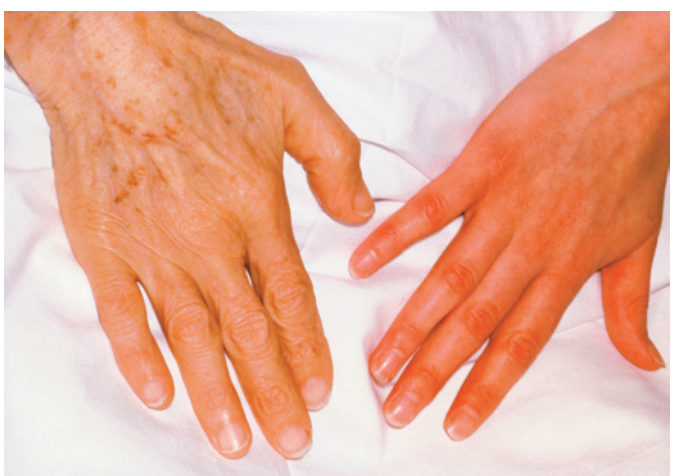


FIGURE 32.1 ■ The skin of the person with anaemia appears pale beside that of a person with a normal haemoglobin and haematocrit

Source: ©Westminster Hospital/Science Source.

In acute blood loss, circulating RBCs are of normal size and shape (*normocytic*). Early in the haemorrhage, the RBC count, haemoglobin and haematocrit may be normal; as fluid shifts from the interstitial space into the vascular space to maintain circulating volume, the RBC count, haemoglobin and haematocrit fall. If sufficient iron is available, haemoglobin levels and the number of circulating RBCs return to normal within 3 to 4 weeks after the bleeding episode. Chronic blood loss, on the other hand, depletes iron stores as RBC production attempts to maintain the RBC supply. The resulting RBCs are *microcytic* (small) and *hypochromic* (pale).

Nutritional anaemias

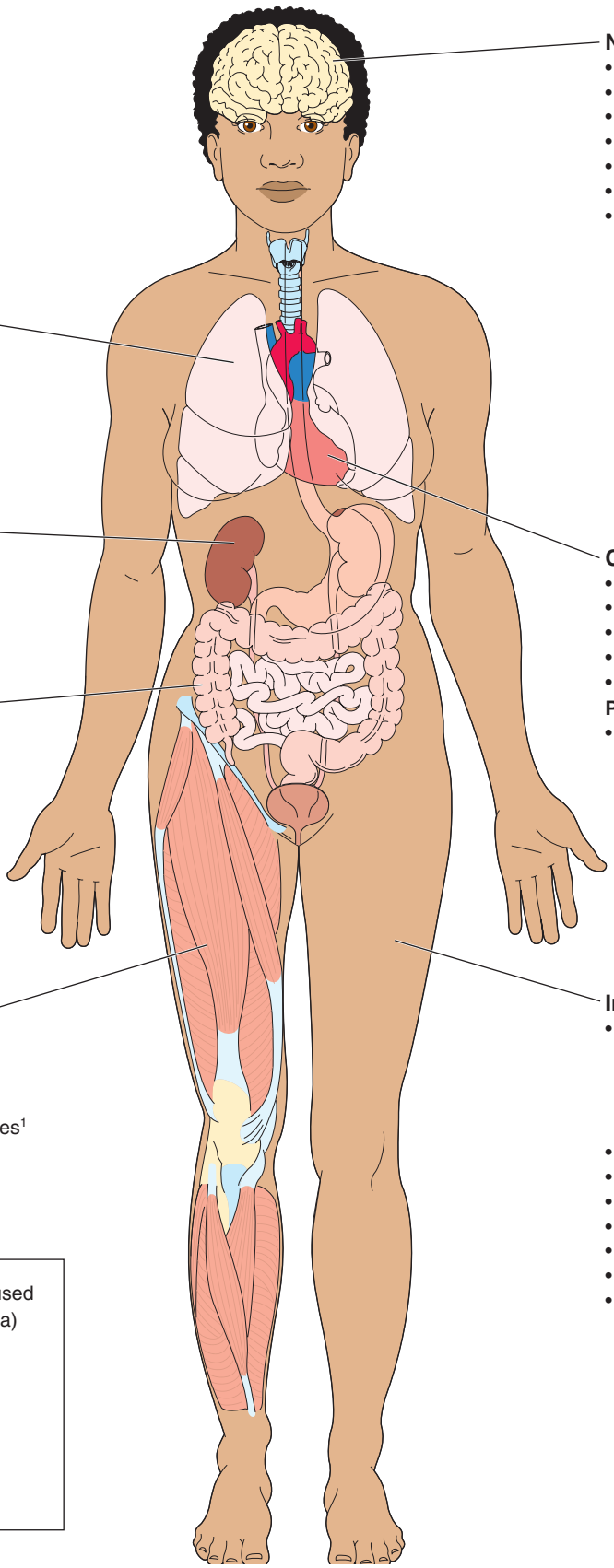
A number of different nutrients are required for normal red blood cell development (erythropoiesis). Iron is a key nutrient necessary for haemoglobin synthesis. In addition, adequate supplies of protein (and its building blocks, amino acids), vitamins and other minerals are required. The B group vitamins, particularly B_{12} (cobalamin) and folate (B_9), play a key role in RBC development. Vitamins C and E are also necessary. Nutritional anaemias result from nutrient deficits that affect RBC formation or haemoglobin synthesis. The nutrient deficit may be caused by inadequate diet, malabsorption of the nutrient, or an increased need for the nutrient. The most common types of nutritional anaemias are iron deficiency anaemia, vitamin B_{12} anaemia and folic acid deficiency anaemia. Vitamin B_{12} and folic acid anaemias are sometimes called *megaloblastic* anaemias, because enlarged nucleated RBCs called megaloblasts are seen in these anaemias.

IRON DEFICIENCY ANAEMIA **Iron deficiency anaemia** is the most common type of anaemia. It develops when the supply of iron is inadequate for optimal RBC formation, as the body cannot synthesise haemoglobin without iron. Normally, the body efficiently recycles and stores iron, reusing much of the iron contained in RBCs that are removed from circulation due to age or damage. However, small amounts of iron are continually lost in the faeces; therefore adequate iron intake is necessary for normal haemoglobin synthesis and RBC production. Iron deficiency anaemia results in fewer numbers of RBCs, microcytic and hypochromic RBCs, as well as malformed RBCs (*poikilocytosis*) (see Figure 32.2).

Excessive iron loss due to chronic bleeding is the usual cause of iron deficiency anaemia in adults. Menstrual blood loss is the most common cause in adult females. Iron deficiency anaemia also may result from inadequate dietary iron intake (less than 1 mg/day), malabsorption syndromes or the increased iron requirements associated with pregnancy and lactation. Box 32.2 summarises common causes of iron deficiency anaemia.

Iron deficiency anaemia is particularly common in older adults. Chronic, occult (hidden) blood loss may occur from slowly bleeding peptic ulcers, gastrointestinal inflammation,

MULTISYSTEM EFFECTS OF ANAEMIA



Neurological
- Paraesthesias[4]
- Proprioception deficits[4]
- Headache[5]
- Fainting[5]
- Forgetfulness[5]
- Pain[6]
- Behavioural disturbances (pica)[3]

Respiratory
- Increased rate
- Dyspnoea on exertion

Urinary
- Haemoglobinuria[7]

Cardiovascular
- Tachycardia
- Palpitations
- Systolic murmur
- Ventricular hypertrophy
- Angina

Potential complication
- Heart failure[6]

Gastrointestinal
- Diarrhoea[4]
- Anorexia[5]
- Nausea[5]
- Gallstones[6]
- Splenomegaly[1]
- Abdominal pain[6]

Integumentary
- Pallor
  - Skin
  - Mucous membranes
  - Conjunctiva
  - Nail beds
- Jaundice[1]
- Petechiae[2]
- Purpura[2]
- Spoon-shaped nails[3]
- Cheilosis[3]
- Sore, beefy red tongue[4]
- Chronic leg ulcers[6]

Musculoskeletal
- Night cramps[5]
- Bone pain
- Joint pain[6]
- Bone deformity and fractures[1]

Key (symptoms usually caused by a specific form of anaemia)
1 Haemolytic anaemias
2 Aplastic anaemia
3 Iron deficiency anaemia
4 Pernicious anaemia
5 Vitamin B_{12} anaemia
6 Sickle cell anaemia
7 G6PD anaemia

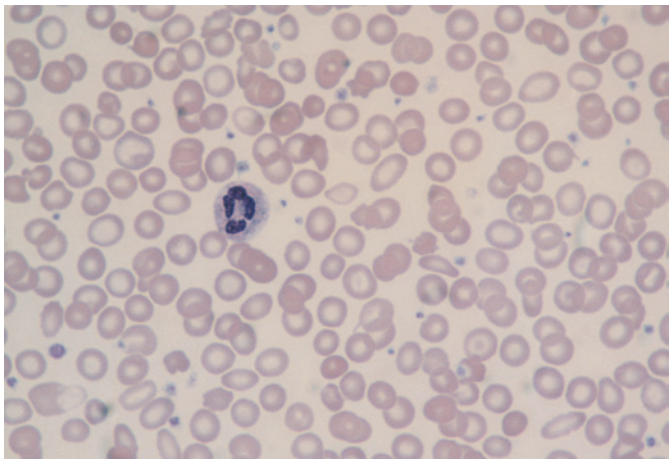


FIGURE 32.2 ■ A blood smear showing RBCs characteristically seen in iron deficiency anaemia. Note the pale colour of the RBCs (hypochromic). Many of the cells also are smaller than normal (microcytic) and misshapen, reducing their oxygen-carrying capacity

Source: © Dr E. Walker/Science Source.

BOX 32.2 Causes of iron deficiency anaemia

- Dietary deficiencies
- Vegetarian diet
- Inadequate protein intake
- Decreased absorption
- Partial or total gastrectomy
- Chronic diarrhoea
- Malabsorption syndromes
- Increased metabolic requirements
- Pregnancy
- Lactation
- Blood loss
- Gastrointestinal bleeding (especially due to ulcers or chronic aspirin use)
- Menstrual losses
- Chronic haemoglobinuria

haemorrhoids and cancer. Inadequate dietary iron intake also contributes to anaemia in the older adult. Access to transportation may limit fresh food consumption, a factor contributing to poor iron intake among all adults, especially people with limited or fixed incomes.

Manifestations In addition to the general manifestations of anaemia described earlier, chronic iron deficiency may lead to brittle, spoon-shaped nails; cheilosis (cracks at the corners of the mouth); a smooth, sore tongue; and *pica* (a craving for unusual substances, such as clay or starch).

VITAMIN B_{12} DEFICIENCY ANAEMIA

Vitamin B_{12} is necessary for DNA synthesis and is almost exclusively found in foods derived from animals. *Vitamin B_{12} deficiency anaemia* occurs when inadequate vitamin B_{12} is consumed or, more commonly, when it is poorly absorbed from the gastrointestinal tract. Deficiency of this vitamin impairs cell division and maturation of the cell nucleus, especially in rapidly proliferating RBCs. As a result, macrocytic (large), misshapen (oval rather than concave) RBCs with thin membranes are produced. Great numbers of these large, immature RBCs enter the circulation. These cells are fragile, incapable of carrying adequate amounts of oxygen and have a shortened lifespan.

Failure to absorb dietary vitamin B_{12} is called **pernicious anaemia**. It develops due to lack of intrinsic factor, a substance secreted by the gastric mucosa. Intrinsic factor binds with vitamin B_{12} and travels with it to the ileum, where the vitamin is absorbed. In the absence of intrinsic factor, vitamin B_{12} cannot be absorbed into the body, most commonly seen in elderly people.

Vitamin B_{12} deficiency may also result from other malabsorption disorders and dietary factors. Resection of the stomach or ileum, loss of pancreatic secretions and chronic gastritis can affect vitamin B_{12} absorption. Dietary deficiencies of vitamin B_{12} are rare, usually occurring only among strict vegetarians.

Manifestations Manifestations of vitamin B_{12} deficiency anaemia develop gradually as bodily stores of the vitamin are depleted. Pallor or slight jaundice and weakness develop. In pernicious anaemia, a smooth, sore, beefy red tongue and diarrhoea may occur. Because vitamin B_{12} is important for neurological function, paraesthesia (altered sensations, such as numbness or tingling) in the extremities and problems with proprioception (the sense of one's position in space) develop. These manifestations may progress to difficulty maintaining balance due to spinal cord damage. Central nervous system (CNS) manifestations of relatively short duration (a few months) are reversible with treatment, but may be permanent if treatment is delayed (Katzung, Masters & Trevor, 2012; Papadakis, McPhee & Rabow, 2013).

FOLIC ACID DEFICIENCY ANAEMIA

Like vitamin B_{12}, folic acid is required for DNA synthesis and normal maturation of red blood cells. *Folic acid deficiency anaemia* is characterised by fragile, megaloblastic (large and immature) cells. Folic acid is found in green leafy vegetables, fruit, cereals and meats, and is absorbed from the intestines.

Folic acid deficiency anaemia due to inadequate intake is more common among people who are chronically undernourished. This includes older adults, alcoholics and the drug addicted. Alcoholics are especially at risk because alcohol suppresses folate metabolism, which forms folic acid. Increased folic acid requirements also may lead to anaemia, with pregnant women at greatest risk. Infants and teenagers can also develop temporary folic acid deficiencies during periods of rapid growth. Impaired folic acid absorption and metabolism can cause folic acid deficiency anaemia. Malabsorption disorders, such as coeliac sprue (a hereditary gastrointestinal disorder characterised by inability to metabolise amino acids found in gluten), and certain medications, such as methotrexate and some chemotherapeutic agents, may be contributing factors. Causes of folic acid deficiency anaemia are summarised in Box 32.3.

BOX 32.3 Causes of folic acid deficiency anaemia

- Inadequate dietary intake
  At risk:
  a. Older adults
  b. Alcoholics
  c. People receiving total parenteral nutrition (TPN)
- Increased metabolic requirements
  At risk:
  a. Pregnant women
  b. Infants and teenagers
  c. People undergoing haemodialysis
  d. People with forms of haemolytic anaemia
- Folic acid malabsorption and impaired metabolism
  a. Coeliac sprue
  b. Chemotherapeutic agents, folate antagonists (methotrexate, pentamidine) or anticonvulsants
  c. Alcoholism

Manifestations The manifestations develop gradually as folic acid stores are depleted. Signs and symptoms may include pallor, progressive weakness and fatigue, shortness of breath and heart palpitations. Manifestations similar to those associated with vitamin B_{12} anaemia, such as glossitis, cheilosis and diarrhoea, are common. No neurological symptoms occur with folic acid deficiency anaemia, thus helping to differentiate it from vitamin B_{12} deficiency anaemia. These two nutritional anaemias do, however, sometimes coexist.

Maternal levels of folic acid are strongly associated with the prevention of neural tube defects (NTD). NTDs, including spina bifida, encephalocoele and anencephaly, result from the failure of the spinal cord or brain to develop normally during early foetal development, often before pregnancy is recognised. The seriousness of these abnormalities is reflected by the fact that fewer than 40% of babies affected survive to birth. People born with an NTD, especially those with spina bifida, will experience lifelong disability. Hence the National Health and Medical Research Council (2005) recommends folic acid supplements for 3 months preconception and during the first trimester of pregnancy.

Haemolytic anaemias

Haemolytic anaemias are characterised by premature destruction (*lysis*) of RBCs. When RBCs break down, iron and other by-products of their destruction remain in the plasma. RBC lysis (haemolysis) may occur within the circulatory system or due to phagocytosis by white blood cells (WBCs) such as circulating monocytes and macrophages in the spleen. In response to haemolysis, the haematopoietic activity of bone marrow increases, leading to increased reticulocytes (immature RBCs) in circulating blood. Most types of haemolytic anaemia are characterised by normocytic and normochromic RBCs.

There are many different causes of haemolytic anaemia (see Box 32.4). The cause may be *intrinsic*, arising from disorders within the RBC itself, or *extrinsic*, originating outside the RBC. Intrinsic disorders include cell membrane defects, defects in haemoglobin structure and function, and inherited enzyme deficiencies. See the accompanying 'Focus on cultural diversity' box for more information about inherited intrinsic RBC disorders associated with haemolytic anaemia. Extrinsic causes of haemolytic anaemia include drugs, bacterial and other toxins, and trauma. This section discusses sickle cell anaemia, thalassaemia, acquired haemolytic anaemia and glucose-6-phosphate dehydrogenase (G6PD) anaemia.

BOX 32.4 Causes of haemolytic anaemia

Intrinsic
- RBC cell-membrane defects
- Haemoglobin structure defects (e.g. sickle cell anaemia, thalassaemia)
- Inherited enzyme defects (e.g. G6PD deficiency (glucose-6-phosphate dehydrogenase deficiency))

Extrinsic
- Drugs, chemicals
- Toxins and venoms
- Bacterial and other infections
- Trauma, burns
- Mechanical damage (prosthetic heart valves)

SICKLE CELL ANAEMIA **Sickle cell anaemia** is a hereditary, chronic haemolytic anaemia. It is characterised by episodes of *sickling*, during which RBCs become abnormally crescent shaped. The disorder is transmitted as an autosomal recessive genetic defect (see Figure 32.3). This defect causes synthesis of an abnormal form of haemoglobin (HbS) within red blood cells. Sickle cell anaemia can significantly shorten lifespan, with most deaths occurring due to infection (Huether & McCance, 2013).

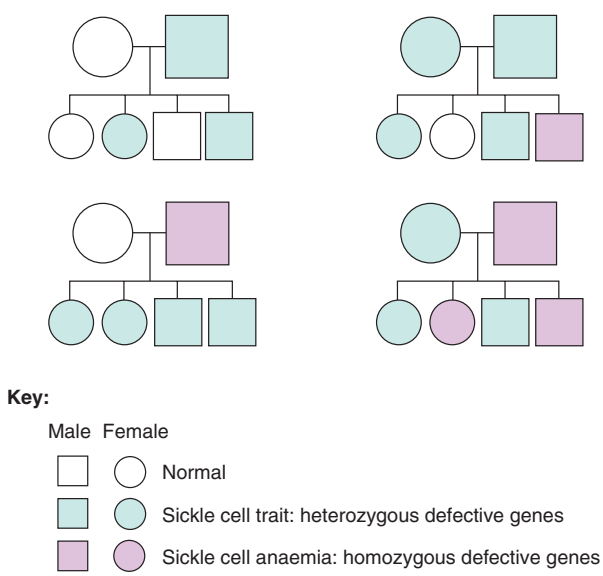


FIGURE 32.3 ■ Inheritance pattern for sickle cell anaemia

The disease is one of the most common inherited conditions worldwide and is most common among people from African, Middle Eastern and Southern Europe populations, as well as from India, Pakistan, South America and the Caribbean (see 'Focus on cultural diversity' box below). However, in Australia this disease is quite rare, with the majority of cases originating from New South Wales. Of the children born in Australia with sickle cell anaemia, more than half were from South-East Asia and the Middle East (Argent et al., 2012).

The HbS gene changes the structure of the beta chain of the haemoglobin molecule. When hypoxaemia develops and HbS is deoxygenated, it crystallises into rod-like structures. Clusters of these rods form long chains that deform the erythrocyte into a crescent or sickle shape (see Figure 32.4). The sickled cells tend to clump together and obstruct capillary blood flow, causing ischaemia and possible infarction of surrounding tissue. See the following 'Pathophysiology illustrated: sickle cell anaemia'.

When normal oxygen tension is restored, the sickled RBCs resume their normal shape; that is, they 'unsickle'. Repeated episodes of sickling and unsickling weaken RBC cell membranes. The weakened RBCs are haemolysed and removed. Consequently, the normal lifespan of RBCs is greatly reduced in sickle cell anaemia, increasing the demand for RBC production. Conditions likely to trigger sickling include hypoxia, low environmental or body temperature, excessive exercise, anaesthesia, dehydration, infections or acidosis.

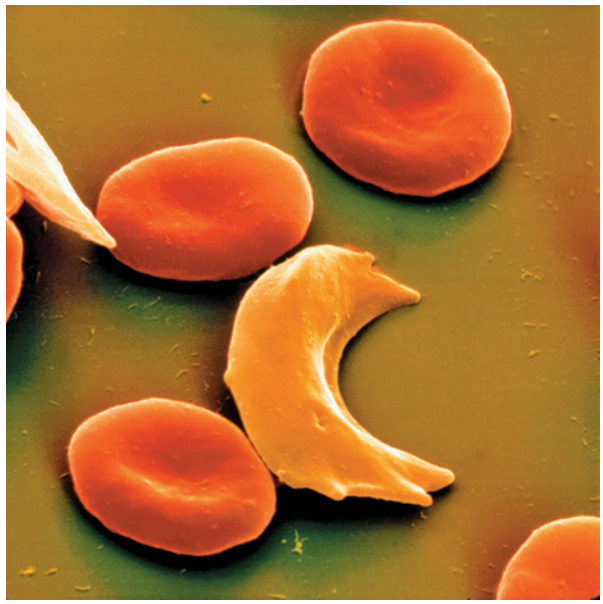


FIGURE 32.4 ■ Blood smear containing normal red blood cells and sickle cells

Source: © Eye of Science/Science Source.

Manifestations and complications The acute and chronic manifestations of sickle cell anaemia arise from episodes of RBC sickling. Sickling causes general manifestations of haemolytic anaemia, including pallor, fatigue, jaundice and irritability. Extensive sickling can precipitate a crisis due to occluded circulation, impaired erythropoiesis or sequestration of large amounts of blood in the liver or spleen.

A vaso-occlusive or thrombotic crisis occurs when sickling develops in the microcirculation. Obstruction of blood flow triggers vasospasm that halts all blood flow in the vessel. Lack of blood flow leads to tissue ischaemia and infarction. Vaso-occlusive crises are painful and last an average of 4 to 6 days. Infarction of small vessels in the extremities causes painful swelling of the hands and feet; large joints also may be affected. Priapism (persistent, painful erection of the penis) may develop. Abdominal pain may signal infarction of abdominal organs and structures. Infarction may affect bone marrow or lead to aseptic necrosis of affected bones. Stroke may result from cerebral vessel occlusion (Huether & McCance, 2013; Marieb & Hoehn, 2014). Skin ulcers may develop as the result of occluded vessels supplying the dermis. Repeated infarcts associated with sickling can affect the structure and function of nearly every organ system. People with sickle cell disease may develop an

FOCUS ON CULTURAL DIVERSITY Inherited haemolytic anaemias

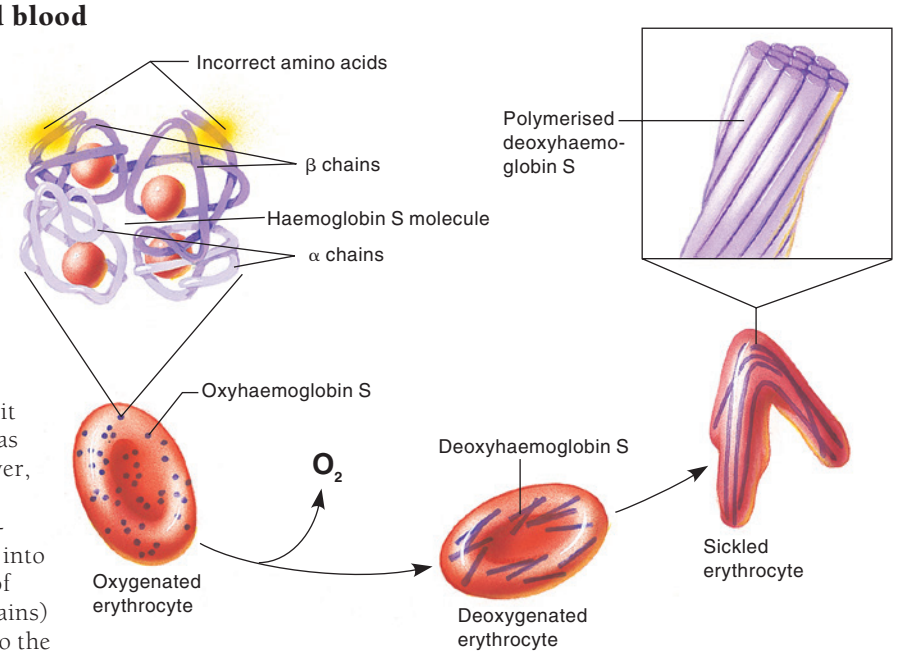
- Sickle cell disease is one of the most common inherited conditions of haemoglobin in the world.
- It is projected that the number of children born with sickle cell anaemia will increase from 305 800 in 2010 to 404 200 by 2050, with Nigeria and the Democratic Republic of Congo needing most assistance in prevention and management strategies (Piel et al., 2013).
- People from Central and South America, the Caribbean, Saudi Arabia and India, and Mediterranean countries such as Greece, Italy, Lebanon and Turkey, are affected. Additionally, peoples from sub-Saharan African countries are also at risk. The World Health Organization (WHO) (2013) estimates that 1 in 500 African-American births and 1 in every 1000 to 1400 Hispanic-American births are affected by sickle cell disease.
- Immigration of sub-Saharan African and Indian subcontinent peoples means that sickle cell disease is becoming more prevalent in Australia.
- Alpha-thalassaemia primarily affects people of South-East Asian, Indian, Chinese or Filipino ancestry. However, the disorder also occurs in other groups, including people from the Pacific Islands and New Zealand, and some Indigenous Australian communities in the Northern Territory and the north of Western Australia.
- Indigenous populations from the Kimberley region of Australia have the greatest incidences of thalassaemia in Australia (Babey & Manias, 2013).

Sickle cell anaemia

Haemoglobin S and red blood cell sickling

Sickle cell anaemia is caused by an inherited autosomal recessive defect in Hb synthesis. Sickle cell haemoglobin (HbS) differs from normal haemoglobin only in the substitution of the amino acid valine for glutamine in both beta chains of the haemoglobin molecule.

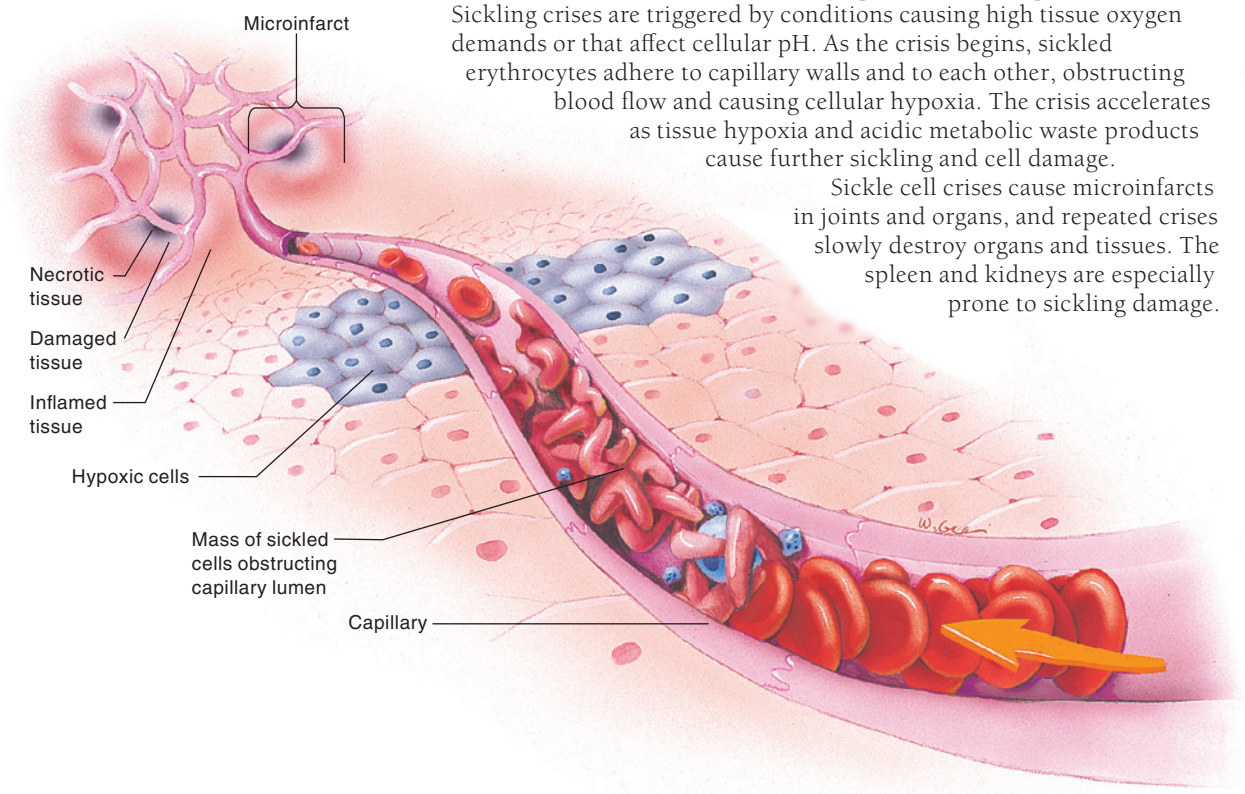
When HbS is oxygenated, it has the same globular shape as normal haemoglobin. However, when HbS offloads oxygen, it becomes insoluble in intracellular fluid and crystallises into rodlike structures. Clusters of rods form polymers (long chains) that bend the erythrocyte into the characteristic crescent shape of the sickle cell.



The sickle cell disease process

Sickle cell disease is characterised by episodes of acute painful crises. Sickling crises are triggered by conditions causing high tissue oxygen demands or that affect cellular pH. As the crisis begins, sickled erythrocytes adhere to capillary walls and to each other, obstructing blood flow and causing cellular hypoxia. The crisis accelerates as tissue hypoxia and acidic metabolic waste products cause further sickling and cell damage.

Sickle cell crises cause microinfarcts in joints and organs, and repeated crises slowly destroy organs and tissues. The spleen and kidneys are especially prone to sickling damage.



enlarged spleen and liver, renal insufficiency, gallstones and other manifestations of organ dysfunction. *Acute chest syndrome*, a symptom complex that includes fever, chest pain, an increasing WBC count and pulmonary infiltrates, may develop, as well as other pulmonary complications such as pneumonia, pulmonary infarction and pulmonary embolism. Treatment for sickle cell crisis is still predominately blood transfusion, but there have been recent advances in the use of nitric oxide inhalation for its vasodilatory properties (Marieb & Hoehn, 2014).

The shortened RBC lifespan and compromised erythropoiesis can lead to profound *aplastic anaemia* in sickle cell disease. *Sequestration crises* are marked by pooling of large amounts of blood in the liver and spleen. This sickle cell crisis only occurs in children, but is thought to be the cause of deaths in early childhood related to sickle cell disease (Huether & McCance, 2013).

THALASSAEMIA The **thalassaemias** are inherited disorders of haemoglobin synthesis in which either the alpha or the beta chains of the haemoglobin molecule are missing or defective. This leads to deficient haemoglobin production and fragile hypochromic, microcytic RBCs called *target cells* because of their distinctive bull's-eye appearance.

Thalassaemia usually affects certain populations. People of Mediterranean descent (southern Italy and Greece) are more likely to have beta-defect thalassaemias (often called *Cooley's anaemia* or Mediterranean anaemia). People of Asian ancestry, especially from Thailand, the Philippines and China, more often have alpha-defect thalassaemia. As with sickle cell anaemia, only one defective beta-chain forming gene may be present (*beta-thalassaemia minor*), causing mild symptoms; or both may be defective (*beta-thalassaemia major*), leading to more severe symptoms. Children with thalassaemia major rarely reach adulthood, although repeated blood transfusions may extend their lifespan (Huether & McCance, 2013). Four genes are responsible for alpha chain formation; one, two, three or all four may be defective. In the last case (*alpha-thalassaemia major*), death is inevitable and usually occurs in utero. Genetic studies and counselling are recommended for people at risk of this illness.

Manifestations and complications People with thalassaemia minor are often asymptomatic. When manifestations do occur, they include mild to moderate anaemia, mild splenomegaly, bronze skin colouring and bone marrow hyperplasia. The major form of the disease causes severe anaemia, heart failure and liver and spleen enlargement from increased red cell destruction. Fractures of the long bones, ribs and vertebrae may result from bone marrow expansion and thinning due to increased haematopoiesis. Jaundice may develop due to haemolysis, as well as hepatomegaly and splenomegaly. Accumulation of iron in the heart, liver and pancreas following repeated transfusions for treatment may eventually cause failure of these organs.

ACQUIRED HAEMOLYTIC ANAEMIA *Acquired haemolytic anaemia* results from haemolysis due to factors outside of the RBC. Causes of acquired haemolytic anaemias include:

- mechanical trauma to RBCs produced by prosthetic heart valves, severe burns, haemodialysis or radiation
- autoimmune disorders
- bacterial or protozoal infection
- immune-system-mediated responses, such as transfusion reactions
- drugs, toxins, chemical agents or venoms.

The manifestations of acquired haemolytic anaemia depend on the extent of haemolysis and the body's ability to replace destroyed RBCs. The anaemia itself often is mild to moderate as erythropoiesis increases to replace the destroyed RBCs. The spleen enlarges as it removes damaged or destroyed RBCs. If the breakdown of haem units exceeds the liver's ability to conjugate and excrete bilirubin, jaundice develops. When the condition is severe, bone marrow expands and bones may be deformed or may develop pathological fractures. The severity of generalised manifestations of anaemia (tachycardia, pallor, etc.) depends on the degree of anaemia and deficiency of tissue oxygenation.

GLUCOSE-6-PHOSPHATE DEHYDROGENASE (G6PD) ANAEMIA *Glucose-6-phosphate dehydrogenase (G6PD) anaemia* is caused by a hereditary defect in RBC metabolism. It is relatively common in people of African and Mediterranean descent. The defective gene is located on the X chromosome and therefore affects more males than females. There are many variations of this genetic defect.

G6PD is an enzyme that catalyses glycolysis, the process in which an RBC derives cellular energy. A defect in G6PD action causes direct oxidation of haemoglobin, damaging the RBC. Haemolysis usually occurs only when the affected person is exposed to stressors (e.g. drugs such as aspirin, sulfonamides or vitamin K derivatives) that increase the metabolic demands on RBCs. The G6PD deficiency impairs the necessary compensatory increase in glucose metabolism and causes cellular damage. Damaged RBCs are destroyed over a period of 7 to 12 days.

When exposed to a stressor triggering G6PD anaemia, symptoms develop within several days. These may include pallor, jaundice, haemoglobinuria (haemoglobin in the urine) and an elevated reticulocyte count. As new RBCs develop, counts return to normal.

Aplastic anaemia

In **aplastic anaemia**, the bone marrow fails to produce all three types of blood cells, leading to *pancytopenia*. Normal bone marrow is replaced by fat. Fortunately, aplastic anaemia is rare. *Fanconi anaemia* is a rare aplastic anaemia caused by defects of DNA repair. The underlying cause of about 50% of acquired aplastic anaemia is unknown (*idiopathic aplastic anaemia*). Other cases follow stem cell damage caused by exposure to radiation or certain chemical substances such as benzene, arsenic, nitrogen mustard, certain antibiotics (especially chloramphenicol) and chemotherapeutic drugs (Huether & McCance, 2013; Marieb & Hoehn, 2014). Aplastic anaemia also may occur with viral infections such as mononucleosis, hepatitis C and HIV disease.

In aplastic anaemia, the number of stem cells in the bone marrow is significantly reduced. The stem cell pool may be less

than 1% of normal when the disease is recognised. Anaemia develops as the bone marrow fails to replace RBCs that have reached the end of their lifespan. Remaining RBCs may be normochromic and normocytic, or may be large with increased mean corpuscular volume.

MANIFESTATIONS Manifestations of aplastic anaemia vary with the severity of the pancytopenia. Its onset usually is insidious, but may be sudden. Manifestations include fatigue, pallor, progressive weakness, exertional dyspnoea, headache and, ultimately, tachycardia and heart failure. Platelet deficiency leads to bleeding problems; bleeding gums, excessive bruising and nosebleeds may be the initial symptoms. A deficiency of white blood cells increases the risk of infection, causing manifestations such as sore throat and fever.

INTERPROFESSIONAL CARE

Ensuring adequate tissue oxygenation is the priority of care in treating anaemia. Specific therapy is determined by the underlying cause of the disorder. Usual treatments include medications, dietary modifications, blood replacement or supportive interventions. Table 32.1 outlines interprofessional care measures for selected types of anaemia.

Diagnosis

When anaemia is suspected, the following laboratory and diagnostic tests may be ordered:

- *Full blood count (FBC)* is done to determine blood cell counts, haemoglobin, haematocrit and red blood cell indices. The severity of the anaemia and the shape, volume and iron content of the RBCs can help determine the cause of anaemia.
- *Iron levels* and *total iron-binding capacity* are performed to detect iron deficiency anaemia. A low serum iron concentration and elevated total iron-binding capacity are indicative of iron deficiency anaemia.
- *Serum ferritin* is low due to depletion of the total iron reserves available for haemoglobin synthesis. Ferritin is an iron-storage protein produced by the liver, spleen and bone marrow. Ferritin mobilises stored iron when metabolic needs are higher than dietary intake.
- *Sickle cell test* is a screening test to evaluate haemolytic anaemia and detect HbS.
- *Haemoglobin electrophoresis* separates normal haemoglobin from abnormal forms. It is used to evaluate haemolytic anaemia, diagnose thalassaemia and differentiate sickle cell trait from sickle cell disease.
- *Schilling test* measures vitamin B_{12} absorption before and after intrinsic factor administration to differentiate between pernicious anaemia and intestinal malabsorption of the vitamin. A 24-hour urine sample is collected following administration of radioactive vitamin B_{12}. Lower than normal levels of the tagged B_{12} when intrinsic factor is given concurrently indicate malabsorption rather than pernicious anaemia.
- *Bone marrow examination* is done to diagnose aplastic anaemia. In aplastic anaemia, normal marrow elements are significantly decreased as they are replaced by fat cells. Nursing implications for bone marrow collection are described in the 'Nursing care of the person having bone marrow studies' box in Chapter 28.
- *Quantitative assay of G6PD* may be performed to confirm a diagnosis of glucose-6-phosphate dehydrogenase deficiency.

Medications

Medications used to treat anaemia depend on its cause. Iron replacement therapy is ordered for iron deficiency anaemia. Supplemental iron may be given by mouth or parenterally. Intravenous administration of iron is becoming more common, particularly in those with an acute deficiency and in anaemia associated with chronic GI blood loss, chronic renal failure and other chronic conditions that increase the need for blood cell production (e.g. cancers). When administering iron intravenously it needs to be diluted in normal saline and infused slowly to reduce the risk of anaphylaxis (MIMS, 2013). The risk of

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 7:
Blood and Blood Products

'The intention of this standard is to ensure that the patients who receive blood and blood products do so appropriately and safely.' (ACSQHC, 2011, p. 48)

Implementing this standard is achieved by the establishment of systems to ensure safe and appropriate prescription and administration of blood and blood products. These systems include processes facilitating accurate documentation, storage, transport, use and disposal. Effective communication regarding risks, benefits and use should exist between all individuals involved in a person's care (including the person themself and their significant others).

Caring for individuals experiencing haematological conditions will often result in the need to administer blood or blood products in order to manage the person's condition. As with any biological material, various risks are involved in all facets of this treatment. Efficient and appropriate systems are imperative to ensure the safety of not only the person receiving the product, but also any other individual involved in their care.

Source: © Australian Commission on Safety and Quality in Health Care.

TABLE 32.1 Interprofessional care focus for major anaemias

TYPE OF ANAEMIA	INTERPROFESSIONAL CARE
Iron deficiency anaemia	• Increased dietary intake of iron-rich foods • Oral or parenteral iron supplements
Vitamin B_{12} deficiency	• Increased dietary intake of foods containing vitamin B_{12} (e.g. meats, eggs and dairy products) • Oral or parenteral vitamin B_{12} supplements • Parenteral vitamin B_{12} for deficiency due to malabsorption or lack of intrinsic factor
Folic acid deficiency	• Increased dietary intake of foods rich in folic acid (folate) • Oral folic acid supplements • Folic acid supplements recommended for women who are pregnant or may become pregnant to prevent neural tube defects

Sickle cell anaemia	• Treatment is primarily supportive
	• Hydroxyurea 10–30 mg/kg per day
	• Sickle cell crisis:
	  • Rest
	  • Oxygen therapy to maintain SaO_2
	  • Narcotic analgesia
	  • Vigorous hydration
	  • Treatment of precipitating factors
	  • Nitric oxide inhalation
	• Acute chest syndrome:
	  • Careful hydration; haemodynamic monitoring
	  • Oxygen therapy
	  • Transfusion
	• Folic acid supplements
	• Blood transfusions during surgery or pregnancy as necessary
	• Genetic counselling recommended

Thalassaemia	• Regular blood transfusions • Folic acid supplements • Possible splenectomy • Genetic counselling
Aplastic anaemia	• Withdrawal of the causative agent, if known • Blood transfusions • Bone marrow transplant as indicated • Stem cell transplant—donor blood, umbilical cord blood, bone marrow sources

anaphylaxis is a major concern when iron dextran is given intravenously. Other parenteral iron solutions, including intravenous sodium ferric gluconate and iron sucrose (Venofer), carry a much lower risk of adverse and allergic reactions (Bryant & Knights, 2015; Crisp et al., 2013). For intramuscular administration of iron, a large deep muscle such as the ventrogluteal muscle is the preferred site. Utilising the Z track method of injection is recommended for the intramuscular administration of iron because it decreases irritation and staining and assists with sealing the medication in muscle tissue (Crisp et al., 2013).

Parenteral vitamin B_{12} is given when malabsorption or lack of intrinsic factor leads to vitamin B_{12} deficiency anaemia. Folic acid is ordered for women of childbearing age, pregnant women and those with folic acid deficiency or sickle cell anaemia to meet the increased demands of the bone marrow. Hydroxyurea, a drug that promotes foetal haemoglobin production, may be prescribed for people with sickle cell disease, particularly those with frequent crises or severe disease. Resulting increased levels of foetal haemoglobin interfere with the sickling process and reduce the incidence of painful crises. Nursing implications for people receiving iron, vitamin B_{12} and folic acid are found in the 'Medication administration' box below.

Erythropoietin may be ordered for people with low erythropoietin levels (e.g. those with chronic renal failure) and anaemia associated with other chronic diseases. Erythropoietin (Aranesp) is given subcutaneously or intravenously and may be given as often as three times a week in chronic renal failure. Because erythropoietin stimulates RBC production, adequate iron must be present. People receiving erythropoietin may require regular intravenous iron therapy as well.

Immunosuppressive therapy with antithymocyte globulin (ATG), corticosteroids and cyclosporine may be used to treat aplastic anaemia. Androgens may stimulate blood cell production in some people with aplastic anaemia. See Chapter 12 for more information about immunosuppression.

Nutrition

Dietary modifications are recommended for nutritional deficiency anaemias, such as iron deficiency anaemia, vitamin B_{12} deficiency anaemia or folic acid deficiency anaemia. Box 32.5 identifies good sources of dietary iron, folic acid and vitamin B_{12}.

MEDICATION ADMINISTRATION Drugs to treat anaemia

IRON SOURCES

Ferric carboxymaltose (Ferinject)
Ferrous sulfate (Ferro-Gradumet, Fefol, FGF)
Ferro fumerate (Ferro-tab)
Iron polymaltose (Ferrosig)
Iron sucrose (Venofer)

Iron preparations are normally taken by mouth and are absorbed from the gastrointestinal tract. They are given to treat anaemias resulting from iron deficiency or blood loss. When absorbed, iron combines with transferrin. This complex then is transported to the bone marrow and incorporated into haemoglobin.

Nursing responsibilities

- Prior to giving the drug, assess for use of drugs that might interact with iron (e.g. antacids, allopurinol, chloramphenicol, tetracyclines, vitamin E), gastrointestinal bleeding and manifestations of anaemia.
- Administer iron preparations with orange juice to enhance absorption.
- If using an elixir, give it through a straw to prevent staining the teeth.
- Monitor for manifestations of iron toxicity: nausea, diarrhoea or constipation; symptoms of anaphylactic shock (extreme cases).
- Monitor haemoglobin and reticulocyte counts.
- If the person is also taking tetracyclines, schedule the dose of iron 2 hours before the tetracycline (iron reduces the absorption of tetracyclines).
- When administering IM or IV, monitor closely for anaphylaxis.

Health education for the person and family

- Gastrointestinal side effects may be reduced by taking iron with food (but not milk, which decreases absorption).
- Stools may be dark green or black; this is harmless.
- Increase fluids and fibre in diet to decrease constipation.

VITAMIN B_{12} SOURCES

Cyanocobalamin oral, parenteral

Cyanocobalamin is used to treat vitamin B_{12} deficiencies or malabsorption and pernicious anaemia. It is rapidly absorbed when administered orally or by injection and it is stored in the liver. Intrinsic factor is necessary for absorption from the gastrointestinal tract.

Nursing responsibilities

- Do not expose crystalline injection to light.
- Assess for other drugs that might interfere with the therapeutic response: chloramphenicol, cimetidine, colchicine and timed-release potassium decrease its effectiveness.
- Do not mix cyanocobalamin in a syringe with other medications.
- Administer parenteral doses intramuscularly or deep subcutaneously to decrease local irritation.
- Monitor haemoglobin, RBC counts, reticulocyte counts and potassium levels.

Health education for the person and family

- A burning sensation with injection is temporary.
- Avoid alcohol, which interferes with absorption.
- If used to treat pernicious anaemia, the medication must be taken for life.

FOLIC ACID SOURCES

Folic acid (folate)

Synthetic folic acid is used to treat folic acid deficiency and megaloblastic or macrocytic anaemia. It is absorbed from the gastrointestinal tract and stored in the liver.

Nursing responsibilities

- Prior to giving the medication, assess for use of drugs that alter its effect: corticosteroids, methotrexate, oral contraceptives, phenytoin, sulfonamides.
- Do not mix folic acid with other medications in the same syringe.
- Monitor for possible hypersensitivity response of skin rash.

Health education for the person and family

- Large doses of folic acid may cause the urine to become darker yellow.
- Excess alcohol intake increases folic acid requirements.

Blood transfusion

Blood transfusions may be indicated to treat anaemias resulting from major blood loss, such as from trauma or major surgery and severe anaemia regardless of cause. In acute haemorrhage, whole blood may be given to replace both blood cells and volume. A unit of packed red blood cells may be given when anaemia is severe and the person demonstrates cardiovascular instability or compromise. Blood transfusions are fully discussed in Chapter 10.

Complementary therapies

Complementary healthcare practitioners may recommend specific plant enzymes to treat nutritional anaemias. There is limited quality evidence that complementary therapies can assist with the pathology or symptoms of anaemia.

Nursing care

For nursing care specific for a person with folic acid deficiency anaemia, see the accompanying nursing care plan.

Health promotion

Nursing measures to prevent anaemia focus on teaching good dietary habits to everyone, regardless of age. Stress the importance of consuming adequate amounts of iron, folate and the B vitamins. Provide a list of dietary sources of these nutrients. Discuss alternate iron sources with those with vegetarian diets and teach them that foods high in vitamin C enhance the

BOX 32.5 Dietary sources of iron, folic acid and vitamin B_{12}

Iron

Iron in the diet comes from two sources. *Haem iron* makes up about one-half of the iron from animal sources. *Non-haem iron* includes the remaining iron from animal sources and all the iron from plants, legumes and nuts. Haem iron promotes absorption of non-haem iron from other foods when both forms are consumed at the same time. Absorption of non-haem iron is also enhanced by vitamin C and inhibited by tea and coffee.

Sources of haem iron

- Beef
- Chicken
- Egg yolk
- Oysters
- Pork loin
- Turkey
- Veal

Sources of non-haem iron

- Bran flakes
- Brown rice
- Wholegrain breads
- Oatmeal
- Dried fruits
- Leafy green vegetables
- Dried beans

Sources of folic acid

- Green leafy vegetables
- Broccoli
- Organ meats
- Eggs
- Wheat germ
- Asparagus
- Liver
- Milk
- Yeast
- Kidney beans

Sources of vitamin B_{12}

- Liver
- Prawns and oysters
- Eggs
- Milk
- Kidney
- Meats (muscle)
- Cheese

absorption of iron from grains, legumes and other sources. Emphasise the importance of adequate iron intake in women of childbearing age and older adults. Stress the increased need for these nutrients during pregnancy and discuss strategies to ensure an adequate intake.

Assessment

Assessment data to collect for people with suspected anaemia include:

- *Health history:* complaints of shortness of breath with activity, fatigue, weakness, dizziness or fainting, palpitations; history of previous anaemia, bleeding episodes; menstrual history (if appropriate); medications; chronic diseases; usual diet and patterns of alcohol intake or cigarette smoking.
- *Physical examination:* general appearance, skin colour; vital signs, including temperature; heart and lung sounds; peripheral pulses, capillary refill; abdominal tenderness; obvious bleeding or bruising.
- *Diagnostic tests:* FBC, haemoglobin and haematocrit; bone marrow studies; specialised tests (e.g. haemoglobin electrophoresis, Schilling test).

Nursing diagnoses and interventions

Anaemia affects circulating oxygen levels and tissue oxygenation. Priority nursing diagnoses include activity intolerance, altered oral mucous membranes and self-care deficits. With acute blood-loss anaemia, risk of insufficient cardiac output also is a priority. People with sickle cell disease have specific needs related to the effects of the disease on tissue perfusion. See the section on disseminated intravascular coagulation later in this chapter for nursing interventions appropriate to ineffective tissue perfusion, associated pain and maintaining oxygenation.

Activity intolerance

Anaemia causes weakness and shortness of breath on exertion. These symptoms are due to decreased circulating oxygen levels secondary to low haemoglobin levels. Weakness, fatigue and/or vertigo may occur even during activities of daily living, including those associated with self-care, home life, job performance and social roles.

- Help identify ways to conserve energy when performing necessary or desired activities. *Modifying the approach to a particular activity may reduce cardiorespiratory symptoms and activity-related fatigue. Alternative ways of performing tasks (e.g. sitting when performing hygiene care and kitchen tasks) may reduce oxygen demands. In some cases, assistance from others is necessary to conserve energy and reduce symptoms.*
- Help the person and family establish priorities for tasks and activities. *Because family members may need to assume responsibility for additional tasks, the plan's success depends on mutually established goals.*
- Assist to develop a schedule of alternating activity and rest periods throughout the day. *Rest periods decrease oxygen needs, reducing strain on the heart and lungs and allowing restoration of homeostasis before further activities.*
- Encourage 8 to 10 hours of sleep at night. *Rest decreases oxygen demands and increases available energy for morning activities.*
- Monitor vital signs before and after activity. *Vital signs provide a measure of activity tolerance. Increased heart and respiratory rates or a change in blood pressure may indicate intolerance of the activity.*
- Discontinue activity if any of the following occurs:
  a. complaints of chest pain, breathlessness or vertigo
  b. palpitations or tachycardia that does not return to normal within 4 minutes of resting
  c. bradycardia
  d. tachypnoea or dyspnoea
  e. decreased systolic blood pressure.

  These changes may signify cardiac decompensation due to insufficient oxygenation. The intensity, duration or frequency of the activity needs to be reduced.

- Instruct the person not to smoke. *Smoking causes vasoconstriction and increases carbon monoxide levels in the blood, interfering with tissue oxygenation.*

Impaired oral mucous membrane

Glossitis and cheilosis may occur with nutritional deficiencies of iron, folate and vitamin B_{12}. The tongue and lips become very red and fissures or cracks may form at the corners of the mouth.

- Monitor condition of lips and tongue daily. *Glossitis and cheilosis increase the risk of bleeding and infection and may require medical treatment. Pain and discomfort may interfere with oral intake, further worsening the nutritional deficiency.*
- Use a mouthwash of saline, saltwater or half-strength peroxide and water to rinse the mouth every 2 to 4 hours. Avoid alcohol-based mouthwashes. *This cleanses and soothes oral mucous membranes. Alcohol-based mouthwashes further irritate and dry oral tissues.*
- Provide frequent oral hygiene (after each meal and at bedtime) with a soft-bristle toothbrush or sponge. *Removing food debris from painful fissures promotes comfort. A soft toothbrush reduces irritation or bleeding of oral mucosa. Keeping the oral cavity clean also reduces the risk of infection.*
- Apply a petroleum-based lubricating jelly or ointment to the lips after oral care. *Lubricating ointment helps to retain moisture, facilitate healing and protect the lips from other drying agents.*
- Instruct to avoid hot, spicy or acidic foods. *Such foods may further irritate and dry mucous membranes.*
- Encourage soft, cool, bland foods. *Foods that are soothing to the mucous membranes promote comfort and help maintain adequate food and fluid intake. Minimising oral pain may also promote compliance with oral care routines.*

NURSING CARE PLAN A person with folic acid deficiency anaemia

Iris Matthews is a 76-year-old widow who lives alone. She tells Lisa Kennedy RN, the practice nurse in the general practice surgery, that she liked to cook when her husband was alive, but preparing an entire meal just for herself seems senseless. She relates that her typical day's menu includes tea for breakfast, a ham sandwich and tea for lunch, a cup of soup, a few biscuits and a glass of milk for dinner.

ASSESSMENT

Mrs Matthews's nursing history includes a 9 kg weight loss since her husband died 8 months ago. She states that she sometimes has heart palpitations and always feels weak. Physical assessment shows: 37.1°C, P 110, R 22, BP 90/52. Skin warm, pale and dry. Diagnostic tests indicate folic acid deficiency anaemia. Mrs Matthews is started on an oral folic acid supplement and instructed about foods containing folic acid.

DIAGNOSES

- *Activity intolerance* related to weakness secondary to decreased tissue oxygenation.
- *Imbalanced nutrition: less than body requirements* related to lack of motivation to cook and understanding of nutritional needs, as manifested by weight loss of 9 kg and folic acid deficiency.
- *Deficient knowledge* related to lack of information about a well-balanced diet and foods containing folic acid.

PLANNING

- Discuss foods required for a well-balanced diet, as well as dietary sources of folic acid.
- Discuss the importance of taking the folic acid supplement. Advise to continue taking it even after she begins to feel better.

Expected outcomes

- Verbalise and understand the importance of taking folic acid supplements and eating a balanced diet.
- Gain at least 0.45 kg per week.
- Return to previous level of physical energy.
- Consume a balanced diet, including foods containing folic acid.

IMPLEMENTATION

- Develop a balanced dietary plan with Mrs Matthews that includes food preferences and foods that are easy and quick to prepare.
- Include foods that contain folic acid, or assist her in organising supplements.
- Help Mrs Matthews develop a schedule of activities that provides adequate rest and energy for cooking.

EVALUATION

Mrs Matthews gained 0.5 kg during the first week of treatment. She has met with a dietitian and has a better understanding of nutritional needs. She states that she can prepare hot meals when she schedules a rest period before and after lunch. She has provided written and verbal information about the folic acid supplement and diet. Mrs Matthews verbalises understanding, stating, 'I will continue to take the folic acid until the doctor tells me to stop. I'm beginning to enjoy cooking again, now that I have a reason to cook!' Ms Kennedy contacts the local seniors group to determine if Mrs Matthews is able to participate in the local Meals on Wheels program.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the pathophysiological basis for Mrs Matthews's abnormal vital signs during her initial assessment?
2. Design a week's menu that includes foods high in folic acid.
3. Why was Mrs Matthews placed on a folic acid supplement in addition to dietary modifications?
4. Why is the older adult at increased risk of developing folic acid deficiency anaemia? Consider physiological, economic and social factors.

REFLECTION ON THE NURSING PROCESS

1. Outline the most important communication and education points you have learned from this case study.
2. How can you use this information in your practice?

- Encourage eating four to six small meals daily with high protein and vitamin content. *Small, frequent meals may be better tolerated, increasing intake. Nutrient-rich meals promote healing of the mucous membranes.*

Risk of decreased cardiac output

Cardiac output may be affected by acute bleeding and volume loss or by heart failure resulting from severe anaemia. In addition, impaired tissue oxygenation leads to an increased respiratory rate and dyspnoea.

- Monitor vital signs, breath sounds and apical pulse. *Increased cardiac workload can affect the blood pressure, heart and respiratory rates. Increased blood flow can lead to heart murmur or abnormal heart sounds such as S_3 or S_4. Tachypnoea and dyspnoea may affect the depth of respirations, alveolar ventilation and blood and tissue oxygenation.*
- Assess for pallor, cyanosis and dependent oedema. *Blood is shunted to the vital organs, causing vasoconstriction of skin vessels. This, in addition to lower levels of haemoglobin, causes pallor. Cyanosis, especially of the lips and nail beds, indicates inadequate oxygenation of blood. Dependent oedema occurs in response to right ventricular failure.*

CONSIDERATION FOR PRACTICE

Report signs of decreased cardiac output to the medical officer. Severe anaemia can lead to heart failure, necessitating additional treatment.

- Closely monitor for manifestations of anaphylaxis (urticaria, erythema or flushing, oedema, wheezing, dyspnoea, nausea and vomiting, anxiety) when administering parenteral iron preparations, particularly iron dextran. Immediately notify the doctor and prepare to administer prescribed drugs such as adrenaline as ordered. Institute cardiopulmonary resuscitation measures as necessary. *Anaphylaxis, a systemic type I hypersensitivity (allergic) reaction, is a risk when administering parenteral iron preparations—iron dextran, in particular. Anaphylaxis can lead to severe cardiopulmonary compromise, necessitating emergency measures to preserve life.*

Self-care deficit

Energy expenditures for activities of daily living (ADLs) may cause oxygen demands to exceed supply in the person with severe anaemia.

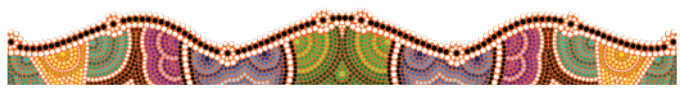
- Assist with ADLs, such as bathing, grooming and eating, as needed. *Assistance decreases energy expenditures and tissue requirements for oxygen, reducing cardiac workload.*
- Discuss the importance of rest periods prior to such activities as dressing. *Rest reduces oxygen demand and cardiac workload. The person who is able to perform self-care in ADLs maintains independence, self-esteem and morale.*

Community-based care

With the exception of anaemia resulting from acute haemorrhage, most people with anaemia are treated in the home and community setting. Include the following topics when preparing the person and family for home care:

- nutritional strategies to address deficiencies
- prescribed medications, vitamins or mineral supplements, and their appropriate use, intended effect, possible adverse effects, and interactions with food or other medications
- energy conservation strategies
- other recommended treatment measures and follow up
- if the anaemia is genetically transmitted, such as sickle cell anaemia, include inheritance patterns of the disorder, symptoms of crisis and manifestations to report to the doctor.

Provide referrals for counselling to facilitate decisions about pregnancy as indicated. Also refer for nutritional assistance and teaching, home healthcare or assistance with self-care and home maintenance activities as indicated. Older adults with nutritional anaemias may benefit from community services such as Meals on Wheels.



THE PERSON WITH MYELODYSPLASTIC SYNDROME

Myelodysplastic syndrome (MDS) is a group of blood disorders characterised by abnormal-appearing bone marrow and cytopenia (low numbers of circulating blood cells). MDS is not a single disease; at least five variations of the disorder have been identified. Anaemia that does not respond to treatment (*refractory anaemia*) is a characteristic of most forms of myelodysplasia.

Idiopathic MDS primarily affects older adults; men have a slightly higher incidence of the disorder than women. Risk factors for secondary MDS include exposure to environmental toxins such as cigarette smoke, benzene, radiation, radiation therapy or chemotherapy for cancer treatment, and other anaemias such as aplastic anaemia or, more rarely, Fanconi's anaemia (American Cancer Society, 2011; National Heart, Lung and Blood Institute, 2012).

FAST FACTS

- Idiopathic or primary MDS accounts for 70–80% of all identified cases.
- 20–30% of MDS cases occur as a secondary condition, related to factors such as age, smoking or exposure to environmental toxins, radiation, chemotherapy or other risk factors (Chantal et al., 2014; Demakos & Linebaugh, 2005).

Pathophysiology

MDS is a stem cell disorder in which stem cells fail to reproduce and differentiate into the various types of blood cells. The genetic components of stem cells (nuclear DNA and/or

mitochondrial DNA) are altered, The bone marrow loses its ability to produce normal blood cells, instead producing abnormal (*dysplastic*) cells, and ineffective haematopoiesis results. With significant alterations, leukaemia (proliferation of abnormal white blood cells) may develop in people with MDS.

Manifestations

Anaemia is the predominant early manifestation of MDS. The person may develop symptoms of the anaemia with increasing fatigue, weakness, dyspnoea and pallor. In many cases, the disorder is asymptomatic, identified when a routine blood test shows anaemia. Splenomegaly may develop, leading to discomfort and a feeling of fullness in the left upper quadrant of the abdomen. Hepatomegaly also may develop, leading to right upper quadrant discomfort. Thrombocytopenia can lead to abnormal bleeding tendencies, and neutropenia increases the risk of infection (Svensson et al., 2014).

INTERPROFESSIONAL CARE

People with MDS require long-term supportive care and therapy to maintain their quality of life. Stem cell transplant offers the only real hope for cure in MDS. See the 'Interprofessional care' section of 'The person with leukaemia' later in this chapter for more information about stem cell transplant and associated nursing care.

Diagnosis

- The *FBC* reveals anaemia. Although anaemia may be the only abnormality of the blood count, the WBC count also may be low, as may the platelet count. Abnormalities of size and shape may be noted in all blood cells.
- The *bone marrow* often appears normal, although precursor cells may have an abnormal appearance. Increased numbers of myeloblasts (granulocyte precursor cells) may be present in the bone marrow.
- *Serum erythropoietin*, *vitamin B_{12}*, *serum iron*, *total iron-binding capacity*, *ferritin levels* and *RBC folate levels* are drawn to help guide supportive therapy.

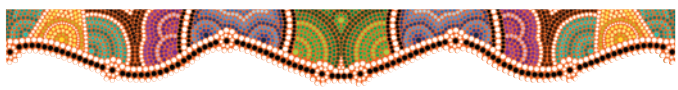
Treatment

Management of MDS is based on the severity of the disease. Several classification systems are available, including the French–American–British (FAB) classification system, the International Prognostic Scoring System (IPSS) and the World Health Organization classification system (Leukaemia Foundation, 2014). These systems are used to guide therapy for the person with MDS. One third of newly diagnosed patients with MDS are classed as high risk, with poor prognosis and survival rates (Svensson et al., 2014).

All those with MDS require monitoring, with regular GP visits and pathology evaluations. Psychosocial support is provided to assist the person and their family in dealing with a chronic, progressive and ultimately fatal disease.

People with MDS may require frequent red blood cell transfusions to treat the predominant anaemia. Each unit of packed RBCs contains 250 to 300 mg of iron. The body is unable to excrete this excess iron, so it accumulates, leading to problems such as endocrine dysfunction, cirrhosis, pericarditis and heart failure. *Iron chelation therapy* is used to remove excess iron from the body. Desferrioxamine (Desferal) is administered by slow intravenous infusion or continuous subcutaneous infusion using an infusion pump to maintain a normal or negative iron balance. This drug is relatively safe, although local skin reactions such as rash and urticaria may develop. An oral form of the drug, ferriprox, is available but is not widely used.

Blood cell growth factors may be administered to stimulate stem cell development in MDS, although the response rate is low. Platelet transfusions are given when bleeding occurs due to low platelet levels. Antibiotic therapy is initiated for bacterial infections (Leukaemia Foundation, 2014). Chemotherapy regimens similar to those employed to treat leukaemia may be used, but rarely are effective in treating MDS. Azacitidine (Vidaza), an antileukaemic agent that acts on abnormal blood-forming cells in the bone marrow, is more effective in treating MDS than standard chemotherapy regimens, and is now one of the first-line treatments for high-risk MDS patients (Svensson et al., 2014). As previously noted, stem cell transplant offers the only hope for cure. This high-risk therapy, however, is reserved for higher-risk people. Factors such as age, functional ability and other existing disease conditions help guide the decision to undergo stem cell transplant (Leukaemia Foundation, 2014).



Nursing care

Nursing diagnoses and interventions

Activity intolerance and the need for education about this disorder are the priorities of nursing care for the person with MDS being managed in a community-based setting. Although neutropenia and thrombocytopenia may accompany the anaemia of MDS, these problems are less common. See the section of this chapter on leukaemia for additional potential nursing diagnoses and interventions for the person with MDS.

Activity intolerance

The person with MDS experiences fatigue, weakness and shortness of breath on exertion related to the lack of RBCs and ineffective oxygen transport. These symptoms may affect the person's ability to maintain self-care, home life, job performance and social roles.

- Monitor vital signs, breath sounds and apical pulse. *Increased cardiac workload due to anaemia and impaired oxygen transport can affect the blood pressure, heart and respiratory rates. Increased blood flow can lead to heart murmur or abnormal heart sounds such as S_3 or S_4. Accumulated iron can lead to pericarditis and a pericardial friction rub.*
- Help identify energy-conserving ways of performing necessary or desired activities. *Alternative ways of*

performing tasks (e.g. sitting while performing hygiene measures) may reduce oxygen demands and fatigue.
- Help the person and family establish priorities for tasks and activities. *Because family members may need to assume responsibility for additional tasks, the plan's success depends on mutually established goals.*
- Suggest planning recreational activities following a transfusion and adjusting activity level between transfusions to match energy and minimise fatigue. *The person with MDS will have more energy and activity tolerance following a transfusion when RBC counts, haemoglobin and haematocrit approach normal levels and oxygen transport is optimal.*
- Encourage 8 to 10 hours of sleep at night. *Rest decreases oxygen demands and increases available energy for morning activities.*
- Discontinue activity if any of the following occurs:
  a. complaints of chest pain, breathlessness or vertigo
  b. palpitations or tachycardia that does not return to normal within 4 minutes of resting
  c. bradycardia
  d. tachypnoea or dyspnoea
  e. decreased systolic blood pressure.

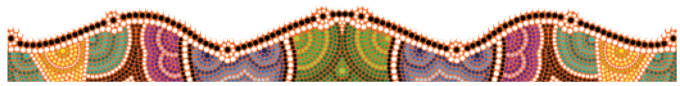
  These changes may signify cardiac decompensation due to insufficient oxygenation. The intensity, duration or frequency of the activity needs to be reduced.
- Instruct the person not to smoke. *Smoking causes vasoconstriction and increases carbon monoxide levels in the blood, interfering with tissue oxygenation.*

Risk of ineffective health maintenance

MDS is a chronic, usually progressive disorder, requiring active management to maintain functional status and quality of life. Regular visits to the doctor may be necessary. In addition, the person or family members may need to learn to administer iron chelation therapy or chemotherapy drugs and measures to prevent complications. The chronic nature of the disorder, and the often advanced age of the person and family caregivers, may interfere with effective management of the disorder.
- Assess knowledge of the disorder and the related treatments. *Assessment allows identification of knowledge gaps and provides a basis on which to provide additional information. Impaired disease management may be due to lack of knowledge or an inability to learn and perform psychomotor skills (e.g. administration of parenteral drug therapy).*
- Provide information about the disorder, its effects and prescribed medications and treatments. *Individualised instruction is more effective than general, possibly irrelevant information. The person and caregivers need to be able to identify and manage possible adverse effects of drug therapy, as well as recognise potential complications to be reported to the doctor.*
- Provide emotional support, expressing confidence in the person's and caregivers' abilities to manage care. *Emotional support helps the person and family caregivers incorporate the care regimen into their lifestyle.*
- Provide supervised learning and practice opportunities for administering parenteral medications if ordered. *Successful practice sessions instil confidence in the ability to manage care and provide an opportunity for questions and exploring alternatives.*

Community-based care

The person with myelodysplastic syndrome needs information about this chronic and ultimately fatal disease. Provide information about the various treatment options available, including management of any infusion device if ordered. Discuss the timing of and options for stem cell transplant and assist the person to evaluate the potential benefits and risks of this treatment option.



THE PERSON WITH POLYCYTHAEMIA

Polycythaemia, or *erythrocytosis*, is an excess of red blood cells characterised by a haematocrit higher than 55%. The two main types of polycythaemia are primary and secondary. A third type of polycythaemia, relative polycythaemia, results from a fluid volume deficit, not excess RBCs.

FAST FACTS

- *Primary polycythaemia* (*polycythaemia vera*) is uncommon.
  - In primary polycythaemia, RBC production is increased.
  - Primary polycythaemia more commonly affects men of European Jewish ancestry between the ages of 40 and 70.
- *Secondary polycythaemia* (*erythrocytosis*) is the most common form of polycythaemia.
  - Secondary polycythaemia occurs when erythropoietin levels are elevated.
  - It may affect people of any age or origin.
  - It usually develops in response to hypoxia (living at a high altitude, smoking or chronic lung disease).
- *Relative polycythaemia* occurs due to fluid deficit, not excess RBCs.
  - In relative polycythaemia the total RBC count is normal.
  - The haematocrit is elevated because of increased cell concentration.
  - It is corrected by rehydration.

Pathophysiology

Primary polycythaemia

Primary polycythaemia, or polycythaemia vera (PV), is a neoplastic stem cell disorder characterised by overproduction of RBCs and, to a lesser extent, white blood cells and platelets. It is classified as a myeloproliferative disorder. Its cause is unknown. In PV, colonies of endogenous erythroid stem cells develop. These colonies produce RBCs in the absence of erythropoietin, leading to excess RBC production.

MANIFESTATIONS Initially, PV is asymptomatic and the diagnosis may be made during routine blood tests. Its manifestations are caused by increased blood volume and viscosity. Hypertension is common and may lead to complaints of headaches, dizziness and vision and hearing disruptions. Venous stasis causes *plethora*, a ruddy, red colour of the face, hands, feet and mucous membranes. This often is accompanied by severe, painful itching of the fingers and toes. Retinal and cerebral vessels may be engorged. Hypermetabolism develops, causing weight loss and night sweats. Mental status may be altered, leading to drowsiness or delirium.

Thrombosis and haemorrhage are potential complications of PV. Thrombosis may cause transient ischaemic attacks, angina or manifestations of peripheral vascular disease. Gastrointestinal bleeding may occur and portal hypertension may develop.

Secondary polycythaemia

Secondary polycythaemia, or erythrocytosis, is increased numbers of RBCs in response to excess erythropoietin secretion or prolonged hypoxia. Secondary polycythaemia is the most common form of polycythaemia.

Abnormally high erythropoietin levels can result from kidney disease or erythropoietin-secreting tumours (e.g. renal cell carcinoma). Chronic hypoxia that stimulates erythropoietin release is a more common cause of secondary polycythaemia. People living at high altitudes where the atmospheric oxygen pressure is lower develop a degree of polycythaemia, as do people with chronic heart or lung disease and smokers. Abnormal haemoglobin that forms tighter bonds with oxygen also may lead to secondary polycythaemia. Blood doping in sport causes a purposeful artificial polycythaemia. Red blood cells are drawn off and stored, and the body responds by producing erythropoietin and increasing RBC production. The stored cells are reinfused, thus producing excess RBC or polycythaemia. This was done to increase the oxygen-carrying capacity for the athlete, but has since been banned (Marieb & Hoehn, 2014).

MANIFESTATIONS The manifestations of secondary polycythaemia are similar to those of primary polycythaemia. Splenomegaly, however, does not develop. Early symptoms often are overshadowed by the manifestations of the underlying disorder. For the manifestations of polycythaemia, see the box below.

MANIFESTATIONS Polycythaemia

- Hypertension
- Headache, tinnitus, blurred vision
- Plethora: dark redness of the lips, feet, ears, fingernails and mucous membranes
- Splenomegaly (polycythaemia vera)
- Severe pruritus, extremity pain
- Weight loss, night sweats
- Gastrointestinal bleeding
- Intermittent claudication
- Symptoms from thrombosis within various organs

INTERPROFESSIONAL CARE

Diagnosis

In polycythaemia vera, serum erythropoietin levels are low. Bone marrow studies show hyperplasia of all haematopoietic elements. With secondary polycythaemia, serum erythropoietin levels usually are high and bone marrow studies show only red stem cell hyperplasia.

Treatments

For secondary polycythaemia, treatment focuses on the underlying cause of the disorder. It is a physiological response in people living at high altitudes and, unless the haematocrit is too high or oxygen saturation levels are low, no treatment is usually necessary. Smokers are urged to quit. Measures to raise oxygen saturation levels and reduce tissue hypoxia often will relieve the polycythaemia. People with both primary and secondary polycythaemia benefit from periodic phlebotomy, removing 300 to 500 mL of blood, to keep blood volume and viscosity within normal levels. For PV, chemotherapeutic agents such as hydroxyurea may be used to suppress marrow function but may increase the risk of developing leukaemia (discussed later in this chapter). Pruritus may be relieved by antihistamines or may require more aggressive treatment with interferon alpha or other treatments. Aspirin may be prescribed daily to control thrombosis without increasing the risk of bleeding.

Nursing care

Preventing polycythaemia begins with educating children and adults about the dangers of smoking. Measures to reduce risk factors for cardiovascular disease also may be beneficial.

This chronic condition is managed in community-based settings unless a complication develops. Teach the person and family the importance of maintaining adequate hydration and increasing fluid intake during hot weather and when exercising. Discuss measures to prevent blood stasis: elevating legs and feet when sitting, using support stockings and continuing treatment measures. Instruct to report manifestations of thrombosis (leg or calf pain, chest pain, neurological symptoms) or bleeding (black, tarry stools, vomiting of blood or coffee-grounds emesis) immediately. Monitor the haematocrit and cell counts throughout treatment.

Examples of nursing diagnoses appropriate for the person with polycythaemia follow:

- *Pain* related to effects of altered blood flow in distal extremities.
- *Risk of ineffective tissue perfusion* related to sluggish blood flow and increased risk of thrombosis.

WHITE BLOOD CELL AND LYMPHOID TISSUE DISORDERS

Important disorders of the white blood cells (WBCs) and lymphoid tissue include the leukaemias, multiple myeloma and malignant lymphomas (Hodgkin's disease and non-Hodgkin's lymphoma). Review the physiology of WBCs and lymphoid tissues and assessment of their function in Chapter 28 before proceeding with this section.

THE PERSON WITH LEUKAEMIA

Leukaemia (literally, 'white blood') is a group of chronic malignant disorders of white blood cells and white blood cell precursors. Precursor cells are stem cells that have developed to the stage where they are committed to forming a particular kind of new blood cell. All leukaemias start in the bone marrow where developing blood cells, usually developing white cells, undergo a malignant change.

In leukaemia, the usual ratio of red to white blood cells is reversed. Leukaemias are characterised by replacement of bone marrow by malignant immature white blood cells, abnormal immature circulating WBCs and infiltration of these cells into the liver, spleen and lymph nodes throughout the body. There are four main types of leukaemia. They are discussed later in this section.

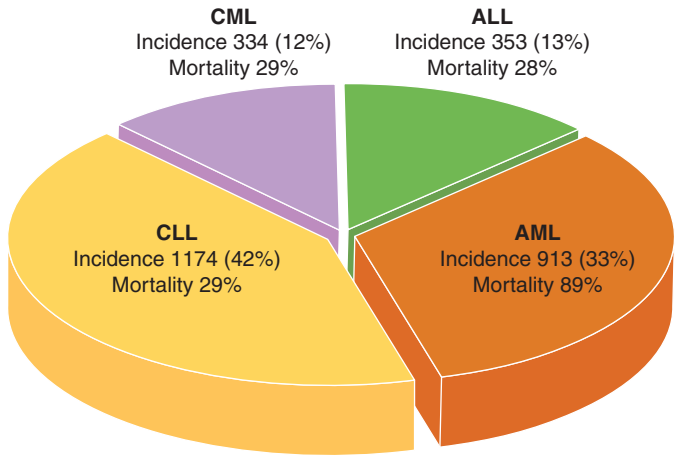


FIGURE 32.5 ■ Australian incidence and mortality data for the four main types of leukaemia, 2011

ALL = acute lymphoblastic leukaemia. AML = acute myeloid leukaemia. CLL = chronic lymphocytic leukaemia. CML = chronic myeloid leukaemia.

Source: Generated using data from the Australian Cancer Incidence and Mortality (ACIM) books, 2015a–d.

Incidence and risk factors

Although leukaemia is often thought of as a childhood disease, it is diagnosed 10 times more often in adults than in children; the majority over 50 years old (Leukaemia Foundation, 2014). Each year in Australia, over 3000 people are diagnosed with leukaemia, making it the seventh most common type of cancer in men and the eighth most common type of cancer in women (Australian Institute of Health and Welfare (AIHW), 2014). See Figure 32.5 for leukaemia incidence and mortality statistics.

Although the cause of most leukaemia is unknown, certain risk factors have been identified. Men are affected more frequently than women. People with certain genetic disorders such as Down syndrome have a higher incidence of leukaemia. Environmental risk factors play a role as well. Risk factors for myeloid leukaemia include cigarette smoking and chemicals such as benzene (present in cigarette smoke and petrol). Exposure to ionising radiation increases the risk of several types of leukaemia. People who have undergone treatment for cancer have an increased risk. The human T-cell leukaemia/lymphoma virus-1, a retrovirus, is known to cause certain leukaemias and lymphomas (Leukaemia Foundation, 2014).

Physiology review

White blood cells are the most diverse of the cellular components of the blood. White blood cells arise from three different precursor cells: myeloblasts, which further differentiate into the granular leucocytes (granulocytes), neutrophils, eosinophils and basophils; monoblasts, which mature into circulating monocytes and ultimately into macrophages; and lymphoblasts, which become lymphocytes and mature in lymphoid tissue to B cells and T cells.

As a whole, the primary function of WBCs is to help maintain the body's immune defences. Neutrophils, the most numerous WBC in circulation, are active phagocytes, the first cells to arrive to injured tissue. Monocytes and macrophages also are phagocytic cells that dispose of foreign and waste material from tissues. Eosinophils and basophils are more specialised. Eosinophils are primarily involved in allergic responses and parasitic infections. Basophils are actively involved in the inflammatory response, releasing substances such as histamine and heparin into inflamed tissues. Lymphocytes, the smallest of the WBCs, are an integral part of the immune system. B cells are part of the humoral immune response, producing antibodies to specific antigens. T cells are part of the cell-mediated immune response. For more information about the inflammatory and immune responses, see Chapter 12. The normal WBC count and differential are presented in Table 32.2 (Van Leeuwen, Poelhuis-Leth & Blandh, 2013).

Pathophysiology

Leukaemia begins with malignant transformation of a single stem cell. Leukaemic cells proliferate slowly, but do not differentiate normally. They have a prolonged lifespan and accumulate in the bone marrow. As they accumulate, they compete with the proliferation of normal cells. Leukaemic cells do not function as mature WBCs and are ineffective in the inflammatory and immune processes. Leukaemic cells replace normal

TABLE 32.2 Normal white blood cell count and differential

LABORATORY TEST	VALUE
WBC count	3.7–11.0 × 10^9/L
Differential WBC count:	
Neutrophils	2.0–7.5 × 10^9/L
Eosinophils	0.05–0.5 × 10^9/L
Basophils	< 0.1 × 10^9/L
Lymphocytes	1.5–3.7 × 10^9/L
Monocytes	0.2–0.4 × 10^9/L

haematopoietic elements in the marrow. Because erythrocyte- and platelet-producing cells are crowded out, severe anaemia, splenomegaly and bleeding difficulties result.

Leukaemic cells leave the bone marrow and travel through the circulatory system, infiltrating other body tissues such as the central nervous system, testes, skin, gastrointestinal tract and the lymph nodes, liver and spleen. Death usually is due to internal haemorrhage and infections.

Manifestations

The general manifestations of leukaemia (regardless of type) result from anaemia, infection and bleeding. These include pallor, fatigue, tachycardia, malaise, lethargy and dyspnoea on exertion. Infection may cause fever, night sweats, oral ulcerations and frequent or recurrent respiratory, urinary, integumentary or other infections. Increased bleeding due to thrombocytopenia leads to bruising, petechiae, bleeding gums and bleeding within specific organs and tissues. Multisystem effects of leukaemia are shown below.

Other manifestations result from leukaemic cell infiltration, increased metabolism and increased leucocyte destruction. Infiltration of the liver, spleen, lymph nodes and bone marrow causes pain and tissue swelling in the involved areas. Meningeal infiltration may cause manifestations of increased intracranial pressure, such as headache, altered level of consciousness, cranial nerve impairment, nausea and vomiting. Infiltration of the kidneys may affect renal function, with decreased urine output and increased blood urea nitrogen and creatinine. Increased metabolism causes heat intolerance, weight loss, dyspnoea on exertion and tachycardia. Destruction of large numbers of WBCs releases substantial amounts of uric acid into the circulation; uric acid crystals may obstruct renal tubules, causing renal insufficiency.

Without treatment, leukaemia is invariably fatal, usually due to complications of leukaemic cell infiltration of bone marrow or vital organs. With treatment, prognosis varies. The overall 5-year survival rate is 48% (Leukaemia Foundation, 2014). Survival rates differ by type of leukaemia: people with acute myeloid leukaemia can have a 10–30% 5-year survival rate, with a mean of 24%, favouring the younger generation in response to treatment. Those with chronic lymphocytic leukaemia may not even require treatment, or may need it only after 10 years or more, as their 5-year survival rate is around 73% (Leukaemia Foundation, 2010; Cancer Council Australia, 2015; Australian Cancer Research Foundation, 2012). The types, pathology, manifestations and treatment for the major leukaemias are outlined in Table 32.3.

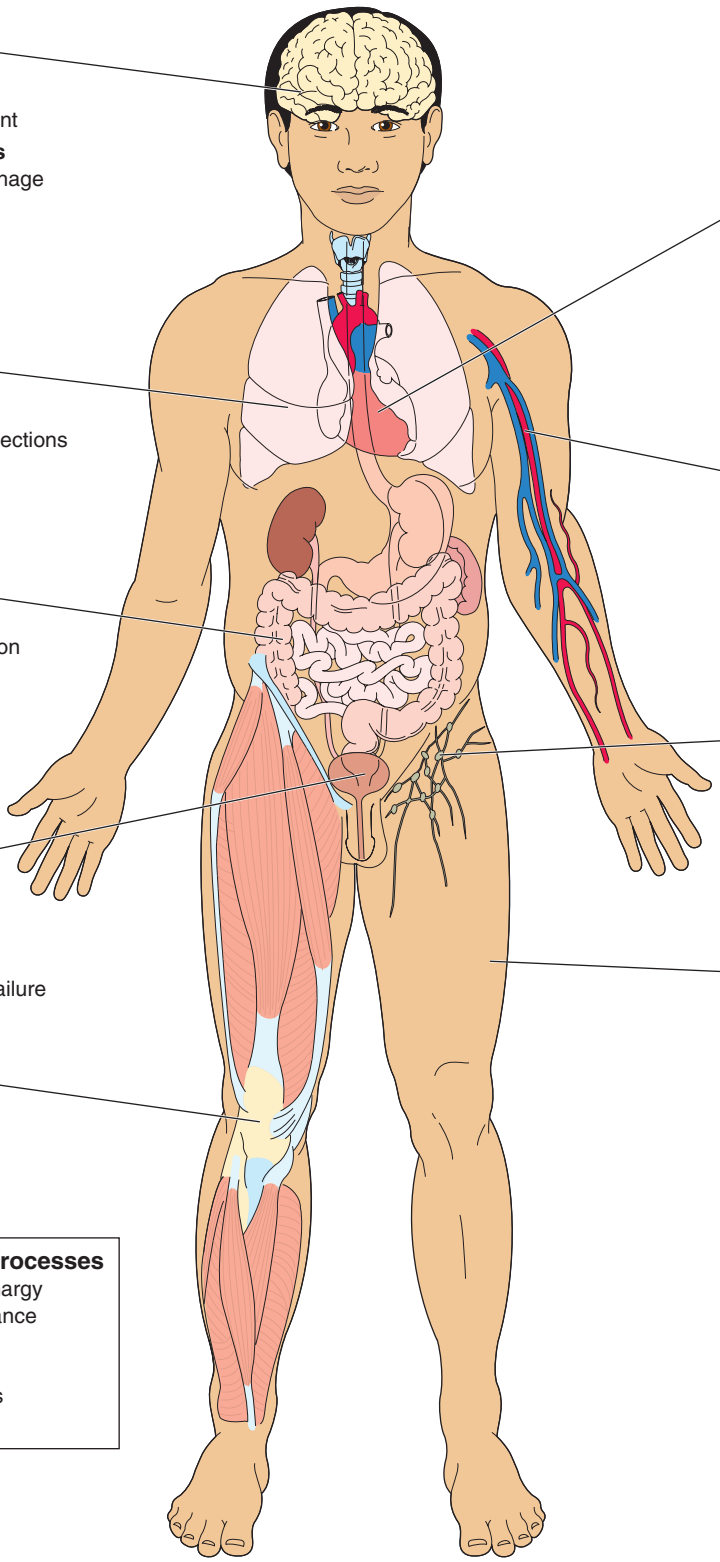
Classifications

Leukaemias are classified by their acuity and by the predominant cell type involved. The *acute* leukaemias are characterised by an acute onset, rapid disease progression and immature or undifferentiated blast cells. *Chronic* leukaemias, on the other hand, have a gradual onset, prolonged course and abnormal, mature-appearing cells. *Lymphocytic* (or *lymphoblastic*) leukaemias involve immature lymphocytes and their precursor cells in the bone marrow. Lymphocytic leukaemias infiltrate

TABLE 32.3 Major types of leukaemia

CLASSIFICATION	CHARACTERISTICS	MANIFESTATIONS	TREATMENT
Acute lymphoblastic leukaemia (ALL)	Primarily affects children and young adults; leukaemic cells may infiltrate CNS	Recurrent infections; bleeding; pallor, bone pain, weight loss, sore throat, fatigue, night sweats, weakness	Chemotherapy; bone marrow transplant (BMT) or stem cell transplant (SCT)
Chronic lymphocytic leukaemia (CLL)	Primarily affects older adults; insidious onset and slow, chronic course	Fatigue; exercise intolerance; lymphadenopathy and splenomegaly; recurrent infections, pallor, oedema, thrombophlebitis	Often requires no treatment; chemotherapy; BMT
Acute myeloid leukaemia (AML)	Common in older adults, may affect children and young adults. Strongly associated with toxins, genetic disorders and treatment of other cancers	Fatigue, weakness, fever; anaemia; headache; bone and joint pain; abnormal bleeding and bruising; recurrent infection; lymphadenopathy, splenomegaly and hepatomegaly	Chemotherapy; SCT
Chronic myeloid leukaemia (CML)	Primarily affects adults; early course slow and stable, progressing to aggressive phase in 3–4 years	Early: weakness, fatigue, dyspnoea on exertion; possible splenomegaly Later: fever, weight loss, night sweats	Interferon alpha; chemotherapy with imatinib, hydroxyurea and combination chemotherapy

MULTISYSTEM EFFECTS OF LEUKAEMIA



Neurological
- Headache
- Altered LOC
- Cranial nerve impairment

Potential complications
- Subarachnoid haemorrhage
- Retinal haemorrhage
- Seizures, coma

Respiratory
- Dyspnoea on exertion
- Pharyngitis, sore throat
- Frequent respiratory infections

Potential complication
- Pulmonary bleeding

Gastrointestinal
- Anorexia, nausea
- Oral ulcerations, infection
- Bleeding gums
- Gingival hyperplasia (gum overgrowth)
- Abdominal pain
- Hepatomegaly
- Occult GI bleeding

Urinary
- Urinary tract infection
- Haematuria

Potential complication
- Renal insufficiency or failure

Musculoskeletal
- Weakness
- Bone tenderness, pain
- Joint pain

Cardiovascular
- Tachycardia, palpitations
- Orthostatic hypotension
- Heart murmurs
- Haematomas
- Oedema

Potential complications
- Haemorrhage
- Thrombophlebitis

Hematological
- Anaemia
- Thrombocytopenia
- Leucopenia
- Bleeding (epistaxis)
- Splenomegaly

Potential complication
- DIC

Immunological
- Frequent or recurrent infections
- Lymphadenopathy

Potential complications
- Abscesses
- Septicaemia

Integumentary
- Skin and mucous membrane pallor
- Petechiae
- Bruising, purpura
- Ulcerations
- *Chloromas* (skin infiltrations near bony prominences)

Metabolic processes
- Malaise, lethargy
- Heat intolerance
- Diaphoresis
- Chills, fever
- Night sweats
- Weight loss

TABLE 32.4 FAB classification of acute leukaemia

TYPE	CLASS	PREDOMINANT CELLS	PROGNOSIS
Acute lymphocytic leukaemia	L_1	Immature lymphoblasts	> 90% remission rate in children
	L_2	Mature lymphoblasts	Relapse common after 2 or more years of remission
Acute myeloid leukaemia	M_0	Undifferentiated cells	Poor
	M_1	Immature myeloblasts	Good; complete response in 65% or more
	M_2	Mature myeloblasts	Good for 2 or more years of remission
	M_3	Promyelocytes	Good in adults
	M_4	Myelocytes and monocytes	Poorest in adults
	M_5	Poorly or well-differentiated monocytes	Poor
	M_6	Predominant erythroblasts	Variable
	M_7	Megakaryocytes	Poor

the spleen, lymph nodes, CNS and other tissues. *Myeloid* (also called *myelogenous*, *myelocytic* or *myeloblastic*) leukaemias involve myeloid stem cells in the bone marrow, interfering with the maturation of all types of blood cells, including granulocytes, RBCs and thrombocytes (Huether & McCance, 2013). Acute lymphoblastic leukaemia is the most common type of leukaemia in children. In adults, acute myeloid leukaemia and chronic lymphocytic leukaemia are the most common types (Huether & McCance, 2013). In summary, the general types of leukaemia are as follows:

- acute myeloid (myeloblastic) leukaemia (AML)
- chronic myeloid (myelogenous) leukaemia (CML)
- acute lymphocytic (lymphoblastic) leukaemia (ALL)
- chronic lymphocytic leukaemia (CLL).

This general system of classifying leukaemias does not differentiate subtypes of acute leukaemias. The FAB system for classifying acute leukaemias further differentiates acute leukaemias by the predominant cell involved and the degree of cell differentiation (see Table 32.4).

Acute myeloid leukaemia

Acute myeloid leukaemia (AML) is characterised by uncontrolled proliferation of myeloblasts (the precursors of granulocytes) and hyperplasia of the bone marrow and spleen (see Figure 32.6). Treatment induces complete remission in 66% of people, although only about 30–40% achieve cure or long-term remission (Huether & McCance, 2013).

The manifestations of AML result from neutropenia and thrombocytopenia. Decreased neutrophils lead to recurrent severe infections, such as pneumonia, septicaemia, abscesses and mucous membrane ulceration. The manifestations of thrombocytopenia include petechiae, purpura, ecchymoses (bruising), epistaxis (nosebleeds), haematomas, haematuria and gastrointestinal bleeding. Bone infarctions or subperiosteal infiltrates of leukaemic cells may cause bone pain. Anaemia is a late manifestation, causing fatigue, headaches, pallor and dyspnoea on exertion. Death usually results from infection or haemorrhage.

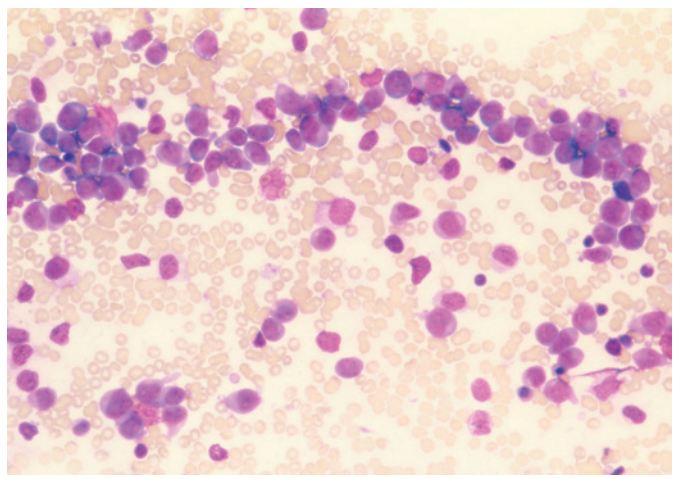


FIGURE 32.6 ■ A blood smear from the bone marrow of a person with acute myeloid leukaemia. Note the abnormally large number of myelocyte WBCs (stained purple) among the small RBCs

Source: Dr Gopal Murti/Science Source.

Bone marrow aspiration shows a proliferation of immature WBCs. The FBC shows thrombocytopenia and normocytic, normochromic anaemia.

Chronic myeloid leukaemia

Chronic myeloid leukaemia (CML) is characterised by abnormal proliferation of all bone marrow elements. This type of leukaemia constitutes approximately 15% of adult leukaemias, and 0.03% of all diagnosed cancers. It affects men more frequently than women. The onset of CML is most common after the age of 50, although it is seen in children and adolescents as well (Leukaemia Foundation, 2014).

CML is usually associated with a chromosome abnormality called the Philadelphia chromosome, a balanced translocation of chromosome 22 to chromosome 9 (see Figure 32.7). The fusion gene produced by this translocation, known as *bcr/abl*,

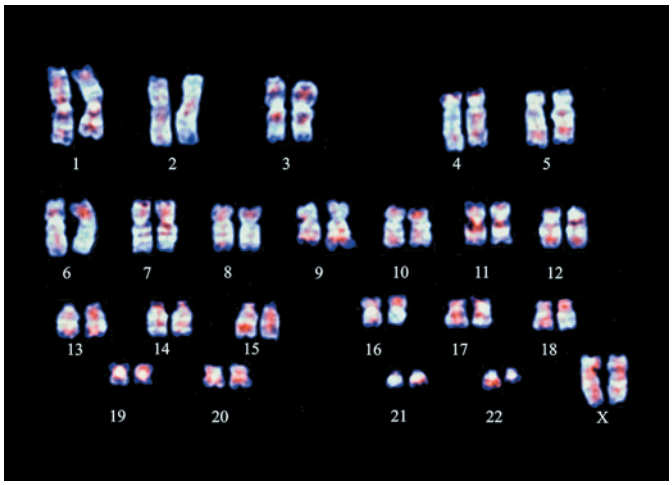


FIGURE 32.7 ■ The Philadelphia chromosome. Note the chromosomes of pairs 9 and 22. In each instance, the left-hand chromosome of the pair is normal, whereas an exchange of material between chromosomes has made the right-hand chromosome 9 larger and the right-hand chromosome 22 smaller. In stem cells within the bone marrow, the chromosome 22 defect leads to chronic myeloid leukaemia

Source: © Addenbrooke's Hospital/Science Source.

is an *oncogene* capable of initiating a malignancy. Very large doses of ionising radiation also may induce CML in some people (Papadakis et al., 2013). The incidence of CML in Australia has increased from around 200 to 330 new cases each year, with excessive radiation the only recognised cause (Leukaemia Foundation, 2014).

People with CML are often asymptomatic in the early stages and, in fact, are often diagnosed when a routine blood test reveals abnormal cell counts. Anaemia causes weakness, fatigue and dyspnoea on exertion. The spleen often is enlarged, causing abdominal discomfort. Within 3 to 4 years, disease progresses to a more aggressive phase. Rapid cell proliferation and hypermetabolism cause fatigue, weight loss, sweating and heat intolerance. The spleen enlarges, leading to a sensation of abdominal fullness and discomfort. Platelet function is affected in this stage, leading to bleeding and increased bruising. Finally, the disease evolves to acute leukaemia, with blast cell proliferation. This stage, known as the *terminal blast crisis phase*, is characterised by significant constitutional manifestations, splenomegaly and infiltration of leukaemic cells into the skin, lymph nodes, bones and CNS (Huether & McCance, 2013). Survival following the onset of this final stage averages only 2 to 4 months.

Acute lymphocytic leukaemia

Acute lymphocytic leukaemia (ALL) is the most common type of leukaemia in children and young adults. In adults, ALL is rarely seen until late middle age and then its incidence increases with ageing. Genetic factors may play a role in its development, particularly the *bcr/abl* translocation also implicated in CML.

Most (80%) cases of ALL result from malignant transformation of B cells, with the remaining 20% arising from T cells. The malignant cells resemble immature lymphocytes (*lymphoblasts*); however, they do not mature or function effectively to maintain immunity. These lymphoblasts accumulate in the bone marrow, lymph nodes and spleen, as well as in circulating blood. Some types of lymphoma (discussed later in this chapter) are thought to represent a later stage of the same disease.

The onset of ALL is usually rapid. Lymphoblasts proliferating in bone marrow and peripheral tissues crowd the growth of normal cells (see Figure 32.8). Normal haematopoiesis is suppressed, leading to thrombocytopenia, leucopenia and anaemia. Manifestations of infections, bleeding and anaemia develop. Bone pain resulting from rapid generation of marrow elements, lymphadenopathy and liver enlargement are also common. Infiltration of the CNS causes headaches, visual disturbances, vomiting and seizures.

The FBC shows an elevated WBC count with increased lymphocytes on the differential. RBC and platelet counts are decreased. Bone marrow studies reveal a hypercellular marrow with growth of lymphoblasts. Combination chemotherapy produces complete remission in 80–90% of adults with ALL.

Chronic lymphocytic leukaemia

Chronic lymphocytic leukaemia (CLL) is characterised by proliferation and accumulation of small, abnormal, mature lymphocytes in the bone marrow, peripheral blood and body tissues. The abnormal cells are usually B lymphocytes that are unable to produce adequate antibodies to maintain normal immune function. Only about 5% of CLL involves T cells (Noonan, 2007). CLL occurs more commonly in adults, especially in older adults (median age 65). CLL is the least common type of the major leukaemias.

CLL has a slow onset and is often diagnosed during a routine physical examination. If symptoms are present, they usually include vague complaints of weakness or malaise. Possible

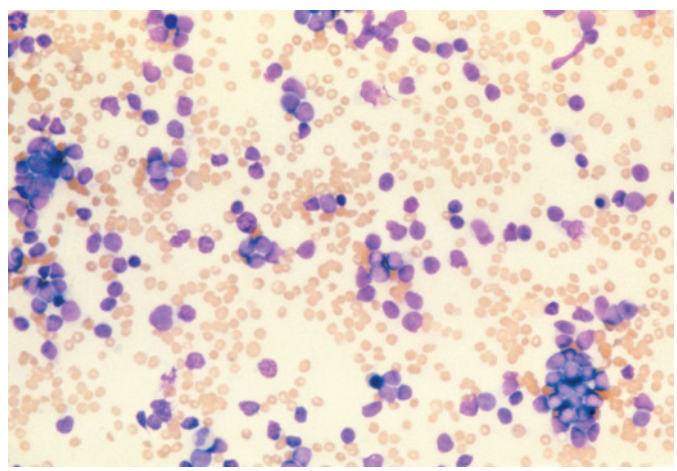


FIGURE 32.8 ■ A blood smear from the bone marrow of a person with acute lymphocytic leukaemia. Note the abnormally large number of lymphocytes (stained purple) crowding the bone marrow. As a result, normal production of RBCs, functional WBCs and platelets is suppressed

Source: Dr Gopal Murti/Science Source.

clinical findings include anaemia, infection and enlarged lymph nodes, spleen and liver. As in other leukaemias, bone marrow hyperplasia is present. Erythrocyte and platelet counts are reduced. Leucocyte counts may either be elevated or reduced, but abnormal cells are always present. In CLL, years may elapse before treatment is required. Survival of this disease averages approximately 7 years.

INTERPROFESSIONAL CARE

Treatment for leukaemia focuses on achieving remission or cure and relieving symptoms. The methods of treatment may include chemotherapy, radiation therapy and bone marrow or stem cell transplantation. Cure is more often achieved in children with acute leukaemia than in adults, although long-term remissions (disease-free periods with no signs or symptoms) often can be achieved.

Diagnosis

The following diagnostic tests are ordered when leukaemia is suspected:

- *FBC* with differential is done to evaluate cell counts, haemoglobin and haematocrit levels, and the number, distribution and morphology (size and shape) of WBCs.
- *Platelets* are measured to identify possible thrombocytopenia secondary to the leukaemia and the risk of bleeding.
- *Bone marrow examination* provides information about cells within the marrow, the type of erythropoiesis and the maturity of erythropoietic and leucopoietic cells.
- Table 32.5 outlines usual diagnostic test results in the various forms of leukaemia.

Chemotherapy

Single agent or combination chemotherapy is the treatment of choice for most types of leukaemia, with the goal of eradicating leukaemic cells and producing remission. Table 32.6 outlines typical chemotherapy regimens for different types of leukaemia. Combination chemotherapy reduces drug resistance and toxicity and interrupts cell growth at various stages of the cell cycle, producing a complementary effect of the drugs used. Cancer treatment with chemotherapy is discussed in detail in Chapter 13.

Chemotherapy for leukaemia generally is divided into the induction phase and post-remission therapy. During *induction*, drug doses are high to eradicate leukaemic cells from the bone marrow. These high doses often also damage stem cells and interfere with production of normal blood cells. Circulating mature blood cells are not affected because they are no longer dividing. The degree of bone marrow suppression is influenced by a number of factors, including age, nutritional status, concurrent chronic diseases such as impaired liver or renal function, the drug and drug dose, and prior treatment.

Colony-stimulating factors (CSFs), also called haematopoietic growth factors, often are administered to 'rescue' the bone marrow following induction chemotherapy. CSFs are cytokines that regulate the growth and differentiation of blood cells. Factors that support neutrophil maturation, *granulocyte-macrophage CSF (GM-CSF)* and *granulocyte CSF (G-CSF)*, are commonly used. Bone pain is a common side effect of therapy with these agents. People may also experience fevers, chills, anorexia, muscle aches and lethargy.

Once remission has been achieved, post-remission chemotherapy is continued to eradicate any additional leukaemic cells, prevent relapse and prolong survival. A single chemotherapeutic agent, combination therapy or bone marrow transplant may be used for post-remission treatment.

Radiation therapy

Radiation therapy damages cellular DNA. While the cell continues to function, it cannot divide and multiply. Cells that divide rapidly, such as bone marrow and cancer cells (radiosensitive cells), respond quickly to radiation therapy. Although

TABLE 32.5 Diagnostic findings by type of leukaemia

TEST	AML	CML	ALL	CLL
RBC count	Low	Low	Low	Low
Haemoglobin	Low	Low	Low	Low
Haematocrit	Low	Low	Low	Low
Platelet count	Very low	High early, low late	Low	Low
WBC count	Varies	Increased	Varies	Increased
Myeloblasts	Present			
Neutrophils	Decreased	Increased	Decreased	Normal
Lymphocytes		Normal		Increased
Monocytes		Normal/low		
Bone marrow	Hypercellular		Hypercellular	
Myeloblasts	Present			
Lymphoblasts			Present	
Lymphocytes				Present

TABLE 32.6 Chemotherapeutic regimens used to treat leukaemia

Acute myeloid leukaemia	• Cytarabine (Cytoxan, an alkylating agent), *with* daunorubicin (Cerubidine, an antitumour antibiotic) *or* idarubicin (Idamycin, an antitumour antibiotic)
Chronic myeloid leukaemia	• Imatinib mesylate (Gleevec) or dasatinib (*bcr/abl* tyrosine kinase (enzyme) inhibitors), nilotinib (Tasigna) (2nd generation tyrosine kinase inhibitor)
	• Hydroxyurea (a DNA inhibitor) if imatinib not tolerated
Acute lymphocytic leukaemia	• Daunorubicin (Cerubidine, an antitumour antibiotic) *with* vincristine (Oncovin, a plant alkaloid) *with* prednisone *with* asparaginase
Chronic lymphocytic leukaemia	• Fludarabine (Fludara, an antimetabolite) *or* chlorambucil (Chloromycetin, an antitumour antibiotic)
	• Cyclophosphamide (Cytoxan, an alkylating agent), vincristine and prednisone
	• Cyclophosphamide, doxorubicin (Adriamycin, an antitumour antibiotic), vincristine and prednisone

normal cells are affected, they are better able to recover from the damage caused by the radiation than are cancer cells. The types of delivery, effects and toxicities of radiation are discussed in greater detail in Chapter 13.

Bone marrow transplant

Bone marrow transplant (BMT) is the treatment of choice for some types of leukaemia (see Table 32.3). BMT often is used in conjunction with or following chemotherapy or radiation. There are two main categories of BMT: in allogeneic BMT, the bone marrow of a healthy donor is infused into the person with the illness; in autologous BMT, the person is infused with their own bone marrow.

ALLOGENEIC BMT *Allogeneic BMT* uses bone marrow cells from a donor (often from a sibling with closely matched tissue antigens; closely matched unrelated donors also may be used). Prior to allogeneic BMT, high doses of chemotherapy and/or total body irradiation are used to destroy leukaemic cells in the bone marrow. The donor's bone marrow is aspirated (see Figure 32.9) and infused through a central venous line into the recipient. Prior to BMT and re-establishment of bone marrow function, the person is critically ill and at significant risk of infection and bleeding due to depletion of WBCs and platelets.

AUTOLOGOUS BMT *Autologous BMT* uses the person's own bone marrow to restore bone marrow function after chemotherapy or radiation. This procedure is often called *bone marrow rescue*. In autologous BMT, about 1 L of bone marrow is aspirated (usually from the iliac crests) during a period of disease remission. The bone marrow is then frozen and stored for use after treatment. If relapse occurs, lethal doses of chemotherapy or radiation are given to destroy the immune system and malignant cells and to prepare space in the bone marrow for new cells. The filtered bone marrow is then thawed and infused intravenously through a central line. The infused marrow cells slowly become a part of the person's bone marrow, the neutrophil count increases and normal haematopoiesis takes place.

As in allogeneic BMT, the person is critically ill during the period of bone marrow destruction and immunosuppression. The person is hospitalised in a private room for 6 to 8 weeks or more. Potential complications include malnutrition, infection and bleeding.

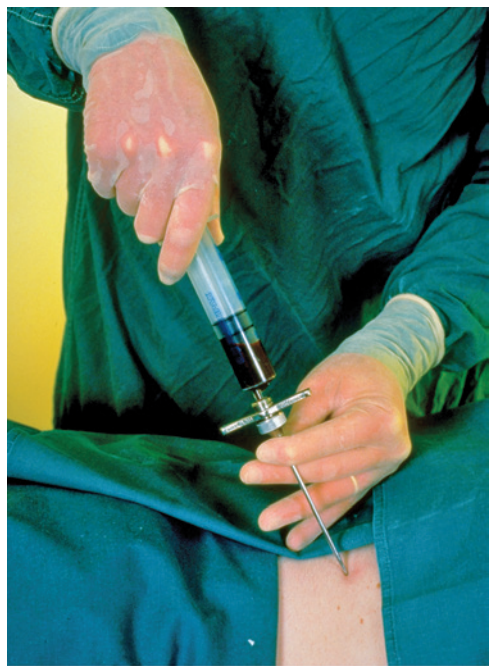


FIGURE 32.9 ■ Allogeneic bone marrow transplant. Bone marrow from the donor is aspirated, then filtered and infused into the recipient

Source: © Simon Fraser/Science Source.

Stem cell transplant

Allogeneic **stem cell transplant (SCT)** is an alternative to bone marrow transplant. SCT results in complete and sustained replacement of the recipient's blood cell lines (WBCs, RBCs and platelets) with cells derived from the donor stem cells.

Donors must have tissue that is closely matched with that of the recipient. Prior to harvesting, haematopoietic growth factors, including G-CSF and GM-CSF, are administered to the donor for 4 to 5 days. This increases the concentration of stem cells in peripheral blood, allowing it to be used for the transplant instead of bone marrow. Peripheral blood is removed and white cells are separated from the plasma, then administered via a large central venous catheter. Large concentrations of stem cells also are present in umbilical cord blood. This may be stored and used (Ballen, Gluckman & Broxmeyer, 2013).

The recipient undergoes similar treatment prior to SCT as for BMT. The risks of infection and other complications, as well as graft-versus-host disease, are similar.

Graft-versus-host disease

Allogeneic BMT or SCT may precipitate *graft-versus-host disease (GVHD)*, which develops in up to 80% of all people receiving an allogeneic BMT or SCT, depending on their risk factors (Dignam et al., 2012). In GVHD, immune cells of the donated bone marrow identify the recipient's body tissue as foreign. Consequently, T lymphocytes in the donated marrow attack the liver, skin and GI tract, causing skin rashes progressing to desquamation (loss of skin), diarrhoea, GI bleeding and liver damage. *Acute GVHD* develops within days or weeks of the transplant and is usually marked by a pruritic, maculopapular rash that begins on the palms and soles of the feet and may extend over the entire body. Vaso-occlusive disease of the liver affects up to 25% of allogeneic bone marrow transplant recipients, with jaundice and elevated liver function tests (Grossman & Mattson, 2013). *Chronic GVHD* develops later, 100 or more days after the transplant, affecting 20–50% of people who survive 6 months or more following allogeneic BMT or SCT. It may follow acute GVHD or develop in people with no prior symptoms. GVHD is treated with antihistamines, calcineurin inhibitors, antibiotics and steroids. Immunosuppressant drugs such as thalidomide are now not recommended as being effective, but sirolimus (Rapamycin) has been shown to be more useful. Other second-line treatments can be trialled depending on severity of symptoms and response to treatment. Such agents include extracorporeal photopheresis, anti-TNF antibodies and interleukin-2 receptor antibodies (Dignam et al., 2012).

Biological therapy

Cytokines such as interferons and interleukins are biological agents that may be used to treat some leukaemias. These agents modify the body's response to cancer cells; in some cases they are cytotoxic as well. Interferons are a complex group of messenger proteins normally produced in response to antigens such as viruses (see Chapter 11). They have multiple effects, including moderating immune function and inhibiting abnormal cell proliferation and growth. Side effects commonly associated with interferon therapy include flu-like symptoms, persistent fatigue and lethargy, weight loss and muscle and joint pain.

Complementary therapies

Although many complementary and alternative medicine therapies have been purported to treat cancer in general, at this time none has been shown to have sustained benefit in treating leukaemia. Clinical trials have demonstrated the efficacy of both coping skills training (relaxation and imagery) and hypnosis to significantly reduce oral discomfort associated with leukaemia and its treatment.

Nursing care

For nursing care specific to the person undergoing diagnostic testing for leukaemia, see the accompanying nursing care plan.

Health promotion

Health promotion activities related to leukaemia include teaching about leukaemia risk factors, particularly those that can be controlled. Discuss the potential dangers of exposure to ionising radiation and certain chemicals such as benzene. Encourage all persons to avoid smoking cigarettes. Discuss genetic counselling with people at high risk of having a child with Down syndrome (women over age 35).

Assessment

Focused assessment data related to leukaemia include:

- *Health history:* complaints of fatigue, weakness, dyspnoea on exertion, frequent infections, sore throat, night sweats, bleeding gums or nosebleeds; recent weight loss; exposure to ionising radiation (multiple x-rays, residence near a site of radiation or atomic testing) or chemicals (occupational); prior treatment for cancer; history of an immune disorder.
- *Physical examination:* skin and mucous membranes for bruising, purpura, petechiae, ulcers or lesions; pallor; vital signs, including orthostatic vitals; heart and lung sounds; abdominal examination; stool for occult blood.
- *Diagnostic tests:* blood count with differential; bone marrow studies.

Nursing diagnoses and interventions

When caring for the person with leukaemia, the nurse considers the chronic and life-threatening nature of the disease as well as the effects of treatment. See the 'Translation to practice' box below. Priority nursing problems may include *Risk of infection*, *Imbalanced nutrition: less than body requirements*, *Impaired oral mucous membranes*, *Ineffective protection (bleeding)* and *Anticipatory grieving*.

Risk of infection

Changes in white blood cell function impair the immune and inflammatory responses in leukaemia, increasing the risk of infection. WBCs may be immature and ineffective or, in some cases, deficient. Chemotherapy or radiation therapy further depresses bone marrow function and increases the risk of infection.

- Promptly report manifestations of infection: fever, chills, throat pain, cough, chest pain, burning on urination, purulent drainage and itching and burning in vaginal or rectal areas. *Prompt reporting allows timely intervention to prevent overwhelming infection and sepsis.*
- Institute infection protection measures:
  a. Maintain protective isolation as indicated.
  b. Ensure meticulous hand hygiene among all people in contact with the person.
  c. Assist as needed with appropriate hygiene measures.
  d. Restrict visitors with colds, flu or infections.
  e. Provide oral hygiene after every meal.
  f. Avoid invasive procedures when possible, including injections, intravenous catheters, catheterisations and rectal and vaginal procedures. When necessary, use strict aseptic technique for all invasive procedures and monitor carefully for infection.

NURSING CARE PLAN A person with acute myelocytic leukaemia

Catherine Cole is a 37-year-old secretary who lives with her husband, Ray, and teenage daughter, Amy, in an apartment in a large metropolitan area. About 2 months ago, Mrs Cole began to tire easily and experience night sweats several times a week. She also noted that she was pale, bruised easily and was having heavier menstrual periods. Blood tests ordered by her local general practitioner (GP) are abnormal. She is admitted for a bone marrow biopsy.

ASSESSMENT

Mary Grant, RN, obtains a nursing history and physical assessment for Mrs Cole. Catherine tells her, 'I'm so tired and I have these bruises all over me. I'm so afraid of the results of the bone marrow examination. I don't know what we will do if I have cancer.' Mrs Cole clutches her husband's hand and begins to cry. Physical assessment data include height 156 cm; weight, 48.1 kg; vital signs: T 36.5°C, P 102, R 22, BP 130/82. Numerous petechiae scattered over trunk and arms; ecchymoses noted on lower right arm and right calf. Oral mucosa is red, with several small ulcerations in buccal areas.

Blood count shows reduced RBCs, haemoglobin and haematocrit levels. The WBC is high, with myeloblasts seen on differential. The platelet count is very low. A tentative diagnosis of acute myelogenous leukaemia is made.

DIAGNOSES

- *Risk of infection* related to altered WBC production and immune function.
- *Ineffective protection* related to reduced platelet count and risk of bleeding.
- *Impaired oral mucous membrane* secondary to anaemia and reduced platelets.
- *Fatigue related to anaemia.*
- *Anxiety* related to fear of leukaemia diagnosis.

PLANNING

- Monitor and reduce the risk of infection and bleeding.
- Assist with management of self-care issues.
- Assist with management of emotional state.

Expected outcomes

- Remain free of infection.
- Experience no significant bleeding.
- Have intact oral mucous membranes.
- Manage self-care activities despite fatigue.
- Develop strategies for managing anxiety.

IMPLEMENTATION

- Place in a private room, if available.
- Limit visitors to immediate family/significant others for the present (consider cultural implications around family, illness and hospitalisation).
- Instruct all staff, the family and person to carefully wash hands. Post a sign on the door and over the washbasin in the room as a reminder.
- Record vital signs every 4 hours.
- Avoid invasive procedures unless absolutely necessary.
- Monitor for bleeding every 4 hours, including skin, oral mucosa, abdominal assessment, body fluids and menstrual pad count.
- Instruct to perform oral hygiene every 2 to 4 hours, using a soft-bristle toothbrush.
- Ask the dietitian to work with Mrs Cole to identify preferred foods. Instruct to avoid foods that may damage oral mucosa, such as very hot, very cold or highly acidic or spicy foods.
- Provide for periods of rest alternating with activity.
- Teach about the bone marrow biopsy. Allow time for questions and to talk through fears.
- Refer to the oncology nurse specialist for further teaching and support.
- Refer to an oncology social worker where economic or social challenges are pressing.
- Provide written and/or online links to further information and support services for people living with a diagnosis of acute myelocytic leukaemia.

EVALUATION

The bone marrow biopsy confirms the diagnosis of acute myelogenous leukaemia. Mrs Cole is very upset, but calms as the doctor and the oncology nurse discuss treatment plans and the possibility of remission. She decides to have outpatient chemotherapy. During her hospital stay, Mrs Cole remained free of infection or further bleeding. She tells RN Grant that her mouth feels better, although it is still painful. During routine assessment, Mrs Cole remarks, 'You know, I was so scared when I came here, but I think I am a little less so now. Sometimes not knowing what is wrong is worse than knowing.'

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe how alterations in WBCs can increase a person's susceptibility to infection.
2. List sources of potential infection for the hospitalised person.
3. What is the rationale for having the person do her own oral and physical hygiene?
4. Outline a teaching plan for this person and her family for home care to prevent infection.
5. Develop a care plan for Mrs Cole for the nursing diagnosis *Activity intolerance*.

REFLECTION ON THE NURSING PROCESS

1. What are the take-home points that you have learned from this case study?
2. How can you use them in your daily practice area? Where will you find further information about this disease and its effects on people who live with it?

These precautions minimise exposure to bacterial, viral and fungal pathogens. Infection is the main cause of death in people with leukaemia. Mucous membranes are especially susceptible to breakdown and infection as a result of tissue damage from chemotherapy or radiation.

- Monitor vital signs, including temperature and oxygen saturation, every 4 hours. Report temperature spikes with chilling, tachypnoea, tachycardia, restlessness, change in PaO_2 and hypotension. *The inflammatory response may be impaired in leukaemia, masking signs of infection until*

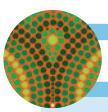
sepsis develops, indicated by manifestations such as those above.

- Monitor neutrophil levels (measured in cubic millimetres) for relative risk of infection:
  - 2.0 to 2.5 $\times 10^9$/L: no risk
  - 1.0 to 2.0 $\times 10^9$/L: minimal risk
  - 0.5 to 1.0 $\times 10^9$/L: moderate risk
  - Below 0.5 $\times 10^9$/L: severe risk.
  - *Neutrophils are the first line of defence against infection. As levels decrease, the risk of infection increases.*
- Explain infection precautions and restrictions and their rationale; explain that these measures are usually temporary. *Understanding of these aspects by the person and family increases compliance and lowers the risk of infection.*

Imbalanced nutrition: less than body requirements

The person with leukaemia may have difficulty meeting nutritional needs due to increased metabolism, fatigue, loss of appetite from radiation, nausea and vomiting from chemotherapy, or painful oral mucous membranes that make chewing and swallowing difficult and/or painful.

- Weigh regularly and evaluate weight loss over time to determine degree of malnutrition. *A weight loss of 10–20% may indicate malnutrition. A minimum intake of nutrients is necessary for health and tissue repair; cancer increases metabolic needs over this basal requirement. Weight loss occurs when metabolic requirements are not met. Both the disease process and its treatment can interfere with nutrient intake.*
- Address causative or contributing factors to inadequate food and fluid intake:
  a. Provide mouth care before and after meals; use a soft toothbrush or sponges as necessary.
  b. Provide liquids with different textures and tastes.
  c. Increase liquid intake with meals.
  d. Reduce intake of milk and milk products, which makes mucus more tenacious.
  e. Assist to a sitting position for eating.
  f. Ensure that the environment is clean and odour free.
  g. Provide medications for pain or nausea 30 minutes before meals, if prescribed.
  h. Provide rest periods before meals.
  i. Offer small, frequent meals including low-fat, high-kilojoule foods throughout the day.



TRANSLATIONTO PRACTICE Evidence-based practice for people with acute leukaemia and lymphoma

People with acute leukaemia and malignant lymphoma experience a number of distressing manifestations of their disease, including malaise and fatigue, fever, night sweats, infections and possible haemorrhage. Treatments such as radiation therapy and chemotherapy often have numerous adverse effects as well, including anorexia and nausea, stomatitis, lethargy, malaise and fatigue. In studies, people in remission from acute leukaemia or malignant lymphoma were surveyed regarding physical problems, their view of help they received and how supported they felt afterwards, and the impact of the disease and treatment on their current life, including what stressors played the largest part (Jones et al., 2015; Parry et al., 2011).

Jones et al. (2015) outlined changing stressors impacting on quality of life dependent on the age of the person in remission. They clustered post-treatment distress into physical, emotional and economic clusters. There was less distress with increasing age, but the physical impact, predominately fatigue, remained larger throughout the ages. Parry et al. (2010) in their study found that there was limited follow up and continuity of care post active treatment, and ongoing needs were not met, suggesting that there is a need for nurses to continue with care and resources beyond the actual treatment time. Patients felt abandoned and unsupported once treatment had been completed, but stressors still continued. They were not aware of the psychological support that was necessary.

IMPLICATIONS FOR NURSING

Nurses need to actively focus their care on the physical problems experienced during treatment, especially energy loss and nutritional problems. Overwhelming fatigue interferes with the person's ability to provide self-care, but its effects may not be readily apparent to nurses. People need to become equipped to deal with the remission phase as well as the treatment phase, and feel safe and supported when home from treatment. The long-term effects physically, emotionally and financially indicate a need for continued follow-up care, teaching, resource availability and ongoing access to support, with possible referral to counselling services as required.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Explain the physiological responses to malignancies and cancer treatments that cause fatigue, malaise and nutritional problems.
2. People undergoing treatment for leukaemia, malignant lymphoma and other cancers may have few outward manifestations of their disease or responses to treatment. Discuss how this apparent wellbeing may affect nurses' perceptions of care needs.
3. How may continued physical and emotional problems of fatigue and lack of psychological and sexual energy affect family relations?
4. Develop a nursing care plan for a person with acute leukaemia to address the nursing diagnosis of *Ineffective sexuality patterns* related to fatigue and lack of energy.
5. Appreciate the experiences of people who live with these diseases. Everyone's experience is different. Read the stories of people who are living with leukaemias, lymphomas, myeloma or a related blood disorder. Available from Leukaemia Australia: www.leukaemia.org.au/web/aboutdiseases/living_patientsstories.php.

j. Provide commercial supplements, such as Ensure.
k. Avoid painful or unpleasant procedures immediately before or after meals.
l. Suggest measures to improve food tolerance, such as eating dry foods when arising, consuming salty foods if allowed and avoiding very sweet, rich or greasy foods.

Anorexia, nausea and vomiting, diarrhoea, stomatitis, taste changes and dysphagia often make eating difficult during cancer treatment when good nutrition is most important. Maintaining nutritional status decreases morbidity and mortality by preventing weight loss, improving the response to treatment, minimising adverse effects and improving quality of life. Small, frequent meals are often better tolerated, especially high-protein, high-kilojoule foods.

Impaired oral mucous membrane

Stomatitis, inflammation and ulceration of the oral mucous membrane, is common in leukaemia. Chemotherapy can further impair the integrity of constantly dividing oral tissues.

- Inspect the buccal region, gums, sublingual area and the throat daily for swelling or lesions. Ask about oral pain or burning. *Breakdown of the oral mucous membrane increases the risk of infection and bleeding, causes pain and discomfort with eating and swallowing, and may cause swelling that interferes with the airway.*
- Culture any oral lesions. *Herpes simplex virus and* Candida *(yeast) are more common in those with neutropenia. Herpes lesions are usually red, raised, fluid-filled blisters;* Candida *causes a white coating and patches of white plaque.*
- Assist with mouth care and oral rinses with saline or a baking soda solution. Apply lip balm to the lips to prevent dryness and cracking. *These measures help prevent infection and increase comfort.*
- Encourage use of soft-bristle toothbrush or sponge to clean teeth and gums. *Toothbrushes with hard bristles may abrade inflamed mucosa, causing bleeding and increasing the risk of infection.*
- Administer medications as ordered to treat infection or relieve pain. *Topical antifungal agents such as nystatin may be prescribed to treat* Candida *infections. Topical anaesthetics such as lignocaine may be prescribed to relieve discomfort and facilitate good oral care.*
- Instruct to avoid alcohol-based mouthwashes, citrus fruit juices, spicy foods, very hot or very cold foods, alcohol and crusty foods. Suggest bland, cool foods and cool liquids at least every 2 hours. *Avoiding mucosa-traumatising foods and liquids increases comfort; bland, cool foods and liquids cause the least pain. Intake of adequate fluids is necessary to prevent dehydration.*
- If the patient is undergoing treatments such as haematopoetic stem cell transplant they may benefit from palifermin—a recombinant human keratinocyte growth factor-1, which decreases the duration and incidence of mucositis (BMJ Best Practice, 2015).

Ineffective protection

Bleeding is the second most common cause of leukaemia deaths. As platelet counts decrease, the risk of bleeding increases (see the section later in this chapter on thrombocytopenia). Tumour lysis syndrome also is a risk in people with leukaemia who are undergoing their initial treatment with chemotherapy. Tumour lysis syndrome develops when a large number of malignant cells are destroyed by treatment with chemotherapy or radiation. The resultant by-products of cell lysis can overwhelm the body's ability to effectively eliminate them, leading to hyperkalaemia, hyperphosphataemia with secondary hypocalcaemia and hyperuricaemia.

- Assess vital signs every 4 hours and body systems every shift for bleeding:
  a. skin and mucous membranes for petechiae, ecchymoses and purpura
  b. gums, nasal membranes and conjunctiva for bleeding
  c. vomitus, stool and urine for visible or occult blood
  d. vaginal bleeding
  e. prolonged bleeding from puncture sites
  f. neurological changes such as headache, visual changes, altered mentation, decreased level of consciousness, seizures
  g. abdomen for complaints of epigastric pain, diminished bowel sounds, increasing abdominal girth, rigidity or guarding.

  Early identification of bleeding helps prevent significant blood loss and potential shock. Internal haemorrhage may lead to tachycardia, hypotension, pallor and diaphoresis. Bleeding into the lungs may cause dyspnoea; bleeding into the abdomen causes increased girth, pain and guarding. Intracranial bleeding affects mental status and level of consciousness.
- Avoid invasive procedures such as rectal temperatures and suppositories, vaginal douches, suppositories, tampons, urinary catheterisation and parenteral injections if possible. *Diagnostic procedures such as biopsy or lumbar puncture should not be done if the platelet count is low and there is risk of bleeding. Invasive procedures can cause tissue trauma and bleeding. Procedures that use large-bore needles should be delayed until the platelet count is increased.*
- Apply pressure to injection sites for 3 to 5 minutes and to arterial punctures for 15 to 20 minutes. *Pressure prevents prolonged bleeding by prompting haemostasis and clot formation.*
- Instruct to avoid forcefully blowing or picking the nose, forceful coughing or sneezing, and straining to have a bowel movement. *These activities can damage mucous membranes, increasing the risk of bleeding.*
- Monitor and promptly report abnormal blood levels of electrolytes, uric acid, urea nitrogen and creatinine, or manifestations of tumour lysis syndrome. *Significant alterations in electrolyte levels can lead to complications such as cardiac arrhythmias, muscle weakness or tetany, paraesthesias and mental status changes. Excess uric acid can compromise renal function and lead to metabolic acidosis and gout.*

- Maintain adequate hydration and administer prescribed medications such as allopurinol and diuretics as ordered. *Hydration is vital to maintain renal function and promote elimination of tumour lysis by-products. Allopurinol reduces the risk of uric acid crystallisation in the kidneys and other tissues* (Bryant & Knights, 2015).

Anticipatory grieving

The diagnosis of cancer and a potentially life-threatening illness causes actual or perceived losses, such as loss of function, independence, normal appearance, friends, self-esteem and self. Grieving is the emotional response to those losses. The adaptive process of mourning a loss and resolving grief is called grief work; grief work cannot begin until a loss is acknowledged. See Chapter 4 for a detailed discussion of grief and loss.

- Discuss roles of the person and family and ways in which they managed stressful situations in the past. Assess coping strategies and their effectiveness. Help identify sources of strength and support. Discuss changing roles resulting from leukaemia diagnosis and its effect on spiritual, social and economic status and usual lifestyle. Evaluate cultural or ethnic factors that affect grief reactions. *Grieving is a normal response to a real or potential loss that begins at the time of diagnosis. The timing, duration and intensity of grief and responses to grief may differ among family members. Share information on diagnosis, role change and physical loss among all family members to build the foundation for mutual understanding and trust.*
- Use therapeutic communication skills to facilitate open discussion of losses and provide permission to grieve. *Encouraging discussion of the meaning of the loss helps decrease some of the anxiety associated with loss. This in turn allows the person and their family to examine the current situation and compare it with past situations that they have coped with successfully.*
- Provide information about agencies that may help in resolving grief and make referrals as indicated. Consider self-help groups, cancer support groups and bereavement groups. *Participating in support groups with others who are anticipating or experiencing a similar loss can decrease feelings of isolation.*

Community-based care

Person and family teaching for home care after treatment for leukaemia focuses on encouraging self-care, providing information about the disease and the treatment, preventing infection and injury, and promoting nutrition. Teaching topics for each of these areas are as follows.

Encouraging self-care

- Hygiene measures and energy conservation during self-care activities.
- Oral hygiene, including using a soft-bristle toothbrush several times daily; avoiding flossing.
- Reporting lesions, bleeding or signs of infection promptly.
- Maintaining a balance of rest and activity.

Information about leukaemia and treatment

- Bone marrow function, the pathophysiology of leukaemia and potential complications of leukaemia.
- Prognosis for the specific type of leukaemia.
- Treatment measures such as chemotherapy, radiation, bone marrow or stem cell transplant, their purpose and effects, where treatment is available, and potential adverse effects or risks.
- Community, regional and national resources for people with leukaemia.

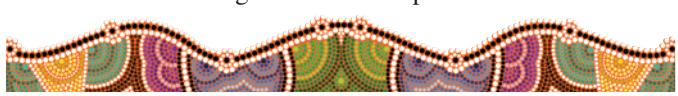
Preventing infection and injury

- Handwashing and other measures to reduce exposure to pathogens, such as avoiding people who are ill and avoiding crowds.
- Avoiding food-borne illnesses by washing fruits and vegetables, proper food storage.
- Dental hygiene measures.
- Avoiding immunisations.
- Manifestations to report: fever, chills, burning on urination, foul-smelling urine, vaginal or rectal discharge, skin lesions.
- Avoiding contact sports or strenuous exercise if platelet count is low.
- Using an electric razor for shaving, avoiding rectal or vaginal suppositories, vaginal tampons or enemas.
- Increasing dietary fibre and using a bulk-forming laxative as needed to prevent straining.
- Avoiding over-the-counter or prescription drugs that interfere with platelet function (see Box 32.6).
- The importance of reporting any bleeding (nosebleeds, rectal bleeding, vomiting blood, excessive menstrual periods, blood in the urine, bleeding gums, bruises or collections of blood under the skin) or changes in behaviour to the healthcare provider.

Promoting nutrition

- Eating several small, low-fat, high-kilojoule meals and drinking five to eight glasses of water daily; ensure culturally appropriate food choices available.
- Reporting continued weight loss, loss of appetite or inability to eat for 24 hours.
- Discussing dietary needs with the dietitian.

Assistance with physical care, finances and transportation may be required following discharge, especially for people who live in rural and remote locations. Refer the person and family to social services, support groups, home-care services as needed and other agencies that can provide needed services.



THE PERSON WITH MALIGNANT LYMPHOMA

Lymphomas are malignancies of lymphoid tissue. They are characterised by the proliferation of lymphocytes, histiocytes (resident monocytes or macrophages) and their precursors or derivatives. Lymphomas are closely related to lymphocytic leukaemias. Some experts consider them to be different forms or stages of the same disease processes.

BOX 32.6 Medications that may interfere with platelet function

Over-the-counter medications

- Aspirin and salicylates, including:
  - Ecotrin
  - Aspalgin
  - Astrix
  - Cartier
  - Cardiprin
- NSAIDs such as:
  - Naproxen
  - Ibuprofen

Prescription medications

- Aspirin-containing analgesics
- Chemotherapy drugs
- Antibiotics such as penicillin
- Carbamazepine (Tegretol)
- Colchicine
- Dipyridamole (Persantin)
- Gold salts
- Heparin
- Quinine derivatives
- Sulfonamides
- Thiazide diuretics

Although there are many types of malignant lymphoid cells, at this time lymphomas commonly are identified as Hodgkin's disease or non-Hodgkin's lymphoma.

Incidence and risk factors

In Australia, over 5400 people are diagnosed with lymphoma each year; lymphoma is the sixth most common cancer for both men and women (AIHW, 2014). The incidence of non-Hodgkin's lymphoma has nearly doubled since 1970, but currently has stabilised, primarily due to a fall in its incidence related to HIV infection and AIDS. The incidence of Hodgkin's disease has significantly declined since 1990 (Leukaemia Foundation, 2014). See Figure 32.10 for Australian lymphoma incidence and mortality statistics.

While the cause of lymphoma is unknown, some risk factors have been identified. Immunosuppression due to drug therapy following organ transplant or to HIV disease increases the risk of non-Hodgkin's lymphoma. Infectious agents such as HTLV-1 and Epstein–Barr virus (EBV) also have been identified as risk factors. Others may include occupational herbicide or chemical exposure (Leukaemia Foundation, 2014).

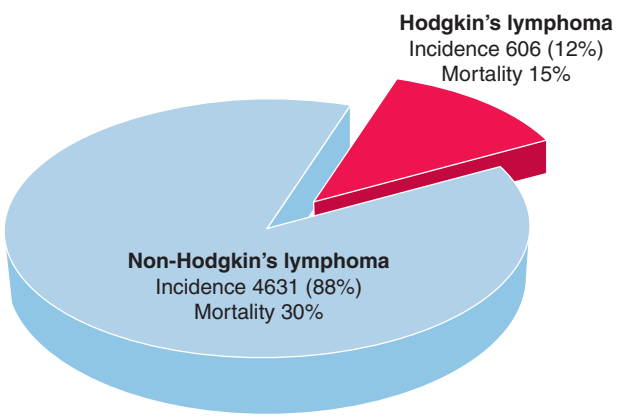


FIGURE 32.10 ■ Australian incidence and mortality data for the lymphomas, 2011

Source: Generated using data from the Australian Cancer Incidence and Mortality (ACIM) books (2015e, f).

Pathophysiology

Hodgkin's disease

Hodgkin's disease is a rare lymphatic cancer, occurring most often in people between the ages of 15 and 35. Approximately 400 new cases of Hodgkin's disease are diagnosed each year in Australia (Leukaemia Foundation, 2014). The exact cause of Hodgkin's disease is unknown, but both EBV infection and genetic factors appear to play a role in its development. Hodgkin's disease is one of the most curable cancers and most people with Hodgkin's lymphoma can be cured. While as many as 60–90% of people with localised disease achieve cure with a normal lifespan, there may be an increased risk of a second cancer occurrence throughout life (Leahy, 2008).

Hodgkin's disease develops in a single lymph node or chain of nodes, spreading to adjoining nodes. Involved lymph nodes contain *Reed–Sternberg cells* (malignant cells) surrounded by host inflammatory cells. These malignant cells secrete inflammatory mediator substances, attracting inflammatory cells to the tumour site. They may invade almost any tissue in the body. The spleen often is involved; as the disease progresses, the liver, lungs, digestive tract and CNS may be affected (Huether & McCance, 2013). Rapid proliferation of abnormal lymphocytes impairs the immune response, especially cell-mediated immune responses. Infections are common.

Hodgkin's disease is classified as classic Hodgkin's disease or as nodular-lymphocyte-predominant Hodgkin's disease. The classic form of the disease accounts for 95% of all cases; nodular-lymphocyte-predominant Hodgkin's is rare. Classic Hodgkin's can be further divided into four subtypes by cells identified within the tumour, but the subtype does not affect the prognosis.

MANIFESTATIONS The most common symptom of Hodgkin's disease is one or more painlessly enlarged lymph nodes, usually in the cervical or subclavicular region. Systemic manifestations such as persistent fever, night sweats, fatigue and weight loss are associated with a poorer prognosis for the disease. Late symptoms such as malaise, pruritus and anaemia indicate spread of the disease (Leukaemia Foundation, 2014). The spleen may be enlarged and other organ systems such as

the lungs and gastrointestinal tract are occasionally involved, causing difficulty breathing or bloating symptoms.

Non-Hodgkin's lymphoma

Non-Hodgkin's lymphoma is a diverse group of lymphoid tissue malignancies that do not contain Reed–Sternberg cells. **Non-Hodgkin's lymphomas** tend to arise in peripheral lymph nodes and spread early to tissues throughout the body. Non-Hodgkin's lymphoma is more common than Hodgkin's disease, affecting an estimated 4550 people in Australia annually, as opposed to around 600 diagnosed with Hodgkin lymphoma types (Leukaemia Foundation, 2014). Older adults are more often affected and it occurs more frequently in men than in women. Like Hodgkin's disease its cause is unknown, although both genetic and environmental factors (e.g. viral infections such as EBV, human T-cell leukaemia/lymphoma virus-1 or -2 and HIV) are thought to play a role.

As in most malignancies, non-Hodgkin's lymphoma begins as a single transformed cell; it may arise from T cells, B cells or tissue macrophages (histocytes). The primary types of non-Hodgkin's lymphomas are identified in Table 32.7. Although non-Hodgkin's lymphoma usually arises in a lymph node, it can originate in any lymphoid tissue. It tends to spread early and unpredictably to other lymphoid tissues and organs. Extranodal spread may involve the nasopharynx, GI tract, bone, CNS, thyroid, testes and soft tissue.

The prognosis for non-Hodgkin's lymphoma ranges from excellent to poor, depending on the identified cell type and grade of differentiation. Low-grade tumours (better differentiated) tend to be less aggressive and more curable. Higher-grade tumours often are disseminated at the time of diagnoses and have a poorer prognosis.

MANIFESTATIONS The early manifestations of non-Hodgkin's lymphoma are similar to those for Hodgkin's disease. Painless lymphadenopathy may be localised or widespread (see Figure 32.11). Systemic manifestations such as fever, night sweats, fatigue and weight loss may be present, but are less common in non-Hodgkin's lymphoma. Organ system involvement may cause symptoms such as abdominal pain, nausea and vomiting. Headaches, peripheral or cranial nerve symptoms, altered mental status or seizures may signal CNS involvement.

The manifestations and clinical features of Hodgkin's disease and non-Hodgkin's lymphoma are compared in Table 32.8.

Course

In both Hodgkin's disease and non-Hodgkin's lymphoma, the stage of the disease, the presence of systemic manifestations and factors such as age help determine the prognosis. The prognosis is good when the disease is localised to one or two node regions. Factors such as anaemia, thrombocytopenia and older age reduce the likelihood of disease cure.

TABLE 32.7 Subtypes of non-Hodgkin's lymphoma

SUBTYPE	INCIDENCE	COURSE AND PROGNOSIS
B-cell lymphomas		
Diffuse large B-cell lymphomas	Most common adult type (40–50% of adult lymphomas) More common in males Incidence increases with ageing	Aggressive tumour 45–50% cure rate
Follicular lymphoma	Accounts for 40% of adult lymphomas, rare in children Incidence increases with ageing	Bone marrow frequently involved, course slow, indolent 72% 5-year survival
Extranodal marginal zone lymphoma (MALT lymphoma)	Accounts for about 5% of adult lymphomas, rare in children Incidence increases with ageing More common in Italy	Presents with tumours outside lymphatic system: GI tract, lung, thyroid, urinary tract, skin, CNS. Slow, indolent course 74% 5-year survival
Mantle cell lymphoma	Accounts for 3–4% of adult lymphomas, rare in children Predominantly affects older men (74%)	Aggressive, difficult to cure 27% 5-year survival
Burkitt lymphoma	Rare in adults (< 1% of lymphomas), more common in children (~30% NHL)	Rapidly progressive but responds well to therapy 45% 5-year survival
T-cell lymphomas		
Precursor T-cell lymphoblastic leukaemia/lymphoma	More common in children and young adults More common in males than females	Can present either as ALL or lymphoma Aggressive disease 26% 5-year survival
Peripheral T-cell lymphoma	Most common T-cell lymphoma in adults	Often presents as disseminated disease 25% 5-year survival
Mycosis fungoides/cutaneous T-cell lymphoma	Onset typically during mid-fifties; more common in African Americans	Cutaneous lymphoma Slow course, progressing from patchy skin lesions to plaque to cutaneous tumours

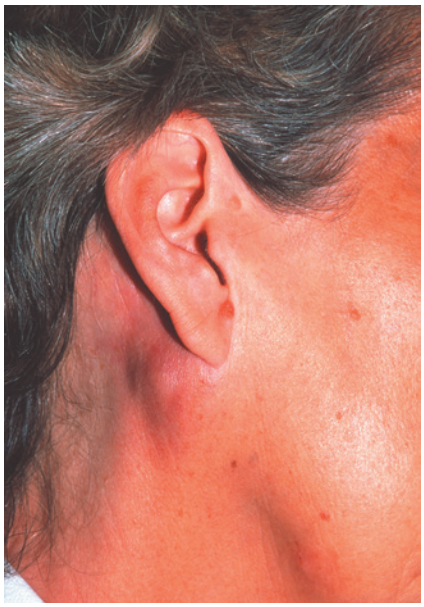


FIGURE 32.11 ■ Cervical lymphadenopathy in a person with lymphoma of the neck

Source: Dr P. Marazzi/Science Source.

INTERPROFESSIONAL CARE

Chemotherapy and radiation therapy, either alone or in combination, are the primary treatments for Hodgkin's and non-Hodgkin's lymphomas. Use of monoclonal antibodies to target lymphoma cells and bone marrow and peripheral stem cell transplants are under investigation for treating lymphomas as well. See the previous section on treatment of leukaemia for more information about these transplants.

Diagnosis

The following diagnostic tests may be ordered for lymphomas:

- *FBC* often shows a mild normochromic, normocytic anaemia in Hodgkin's disease; other findings in Hodgkin's disease may include leucocytosis with high neutrophil and eosinophil counts and an elevated erythrocyte sedimentation rate (ESR). In non-Hodgkin's lymphoma, the FBC typically remains normal until late in the disease, when pancytopenia may develop.
- An *ESR* is done to identify possible inflammatory causes of lymph node enlargement.
- *Blood studies* of major organ function (including liver function tests and renal function studies) are performed to identify possible organ involvement. Serum LDH levels and protein electrophoresis also may be done when Hodgkin's disease is suspected.
- *Chest x-ray* is done to identify possible enlarged mediastinal lymph nodes and pulmonary involvement.
- *CT scans* of the chest, abdomen and pelvis are performed to identify abnormal or enlarged nodes.
- *Positron emission tomography (PET* or *gallium scans)* may be performed in diagnosing the disease, as well as to evaluate the effectiveness of treatment.
- *Biopsy* of the largest, most central enlarged lymph node and of the bone marrow is done to establish the diagnosis for both Hodgkin's disease and non-Hodgkin's lymphoma. The presence of Reed–Sternberg cells confirms the diagnosis of Hodgkin's disease.

Staging

Staging is used to determine the extent of the disease and appropriate treatment (Leukaemia Foundation, 2011). The staging system used in Australia is the Ann Arbor Staging System (AIHW, 2011), which assesses the extent and severity of lymphomas. The stages are:

- Stage I: involvement of a single lymph node region or lymphoid structure (e.g. spleen, thymus, lymphoid tonsillar tissue).
- Stage II: involvement of two or more lymph node regions on the same side of the diaphragm.
- Stage III: involvement of lymph node regions or structures on both sides of the diaphragm.
  - III_1: limited to upper abdomen (spleen, splenic, coeliac or portal nodes).
  - III_2: involvement of lower abdominal nodes (para-aortic, iliac or mesenteric).
- Stage IV: involvement of an extranodal site (not proximal or contiguous with an involved node) such as the liver, lung or pleura, bone or bone marrow, or skin.

TABLE 32.8 Features and manifestations of Hodgkin's disease and non-Hodgkin's lymphoma

FEATURE OR MANIFESTATION	HODGKIN'S DISEASE	NON-HODGKIN'S LYMPHOMA
Lymphadenopathy	Localised to a single node or chain, often cervical, subclavicular or mediastinal	Multiple peripheral nodes, nodes of the mesentery often involved
Spread	Orderly and continuous	Diffuse and unpredictable
Extranodal involvement	Rare	Early and common
Bone marrow involvement	Uncommon	Common
Fever, night sweats, weight loss	Common	Uncommon until disease is extensive
Other manifestations	Fatigue, pruritus, splenomegaly; anaemia, neutrophilia	Abdominal pain, nausea, vomiting; dyspnoea, cough; CNS symptoms; lymphocytopenia

The presence or absence of systemic symptoms is indicated by either an 'A' (no systemic symptoms) or 'B' (systemic symptoms of fever, night sweats, weight loss), or 'E' is used when lymphoma has spread to an area or organ outside the lymph nodes. The letter is always placed after the stage; for example, 2A or 4E (Leukaemia Foundation, 2011).

Chemotherapy

Combination chemotherapy is used to treat both Hodgkin's disease and non-Hodgkin's lymphoma. In both cases, chemotherapy often is followed by radiation therapy to involved lymph node regions. The choice of drug combination depends on the stage of the disease as well as the person's age and general condition. Combination regimens used may include CHOP (cyclophosphamide, doxorubicin, vincristine and prednisone); ABVD (doxorubicin, bleomycin, vinblastine and dacarbazine); MOPP (nitrogen mustard, vincristine, procarbazine and prednisone); and ChlVPP (chlorambucil, vinblastine, procarbazine and prednisone). These regimens also may be combined in alternating months to reduce the adverse effects and improve tumour cell kill. More than 75% of people with Hodgkin's disease who do not have systemic symptoms achieve complete remission with treatment. The prognosis for those with non-Hodgkin's lymphoma varies by the type and stage of the disease. For more information about nursing care of the person receiving combination chemotherapy, see Chapter 13.

Radiation therapy

Radiation therapy may be the primary treatment for early-stage Hodgkin's disease, although early chemotherapy is becoming more common. In later stages and in non-Hodgkin's lymphoma it is usually combined with chemotherapy. Many lymphomas are highly responsive to radiation. The involved lymph node region is treated, with careful shielding to protect unaffected areas and minimise the extent of radiation burn and normal cell destruction (see Figure 32.12). If the disease is advanced, total nodal irradiation may be done. See Chapter 13 for nursing care of the person receiving radiation therapy.

Stem cell transplant

Autologous peripheral blood stem cell transplant (PBSCT) is a treatment option for people who experience remission of malignant lymphoma. Autologous PBSCT uses the person's own stem cells to restore bone marrow function after chemotherapy or radiation. In autologous PBSCT, stem cells are obtained from peripheral blood following chemotherapy and treatment with colony-stimulating factors to promote development of normal blood cells. The blood containing these normal stem cells is then frozen and stored for use after treatment. If relapse occurs, lethal doses of chemotherapy or radiation are given to destroy the immune system and malignant cells. The frozen blood is then thawed and infused intravenously through a peripheral line. The infused stem cells become a part of the person's bone marrow and normal haematopoiesis takes place.

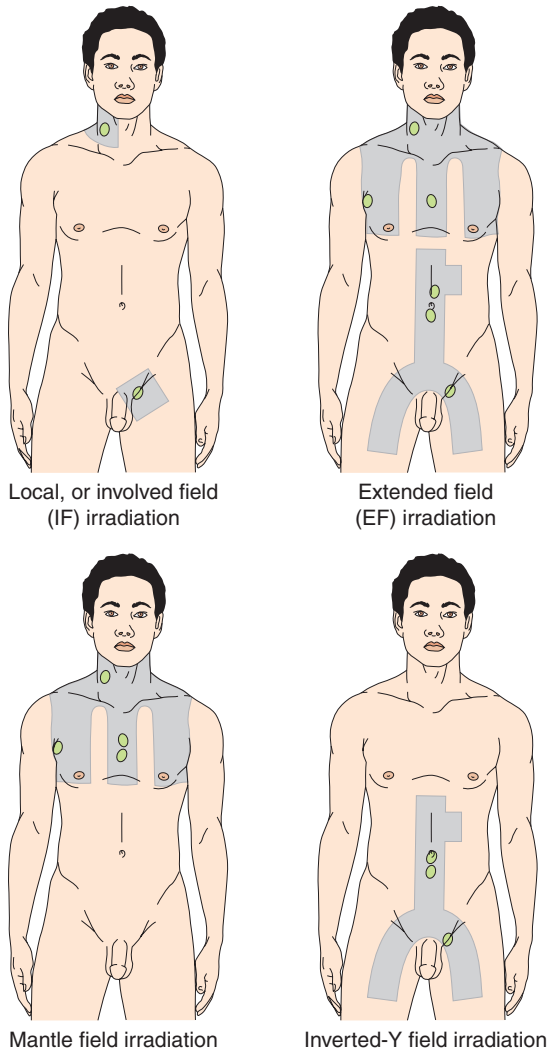


FIGURE 32.12 ■ Patterns of radiation therapy used to treat lymphoma based on the location and extent of the disease

The person is critically ill during the period of bone marrow destruction and immunosuppression, and is hospitalised in a private room for 6 to 8 weeks or more. See the accompanying 'Translation to practice' box for discussion of fatigue related to autologous PBSCT.

Complications of treatment

Both chemotherapy and radiation therapy may have long-term effects. Permanent sterility is common, especially in older adults. Bone marrow depression can lead to immunosuppression, anaemia and bleeding. Secondary cancers and cardiac injury are the most serious late adverse effects of treatment. Chemotherapy regimens using the MOPP or a related protocol carry a risk of acute leukaemia. Cancers such as breast or lung cancer may develop 10 or more years after thoracic radiation. Thoracic radiation also increases the risk of coronary heart disease and hypothyroidism (Sausville & Longo, 2015).

TRANSLATION TO PRACTICE Evidence-based practice: risk factors for depression and fatigue among hematopoietic cell transplant (HCT) recipients

Fatigue and depression are common adverse effects of chemotherapy and radiation therapy in people undergoing cancer treatment. Fatigue is prevalent, affecting 80–100% of people undergoing standard chemotherapy and often leading to lost work time and difficulty maintaining functional roles within the family and society. A study by Jim et al. (2015) found that depression and fatigue levels were closely linked, with increasing fatigue severity leading to greater levels of depression. The cohort most susceptible to ongoing fatigue and depression were female, younger in age, had chronic pain issues and had received peripheral blood stem cells. There were high rates of depression and fatigue, with 13% reporting depression classed as moderated to severe, and 42% reporting fatigue that was moderate to severe.

IMPLICATIONS FOR NURSING

This study suggests that nurses should assess for fatigue and depression in people following stem cell transplant (SCT). Nursing measures to help conserve the person's energy are appropriate to manage fatigue. Early screening, referral and intervention for fatigue may actually reduce the intensity of fatigue at its peak. Nurses can use this information to prepare people for common symptom patterns following SCT, thus reducing anxiety and concern that their condition may be declining rather than improving after SCT. Families and caregivers are also at risk of depression and psychological distress and should be screened accordingly. Assessment tools to measure fatigue and depression in people undergoing SCT can be incorporated into nursing care. Majhail et al. (2012) recommend screening 6–12 months post transplantation, and then annually thereafter.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. The average age of participants (N = 1869) in this study was 56 years; 53% were male, 47% female. Does this sample reflect your local demographic of people undergoing haematopoietic cell transplant? How can information about the sample be used to guide application of study findings to the general population of people undergoing this treatment? To older adults? To adolescents and young adults?
2. In this study, participants who were female, younger, and had chronic pain or secondary malignancy were at greater risk of depression and fatigue. How would understanding of this result improve your care for individuals who have received haematopoietic stem cell transplant?
3. Interestingly, myeloablative regimen, total body irradiation dose and cytomegalovirus status was not predictive of depression or fatigue. What is the significance of this finding?

Nursing care

Assessment and priority nursing care for the person with lymphoma follows. See also the nursing care plan later in this section for application of nursing care strategies for a person with Hodgkin's disease.

Assessment

Focused assessment of the person with Hodgkin's disease or non-Hodgkin's lymphoma includes:

- *Health history:* complaints of enlarged lymph node(s), fever, night sweats, weight loss, fatigue or general malaise, abdominal pain, respiratory symptoms, numbness or tingling of extremities, visual changes or changes in mentation; history of infectious mononucleosis, HIV disease or other immunosuppressive disorders.
- *Physical examination:* mental status exam; inspect and palpate lymph nodes (cervical, subclavicular, axillary and inguinal) for enlargement, tenderness; heart and lung sounds; abdominal examination for tenderness, masses, liver or spleen enlargement.
- *Diagnostic tests:* FBC, haemoglobin and haematocrit, ESR; serum blood results; x-ray, scan and biopsy results.

Nursing diagnoses and interventions

Nursing care of the person with malignant lymphoma involves both physical and emotional support during diagnosis and treatment. Common nursing care problems include impaired protection due to bone marrow suppression, fatigue, nausea and altered body image. See the nursing care section for leukaemia, above, for specific nursing interventions for *Ineffective protection*.

Fatigue

General malaise and fatigue may accompany malignant lymphoma and are side effects of chemotherapy. In addition, the physical and psychological stress of dealing with a chronic, debilitating disease and its treatment may cause fatigue.

- Inquire about feelings of malaise (a vague feeling of body weakness or discomfort) and fatigue (a pervasive, drained feeling that cannot be eliminated). *Both malaise and fatigue are subjective experiences with physiological, situational and psychological components.*
- Encourage verbalisation of feelings about the impact of the disease and fatigue on lifestyle. *Discussion of feelings helps the person clarify values and may assist in identifying priorities.*
- Encourage enjoyable but quiet activities, such as reading, listening to music or hobbies. Enjoyable activities help decrease feelings of fatigue. *Quiet activities conserve energy while yielding a sense of accomplishment.*

- Encourage establishing priorities and including rest periods or naps when scheduling daily activities. *This provides a sense of control over activities and helps maintain self-esteem. Scheduled rest periods help restore energy and decrease fatigue.*
- Encourage delegation of some responsibilities to family members. *Delegation helps maintain the person's involvement and role in family decisions and responsibilities, while conserving energy for those activities identified as high priority by the person.*
- Identify and encourage the person to use energy-saving equipment. *Performing tasks with less exertion and in less time helps conserve energy.*
- Encourage a diet high in carbohydrates and fluids. *A high-carbohydrate diet helps maintain muscle glycogen stores. A liberal fluid intake promotes excretion of metabolic by-products that may contribute to malaise and fatigue.*

Nausea

The effects of malignant lymphoma and its treatment with chemotherapy and/or radiation therapy can contribute to nausea and interfere with nutritional status. Nausea, a sensation of abdominal fullness and fear of vomiting often limit food intake. See also the nursing diagnosis of *Imbalanced nutrition* in the section on leukaemia, above, for additional interventions.

- Assess precipitating factors for nausea and/or vomiting, the frequency of vomiting and relief measures used by the person. *Careful assessment allows development of interventions tailored to the person's situation and needs.*

CONSIDERATION FOR PRACTICE

Provide ordered anti-emetics before chemotherapy is started. Administering prescribed anti-emetics before chemotherapy helps prevent nausea and the psychological association of nausea with chemotherapy.

- Teach measures to prevent or relieve nausea and vomiting:
  a. Eat cracker biscuits and suck on hard lollies.
  b. Eat soft, bland foods that are cold or at room temperature.
  c. Avoid unpleasant odours and get fresh air.
  d. Eat prior to but not immediately before chemotherapy.
  e. Use distraction or progressive muscle relaxation when nauseated.
  f. If vomiting occurs, gradually resume oral intake with frequent sips of clear liquids or ice, progressing to bland foods.

  Cracker biscuits and lollies often relieve queasiness, whereas hot, spicy, sweet or strong-smelling foods may increase nausea. Alternative nausea relief measures may be effective.
- Provide small feedings of high-kilojoule, high-protein foods and fluids. *This increases nutritional intake.*
- Assist with oral care, general hygiene and environmental control of temperature, appearance and odours. *These measures enhance appetite.*
- Identify and provide preferred foods. *This promotes nutritional intake.*
- Assist to a sitting position during and immediately after meals. *The sitting position helps decrease early feelings of fullness.*

Disturbed body image

The diagnosis of cancer is often devastating to the sense of trust in and the perception of one's body. Radiation and chemotherapy lead to changes in appearance and body function (e.g. hair loss, reduced libido and infertility), further altering body image. Reactions to this diagnosis vary and may include refusal to look in a mirror or discuss the effects of the disease or treatment, unwillingness to participate in rehabilitation, inappropriate treatment decisions, increasing dependence on others or refusal to provide self-care, hostility, withdrawal and signs of grieving.

- Assess perception of body image through subjective information such as:
  a. what the person likes most and least about their body
  b. pre-illness perception of people who are sick or have a disability
  c. current understanding of health and limitations imposed by illness or treatment
  d. feelings about the illness and its effect on perception of self and others.

  Body image is one's mental idea or picture of the body. It is based on past and present experiences and includes components of one's actual body and emotional responses to that body. Body image changes constantly. There is often a time lag between an actual body change and the changed body image; during this time, the diagnosis, teaching and treatment may be rejected.
- Discuss the risk of and measures to cope with alopecia. Suggest wearing wigs, scarves, hats or caps. Teach proper scalp care using baby shampoo or mild soap, a soft brush, sunscreen and mineral oil to reduce itching. If eyelashes and eyebrows are lost, teach eye protection, such as wearing eyeglasses and caps with wide brims. *Chemotherapeutic agents attack rapidly dividing cells such as those responsible for hair growth. Hair loss usually begins 1 to 2 weeks after initiation of chemotherapy, with maximum loss 1 to 2 months later. Alopecia may range from thinning to total hair loss. Regrowth depends on the treatment schedule and doses; however, it usually begins 2 to 3 months after treatment ends. New hair may be softer, more curly and slightly different in colour. Teaching and emotional support help the person anticipate hair loss, discuss its potential effect on body image and learn self-care techniques.*
- Discuss available resources for financial assistance with purchase of wigs, including assistance from the Australian Cancer Council. *A well-matched wig (or one the colour the person has always wished for!) can help maintain a positive body image.*

Sexual dysfunction

Sexual dysfunction may result from the malignancy and the effects of radiation and chemotherapy. Reproductive tissues are made of rapidly dividing cells and cancer treatment may cause

NURSING CARE PLAN A person with Hodgkin's disease

Max Patterson, RN, aged 28, is the nurse manager of a thoracic critical care unit in a large teaching hospital. Lately he has been more tired than usual, often wakes up at night covered with sweat and just does not feel well. He had thought that his symptoms were due to a viral illness and his busy work schedule. However, yesterday morning Max noticed a large swollen area on the right side of his neck. He made an appointment with his general practitioner who found a large cervical lymph node. A biopsy of the node and a CT scan of the chest were scheduled.

ASSESSMENT

David Smart, RN, the practice nurse at the medical centre, obtains a nursing history and assessment of Mr Patterson. His physical examination is essentially normal, with the exception of the enlarged node, which is not tender to palpation. When Mr Patterson is weighed, he tells RN Smart that he has lost 3.2 kg in the past 2 months. In reviewing the results of the blood studies, RN Smart notes mild anaemia and an increased neutrophil count. The lymph node biopsy shows Reed–Sternberg cells. The doctor and RN Smart tell Mr Patterson that the findings indicate stage 1B Hodgkin's disease but that the prognosis is very good. The doctor recommends a short course of combination chemotherapy followed by radiation therapy to involved sites.

DIAGNOSES

- *Anxiety* related to the diagnosis of Hodgkin's disease and effects of treatment on job performance.
- *Risk of infection* related to potential bone marrow depression due to chemotherapy.
- *Fatigue* related to effects of cancer, chemotherapy and radiation therapy.

PLANNING

- Reduce anxiety through discussion and education.
- Improve and extend energy levels.
- Monitor and reduce risk of infection.

Expected outcomes

- Verbalise reduced anxiety.
- Remain free of infection.
- Identify and use methods to preserve energy.

IMPLEMENTATION

- Encourage person to consider a leave of absence from work during course of treatment.
- Discuss joining a support group for people with cancer.
- Provide information about the illness, combination chemotherapy and radiation therapy.
- Reinforce knowledge of actions to decrease the risk of infection.
- Discuss ways to decrease fatigue and maintain energy:
- Take a 1- to 2-hour nap once or twice a day.
- Avoid overexertion during weekends and time off.
- Maintain a well-balanced diet.

EVALUATION

When Mr Patterson returns the following week to begin chemotherapy, he brings his friend Nina to meet RN Smart and asks him to discuss his treatment with her. Mr Patterson says, 'I am still really scared, but being able to talk about this with Nina will help a lot.' Mr Patterson has made arrangements to take 4 months' leave from work, with the understanding that his job will be held for him. He states that he will have some problems with money but is working them out. He also says he feels that taking a daytime sleep is silly but that he will rest to maintain his energy level. Mr Patterson and Nina express confidence that he will be cured and say they plan to be active members of the cancer support group—even after recovery.

CRITICAL THINKING IN THE NURSING PROCESS

1. Discuss the rationale for treating Hodgkin's disease with chemotherapy and radiation.
2. Design a teaching plan to help Mr Patterson prevent infection while he is at home.
3. What effect does the diagnosis of cancer have on the developmental tasks of a young adult?
4. Develop a care plan for Mr Patterson for the nursing diagnosis of *Ineffective role performance*.

REFLECTION ON THE NURSING PROCESS

1. What are the take-home points that you have learned within this case study?
2. How can you use them within your daily practice?
3. Where would you find good-quality information for a person in Mr Patterson's situation?

temporary or permanent sterility, changes in menstruation and changes in libido.

- Encourage discussion of actual or potential sexual dysfunction or sterility with the person and significant other. *People may be reluctant to discuss this unintended effect of treatment unless encouraged.*
- Assess knowledge, provide information and clarify misconceptions. Discuss realistic measures for coping (e.g. sperm banking prior to chemotherapy or radiation therapy). *People and their partners may be unclear about expected effects on sexuality, reproduction and the permanency of these effects.*
- Refer for counselling as indicated. *Sexual counselling can help the person and partner develop alternative strategies for expressing their sexuality.*

Risk of impaired skin integrity

Malignant lymphomas may cause significant pruritus and drenching night sweats. As a result, skin integrity may be impaired. In addition, radiation therapy can cause superficial burns, which also may affect skin integrity.

- Frequently assess skin, especially in areas undergoing radiation. *Early identification of lesions allows timely*

treatment and can prevent further disruption of this important line of defence against infection.

- Provide and teach measures to promote comfort and relieve itching: use cool water and a mild soap to bathe; blot (rather than rub) dry skin; apply plain cornflour or non-perfumed lotion or powder to the skin unless contraindicated, being careful not to clog pores; use lightweight blankets and clothing; maintain adequate humidity and a cool room temperature; wash bedding and clothes in mild detergent and put them through a second rinse cycle. *Pruritus is aggravated by excessive warmth, excessive dryness, rough fabrics, fatigue and stress. Lotions and some powders may be contraindicated during radiation therapy.*

Community-based care

When teaching the person and family for home care, include the following topics in addition to those previously identified for specific nursing diagnoses:

- information about the illness, planned treatment and anticipated side effects of treatment
- skin care and measures to relieve itching and protect areas of radiation
- symptoms to report to the doctor, including those of vertebral compression (decreased sensation or strength in lower extremities)
- use of analgesics and alternative relief strategies for abdominal pain and peripheral neuropathies
- respiratory care if mediastinal nodes are enlarged or lungs or pleurae are involved
- planning activities of daily living to ensure adequate rest and exercise
- measures to relieve nausea and maintain adequate nutrition.

Refer the person and their family members to the local oncology clinic, cancer support group or Cancer Council for information, assistance and counselling. A list of state and local agencies that offer information about malignant lymphoma and financial assistance can be obtained from the Leukaemia Foundation: www.leukaemia.org.au.

THE PERSON WITH MULTIPLE MYELOMA

Multiple myeloma is a malignancy in which plasma cells multiply uncontrollably and infiltrate the bone marrow, lymph nodes, spleen and other tissues. *Plasma cells* are B-cell lymphocytes that develop to produce antibodies (immunoglobins).

Incidence and risk factors

The incidence of multiple myeloma is increasing slightly, with an estimated 1500 people diagnosed each year in Australia (Leukaemia Foundation, 2014). The incidence of multiple myeloma increases with age, rarely occurring before age 40, and 80% of new cases occur over the age of 60, more frequently in men than in women (Huether & McCance, 2013). Its cause is unknown. Possible contributing factors include genetic alterations, radiation exposure, oncogenic virus, inflammatory stimuli and chronic antigenic stimulation. The risk of developing multiple myeloma is higher in people of lower socioeconomic status. This increased risk may relate to environmental factors such as poor housing, occupational hazards, poor nutritional status and other physical and psychosocial stressors such as exposure to infectious agents.

Pathophysiology

Malignant plasma cells arise from one clone (*monoclonal*) of B cells that produce abnormally large amounts of a particular immunoglobin called the *M protein*. This abnormal protein interferes with normal antibody production and impairs the humoral immune response. It also increases blood viscosity and may damage kidney tubules. As myeloma cells proliferate, they replace the bone marrow and infiltrate the bone itself. Cortical bone is progressively destroyed by tumour growth and enzymes produced by myeloma cells. These enzymes facilitate bone destruction, its infiltration by tumour cells, development of new blood vessels to sustain the tumour and growth of myeloma cells (Huether & McCance, 2013). Affected bones (primarily the vertebrae, ribs, skull, pelvis, femur, clavicle and scapula) are weakened and may break without trauma (*pathological fracture*). With disease progression, malignant cells spread via the bloodstream to invade other organs (see Figure 32.13).

Manifestations

The disease develops slowly, with many people diagnosed during evaluation for unrelated problems. Manifestations of multiple myeloma are due to its effects on the bone and the impaired immune response due to M-protein production. The most common presenting problem is bone pain that is usually felt in the back or ribs and is increased with movement (Leukaemia Foundation, 2014). With progression of the disease, the pain may increase in severity and become more localised. Rapid bone destruction releases calcium from the bone, leading to hypercalcaemia and manifestations of neurological dysfunction, such as lethargy, confusion and weakness.

As functional antibody formation decreases and the humoral immune response is suppressed, recurrent infections develop. Cell-mediated immunity remains intact. Bence Jones proteins are found in the urine in multiple myeloma. These proteins are toxic to the renal tubules and may lead to renal failure with azotaemia and uraemia. (See Chapter 27 for more information about renal failure.)

About 15% of people with multiple myeloma die within 3 months of the diagnosis. More frequently, the disease course is chronic, progressing more rapidly with each relapse after remission. The acute terminal stage of the disease is marked by pancytopenia and widespread organ infiltration by myeloma cells (Huether & McCance, 2013).

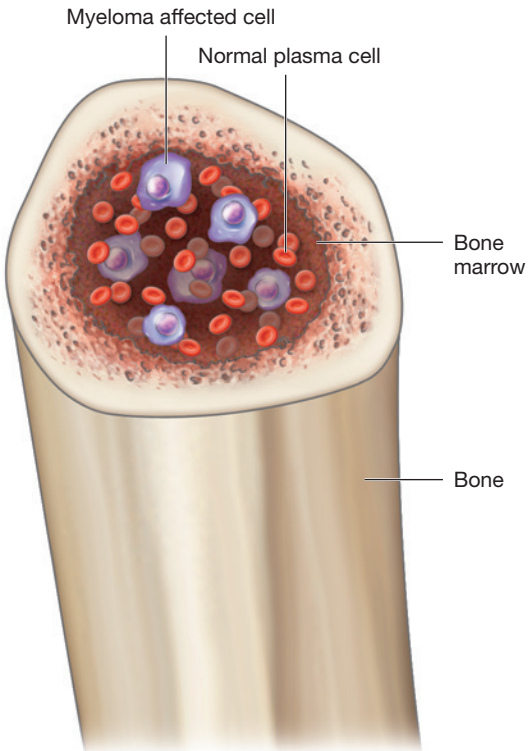


FIGURE 32.13 ■ An illustration of the progress of multiple myeloma in a male. Abnormal plasma cells proliferate uncontrollably, gradually replacing bone marrow and infiltrating bone itself. As the disease progresses, these cells spread to other organs via the bloodstream

INTERPROFESSIONAL CARE

Diagnosis and staging

Diagnostic tests for multiple myeloma include the following:

- *X-rays* and other radiological studies of the bone may reveal multiple punched-out lesions.
- *Bone marrow examination* shows an abnormal number of immature plasma cells.
- *FBC* shows moderate to severe anaemia and the *ESR* usually is elevated.
- *Protein electrophoresis* shows a spike of one type of antibody, usually immunoglobulin G (IgG).
- *Serum calcium, creatinine, uric acid* and *blood urea nitrogen (BUN)* levels often are elevated.
- *Urinalysis* shows Bence Jones protein in the urine.
- *Biopsy* of myeloma lesions confirms the diagnosis of multiple myeloma.

Staging of multiple myeloma is based on the haemoglobin and serum calcium levels, the amount of abnormal protein present and the degree of bone involvement.

Treatment

There is no cure for multiple myeloma. In some people, active observation is indicated, as the disease may continue with a slow, *indolent* (sluggish, not developing or progressing) course for many years (Leukemia Foundation, 2014). When indicated by disease stage or progression, standard treatment includes induction chemotherapy followed by stem cell transplant and maintenance chemotherapy to control progression of the disease. Supportive care is provided to reduce complications of the disease and their effects.

Combination chemotherapy with an alkylating agent melphalan (Alkeran), cyclophosphamide (Cytoxan) or chlorambucil (Chloromycetin)) and prednisone administered for 4 to 7 days every 4 to 6 weeks is commonly used. Chemotherapy typically reduces bone pain, hypercalcaemia, anaemia and the number of infections (Fauci et al., 2008). Novel therapies that include immunomodulatory drugs and proteasome inhibitors can contribute to improved survival rates and better response rates when compared to conventional chemotherapy. Localised radiation therapy may be used to treat painful bone lesions. High-dose chemotherapy followed by peripheral allogeneic stem cell transplant (SCT) may be more effective in achieving a cure, but is associated with a high mortality rate. When autologous SCT is used, granulocyte colony-stimulating factor is administered prior to harvesting and preserving peripheral stem cells for transplant.

Supportive care may include treatment of hypercalcaemia with hydration, possible bisphosphonate therapy to reduce bone loss (see Chapter 39) and calcium, vitamin D and fluoride supplements to support bone structure. Plasma exchange therapy (plasmapheresis) to remove circulating M proteins is used as needed to treat acute renal failure. Infections are treated promptly when they develop.

Nursing care

Assessment

Focused assessment data for the person with multiple myeloma include the following:

- *Health history:* complaints of back or bone pain, onset, duration and intensity; complaints of weakness, fatigue, anorexia; history of frequent or recurrent infections; neurological symptoms such as numbness and tingling or clumsiness.
- *Physical examination:* level of consciousness and mental status; mobility, gait; localised tenderness or pain, bony crepitus with movement or palpation; movement and sensation in extremities.

Nursing diagnoses and interventions

Nursing care of the person with multiple myeloma focuses on problems of chronic pain, impaired mobility and the risk of injury. Risk of infection is a major nursing care focus; see the previous section on leukaemia for specific interventions to reduce this risk. Other nursing care needs are similar to those

of people with other cancers and chronic pain. See Chapters 8 and 13 for additional specific nursing interventions for these problems.

Chronic pain

People with multiple myeloma typically experience chronic back pain and deep bone pain as myeloma cells saturate the bone marrow and invade the bone structure. Pathological fractures are a common and reoccurring problem.

- Assess pain, including intensity (use a standard pain scale), onset, duration, precipitating factors and effective relief measures. *Identifying the intensity, causes and precipitating factors of pain helps determine and evaluate effective pain relief measures.*
- Determine position of greatest comfort and assist as needed into this position. *The person is best able to identify positions that minimise pain, but may need assistance with repositioning.*
- Support position with pillows. *Bony prominences may be painful due to infiltrates. Pillows can help relieve pressure on these prominences, thus reducing pain.*
- Provide uninterrupted rest periods. *Adequate rest facilitates pain relief and improves pain tolerance.*
- Teach adjunctive pain relief strategies such as relaxation or guided imagery. *A combination of pharmacological and non-pharmacological methods provides better management of chronic pain, especially bone pain.*
- Teach effective analgesic use, including the family in instruction. *Analgesics are most effective when taken before pain becomes severe. People and their families may be reluctant to use prescription analgesics on a regular basis.*
- Report unrelieved pain to the doctor. *A different analgesic or addition of an adjunctive medication such as a non-steroidal anti-inflammatory drug (NSAID) may be needed to effectively control pain.*

Impaired physical mobility

Painful bony infiltrates and pathological fractures may limit mobility. A brace or splint may be used to protect extremities or support the back. In addition, persistent weakness associated with the cancer and anaemia may limit the person's ability to participate in usual activities.

- Assist to change position at least every 2 hours. *Assistance with repositioning is necessary due to weakness. Frequent repositioning improves comfort and reduces the risk of impaired skin and tissue integrity.*

CONSIDERATION FOR PRACTICE

Gently support extremities during repositioning. Weakened extremities due to infiltration of bone by myeloma cells and muscle atrophy from lack of use increase the risk of pathological fractures.

- Provide a trapeze to assist with repositioning. *A trapeze provides better leverage, allowing the person to assist with repositioning and providing a degree of independence. The ability to participate in self-care improves self-esteem.*

Risk of injury

The bone involvement of multiple myeloma places the person at high risk of pathological and traumatic fractures. Pathological fractures can occur with simple activities such as turning or reaching for an item. The spine usually is affected; the ribs and bones of the extremities also may be at risk of fracture.

- Place needed items close at hand. *Straining to reach objects increases the risk of falling or sustaining other injury.*
- Provide safety measures to prevent falls from bed: place the bed in a low position, use side rails as indicated and place the call bell within reach. *Safety measures help prevent accidental injury. A secure environment minimises risk and helps prevent falls.*
- Provide shoes with non-skid soles, a clear pathway, adequate lighting and a level surface free of scatter rugs or other hazards when ambulating. Provide a walker as needed for support and security. *Weight-bearing exercise promotes bone repair. Safety measures, such as an unobstructed pathway and a firm walking surface, help prevent falls.*

Community-based care

When teaching persons and their families for home care, include the following topics:

- strategies for home maintenance management
- signs and symptoms of complications to be reported to the doctor (e.g. symptoms of vertebral and extremity fractures)
- manifestations of infection to report: fever and chills; increased malaise, fatigue or weakness; cough with or without sputum; sore throat; dysuria, nocturia, frequency, urgency or malodourous urine.

Provide referrals for home health and home maintenance services, physical or occupational therapy, social services and hospice care as appropriate.

THE PERSON WITH NEUTROPENIA

Leucopenia is a decrease in the total circulating white blood cell count. Although any type of WBC may be affected, neutrophils, which make up the majority of WBCs, are affected most often. *Neutropenia* is a decrease in circulating neutrophils, usually less than 1×10^9/L to 1.5×10^9/L. Neutropenia may be either congenital or acquired, developing secondarily to prolonged infection, haematological disorders, starvation or autoimmune disorders (such as rheumatoid arthritis). Chemotherapy and other drugs can suppress the bone marrow. Neutropenia develops in approximately half of people undergoing chemotherapy to treat cancer. *Agranulocytosis* is severe neutropenia, with less than 0.5×10^9/L. Numbers of other granulocytes also are reduced. It is usually due to impaired leucocyte formation in the bone marrow or increased cell destruction in circulating blood. Agranulocytosis significantly increases the risk of infection. *Aplastic anaemia* affects production of all blood cells, resulting in anaemia, thrombocytopenia and agranulocytosis.

Pathophysiology and manifestations

Neutrophils are an integral component of the immune response. They are phagocytes, drawn to and activated by infection and inflammation to engulf and degrade invading microorganisms. Their lifespan in peripheral blood is short, less than 1 day. When *granulopoiesis* (the development and maturation of granulocytes) in the bone marrow is suppressed, the number of circulating neutrophils falls rapidly. As a result, the body's ability to defend itself against infection is significantly reduced.

The manifestations of neutropenia reflect the resulting impaired immunity and inflammatory response. Opportunistic bacterial, fungal and protozoal infections develop, commonly affecting the respiratory tract and mucosa of the mouth, GI tract and vagina. Malaise, chills and fever with extreme weakness and fatigue are common manifestations.

INTERPROFESSIONAL CARE

The diagnosis of neutropenia is made based on the person's manifestations, risk factors and the FBC. The total white blood count is low, 4×10^9/L.

Haematopoietic growth factors such as granulocyte-macrophage colony-stimulating factor are administered to stimulate granulocyte maturation and differentiation. Infections are treated with antibiotic therapy. Protective isolation procedures may be initiated to prevent exposure to pathogens. When neutropenia is related to chemotherapy, cancer treatment often must be halted, at least temporarily, to allow the bone marrow to recover.

Nursing care

The primary nursing care focus is early identification of neutropenia and protecting the person from infection. The WBC count is monitored on a regular basis and any decline reported to the doctor. Protective isolation may be indicated, including restricting the number of visitors and people with apparent illness. See *Risk of infection* in the earlier section on leukaemia for specific nursing interventions for the person with neutropenia.

PLATELET AND COAGULATION DISORDERS

Platelet and coagulation disorders affect **haemostasis**, control of bleeding. Haemostasis maintains a relatively steady state of blood volume, blood pressure and blood flow through injured vessels. Bleeding disorders result from deficient platelets, disruption of the clotting cascade or a combination of factors.

THE PERSON WITH THROMBOCYTOPENIA

Thrombocytopenia is a platelet count of less than 150×10^9/L of blood. It can lead to abnormal bleeding. A continuing decline in circulating platelets can lead to spontaneous bleeding and haemorrhage from minor trauma (see Figure 32.14). Bleeding due to platelet deficiency usually occurs in small vessels, causing manifestations such as *petechiae* and *purpura*. The mucous membranes of the nose, mouth, GI tract and vagina often bleed. Serious and potentially fatal bleeding may occur when the platelet count is less than 150×10^9/L.

Thrombocytopenia results from one of three mechanisms: decreased production, increased sequestration in the spleen or accelerated destruction. Primary thrombocytopenia that leads to increased platelet destruction is discussed below. Secondary thrombocytopenia may be caused by aplastic anaemia, bone marrow malignancy, infection, radiation therapy or drug therapy (see Box 32.7). Heparin therapy is the most common drug-induced thrombocytopenia; it is included in the discussion that follows. Platelet sequestration usually is due to an enlarged spleen. Up to 80% of platelets may be removed from circulation with significant splenomegaly (Huether & McCance, 2013). Finally, thrombocytopenia may result from premature platelet destruction associated with disseminated intravascular coagulation (DIC).

Physiology review

Effective control of bleeding requires a series of complex interactions between the damaged tissue and blood vessel, platelets, clotting factors and processes to dissolve clots once bleeding has been controlled. Platelets are formed in the bone marrow under

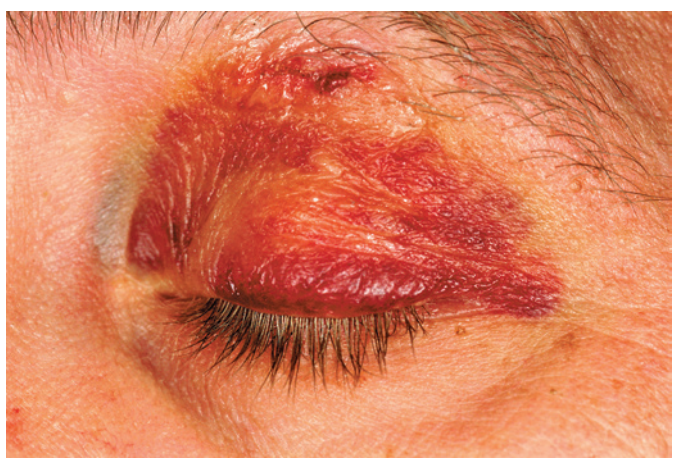


FIGURE 32.14 ■ Significant ecchymosis of the eyelid associated with minor trauma in a person with thrombocytopenia

Source: © Scott Camazine/Science Source.

BOX 32.7 Selected causes of secondary thrombocytopenia

Diseases
- Vitamin B_{12} anaemia
- Folic acid anaemia
- Aplastic anaemia
- Leukaemia
- Alcoholism
- DIC
- Infectious mononucleosis
- Viral infections
- HIV disease

Drugs
- Thiazide diuretics
- Aspirin
- Ibuprofen
- Indomethacin
- Naproxen
- Sulfonamides
- Phenytoin
- Cimetidine
- Digoxin
- Frusemide
- Heparin
- Morphine

Treatments
- Radiation therapy
- Chemotherapy
- Massive transfusion of stored blood

control of thrombopoietin, a protein produced by the liver, kidney, smooth muscle and bone marrow. Platelets are attracted to the damaged vessel wall, where they aggregate and release mediators that activate the clotting process. See Chapter 28 for a more complete discussion about platelets, clotting and haemostasis.

Pathophysiology

The two types of primary thrombocytopenia are immune thrombocytopenic purpura and thrombotic thrombocytopenic purpura.

Immune thrombocytopenic purpura

Immune thrombocytopenic purpura (ITP), also known as *idiopathic thrombocytopenic purpura*, is an autoimmune disorder in which platelet destruction is accelerated. In ITP, proteins on the platelet cell membrane stimulate autoantibody production, usually IgG antibodies. These autoantibodies adhere to the platelet membrane. Although the platelets function normally, the spleen reacts to them as being foreign and destroys the altered platelets after only 1 to 3 days of circulation.

MANIFESTATIONS The manifestations of ITP are due to bleeding from small vessels and mucous membranes. Petechiae and purpura develop, often on the anterior chest, arms, neck and oral mucous membranes. Bruising also may be apparent. As bleeding progresses, epistaxis (nosebleed), haematuria, excess menstrual bleeding and bleeding gums occur. Spontaneous intracranial bleeding is rare but does occur. Associated symptoms include weight loss, fever and headache.

INCIDENCE AND COURSE Acute ITP affects people of any age following a viral illness (Kessler, 2014). Acute ITP typically lasts only 1 to 2 months, resolving without long-term consequences. In its chronic form, ITP typically affects adults between ages 20 and 50; women are affected more often than men. Its onset is insidious. Chronic (or adult) ITP often occurs in people with other immune-associated disorders such as systemic lupus erythematosus or HIV disease.

Thrombotic thrombocytopenic purpura

Thrombotic thrombocytopenic purpura (TTP) is a rare disorder in which thrombi occlude arterioles and capillaries of the microcirculation. Many organs are affected, including the heart, kidneys and brain. The incidence of TTP is increasing (Huether & McCance, 2013). Its cause is unknown. Platelet aggregation is a key feature of the disorder. As RBCs circulate through partially occluded vessels, they fragment, leading to haemolytic anaemia.

MANIFESTATIONS TTP may be acute, the more common and severe form, or chronic. Acute idiopathic TTP may be fatal within months if untreated. The manifestations of TTP include purpura and petechiae and neurological symptoms such as headache, seizures and altered consciousness.

Heparin-induced thrombocytopenia

Heparin-induced thrombocytopenia (HIT) develops as a result of an abnormal response to heparin therapy. Unfractionated heparin carries a greater potential to precipitate HIT; it can, however, develop in people receiving low-molecular-weight heparin who have previously been treated with unfractionated heparin. See Chapter 31 for further discussion of heparin therapy and the forms of heparin.

Heparin is a protein that occurs naturally in human tissues and inflammatory cells. It can react directly with platelets, causing them to agglutinate (clump) and be removed from circulation by phagocytosis. This form of HIT, called type I HIT, typically causes mild thrombocytopenia. The more severe form, type II HIT, results from an immune reaction to heparin. In type II HIT, heparin forms an immune complex with a platelet protein known as platelet factor 4 (PF4). This complex acts as a foreign antigen in some people, stimulating antibody production. The antibody binds with the heparin–PF4 complex and these heparin–PF4–antibody complexes subsequently bind with circulating platelets, causing them to aggregate. As affected platelets aggregate, they are removed from circulation, leading to thrombocytopenia. In addition, small pieces of platelets can break loose, stimulating the clotting cascade and the development of thrombosis (clotting). The thrombocytopenia and thrombosis can be reversed by prompt withdrawal of heparin therapy, and an alternative replacement commenced (Lee & Arepally, 2013).

MANIFESTATIONS Despite thrombocytopenia, bleeding is usually a manifestation of HIT, probably because of the increased tendency to form clots that deplete clotting factors. The person may develop manifestations of an arterial thrombosis (severe pain, paresthesias, pallor and cool skin temperature, and pulselessness distal to the arterial occlusion) or of venous thrombosis (oedema, redness and warmth of the affected area). On rare occasions, an intravenous bolus of unfractionated heparin can precipitate an acute inflammatory response with manifestations that may mimic an acute pulmonary embolism: fever, chills, hypertension, tachycardia, dyspnoea, chest pain and cardiopulmonary arrest.

INTERPROFESSIONAL CARE

The diagnosis of thrombocytopenia is based on history, manifestations and diagnostic test results. Management focuses on treating or removing any causative factors and treating the platelet deficiency.

Diagnosis

The following diagnostic tests are used to identify thrombocytopenia:

- *FBC with platelet count* is done to evaluate blood cell counts, haemoglobin and haematocrit.
- *Antinuclear antibodies (ANA)* are measured to assess for autoantibodies and identify possible contributing disorders such as systemic lupus erythematosus.
- *Serological studies* for hepatitis viruses, cytomegalovirus (CMV), EBV, toxoplasma and HIV may be done. Serological testing also may be performed when HIT is suspected.
- *Bone marrow examination* evaluates for aplastic anaemia and megakaryocyte production.

Medications

Oral glucocorticoids, such as prednisone, are prescribed to suppress the autoimmune response. Many people who respond to glucocorticoid treatment relapse when the drug is withdrawn, however. Immunosuppressive drugs such as azathioprine, cyclophosphamide and cyclosporin may be used.

Prompt withdrawal of heparin therapy is vital when HIT is the cause of thrombocytopenia. All sources of heparin are removed, including heparin used to flush intravenous or other catheters and heparin-coated catheters. A non-heparin anticoagulant such as lepirudin (Refludan), a thrombin inhibitor, may be used. It is a recombinant form of hirudin, originally isolated from the salivary glands of leeches. Its primary adverse effect is bleeding; as a protein, it also can stimulate antibody development, resulting in rare instances of anaphylaxis. Argatroban is a synthetic direct thrombin inhibitor with a short half-life. It clears quickly when the infusion is discontinued, an advantage if excessive bleeding develops or invasive procedures must be performed.

Treatments

Platelet transfusions may be required to treat acute bleeding due to thrombocytopenia. Platelets are prepared from fresh whole blood; one unit contains 30 to 60 mL of platelet concentrate. The expected increase in platelets after one unit is infused is 10 000/mL. *Plasmapheresis*, or *plasma exchange therapy*, is the primary treatment for acute thrombotic thrombocytopenic purpura. The person's plasma is removed and replaced with fresh frozen plasma to remove autoantibodies, immune complexes and toxins.

Surgery

A *splenectomy* (surgical removal of the spleen) is the treatment of choice if the person with ITP relapses when glucocorticoids are discontinued. The spleen is the site of platelet destruction and antibody production. This surgery often cures the disorder, although relapse may occur years after splenectomy.

Nursing care

Assessment

- *Health history:* complaints of bruising with minor or no trauma, bleeding gums, nosebleed, heavy or prolonged menstrual periods, black, tarry or bloody stools, haematemesis, headache, fever or neurological symptoms; recent weight loss; recent viral or other illness; current and recent medications; exposure to toxins; previous exposure to heparin.
- *Physical examination:* skin and mucous membranes for colour, temperature, petechiae, purpura or bruises; vital signs; weight; mental status and level of consciousness; heart and breath sounds; abdominal exam; body fluids for occult blood.
- *Diagnostic tests:* FBC, haemoglobin and haematocrit, platelet count; serological and ANA test results; bone marrow examination results.

Nursing diagnoses and interventions

Inadequate platelets impair haemostasis, placing the person at risk of bleeding. Bleeding gums, an early sign of the disorder, affect oral mucous membrane integrity as well.

Ineffective protection

Bleeding is a serious complication associated with thrombocytopenia. As platelet counts decrease, the risk of bleeding increases. The risk is minimal with counts greater than 100×10^9/L, moderate when the count is around 50×10^9/L and significant when the count falls below 20×10^9/L.

- Monitor vital signs, heart and breath sounds every 4 hours. Frequently assess for other manifestations of bleeding:
  a. skin and mucous membranes for petechiae, ecchymoses and haematoma formation
  b. gums, nasal membranes and conjunctiva for bleeding
  c. overt or occult blood in emesis, urine or stool
  d. vaginal bleeding
  e. prolonged bleeding from puncture sites
  f. neurological changes: headache, visual changes, altered mental status, decreasing level of consciousness, seizures
  g. abdominal: epigastric pain, absence of bowel sounds, increasing abdominal girth, abdominal guarding or rigidity.

  Early identification of bleeding is important to prevent serious blood loss and shock.

CONSIDERATION FOR PRACTICE

Avoid invasive procedures such as rectal temperatures, urinary catheterisation and parenteral injections to the extent possible. Diagnostic procedures such as biopsy or lumbar puncture should be avoided if the platelet count is less than 150×10^9/L. Invasive procedures can cause tissue trauma and bleeding. Procedures that use large-bore needles should be delayed until the platelet count is increased.

- Apply pressure to puncture sites for 3 to 5 minutes; apply pressure to arterial puncture sites for 15 to 20 minutes. *Pressure promotes haemostasis and clot formation.*
- Instruct to avoid forcefully blowing the nose or picking crusts from the nose, straining to have a bowel movement and forceful coughing or sneezing. *These activities increase the risk of external and internal bleeding.*

Impaired oral mucous membranes

Thrombocytopenia frequently leads to bleeding of the gums and oral mucosa. As a result, risk of infection and impaired nutrition increases.

- Frequently assess the mouth for bleeding. Inquire about oral pain or tenderness. *Breakdown of oral mucous membranes increases the risk of infection and bleeding and causes discomfort with eating.*
- Encourage use of a soft-bristle toothbrush or sponge to clean teeth and gums. *Hard bristles may abrade oral mucosa, causing bleeding and increasing the risk of infection.*
- Instruct to rinse the mouth with saline every 2 to 4 hours. Apply petroleum jelly to lips as needed to prevent dryness and cracking. *Saline mouth rinses and petroleum jelly help maintain oral tissue integrity and promote cleansing and healing.*
- Instruct to avoid alcohol-based mouthwashes, very hot foods, alcohol and crusty foods. Teach to drink cool liquids at least every 2 hours. *Avoiding foods and liquids that traumatise oral mucosa increases comfort; fluid intake prevents dehydration and helps maintain mucous membrane integrity.*

Community-based care

In the adult, ITP often is a chronic disorder that the person and their family must learn to manage. Secondary thrombocytopenia may be either acute or chronic. Discuss the following topics when preparing the person and family for home care:

- nature of the disorder, its usual course and the treatment plan
- use of and desired and potential adverse effects of prescribed medications
- risks and benefits of surgery or treatments such as plasma replacement therapy
- the importance of follow-up tests and visits for care
- measures to reduce the risk of bleeding: safety measures such as a soft-bristle toothbrush, electric razor, avoidance of contact sports and hazardous activities, and avoiding medications that further interfere with platelet function (see Box 32.6).

Refer for home health or other community services (e.g. housekeeping, shopping) as indicated.

THE PERSON WITH HAEMOPHILIA

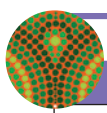
Haemophilia is a group of hereditary clotting factor disorders that lead to persistent and sometimes severe bleeding (see the accompanying 'Genetic considerations' box). Although often considered a disease of children, haemophilia may be diagnosed in adults. Deficiencies of three clotting factors—VIII, IX and XI—account for 90–95% of the bleeding disorders collectively called haemophilia (Huether & McCance, 2013).

Physiology review

When tissue injury occurs, platelets collect at the site, adhering to the damaged vessel wall (the platelet plug). Activation of the clotting cascade, a sequential process of interactive reactions of clotting factors, is vital to form a stable clot. Clotting factors are plasma proteins primarily produced by the liver. A number of these factors require the presence of vitamin K for synthesis and activation. Once the clot has been formed and stabilised, it begins to retract, pulling together the edges of the damaged blood vessel to initiate the healing process.

Pathophysiology

Haemophilia A (or *classic haemophilia*) is the most common type of haemophilia, caused by deficiency or dysfunction of clotting factor VIII. It is transmitted as an X-linked recessive disorder from mothers to sons (see Figure 32.15). The genetic defect of haemophilia A on the X chromosome may cause deficient factor VIII production or a defective form of the protein. When the concentration of the clotting factor is 5–35% of normal, the disease is *mild*. Bleeding is infrequent and usually associated with trauma. Concentrations of 1–5% of normal result in *moderate* disease. Again, bleeding usually occurs secondarily to trauma. *Severe* haemophilia occurs when concentrations are less



GENETIC CONSIDERATIONS
Focus on haemophilia

The incidence and pattern of inheritance for the forms of haemophilia differ:

- Haemophilia A occurs in about 1 in 10 000 male births, transmitted on the X chromosome: each male offspring has a 50% risk of inheriting the defective gene; each female offspring has a 50% risk of becoming a carrier.
- Haemophilia B occurs in about 1 in 100 000 male births, transmitted on the X chromosome.
- von Willebrand's disease affects about 1 in 100–500 people, usually inherited as an autosomal dominant trait: offspring of an affected person have a 50% risk of inheriting the trait and the disorder.
- Factor XI deficiency inherited as an autosomal recessive trait: each offspring of a carrier and an unaffected individual has as 50% risk of inheriting the trait; each offspring of two carriers has a 50% risk of being a carrier and a 25% risk of having the disorder. This deficiency is common in Ashkenazi Jews.

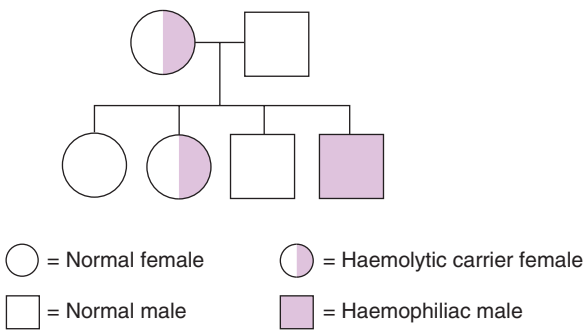


FIGURE 32.15 ■ The inheritance pattern of haemophilia A and B. Both are transmitted as X-linked recessive disorders. Females may be carriers, but males develop these disorders

than 1% of normal. Bleeding is frequent, often occurring without trauma (Fauci et al., 2008; Huether & McCance, 2013).

Haemophilia B (also called *Christmas disease*) accounts for about 15% of cases and is caused by a deficiency in factor IX. Despite the difference in clotting factor deficits, haemophilia A and B are clinically identical.

von Willebrand's disease, often considered a type of haemophilia, is the most common hereditary bleeding disorder (Grossman & Mattson, 2013). It is caused by a deficit of or defective von Willebrand (vW) factor, a protein that mediates platelet adhesion (Papadakis et al., 2013). Reduced levels of factor VIII often also are present, because vW factor carries factor VIII. This clotting disorder affects men and women equally. Bleeding associated with von Willebrand's disease rarely is severe. It often is diagnosed when prolonged bleeding follows surgery or a dental extraction.

Factor XI deficiency (or *haemophilia C*) is usually a mild disorder, identified when postoperative bleeding is prolonged. A comparison of the types of haemophilia is found in Table 32.9.

People with haemophilia form a platelet plug at the site of bleeding, but the clotting factor deficit impairs formation of a stable fibrin clot. The effect of vW factor deficiency is somewhat different, in that platelet aggregation at the site of injury is impaired. In either case, prolonged or extensive bleeding may result. Often bleeding occurs in response to injury or as a result of surgery. However, a severe clotting factor deficit can lead to spontaneous bleeding into the joints (*haemarthrosis*), deep tissues and CNS. Haemarthrosis often causes joint deformity and disability, usually of the elbows, hips, knees and ankles.

Manifestations

The following are manifestations of haemophilia:

- haemarthrosis
- easy bruising and cutaneous haematoma formation with minor trauma (e.g. an injection)
- bleeding from the gums and prolonged bleeding following minor injuries or cuts
- gastrointestinal bleeding, with haematemesis (vomiting blood), occult blood in the stools, gastric pain or abdominal pain
- spontaneous haematuria or epistaxis (nosebleed)
- pain or paralysis due to the pressure of haematomas on nerves
- intracranial haemorrhage is a potentially life-threatening manifestation of haemophilia.

INTERPROFESSIONAL CARE

Treatment of haemophilia focuses on preventing and/or treating bleeding, primarily by replacing deficient clotting factors. Specific treatment depends on the severity of the disorder and the specific factor deficiency. Care may be complicated by hepatitis or HIV disease in people with haemophilia treated with clotting factor concentrates prepared from multiple units of donated blood. Today, routine testing of all blood, improved blood donor screening and current methods of treating haemophilia have significantly reduced the risk of these blood-borne diseases.

Diagnosis

The following laboratory tests may be ordered:

- *Serum platelet levels* are measured and are usually normal.
- *Coagulation studies* such as APTT, bleeding time and prothrombin time are used to screen for haemophilia when abnormal bleeding occurs. APTT is increased in all types of haemophilia. Prothrombin time is unaffected in these

TABLE 32.9 Types of haemophilia

TYPE/NAME	DEFICIENCY	CHARACTERISTICS	TREATMENT
Haemophilia A (classic haemophilia)	Factor VIII	Transmitted by females; occurs primarily in males; bleeding time normal; coagulation time prolonged	Factor VIII concentrate or cryoprecipitate
Haemophilia B	Factor IX	Transmitted by females; occurs primarily in males; bleeding time normal; coagulation time prolonged	Factor IX (Christmas disease concentrate)
von Willebrand's disease	vW factor Factor VIII	Occurs in both females and males; bleeding time and coagulation time are both prolonged	Cryoprecipitate and desmopressin acetate (DDAVP)
Factor XI deficiency	Factor XI	Occurs in both males and females; the activated partial thromboplastin time is prolonged	Fresh frozen plasma

disorders but may be measured to rule out other disorders. Bleeding time is prolonged in von Willebrand's disease but normal in haemophilia A and B.

- *Factor assays* are performed; factor VIII is decreased in haemophilia A and often in von Willebrand's disease, factor IX is decreased in haemophilia B and factor XI in haemophilia C.
- *Amniocentesis* or *chorionic villus sampling* is used to identify the genetic defect of haemophilia when there is a known family history of the disease.

Medications

Deficient clotting factors are replaced regularly, as a prophylactic measure before surgery and dental procedures, and to control bleeding. Clotting factors may be given as fresh frozen plasma, cryoprecipitates or concentrates. Factor levels are measured on a regular basis to determine whether the treatment is adequate. Clotting factors are often self-administered and may be taken on either a regular or intermittent schedule.

Fresh frozen plasma replaces all clotting factors (including both factor VIII and factor IX) except platelets. When the cause of bleeding is not yet determined, fresh frozen plasma may be administered intravenously until a definitive diagnosis is made.

Haemophilia A is usually treated with either heat-treated factor VIII concentrate (heat treating reduces the risk of transmitting disease) or recombinant factor VIII. Although recombinant factor VIII, produced using recombinant DNA technology, eliminates the risk of viral disease transmission, its use is limited by cost. The dose of factor VIII is determined by the severity of the deficit and the presence or prospect of active bleeding (e.g. planned surgery).

Desmopressin acetate (DDAVP) may be given to people with mild haemophilia A or von Willebrand's disease prior to minor surgeries. This drug causes release of factor VIII and will raise blood levels two- or threefold for several hours, reducing the risk of bleeding and the need for clotting factor concentrate (Papadakis et al., 2013).

Factor IX concentrate (administered intravenously) is used to treat haemophilia B. Because factor IX concentrates also contain a number of other proteins, there is risk of thrombosis with recurrent use. They are used judiciously, only when needed. Products produced by recombinant technology or that are monoclonally purified carry a lower risk of stimulating thrombus formation (Fauci et al., 2008). Fresh frozen plasma replaces factor XI and is used when necessary. It may be given daily until the risk of bleeding decreases.

Factor VIII concentrates contain functional vW factor and may be used to treat von Willebrand's disease. Aspirin is avoided in all types of haemophilia.

Nursing care

Although primary responsibility for care falls to the person and family, nursing care presents challenges. For additional assessment and nursing care strategies for a person with haemophilia, see the accompanying nursing care plan.

Health promotion

Encourage people with a family history of haemophilia or bleeding disorders to seek genetic counselling during their family planning process. Although tests are available for the haemophilia gene, the technology to correct the disorder in utero does not yet exist. See Chapter 7.

Assessment

While severe haemophilia usually is diagnosed in childhood, milder cases may not be identified until surgery, invasive dental work or a traumatic injury causes extensive or prolonged bleeding. Focused assessment related to haemophilia includes the following:

- *Health history:* previous bleeding episodes with or without trauma; history of easy bruising, haematomas, epistaxis, bleeding gums, haematuria, vomiting blood or joint pain; aspirin use; family history of haemophilia or bleeding disorders.
- *Physical examination:* vital signs; bruising or bleeding of skin or mucous membranes; mental status; abdominal assessment; presence of joint deformity, decreased range of motion.
- *Diagnostic tests:* FBC including haemoglobin, haematocrit and platelet count; clotting factor assays; tests for occult blood (urine, stool, emesis); x-ray and scan results for evidence of bleeding.

Nursing diagnoses and interventions

Impaired blood clotting, the need for continuing care and disease management, and the risk of genetic transmission of haemophilia are priority problems for the person with haemophilia.

Ineffective protection

The inability to form stable clots and stem bleeding from injured blood vessels creates a significant risk for the person with haemophilia. Nursing care measures focus on preventing injury and protecting the skin from damage.

- Monitor for signs of bleeding, including haematomas, ecchymoses and purpura, as well as surface oozing or bleeding. Check emesis and stool for occult blood. *Bleeding may occur in cutaneous tissues as well as internal organs. Bleeding in the upper gastrointestinal tract may not be readily apparent in the stool.*
- Notify the doctor of any apparent bleeding. *Prompt intervention with administration of clotting factor concentrate decreases the risk of haemorrhage and subsequent hypovolaemia.*
- Avoid intramuscular injections, rectal temperatures and enemas. *These can pose a risk of tissue and vascular trauma, which can precipitate bleeding.*
- Use safety measures in personal care. For example, use an electric razor rather than a razor blade to shave. *Use of an electric razor minimises the opportunity to develop superficial cuts that may result in bleeding.*
- If bleeding occurs, control blood loss using gentle pressure, ice or a topical haemostatic agent, such as topical thrombin. *Direct pressure occludes bleeding vessels. Ice, a vasoconstrictor, may facilitate bleeding control, as do topical haemostatic agents.*

- Instruct to avoid activities that increase the risk of trauma, including contact sports and physical exertion associated with job performance, and to eliminate safety hazards in the home. *Depending on the severity of the clotting factor deficit, even minor trauma can lead to serious bleeding episodes. Safer activities such as non-contact sports (e.g. swimming, golf) and occupations that do not require physical labour may be substituted.*

Risk of ineffective health maintenance

Haemophilia is a chronic disorder, requiring active management to prevent and control bleeding and complications. Frequent visits to the doctor or clinic may be necessary. In addition, the person may need to learn to self-administer clotting factors and measures to prevent complications. The lifelong nature of the disorder may interfere with compliance, especially during early adulthood.

- Assess knowledge of disorder and the related treatments. Assessment allows identification of knowledge gaps and provides a basis on which to provide additional information. *Impaired disease management may be due to lack of knowledge or a conscious decision not to follow the recommendations of the healthcare provider.*
- Provide information about the bleeding disorder and prescribed medications and treatments. *Individualised instruction is more effective than general, possibly irrelevant information.*
- Provide emotional support, expressing confidence in the person's self-care abilities. *Emotional support helps the person incorporate the care regimen into their lifestyle.*
- Provide supervised learning and practice opportunities for administering clotting factors and topical haemostatic agents. *Successful practice sessions instil confidence in the ability to manage care and provide an opportunity for questions and exploring alternatives.*

Community-based care

Discuss the following topics when preparing the person with a bleeding disorder and the family for home care:

- Recognising the manifestations of internal bleeding: pallor, weakness, restlessness, headache, disorientation, pain, swelling. These manifestations

NURSING CARE PLAN A person with haemophilia

John Cruise is a 20-year-old TAFE student. He is admitted to the emergency department with a nosebleed that began when he fell during a touch football game. It has continued to bleed for over an hour.

ASSESSMENT

Mr Cruise states that he has haemophilia and realises that playing contact sport 'is probably a dumb thing to do'. He adds that he has not had any recent bleeding episodes. An icebag and manual pressure are applied in the emergency department. The doctor orders factor VIII concentrate to be administered. Physical assessment findings are 36.2°C, BP 118/64, R 18. Skin pale but warm. Laboratory tests reveal a prolonged APTT and a normal bleeding time and PT. Following treatment, Mr Cruise's bleeding subsides.

DIAGNOSES

- *Risk of aspiration* related to uncontrolled nosebleed.
- *Non-compliance* with activity recommendations.
- *Ineffective protection* related to lack of clotting factor VIII.

PLANNING

- Monitoring and reduction of potential complications required.
- Education required regarding disease risks and how to treat symptoms.

Expected outcomes

- Maintain an open airway.
- Maintain vital signs within his usual range.
- Exhibit no further signs of bleeding.
- Identify sports and recreation activities in which he can safely participate.
- Verbalise self-care measures to control bleeding.

IMPLEMENTATION

- Monitor vital signs and for further signs of bleeding.
- Assess airway and auscultate breath sounds.
- Review emergency measures to help stop bleeding.
- Reiterate the importance of seeking prompt medical attention if bleeding should occur.
- Advise regarding the importance of wearing a MedicAlert® bracelet identifying him as a haemophiliac.
- Discuss alternative non-contact sports and recreational activities.

EVALUATION

On discharge, Mr Cruise has no further signs of bleeding, shock or aspiration. He is able to verbalise methods to help stop local bleeding and the importance of seeking medical attention promptly when bleeding continues. Mr Cruise agrees to stop at a local chemist on the way home to order a MedicAlert® bracelet. In addition, Mr Cruise verbalises an understanding of the importance of avoiding contact sports and has identified swimming and golf as alternative leisure activities that he might enjoy.

CRITICAL THINKING IN THE NURSING PROCESS

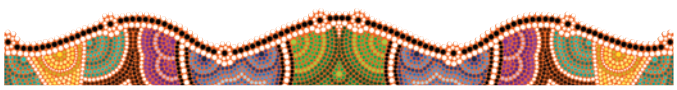
1. What is the pathophysiological basis for the bleeding that occurs in haemophilia A and B?
2. What was Mr Cruise's priority nursing diagnosis? Why?
3. Why is family planning a special consideration with a person who has haemophilia?
4. Outline a plan to teach the family of a person diagnosed with haemophilia how to administer an intravenous infusion.
5. Develop a care plan for Mr Cruise for the nursing diagnosis of *Impaired social interaction*. Consider Mr Cruise's age and developmental level in creating the plan.

REFLECTION ON THE NURSING PROCESS

1. What are the take-home messages you have learned in this case study?
2. How can they be used within your daily practice?

require emergency medical care and should be reported immediately.
- Applying cold packs and immobilising the joint for 24 to 48 hours if haemarthrosis occurs.
- Using analgesics for pain; avoiding prescription and over-the-counter drugs containing aspirin.
- Ensuring a safe home environment (e.g. padding sharp edges of furniture, using transition lighting or a night light, avoiding scatter rugs and wearing protective gloves when working in the house or garden).
- Using safe grooming practices such as electric razors.
- Wearing a MedicAlert® bracelet in case of accident.
- Practising good dental hygiene to decrease potential tooth decay and extractions. If dental procedures are necessary, discuss the need for prophylactic factor administration with the dentist and doctor.
- Following safer sex practices.
- Preparing and administering intravenous medications.

Refer the person and family to a local haemophilia or bleeding disorders support group. Provide contact information for national organisations and support services, such as the Australian Haemophilia Foundation: www.haemophilia.org.au.



THE PERSON WITH DISSEMINATED INTRAVASCULAR COAGULATION

Disseminated intravascular coagulation (DIC) is a disruption of haemostasis characterised by widespread intravascular clotting and bleeding. It may be acute and life threatening or relatively mild. DIC is a clinical syndrome that develops as a complication of a wide variety of other disorders (see Box 32.8). Sepsis is the most common cause of DIC. Gram-negative and gram-positive bacteria as well as viruses, fungi and protozoal infections may lead to DIC (Huether & McCance, 2013).

Pathophysiology

DIC is triggered by endothelial damage, release of tissue factors into the circulation or inappropriate activation of the clotting cascade by an endotoxin. Both the intrinsic and the extrinsic clotting cascade may be activated, although the extrinsic cascade usually is the one activated. Extensive thrombin entering the systemic circulation overwhelms natural anticoagulants, leading to unrestricted clot formation (Huether & McCance, 2013). Clotting may be localised to an individual organ or widespread with deposition of small thrombi and emboli throughout the microvasculature (Fauci et al., 2008). The widespread clotting consumes clotting factors (prothrombin, platelets, factor V and factor VIII, in particular) and activates fibrinolytic processes with anticoagulant production. As a result, haemorrhage occurs (see Figure 32.16).

The sequence of DIC follows:

1. Endothelial damage, tissue factors or toxins stimulate the clotting cascade.
2. Excess thrombin within the circulation overwhelms naturally occurring anticoagulants.

BOX 32.8 Conditions that may precipitate disseminated intravascular coagulation

Tissue damage
- Trauma: burns, gunshot wounds, frostbite, head injury
- Obstetric complications: septic abortion, abruptio placentae, amniotic fluid embolus, retained dead foetus
- Neoplasms: acute leukaemia, adenocarcinomas
- Haemolysis
- Fat embolism

Vessel damage
- Aortic aneurysm
- Acute glomerulonephritis
- Haemolytic uraemic syndrome

Infections
- Bacterial infection or sepsis
- Viral or mycotic infections
- Parasitic or rickettsial infection

3. Widespread clotting occurs within the microvasculature.
4. Thrombi and emboli impair tissue perfusion, leading to ischaemia, infarction and necrosis.
5. Clotting factors and platelets are consumed faster than they can be replaced.
6. Clotting activates fibrinolytic processes, which begin to break down clots.
7. Fibrin degradation products (FDPs, potent anticoagulants) are released, contributing to bleeding.
8. Clotting factors are depleted, the ability to form clots is lost and haemorrhage occurs.

Manifestations

The manifestations of DIC result from both clotting and bleeding, although bleeding is more obvious, especially in acute DIC. Bleeding ranges from oozing blood following an injection to frank haemorrhage from every body orifice (see the 'Manifestations' box below). Chronic DIC may be asymptomatic or may present with peripheral cyanosis, thrombosis and pre-gangrenous changes in the fingers and toes, nose and

MANIFESTATIONS DIC

- Frank haemorrhage from incisions
- Oozing of blood from punctures, intravenous catheter sites
- Purpura, petechiae, bruising
- Cyanosis of extremities
- Gastrointestinal bleeding or haemorrhage
- Dyspnoea, tachypnoea, bloody sputum
- Tachycardia, hypotension
- Haematuria, oliguria, acute renal failure
- Manifestations of increased intracranial pressure: decreased level of consciousness, pupillary, motor and sensory changes
- Mental status changes

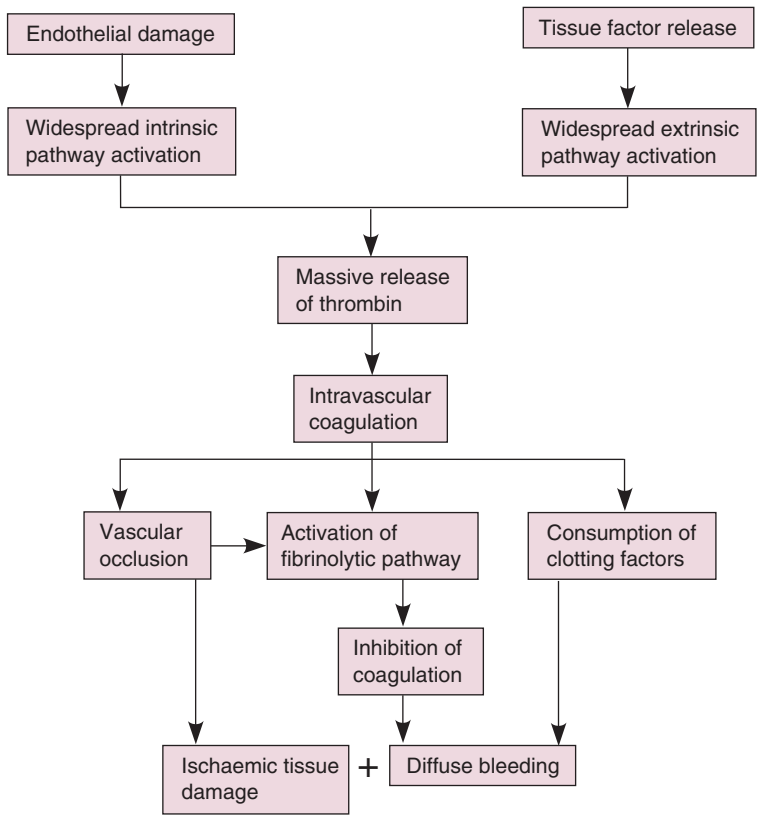


FIGURE 32.16 ■ Disseminated intravascular coagulation (DIC). Endothelial cell injury or release of tissue factors activates the intrinsic or extrinsic clotting pathway (or both). As a result, numerous microthrombi form throughout the vasculature, causing ischaemic tissue damage. Simultaneously, rapid consumption of clotting factors and activation of fibrinolytic mechanisms trigger widespread bleeding

genitalia with superficial thrombophlebitis, as well as deep venous thrombosis (Papadakis et al., 2013; Hampton, 2012).

INTERPROFESSIONAL CARE

Treatment of DIC is directed towards treating the underlying disorder and preventing further bleeding or massive thrombosis. Prompt treatment stabilises the person, reduces complications and allows recovery to occur; it does not cure DIC.

Diagnosis

Diagnostic tests are used to confirm the diagnosis of DIC and evaluate the risk of haemorrhage.

- *FBC* and *platelet count* are used to evaluate the haemoglobin, haematocrit and number of circulating platelets. *Schistocytes*, fragmented RBCs, may be noted due to cell trapping and damage within fibrin thrombi. The platelet count is decreased.
- *Coagulation studies* show prolonged prothrombin time (PT), *partial thromboplastin time (PTT)* and thrombin time, and a *low fibrinogen level* due to depletion of clotting factors. The fibrinogen level helps predict bleeding in DIC: as it falls, the risk of bleeding increases.
- *Fibrin degradation products (FDPs)* or *fibrin split products (FSPs)*, and plasma D-dimers are increased due to the fibrinolysis that occurs with DIC (Hampton, 2012).

Treatment

When bleeding is the major manifestation of DIC, fresh frozen plasma and platelet concentrates are given to restore clotting factors and platelets. Heparin, although controversial, may be administered. Heparin interferes with the clotting cascade and may prevent further clotting factor consumption due to uncontrolled thrombosis. It is used when bleeding is not controlled by plasma and platelets, as well as when the person has manifestations of thrombotic problems such as acrocyanosis and possible gangrene. Long-term heparin therapy (administered by injection or continuous infusion using a portable pump) may be necessary for people with chronic DIC.

Nursing care

Assessment

Nurses can be instrumental in identifying early manifestations of DIC, facilitating timely intervention. Focused nursing assessment for DIC includes:

- *Health history:* recent abortion (spontaneous or therapeutic) or current pregnancy; presence of a known malignant

tumour; history of abnormal bleeding episodes or a haematological disorder.
- *Physical examination:* bleeding from puncture wounds (e.g. injections), IV sites, incisions; haematuria, obvious or occult blood in emesis or stool, epistaxis, other abnormal bleeding; vital signs; heart and breath sounds; abdominal assessment, including girth, contour, bowel sounds, tenderness or guarding to palpation; colour, temperature, skin condition of hands, feet and digits; petechiae or purpura of skin, mucous membranes.
- *Diagnostic tests:* FBC with haemoglobin, haematocrit; platelet count; coagulation studies; evaluations of organ system function (e.g. liver and renal function tests); CT scans of the head and abdomen.

Nursing diagnoses and interventions

People with acute DIC often are critically ill, with multiple nursing care needs. Priority nursing diagnoses discussed in this section focus on impaired tissue perfusion and gas exchange, pain and fear. Septic shock may precipitate DIC; haemorrhagic shock may occur as a complication of DIC. See Chapter 10 for nursing diagnoses and interventions related to these problems.

Ineffective tissue perfusion

Thrombi and emboli forming throughout the microcirculation affect the perfusion of multiple organs and tissues. Additionally, bleeding due to clotting factor consumption affects cardiac output and blood flow to these tissues.
- Assess extremity pulses, warmth and capillary refill. Monitor level of consciousness (LOC) and mental status. *Monitoring central and peripheral tissue perfusion facilitates early treatment of impaired perfusion.*
- Carefully reposition person at least every 2 hours. *Position changes facilitate circulation and tissue perfusion and also provide an opportunity to assess for purpura, pallor and bleeding.*
- Discourage crossing the legs and do not elevate the knees on the bed or with a pillow. *These positions may impair arterial and venous flow to the lower legs and feet, increasing vascular stasis and the risk of thrombosis.*
- Minimise use of tape on the skin, using binders, non-adhesive dressings and other devices as needed. *Preventing skin trauma reduces the risk of bleeding and potential infection.*

CONSIDERATION FOR PRACTICE

Promptly report complaints of chest pain, changes in mental status, LOC, tissue perfusion, respirations, gastrointestinal function and urinary output. Chest pain or respiratory changes (tachypnoea, dyspnoea, orthopnoea) may be due to angina, pulmonary embolism or bleeding into lung tissue. Changes in mentation or LOC can indicate cerebral ischaemia. A painful, pale and cold extremity with no or diminished pulses indicates arterial occlusion. Prompt intervention is critical to save the extremity. Acute abdominal pain, decreased bowel sounds and GI bleeding may indicate mesenteric occlusion, a surgical emergency. Decreased urine output may signify renal artery thrombosis; renal failure may develop.

Impaired gas exchange

Microclots in the pulmonary vasculature are likely to interfere with gas exchange in the person with DIC.
- Monitor oxygen saturation continuously. Administer oxygen as required/ordered. *Oxygen saturation levels are a non-invasive means of assessing gas exchange. Supplemental oxygen promotes gas exchange and reduces cardiac work, relieving dyspnoea.*

CONSIDERATION FOR PRACTICE

Monitor arterial blood gas results; report abnormal results to the doctor. Low PaO_2 and rising $PaCO_2$ levels indicate impaired gas exchange and may signify the need for additional treatment.

- Place in Fowler's or high-Fowler's position as tolerated. *Elevating the head of the bed improves diaphragmatic excursion and alveolar ventilation.*
- Maintain bed rest. *Bed rest reduces oxygen demands and cardiac work.*
- Encourage deep breathing and effective coughing. *Increased respiratory depth and clearance of secretions from airways improves alveolar ventilation and oxygenation.*
- Cautious nasotracheal suctioning may be instituted if cough is ineffective or an endotracheal tube is in place. *Removal of secretions facilitates ventilation and oxygenation. However, care must be used to minimise suction-induced hypoxia and airway trauma.*
- Administer analgesics and anti-anxiety drugs as needed to control pain and anxiety. Provide reassurance and comfort measures. *Pain and anxiety increase the respiratory rate and decrease the depth of respirations, reducing effective ventilation and gas exchange.*

Pain

Both the underlying cause of DIC and tissue ischaemia from microvascular clots can cause pain. Identifying the aetiology of pain is important to identify potential complications or harmful effects of DIC and to institute effective treatment.
- Use a standard pain scale to evaluate and monitor pain and analgesic effectiveness. *Monitoring pain and response to medication facilitates development of an appropriate and effective treatment plan.*
- Handle extremities gently. *Gentle handling reduces the risk of further injury to, and pain in, ischaemic tissues.*
- Apply cool compresses to painful joints. *Application of cold decreases pain through the gate-control mechanism, inhibiting the dorsal horn of the spinal cord and reducing the sensation of pain.*

CONSIDERATION FOR PRACTICE

Notify the doctor promptly of new or a sudden increase in pain, especially when accompanied by changes in assessment findings. New or increased complaints of pain may signify increased circulatory impairment and ischaemic changes in tissues such as the heart, bowel or extremities. Circulation to a painful, pale or cyanotic, or cold extremity may be occluded by an arterial clot. Prompt intervention is necessary to save the extremity. Acute abdominal pain may signify mesenteric occlusion, a surgical emergency. Anginal pain may indicate occlusion of coronary arteries.

CONSIDERATION FOR PRACTICE

Continuously monitor effects of analgesics and mental and respiratory status. Analgesics may mask manifestations of neurological impairment due to thromboembolism and may depress the respiratory centre, further impairing gas exchange. Judicious analgesic administration with careful monitoring is vital to safely provide effective pain relief.

Fear

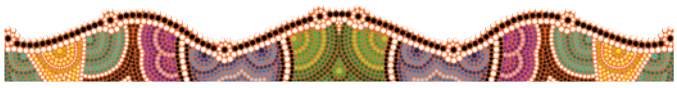
The underlying serious illness and a complication such as DIC result in an uncertain prognosis, often accompanied by fear.

- Encourage the person and family to verbalise concerns. *This helps the person and family identify their concerns and frame questions.*
- Answer questions truthfully. Providing honest answers is vital to developing a therapeutic nurse–individual relationship. *Accurate responses allow the person and their family to set priorities as they plan for an uncertain future.*
- Help the person and family identify coping strategies to manage this significant situational stressor. *Implementing past effective coping methods may provide the skills to manage the current crisis.*
- Provide emotional support. *The presence of a caring nurse helps reduce the fear and anxiety associated with a crisis.*
- Maintain a calm environment. *A calm environment provides reassurance that the situation is in control, reduces anxiety and promotes rest.*
- Respond promptly when the person calls for help. *Prompt response to expressed needs helps develop a trusting relationship and a sense of security that assistance is readily available.*
- Teach relaxation techniques. *Relaxation techniques can reduce muscle tension and other signs of anxiety. Gaining control over physical responses can help the person gain a sense of control over the situation.*

Community-based care

Although the immediate crisis of acute DIC is resolved prior to discharge, the person may have some continuing effects of the disorder, such as impaired tissue integrity of distal extremities. Teach the person and family about specific care needs, such as foot care (see Box 31.6) or dressing changes. Provide instruction about any continuing medications and follow-up care.

People with chronic DIC may require continuing heparin therapy, using either intermittent subcutaneous injections or a portable infusion pump. Teach the person and family members how to administer the injection or manage the infusion pump. Provide a referral to home healthcare or a home intravenous management service for assistance. Discuss the manifestations of excessive bleeding or recurrent clotting that need to be reported to the doctor.



CHAPTER HIGHLIGHTS

- Anaemia is the most common disorder of the red blood cells; nutritional deficiencies are the most common causes of anaemia. Its manifestations relate to the function of RBCs and haemoglobin, transporting oxygen to the cells: fatigue, increased respiratory and heart rates, shortness of breath with activity and pallor.
- Genetically transmitted disorders such as sickle cell disease and thalassaemia can cause significant anaemia and associated problems in affected populations. These people require teaching and episodic acute care for crises such as vaso-occlusive crisis in sickle cell disease.
- Nursing care related to anaemia is primarily educational to prepare the person for effective self-care, including diet, prescribed medications and measures to prevent sickling episodes (for those with sickle cell disease).
- Leukaemia and lymphomas are the primary disorders of white blood cells and lymphoid tissues.
- Manifestations of the leukaemias reflect the altered ability of abnormal WBCs to perform effective immune surveillance and crowding of the bone marrow and other organs by rapidly proliferating cells. Frequent sore throats, increased risk of infection and manifestations of anaemia and thrombocytopenia are seen, as well as an enlarged spleen and abdominal pain.
- Four major subgroups of leukaemia are identified: acute and chronic myeloid leukaemias, and acute and chronic lymphocytic (or lymphoblastic) leukaemias. The primary population affected differs for each of these leukaemias, as does their course.
- Genetic alterations and certain viruses are linked to the development of leukaemia, as are exposure to chemotherapy drugs, environmental toxins and ionising radiation.
- Lymphocytic leukaemias and lymphomas are closely related disorders.
- Nursing care for people with leukaemia and lymphoma focuses on reducing the risk of infection and bleeding, managing the effects of chemotherapy and radiation therapy and, in some cases, caring for people before and after bone marrow or stem cell transplant.

- The major risks associated with bone marrow and stem cell transplant are infection prior to, and immediately following, the transplant; and graft-versus-host disease, a potentially fatal condition. A pruritic rash and desquamation of the palms and soles; abdominal pain, nausea and diarrhoea; and jaundice and elevated liver enzymes are common early manifestations of GVHD.
- The treatment of and nursing care for people with lymphomas (including Hodgkin's disease and non-Hodgkin's lymphoma) is similar to that provided for people with leukaemia.
- Multiple myeloma is a malignancy of plasma cells, which are B lymphocytes that produce antibodies. Circulating M proteins and Bence Jones proteins in the urine are seen in multiple myeloma. The usual presenting manifestation is bone pain. Pathological fractures and hypercalcaemia are common complications of multiple myeloma as bone is destroyed.
- Bleeding and clotting disorders can result from either inadequate platelets (thrombocytopenia) or disruption of the clotting mechanisms (haemophilia, DIC). Petechiae and purpura are common manifestations of bleeding/clotting disorders.
- Haemophilias are genetically transmitted disorders. Haemophilia A and B are transmitted on the X chromosome (sex-linked) from mother to son. von Willebrand's disease, the most common bleeding disorder, is transmitted as an autosomal dominant disorder and affects men and women equally.
- Haemophilias are treated by replacement of the missing clotting factor and measures to prevent injury and bleeding.
- Disseminated intravascular coagulation is a disorder of widespread microvascular clotting. It commonly is precipitated by sepsis, but also may occur with conditions such as major trauma, malignancy or as an obstetric emergency.
- In DIC, platelets and clotting factors are consumed by the abnormal clotting processes, leading to the manifestations of bleeding, including frank haemorrhage, haematuria, oozing blood from parenteral and intravenous injection sites, and GI bleeding. Blood flow to organs and tissues is compromised by clot formation, leading to manifestations such as cyanosis of extremities, abdominal pain, kidney failure and changes in mental status and level of consciousness. Nursing care is supportive, focusing on administering prescribed treatments and monitoring and supporting cardiovascular, respiratory and kidney function.

CONCEPT CHECK

1 In assessing a woman with moderate anaemia, the nurse would expect to find which of the following?
1 haematocrit 45%
2 pulse rate 140
3 complaints of shortness of breath with exercise
4 WCC 150×10^9/L

2 The nurse caring for a person after gastric resection observes carefully for evidence of nutritional deficiency anaemia related to malabsorption, including:
1 numbness and tingling of extremities
2 steatorrhoea
3 dark yellow or bronze skin colour
4 bone pain

3 The nurse caring for a person with acute myeloid leukaemia plans which of the following nursing interventions during hospitalisation? (Select all that apply.)
1 Place in a private room.
2 Implement airborne infection control precautions.
3 Assist with oral hygiene after meals.
4 Monitor rectal temperature q4h.
5 Request soft, bland diet.

4 The nurse caring for a person with lymphoma who is being started on chemotherapy regimens understands that chemotherapy drugs are used in combination to:
1 target malignant cells in different organs
2 prevent the development of adverse effects
3 target different phases of the cell cycle
4 support growth and development of normal cells

5 A person with multiple myeloma calls the home health nurse complaining of new-onset severe back pain. The appropriate response by the nurse is to:
1 reassure the person that bone pain is expected with this disease
2 inquire about the person's use of NSAIDs and analgesics to manage pain
3 suggest use of a back brace to reduce pain
4 notify the doctor of the onset of new pain

6 The nurse observes reddish-purple spots and areas of purple bruising on a newly admitted person. Which laboratory results support this assessment finding?
1 haematocrit 28%
2 platelets 100×10^9/L
3 INR 4.0
4 WCC 150×10^9/L

7 A person whose husband has haemophilia asks if her newborn baby girl could have the disease. The nurse's response is based on the knowledge that:
1 the most common forms of haemophilia are transmitted as sex-linked recessive disorders; her daughter is at risk of carrying the defective gene
2 because haemophilia is a sex-linked recessive disorder carried on the Y chromosome, her daughter has no risk of having or carrying the disease
3 haemophilia is an autosomal dominant disorder; therefore, her daughter has a 50% chance of having the disorder
4 although haemophilia is genetically transmitted, its pattern of inheritance is unknown and her daughter will need to be tested for the defective gene

8 The nurse administering platelets to a person with disseminated intravascular coagulation (DIC) understands that the intended effect of this treatment is to:
1 replace specific clotting factors
2 promote intravascular clotting
3 restore tissue oxygenation
4 replace depleted platelets

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UNIT 8 BUILDING CLINICAL COMPETENCE

Responses to altered cardiac function

CLINICAL SCENARIO

You have been assigned to work with the following four people for the 0700 shift on a cardiac telemetry unit. Significant data obtained during report are as follows:

- Betty Williams, aged 62, was admitted with acute anterior MI and had successful fibrinolytic therapy 3 days ago. Significant history includes type 2 diabetes, angina, hypertension and a history of smoking (1.5 to 2 packets per day for 45 years). Her CCU course was uneventful. Current vital signs are T 37.2°C, P 76, R 16, BP 148/88. Cardiac monitor shows normal sinus rhythm with no ectopy. She has been pain free since her glyceryl trinitrate infusion was titrated off in the CCU 2 days ago. Lung sounds are clear.
- Arnold Markus, aged 71, was admitted with acute heart failure 2 days ago, treated and stabilised in the CCU, and transferred to the cardiac telemetry unit. Current vital signs are T 36.9°C, P 88, R 18, BP 112/74. Cardiac monitor shows normal sinus rhythm with new isolated premature ventricular contractions (PVCs). Lung sounds are clear in upper lobes with crackles in the left base and he was able to sleep intermittently during the night using 2 pillows.
- Theresa Cartwright is a 34-year-old female admitted for anticoagulant therapy after developing a deep venous thrombosis after a fall down the steps and hitting her calf. She was started on heparin yesterday and needs blood drawn for an activated partial thromboplastin time (APTT) to determine her morning dose of heparin.
- Scott Jacoby is a 25 year old with Down syndrome. He was admitted yesterday with an upper respiratory infection. On assessment he was pale, T 38.3°C, P 100, R 30 with dyspnoea on exertion, BP of 118/86 and multiple bruises and petechiae on his arms and legs. He is scheduled for a bone marrow examination this morning.

Critical thinking questions

1 In what order would you visit these people after report?

1.
2.
3.
4.

2 What top two priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Betty Williams		
Arnold Markus		
Theresa Cartwright		
Scott Jacoby		

3 You are completing a beginning-of-shift assessment with Mr Markus. Which of the following items would take priority?

1. rhythm strip analysis
2. lung sounds
3. oxygen saturation
4. heart sounds

4 The charge nurse tells you that the monitor at the nursing station shows that Mr Markus is having an increase in PVCs. You would be most concerned after reviewing a rhythm strip if the PVCs had which of the following characteristics?

1. a frequency of 4 per minute
2. could be felt by the person
3. were unifocal in morphology
4. fell on the T wave of the preceding beat

5 Ms Williams rings the call bell and tells you that she is having pressure in her chest. On assessment, she rates it as a 5. Which of the following nursing actions should take priority?

1. Obtain a full symptom assessment.
2. Administer a prn glyceryl trinitrate tablet SL.
3. Call for an electrocardiogram.
4. Notify the doctor.

6 Which is the most important for you to report when caring for Mr Jacoby?

1. constipation and straining with bowel movements
2. fever and burning on urination
3. weight loss and decreased appetite
4. dyspnoea and shortness of breath with exercising

7 When a person is placed on warfarin therapy, which laboratory studies would you expect to draw? (Select all that apply.)

1. activated partial thromboplastin time (APTT)
2. International Normalized Ratio (INR)
3. partial thromboplastin time (PTT)
4. full blood cell count (FBC)
5. white blood cell count (WBC)
6. prothrombin time (PT)

8 Mrs Cart wright needs further teaching regarding anticoagulant therapy when she makes which statement?

1. 'The heparin will be continued for four to five days for the Coumadin to reach a good effect.'
2. 'I need to continue to have blood drawn to watch my drug levels as long as I am taking these drugs.'
3. 'I cannot continue to take birth control pills while I am taking these drugs.'
4. 'I need to take the medication at the same time every day for the drug to be effective.'

9 After a person has undergone mitral valve replacement surgery, the nurse would be most concerned about possible risks to medication therapy if the person had a history of which of the following disorders listed in the medical record?

1. glaucoma
2. gout
3. duodenal ulcer
4. osteoarthritis

10 Which nursing diagnosis has the highest priority for the person with varicose veins?

1. *Body image disturbance*
2. *Impaired tissue perfusion*
3. *Activity intolerance*
4. *Risk of infection*

11 Which is a priority teaching instruction for the person with a history of Raynaud's disease?

1. Enter a smoking cessation program.
2. Reduce dietary fats and carbohydrates.
3. Wear gloves and socks in cold weather.
4. Begin an exercise program.

12 A prescription for the calcium channel blocker diltiazem is ordered for a person with hypertension. What will the nurse instruct the person regarding medication administration?

1. Limit fluids to decrease the development of peripheral oedema.
2. Notify the doctor for a pulse of less than 60 bpm.
3. Increase fibre in the diet as diarrhoea may be a side effect.
4. Report tachycardia and an increase in blood pressure.

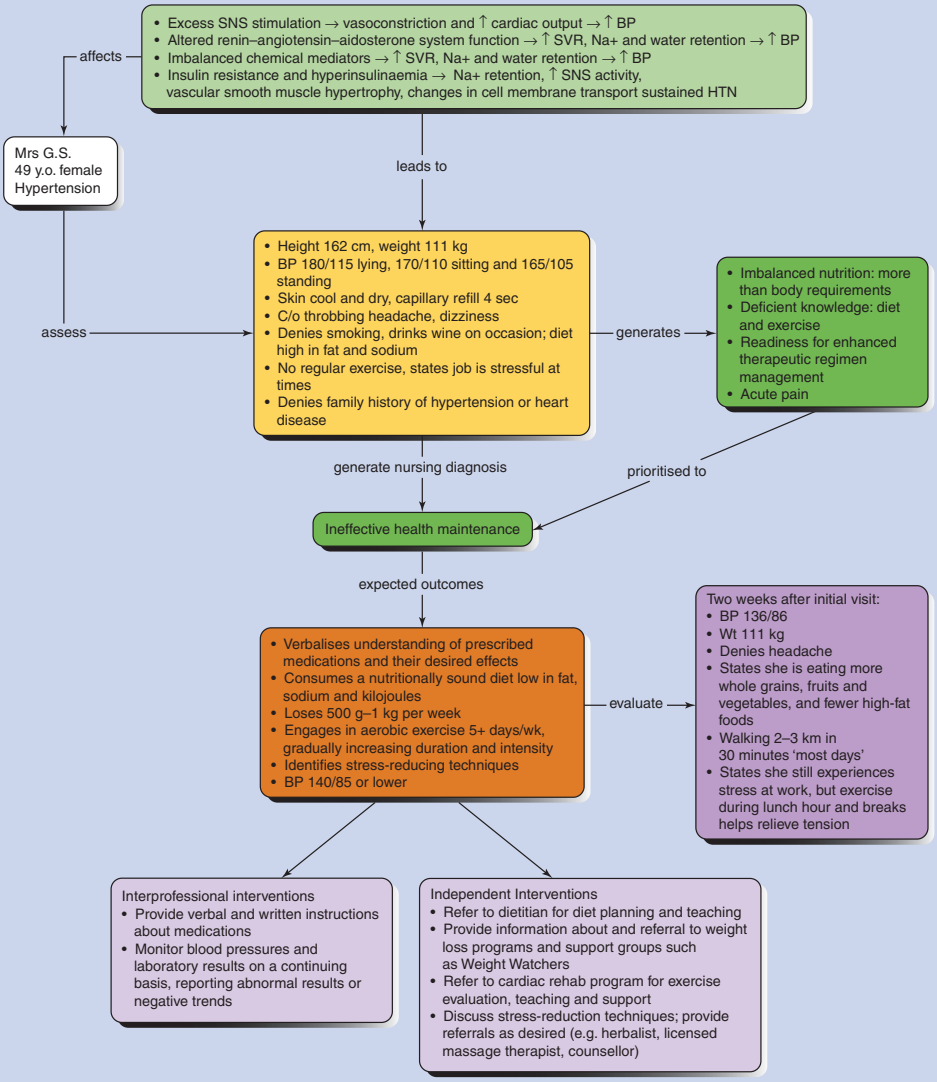
13 A person with history of haemophilia fell and cut his leg while bushwalking. Which intervention should the person perform until help arrives?

1. Apply a tourniquet above the cut.
2. Splint the leg to prevent movement.
3. Apply pressure to the femoral artery.
4. Apply gentle pressure over the cut.

CASE STUDY

Grace Schmidt is a 49-year-old female who works as a teacher at a high school. She comes to the medical centre complaining of a throbbing headache and dizziness. Her height and weight are 162 cm and 111 kg. Upon assessment her vital signs are P 100, R 16, BP lying is 180/115, sitting is 170/110 and standing is 165/105. Her skin is cool and dry. Her capillary refill is 4 seconds. She denies smoking, drinks an occasional glass of wine and does not participate in a regular exercise program. She states that her job can be stressful at times. Nutrition assessment indicates a diet high in fats and sodium. She denies any family history of hypertension or heart disease. She is married and has a daughter and son who live in the same town. A medical diagnosis of hypertension is determined.

Based on Mrs Schmidt's assessment, blood pressure readings and weight, the priority nursing diagnosis of *Ineffective health maintenance* is identified for planning nursing care.



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UNIT 9

RESPONSES TO ALTERED RESPIRATORY FUNCTION

CHAPTER 33

A PERSON-CENTRED APPROACH TO ASSESSING THE RESPIRATORY SYSTEM

MAJELLA HALES

KEY TERMS

LEARNING OUTCOMES

- Describe the anatomy, physiology and functions of the respiratory system.
- Explain the mechanics of ventilation.
- Differentiate between oxygen and carbon dioxide transport and how affinity influences loading and unloading.
- Examine investigations and observations important for assessing a person's respiratory system function.
- Demonstrate accurate interpretation of data obtained from a person requiring review of their respiratory system.

CLINICAL COMPETENCIES

- Conduct and document a health history for people at risk of or currently experiencing alterations in the respiratory system.
- Conduct and document a physical assessment of respiratory structures and functions.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Tongue depressor
- Penlight
- Nasal speculum
- Metric ruler
- Marking pen
- Stethoscope with diaphragm

The respiratory system provides the cells of the body with oxygen and eliminates carbon dioxide, formed as a waste product of cellular metabolism. The events in this process, called respiration, are:

- *Pulmonary ventilation:* Air is moved into and out of the lungs.
- *External respiration:* Exchange of oxygen and carbon dioxide occurs between the alveoli and the blood.
- *Gas transport:* Oxygen and carbon dioxide are transported to and from the lungs and the cells of the body via the blood.
- *Internal respiration:* Exchange of oxygen and carbon dioxide occurs between the blood and the cells.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE RESPIRATORY SYSTEM

The respiratory system functions as a whole, but is divided into the upper respiratory system and the lower respiratory system for discussion of respiratory disorders in the following chapters.

THE UPPER RESPIRATORY SYSTEM

The upper respiratory system serves as a passageway for air moving into the lungs and carbon dioxide moving out to the external environment (see Figure 33.1). As air moves through these structures, it is cleaned, humidified and warmed.

The nose

The nose is the external opening of the respiratory system. The external nose is given structure by the nasal, frontal and maxillary bones as well as by plates of hyaline cartilage. The nostrils (also called the external nares) are two cavities within the nose, separated by the nasal septum. These cavities open into the nasal portion of the pharynx through the internal nares. The nasal cavities just behind the nasal openings are lined with skin that contains hair follicles, sweat glands and sebaceous glands. The nasal hairs filter the air as it enters the nares. The rest of the cavity is lined with mucous membranes that contain olfactory neurons and goblet cells that secrete thick mucus. The mucus not only traps dust and bacteria but also contains lysozyme, an enzyme that destroys bacteria as they enter the nose. As mucus and debris accumulate, mucosal ciliated cells move them towards the pharynx, where they are swallowed. The mucosa is highly vascular, warming air that moves across its surface.

Three structures project outward from the lateral wall of each nasal cavity: the superior, middle and inferior turbinates. The turbinates cause air entering the nose to become turbulent and also increase the surface area of mucosa exposed to the air. As air moves through this area, heavier particles of debris drop out and are trapped in the mucosa of the turbinates.

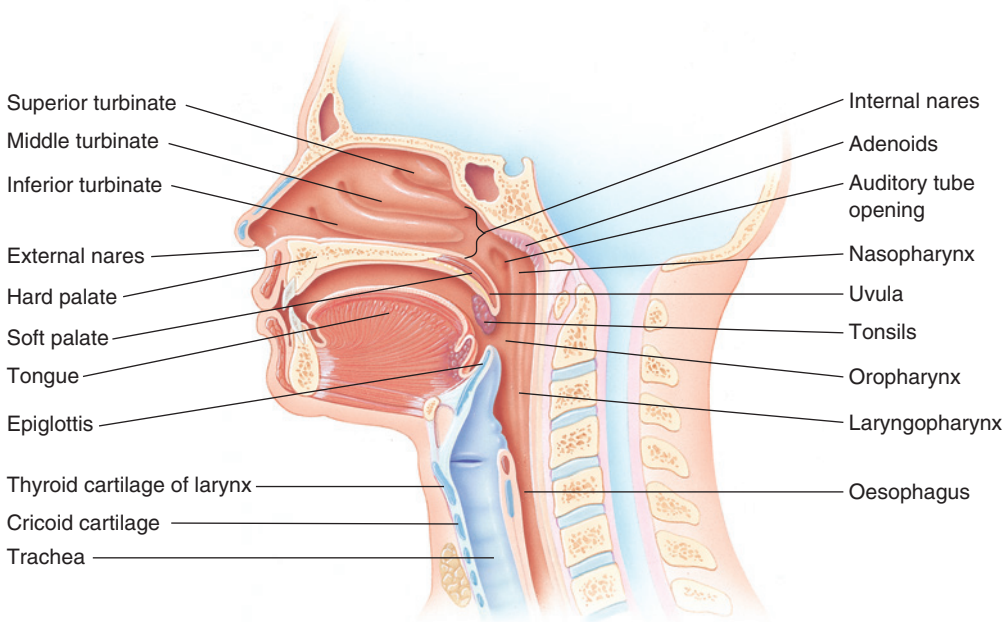


FIGURE 33.1 ■ The upper respiratory system

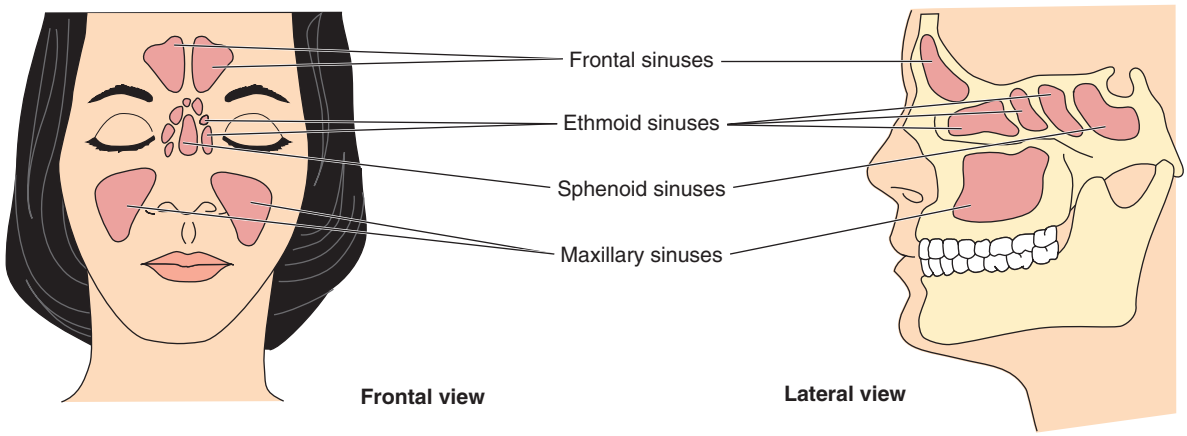


FIGURE 33.2 ■ Sinuses—frontal and lateral views

The sinuses

The nasal cavity is surrounded by paranasal sinuses (see Figure 33.2), located in the frontal, sphenoid, ethmoid and maxillary bones. Sinuses lighten the skull, assist in speech and produce mucus that drains into the nasal cavities to help trap debris.

The pharynx

The pharynx, a funnel-shaped passageway approximately 13 cm long, extends from the base of the skull to the level of the C6 vertebra. The pharynx serves as a passageway for both air and food. It is divided into three regions: the nasopharynx, the oropharynx and the laryngopharynx.

The nasopharynx serves only as a passageway for air. Located beneath the sphenoid bone and above the level of the soft palate, the nasopharynx is continuous with the nasal cavities. This segment is lined with ciliated epithelium, which continues to move debris from the nasal cavities to the pharynx. Masses of lymphoid tissue (the tonsils and adenoids) are located in the mucosa high in the posterior wall; these tissues trap and destroy infectious agents entering with the air. The auditory (eustachian) tubes also open into the nasopharynx, connecting it with the middle ear.

The oropharynx lies behind the oral cavity and extends from the soft palate to the level of the hyoid bone. It serves as a passageway for both air and food. An upward rise of the soft palate prevents food from entering the nasopharynx during swallowing. The oropharynx is lined with stratified squamous epithelium that protects it from the friction of food and damage from the chemicals found in food and fluids.

The laryngopharynx extends from the hyoid bone to the larynx. It is also lined with stratified squamous epithelium and serves as a passageway for both food and air. Air does not move into the lungs while food is being swallowed and moved into the oesophagus.

The larynx

The larynx is approximately 5 cm long. It opens superiorly at the laryngopharynx and is continuous inferiorly with the trachea. The larynx provides an airway and directs air and food into the proper passageway. As long as air is moving through the larynx, its inlet is open; however, the inlet closes during swallowing. The larynx also contains the vocal cords, necessary for voice production.

The larynx is formed by cartilages, connected by ligaments and membranes. The thyroid cartilage is formed by the fusion of two cartilages; the fusion point is visible as the Adam's apple. The cricoid cartilage lies below the thyroid cartilage; other pairs of cartilages form the walls of the larynx. The epiglottis, also a cartilage, normally projects upwards to the base of the tongue; however, during swallowing the larynx moves upwards and the epiglottis tips to cover the opening to the larynx. If anything other than air enters the larynx, a cough reflex expels the foreign substance before it can enter the lungs. This protective reflex does not work if the person is unconscious or has a decreased level of consciousness that results in an inability to manage this reflex.

The trachea

The trachea begins at the inferior larynx and descends anteriorly to the oesophagus to enter the mediastinum, where it divides to become the right and left primary bronchi of the lungs. The trachea is approximately 12 to 15 cm long and 2.5 cm in diameter. It contains 16 to 20 C-shaped rings of cartilage joined by connective tissue. The mucosa lining the trachea consists of pseudostratified ciliated columnar epithelium containing seromucous glands that produce thick mucus. Dust and debris in the inspired air are trapped in this mucus, moved towards the throat by the cilia and then either swallowed or coughed out through the mouth.

THE LOWER RESPIRATORY SYSTEM

The lower respiratory system includes the lungs and the bronchi (see Figures 33.3 and 33.4).

The lungs

The centre of the thoracic cavity is filled by the *mediastinum*, which contains the heart, great blood vessels, bronchi, trachea and oesophagus. The mediastinum is flanked on either side by the lungs (see Figure 33.3). Each lung is suspended in its own pleural cavity, with the anterior, lateral and posterior lung surfaces lying close to the ribs. The hilus, on the mediastinal surface of each lung, is where blood vessels of the pulmonary

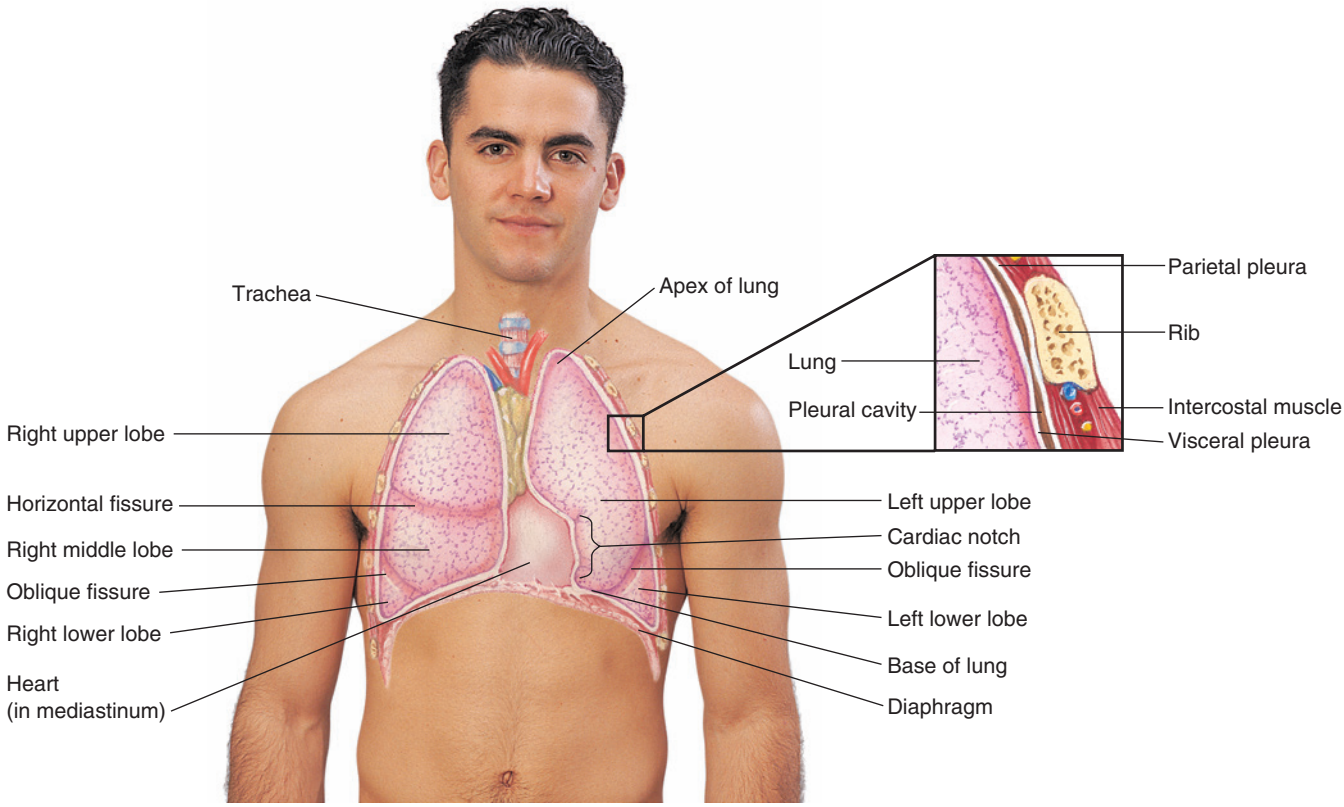


FIGURE 33.3 ■ The lower respiratory system, showing the location of the lungs, the mediastinum and layers of visceral and parietal pleura

and circulatory systems enter and exit the lungs. The primary bronchus also enters in this area. The apex of each lung lies just below the clavicle, whereas the base of each lung rests on the diaphragm. The lungs are elastic connective tissue, called stroma, and are soft and spongy.

The two lungs differ in size and shape. The left lung is smaller and has two lobes, whereas the right lung has three lobes. Each of the lung lobes contains a different number of bronchopulmonary segments, separated by connective tissue. There are 8 segments in the two lobes of the left lung and 10 segments in the three lobes of the right lung.

The vascular system of the lungs consists of the pulmonary arteries, which deliver blood to the lungs for oxygenation, and the pulmonary veins, which deliver oxygenated blood to the heart. Within the lungs, the pulmonary arteries branch into a pulmonary capillary network that surrounds the alveoli. Lung tissue receives its blood supply from the bronchial arteries and drains by the bronchial and pulmonary veins.

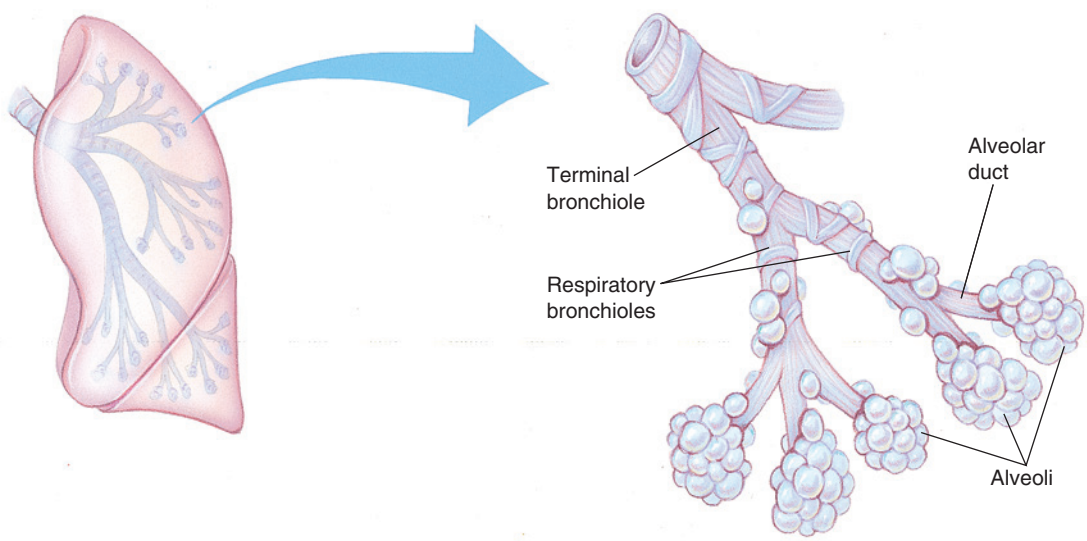


FIGURE 33.4 ■ Respiratory bronchi, bronchioles, alveolar ducts and alveoli

The pleura

The pleura is a double-layered membrane that covers the lungs and the inside of the thoracic cavities (see Figure 33.3). The parietal pleura lines the thoracic wall and mediastinum. It is continuous with the *visceral pleura*, which covers the external lung surfaces. The pleura produces pleural fluid, a lubricating, serous fluid that allows the lungs to move easily over the thoracic wall during breathing. The pleura's two layers also cling tightly together and hold the lungs to the thoracic wall. The structure of the pleura creates a slightly negative pressure in the pleural space (which is a potential rather than an actual space), necessary for lung function.

The bronchi and alveoli

The trachea divides into right and left primary bronchi; in comparison to the left primary bronchus, the right primary bronchus is shorter, wider and situated more vertically (making aspiration of foreign bodies into the right primary bronchus more likely). The point where the trachea divides is innervated with sensory neurons; activities such as tracheal suctioning may induce coughing and bronchospasm from stimulation of these neurons. These main bronchi subdivide into the secondary (lobar) bronchi, with the right middle lobe bronchus being smaller in diameter and length, and sometimes bending sharply near its bifurcation. The secondary bronchi then branch into the tertiary (segmental) bronchi and then into smaller and smaller bronchioles, ending in the terminal bronchioles (see Figure 33.4). These branching passageways collectively are called the bronchial or respiratory tree. From the terminal bronchioles, air moves into the respiratory bronchioles (air sacs), which further branch into alveolar ducts that lead to alveolar sacs and then to the alveoli. During inspiration, air enters the lungs through the primary bronchus and then moves through the increasingly smaller passageways of the lungs to the alveoli, where oxygen and carbon dioxide exchange occurs in the process of external respiration. During expiration, the carbon dioxide is expelled.

Alveoli cluster around the alveolar sacs, which open into a common chamber called the atrium. The adult lung has approximately 300 million alveoli, providing an enormous surface for gas exchange (Grossman & Mattson, 2013). Alveoli have extremely thin walls of a single layer of squamous epithelial cells over a very thin basement membrane. The external surface of the alveoli is covered with pulmonary capillaries. The alveolar and capillary walls form the respiratory membrane. Gas exchange across the respiratory membrane occurs by simple diffusion. The alveolar walls also contain cells that secrete a surfactant-containing fluid, necessary for maintaining a moist surface and reducing the surface tension of the alveolar fluid to help prevent collapse of the lungs.

The rib cage and intercostal muscles

The lungs are protected by the bones of the rib cage and the intercostal muscles. There are 12 pairs of ribs, which all articulate with the thoracic vertebrae (see Figure 33.5). Anteriorly, the first 7 ribs articulate with the body of the sternum. The 8th, 9th and 10th ribs articulate with the cartilage immediately above the ribs. The 11th and 12th ribs are called floating ribs, because they are unattached.

The sternum has three parts: the manubrium, the body and the xiphoid process. The junction between the manubrium and the body of the sternum is commonly called the angle of Louis. The depression above the manubrium is called the suprasternal notch.

The spaces between the ribs are called the intercostal spaces. Each intercostal space is named for the rib immediately above it (e.g. the space between the third and fourth ribs is designated as the third intercostal space). The intercostal muscles between the ribs, along with the diaphragm, are called the inspiratory muscles.

MECHANICS OF VENTILATION

Many factors affect ventilation and respiration. Those discussed here include changes in volume and capacity; air pressures; oxygen, carbon dioxide and hydrogen ion concentrations in the blood; airway resistance, lung compliance and elasticity; and alveolar surface tension.

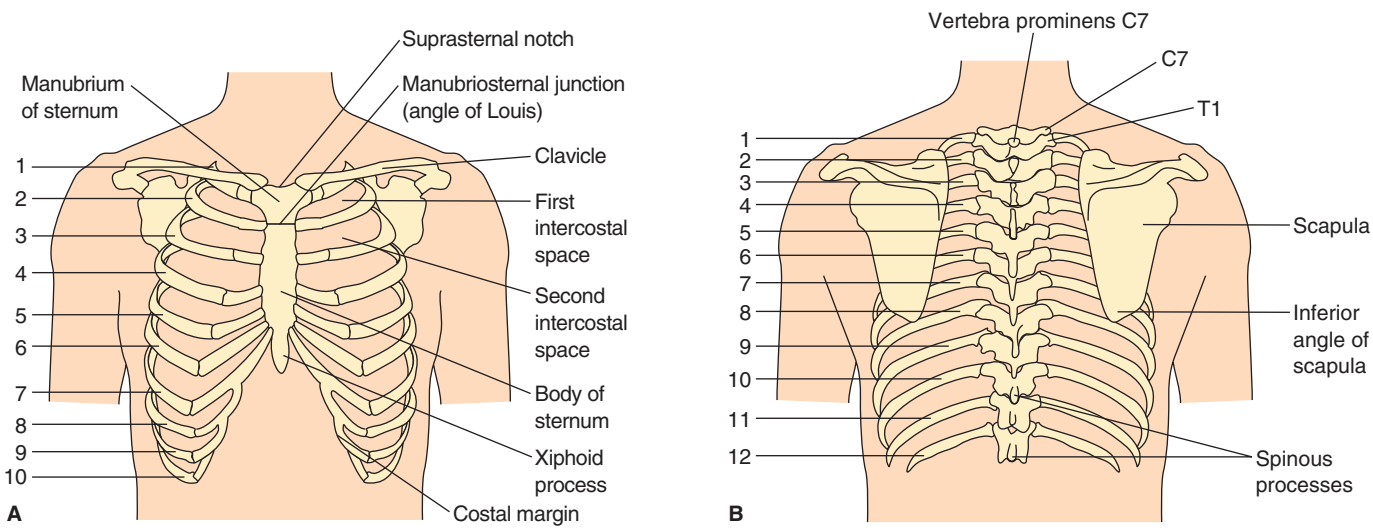


FIGURE 33.5 ■ *A*, Anterior rib cage, showing intercostal spaces. *B*, Posterior rib cage

Respiratory volume and capacity

Respiratory volume and capacity are affected by gender, age, weight and health status.

- **Tidal volume (TV)** is the amount of air (approximately 500 mL) moved in and out of the lungs with each normal, quiet breath.
- *Inspiratory reserve volume (IRV)* is the amount of air (approximately 2100 to 3100 mL, depending on body size) that can be inhaled forcibly over the tidal volume.
- *Expiratory reserve volume (ERV)* is the volume of air (approximately 1000 mL) that can be forced out over the tidal volume.
- The *residual volume* is the volume of air (approximately 1100 mL) that remains in the lungs after a forced expiration.
- **Vital capacity (VC)** refers to the sum of TV + IRV + ERV and is approximately 4500 mL in the healthy person.
- About 150 mL of air never reaches the alveoli (the amount remaining in the passageways) and is called anatomical dead space volume.

Pulmonary function tests measure these and other respiratory volumes and capacities and are discussed in Box 33.1.

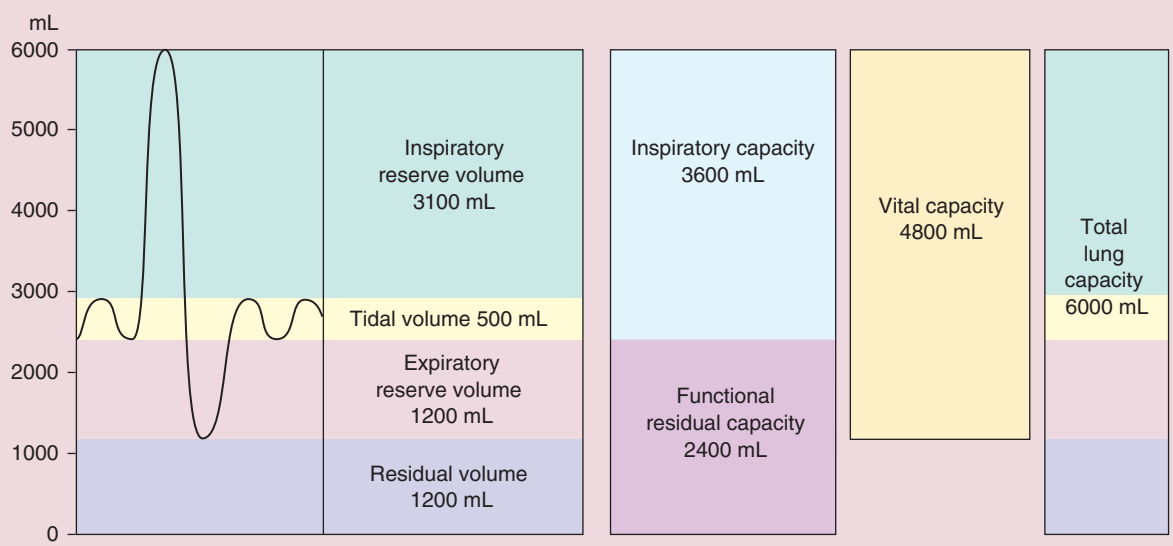
BOX 33.1 Pulmonary function tests

Pulmonary function tests (PFTs) are performed in a pulmonary function laboratory. After preparing the person, a nose clip is applied and the person breathes into a spirometer or body plethysmograph, a device for measuring and recording lung volume in litres versus time in seconds. The person is instructed how to breathe for specific tests; for example, to inhale as deeply as possible and then exhale to the maximal extent possible. Using measured lung volumes, respiratory capacities are calculated to assess pulmonary status. The specific values determined by PFTs and illustrated in the figure include the following.

- *Total lung capacity (TLC)* is the total volume of the lungs at their maximum inflation. Four values are used to calculate TLC:
  1. *tidal volume (TV)*, the volume inhaled and exhaled with normal quiet breathing (also called total volume)
  2. *inspiratory reserve volume (IRV), the maximum amount that can be inhaled over and above a normal inspiration*
  3. *expiratory reserve volume (ERV)*, the maximum amount that can be exhaled following a normal exhalation
  4. *residual volume (RV)*, the amount of air remaining in the lungs after maximal exhalation.
- *Vital capacity (VC)* is the total amount of air that can be exhaled after a maximal inspiration. It is calculated by adding together the IRV, TV and ERV.
- *Inspiratory capacity* is the total amount of air that can be inhaled following a normal quiet exhalation. It is calculated by adding the TV and IRV.
- *Functional residual capacity (FRC)* is the volume of air left in the lungs after a normal exhalation. The ERV and RV are added to determine the FRC.
- *Forced expiratory volume (FEV1)* is the amount of air that can be exhaled in 1 second.
- *Forced vital capacity (FVC)* is the amount of air that can be exhaled forcefully and rapidly after maximum air intake.
- *Minute volume (MV)* is the total amount or volume of air breathed in 1 minute.

In older people, residual capacity is increased and vital capacity is decreased. These age-related changes result from the following:

- calcification of the costal cartilage and weakening of the intercostal muscles, which reduce movement of the chest wall
- vertebral osteoporosis, which decreases spinal flexibility and increases the degree of kyphosis, further increasing the anterior–posterior diameter of the chest
- diaphragmatic flattening and loss of elasticity.



The relationship of lung volumes and capacities. Volumes (mL) shown are for an average adult male

Air pressures

Pulmonary ventilation depends on volume changes within the thoracic cavity. A change in the volume of air in the thoracic cavity leads to a change in the air pressure within the cavity. Because gases always flow along their pressure gradients, a change in pressure results in gases flowing into or out of the lungs to equalise the pressure.

The pressures normally present in the thoracic cavity are intrapulmonary and intrapleural pressure. The intrapulmonary pressure, within the alveoli of the lungs, rises and falls constantly as a result of the acts of ventilation (inhalation and exhalation). The intrapleural pressure, within the pleural space, also rises and falls with the acts of ventilation, but it is always less than (or negative to) the intrapulmonary pressure. Intrapulmonary and intrapleural pressures are necessary not only to expand and contract the lungs, but also to prevent their collapse.

Pulmonary ventilation has two phases: inspiration, during which air flows into the lungs; and expiration, during which gases flow out of the lungs. The two phases make up a single breath and normally occur from 12 to 20 times each minute. Inspiration occurs generally in a 1:2 ratio. A single inspiration lasts for approximately 1 to 1.5 seconds, whereas an expiration lasts for approximately 2 to 3 seconds.

During inspiration, the diaphragm contracts and flattens out to increase the vertical diameter of the thoracic cavity (see Figure 33.6). The external intercostal muscles contract, elevating the rib cage and moving the sternum forward to expand the lateral and anteroposterior diameter of the thoracic cavity, decreasing intrapleural pressure. The lungs stretch and the intrapulmonary volume increases, decreasing intrapulmonary pressure slightly below atmospheric pressure. Air rushes into the lungs as a result of this pressure gradient until the intrapulmonary and atmospheric pressures equalise.

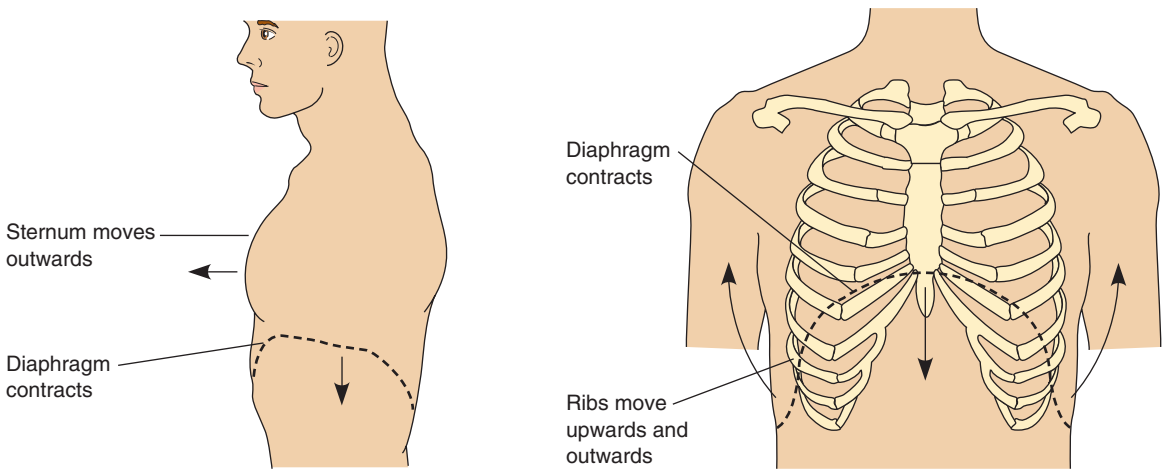


FIGURE 33.6 ■ Respiratory inspiration: lateral and anterior views. Note the volume expansion of the thorax as the diaphragm flattens

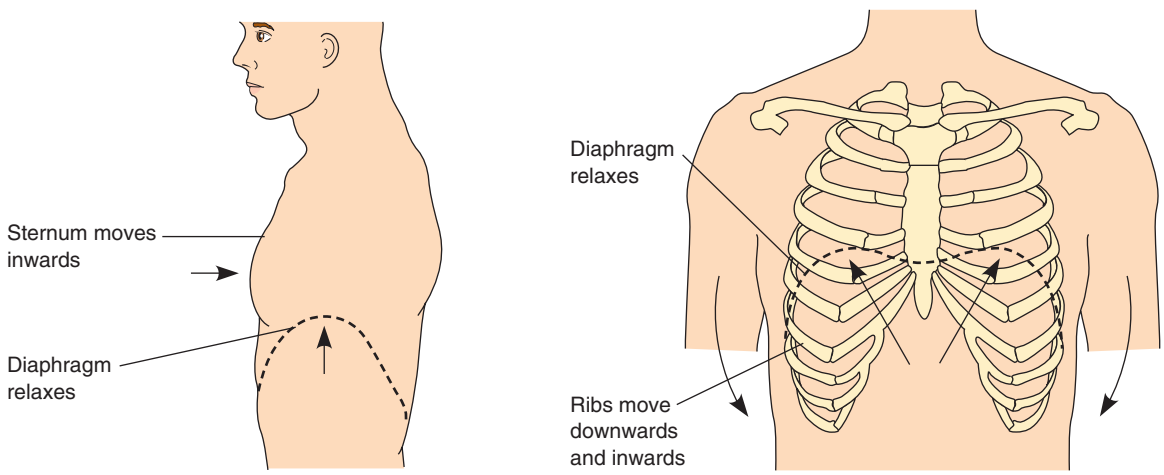


FIGURE 33.7 ■ Respiratory expiration: lateral and anterior views

Expiration is primarily a passive process that occurs as a result of the elasticity of the lungs (see Figure 33.7). The inspiratory muscles relax, the diaphragm rises, the ribs descend and the lungs recoil. Both the thoracic and the intrapulmonary pressures increase, compressing the alveoli. The intrapulmonary pressure rises to a level greater than atmospheric pressure and gases flow out of the lungs.

Oxygen, carbon dioxide and hydrogen ion concentrations

The rate and depth of respirations are controlled by respiratory centres in the medulla oblongata and pons of the brain and by chemoreceptors located in the medulla and in the carotid and aortic bodies. The centres and chemoreceptors respond to changes in the concentration of oxygen, carbon dioxide and hydrogen ions in arterial blood. For example, when carbon dioxide concentration increases or the pH decreases, the respiratory rate increases. This process is further described in Chapter 9.

Airway resistance, lung compliance and elasticity

Respiratory passageway resistance, lung compliance and lung elasticity also affect respiration.

- Respiratory passageway resistance is created by the friction encountered as gases move along the respiratory passageways, by constriction of the passageways (especially the larger bronchioles), by accumulations of mucus or infectious material, and by tumours. As resistance increases, gas flow decreases.
- **Lung compliance** depends on the elasticity of the lung tissue and the flexibility of the rib cage. Compliance is decreased by factors that decrease the elasticity of the lungs, block the respiratory passageways or interfere with movement of the rib cage.
- Lung elasticity is essential for lung distension during inspiration and lung recoil during expiration. Decreased elasticity from disease such as emphysema impairs respiration.

Alveolar surface tension

A liquid film of mostly water covers the alveolar walls. At any gas–liquid boundary, the molecules of liquid are more strongly attracted to each other than to gas molecules. This produces a state of tension, called surface tension, which draws the liquid molecules even more closely together. The water content of the alveolar film compacts the alveoli and aids in the lungs' recoil during expiration. In fact, if the alveolar film were pure water, the alveoli would collapse between breaths.

Surfactant, a lipoprotein produced by the alveolar cells, interferes with this adhesiveness of the water molecules, reducing surface tension and helping to expand the lungs. With insufficient surfactant, the surface tension forces can become great enough to collapse the alveoli between breaths, requiring tremendous energy to reinflate the lungs for inspiration.

GAS TRANSPORT AND AFFINITY

Blood gases

Gases are transported by the blood to provide cells with oxygen and to remove carbon dioxide produced during cellular activities.

Oxygen transport and unloading

Oxygen is carried in the blood either bound to haemoglobin or dissolved in the plasma. Oxygen is not very soluble in water, so almost all oxygen that enters the blood from the respiratory system is carried to the cells of the body by haemoglobin. This combination of haemoglobin and oxygen is called **oxyhaemoglobin**.

Each haemoglobin molecule is made of four polypeptide chains, with each chain bound to an iron-containing haem group. The iron groups are the binding sites for oxygen; each haemoglobin molecule can bind with four molecules of oxygen.

Oxygen binding is rapid and reversible. It is affected by temperature, blood pH, partial pressure of oxygen (PO_2), partial pressure of carbon dioxide (PCO_2) and serum concentration of an organic chemical called 2,3-DPG. These factors interact to ensure adequate delivery of oxygen to the cells.

The relative saturation of haemoglobin depends on the PO_2 of the blood, as illustrated in the oxygen–haemoglobin dissociation curve (see Figure 33.8).

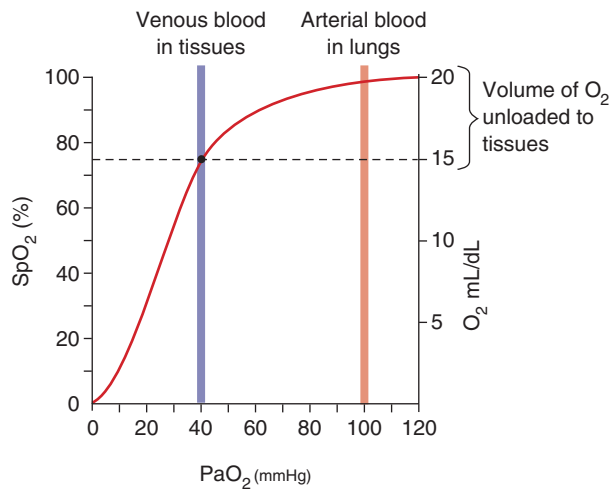


FIGURE 33.8 ■ Oxygen–haemoglobin dissociation curve. The percentage of O_2 saturation of haemoglobin and total blood oxygen volume are shown for different oxygen partial pressures (PO_2). Arterial blood in the lungs is almost completely saturated. During one pass through the body, about 25% of haemoglobin-bound oxygen is unloaded to the tissues. Thus, venous blood is still about 75% saturated with oxygen. The steep portion of the curve shows that haemoglobin readily off-loads or on-loads oxygen at PO_2 levels below about 50 mmHg

- Under normal conditions, the haemoglobin in arterial blood is 97.4% saturated with oxygen. Haemoglobin is almost fully saturated at a PO_2 of 70 mmHg. As arterial blood flows through the capillaries, oxygen is unloaded, so that the oxygen saturation of haemoglobin in venous blood is 75% under normal conditions.
- The affinity of oxygen and haemoglobin decreases as the temperature of body tissues increases above normal. As a result, less oxygen binds with haemoglobin and oxygen unloading is enhanced. Conversely, as the body is chilled, oxygen unloading is inhibited.
- The oxygen–haemoglobin bond is weakened by increased hydrogen ion concentrations. As blood becomes more acidotic, oxygen unloading to the tissues is enhanced. The same process occurs when the partial pressure of carbon dioxide increases because this decreases the pH.
- The organic chemical 2,3-DPG is formed in red blood cells and enhances the release of oxygen from haemoglobin by binding to it during times of increased metabolism (as when body temperature increases). This binding alters the structure of haemoglobin to facilitate oxygen unloading.

Carbon dioxide transport

Active cells produce about 200 mL of carbon dioxide each minute; this amount is exactly the same as that excreted by the lungs each minute. Excretion of carbon dioxide from the body requires transport by the blood from the cells to the lungs. Carbon dioxide is transported in three forms: dissolved in plasma, bound to haemoglobin and as bicarbonate ions in the plasma (the largest amount is in this form.)

The amount of carbon dioxide transported in the blood is strongly influenced by the oxygenation of the blood. When the PO_2 decreases, with a corresponding decrease in oxygen saturation, increased amounts of carbon dioxide can be carried in the blood. Carbon dioxide entering the systemic circulation from the cells causes more oxygen to dissociate from haemoglobin, in turn allowing more carbon dioxide to combine with haemoglobin and more bicarbonate ions to be generated. This situation is reversed in the pulmonary circulation, where the uptake of oxygen facilitates the release of carbon dioxide.

ASSESSING A PERSON'S RESPIRATORY FUNCTION

Respiratory system function is assessed by findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests. Sample documentation of an assessment of the respiratory system is included in the box below.

Health assessment interview

A health assessment interview to determine problems with respiratory structure and function may be conducted during a health screening, may focus on a chief complaint (such as shortness of breath) or may be part of a total health assessment. If the person has a problem with respiratory function, analyse its onset, characteristics, course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, ask the person:

- Describe the problems you are having with your breathing. Is your breathing more difficult if you lie flat? Is it painful to breathe in or out?
- When did you first notice that your cough was becoming a problem? Do you cough up mucus? What colour is the mucus?
- Have you had nosebleeds in the past?

During the interview, carefully observe the person for difficulty in breathing, pausing to breathe in the middle of a sentence, hoarseness, changes in voice quality, audible wheeze and cough. Ask about present health status, medical history, family health history and risk factors for illness.

To determine present health status, ask about pain in the nose, throat or chest. Information about cough includes what type of cough, when it occurs and how it is relieved. The person should describe any sputum associated with the cough. Is the person experiencing any dyspnoea (difficult or laboured breathing)? How is the dyspnoea associated with activity levels and time of day? Is the person having chest pain? How is this related to activity and time of day? Note the severity, type and location of the pain. Explore problems with swallowing, smelling or taste. Also ask about nosebleeds and nasal or sinus stuffiness or pain, and about current medication use, aerosols or inhalants, and oxygen use.

SAMPLE DOCUMENTATION
Assessment of respiratory function

14/4/2014 NURS 1100	57-year-old male, history of smoking two packets cigarettes/day for 37 years; continues to smoke despite previous discussions. Works as a fibreglass boat builder. No family history of cancer or TB. States he has trouble breathing, especially at night. Often sleeps on a recliner 'to breathe better'. Complains of a cough, but denies sputum production. Diagnosed 3 years ago with emphysema. Colour of face is dusky red. Fingernails pink. No digital clubbing observed. Respirations 30/min, unlaboured, regular. (Respiration rate varies from 26 to 32 on visits to the clinic.) SpO_2 91%. Afebrile, heart rate 104 bpm; BP 134/78. Thoracic assessment L = R, intercostal recession, barrel chest, and use of accessory muscles of respiration. Diminished lung sounds noted bilaterally in lower lobes. Crackles present in upper lobes, not cleared by coughing. Discussed with RMO. For oxygen via nasal prongs at 2 Lpm. Will review within a few hours. RN Nguyen L NGUYEN RN

TABLE 33.1 Age-related changes in the respiratory system

AGE-RELATED CHANGE	SIGNIFICANCE
• ↓elastic recoil of lungs during expiration because of less elastic collagen and elastin. • Loss of skeletal muscle strength in the thorax and diaphragm. • Alveoli are less elastic, more fibrotic and have fewer functional capillaries. • Cough is less effective. • PO_2 reduces as much as 15% by age 80.	The older adult often has an increased anterior–posterior chest diameter, with kyphosis and barrel chest. There is a reduction in vital capacity and an increase in residual volume, with decreased effectiveness in coughing up phlegm or sputum. All of these changes greatly increase the risk of respiratory infections (such as pneumonia), especially if the person becomes immobile. They also mean that respiratory infections are more difficult to treat.

Document past medical history by asking questions about a history or family history of allergies, asthma, bronchitis, emphysema, pneumonia, tuberculosis or congestive heart failure. Other questions include a history of surgery or trauma to the respiratory structures and a history of other chronic illnesses such as cancer, kidney disease and heart disease and medications used. The person's personal lifestyle, environment and occupation may provide clues to risk factors for actual or potential health problems. Ask the person about a history of smoking and/or exposure to environmental chemicals (including smog), dust, vapours, animals, coal dust, asbestos, fumes or pollens. Other risk factors include a sedentary lifestyle and obesity. Also ask the person about use of alcohol and illicit substances.

Physical assessment

Physical assessment of the respiratory system may be performed either as part of a total assessment or alone for a person with known or suspected problems. The techniques used to assess the respiratory system are inspection, palpation, percussion and auscultation. In addition, note the person's level of consciousness, restlessness and anxiety level, and assess the colour of the lips and nail beds. Normal age-related findings for the older adult are summarised in Table 33.1.

The room should be warm and well lit. Ask the person to remove all clothing above the waist; give them a gown to wear during the examination. Conduct the examination with the person in the sitting position. Prior to the examination, collect all necessary equipment and explain the techniques to the person to decrease anxiety.

Diagnostic tests

The results of diagnostic tests of respiratory function are used to support the diagnosis of a specific disease, to provide information to identify or modify the appropriate medications or therapy used to treat the disease, and to help nurses monitor the person's response to treatment and nursing care interventions. Diagnostic tests to assess the structures and functions of the respiratory system are described in the 'Diagnostic tests' box below and summarised in the bulleted list below. More information is included in the discussion of specific disorders in Chapters 34, 35 and 36.

- Sputum tests include a culture and sensitivity to identify organisms causing infections as well as the most effective antibiotic to treat the infection, an acid-fast smear and culture to identify the tuberculosis bacillus and cytology to identify malignancies (see Procedure 33.1).
- Arterial blood gases are conducted to evaluate alterations in acid–base balances.
- Pulse oximetry is used to evaluate or monitor the oxygen saturation of the blood.
- Many different radiological examinations are used to diagnose respiratory disorders, including a chest x-ray to evaluate structures and tissues, a computed tomography (CT) scan to differentiate pathological conditions, magnetic resonance imaging (MRI) to more accurately identify abnormal masses and fluid accumulation, a positron emission tomography (PET) to identify lung cancers and a pulmonary angiogram to identify various disorders including pulmonary emboli and emphysema.
- A bronchoscopy is a direct visualisation of the larynx, trachea and bronchi. During the test, lesions can be identified, foreign bodies or mucus plugs removed, and tissue taken for biopsy. In addition, a biopsy of lung tissue may be done through an incision through the chest wall.
- A thoracentesis, when done for diagnostic purposes, is conducted to obtain a specimen of pleural fluid.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, for assessing for medication use that may affect the outcome of the tests, for supporting the person during the examination as necessary, for documenting the procedures as appropriate and for monitoring the results of the tests.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of the adult. During the health assessment interview, ask about family members with health problems affecting respiratory function. In addition, ask about a family history of emphysema, asthma, cystic fibrosis or lung cancer. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the 'Genetic considerations' box). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

PROCEDURE 33.1 Obtaining a sputum specimen

GATHER SUPPLIES

- Sterile sputum container, specimen cup or mucus trap
- Mouth care supplies
- Sterile suction kit, if necessary
- Gloves

BEFORE THE PROCEDURE

If the sputum specimen is to establish the initial diagnosis, obtain the specimen before starting oxygen and/or antibiotic therapy. Antibiotics reduce the bacterial count, making it difficult to identify the infecting organism. Oxygen therapy dries mucous membranes, making it more difficult to obtain a specimen. Unless otherwise instructed, obtain the specimen early in the morning, just after awakening. Respiratory secretions tend to pool during sleep; it is easier to obtain a specimen before normal coughing and daily activity have cleared them.

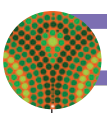
Provide for privacy and explain the procedure. Emphasise the importance of coughing deeply to obtain sputum from the lower respiratory tract, avoiding expectoration of saliva. Increasing fluid intake prior to obtaining the specimen can help liquefy secretions, making them easier to expectorate.

DURING THE PROCEDURE

1. Provide for mouth care prior to obtaining the specimen to reduce contamination by oral flora.
2. Instruct to cough deeply several times, expectorating mucus into the container.
3. Close the container securely.
4. Label the container according to the institution's requirements, usually with the person's name, date of birth, hospital identification number, time and date, signature of specimen collector and any special conditions, such as antibiotic or oxygen therapy. Enclose specimen container in a specimen bag and send to the laboratory with the pathology request slip, or refrigerate as ordered to preserve the specimen.
5. To obtain a specimen by suctioning:
   - Provide mouth care.
   - Obtain a sterile mucus trap. Using aseptic technique, attach the trap to the suction apparatus between the suction catheter and tubing.
   - Pre-oxygenate for suctioning as needed.
   - Perform tracheal suctioning using aseptic technique via the nasotracheal route, endotracheal tube or tracheostomy. Lubricate the catheter with sterile normal saline. Do not apply suction as the catheter is being inserted into the trachea; apply suction for no longer than 10 seconds while withdrawing the catheter.
   - If the secretion remains in the tubing of the trap, suction sterile water into the trap to encourage the secretion into the container section.
   - Detach the mucus trap; close and label. Reattach the catheter and clear the suction catheter and tubing with normal saline after removing the mucus trap. Dispose of equipment appropriately.
   - Label the specimen as identified above before sending to the laboratory.
6. A sputum specimen also may be obtained during a bronchoscopy procedure.

AFTER THE PROCEDURE

Provide mouth care as needed. Teach the importance of completing all ordered antibiotic prescriptions to ensure complete eradication of microorganisms. Document the time and date that the specimen was obtained; and note colour, consistency and odour of sputum.



GENETIC CONSIDERATIONS Respiratory disorders

- Deficiency of alpha-1 antitrypsin (a protein that protects the body from damage by its immune cells) is caused by a mutation of a gene located on chromosome 14. Deficiency of this protein leaves the lung susceptible to emphysema.
- Asthma is a respiratory disease which affects 10% of the population. Although mortality has declined substantially in the past 20 years, it remains high in Australia compared with international rates. Unacceptably, over 350 people died from asthma in 2011 (Poulos et al., 2014). It is an obstructive respiratory condition and is associated with inheritable factors.
- The prevalence of cystic fibrosis in Australia in 2013 was 3235, with 92 new cases diagnosed, and mortality was 38 (Cystic Fibrosis Australia, 2015). All gene defects result in defective transport of chloride and sodium by epithelial cells. As a result, the amount of sodium chloride is increased in body secretions. Thick mucus is produced that clogs the lungs, leads to infection and blocks pancreatic enzymes from reaching the intestines to digest food.
- Lung cancer is the fifth most common cancer in Australia and accounts for almost 19% of all cancer deaths (Cancer Australia, 2015). A familial history of lung cancer increases the risk of developing lung cancer, and small-cell lung cancer has a definite genetic component. In addition, researchers have found that people with lung cancer who never smoked are more likely than smokers to have one of two genetic mutations linked to the disease.

DIAGNOSTIC TESTS The respiratory system

NAME OF TEST Sputum studies
- Culture and sensitivity
- Acid-fast bacilli smear and culture
- Cytology

PURPOSE AND DESCRIPTION Culture and sensitivity of a single sputum specimen is performed to diagnose bacterial infections, identify the most effective antibiotic and evaluate treatment.

Sputum is examined for presence of acid-fast bacillus, specifically tuberculosis. A series of three early-morning sputum specimens is used.

Sputum is examined for presence of abnormal (malignant) cells. A single sputum specimen is collected in a special container of fixative solution.

RELATED NURSING CARE See Procedure 33.1 for obtaining a sputum specimen. Sputum specimens may also be obtained during bronchoscopy (described later) if the person is unable to provide a specimen.

NAME OF TEST Arterial blood gases (ABGs)

PURPOSE AND DESCRIPTION This test of arterial blood is performed to assess alterations in acid–base balance caused by a respiratory disorder, a metabolic disorder or both. A pH of less than 7.35 indicates acidosis and a pH of more than 7.45 indicates alkalosis (see Chapter 9). To determine a respiratory cause, assess the $PaCO_2$: If pH is decreased and $PaCO_2$ is increased, respiratory acidosis is indicated.

Normal values:
pH: 7.35–7.45
$PaCO_2$: 35–45 mmHg
PaO_2: 75–100 mmHg
HCO_{3-}^{-}: 22–26 mEq/L
BE: ± 2 mEq/L

RELATED NURSING CARE Arterial blood is collected in a heparinised needle and syringe. Sample is placed on an ice bag and taken immediately to the lab. If the person is receiving oxygen, you are required to indicate this on the lab slip. Apply pressure to puncture site for 2–5 minutes post test or longer if needed. Do not collect blood from the same arm used for an IV infusion as this can make the results of the test inaccurate.

NAME OF TEST Pulse oximetry

PURPOSE AND DESCRIPTION This non-invasive test is used to evaluate or monitor oxygen saturation of the blood. A device that uses infrared light is attached to an extremity (most commonly the finger, but can also be the toe, earlobe, nose or forehead) and light is passed through the tissues or reflected off bony structures.

Normal value: 95–100%

RELATED NURSING CARE Assess for factors that may alter findings, including faulty placement, movement, dark skin colour, acrylic nails, ambient light, peripheral hypothermia, and peripheral vasoconstriction.

NAME OF TEST Chest x-ray

PURPOSE AND DESCRIPTION Chest x-rays are used to identify abnormalities in chest structure and lung tissue, for diagnosis of diseases and injuries of the lungs, and to monitor treatment.

RELATED NURSING CARE Remove any metal objects around area such as jewellery or brassieres containing underwire.

NAME OF TEST Computed tomography (CT)

PURPOSE AND DESCRIPTION CT of the thorax may be performed when x-rays do not show some areas well, such as the pleura and mediastinum. It is also performed to differentiate pathological conditions (such as tumours, abscesses and aortic aneurysms), to identify pleural effusion and enlarged lymph nodes and to monitor treatment. Images are shown in cross-section.

RELATED NURSING CARE Need to assess whether the person is able to lie still for extended periods and is not claustrophobic.

NAME OF TEST Magnetic resonance imaging (MRI)

PURPOSE AND DESCRIPTION An MRI of the thorax is used to diagnose alterations in lung tissue more difficult to visualise by CT scan and to identify abnormal masses and fluid accumulation.

RELATED NURSING CARE Assess for any metallic implants (such as pacemaker, pacemaker wires, implants) or history of metal shavings etc in eyes. Also assess for any metal worn on the body. Test will not be performed if present.

(continued)

DIAGNOSTIC TESTS The respiratory system (continued)

NAME OF TEST Positron emission tomography (PET)

PURPOSE AND DESCRIPTION This relatively non-invasive test, when used to examine the lungs, is performed to identify lung nodules (cancers). The person is given a radioactive substance and cross-sectional images are displayed on a computer. Radiation from PET is only 25% of that from a CT scan.

RELATED NURSING CARE No alcohol, coffee or tobacco is allowed for 24 hours prior to the test. Encourage increased fluid intake post-test to help eliminate the radioactive material.

NAME OF TEST Pulmonary angiography

PURPOSE AND DESCRIPTION This test is performed to identify pulmonary emboli, tumours, aneurysms, vascular changes associated with emphysema and pulmonary circulation. A catheter is inserted into the brachial or femoral artery, threaded into the pulmonary artery and dye is injected. Electrocardiograph leads are applied to the chest for cardiac monitoring. Images of the lungs are taken.

RELATED NURSING CARE Monitor injection site and pulses distal to the site after the test.

NAME OF TEST Pulmonary ventilation–perfusion scan (V/Q scan)

PURPOSE AND DESCRIPTION This test is performed to measure breathing (ventilation) and circulation (perfusion) in all parts of the lungs. A perfusion scan is performed by injecting radioactive albumin into a vein and scanning the lungs. A ventilation scan is performed by scanning the lungs as the person inhales radioactive gas. A decreased uptake of radioisotope during the perfusion scan indicates a blood flow problem, such as from a pulmonary embolus or pneumonitis. A decreased uptake of gas during the ventilation scan may indicate airway obstruction, pneumonia or chronic obstructive pulmonary disease (COPD).

RELATED NURSING CARE No special preparation is needed other than ensuring the person has a patent intravenous catheter.

NAME OF TEST Bronchoscopy

PURPOSE AND DESCRIPTION A bronchoscopy is the direct visualisation of the larynx, trachea and bronchi through a bronchoscope to identify lesions, remove foreign bodies and secretions, obtain tissue for biopsy and improve tracheobronchial drainage (see Figure 33.9). During the test, a catheter brush or biopsy forceps can be passed to obtain secretions or tissue for examination for cancer.

RELATED NURSING CARE Provide routine preoperative care as ordered. Bronchoscopy is an invasive procedure requiring conscious sedation or anaesthesia. *Care provided prior to the procedure is similar to that provided before many minor surgical procedures.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Fibre-optic bronchoscopy requires 30 to 45 minutes to complete. It may be done at the bedside, in a special procedure room or in the surgical suite.
- The procedure usually causes little pain or discomfort because an anaesthetic is given. You will be able to breathe during the bronchoscopy.
- Some voice hoarseness and a sore throat are common following the procedure. Throat lozenges or warm saline gargles may help relieve discomfort.
- You may develop a mild fever within the first 24 hours following the procedure. This is a normal response.
- Persistent cough, bloody or purulent sputum, wheezing, shortness of breath or chest pain may indicate a complication. Notify your doctor if they develop.

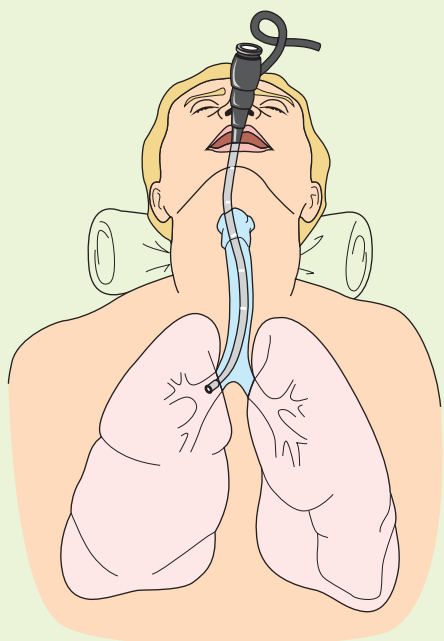


FIGURE 33.9 ■ Fibre-optic bronchoscopy

DIAGNOSTICTESTS The respiratory system (continued)

NAME OF TEST Lung biopsy

PURPOSE AND DESCRIPTION Performed to obtain tissue to differentiate benign from malignant tumours of the lungs. May be performed during a bronchoscopy or by surgical procedure.

RELATED NURSING CARE Same as for bronchoscopy or the same as for a thoracotomy (incision through the chest wall) if a surgical biopsy is performed.

NAME OF TEST Thoracentesis

PURPOSE AND DESCRIPTION Performed to obtain a specimen of pleural fluid for diagnosis (and used as a procedure to remove pleural fluid or instil medication). A large-bore needle is inserted through the chest wall and into the pleural space. Following the procedure, a chest x-ray is taken to check for a pneumothorax.

RELATED NURSING CARE Nursing care of the person having a thoracentesis is provided in Chapter 35.

RESPIRATORY ASSESSMENTS

Technique/normal findings	Abnormal findings
Nasal assessment Inspect the nose for changes in size, shape or colour. *The nose should be midline in the face, of the same colour as the face and the nares should be symmetrical.*	■ The nose may be asymmetrical as a result of previous surgery or trauma. ■ The skin around the nostrils may be red and swollen with allergies or upper respiratory infections.
Inspect the nasal cavity. Use an otoscope with a broad, short speculum. Gently insert the speculum into each of the nares and assess the condition of the mucous membranes and the turbinates. *The septum should be midline with pink mucosa and without drainage.*	■ The septum may be deviated. ■ Perforation of the septum may occur with chronic cocaine abuse. ■ Red mucosa indicates infection. ■ Purulent drainage indicates nasal or sinus infection. ■ Allergies may be indicated by watery nasal drainage, pale turbinates and polyps on the turbinates.
Assess ability to smell (cranial nerve I, olfactory). Ask the person to breathe through one nostril while pressing the other one closed. Ask the person to close their eyes. Place a substance with an aromatic odour under the person's nose (use ground coffee or alcohol) and ask the person to identify the odour. Test each nostril separately. *This test is usually done only if the person has problems with the sense of smell, but the person should be able to distinguish different odours.*	■ Changes in the ability to smell may be the result of damage to the olfactory nerve or to chronic inflammation of the nose. ■ Zinc deficiency may cause a loss of the sense of smell.
Sinus assessment Palpate the frontal and maxillary sinuses. *The sinuses should not be tender to palpation.*	■ Frontal and maxillary sinuses are tender to palpation with allergies or sinus infections.
Thoracic assessment Assess respiratory rate. *The normal respiratory rate is 12 to 20 breaths per minute.*	■ **Tachypnoea** (rapid respiratory rate) is seen in atelectasis (collapse of lung tissue following obstruction of the bronchus or bronchioles), pneumonia, asthma, pleural effusion, pneumothorax, congestive heart failure, anxiety and in response to pain. ■ Damage to the brainstem from a stroke or head injury may result in either tachypnoea or **bradypnoea** (low respiratory rate). ■ Bradypnoea is seen with some circulatory disorders, lung disorders and as a side effect of some medications. ■ **Apnoea**, cessation of breathing lasting from a few seconds to a few minutes, may occur following a stroke or head trauma, as a side effect of some medications or following airway obstruction.

Technique/normal findings	Abnormal findings
Inspect the anteroposterior diameter of the chest. *The anteroposterior diameter of the chest should be less than the transverse diameter. Normal ratio is 1:2.*	■ The anteroposterior diameter is equal to the transverse diameter in barrel chest, which typically occurs with emphysema.
Inspect for intercostal retraction or bulging. *There should be no retraction or bulging.*	■ Retraction of intercostal spaces may be seen in asthma. ■ Bulging of intercostal spaces may be seen in pneumothorax.
Inspect and palpate for chest expansion. Place your hands with the fingers spread apart palm down on the person's posterolateral chest. Gently press the skin between your thumbs (see Figure 33.10). Ask the person to breathe deeply. As the person inhales, watch your hands for symmetry of movement. *Chest expansion should be bilaterally symmetrical with the examiner's hands moving 5 to 10 cm apart.*	■ Thoracic expansion is decreased on the affected side in atelectasis, pneumonia, pneumothorax and pleural effusion. ■ Bilateral chest expansion is decreased in emphysema.

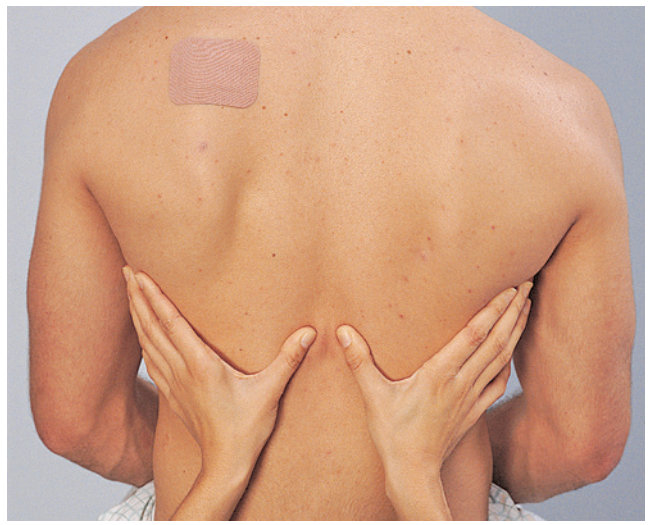


FIGURE 33.10 ■ Palpating for chest expansion

Gently palpate the location and position of the trachea. *The trachea should be midline.*	■ The trachea shifts to the unaffected side in pleural effusion and pneumothorax and shifts to the affected side in atelectasis.
Palpate for tactile fremitus. Ask the person to say 'ninety-nine' as you palpate at three different levels for a vibratory sensation called tactile fremitus, which occurs as sound waves from the larynx travel through patent bronchi and lungs to the chest wall. *Fremitus is symmetrical and easily palpated in the upper regions of the lungs.*	■ Tactile fremitus is decreased in atelectasis, emphysema, asthma, pleural effusion and pneumothorax. It is increased in pneumonia if the bronchus is patent.
Percuss the lungs for dullness over shoulder apices and over anterior, posterior and lateral intercostal spaces (see Figure 33.11). *The normal percussion tone over normal lung tissue is resonance.*	■ Dullness is heard in people with atelectasis, lobar pneumonia and pleural effusion. ■ Hyper-resonance is heard in those with chronic asthma, emphysema and pneumothorax.

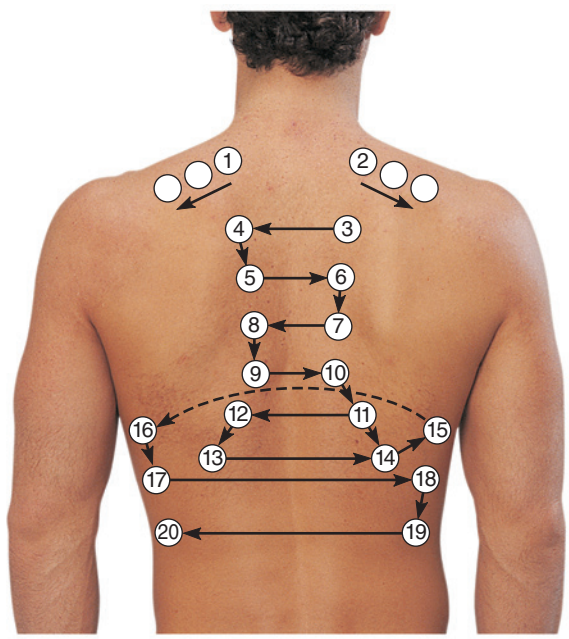


FIGURE 33.11 ■ Sequence for lung percussion

Technique/normal findings	Abnormal findings
Percuss the posterior chest for diaphragmatic excursion. Systematic percussion of the posterior chest from a level of lung resonance to the level of diaphragmatic dullness reveals *diaphragmatic excursion*, a measurement of the level of the diaphragm. First percuss downward over the posterior thorax while the person exhales fully and holds the breath. Mark the spot at which the sound changes from resonant to dull. Then ask the person to inhale and hold the breath while you percuss downward again to note the descent of the diaphragm. Again mark the spot where the sound changes. *Measure the difference in diaphragmatic excursion, which normally varies from about 3 to 5 cm (see Figure 33.12).*	■ Diaphragmatic excursion is decreased in emphysema, ascites, on the affected side in pleural effusion and in pneumothorax. ■ A high level of dullness or a lack of excursion may indicate atelectasis or pleural effusion.

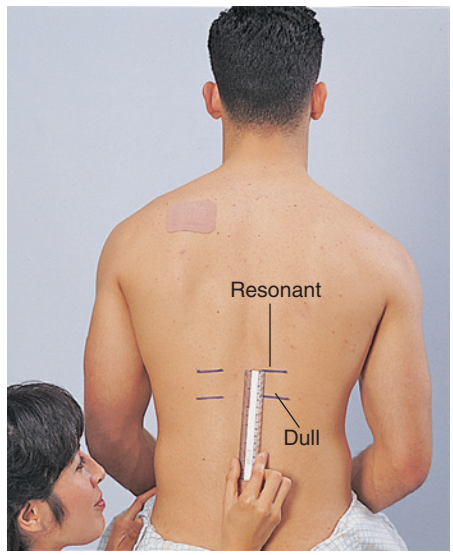


FIGURE 33.12 ■ Measuring diaphragmatic excursion

Breath sound assessment

Auscultate the lungs for breath sounds with the diaphragm of the stethoscope by having the person take slow deep breaths through the mouth. Listen over anterior, posterior and lateral intercostal spaces (see Figure 33.13). *The three different types of normal breath sounds are vesicular, bronchovesicular and bronchial (see Table 33.2).*

- Bronchial breath sounds (expiration > inspiration) and bronchovesicular breath sounds (inspiration = expiration) are heard over lungs filled with fluid or solid tissue.
- Breath sounds are decreased or diminished over atelectasis, emphysema, asthma, pleural effusion and pneumothorax.
- Breath sounds are increased over lobar pneumonia.
- Breath sounds are absent over collapsed lung, surgical removal of lung, pleural effusion and primary bronchus obstruction.

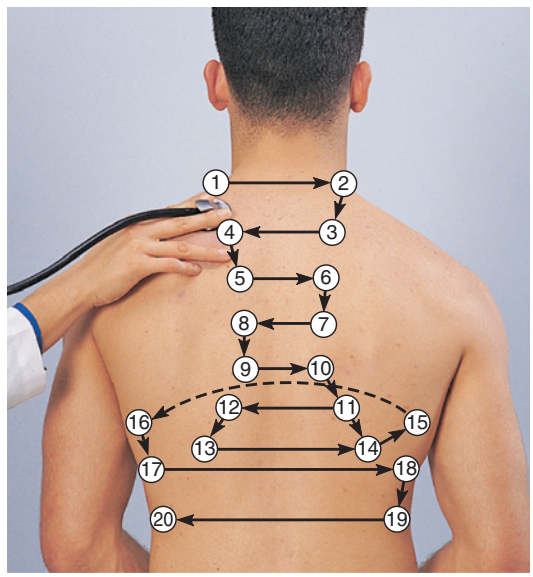


FIGURE 33.13 ■ Sequence for lung auscultation

Auscultate for crackles, wheezes and friction rubs. If crackles or wheezes are heard, ask the person to cough and note if adventitious sound is cleared. *Normally, there are no crackles, wheezes or friction rubs.*

- **Crackles** (short, discrete, crackling or bubbling sounds) may be noted in pneumonia, bronchitis and congestive heart failure.
- **Wheezes** (continuous, musical sounds) may be heard in people with bronchitis, emphysema and asthma.
- A **friction rub** is a loud, dry, creaking sound that indicates pleural inflammation.

Auscultate voice sounds where any abnormal breath sound is noted by having the person say 'ninety-nine' (bronchophony); whisper 'one, two, three' (whispered pectoriloquy); and say 'ee' (egophony). *Normally, these sounds are heard by the examiner, but are muffled.*

- Voice sounds are decreased or absent over areas of atelectasis, asthma, pleural effusion and pneumothorax.
- Voice sounds are increased and clearer over lobar pneumonia.
- When testing egophony, the sound becomes louder and changes to 'a' over areas of consolidation or compression.

TABLE 33.2 Normal breath sounds

TYPE OF BREATH SOUND	CHARACTERISTICS
Vesicular	• Soft, low-pitched, gentle sounds • Heard over all areas of the lungs except the major bronchi • Have a 3:1 ratio for inspiration and expiration, respectively
Bronchovesicular	• Medium pitch and intensity of sounds • Have a 1:1 ratio, with inspiration and expiration being equal in duration • Heard anteriorly over the primary bronchus on each side of the sternum and posteriorly between the scapulae
Bronchial	• Loud, high-pitched sounds • Gap between inspiration and expiration • Have a 2:3 ratio for inspiration and expiration, respectively • Heard over the manubrium

TRANSLATION TO PRACTICE

Evidence-based practice: nursing care of dyspnoea, the 6th vital sign in individuals with COPD

Research by Bailey et al. (2013) discussed the concept that in individuals with a chronic obstructive respiratory disease, the type, quality and duration of dyspnoea should be quantified and considered as the 6th vital sign. The recommendation is that the characteristics of a person's dyspnoea can convey a greater understanding of the respiratory compromise that person may be experiencing.

IMPLICATIONS FOR NURSING

Historically, measuring vital signs called for the quantification of an individual's respiratory rate as the lone parameter recorded for the respiratory system. Bailey et al.'s research indicated that collecting and undertaking a more comprehensive analysis of the characteristics of a person's dyspnoea may encourage faster or different intervention. Nurses need to be aware of the presence of dyspnoea rating scales such as the Medical Research Council (MRC) scale so that acute deterioration may be identified more readily.

CRITICAL THINKING IN PERSON-CENTRED CARE

1 In this study, the findings indicated that enhanced identification of deterioration and improved management may result from a formal assessment and documentation of a person's dyspnoea. How will you accommodate these findings into the health management plan of people with respiratory compromise in the future?
2 How will the findings of this study impact on the assessment of and care that you give to a person with chronic obstructive pulmonary disease?

FOCUS ON CULTURAL DIVERSITY

Are we working cooperatively with our Pacific partners to control multidrug-resistant tuberculosis at our borders?

An article by Kirby (2013) on multidrug-resistant tuberculosis on Australia's northern border, called 'Extensively drug-resistant tuberculosis hovers threateningly at Australia's door', discussed the spread of multidrug-resistant tuberculoses from Papua New Guinea to the Torres Strait and Australia's northern coast.

Implications for nursing

The Department of Health (2015) reported that 88% of all cases of people newly diagnosed with tuberculosis (TB) in 2013 occurred in individuals born overseas. The rate of TB in overseas-born individuals is 19 times higher than for those born in Australia (Toms et al., 2015). Although there has been a 45% reduction is mortality since 1990, there is still a worrying prevalence of TB and multidrug-resistant TB (MDR-TB) in Papua New Guinea. World Health Organization (WHO) reports suggest that up to 24% of people who have been treated for TB have developed MDR-TB. In comparison, Australia's MDR-TB rate is estimated at between 2.3% and 4.2% (WHO, 2015).

Critical thinking in person-centred care

1. How could you find out more information about the epidemiology of tuberculosis within the local region?
2. How might cultural and language diversity complicate care of persons requiring treatment for tuberculosis?
3. How might people with cultural and language diversity contribute to multidrug-resistant tuberculosis following discharge? Identify mechanisms and interventions to avoid this occurrence.

CHAPTER HIGHLIGHTS

- The respiratory system is divided into the upper respiratory system and the lower respiratory system.
- The upper respiratory system functions mostly as protection, filtration and the conductive section directing air to the lower respiratory system.
- The lower system has some conducting function but is largely responsible for provision of oxygen and elimination of carbon dioxide.
- Critical factors affecting ventilation and respiration include changes in volume and capacity; air pressures; oxygen, carbon dioxide and hydrogen ion concentrations in the blood; airway resistance, lung compliance and elasticity; and alveolar surface tension.
- Important components of a health history for an individual with a respiratory condition should include questions which can elicit answers to onset, characteristics, course, severity, precipitating and relieving factors, and any associated symptoms.
- Physical assessment techniques for assessing the respiratory system of an individual include inspection, palpation, percussion and auscultation. Other factors that should be noted include the person's level of consciousness, restlessness and anxiety level, and the colour of the lips and nail beds.

CONCEPT CHECK

1 Where is the apex of each lung located?
1. in the mediastinum
2. resting on the diaphragm
3. within the parietal pleura
4. just below the clavicle

2 Which physiological process is involved in gas exchange at the respiratory membrane?
1. facilitated transport
2. active transport
3. simple diffusion
4. hydrostatic pressure

3 Which structures cover the external surface of the alveoli?
1. terminal bronchioles
2. pulmonary arteries
3. pulmonary veins
4. pulmonary capillaries

4 Which process is initiated between oxygen and haemoglobin as the temperature of body tissues increases?
1. oxygen unloading is enhanced
2. oxygen unloading is inhibited
3. respiratory rate is decreased
4. lung compliance is increased

5 Which of the following questions should be included when conducting a health history to identify a genetic risk of respiratory disease?
1. 'Tell me how many colds you have each year.'
2. 'Has anyone in your family had a stroke or heart attack?'
3. 'Has lung cancer ever been diagnosed in your family?'
4. 'Do your children have trouble breathing at night?'

6 While auscultating the person's breath sounds, you note continuous musical sounds. You document these sounds as:
1. murmurs
2. wheezes
3. crackles
4. rales

7 The person you are assessing has had a lung removed. What type of breath sound would you expect to assess over the affected side?
1. resonance
2. crackles
3. bronchovesicular
4. absent

8 While assessing the person with a left pneumothorax you note decreased diaphragmatic excursion on the left. What would you do next?
1. Notify the doctor immediately.
2. Document the assessment.
3. Repeat the assessment several times.
4. Tell the person to hold their breath.

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CHAPTER 34

NURSING CARE OF PEOPLE WITH UPPER RESPIRATORY DISORDERS

MAJELLA HALES

KEY TERMS

coryza 1223
epistaxis 1236
influenza 1225
laryngectomy 1247
laryngitis 1234
pertussis 1234
pharyngitis 1231
rhinitis 1223
rhinoplasty 1240
sinusitis 1229
sleep apnoea 1242
tonsillitis 1231

LEARNING OUTCOMES

- Discuss the causes and implications of individuals experiencing selected infectious or inflammatory disorders of the upper respiratory system.
- Explore the occurrence and management of people experiencing obstructive conditions to the upper respiratory system such as trauma, epistaxis or surgery.
- Describe the critical considerations related to caring for a person with laryngeal obstruction from trauma or obstructive sleep apnoea.
- Examine the biopsychosocial implications and potential outcomes for a person with laryngeal tumour.

CLINICAL COMPETENCIES

- Assess functional health status of people with upper respiratory tract disorders, using data to identify and prioritise holistic nursing care needs.
- Use nursing research and evidence-based practice to plan and implement nursing care for people with upper respiratory disorders.
- Provide safe and effective nursing care for people having surgery involving the upper respiratory system and/or with a tracheostomy.
- Safely and knowledgeably administer medications and prescribed treatments for people with disorders of the upper respiratory tract.
- Provide appropriate teaching for the person and family affected by upper respiratory tract disorders.
- Evaluate the effectiveness of care, reassessing and modifying the plan of care as needed to achieve desired outcomes.

Upper respiratory disorders may affect the nose, paranasal sinuses, tonsils, adenoids, larynx and pharynx. Upper respiratory disorders may be minor, such as the common cold. However, a patent upper airway is necessary for effective breathing. Acute and even life-threatening problems develop when upper airway patency is affected (e.g. by laryngeal oedema). Upper respiratory disorders can affect breathing, communication and body image. When breathing is compromised because of swelling, bleeding or accumulation of secretions, fear and anxiety may develop.

Nursing care focuses on maintaining the airway, managing pain and symptoms, promoting effective communication and providing psychological support for the person and family. Prior to proceeding with this chapter, review the anatomy and physiology, diagnostic tests and assessment of the upper respiratory system in Chapter 33 as needed.

INFECTIOUS AND INFLAMMATORY DISORDERS OF THE UPPER RESPIRATORY SYSTEM

Constant exposure of the upper respiratory tract to the environment makes it vulnerable to a variety of infectious and inflammatory conditions. Although most upper respiratory infections and inflammations are minor, complications may result. In the older adult, the risk of serious problems following an upper respiratory infection can be significant.

Rhinitis, inflammation of the nasal cavities, is the most common upper respiratory disorder. Rhinitis may be either acute or chronic. *Acute viral rhinitis*, or the common cold, is discussed below. Chronic rhinitis includes allergic, vasomotor and atrophic rhinitis. *Allergic rhinitis*, or hay fever, results from a sensitivity reaction to allergens such as plant pollens. It tends to occur seasonally. The aetiology of *vasomotor rhinitis* is unknown. Although its manifestations are similar to those of allergic rhinitis, it is not linked to allergens. *Atrophic rhinitis* is characterised by changes in the mucous membrane of the nasal cavities.

THE PERSON WITH VIRAL UPPER RESPIRATORY TRACT INFECTION

Viral upper respiratory tract infections (URTIs, or the common cold) are the most common respiratory tract infections and are among the most common human diseases. URTIs are highly contagious and are prevalent in schools and work environments.

FAST FACTS

- Rhinoviruses are the most common cause of viral URTIs (Rajnik, 2014).
- Colds due to rhinovirus are more common in early autumn and late spring.
- More than 100 different serotypes of rhinovirus have been identified.
- Parainfluenza viruses, respiratory syncytial virus (RSV), coronaviruses and adenoviruses also can cause URTIs (Lee & Bishop, 2015).

Pathophysiology

More than 200 strains of virus cause URTI, including rhinoviruses, adenoviruses, parainfluenza viruses, coronaviruses and respiratory syncytial virus. (See the section that follows for more information about RSV.) Occasionally, more than one virus may be present. Viruses causing acute URTIs spread by aerosolised droplet nuclei during sneezing or coughing, or by direct contact. The virus usually spreads when the hands and fingers pick it up from contaminated surfaces and carry it to the eyes and mucous membranes of the susceptible host. People who have been infected are highly contagious, shedding the virus for a few days prior to and after the appearance of symptoms. Although immunity is produced to the individual virus strain, the number of viruses causing URTI ensures that most people continue to experience colds throughout their lifetimes.

Viscous mucus secretions in the upper respiratory tract trap invading organisms, preventing contamination of more vulnerable areas. Cells of the upper respiratory tract are infected when the virus attaches to receptors on the cell. Local immunological defences, such as secretory IgA antibodies in respiratory secretions, then attempt to inactivate the antigen, producing a local inflammatory response. The mucous membranes of the nasal passages swell and become hyperaemic and engorged. Mucus-secreting glands become hyperactive. These responses to the virus produce the typical manifestations of viral URTI.

Manifestations and complications

Acute viral URTI often presents as the common cold. Nasal mucous membranes appear red (*erythematous*) and *boggy* (swollen). Swollen mucous membranes, local vasodilation and secretions cause nasal congestion. Clear, watery secretions lead to **coryza**, profuse nasal discharge. Sneezing and coughing are common. Sore throat is common and may be the initial symptom. Systemic manifestations of acute viral URTI may include low-grade fever, headache, malaise and muscle aches. Symptoms generally last for a few days up to 2 weeks. Although acute viral URTI is typically mild and self-limited, its effects on the immune defences of the upper respiratory tract can increase the risk of more serious bacterial infections, such as sinusitis or otitis media.

INTERPROFESSIONAL CARE

Because most acute viral upper respiratory tract infections are self-limiting, self-care is appropriate and encouraged. Medical treatment is usually required only when complications such as sinusitis or otitis media develop.

Diagnosis of acute viral URTI is usually based on the history and physical examination. Diagnostic testing may be indicated if a complication such as bacterial infection is suspected. A white blood count (WBC) may be ordered to assess for leucocytosis (an elevated WBC). Cultures of purulent discharge may also be obtained.

Treatment is symptomatic. Adequate rest, maintaining fluid intake and avoiding chilling help relieve systemic symptoms such as fever, malaise and muscle ache. Instruct people to cover the mouth and nose with tissues when coughing or sneezing, dispose of soiled tissues appropriately and wash hands thoroughly. Additionally, avoiding crowds and maintaining a distance of 1 metre from others helps prevent spread of the infection to others.

Medications

Medications may be recommended to shorten the duration of the illness and relieve symptoms. Mild decongestants or over-the-counter (OTC) antihistamines may help relieve coryza and nasal congestion. Nasal sprays such as phenylephrine rapidly relieve nasal congestion, but may lead to dependence and rebound congestion if used for more than a few days at a time. Warm saltwater gargles, throat lozenges or mild analgesics may be used for sore throat. Although no specific antiviral therapy has been shown to be effective in shortening the duration of a URTI, experimental vaccines to prevent acute viral URTI are in developmental stages. For the nursing implications of decongestants and common antihistamines, see the 'Medication administration' box below.

Complementary therapies

Currently there is little evidence to demonstrate that complementary therapies are useful in the management of respiratory infections. Herbal remedies, such as echinacea and garlic, may have antiviral and antibiotic effects (Kumar & Ramaiah, 2011). Echinacea is also thought to stimulate the immune system, improving the body's response to infection. Taken at the first sign of infection, echinacea may reduce the duration and symptoms, although clinical trials have shown no consistent benefit (Karsch-Völk et al., 2014). Studies of the use of garlic in acute respiratory tract infections also suggest limited or uncertain effectiveness (Lissiman, Bhasale & Cohen, 2012).

Vitamin C is also promoted as a measure to reduce the severity and duration of URTI. Again, however, little consistent

MEDICATION ADMINISTRATION Decongestants and antihistamines

DECONGESTANTS

Decongestants promote vasoconstriction, reducing the inflammation and oedema of nasal mucosa and relieving nasal congestion. They are very effective when applied topically (by nasal spray) because of their rapid onset of action. However, the duration of effect is short, followed by vasodilation and rebound congestion. Because of their rapid effect and short duration, these preparations are habit forming. Chronic use may lead to rhinitis medicamentosa, a rebound phenomenon of drug-induced nasal irritation and inflammation.

Nursing responsibilities

- Assess for contraindications, such as hypertension or chronic heart disease. These drugs stimulate the sympathetic nervous system, increasing peripheral vascular resistance, blood pressure and heart rate.
- Evaluate medication regimen for potential interactions such as antihypertensive medications and monoamine oxidase (MAO) inhibitors.

Health education for the person and family

- Do not use more than the recommended dose.
- Check with the doctor before taking decongestants if you are taking any prescription medications or are being treated for high blood pressure or heart disease.
- Use nasal sprays for no more than 3 to 5 days.
- Increase fluid intake to relieve mouth dryness.
- These drugs may cause nervousness, shakiness or difficulty sleeping. Stop the drug if these effects occur.
- In some states, drugs containing pseudoephedrine may require a prescription or be kept behind the counter to reduce its use in preparing methamphetamine.

ANTIHISTAMINES

Antihistamines are widely available with and without a prescription. They are frequently combined with decongestants in over-the-counter cold and allergy preparations. Antihistamines relieve the systemic effects of histamine and dry respiratory secretions through an anticholinergic effect. Most antihistamines cause drowsiness; non-sedating forms are less likely to interfere with alertness. Diphenhydramine is used in numerous over-the-counter sleep aids as well as in cold and allergy preparations.

Nursing responsibilities

- Before administering or recommending these drugs, assess for possible contraindications, including the following:
  - acute asthma or lower respiratory disease that may be aggravated by drying of secretions
  - hypersensitivity to antihistamines
  - glaucoma (increased intraocular pressure)
  - impaired gastrointestinal motility or obstruction
  - prostatic hypertrophy or other urinary tract obstruction
  - heart disease.
- For people who must remain alert while on antihistamine therapy, recommend non-sedating forms.

Health education for the person and family

- Do not drive or operate machinery while taking over-the-counter or prescription forms of antihistamines known to be sedating.
- Stop the drug and notify your doctor immediately if you develop confusion, excessive sedation, chest tightness, wheezing, bleeding or easy bruising while taking antihistamines.
- Do not use alcohol or other central nervous system depressants while taking antihistamines.
- Hard lollies, chewing gum, ice chips and liquids help relieve mouth dryness caused by antihistamines.

benefit is demonstrated in well-designed rigorous clinical trials. It is possible that zinc helps reduce the length and severity of a cold; however, it can cause nausea and other gastrointestinal issues (Fashner, Ericson & Werner, 2012).

Aromatherapy with essential oils such as basil, cedarwood, eucalyptus, frankincense, lavender, marjoram, peppermint or rosemary may reduce congestion and promote comfort and recovery. Teach people that these essential oils are to be used only for inhalation, not for internal consumption.

Nursing care

Health promotion

Most importantly, people can limit their incidence of acute viral URTI by frequent handwashing and avoiding exposure to crowds. Maintaining good general health and stress-reducing activities support the immune system and help prevent acute viral URTI. Teach the person that becoming chilled or going out in the rain does not cause colds and that URTI are more likely to occur during periods of physical or psychological stress.

Community-based care

The primary nursing role in caring for people with acute viral URTI is educational. Self-care is appropriate for most people unless the problem is recurrent or a complication occurs. Acute viral URTI may interfere with work and recreational activities. Unless limited by symptoms, normal daily activities and roles usually can be maintained. Additional rest during the acute phase of illness is recommended. Additional fluid intake and a well-balanced diet help support the immune response, hastening recovery.

Include the following topics in teaching for home care:

- using disposable tissues to cover the mouth and nose while coughing or sneezing to reduce airborne spread of the virus
- blowing the nose with both nostrils open to prevent infected matter from being forced into the eustachian tubes
- washing hands frequently, especially after coughing or sneezing, to limit viral transmission
- using OTC preparations for symptomatic relief; precautions related to the sedating effects of antihistamines
- limiting use of nasal decongestants to every 4 hours for only a few days at a time to prevent rebound effect.

THE PERSON WITH RESPIRATORY SYNCYTIAL VIRUS

Respiratory syncytial virus (RSV) is a common virus that is the primary cause of respiratory illnesses in young children and the majority of lower respiratory disease in infants. Older children and adults also are commonly and repetitively infected by RSV, but the disease is milder, usually presenting as a common cold. However, older adults and people who are immunocompromised may develop severe pneumonitis when exposed to RSV (Walsh & Falsey, 2012). Mortality rates for RSV are higher in older adults and people who are immunocompromised (Krilov, 2015).

RSV is transmitted in much the same way as other URTIs: via contaminated hands or objects, and by coarse droplets spread by coughing and sneezing. The incubation period is 4 to 6 days.

In adults, the manifestations of RSV are those of other common URTIs, including rhinorrhoea, sore throat and cough. Headache, malaise and low-grade fever may occur. In older adults, RSV may present as lower respiratory infection with fever or pneumonia (Krilov, 2015). While the illness also presents as URTI in infants, it is more likely to progress to pneumonia, bronchiolitis and tracheobronchiolitis in this population.

Treatment for adults with upper respiratory RSV is symptomatic (see the preceding section on URTI). When the lower respiratory tract is involved, hydration and other measures to mobilise respiratory secretions are important. Intubation and mechanical ventilation may be necessary if hypoxia develops. Nursing care is supportive. The focus of nursing care for the adult with URTI manifestations of RSV is on teaching for self-care, identification of complications and prevention of viral spread. When lower respiratory symptoms are present, nursing care is similar to that provided for people with pneumonia (see Chapter 35).

THE PERSON WITH INFLUENZA

Influenza, or *flu*, is a highly contagious viral respiratory disease characterised by coryza, fever, cough and systemic symptoms such as headache and malaise. In 2014 in Australia, seasonal influenza notifications peaked in August in all states/territories (Department of Health, 2015a). However, this often varies slightly by year and region. Figure 34.1 shows the number of laboratory-confirmed influenza notifications per 100 000 since 2010. It is interesting to note

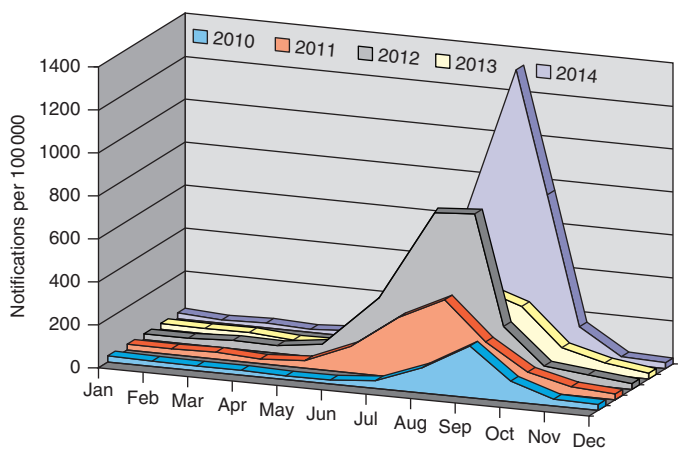


FIGURE 34.1 ■ Laboratory-confirmed influenza notifications per 100 000, Australia 2010–2014

Source: Derived using data from National Notifiable Diseases Surveillance System (NNDSS) (Department of Health, 2015b). *Notifications of influenza by month and year.* Retrieved from www9.health.gov.au/cda/source/cda-index.cfm.

that 2014 was a particularly busy year with almost two-and-a-half times the number of notifications than 2013. At the time of publication, the notifications for 2015 were again higher than the previous year when comparing the progress through the influenza season.

Influenza usually occurs in epidemics or pandemics, although sporadic cases do occur. Localised outbreaks of influenza usually occur about every 1 to 3 years. Global epidemics (pandemics) are less frequent, developing every 10 to 15 years until the past two decades. A recently identified strain of influenza A, H1N1 (swine flu), reached pandemic stage in 2009 and is currently classified in the post-pandemic stage. It demonstrated the ability to spread between humans. This strain of virus is thought to be a reassortment of four known strains of influenza A virus subtype H1N1, one strain originating from humans, one from birds and two from swine. See Box 34.1 for more information about swine influenza.

Although influenza tends to be mild and self-limited in healthy adults, older adults and people with chronic heart or pulmonary disease have a high incidence of complications (such as pneumonia) and a higher risk of mortality related to the disease and its complications (Rothberg & Haessler, 2010).

Pathophysiology

Influenza virus is transmitted by airborne droplet and direct contact. Three main strains of the virus have been identified as influenza A virus, influenza B virus and influenza C virus. Influenza A is responsible for most infections and the most severe outbreaks of influenza. This is primarily due to its ability to alter its surface antigens, bypassing previously developed immune defences to the virus. New strains of influenza virus are named according to the strain, geographical origin and the year the strain was identified (e.g. A/Taiwan/89). Surface antigens of the specific virus may be used to further differentiate influenza A viruses. Outbreaks of influenza B virus are generally less extensive and less severe than those caused by influenza A virus. Illness associated with influenza C virus is mild and often goes unrecognised.

The incubation period for influenza is short, only 18 to 72 hours. The virus infects the respiratory epithelium. It rapidly replicates in infected cells and is released to infect neighbouring cells. Inflammation leads to necrosis and shedding of serous and ciliated cells of the respiratory tract. This allows extracellular fluid to escape, producing rhinorrhoea. With recovery, serous

FAST FACTS

- Type A influenza viruses are found in many animals including birds, bats, pigs, whales, horses, seals and humans (Centers for Disease Control, 2014).
- Type A influenza is believed to have caused four pandemics: 1918 (Spanish flu), 1957 (Asian flu), 1968 (Hong Kong flu) and most recently the H1N1 'swine flu' in 2009 (worldwide).
- Type B influenza viruses are commonly found in humans and often are responsible for influenza outbreaks but not pandemics.
- Type C influenza viruses, found in humans, pigs and dogs, typically cause mild respiratory infections (National Institute of Allergy and Infectious Diseases, 2012).

BOX 34.1 Focus on swine influenza

On 11 June 2009, the World Health Organization (WHO) declared the worldwide pandemic alert level at 6. The alert levels are defined by spread more than by severity of illness and are classified as follows:

Phase 1: influenza virus circulating among animals (most commonly birds); however, no virus has been reported in humans.

Phase 2: a virus circulating among animals is known to have caused infection in humans.

Phase 3: sporadic cases or clusters of disease have been found; however, level of human-to-human transmission is not severe enough to cause community spread.

Phase 4: human-to-human transmission of virus able to cause 'community-level outbreaks'. Indicates a significant increase in risk of a pandemic but does not necessarily mean that a pandemic is a foregone conclusion.

Phase 5: human-to-human spread of the virus into at least two countries in one WHO region. While most countries will not be affected at this stage, the declaration of Phase 5 is a strong signal that a pandemic is imminent.

Phase 6: community-level outbreaks in at least one other country in a different WHO region in addition to the criteria defined in Phase 5. Designation of this phase will indicate that a global pandemic is underway (WHO, 2009).

The alert was released relating to an influenza A strain subtype H1N1 (commonly referred to as 'swine flu') which was initially identified in April 2009 in Mexico. This strain, however, was thought to be in existence in pigs for a number of years before its transmission to humans. By June the outbreak had reached a global level, and in August 2010 WHO declared the world to be in the post-pandemic phase. In 2009 in Australia, revised data reported 37636 cases of pandemic (H1N1), of which 191 people died (Department of Health, 2015c).

Symptoms of 'swine flu' mimic those of seasonal flu and may include fever, sneezes, sore throat, muscle/joint aches and pains, coughs and headache. The virus is spread by coughs and sneezes or by touching contaminated surfaces before touching the mouth or nose. WHO has released a number of fact sheets targeted at educating the community regarding correct handwashing techniques and techniques to limit the spread of the virus.

Currently the only treatment is by antiviral medications (e.g. Tamiflu). In the last quarter of 2009, a monovalent vaccine (Panvax®) was introduced by the Commonwealth Serum Laboratories in Australia; however, from December 2010, expired supplies were withdrawn (Department of Health, 2015d). Since 2010, the formulation against the commonly circulating A(H1N1)pdm09 strain has been included in the seasonal trivalent vaccine (Department of Health, 2015d).

cells are replaced more rapidly than ciliated cells, leading to continued cough and coryza. Systemic manifestations of influenza likely are caused by release of inflammatory mediators such as tumour necrosis factor alpha, interleukin-alpha and interleukin-6. The humoral and cell-mediated immune responses are activated by influenza infection and are supplemented by other local and systemic responses (such as interferons). Viral shedding lasts 5–10 days, starting just before the onset of symptoms; however, young children and immunocompromised individuals may shed for much longer (Derlet, 2015).

Manifestations

Infection with influenza virus produces one of three syndromes: uncomplicated nasopharyngeal inflammation, viral upper respiratory infection followed by bacterial infection, or viral pneumonia. The onset is rapid; profound malaise may develop in a matter of minutes.

Manifestations of influenza include abrupt onset of chills and fever, malaise, muscle aches and headache. Respiratory manifestations include dry, non-productive cough, sore throat, substernal burning and coryza (see the box below). Acute symptoms subside within 2 to 3 days, although fever may last as long as a week. The cough may be severe and productive. Along with fatigue and weakness, the cough can persist for days or several weeks.

MANIFESTATIONS Influenza

RESPIRATORY MANIFESTATIONS
- Coryza
- Cough, initially dry becoming productive
- Substernal burning
- Sore throat

SYSTEMIC MANIFESTATIONS
- Fever and chills
- Malaise
- Muscle aches
- Fatigue

Complications

The respiratory epithelial necrosis caused by influenza increases the risk of secondary bacterial infections. Sinusitis and otitis media are frequent complications of influenza. Tracheobronchitis, inflammation of the trachea and bronchi, may develop. Although tracheobronchitis is not a serious health risk, its manifestations may persist for up to 3 weeks.

Influenza is clearly linked to an increased risk of pneumonia, particularly in older adults. Changes in respiratory function associated with ageing, including decreased effectiveness of cough and increased residual lung volume, pose little risk in the healthy older adult but greatly increase the risk of pneumonia associated with influenza. Primary influenza viral pneumonia, while uncommon, is a serious complication that may be fatal. It typically develops within 48 hours of the onset of influenza, often in people with pre-existing heart valve or pulmonary disease. Influenza pneumonia progresses rapidly and can cause hypoxaemia and death within a few days. Bacterial pneumonia is more likely to occur in older at-risk adults but may also affect otherwise healthy adults. It usually presents as a relapse of influenza, with a productive cough and evidence of pneumonia on the chest x-ray. See Chapter 35 for more information about pneumonia.

Other respiratory complications of influenza include exacerbation of chronic obstructive pulmonary disease (COPD), chronic bronchitis or asthma. Sinusitis (discussed later in this chapter) may also develop.

Reye's syndrome is a rare but potentially fatal complication of influenza. Although it is more likely to affect children, it also has been identified in older adults. It is most often associated with influenza B virus. Reye's syndrome develops within 2 to 3 weeks after the onset of influenza. It has a 30% mortality rate. Hepatic failure and encephalopathy develop rapidly in people with Reye's syndrome.

While uncommon, other potential complications of influenza include myositis (inflammation of skeletal muscles), myocarditis (inflammation of the heart muscle) and central nervous system (CNS) disorders such as encephalitis and Guillain–Barré syndrome.

INTERPROFESSIONAL CARE

Preventing community outbreaks and protecting vulnerable populations (e.g. older adults and people with chronic diseases) are the primary focus for interprofessional care related to influenza. Medical treatment of influenza focuses on establishing the diagnosis, providing symptomatic relief and preventing complications.

Prevention

Preventing influenza by immunising at-risk populations is an important aspect of care. Immunisation with polyvalent (containing antigens of several viral strains) influenza virus vaccine is largely effective in preventing influenza infection for several months to a year (Derlet, 2015). Annual immunisation is recommended for at-risk people, including people over the age of 65, residents of nursing homes, adults and children with chronic cardiopulmonary disorders (e.g. asthma) or chronic metabolic diseases such as diabetes, and healthcare workers who have frequent contact with high-risk people. Additionally, family members of at-risk people should be vaccinated to reduce the person's risk of exposure. The vaccine is given in autumn, prior to the annual winter outbreak.

Diagnosis

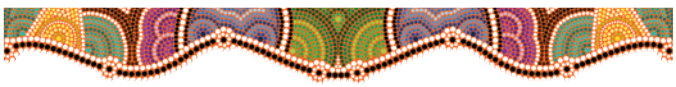
The diagnosis of influenza is based on history, clinical findings and knowledge of an influenza outbreak in the community. A chest x-ray and WBC count may be done to rule out complications such as pneumonia. The WBC count is commonly decreased in influenza; bacterial infections usually cause increased WBCs.

Medications

Yearly immunisation with influenza vaccine is the single most important measure to prevent or minimise symptoms of

influenza. About 5% of people experience mild symptoms of low-grade fever, malaise or myalgia for up to 24 hours after vaccination. Because the vaccine is produced in eggs, it should not be given to people who are allergic to egg protein. Serious adverse reactions to influenza vaccine are rare. *Guillain–Barré syndrome*, an acute neurological disorder characterised by muscle weakness and distal sensory loss, has been associated with certain batches of vaccine.

Over-the-counter analgesics such as aspirin, paracetamol or non-steroidal anti-inflammatory drugs (NSAIDs) provide symptomatic relief of fever and muscle ache. Antitussives may decrease cough, promoting rest. Antibiotics are not indicated unless secondary bacterial infection occurs.



Nursing care

Health promotion

Stress the importance of yearly influenza vaccination for people in high-risk groups and their families. Teach about spread of the disease, including measures to reduce the risk of contracting influenza, such as avoiding crowds and people who are ill.

Assessment

Unless there is a known outbreak of influenza in the community, it can be difficult to differentiate the manifestations of influenza from those of other URTIs.

- *Health history:* known exposure to virus; current symptoms, their onset and duration; presence of dyspnoea, chest pain, productive cough, facial pain or pressure in sinus areas; current medications, history of influenza vaccine; chronic diseases such as heart disease, COPD or diabetes; known medication allergies.
- *Physical examination:* general appearance; vital signs including temperature; skin colour; lung sounds; abdominal exam.
- *Diagnostic tests:* WBC, throat and sputum cultures, and chest x-ray for evidence of bacterial infection or pneumonia.

Nursing diagnoses and interventions

Although the symptoms of influenza are distressing, most people with the illness provide self-care and do not contact a healthcare provider. Recommendations to rest in bed during the acute phase of the illness and to limit activities until recovery are appropriate for influenza.

Severe disease or complications of influenza may necessitate hospitalisation for respiratory support and management. For these people, nursing care focuses on maintaining airway clearance, breathing patterns and adequate rest.

Ineffective breathing pattern

Muscle aches, malaise and elevated temperature may increase the respiratory rate and alter the depth of respirations, decreasing effective alveolar ventilation. Shallow respirations also increase the risk of *atelectasis* (lack of ventilation in an area of lung).

CONSIDERATION FOR PRACTICE

Monitor respiratory rate and pattern. Tachypnoea and/or rapid, shallow respirations may impair effective alveolar ventilation and gas exchange.

- Pace activities to provide for periods of rest. *Tachypnoea increases the work of breathing, causing fatigue; fatigue, in turn, can further impair ventilation and reduce the effectiveness of coughing.*
- Elevate the head of the bed. *The upright position improves lung excursion and reduces the work of breathing by lowering the diaphragm, moving abdominal contents downwards, creating less resistance to diaphragmatic excursion and slightly decreasing venous return.*

Ineffective airway clearance

Swelling and congestion of mucous membranes, extracellular fluid exudate and impaired ciliary action due to cell damage increase the risk of impaired airway clearance in influenza. The older adult is at particular risk because of normally reduced ciliary activity and increased lung compliance.

CONSIDERATION FOR PRACTICE

Monitor the effectiveness of cough and ability to remove airway secretions. Fatigue and general malaise may impair the ability to cough effectively and mobilise secretions.

- Maintain adequate hydration. Assess mucous membranes and skin turgor for evidence of dehydration. *Fever and decreased oral fluid intake may lead to dehydration and increased viscosity of secretions. Thick, viscous secretions are more difficult to expectorate.*
- Increase the humidity of inspired air with a bedside humidifier. *Increasing the water content of inhaled air helps loosen thick secretions and soothe mucous membranes.*
- Teach effective cough techniques. Administer analgesics as ordered. *The huff cough is effective to maintain open airways and it spares energy. (See Box 36.4 in Chapter 36 for teaching of this technique.) Relieving muscle ache increases the ability to cough effectively.*

Disturbed sleep pattern

Airway congestion, malaise, muscle aches and persistent cough may interfere with the ability to rest, increasing fatigue and prolonging recovery.

- Assess sleep patterns using subjective and objective information. *The person may appear to be sleeping but not achieving normal sleep patterns because of influenza symptoms. Both subjective and objective data are important to accurately assess sleep.*

CONSIDERATION FOR PRACTICE

If necessary, request a cough suppressant for night-time use. Cough suppressants are not recommended during the day because coughing promotes airway clearance. They may, however, be necessary at night to allow rest.

- Provide antipyretic and analgesic medications at or shortly before bedtime. *These drugs promote comfort by reducing fever and relieving muscle aches.*

Risk of infection

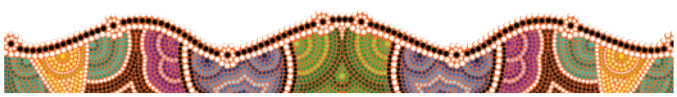
Infection control measures are recommended to prevent person-to-person transmission of influenza and to control influenza outbreaks in healthcare facilities.

- Use standard precautions and encourage all staff and visitors to frequently wash hands. *Handwashing is a primary infection control measure for infections transmitted via respiratory secretions.*
- Instruct people and visitors to control respiratory secretions by using tissues and to maintain a distance of at least 1 metre from others when coughing or sneezing. Handkerchiefs are not recommended due to spread of contaminants with multiple use. Provide masks for people and visitors who are unable to control secretions. *Limiting the spread of aerosolised secretions by covering the nose and mouth and maintaining distance from other people can reduce the spread of the disease to vulnerable populations.*
- Use droplet precautions for people with suspected or confirmed influenza: private room, masks for caregivers and visitors, and a mask for the person when being transported within the facility. *These measures limit the spread of respiratory secretions.*

Community-based care

Encourage appropriate self-care for people with influenza. Discuss the following topics related to home care:

- Increase rest during the acute, febrile phase of the illness.
- Maintain a liberal fluid intake even if anorexic.
- Appropriately use OTC medications for symptom relief.
- Employ hygiene measures such as using disposable tissues and frequent handwashing to reduce spread of the disease.
- Know manifestations of potential complications of influenza to report to the primary care provider.



THE PERSON WITH SINUSITIS

Sinusitis is inflammation of the mucous membranes of one or more of the sinuses (see Figure 33.2 in Chapter 32). Sinusitis is a common condition that usually follows an upper respiratory infection such as acute viral upper respiratory infection or influenza. Common causative organisms include streptococci, *S. pneumoniae, Haemophilus influenzae* and staphylococci. The risk of sinusitis is higher when the immune system is suppressed by immunosuppressive drugs or HIV infection. Sinusitis is common and difficult to treat in people who have AIDS.

Physiology review

The sinuses (or *paranasal sinuses*) are air-filled cavities in the facial bones that open into the turbinates of the nasal cavity. They are lined with ciliated mucous membranes that help move fluid and microorganisms out of the sinuses into the nasal cavity. The sinuses normally are sterile. Air within the sinuses has lower oxygen content than inspired air.

Pathophysiology

Sinusitis develops when nasal mucous membranes swell or other disorders obstruct sinus openings, impairing drainage. Mucus secretions collect in the sinus cavity, serving as a medium for bacterial growth. The nasal and sinus mucous membranes are continuous; therefore, bacteria generally spread to the sinuses via the opening into the nasal turbinates. The inflammatory response provoked by bacterial invasion draws serum and leucocytes to the area to combat the infection, increasing swelling and pressure.

Any process that impairs drainage from the sinuses may precipitate sinusitis. These include nasal polyps, deviated septum, rhinitis, tooth abscess or swimming or diving trauma. In hospitalised people, sinusitis may develop following prolonged nasotracheal intubation. Usually more than one sinus is infected. The frontal and maxillary sinuses are usually involved in adults.

Sinusitis may be acute or chronic. Chronic sinusitis results when acute sinusitis is untreated or inadequately treated. With continued infection, bacteria can become isolated, producing chronic inflammation. Over time, mucous membranes become thickened. Fungal infections may cause chronic infections, especially in immunosuppressed people. Other factors that may contribute to chronic sinusitis are smoking, a history of allergy and habitual use of nasal sprays or inhalants.

Manifestations and complications

The person with acute sinusitis often looks sick. Manifestations of sinusitis include pain and tenderness across the infected sinuses, headache, fever and malaise. The pain usually increases with leaning forward. When the maxillary sinuses are involved, pain and pressure are felt over the cheek. The pain may be referred to the upper teeth. Frontal sinusitis causes pain and tenderness across the lower forehead. Infection of the ethmoid sinus produces retro-orbital pain and pain over the high lateral aspect of the nose. Sphenoid sinusitis, the rarest form, may cause pain in the occiput, vertex or middle of the head. Symptoms often worsen for 3 to 4 hours after awakening and then become less severe in the afternoon and evening as secretions drain. The intensity and location of headache pain may change as sinuses drain. In acute sinusitis, the pain is usually constant and severe. In chronic sinusitis, the pain is described as dull and may be constant or intermittent.

Other symptoms include nasal congestion, purulent nasal discharge and bad breath. The nasal mucous membrane is red and swollen. Purulent drainage may be noted at the opening to the middle turbinate. This may be the only sign of chronic sinusitis. Swallowed secretions irritate and inflame the throat and may cause nausea or vomiting.

Complications develop when the infection spreads to surrounding structures (see Box 34.2). These include periorbital abscess or cellulitis, cavernous sinus thrombosis, meningitis, brain abscess or sepsis. Eustachian tube oedema may lead to hearing loss.

BOX 34.2 Potential complications of sinusitis

Local complications
- Orbital cellulitis
- Subperiosteal abscess
- Orbital abscess
- Cavernous sinus thrombosis
- Mucocoele
- Osteomyelitis

Intracranial complications
- Meningitis
- Epidural abscess
- Subdural abscess
- Brain abscess
- Venous sinus thrombosis

INTERPROFESSIONAL CARE

Treatment of sinusitis focuses on restoring drainage of obstructed sinuses, controlling infection, relieving pain and preventing complications.

Diagnosis

The diagnosis of acute sinusitis usually can be made using the history and physical exam. Diagnostic studies such as computed tomography (CT) scan or sinus x-rays generally are done only when sinusitis is persistent, chronic or recurrent. See Chapter 33 for more information about diagnostic studies and their nursing implications.

- *Sinus x-rays* are evaluated. Sinuses are normally translucent because they are filled with air; affected sinuses appear cloudy or opaque. A visible air–fluid level or thickening of the sinus mucosa may be seen in infected sinuses.
- *CT scan* is a more sensitive indicator of acute and chronic sinusitis and often is performed without preceding x-rays.
- *Magnetic resonance imaging (MRI)* may be ordered if malignancy of the sinus is suspected.

Medications

Antibiotic therapy directed at the usual organisms causing sinusitis typically is prescribed. Amoxicillin (possibly combined with clavulanate (Augmentin)) or cefaclor are commonly used antibiotics for sinusitis. Antibiotic therapy is continued for 10 to 14 days; occasionally a longer course is prescribed to prevent relapse. If the sinusitis does not respond to treatment with oral antibiotics, hospitalisation and intravenous antibiotic therapy may be required. See Chapter 11 for nursing care related to antibiotic therapy.

Oral or topical (in the form of nasal sprays) decongestants such as pseudoephedrine or phenylephrine are also prescribed to reduce mucosal oedema and promote sinus drainage. Antihistamines may decrease nasal congestion and facilitate sinus drainage, but they also tend to increase the viscosity of secretions and hinder drainage. For this reason, they may not be as effective as decongestants. Saline nose drops or sprays promote sinus drainage, as does inhalation of warm steam. Aerobic exercise also promotes mucus flow and may be recommended.

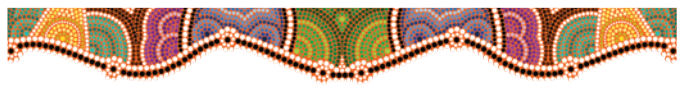
CONSIDERATION FOR PRACTICE

To administer topical drugs, the person's head is tilted backwards and to the side on which the drops are to be instilled. The person may need to remain in position for 5 minutes to allow the drops to reach the posterior nares.

Surgery

People who do not respond to pharmacological measures and who experience persistent facial pain, headache or nasal congestion may require *endoscopic sinus surgery*. People who have endoscopic sinus surgery usually do not require nasal packing postoperatively. Instead, frequent nasal cleaning and irrigation with normal saline are performed. The person is instructed to sneeze with the mouth open and avoid blowing the nose, lifting or straining for a week following surgery.

Antral irrigation can be done in the doctor's office under local anaesthesia. Saline solution is instilled into the maxillary sinus to irrigate the area and wash out the sinus of purulent exudate. The person is seated with the head forward and mouth open to allow drainage of the solution through the nose and mouth. A culture of the exudate may be obtained to determine appropriate antibiotic therapy.



Nursing care

Health promotion

Measures to prevent sinusitis are those that promote nasal drainage: encouraging liberal fluid intake, judicious use of nasal decongestants as needed and treating any obstructive process. Encourage individuals with URTI to blow their nose with both nares open. Advise people that use of saline nasal sprays can help maintain patency of the opening to the sinuses, promoting drainage and reducing the risk of obstruction and infection.

Assessment

Focused assessment of the person with suspected sinusitis includes the following:

- *Health history:* complaints of frontal or periorbital headache, cheek, teeth or ear pain; timing of pain and changes in intensity over course of the day; nasal discharge or post-nasal drip; other symptoms; previous sinus problems; current medications, known medication allergies.
- *Physical examination:* general appearance; vital signs including temperature; inspect nasal and pharyngeal mucous membranes; percuss sinuses for tenderness.
- *Diagnostic tests:* WBC and differential, cultures of sinus drainage, sinus x-rays or other imaging studies.

Nursing diagnoses and interventions

The person with sinusitis is often acutely uncomfortable. Obstructed and congested sinuses cause pain and pressure that increase with position changes and leaning forward. Treatment usually is community based, making education the key nursing role. When the person is hospitalised for intravenous antibiotic therapy or sinus surgery, pain and nutritional considerations are the priority for nursing care.

Pain

Although sinus surgery is relatively minor, both the incision and postoperative swelling can cause discomfort. Nasal packing, if used, contributes to the discomfort.

- Assess pain using a standardised pain scale. Administer analgesics as ordered. *Relief of pain promotes a feeling of wellbeing and enhances recovery.*
- Apply ice packs to the nose. *Cold compresses reduce swelling, control bleeding and provide local analgesia.*
- Elevate the head of the bed to Fowler's or high-Fowler's position for 24 to 48 hours after surgery. *Elevating the operative site minimises tissue swelling and promotes comfort.*

Imbalanced nutrition

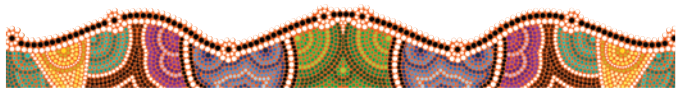
Postoperatively, the sense of smell, an appetite stimulus, is diminished by nasal packing. Mouth discomfort from the incision and numbness of the upper teeth also may affect appetite and eating.

- Provide clear liquid diet progressing to soft foods as tolerated. High-kilojoule dietary supplements may be used. *A progressive diet is used to assess the ability to swallow without choking and allay fears. Foods high in kilojoules and nutritional value provide for metabolic and healing requirements.*
- Monitor intake, output and weight. *This information allows assessment of overall fluid balance and the adequacy of dietary intake.*
- Elevate the head of the bed during meals. *The upright position facilitates swallowing and minimises risk of aspiration.*

Community-based care

Teaching for people with sinusitis and their families focuses on following through with appropriate treatment and promoting comfort. Discuss the following topics when preparing for home care:

- The importance of completing the entire course of prescribed antibiotics to achieve cure and prevent the development of antibiotic-resistant bacteria. Assist in developing a schedule that helps ensure all doses are taken.
- Measures to prevent superinfections (such as vaginitis or oral thrush) during the prolonged course of treatment (e.g. consume 200 g of yoghurt containing live bacterial cultures daily while on antibiotics).
- Use of systemic or topical decongestants to promote sinus drainage.
- Maintaining a liberal fluid intake to reduce the viscosity of mucus drainage.
- Use of a humidifier or steam inhalation to promote sinus drainage.
- Sleeping with the head of the bed elevated to a 45-degree angle and on the unaffected side to promote drainage of affected sinuses.
- Application of a warm, moist pack to the area of pain and tenderness to promote comfort.
- Notify the doctor if symptoms do not improve with treatment or if signs of a complication develop, such as increased pain and redness and swelling on the side of the nose or around the eyes.
- Postoperative instructions to prevent bleeding, such as avoiding blowing the nose for 7 to 10 days and avoiding strenuous activity such as heavy lifting for about 2 weeks.
- Use of saline nasal sprays postoperatively to keep the nasal mucosa moist.



THE PERSON WITH PHARYNGITIS OR TONSILLITIS

Pharyngitis, acute inflammation of the pharynx, is one of the most commonly identified clinical problems. Although it is usually viral in origin, pharyngitis may also be caused by bacterial infection. *Group A beta-hemolytic streptococcus* (strep throat) is the most common cause of bacterial pharyngitis. Other bacteria that may cause pharyngitis include *Neisseria gonorrhoeae*, a Gram-negative diplococcus that is sexually transmitted, *Mycoplasma* and *Chlamydia trachomatis*.

Tonsillitis is acute inflammation of the palatine tonsils. Although it is sometimes viral in origin, tonsillitis is usually due to streptococcal infection. The incidence of streptococcal infections is greatest between late autumn and spring, especially in cold climates. Viral tonsillitis may occur in epidemics in people living in crowded conditions, such as children in boarding schools.

Pathophysiology and manifestations

Pharyngitis and tonsillitis are contagious and spread by droplet nuclei. Incubation varies from a few hours to several days, depending on the organism. Viral infections are communicable for 2 to 3 days. Symptoms usually resolve within 3 to 10 days after onset.

Viral pharyngitis may be attributed to the same viruses as cause the common cold: rhinovirus, coronavirus or parainfluenza virus. Pharyngitis caused by adenovirus, influenza virus or Epstein–Barr virus (associated with infectious mononucleosis) may be particularly severe.

Acute pharyngitis causes pain and fever. The pain may vary from a scratchy sore throat to one so painful that swallowing is difficult. Streptococcal pharyngitis is usually marked by an abrupt onset, with fever of 38.3°C or higher, severe sore throat with dysphagia, malaise and often arthralgias and myalgias. Anterior lymph nodes are often enlarged and tender. Exudate (pus) may be seen on the pharynx and tonsils (see Figure 34.2). By contrast, the onset of viral pharyngitis is often gradual, with manifestations of low-grade fever, sore throat, mild hoarseness, headache and rhinorrhoea. The pharyngeal membranes appear mildly red with vascular congestion. Infectious mononucleosis, caused by the Epstein–Barr virus, often presents as acute pharyngitis, with visible patches of exudate on the pharynx or tonsils. The cervical lymph nodes are enlarged and tender as well. See the accompanying box for the manifestations of pharyngitis and tonsillitis.

In tonsillitis, the tonsils appear bright red and oedematous. White exudate is present on the tonsils; pressing on a tonsil may produce purulent drainage. The uvula may also be reddened and swollen. Cervical lymph nodes are usually tender and enlarged.

The person with tonsillitis complains of a sore throat, difficulty swallowing, general malaise, fever and otalgia (pain referred to the ear). Manifestations are often more severe in adolescents and

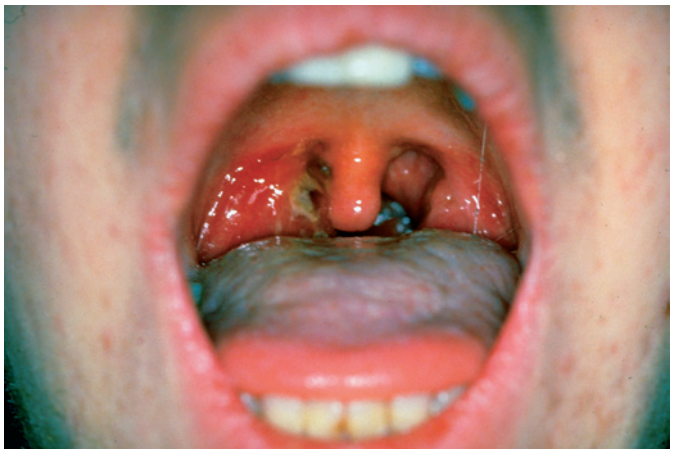


FIGURE 34.2 ■ The appearance of the oral pharynx and tonsils in acute pharyngitis and tonsillitis

Source: © Biophoto Associates/Science Source.

adults than in children. Infection may extend via the eustachian tubes to cause acute otitis media. This may lead to further damage such as spontaneous rupture of the eardrums and mastoiditis. See Chapter 45 for more information about otitis media.

MANIFESTATIONS Pharyngitis and tonsillitis

LOCAL
- Sore throat
- Possible dysphagia and ear pain
- Tender, swollen anterior cervical lymph nodes
- Hoarse voice
- Red, swollen pharyngeal mucous membranes and/or tonsils
- Possible visible exudate on pharyngeal membranes and/or tonsils

GENERAL
- Fever
- General malaise
- Arthralgia, myalgia

Complications

Although bacterial pharyngitis may be mild and indistinguishable from viral pharyngitis by its signs and symptoms, it can lead to significant complications such as abscess, scarlet fever, toxic shock syndrome, rheumatic fever or acute post-streptococcal glomerulonephritis.

Peritonsillar abscess, or *quinsy*, is a potential complication of tonsillitis. It usually results from group A beta-haemolytic streptococcus infection extending from the tonsils to the surrounding tissue. The abscess causes pus formation behind the tonsil with marked swelling and asymmetrical deviation of the uvula. The degree of swelling may make it difficult to swallow anything other than liquids. The person may exhibit thickening of the voice, drooling and trismus (a tonic contraction of the muscles of mastication).

Rare (1–3%) but serious complications of streptococcal pharyngitis and tonsillitis include acute glomerulonephritis and rheumatic fever, abnormal immune responses to the infection. Acute glomerulonephritis generally presents with sudden onset of haematuria, proteinuria and, less commonly, hypertension and oedema within 7 to 10 days after the acute infection. Rheumatic fever typically presents 3 to 5 weeks after acute infection with fever, painful or swollen joints, rash and heart murmur. Other complications of bacterial infection include sinusitis, otitis media, mastoiditis and cervical adenitis.

INTERPROFESSIONAL CARE

Both viral and bacterial pharyngitis are usually self-limited diseases. However, because of the risk of serious complications associated with streptococcal sore throat, an effort is usually made to establish an accurate diagnosis and treat bacterial pharyngitis.

Diagnosis

A *throat swab* is obtained and examined for streptococcus antigen. These tests allow rapid identification of the antigen but are not highly sensitive. When the test is positive, treatment for strep throat is initiated. If the test is negative, the swab is cultured to ensure that streptococcus organisms are not present. Even throat cultures are not always accurate, with approximately 10% false negative and 20% false positive results.

A *full blood count (FBC)* may be done on severely ill people or to rule out other causes of pharyngitis. The WBC count is usually normal or low in viral infections and elevated in bacterial infections.

Medications

Antipyretics and mild analgesics such as aspirin or paracetamol provide symptomatic relief for throat pain and associated myalgias. Penicillin is the drug of choice for group A streptococci. Erythromycin or amoxicillin may be used if the person is allergic to penicillin. Antibiotic therapy is continued for at least 10 days. The person is no longer contagious after 24 hours of antibiotic therapy.

Surgery

Tonsillectomy (surgical removal of the tonsils) is indicated for recurrent or chronic infections that have not responded to antibiotic therapy, hypertrophy of the tonsils with risk of airway obstruction, peritonsillar abscess, repeated attacks of purulent otitis media and tonsil malignancy. Adenoid tissue usually is removed at the same time. Bleeding is the most significant postoperative complication of tonsillectomy and may develop up to 2 weeks following the surgery.

Nursing care

Because of the risk of significant complications associated with streptococcal pharyngitis, encourage all individuals

with symptoms that persist for several days or that include fever, lymphadenopathy and myalgias to seek evaluation and treatment.

Home care is appropriate for acute uncomplicated pharyngitis. Treatment focuses on adequate rest and relief of symptoms. A liquid or soft diet is useful when swallowing is difficult. Increased fluid intake is encouraged, especially when febrile. Warm saline gargles, moist inhalations and application of an ice collar are soothing to the sore throat.

Following tonsillectomy, ensure a patent airway by placing the person in semi-Fowler's position with the head turned to the side to allow secretions to drain from the mouth and pharynx. Keep the airway in place until the gag and swallowing reflexes have returned. Apply an ice collar to reduce swelling and pain. Notify the surgeon immediately if excessive bleeding or haemorrhage occurs. If there is no bleeding, allow water and ice chips as desired. Warm saline mouthwashes are helpful in managing thick oral secretions following tonsillectomy. A liquid or semi-liquid diet is recommended for several days.

Community-based care

Discuss the following topics when preparing the person for home care:

- the importance of completing the full 10 days of antibiotic therapy if prescribed
- using warm saline gargles or throat lozenges for symptomatic relief
- signs and symptoms of possible complications of streptococcal infection such as glomerulonephritis or rheumatic fever
- monitoring temperature in the morning and evening until well to ensure that the infection has not spread to deeper tissues
- proper use and disposal of tissues and frequent handwashing to prevent spreading the infection to others.

For the person who has had a peritonsillar abscess drainage or tonsillectomy, provide the following instructions:

- postoperative mouth and throat care

NURSING CARE PLAN A person with peritonsillar abscess

Monica Wunderman, aged 27, was recently treated for tonsillitis caused by infection by group A streptococcus. She presents to the emergency department 10 days later appearing acutely ill. She states that her throat is so sore that she has difficulty swallowing even liquids. Barbara Wallace, the ED nurse, completes an assessment of Ms Wunderman.

ASSESSMENT

Findings include T 38.8°C. An acutely swollen and reddened area of the soft palate is noted in her mouth, half occluding the orifice from the mouth into the pharynx. Yellow exudate is present. A diagnosis of peritonsillar abscess is made. Needle aspiration of the abscess is performed.

DIAGNOSES

- *Acute pain* related to peritonsillar swelling manifested by difficulty swallowing.
- *Risk of airway compromise* related to pain and swelling manifested by inability to clear secretions and difficulty swallowing.
- *Nutritional imbalance* related to insufficient oral intake manifested by lack of appetite and pain when eating and difficulty swallowing fluids.

PLANNING

- Evaluate current medication regimen and effectiveness of same.
- Evaluate ability to clear secretions and volume of secretions being produced.
- Evaluate current oral intake and assess the need for further nutritional supplements.

Expected outcomes

- Have minimal or no pain.
- Maintain a patent airway as demonstrated by normal respiratory rate and rhythm.
- Maintain optimal fluid intake as evidenced by consumption of fluids and semi-liquid foods, moist mucous membranes, normal skin turgor and normal temperature.

IMPLEMENTATION

- Teach that ice-cold fluids may be easier to swallow than hot or room-temperature beverages and may provide a local analgesic effect.
- Advise to avoid citrus juices, hot or spicy foods, and rough-textured foods for 1 week.
- Teach pain management strategies such as applying an ice collar as desired and gargling with warm saline or mouthwash solution every 1 to 2 hours for the first 24 to 48 hours after aspiration of the abscess.
- Instruct to take medications as prescribed.

EVALUATION

When Ms Wallace contacts Ms Wunderman by telephone 2 days after her visit to the emergency department, she reports complete relief of symptoms. She is afebrile, taking fluids without difficulty and has had no difficulty breathing. She has not experienced any pain.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe common symptoms of infectious or inflammatory diseases of the upper airway and discuss methods of symptom relief.
2. Describe common pharmacological interventions for these disorders.
3. Which themes of nursing diagnoses emerge for people with these types of disorders?

REFLECTION ON THE NURSING PROCESS

1. Which communication and education strategies could you use for people with peritonsillar abscess to improve their knowledge regarding this condition without negatively impacting on their presenting symptoms?
2. Outline what you have learned from this case study and how you will apply this knowledge to your current practice.

- avoiding use of aspirin for 2 weeks to reduce the risk of postoperative bleeding
- manifestations of bleeding to report to the doctor. (Delayed haemorrhage may occur for up to 1 week post surgery.)

THE PERSON WITH A LARYNGEAL INFECTION

The larynx, located between the upper airways and the lungs, protects the lower respiratory tract from inhaled substances other than air and allows speech. The larynx includes the epiglottis, which covers the larynx during swallowing, and the glottis or vocal cords. Either portion of the larynx may become inflamed.

Epiglottitis

Epiglottitis, inflammation of the epiglottis, is an uncommon disorder that presents as a medical emergency. *H. influenzae* infection is the most common cause of epiglottitis. Epiglottitis is a rapidly progressive cellulitis that begins between the base of the tongue and the epiglottis. The epiglottis itself becomes swollen and inflamed; swelling of adjacent tissues pushes the epiglottis posteriorly. This swelling and oedema threaten the airway. Adults usually present with a 1- to 2-day history of sore throat, *odynophagia* (painful swallowing), dyspnoea and possibly drooling and stridor.

Using a tongue blade to view the oropharynx is avoided; this may precipitate laryngospasm and airway obstruction. The epiglottis is visualised using a flexible fibre-optic laryngoscope to establish the diagnosis. The epiglottis appears red, swollen and oedematous. Nasotracheal intubation may be required to ensure airway patency. The person may be admitted to a critical care unit and intravenous antibiotic therapy is initiated. Ceftriaxone may be prescribed. Dexamethasone, a systemic corticosteroid, is also given to suppress the inflammatory response and rapidly reduce swelling of the epiglottis.

Nursing care for the person with acute epiglottitis focuses on monitoring and maintaining airway patency. Monitor oxygen saturation continuously. Observe closely for signs of airway obstruction, including nasal flaring, restlessness, stridor, use of accessory muscles and decreased oxygen saturation measurements. If the person is not intubated, supplies for emergency intubation should be kept in the unit. Epiglottitis is frightening for both the person and the nurse. Maintaining a calm, reassuring manner is an essential nursing role.

Laryngitis

Laryngitis, inflammation of the larynx, is a common disorder that may occur alone or in conjunction with other upper respiratory infections. It is commonly associated with a viral URTI such as influenza. It may also occur with bronchitis, pneumonia or other respiratory infections. Excessive use of the voice, sudden changes in temperature or exposure to dust, irritating fumes, smoke or other pollutants can also cause acute or chronic laryngitis. It is more common in the winter and in colder climates.

In laryngitis, the mucous membrane lining the larynx becomes inflamed; the vocal cords also may become oedematous. The primary symptom of laryngitis is a change in the voice. Hoarseness or *aphonia*, complete loss of the voice, may occur. The throat is often sore and scratchy and a dry, harsh cough may be present.

There is no specific treatment for viral laryngitis. Any identified precipitating factors such as overuse of the voice and exposure to irritants should be eliminated. Voice rest is advised, as is abstinence from tobacco and alcohol, which are chemical irritants. Treatment may also include inhaling steam or spraying the throat with antiseptic solutions. Identifying and eliminating irritants are helpful to prevent future attacks.

Impaired verbal communication is the priority nursing problem for people with laryngitis. The meaning of messages is conveyed not only by the words used but also by the tone and loudness of voice. Instruct to rest the voice as much as possible. Encourage speaking in short sentences or using alternate methods of communication, such as writing. Resting the voice hastens recovery and decreases throat discomfort. Advise the person to use soothing throat lozenges, sprays or other comfort measures such as gargling with a warm antiseptic solution. Help identify potential irritants, such as fumes, chemicals or cold temperature, to prevent future bouts of laryngitis.

THE PERSON WITH PERTUSSIS

Pertussis, or *whooping cough*, is a highly contagious acute upper respiratory infection caused by the bacterium *Bordetella pertussis*. Although it is thought by many to be a childhood disease that has been virtually eliminated by aggressive immunisation of infants, Australia is recovering from an outbreak which started in 2008 and peaked in 2011 at more than four times the average notification rates of the preceding 20 years. Figure 34.3 reports the trend in laboratory-confirmed pertussis infections in Australia from 1992 to 2014. Up to 45% of people affected by pertussis are adolescents and adults. Figure 34.4 shows the distribution of pertussis infection across age.

Pathophysiology

B. pertussis is a Gram-negative rod that is spread by respiratory droplets. The bacteria attach to ciliated epithelial cells of the nasopharynx, multiplying and invading respiratory tissues. The damage and effects of pertussis are not due to the infection itself but to toxins produced by the bacteria. These toxins damage the mucosa and paralyse the cilia. As a result, clearance of respiratory secretions is impaired, increasing the risk of pneumonia. The toxins also prompt an inflammatory response and inhibit immune defences.

Although immunisation does not appear to confer lifetime immunity, the disease tends to be milder in adolescents, adults and people who have been immunised. These infected individuals can, however, transmit the disease to other susceptible people, including unimmunised or under-immunised infants (Teng & Wang, 2012).

Young infants have the highest risk of complications of the disease, such as death, pneumonia and neurological complications. Neurological complications are thought to result from hypoxia due to prolonged paroxysms of coughing.

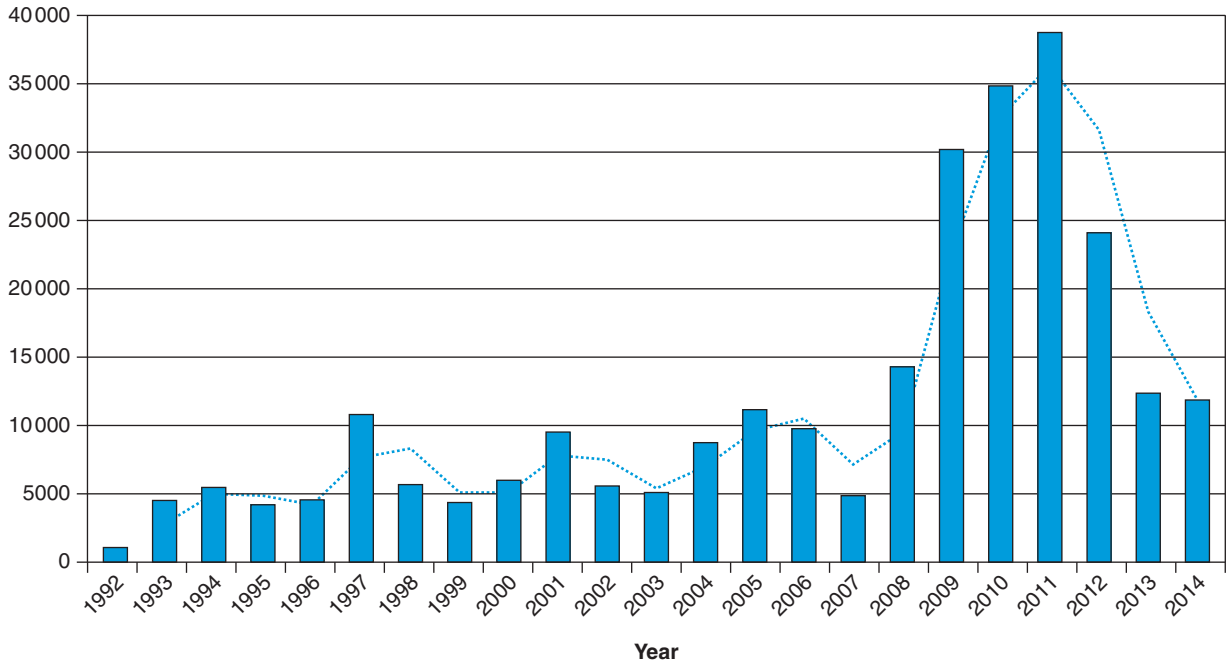


FIGURE 34.3 ■ *Bordetella pertussis* incidence in Australia 1992–2014

Source: Derived using data from National Notifiable Diseases Surveillance System (NNDSS) (Department of Health, 2015e). *Notifications of* Bordetella pertussis *by year.* Retrieved from www9.health.gov.au/cda/source/cda-index.cfm.

Complications in adolescents and adults may occur as a result of increased intrathoracic pressure during prolonged coughing spells. These may include pneumothorax, weight loss, inguinal hernia, rib fracture and *cough syncope* (fainting due to hypoxia) (Teng & Wang, 2012).

Manifestations

Classic pertussis follows a predictable pattern, with typical URTI symptoms (coryza, sneezing, low-grade fever and mild cough) beginning 7 to 10 days after exposure. After 1 to 2 weeks, the cough becomes more frequent, occurring in

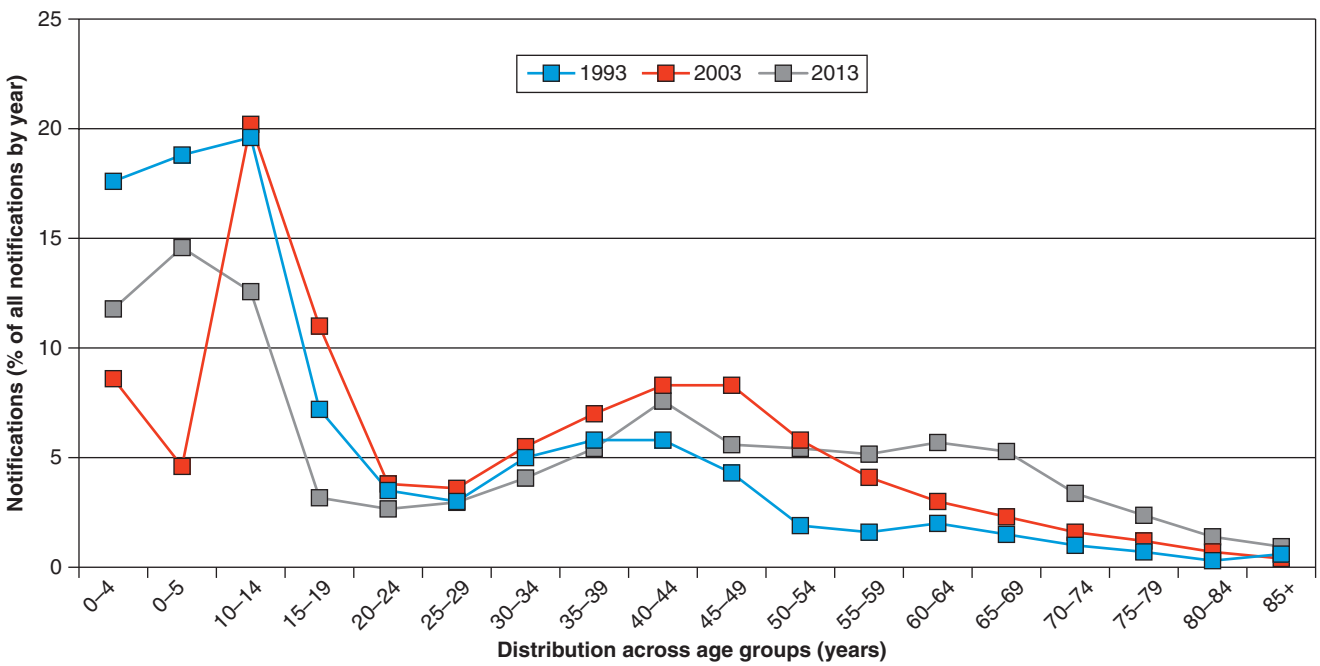


FIGURE 34.4 ■ Distribution of *Bordetella pertussis* laboratory-confirmed infections across age groups for selected years

Source: Derived using data from National Notifiable Diseases Surveillance System (NNDSS) (Department of Health, 2015f). *Notifications of* Bordetella pertussis *by age group and year.* Retrieved from www9.health.gov.au/cda/source/cda-index.cfm.

paroxysms or bursts of rapid coughs, often ending with an audible whoop caused by rapid inspiration. This whoop is less common in adolescents and adults, often delaying diagnosis. Vomiting commonly follows an episode of coughing (Bocka, 2014). Coughing paroxysms vary in frequency from several per hour to 5 to 10 per day, interfering with eating and sleep. This stage of the disease, called the *paroxysmal stage*, usually lasts no more than 6 weeks, after which coughing becomes less severe and gradually resolves over a period of up to 3 months.

In adolescents and adults, pertussis is suspected when an upper respiratory infection produces a cough that persists longer than 7 days, is accompanied by vomiting and is worse at night.

INTERPROFESSIONAL CARE

Active immunisation with pertussis vaccine is the primary preventive strategy for pertussis. Acellular pertussis vaccines that are effective but produce fewer adverse reactions than traditional whole-cell vaccines are available and preferred for immunisation.

The diagnosis of pertussis is established by culture of nasopharyngeal secretions. However, nasopharyngeal secretions may remain positive for the organism for only about 3 weeks after the onset of symptoms, so blood tests for antibodies to the organism may be necessary to confirm the diagnosis. Lymphocytosis (elevated lymphocyte count) may be present.

Azithromycin and clarithromycin are the antibiotics of choice to eradicate *B. pertussis* infection. In individuals where macrolide antibiotics are contraindicated, trimethoprim-sulfamethoxazole may be used (Antibiotic Expert Groups, 2014). Hospitalisation rarely is required for adults, although children and infants with severe disease often are hospitalised to prevent complications such as neurological effects of hypoxia and malnutrition. Respiratory isolation is instituted for 5 days after antibiotic therapy is started. Prophylactic antibiotics may be given to household and close contacts of the infected person depending on age, health, and immunisation status.

Nursing care

Nurses are instrumental in promoting effective immunisation of all infants and young children against pertussis. The Immunise Australia Program recently has started to recommend that parents, grandparents and carers of infants under 6 months of age should receive a single pertussis vaccine (dTpa) booster at least 2 weeks before beginning close contact with the infant (Department of Health, 2015d). Education is a key nursing role related to immunisation, as significant controversy currently exists about potential long-term adverse consequences of the vaccine.

Recommend nasopharyngeal culture for people complaining of persistent cough, especially when the cough is accompanied by vomiting or is significantly worse at night, or if other members of the household or close contacts have a similar illness.

CONSIDERATION FOR PRACTICE

Pertussis is a reportable communicable disease. All probable and confirmed cases must be reported to the local health department.

Education is a primary nursing role related to pertussis. Adults usually remain in the community for treatment. Teach respiratory isolation measures to be used until the disease is no longer communicable. Discuss ways to control respiratory secretions and the importance of disposing of tissues and secretions personally to prevent exposure of others. Stress the importance of prophylactic treatment for all household and close contacts. Discuss measures to maintain fluid and nutrient intake and use of a cough suppressant at night to promote rest. Encourage increased fluid intake to promote expectoration of respiratory secretions. Teach about the prescribed antibiotic, including its potential adverse effects and measures to reduce them, such as taking erythromycin with meals to prevent gastric upset. Contact the state or territory health department for follow up of contacts and compliance with prescribed treatment.

CONSIDERATION FOR PRACTICE

The National Immunisation Program requires the diphtheria, tetanus and acellular pertussis immunisation to be administered at 2, 4 and 6 months of age, with a booster to be administered at 4 years, and also between 10 and 15 years (Department of Health, 2015d).

UPPER RESPIRATORY TRAUMA OR OBSTRUCTION

Obstruction of the upper airway due to trauma (fracture of the nasal septum or the larynx), bleeding (e.g. epistaxis) or a tumour is not only frightening for the person but also may interfere with the ability to breathe.

THE PERSON WITH EPISTAXIS

The nose has a rich blood supply, receiving major arterial vessels from both the internal and external carotid artery systems. **Epistaxis**, or nosebleed, may be precipitated by a number

of factors. Trauma (picking the nose or blunt trauma) can cause epistaxis, as can drying of nasal mucous membranes, infection, substance abuse (e.g. cocaine), arteriosclerosis or hypertension. Epistaxis may also indicate a bleeding disorder related to acute leukaemia, thrombocytopenia, aplastic anaemia or severe liver disease. Additionally, treatment with an anticoagulant or antiplatelet drug may cause nosebleed. In adults, men more frequently have nosebleeds than women.

Pathophysiology and manifestations

Ninety per cent of all nosebleeds arise in the anterior nasal septum from Kiesselbach's area, a rich vascular plexus. Because of their location, these vessels are susceptible to trauma from nose picking, drying and infection. Posterior epistaxis more often develops secondarily to systemic disorders such as blood dyscrasias, hypertension or diabetes. In posterior epistaxis, bleeding is from the terminal branches of the sphenopalatine and internal maxillary arteries. Posterior epistaxis tends to be more severe and occurs more frequently in the older adult.

Anterior nosebleeds usually produce obvious bleeding from the nares, as well as bleeding into the posterior nasal and oral pharynx. The bleeding from a posterior nosebleed may be less apparent, with most of the blood draining into the posterior nasopharynx and swallowed by the person. Nausea and vomiting may occur due to swallowed blood.

INTERPROFESSIONAL CARE

The goal of treatment for epistaxis is to identify and control the source of bleeding.

Anterior bleeding can usually be managed by simple first-aid measures, such as applying pressure (pinching the nose towards the septum) for 5 to 10 minutes and applying ice packs to the nose and forehead to cause vasoconstriction. The person is placed in a sitting position to decrease blood flow to the head and reduce venous pressure. Leaning forward reduces drainage of blood backward into the nasopharynx and decreases swallowing of blood. The person is instructed to spit out the blood to help estimate the amount of bleeding and to prevent nausea and vomiting as a result of swallowed blood.

If applying pressure does not control the bleeding, medications, nasal packing or surgery may be necessary.

Medications

Topical vasoconstrictors such as cocaine (0.5%), phenylephrine (Neo-Synephrine) (1:1000) or adrenaline (1:1000) may be used to control anterior bleeding. These medications may be applied by nasal spray or on a cotton swab held against the bleeding site. Chemical cauterisation of the bleeding vessel may be done using agents such as silver nitrate. A topical anaesthetic such as lignocaine or cocaine may be used prior to nasal packing. If posterior nasal packing is required, prophylactic antibiotic therapy is initiated to prevent sinusitis or possible toxic shock syndrome.

Nasal packing

If bleeding cannot be controlled with pressure and local medications, a nasal tampon (a soft balloon filled with air) may be used to apply direct pressure to the bleeding vessel or the nasal cavity may be packed with 2.5–5 cm petroleum gauze. For an anterior pack, approximately 1–1.5 m of packing may be placed carefully and systematically along the floor of the nasal cavity and then into the vault of the nose. This is often available prepacked, impregnated with triamcinoline and an antibiotic cream. Anterior nasal packs are usually left in place for 24 to 72 hours. If epistaxis is caused by a bleeding disorder, the packing may be left in place for 4 to 5 days while the disorder is treated.

Posterior nosebleeds are more difficult to control, requiring both anterior and posterior packing (see Figure 34.5).

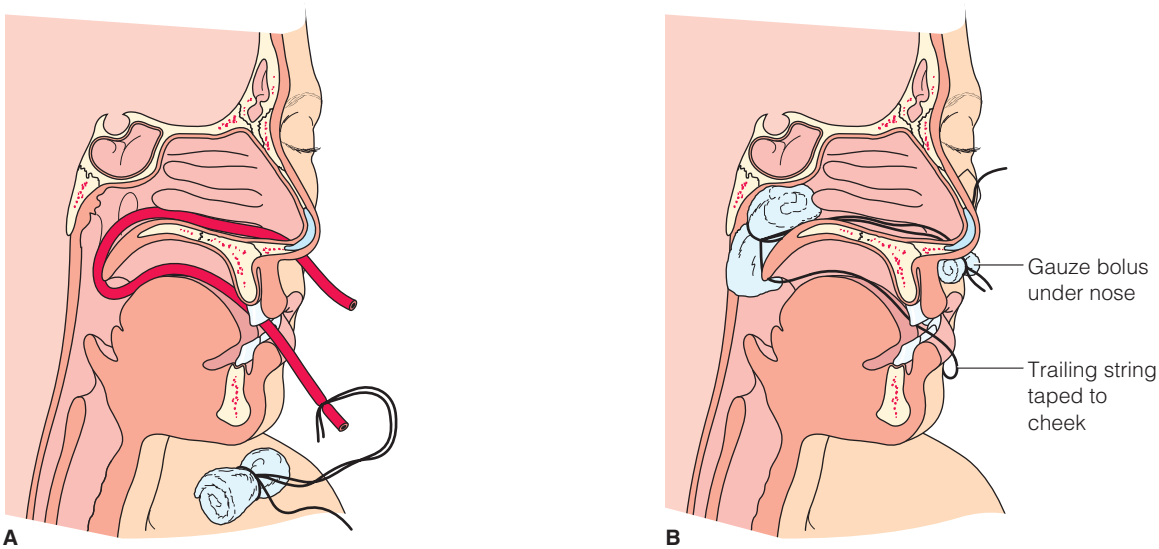


FIGURE 34.5 ■ Posterior nasal packing. *A*, A rubber catheter is inserted through the nose and out the mouth and attached to the packing. *B*, The catheter is withdrawn through the nose to position the packing in the posterior nasopharynx. Ties exiting through the nose and mouth are used to stabilise the packing in position and remove it when it is no longer needed

Posterior packs are usually left in place for 2 to 5 days. A loose anterior nasal pack may also be inserted. Posterior nasal packing is very uncomfortable and can cause respiratory and cardiovascular complications. Hypoxaemia is common; supplementary oxygen is administered. Endotracheal intubation may be necessary to maintain adequate ventilation and gas exchange. Narcotic analgesics are prescribed to manage the discomfort. Hypertension, arrhythmias and even acute myocardial infarction may occur in people with severe cardiovascular disease. Toxic shock syndrome is another potential complication of posterior nasal packing. The pack may occlude the eustachian tube and sinus openings, resulting in ear discomfort, possible otitis media or sinusitis. Oral and nasal dryness can be minimised by use of a high-humidity face tent. Nursing care of the person with nasal packing is outlined in the box below.

An indwelling catheter or inflatable nasal balloon may be used as an alternative to posterior nasal packing for effective tamponade. The catheter or nasal balloon is inserted through the nose into the nasopharynx, inflated and left in place for 2 to 3 days.

Surgery

Chemical or surgical cautery procedures may be used to sclerose involved vessels in the anterior aspect of the nose. The resulting scab must be left undisturbed until the mucosa has healed or further bleeding may occur.

Surgical procedures to control bleeding are often preferred to posterior nasal packing for posterior bleeding. The bleeding vessel may be cauterised using an endoscopic approach. In some cases, surgery is required to occlude the internal maxillary artery by ligation (tying off) or embolisation. These procedures may be done under either conscious sedation and local anaesthesia or general anaesthesia. Facial paralysis, paraesthesia, facial pain and dental injury are potential complications.

Nursing care

Assessment

Nursing assessment of the person with a nosebleed focuses on the immediate problem and possible underlying conditions.

- *Health history:* duration of current bleed; any identified precipitating factors such as trauma; history of prior nosebleeds; current medications; chronic conditions such as hypertension, bleeding disorders and so on.
- *Physical examination:* estimated amount of bleeding; presence of blood in oropharynx; vital signs; evidence of facial or nasal trauma.
- *Diagnostic tests:* haemoglobin, haematocrit and FBC as indicated; oxygen saturation; tests of organ function such as liver function tests (bilirubin, AST, ALT, LDH) or kidney function tests (serum creatinine and urea).

Nursing diagnoses and interventions

Nosebleeds can be frightening, particularly when they occur without preceding trauma. Nurses provide care for people with epistaxis in outpatient and emergency settings and may care for hospitalised people with nasal packing. Support, reassurance and education are important nursing roles related to epistaxis. Priority nursing concerns include assisting possible anxiety and monitoring to reduce the potential risk of aspiration.

Anxiety

The amount of blood lost in a nosebleed can be frightening. The sensation of blood draining down the throat and the inability to breathe through the nose contribute to anxiety. Spontaneous epistaxis may lead to fear of a major health problem such as high blood pressure.

CONSIDERATION FOR PRACTICE

Maintain an attitude of calm reassurance. By remaining calm and confident, the nurse reassures the person that the nosebleed is not a life-threatening event.

- Instruct the person to pinch the nares together at the bridge of the nose. *Most nosebleeds are anterior in origin; direct pressure usually stops the bleeding. Having the person place pressure on the nose provides a focus and helps restore a sense of control, reducing anxiety.*
- Encourage slow, deep breathing through the mouth. *Controlled mouth breathing maintains lung ventilation and reduces anxiety.*
- Provide a basin and tissues; encourage the person to expectorate blood, not swallow it. *These measures give the person greater control and reduce the fear of choking on blood.*
- Apply ice or a cold compress to the nose. *Cold causes vasoconstriction, reducing bleeding.*

CONSIDERATION FOR PRACTICE

Assess the person with nasal packing frequently for adequate oxygenation. Maintain supplemental oxygen as ordered. Cerebral hypoxia produces a sense of apprehension and fear.

Risk of aspiration

Anxiety and blood draining into the nasopharynx increase the risk of aspiration of blood into the trachea. When nasal packing is in place, the person is unable to breathe through the nose, increasing the risk of aspiration when food or fluids are consumed.

CONSIDERATION FOR PRACTICE

Position upright with head forward. Provide a basin for expectorating blood. These measures minimise the amount of blood draining down the nasopharynx and swallowed, reducing the risk of aspiration and minimising nausea from swallowed blood. Vomiting of swallowed blood increases the risk of aspiration.

NURSING CARE OF THE PERSON with nasal packing

- Continuously monitor oxygen saturation. Administer supplementary oxygen as ordered. *Posterior nasal packing causes hypoxaemia. Supplemental oxygen is given to maintain tissue oxygenation.*
- Frequently monitor vital signs and respiratory rate or pattern. *Posterior nasal packing increases the risk of respiratory and cardiovascular complications. Tachycardia and tachypnoea may be early signs of cardiac or respiratory compromise.*
- Inspect the mouth and oropharynx. Notify the doctor if the packing is seen in the oropharynx. *Misplacement of nasal packing can obstruct the upper airway.*
- Elevate the head of the bed. *Elevating the head of the bed facilitates ventilation.*
- Encourage deep, slow breathing through the mouth. Provide psychological support, reassurance and teaching. *Inability to breathe through the nose causes anxiety and fear.*
- Check for blood at the back of the throat and frequent swallowing. *Visible blood or frequent swallowing could indicate posterior bleeding.*
- Report haematemesis. *Bleeding from the posterior portion of the nose often drains down the nasopharynx and is swallowed. Haematemesis may indicate continued bleeding.*
- Apply cold compresses to nose. *An ice or cold compress decreases pain and promotes vasoconstriction, decreasing bleeding and swelling.*
- Provide for rest. *Rest reduces the metabolic demands and oxygen consumption.*
- Ensure adequate oral fluid intake. *Fluid intake helps maintain fluid balance and decreases dryness of oral mucous membranes because of mouth breathing.*
- Provide frequent oral hygiene. Use a bedside humidifier. *These measures reduce drying of oral mucous membranes and promote comfort.*

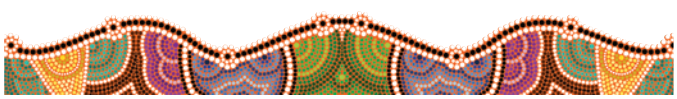
CONSIDERATION FOR PRACTICE

Position the person with nasal packing with the head elevated and on the side when asleep. This position reduces the risk of aspiration of oral secretions.

Community-based care

Following an episode of epistaxis, teaching for home care focuses on measures to prevent further bleeding. Include the following teaching topics:

- Avoid strenuous exercise for several days or weeks, depending on the severity of the nosebleed and its treatment.
- Do not blow the nose or engage in activities such as heavy lifting or bending that could increase pressure and dislodge the crust; sneeze with the mouth open to avoid increasing pressure in nasal vessels.
- For an anterior nosebleed, use petroleum jelly or a water-soluble lubricant to lubricate nasal mucosa and reduce the risk of spontaneous bleeding.
- Use a humidifier or vaporiser to minimise dryness of the mucous membranes.
- Do not forcefully blow the nose or pick the nose.
- For spontaneous nosebleed, seek medical evaluation for any possible underlying problem, such as hypertension or a bleeding disorder.



THE PERSON WITH NASAL TRAUMA OR SURGERY

The nose is the most commonly broken bone of the face. A nasal fracture (broken nose) usually is caused by a sports injury or trauma related to violence or motor vehicle crashes. The nasal septum normally divides the nose into two equal parts. Deviation of the septum can result from nasal trauma. Soft tissue trauma commonly accompanies nasal fracture.

Pathophysiology and manifestations

One or both sides of the nose may be broken. A *unilateral fracture* involves only one side of the nose. It causes little displacement or cosmetic deformity. It is usually not serious, but septal deviation and swelling can obstruct the airway. *Bilateral fractures* are more common, with depression or displacement of both nasal bones to one side. The nose appears flattened or deviated with an S or C configuration. *Complex fractures* may also involve the septum, ascending processes of the maxilla and frontal bones of the face.

Soft tissue trauma commonly accompanies nasal fracture. Mucous membrane tears cause epistaxis. Soft tissue haematomas (black eye) are also frequent. Swelling develops rapidly following the injury and may obscure the fracture. Bony crepitus may be felt on gentle palpation. Septal haematoma may develop, increasing the risk of infection. The manifestations of nasal fracture are listed in the box below.

MANIFESTATIONS Nasal fracture

- Epistaxis
- Deformity or displacement to one side
- Crepitus
- Periorbital oedema and ecchymosis
- Nasal bridge instability

Complications

Potential complications of nasal fracture include septal haematoma and abscess formation, septal perforation or deviation, and

cerebrospinal fluid (CSF) leakage. Septal haematoma can lead to complete and bilateral nasal obstruction. If undrained, haematoma increases the risk of staphylococcal abscess, which can lead to necrosis of septal cartilage and *saddle nose deformity*.

Septal deviation causes varying degrees of nasal obstruction. The septal cartilage bulges or deviates to one side, partially or totally obstructing the nares. Mild deviation is generally asymptomatic. Partial obstruction of air flow through one side may cause noisy breathing while awake and snoring during sleep. Major deviations can cause pain because of sinus obstruction or infection. They may also cause nosebleeds due to dryness of the nasal mucosa. Occasionally, the defect is severe enough to cause cosmetic deformity. Perforations are usually not serious and do not usually require repair unless obstruction or external deformity occurs.

Fractures of other facial bones may accompany a broken nose, particularly when facial trauma is severe. Fractures in the nasoethmoidal or frontal region can disrupt the dura, causing CSF leakage or rhinorrhoea. CSF rhinorrhoea is suspected if watery nasal drainage tests positive for glucose.

INTERPROFESSIONAL CARE

The main treatment goals for nasal fractures are to maintain a patent airway and prevent deformity. Respirations are closely monitored.

Diagnosis

Head and facial x-rays are done to identify the fracture and assess for other facial fractures. The intranasal cavity is examined using a nasal speculum to rule out septal haematoma. If a CSF leak is suspected, a CT scan is done. A radiopaque substance or fluorescein dye may be instilled into the intrathecal or lumbar subarachnoid space to identify the site of leakage.

Treatments

Ideally, the fracture is reduced early, before significant oedema develops. Nasal fractures heal rapidly. Simple reduction may be done in the emergency department with local anaesthesia. An external splint may be applied for 7 to 10 days to maintain proper alignment until healing occurs. The splint is padded to prevent skin breakdown. Ice may be gently applied to the face and nose to control oedema and bleeding. Nasal packing may be used to control epistaxis.

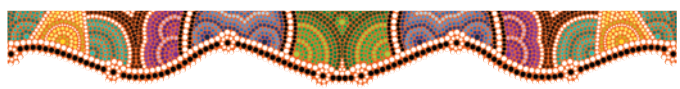
Surgery

Complex nasal fractures, nasal septal deviation or persistent CSF leakage may require surgical repair or realignment of nasal bones. Rhinoplasty with concurrent septoplasty is the most common procedure used to repair nasal fracture or a deviated nasal septum.

Rhinoplasty is surgical reconstruction of the nose. It is done to relieve airway obstruction and repair visible deformity of the nose following fracture. If oedema is excessive after nasal fracture, surgery is delayed for 7 to 10 days to allow swelling to subside. Using an intranasal incision, the nasal skin is lifted and the framework of the nose reshaped by removing, rearranging or augmenting bone or cartilage. The skin is then repositioned over the reconstructed frame. Prosthetic implants may help reshape the nose. Either local or general anaesthesia may be used; hospitalisation is often unnecessary. Following surgery, nasal packing is left in place for up to 72 hours to minimise bleeding and provide tissue support. A temporary plastic splint moulded to the shape of the nose is removed in 3 to 5 days. The splint protects the reshaped nose and helps to control swelling. Most swelling and bruising subside within 10 to 14 days; normal sensation returns within several months following surgery. Rhinoplasty generally has few complications.

Either a septoplasty or a submucosal resection may be done under local anaesthesia to correct a deviated septum. *Septoplasty* involves incising one side of the septum, elevating the mucous membrane and removing or straightening the deviated portion of septal cartilage. In a *submucosal resection*, bone and cartilage are removed. In both procedures, packing is applied to both sides of the nose to prevent bleeding and to keep the septal mucosa in the midline position.

Small defects in the cribriform plate, fovea ethmoidalis or sphenoid sinus associated with persistent CSF leakage may require endoscopic repair. Either a tissue graft or fibrin glue may be used to repair the defect. The graft or glue is held in place with absorbable packing. Large defects may require craniotomy for repair.



Nursing care

Health promotion

Teach all people—children and adolescents, in particular—about the importance of wearing appropriate protective equipment during exercise or sports. Reinforce the compulsory use of seat belts and air bags in vehicles to reduce the risk of facial injury in motor vehicle crashes.

Assessment

Focused nursing assessment for the person with a suspected nasal fracture includes:

- *Health history:* nature and circumstances of the injury; pain; ability to breathe through the nose.
- *Physical examination:* evident trauma, swelling, ecchymosis or deformity of the nose; vital signs, respiratory rate and ease; gently palpate nose and facial bones for crepitus; inspect oropharynx for drainage; test nasal discharge for glucose.

Nursing diagnoses and interventions

Nursing care for the person with nasal fracture focuses on controlling pain, bleeding and swelling. Airway management is a priority. Most nasal fractures are managed on an outpatient basis and education is a vital nursing function.

Risk of airway compromise

Immediately following nasal trauma and fracture, the airway is at risk of obstruction by bleeding and oedema. Deformity resulting from inappropriate fracture position during healing also can impair nasal airway clearance. This is a consideration when inserting nasogastric tubes or suctioning people with septal deviation.

CONSIDERATION FOR PRACTICE

Monitor airway patency. Oedema and bleeding may obstruct the airway, causing signs of respiratory distress such as tachypnoea, dyspnoea, shortness of breath, tachycardia and use of accessory muscles.

- Monitor cough effectiveness and ability to clear airway secretions. *Pain, oedema and nasal bleeding may impair the ability to cough effectively.*
- Maintain adequate hydration. Assess mucous membranes and skin turgor for evidence of dehydration. *Decreased oral fluid intake may lead to dehydration and thick, viscous secretions that are more difficult to expectorate.*

CONSIDERATION FOR PRACTICE

Have suction equipment available. Airway patency is a priority; oropharyngeal suctioning may be necessary to remove secretions and maintain a clear airway. Suctioning of the nasopharynx is avoided to prevent additional tissue trauma.

- Assess patency of both nares before inserting a nasogastric tube or feeding tube. If airflow is obstructed through one side, insert the tube through the unobstructed nare. Carefully monitor respiratory status following tube insertion. *The nasogastric tube is inserted through the unobstructed nare to avoid mucosal trauma; however, a large gastric tube may interfere with nasal breathing, necessitating close monitoring.*

Risk of infection

The person with a nasal fracture is at increased risk of infection. The nasal mucosa is a natural barrier to infection, and trauma increases the risk of invasion by pathogens. Septal haematoma can lead to abscess formation and staphylococcal infection. A cerebrospinal fluid leak indicates disruption of the dura, increasing the risk of ascending infection and meningitis.

CONSIDERATION FOR PRACTICE

Test watery, clear fluid dripping from the ear or nose for glucose. CSF will test positive for glucose on a glucose test strip.

- Avoid suctioning if possible. *Suctioning catheters could introduce microorganisms and cause additional trauma to tissues.*
- Monitor vital signs every 4 hours. *A rise in temperature may indicate infection.*
- Administer antibiotics as ordered. *Antibiotics may be prescribed to prevent abscess formation and, if CSF leakage is present, to prevent meningitis.*

Community-based care

Provide the following teaching when preparing the person with a nasal fracture for home care:

- Elevate the head of the bed and apply ice or cold packs to the nose for 20 minutes four times a day to reduce swelling.
- Swelling usually subsides in several days; bruising may persist for several weeks.
- It is difficult to determine the final cosmetic outcome until swelling has subsided.
- If indicated, discuss the potential benefits of rhinoplasty for fracture reduction or correction of this malformation.

If CSF leakage is present, also include the following instructions:

- Rest in bed with the head of the bed elevated to 30 to 45 degrees.
- Restrict fluid intake as ordered and take the prescribed diuretic to reduce intracranial pressure and CSF leakage.
- Distribute allowed fluids throughout the day.
- List name, purpose, effects and precautions for any prescribed medication.
- Avoid straining, blowing the nose, sneezing or vigorous coughing until allowed by the doctor.
- Immediately report manifestations of infection, including stiff neck, headache and fever, to the doctor.

Following rhinoplasty or septoplasty, provide the following instructions:

- Apply ice packs to the nose to relieve discomfort and reduce swelling.
- Elevate the head of the bed to decrease local oedema.
- Do not blow the nose for 48 hours after the packing is removed to prevent bleeding.
- Vigorous coughing or straining at stool may cause bleeding and should be avoided.
- Clean teeth and mouth frequently and increase fluid intake to decrease oral dryness due to mouth breathing.
- Bruising around the eyes and nose will last for several days.

THE PERSON WITH LARYNGEAL OBSTRUCTION OR TRAUMA

The larynx is the narrowest portion of the upper airway. As such, it is at risk of obstruction. Laryngeal obstruction is a life-threatening emergency. Blows to the neck or traumatic injuries may damage the larynx, interfering with its patency and function.

Pathophysiology and manifestations

The larynx may be partially or fully obstructed by aspirated food or foreign objects or by laryngospasm or oedema due to inflammation, injury or anaphylaxis. Anything that occludes the larynx can obstruct the airway. The most common cause of obstruction in adults is ingested meat that lodges in the airway. Risk factors for food aspiration include ingesting large boluses of food and chewing them insufficiently, consuming excess alcohol and wearing dentures. A foreign body in the larynx causes pain, laryngospasm, dyspnoea and inspiratory stridor. Aspirated foreign bodies may pass through the larynx into the trachea and lungs, causing pneumonitis.

Laryngospasm occurs due to repeated or traumatic intubation attempts, chemical irritation or hypocalcaemia. An acute type I hypersensitivity response may cause anaphylaxis with release of inflammatory mediators, leading to angioedema of upper airways and severe laryngeal oedema.

The most common manifestations of laryngeal obstruction are coughing, choking, gagging, obvious difficulty breathing with use of accessory muscles and inspiratory stridor. As the airway is obstructed, signs of asphyxia become apparent. Respirations are laboured and noisy with wheezing and stridor. Cyanosis may develop. Respiratory arrest and death may result without prompt treatment.

Trauma to the larynx can occur in motor vehicle crashes or assaults (e.g. blows to the neck or attempted strangulation). The larynx also may be traumatised during endotracheal intubation or tracheotomy. Trauma may fracture thyroid and/or cricoid cartilage, resulting in loss of airway patency. Soft tissue injuries can cause swelling that further impairs the airway. Manifestations of laryngeal trauma may include subcutaneous emphysema or crepitus, voice change, dysphagia and pain with swallowing, inspiratory stridor, haemoptysis and cough.

INTERPROFESSIONAL CARE

The treatment goal is to maintain an open airway. If airway obstruction is partial and the person is able to cough and move air in and out of the lungs, radiological and laryngoscopic examination may be done to locate the foreign body. An endotracheal tube may be inserted to maintain airflow through the larynx in spasm or an oedematous larynx. For anaphylaxis, adrenaline may be administered to reduce laryngeal oedema and relieve obstruction.

A CT scan is used to identify laryngeal fractures; however, emergency treatment may be required prior to diagnosis to ensure airway patency and preserve life. Soft tissue injuries may be managed conservatively with a bedside humidifier, intravenous fluids, antibiotics and corticosteroids to reduce oedema. More severe injuries require endotracheal intubation or immediate tracheostomy. Nursing care related to caring for the person with a tra.cheostomy is presented later in this chapter. See Chapter 36 for more information about endotracheal intubation and nursing care for the intubated person.

Nursing care

CONSIDERATION FOR PRACTICE

The priority of nursing care in laryngeal obstruction or trauma is restoring a patent airway to prevent cerebral anoxia and death. Laryngeal obstruction and trauma are medical emergencies requiring immediate intervention.

Closely monitor people at risk of laryngeal obstruction (e.g. following neck trauma, newly extubated people and people receiving medications with a high risk of anaphylaxis, such as intravenous antibiotics or radiological dyes) for manifestations of obstruction, including dyspnoea, nasal flaring, tachypnoea, anxiety, wheezing and stridor. Suction the airway as needed; small aspirated foreign bodies might possibly be removed by suctioning. If obstruction is complete, initiate a cardiopulmonary arrest procedure until the obstruction is relieved or the emergency response team arrives. Prepare to assist with emergency intubation or tracheotomy as needed. Provide emotional support, reassurance and teaching for the person and family to reduce anxiety.

Community-based care

Health promotion and teaching for home care focus on preventing laryngeal obstruction and early intervention techniques. Everyone should be aware of the risk factors for adult aspiration. Caution people who wear dentures to take small bites, chewing each bite carefully before swallowing. Discuss the relationship between excess alcohol intake and food aspiration. Participate in promoting training of the general public in CPR and the appropriate manoeuvres such as back, chest or abdominal thrusts. The more people who are adequately trained in emergency procedures, the more likely it is that emergency procedures will be initiated in a timely manner. Individuals with a known risk of anaphylaxis, such as people with a previous anaphylactic response and those allergic to bee venom, should wear a MedicAlert® tag and carry a bee-sting kit to allow early intervention to prevent severe laryngeal oedema and spasm.

THE PERSON WITH OBSTRUCTIVE SLEEP APNOEA (OSA)

Obstructive **sleep apnoea**, intermittent absence of airflow through the mouth and nose during sleep, is a serious and potentially life-threatening disorder. It affects at least 2% of middle-aged women and 4% of middle-aged men. Sleep apnoea is a leading cause of excessive daytime sleepiness and may

contribute to other problems such as poor work performance and motor vehicle crashes (Morton, 2012). Recent studies have linked sleep apnoea with an increased risk of hypertension, ischaemic heart disease and exacerbation of heart failure.

Types of sleep apnoea include obstructive and central. In *obstructive sleep apnoea*, the more common type, the respiratory drive remains intact but airflow ceases due to occlusion of the oropharyngeal airway. *Central sleep apnoea* is a rare neurological disorder that involves transient impairment of the neurological drive to respiratory muscles.

Risk factors

In addition to male gender, risk factors for obstructive sleep apnoea include increasing age and obesity (Bullock & Hales, 2012). Large neck circumference is also a known risk factor for obstructive sleep apnoea (Downey, 2014). Use of alcohol and other CNS depressants may contribute to sleep apnoea.

Pathophysiology

During sleep, skeletal muscle tone decreases (except the diaphragm). The most significant decrease occurs during rapid eye movement (REM) sleep. Loss of normal pharyngeal muscle tone permits the pharynx to collapse during inspiration as pressure within the airways becomes negative in relation to atmospheric pressure. The tongue is also pulled against the posterior pharyngeal wall by gravity during sleep, causing further obstruction. Obesity or skeletal or soft tissue changes that decrease inspiratory tone, such as a relatively large tongue in a relatively small oropharynx, contribute to the problem. Airflow obstruction causes the oxygen saturation, PO_2 and pH to fall and the PCO_2 to rise. This progressive asphyxia causes brief arousal from sleep, which restores airway patency and airflow. Sleep can be severely fragmented because these episodes may occur hundreds of times each night.

Manifestations

Narrowed upper airways produce loud snoring during sleep, often years before obstructive sleep apnoea occurs. Excessive daytime sleepiness, headache, irritability and restless sleep also are common manifestations. See the box below.

MANIFESTATIONS Obstructive sleep apnoea

- Loud, cyclical snoring
- Periods of apnoea lasting 15 to 120 seconds during sleep
- Gasping or choking during sleep
- Restlessness, thrashing during sleep
- Daytime fatigue and sleepiness
- Morning headache
- Personality changes, depression
- Intellectual impairment
- Impotence
- Hypertension

Complications

Recurrent episodes of apnoea and arousal during sleep have secondary physiological effects. Sleep fragmentation and loss of slow-wave sleep are thought to contribute to neurological and behavioural problems such as excessive daytime sleepiness, impaired intellect, memory loss and personality changes. Recurrent nocturnal asphyxia and negative intrathoracic pressure due to airway obstruction increase the workload of the heart. People with coronary heart disease may develop myocardial ischaemia and angina. Arrhythmias such as significant bradycardia and dangerous tachyarrhythmias may develop. Left ventricular function may be impaired and heart failure may occur. Systemic blood pressure remains high during sleep and may contribute to the systemic hypertension. Pulmonary hypertension also may develop. Sudden cardiac death is believed to be a potential fatal complication of obstructive sleep apnoea (Morton, 2012).

Obstructive sleep apnoea is a common condition in people who are morbidly obese. When these individuals undergo any surgery, sleep apnoea places them at significant risk of postoperative respiratory complications. Obesity not only interferes with chest movement and ventilation, but also increases metabolic demands and carbon monoxide production. Anaesthetic and analgesics used during surgery and in the postoperative period can lead to hypoxaemia due to muscle relaxation and depression of the respiratory drive. Individuals with OSA also experience an increased risk of pneumonia (Su et al., 2014).

INTERPROFESSIONAL CARE

The goal of care for OSA is to restore airflow and prevent the adverse effects of the disorder. Sustained weight loss may cure obstructive sleep apnoea.

Diagnosis

The diagnosis of OSA is based on *polysomnography*, an overnight sleep study. Several variables are recorded during the study, including:

- electroencephalogram and measurements of ocular activity and muscle tone
- recordings of ventilatory activity and airflow
- continuous arterial oxygen saturation readings
- heart rate.

Transcutaneous arterial PCO_2 readings also may be monitored during the study. Because sleep studies are time consuming and expensive, overnight monitoring of oxygen saturation by pulse oximetry may be used to confirm the diagnosis of sleep apnoea when symptoms indicate a high probability of the disorder. Nursing implications for pulmonary function studies and pulse oximetry are presented in Chapter 33. See Chapter 40 for more information about electroencephalography.

FIGURE 34.6 ■ A person using a nasal mask and CPAP to treat sleep apnoea

Treatments

Mild to moderate obstructive sleep apnoea may be treated by weight reduction, alcohol abstinence, improving nasal patency and avoiding the supine position for sleep. Although weight reduction often cures the disorder, maintaining optimal weight is difficult. Oral appliances designed to keep the mandible and tongue forward also may be prescribed.

Nasal continuous positive airway pressure (CPAP) is the treatment of choice for obstructive sleep apnoea. Positive pressure generated by an air compressor and administered through a tight-fitting nasal mask (see Figure 34.6) splints the pharyngeal airway, preventing collapse and obstruction. With proper training, this device is well tolerated by the person. Nasal airways can become dry and irritated with CPAP, so an in-line humidifier or a room humidifier may be utilised. A newer device, the bilateral BiPAP ventilator, delivers higher pressures during inhalation and lower pressures during expiration, providing less resistance to exhaling.

Surgery

Tonsillectomy and adenoidectomy may relieve upper airway obstruction in some people. Excision of obstructive tissue from the soft palate, uvula and posterior lateral pharyngeal wall may be accomplished by *uvulopalatopharyngoplasty (UPPP)*. Although only about 50% of these surgeries are successful in treating sleep apnoea, UPPP is useful in selected cases. In severe cases, tracheostomy may also be performed to bypass the area of obstruction.

Nursing care

Obstructive sleep apnoea usually is treated in the home. Nursing care focuses on teaching the person and family about equipment use and strategies to decrease contributing factors such as obesity and alcohol intake. The following nursing diagnoses are appropriate for individuals with sleep apnoea:

- *Disturbed sleep pattern* related to repeated apnoeic episodes manifested by continual interruptions to the sleep cycle.
- *Fatigue* related to interrupted sleep patterns manifested by tiredness and lack of attention.
- *Ineffective breathing pattern* related to obstruction of upper airway during sleep manifested by alterations in the normal sleep and breathing cycle.
- *Impaired gas exchange* related to altered lung ventilation during obstructive episodes manifested by changes in blood chemistry when assessed.
- *Risk of injury* related to daytime somnolence and altered judgment manifested by increasing number of accidents due to inattention.
- *Risk of sexual dysfunction* related to impotence resulting from sleep apnoea manifested by inability to achieve or maintain an erection.

Community-based care

Effective sleep apnoea management depends on the person's willingness to participate in care. Provide teaching about the following topics:

- relationship between obesity and sleep apnoea
- plans, resources and referrals as needed for weight loss (e.g. programs such as Weight Watchers to provide additional support)
- relationship of alcohol and sedatives to sleep apnoea; referral to an alcohol treatment program or Alcoholics Anonymous as indicated
- how to use CPAP if ordered
- the importance of using CPAP continuously at night
- measures to reduce airway dryness, including supplemental humidity and an adequate fluid intake to maintain moist mucous membranes.

If a support group for people with sleep apnoea syndrome is available in the local area, refer the person and family to the group.

UPPER RESPIRATORY TUMOURS

Although tumours of the upper respiratory tract are relatively uncommon, they have the potential to impair the upper airways and interfere with breathing and ventilation of the lungs. Of the upper respiratory tract structures, the larynx is affected by abnormal growths most often.

THE PERSON WITH NASAL POLYPS

Nasal polyps are benign grape-like growths of the mucous membrane lining the nose. These benign tumours can interfere with air movement through nasal passages or obstruct sinus

openings, leading to sinusitis. They usually affect people who have chronic allergic rhinitis or asthma.

Pathophysiology and manifestations

Chronic irritation and swelling of the mucous membranes from allergic rhinitis may cause slow polyp formation. Polyps form in areas of dependent mucous membrane, presenting as pale, oedematous masses covered with mucous membrane. They are usually bilateral and have a stem-like base, making them fairly movable. Polyps can continue to enlarge, eventually becoming larger than a grape. Polyps may be asymptomatic, although large polyps may cause nasal obstruction, rhinorrhoea and loss of sense of smell. Manifestations of sinusitis may develop. The voice may have a nasal tone. Asthmatics who have nasal polyps may have an associated aspirin allergy of which they are not aware.

INTERPROFESSIONAL CARE

When polyps occur in conjunction with an acute upper respiratory infection, they may regress spontaneously with resolution of the infection. When symptomatic, polyps may be managed with topical corticosteroid nasal sprays or low-dose oral corticosteroids to shrink the oedematous polyps and manage allergic symptoms. However, polyps continue to enlarge when corticosteroid therapy is discontinued and therefore surgery is sometimes required.

Nursing care

Teaching about home care following polypectomy is the primary nursing responsibility for the person with nasal polyps. Provide postoperative care instructions and discuss measures to reduce the risk of bleeding:

- Apply ice or cold compresses to the nose to decrease swelling, promote comfort and prevent bleeding.
- Avoid blowing the nose for 24 to 48 hours after nasal packing is removed.
- Avoid straining at stool, vigorous coughing and strenuous exercise.

Discuss manifestations of possible bleeding, such as frequent swallowing or visible blood at the back of the throat. Swallowed blood may cause nausea and vomiting. Encourage the person to rest for 2 to 3 days after surgery to reduce the risk of bleeding. Instruct to increase fluid intake and clean mouth frequently to reduce oral dryness associated with mouth breathing while nasal packing is in place.

THE PERSON WITH A LARYNGEAL TUMOUR

Laryngeal tumours may be either benign or malignant. Benign tumours of the larynx include papillomas, nodules and polyps. People who chronically shout, project or vocalise in an abnormally high or low tone, abusing the voice, are at risk of developing benign laryngeal tumours. In adults, vocal cord nodules are often referred to as ‘singer’s nodules’; public speakers may also develop them. Voice abuse also contributes to the development of vocal cord polyps, as do cigarette smoking and chronic irritation from industrial pollutants. Malignant tumours of the larynx, although uncommon, can have devastating effects if diagnosis and treatment are delayed.

FAST FACTS

- Age-standardised mortality rates for laryngeal cancer have decreased to 0.8 deaths per 100 000 people in 2012, from 1.9 in 1981 (Australian Institute of Health and Welfare (AIHW), 2015).
- In 2013, 220 people died from laryngeal cancer in Australia (Australian Bureau of Statistics, 2015).
- A person who has had a surgical removal of the larynx (laryngectomy) is known as a laryngectomee.

Risk factors

Men are affected by laryngeal cancer more than three times as often as women. Cancer of the larynx usually develops between ages 50 and 70. Tobacco use is the main risk factor for laryngeal cancer: the risk of developing laryngeal cancer is significantly greater in smokers (cigarette, pipe or cigar) than in non-smokers. A person who smokes one pack a day is 20 times more at risk of laryngeal cancer than a non-smoker (Cancer Council, 2015). Alcohol consumption is a significant co-factor in increasing the risk. Other risk factors include poor nutrition, human papillomavirus infection, exposure to asbestos and other occupational pollutants. Race is also considered a risk factor, with Aboriginal and Torres Strait Islander Australians and Maori New Zealanders experiencing higher rates of laryngeal cancer (AIHW, 2013; Soeberg et al., 2012).

Pathophysiology and manifestations

Papillomas are small, wart-like growths believed to be viral in origin. Polyps and nodules may develop on the vocal cords of the larynx as a result of voice abuse (see Figure 34.7). Nodules occur as paired lesions on the free edges of the vocal cords. Hoarseness and a breathy voice quality are manifestations of benign vocal cord tumours.

Laryngeal cancer

Squamous cell carcinoma is the most common malignancy of the larynx. Changes in the laryngeal mucosa occur over time as it is subjected to noxious irritants such as cigarette smoke. White, patchy, precancerous lesions known as *leucoplakia* appear. Red, velvety patches, called *erythroplakia*, are thought to represent a later stage of carcinoma development. The initial cancerous lesion, carcinoma in situ (CIS), is superficial. Malignant cells replace the lining layer, but do not invade into deeper

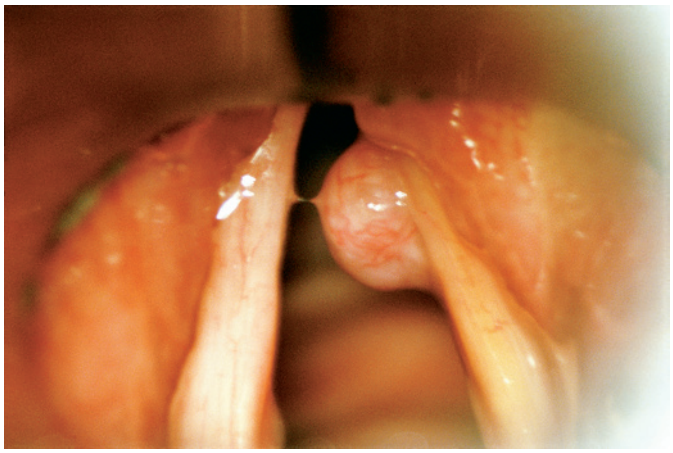


FIGURE 34.7 ■ Laryngoscopy showing a polyp on the left vocal cord

Source: Dr M.A. Ansary/Science Source.

tissues. Untreated, most CIS lesions develop into squamous cell cancer (American Cancer Society, 2015). Laryngeal cancer spreads both by direct invasion of surrounding tissues and by metastasis. It may metastasise to the lungs; however, metastases from other cancers to the larynx are rare.

Laryngeal cancer may develop in any of the three areas of the larynx: the glottis, the supraglottis and the subglottis. Manifestations vary according to the site of the lesion.

Lesions of the true vocal cords or glottis account for nearly 60% of all laryngeal cancers. Fortunately, these cancers tend to be well differentiated and slow growing. Metastasis occurs late in the course of the disease because of a limited lymphatic supply. The usual symptom of glottic cancer is hoarseness or a change in the voice because the tumour prevents complete closure of the vocal cords during speech.

Approximately 35% of laryngeal cancers develop in the supraglottic area, which includes the epiglottis, aryepiglottic folds, arytenoid muscles and cartilage, and false vocal cords (see Figure 34.8). Lymphatic supply to this region of the larynx is rich; tumours often invade locally and metastasise early. Symptoms often do not develop until the tumour is relatively large, delaying diagnosis. Manifestations of supraglottic cancer include painful swallowing, sore throat or a feeling of a lump in the throat. Later manifestations include dyspnoea, foul breath and pain that radiates to the ear.

Subglottic tumours (below the vocal cords) are the least common, accounting for the remaining 5% of laryngeal tumours. They often are asymptomatic until the enlarging tumour obstructs the airway. Common manifestations of laryngeal cancer are listed below.

MANIFESTATIONS Laryngeal cancer

- Hoarseness
- Change in the voice
- Painful swallowing
- Dyspnoea
- Foul breath
- Palpable lump in neck
- Earache

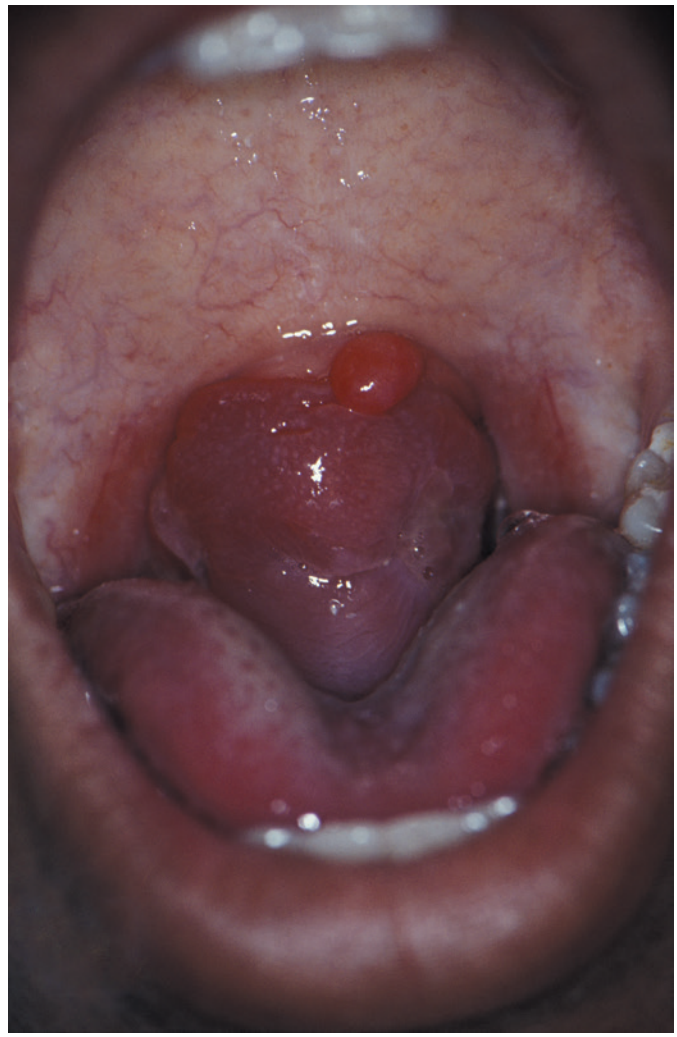


FIGURE 34.8 ■ Cancer of the larynx and epiglottis

Source: Biophoto Associates/Science Source.

INTERPROFESSIONAL CARE

Benign laryngeal tumours may resolve with correction of the underlying problem, such as voice training with a speech therapist or smoking cessation. Treatment of laryngeal malignancy varies with the extent of the cancer. Early diagnosis and treatment are important: 75% of individuals with small, localised cancer are still alive after 5 years. As the stage at diagnosis advances, 5-year survival rates drop to approximately 15% (AIHW, 2015).

Diagnosis

- *Direct* or *indirect laryngoscopy* is used for initial evaluation when laryngeal cancer is suspected. A fibreoptic laryngoscope is used for direct laryngoscopy; mirrors are used to visualise the larynx in indirect laryngoscopy.
- *Biopsy* is obtained from suspicious lesions to examine the cells. Biopsy is usually obtained under general anaesthesia or conscious sedation. Tissue may be obtained via endoscopy or by fine-needle aspiration of the mass.

- *Imaging studies* such as CT scan, MRI and chest x-ray are obtained to evaluate the size of the mass, possible extension into deeper tissues, involvement of lymph nodes and possible metastasis to the lungs. A barium swallow may be done to evaluate the effects of the tumour on swallowing. A positron emission tomography (PET) scan also may be done (possibly in conjunction with CT) to detect tumour metastasis.

Treatments

An inhaled steroid spray may be used for vocal cord polyps. In some cases, surgical excision of benign nodules or polyps is required. This usually is performed via laryngoscopy, using microforceps or a laser. A biopsy of the tumour is done to rule out malignancy.

Laryngeal cancer treatment is determined by *staging* the cancer. Information such as tumour size and location (T), number of involved lymph nodes (N) and presence or absence of metastases (M) is combined to assign a stage, designated by Roman numerals I to IV. The staging also takes into consideration whether the laryngeal cancer is supra-, peri- or subglottic.

RADIATION THERAPY Radiation therapy is often the treatment of choice for early laryngeal cancer. Radiation disrupts the DNA of the cell, causing it to die. External radiation commonly is used; brachytherapy, implants of iridium seeds placed into hollow plastic needles that are inserted directly into or near the tumour site during surgery to deliver radiation, is less frequently used for laryngeal or hypopharyngeal cancer. Radiation therapy is extremely effective for treating glottic cancer, with cure rates equal to those achieved by surgery. Radiation therapy preserves the voice, although the tone or timbre of the voice may be affected.

Radiation therapy may be used in combination with chemotherapy (*chemoradiotherapy*) to treat more advanced laryngeal cancers. In some institutions, and depending on the stage of cancer, chemoradiotherapy may be used in place of laryngectomy (Sheahan, 2014).

Radiation therapy also may be used in conjunction with surgery to destroy any remaining cancerous cells or as a palliative treatment for advanced tumours. See Chapter 13 for more information about radiation therapy and its nursing implications.

CHEMOTHERAPY Chemotherapy is used in combination with radiation therapy as the primary treatment for some laryngeal cancers. It also is used to treat distant metastasis and for palliation when the tumour is unresectable. See Chapter 13 for the nursing implications for chemotherapy.

Surgery

The type of surgery used to treat laryngeal cancer is based on site, size and invasiveness of the tumour into the larynx and surrounding tissues. The goals of surgery are to remove the malignancy, maintain airway patency and achieve optimal cosmetic appearance.

Carcinoma in situ, vocal cord polyps and early vocal cord cancers may be removed by laser during a laryngoscopy procedure. The cure rate for early tumours using this method is excellent. This surgery may be performed on an outpatient basis. The degree of trauma to the vocal cords varies, depending on the size of the lesion. The voice is preserved, but total voice rest with whispering only may be ordered for a week or more following surgery. In some cases, a temporary tracheostomy may be done at the time of surgery to ensure that swelling does not interfere with airway patency. Once the tracheostomy tube is removed and the opening is closed, the person can eat, speak and breathe normally.

Laryngectomy, removal of the larynx, may be necessary. A partial laryngectomy (hemilaryngectomy, vertical partial laryngectomy) may be used for tumours localised to a portion of the larynx with limited extension beyond the larynx. In a partial laryngectomy, 50% or more of the larynx is removed. The voice generally is well preserved, although it may be changed by the surgery. A tracheostomy tube may be inserted for early postoperative airway management. It is usually removed in 5 to 7 days as postoperative swelling subsides and the stoma is allowed to close. Normal speaking, breathing and swallowing are restored. If the epiglottis has been removed, careful monitoring for aspiration is necessary. Enteral tube feedings or parenteral nutrition may be required for several weeks after surgery. Swallowing techniques to prevent aspiration are taught.

A *total laryngectomy* is required for cancers that extend beyond the vocal cords. The entire larynx is removed, along with the epiglottis, thyroid cartilage, several tracheal rings and the hyoid bone. Because the trachea and the oesophagus are permanently separated by this surgery (see Figure 34.9), there

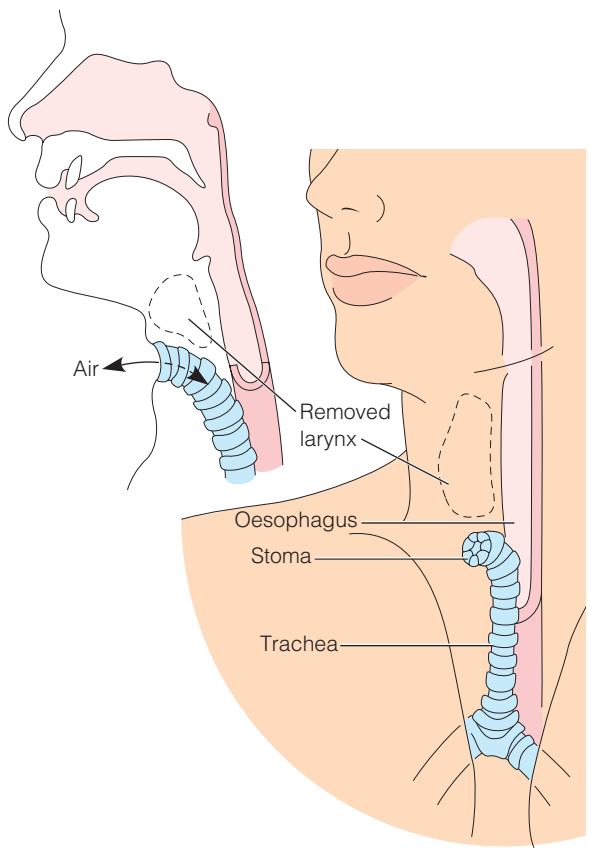


FIGURE 34.9 ■ Following a total laryngectomy, the person has a permanent tracheostomy. No connection between the trachea and oesophagus remains

is no risk of aspiration during swallowing. Normal speech is lost and a permanent tracheostomy is created. The tracheostomy tube inserted during surgery may be left in place for several weeks and then removed, leaving a natural stoma, or it may be left in place permanently. See below for nursing care of the person undergoing a total laryngectomy. Procedure 34.1 below outlines tracheostomy care.

If cervical lymph nodes are involved but there is no evidence of distal metastasis, *radical* or *modified neck dissection* may be performed along with total laryngectomy. In a radical neck dissection, all soft tissue from the lower edge of the mandible down to the clavicle is removed, including cervical lymph nodes, the sternocleidomastoid muscle, internal jugular vein, cranial nerve XI (spinal accessory) and submaxillary

NURSING CARE OF THE PERSON having a total laryngectomy

PREOPERATIVE CARE

- Provide routine preoperative care and teaching as explained in Chapter 3.
- Assess knowledge and understanding of the diagnosis and proposed surgery. Clarify information and reinforce previous teaching as needed. *A clear understanding by the person and family of the purpose, anticipated benefits and consequences of total laryngectomy prior to surgery is vital to promote postoperative recovery.*
- Assess anxiety levels of the person and family related to the diagnosis and proposed surgery. *High levels of anxiety interfere with learning and the ability to cooperate in care. Interventions to reduce anxiety may be required prior to teaching and providing preoperative instructions.*
- Without increasing fear, emphasise that total laryngectomy results in a loss of speech and that the person will breathe through a permanent stoma in the neck. *Although person and family members may verbalise an understanding of the loss of speech following surgery, they may believe that verbal communication will still be possible through the stoma.*
- Establish a means of communicating postoperatively, using an alphabet board, eye or hand signals, or other strategies. *Learning techniques for communicating preoperatively decreases the person's and family's postoperative anxiety. Long-term speech rehabilitation measures, such as the tracheoesophageal puncture, are not appropriate for use in the immediate postoperative period.*
- Point out that surgery will affect the senses of taste and smell, and hence eating, in the initial postoperative period. Reassure that nutritional and fluid needs will be met with intravenous or enteral feedings until eating can be resumed. *The person may not be prepared for the effect of surgery on taste and smell and, therefore, the enjoyment of food.*
- If possible and desired by the person and family, arrange a visit by a person post laryngectomy who effectively uses an alternative form of verbal communication. *The person and family may feel more comfortable expressing their fears and asking questions of someone who has gone through the same experience they are facing.*

POSTOPERATIVE CARE

- Provide routine postoperative nursing care and monitoring as explained in Chapter 3.
- Frequently monitor airway patency and respiratory status, including respiratory rate and pattern, lung sounds and oxygen saturation. *Excessive or retained respiratory secretions can impair gas exchange, increase the work of breathing and lead to complications such as pneumonia.*
- Encourage deep breathing and coughing. *Deep breathing helps ensure adequate ventilation of lower airways; coughing helps to move secretions out of airways.*
- Elevate the head of the bed. *The upright position promotes effective ventilation of the lungs and reduces oedema and swelling of the neck.*
- Maintain humidification of inspired gases. *With a tracheostomy, humidification of inspired air in the upper airways is lost. Humidified air helps maintain moist mucous membranes and secretions, promoting secretion removal by coughing or suctioning.*
- Maintain an adequate fluid intake (intravenously, enterally and orally when allowed). *Adequate hydration keeps secretions liquid and mucous membranes moist.*
- Suction via tracheostomy using sterile technique as needed. *Surgery, impaired nutrition and the effects of radiation therapy may cause fatigue and a weak cough effort. Suctioning may be necessary to clear secretions and maintain airway patency.*
- Provide tracheostomy care as needed (see Procedure 34.1). *Periodic cleaning of the tracheostomy tube is necessary to remove accumulated secretions and maintain airway patency.*
- Teach to protect the stoma from particulate matter in the air with gauze square or other stoma protector. *Permanent tracheostomy results in loss of the protective mechanisms of the upper airway that prevent foreign material from entering the lungs.*
- Instruct to support the head when moving in bed. *Additional head support reduces the strain on tissues in the operative area.*
- Place the call bell within easy reach at all times; answer the call bell promptly. *The person who is unable to speak needs reassurance that help is within reach at all times.*
- Encourage family members to remain present when possible. *Supportive family presence helps reassure the person that they will not be left alone or helpless.*
- Spend as much time as possible with the person. When leaving the room, specify the time when you will return. *These measures help establish trust and relieve anxiety.*

PROCEDURE 34.1 Providing tracheostomy care

GATHER SUPPLIES

- Dressing pack
- Sterile suction catheter and glove kit
- Sterile disposable replacement inner cannula, if appropriate
- Cleaning solutions (e.g. sterile normal saline)
- Sterile 4 × 4 gauze dressings (not cotton filled) or precut dressing
- Sterile cotton-tipped applicators
- Single-use tracheostomy tube holder or cotton twill ties
- Scissors
- Clean exam gloves

BEFORE THE PROCEDURE

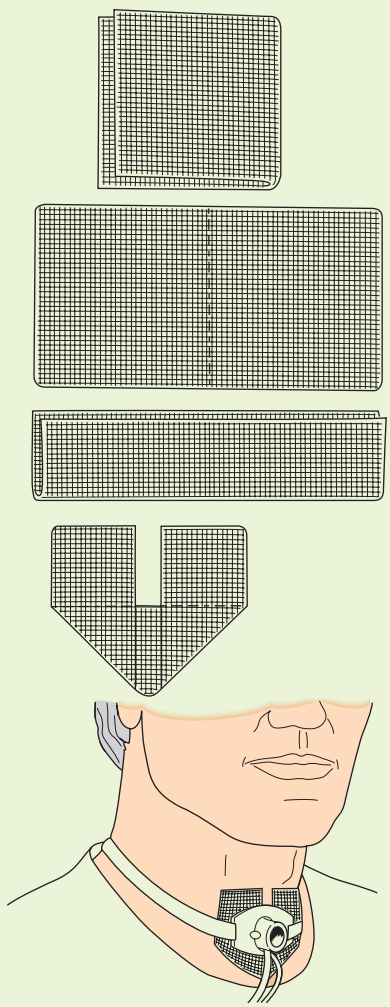
Provide for privacy. Explain the procedure. Provide for a means of communication (e.g. eye blinking or raising a finger to indicate distress). If the person's condition permits, provide a pencil and paper for questions. Place in semi-Fowler's or Fowler's position to facilitate lung ventilation. Assess lung sounds; suction the tracheostomy using sterile technique as needed.

DURING THE PROCEDURE

- Use standard precautions.
- Wearing a clean disposable glove, remove the tracheostomy dressing. Dispose of the glove and dressing.
- Open sterile supplies and add additional materials (dressing, cleaning solution etc). Don gloves.
- Clean area using aseptic technique.
- If the tracheostomy tube has an inner cannula that can be removed for disposal or cleaning, remove the tube and set it aside for disposal or clean with solution. Cleanse the flange of the outer cannula.
- Clean reusable inner cannulas using a small brush, pipe cleaners or cotton-tipped applicators.
- Rinse the inner cannula thoroughly in normal saline. Tap it gently against the inner aspect of the sterile bowl to remove excess liquid.
- Suction the outer cannula using sterile technique.
- Replace the inner cannula into the tracheostomy tube.
- Replace the dressing, using either a commercially prepared tracheostomy dressing or an opened gauze 4 × 4 refolded into a V shape (see the accompanying figure). Do not cut the dressing or use a cotton-filled dressing to prevent aspiration of foreign material into the respiratory tract.
- Apply a clean tracheostomy holder or clean ties.
- Once the clean ties are secured, remove the old ties.

AFTER THE PROCEDURE

Assess breathing and tolerance of the procedure. Dispose of supplies and used solutions. Wash hands. Chart the procedure and any observations made during the procedure such as amount, colour and consistency of sputum and appearance of the incision.



Steps for folding a gauze 4 × 4 into a tracheostomy dressing

salivary gland. Extensive tissue dissection can result in significant deformity. Skin grafts or flaps may be used to close the wound. Bellovac drains are placed in the wound to prevent haematoma and extensive oedema formation. After surgery, the person may have difficulty lifting and turning the head because of muscle loss. Resection of the spinal accessory nerve causes shoulder drop on the affected side. In a modified neck dissection, neck contents are removed, with the exception of the sternocleidomastoid muscle, internal jugular vein and spinal accessory nerve.

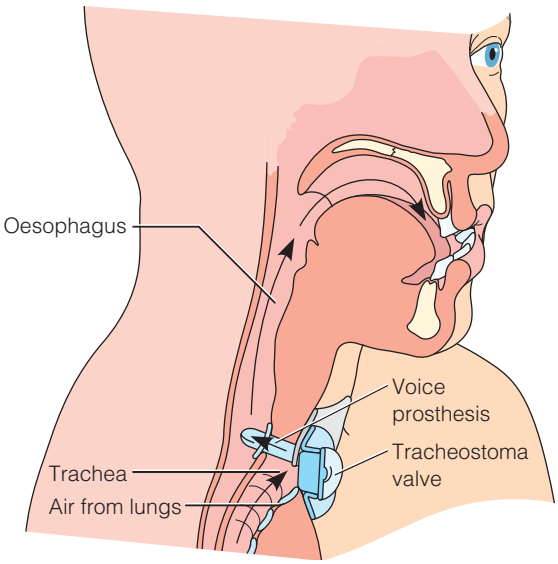


FIGURE 34.10 ■ The tracheoesophageal prosthesis allows diversion of air from the trachea through a one-way valve into the oesophagus and oropharynx, producing speech when the tracheostomy stoma is occluded. The one-way valve prevents food from entering the trachea

A gastrostomy also may be performed to maintain nutrition in the person with laryngeal or hypopharyngeal cancer. See Chapter 22 for more information about caring for the person with a gastrostomy tube.

Speech rehabilitation

Various techniques may be used to restore speech after total laryngectomy. *Tracheoesophageal puncture (TEP)* is the usual method used to restore speech. A small fistula is created between the posterior tracheal wall and the anterior oesophagus. A small, one-way shunt valve is fitted into the fistula (see Figure 34.10). Occluding the tracheostomy stoma with a finger forces exhaled air through the valve into the oesophagus and hypopharynx, creating vibration and sound. The muscles of speech are used to form words. The one-way valve prevents aspiration from the oesophagus into the trachea. An external tracheostoma valve may be used to avoid using the hand to occlude the stoma. This device covers the entire tracheal stoma and closes during exhalation, forcing air directly into the voice prosthesis. Not all post-laryngectomy people are candidates for this device because its use requires motivation and manual dexterity.

Oesophageal speech uses swallowed air to create sound and form words as it is expelled in a controlled belch. The pharyngoesophageal segment vibrates with the belch, creating sound. Muscles of the mouth and tongue are used to control the sound and to form words. This form of speech takes practice and fluent speech may not be restored.

Several speech generators (electrolarynx) are available. One type is held to the neck and creates vibrations that are transmitted to the neck and into the mouth (see Figure 34.11A). The transmitted vibrations are formed into words using the normal muscles of speech. Another device delivers a tone into the mouth via a plastic tube inserted into the corner of the mouth (see Figure 34.11B). The lips, tongue and mouth muscles are used to form the sound into words.

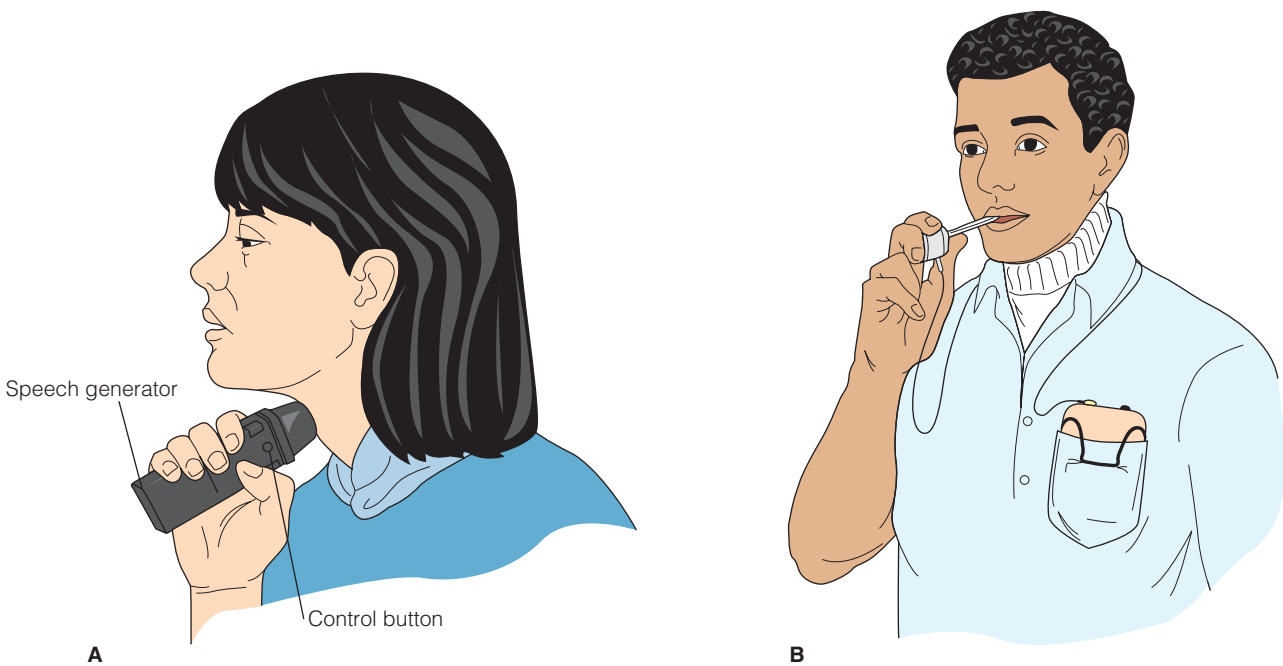
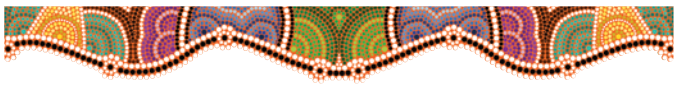


FIGURE 34.11 ■ Speech generators. *A*, The person holds the vibrating tip of the speech generator against the throat, using the mouth to form words. *B*, A plastic handpiece of the generator is held in the corner of the mouth. The audible tone produced by the generator is formed into words



Nursing care

Nurses can be instrumental in early identification and treatment of laryngeal disorders by emphasising the need for the person with new chronic hoarseness to seek treatment.

Health promotion

Health promotion activities to prevent laryngeal cancer focus on preventing smoking by children, adolescents and young adults, and promoting smoking cessation in people who do smoke. Activities to promote abstinence or moderate alcohol use also are beneficial in reducing a significant risk factor for laryngeal cancer.

Assessment

Nurses can be instrumental in identifying early signs of laryngeal cancer, facilitating early diagnosis and treatment.

- *Health history:* current symptoms, including voice change, difficulty swallowing, throat pain; risk factors such as voice abuse, family history of cancer, occupational exposures; smoking history, use of alcohol and amount consumed.
- *Physical examination:* voice character; general appearance and apparent state of health; swallowing ability; visible or palpable mass in neck.

Nursing diagnoses and interventions

Nursing care for the person with a benign tumour of the larynx focuses on maintaining a patent airway and teaching about the disorder and strategies to prevent its recurrence. The person with laryngeal cancer has multiple nursing care needs. The risk of impaired verbal communication is significant. Dysphagia may interfere with swallowing and nutrition. Nutrition also may be impaired by radiation, chemotherapy and surgery. The diagnosis of cancer is frightening for most people, no matter what the potential for cure is with treatment. See the nursing care plan that follows for additional nursing diagnoses and interventions.

CONSIDERATION FOR PRACTICE

During the immediate postoperative period, closely monitor for signs of airway obstruction, such as laboured breathing or inspiratory stridor. The larynx is the narrowest portion of the upper airways. Tissue oedema following surgery can further restrict the airway, interfering with lung ventilation and gas exchange.

Risk of impaired airway clearance

Following resection of a benign or malignant vocal cord nodule, local tissue oedema may interfere with airway patency.

- Apply cold packs to the neck as ordered or indicated. *Cold application constricts local blood vessels and reduces oedema development.*
- Withhold food and fluids until the cough and gag reflexes have returned. *Local anaesthesia used during removal of benign tumours and nodules impairs the cough and gag reflexes, increasing the risk of aspiration.*

Impaired verbal communication

Treatment of laryngeal cancer often alters the quality of the voice, results in short-term restriction of speaking or, in the case of total laryngectomy, causes loss of the voice. The person ultimately determines treatment choices for laryngeal cancer; some choose to forgo laryngectomy to avoid voice loss when the chance for long-term success and cancer cure is minimal.

- Prior to surgery, assess for additional obstacles to communication. *Communication may be impaired by hearing loss, illiteracy or weakness associated with the disease process, altering the ability to use alternative communication strategies.*
- Assess the importance of verbal communication to self-concept, occupation and lifestyle. *Many factors influence adaptation to the loss of normal verbal communication. If the ability to speak is central to an occupation (e.g. school teacher, singer) or self-concept (e.g. politician or barrister), adapting to a total laryngectomy may be difficult. For these people, laryngectomy may mean a loss of employment or career.*

CONSIDERATION FOR PRACTICE

Prior to surgery, introduce non-verbal communication strategies such as pencil and paper or an alphabet board. Encourage the person to practise using each method and to choose the most acceptable one. Having the person determine a means of communication prior to surgery helps to alleviate anxiety and increases the sense of control.

- Arrange consultation with a speech therapist about alternative forms of oral communication prior to surgery if possible. *Determining a means of communicating on a continuing basis prior to surgery helps to relieve fear of inability to communicate and may guide the choice of a surgical procedure.*

CONSIDERATION FOR PRACTICE

After surgery, assess frequently. Place the call bell at hand. The presence of a caring nurse helps to decrease anxiety and promotes communication. Knowing that help is readily available enhances feelings of security and decreases anxiety.

- Reinforce teaching about alternative communication strategies. *Anxiety or information overload may impair the ability to retain information; reinforcement facilitates learning.*

- Maintain a positive attitude about postoperative communication, but do not promote unrealistic expectations. *Not everyone is able to use all alternative methods of verbal communication after the laryngectomy. Some people remain non-verbal.*
- If desired, arrange a visit by a rehabilitated laryngectomy person who has mastered an alternative form of verbal communication and has a positive attitude about rehabilitation. *Many people and their families find that they are better able to communicate their fears with someone who has gone through the same experience they are facing.*

Impaired swallowing

Disruption of laryngeal structures by the tumour itself or due to radiation or surgery can impair the swallowing mechanism. Additionally, even when a total laryngectomy has been performed and a connection between the oropharynx and trachea no longer exists, swallowing may cause fear of choking.

- Maintain intravenous fluids and enteral feedings or parenteral nutrition until adequate food and fluids can be ingested orally. *It is important to maintain nutritional and fluid balance until normal eating can be resumed.*

NURSING CARE PLAN A person with total laryngectomy

David Tom is a 61-year-old accountant who is divorced and has two adult children. He has smoked two packets of cigarettes daily since high school and usually has three or four rum and cokes each evening. After several months of persistent sore throat and hoarseness, Mr Tom was diagnosed with cancer of the larynx. He has been admitted to the surgical care unit from the critical care unit 2 days post total laryngectomy.

ASSESSMENT

Mr Tom's vital signs are stable: BP 146/84, P 92 and regular, R 18, T 36.7°C axillary. A tracheostomy tube is sutured in place and he is receiving humidified oxygen at 28% per tracheostomy collar. Pulse oximetry is 94%. He is receiving continuous tube feeding per nasogastric feeding tube. Two Bellovac wound drains are present in the right neck area. A moderate amount of oedema is noted in the right facial and submandibular area. Mr Tom is ambulatory within the room.

DIAGNOSES

- *Risk of airway compromise* related to postoperative oedema manifested by difficulty in clearing secretions.
- *Risk of ineffective breathing* related to pain and anxiety manifested by decreased oxygen intake and symptoms of increased pain and anxiety.
- *Disturbed body image* related to total laryngectomy and presence of tracheostomy stoma manifested by the person self-reporting body image issues or reluctance to engage in the community.
- *Impaired verbal communication* related to total laryngectomy manifested by difficulty communicating verbally when required.
- *Pain* related to surgical procedure manifested by increased respiratory rate and pulse and the person self-reporting discomfort.
- *Nutritional imbalance* related to insufficient oral intake manifested by lack of appetite and pain when eating and difficulty swallowing fluids.

PLANNING

- Assess respiratory status, including rate, pattern, lung sounds and cough effectiveness, at least every 4 hours.
- Monitor quantity, colour and odour of secretions.
- Assess vital signs and pain at least every 4 hours. Administer analgesics as ordered.

Expected outcomes

- Maintain clear airways and lung sounds.
- Maintain oxygen saturation level greater than 92%.
- Demonstrate interest in providing incision and stoma care.
- Accept information about potential communication strategies.
- Communicate effective pain management.
- Maintain appropriate body weight, intake and output.

IMPLEMENTATION

- Schedule time to sit with Mr Tom and discuss his concerns and feelings intermittently throughout your shift.
- Provide written information as requested.
- Monitor intake, output and daily weight.
- Arrange dietary consultation to determine kilojoule requirements.

EVALUATION

Mr Tom reports in writing that his pain is adequately controlled. His respiratory status is stable with clear breath sounds throughout and an oxygen saturation of 94%. He is afebrile. Mr Tom is tolerating tube feedings well and expresses a desire to begin eating. The dietitian has visited and assisted in planning to begin oral feedings. Intake and output are stable, as is his weight. Mr Tom has been receptive to receiving information about follow-up care and exploration of various modalities of speech.

CRITICAL THINKING IN THE NURSING PROCESS

1. Compare and contrast advantages and disadvantages of various methods to allow speech following total laryngectomy.
2. Develop a plan of care for Mr Tom for the nursing diagnosis of *Disturbed body image*.
3. Discuss nursing interventions to provide wound care for the person with laryngectomy and radical neck dissection.
4. List strategies to optimise ventilation.

REFLECTION ON THE NURSING PROCESS

1. Discuss the various strategies that can be utilised when communicating with Mr Tom and evaluate which patient education strategies may be most useful in this situation.
2. Outline what you have learned from the case study that you will implement in your future practice.

- Postoperatively, initiate oral intake with soft foods, not liquids. *Soft foods are easier to handle and swallow initially. As recovery progresses, thickened liquids can be swallowed and, eventually, a normal diet can be resumed.*
- Following total laryngectomy, reassure that choking is not possible because there is no connection between the oesophagus and trachea. *People often fear that swallowing will result in choking and they will be unable to cough effectively.*
- Instruct to initiate a swallow by placing a small amount of food on the back of the tongue, flex the head forward and then think 'swallow'. *Swallowing is no longer an automatic function and needs to be relearned.*

CONSIDERATION FOR PRACTICE

Provide for privacy during initial attempts at eating. Eating in the presence of others may cause embarrassment until confidence in eating is regained. Privacy also reduces distractions, allowing concentration on swallowing.

Nutritional imbalance related to insufficient oral intake

Large laryngeal tumours often place pressure on the oesophagus and may cause dysphagia (difficulty swallowing) or odynophagia (painful swallowing). In either case, difficulty eating may ultimately impair nutrition. Additionally, cancer often produces a hypermetabolic state, increasing kilojoule requirements. If surgery is performed, difficulty swallowing and a fear of aspiration in the early postoperative period also interfere with eating. Enteral or parenteral feedings are usually needed initially to meet nutritional status. After a total laryngectomy, the senses of taste and smell are disrupted. Although the sense of taste may be partially recovered, people may complain that eating no longer is pleasurable.

- Assess nutritional status using height and weight charts, reported weight loss and anthropometric measurements such as skin folds. *Thorough assessment of nutritional status is important in planning to meet current and anticipated kilojoule needs.*

CONSIDERATION FOR PRACTICE

Monitor food and fluid intake and urinary output. Pain or fatigue, rather than a sensation of fullness, may prompt the decision to stop eating, resulting in inadequate intake.

- Evaluate current and preferred eating habits and foods, as well as understanding of nutrition. *This evaluation provides additional information about nutrition as well as a basis for future planning.*
- Refer to a dietitian for further evaluation, planning and education. *A professional can identify nutritional needs and help plan a diet that will meet them.*
- Encourage experimentation with foods of different textures and temperatures. *Very cold foods, or foods of a soft texture, may be easier to swallow.*

CONSIDERATION FOR PRACTICE

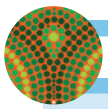
Weigh daily. Daily weight is an accurate measure of both fluid balance and nutritional status.

- Encourage frequent, small meals rather than three large meals per day. *Frequent, small quantities of food improve overall intake when dysphagia, odynophagia or fatigue interfere with nutrition.*
- Recommend liquid supplements, such as Ensure, when kilojoule needs are not being met. Provide information about where to obtain nutritional supplements. *Liquid dietary supplements provide balanced nutrition as well as additional kilojoules and are an effective way of increasing intake. They are available without prescription in major supermarkets.*
- Provide mouth care before meals and supplemental feedings. Provide a topical anaesthetic such as viscous lignocaine before eating for stomatitis or oesophagitis related to radiation or chemotherapy. *The tumour or its treatment may cause bad breath or a foul taste in the mouth, which suppresses appetite. Inflamed mucosa may make eating uncomfortable. A topical anaesthetic may relieve this discomfort and thus promote food intake.*
- Provide an anti-emetic 30 minutes before eating as needed to relieve nausea. *Nausea interferes with food intake. An anti-emetic can relieve nausea and make eating possible.*
- Suggest enteral (tube) feedings via nasogastric or gastrostomy tube if the person is unable to consume enough food to maintain weight and nutritional status. *Both cancer and surgery increase kilojoule needs. Supplemental enteral feedings may be necessary to prevent catabolism and to promote healing and recovery.*

CONSIDERATION FOR PRACTICE

Following laryngectomy, place in semi-Fowler's or Fowler's position. Elevating the head of the bed facilitates swallowing of oral secretions and helps prevent regurgitation of tube feedings.

- Instruct to perform mouth rinses before initiating feeding postoperatively. *Rinsing helps clean the mouth and also provides practice in using tongue and cheek muscles to control fluid in the mouth.*
- Refer to a physical or speech therapist for swallowing rehabilitation following laryngectomy. *Because surgery changes the relationship of the trachea, oesophagus and oropharynx, swallowing needs to be relearned before eating.*
- Reinforce swallowing instructions. *Reinforcement promotes learning.*



TRANSLATION TO PRACTICE Evidence-based practice: quality of life after total laryngectomy

A study by Perry, Casey & Cotton (2015) assessing psychosocial and functional issues following a laryngectomy surveyed 86 people from New South Wales and Victoria, measuring various functional and quality-of-life indicators.

The results demonstrated significant declines in quality of life and social relationship indicators. The individuals also had higher levels of depression and anxiety than normative samples. A strong relationship was demonstrated between the psychological wellbeing scores and important functional measures of speech and swallowing.

IMPLICATIONS FOR NURSING

Psychosocial health is an important part of rehabilitation following a laryngectomy. Follow up with experienced mental health practitioners and methods to support psychosocial health should be pivotal following surgery. Nurses and other healthcare professionals such as speech pathologists need to work together to maximise speech and swallowing function to not only assist individuals with critical safety issues, but also to support and maximise a person's psychosocial wellbeing following laryngectomy and well beyond discharge.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. In this study, there was a strong relationship between swallowing and speech function and the person's psychological wellbeing. As the nurse caring for a person who has had a laryngectomy, what can you do prior to discharge to assist with swallowing and speech function?
2. There were no data comparing male and female outcomes as the number of females in the cohort was too small. Would you expect there to be any difference in quality of life or psychological wellbeing scores between men and women laryngectomees? Explain.
3. How will the findings of this study impact on the care that you give to the person having a laryngectomy or partial laryngectomy?

Anticipatory grieving

The person with laryngeal cancer faces not only the diagnosis of cancer, which is often perceived as a death sentence, but also the prospect of mutilating surgery. If laryngectomy is necessary, the person grieves the loss of both a body part and an important function, speech, which is a vital aspect of social interaction and often necessary for their career. It also enables people to express their needs when they cannot meet them alone. The loss of speech, therefore, is a major loss. In addition, the tracheal stoma changes the manner in which the person breathes. If radical neck dissection is required, loss of neck musculature and function also alters body image and self-concept.

- Provide opportunities for expressing feelings of grief, anger or fear about the diagnosis of cancer, the impending surgery and the anticipated loss of speech. *The person with laryngeal cancer needs the opportunity (and may need permission) to grieve anticipated losses. A cancer diagnosis may precipitate grieving for unfulfilled plans and expectations, even though a cure may be anticipated. Laryngectomy causes a major change in body image, with loss of a vital body part and creation of a stoma. The person also grieves the loss of speech. This loss can have a significant impact on occupation and social interaction.*
- Help the person and family discuss the potential impact of the loss on family structure and function. *Discussion helps family members understand each other's feelings and support one another.*
- Refer for psychological or spiritual counselling as appropriate. *Counselling and spiritual guidance can help the person and family deal with the diagnosis and proposed treatment and help prevent a sense of defeat and hopelessness.*
- Help identify additional resources, such as coping strategies that have been successfully used in the past to deal with crises. *This exercise helps the person and family identify strengths they can use to deal with the present situation.*

CONSIDERATION FOR PRACTICE

Provide a calm, supportive environment with adequate privacy and emotional support for the person and family members as they work through the grieving process. It is important for the person and family to know that their feelings of loss are real and accepted by caregivers.

Community-based care

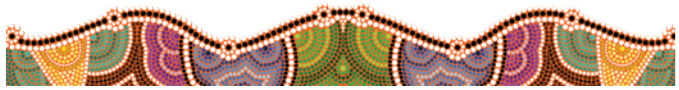
Teaching for the person with a benign laryngeal tumour emphasises management of contributing factors. Stress the importance of not yelling or screaming. Refer people, particularly singers, to a speech therapist for voice training. Emphasise the need to keep the voice within its normal range to reduce vocal cord stress. Encourage smoking cessation, particularly if the person is also a singer. Discuss the relationship of industrial pollutants to laryngeal tumours and help explore ways of reducing pollutant exposure.

Teaching the person and family about laryngeal cancer, treatment options and home care related to those treatments is an important nursing responsibility. Include the following topics when teaching:

- Clarification of treatment options, including risks and benefits.
- Importance of early intervention to reduce the risk of local spread and metastases.
- If a total laryngectomy is proposed, options for communication after surgery, including the pros and cons of each:
  a. The tracheoesophageal puncture device requires some manual dexterity to manipulate.
  b. Only about 30% of people are able to master oesophageal speech.
  c. A trial of the speech generator prior to surgery may reduce frustration in learning to use it postoperatively.
- Care related to radiation therapy, including skin and mouth care, management of secretions (see Chapter 13 for more information about radiation therapy and its effects).
- Strategies and resources for smoking cessation and alcohol abstinence.

- Ways to achieve and maintain optimal nutrition.
- Tracheostomy stoma care and preventing respiratory infection. Provide opportunities to practise and redemonstrate techniques. Clean technique (rather than sterile) is used; the tracheostomy tube may not be needed once the stoma is fully healed. Discuss these additional measures:
  a. Using a humidifier or vaporiser to add humidity to inspired air.
  b. Increasing fluid intake to maintain mucosal moisture and loosen secretions.
  c. Shielding the stoma with a stoma guard, such as a gauze square on a tie around the neck, to prevent particulate matter from entering the lower respiratory tract.
  d. Promptly removing secretions from skin surrounding the stoma to prevent irritation and skin breakdown.
  e. Water sports are contraindicated with a permanent tracheostomy; there is no restriction on other activities, although lifting may be more difficult because of inability to hold the breath (the Valsalva manoeuvre).
  f. Showering and bathing (without submerging the neck or head) are allowed; protect the stoma with a cupped hand or washcloth.
- Manifestations of potential complications of laryngectomy to be reported to the doctor, including loss of hearing or facial expression due to auditory or facial nerve injury or shoulder drop due to damage to the spinal accessory nerve.

The person and family need emotional and motivational support through this trying time. Refer to local support groups. If the person and family are having difficulty adjusting to the diagnosis of cancer and the effects of treatment, provide referral to counselling.



CHAPTER HIGHLIGHTS

- Upper respiratory infections (the common cold) are caused by a multitude of different viruses. Most are mild, self-limiting infections, appropriate for self-care; some viruses, however, such as RSV, can cause serious lower respiratory illness in the very young or very old.
- Three different strains of influenza virus are identified; type A causes most outbreaks of influenza. Because this disease increases the risk of pneumonia in older adults, people with chronic diseases and people who are immunocompromised, annual immunisation is important for these populations and their caregivers.
- Influenza is differentiated from URTI primarily by the presence of systemic manifestations, the duration and degree of fever, and the presence of persistent cough.
- Pharyngitis (sore throat) may be either viral or bacterial in origin; manifestations are similar. People with persistent or severe symptoms that include fever, enlarged lymph nodes and myalgias should be evaluated to rule out streptococcal pharyngitis, which can have significant complications such as rheumatic fever or post-streptococcal glomerulonephritis.
- The incidence of pertussis, a highly contagious reportable disease, is increasing due to waning immunity and improved identification of the infection in adults. In adults, it is often recognised by prolonged and persistent coughing spells. Pertussis is treated in community settings with antibiotic therapy.
- Epistaxis (nosebleed) and nasal fracture are relatively common and pose a risk only when airway clearance is impaired. Emergency care for epistaxis includes pinching the nares or bridge of the nose, sitting upright and leaning forward and applying ice to the nose. When nasal packing is required to control bleeding, close monitoring of respiratory status (respiratory rate and effort, oxygen saturation) is critical.
- Persistent voice hoarseness is the primary manifestation of laryngeal cancer. When identified and treated early, the rate of cure for laryngeal cancer is high. Some laryngeal tumours, however, have few manifestations until advanced. They may be treated by radiation therapy, chemotherapy or surgery (laryngectomy and neck dissection).
- Following total laryngectomy, a permanent tracheostomy is created and the upper trachea and oesophagus are separated, preventing aspiration when feedings are resumed. A tracheoesophageal puncture may be created to allow verbal communication following total laryngectomy.

CONCEPT CHECK

1 A person with hypertension asks the nurse what he can do to relieve the symptoms of an acute URTI. The nurse recommends that he:
1 ask his doctor for an antibiotic prescription
2 use an over-the-counter decongestant such as pseudoephedrine to relieve symptoms
3 take 1000 mg of vitamin C and zinc tablets on a regular basis
4 use an over-the-counter nasal spray for no more than 3 days to relieve congestion

2 Which of the following health promotion activities planned by a nurse working with a group of community-dwelling senior citizens would be most likely to prevent influenza and pneumonia?
1 Indoor exercise programs during winter months.
2 Influenza vaccine clinics at the senior centre.
3 Teaching effective handwashing.
4 Advising seniors to avoid crowds.

3 In teaching a person with bacterial sinusitis about home care, the nurse stresses the importance of:
1 completing the antibiotic prescription as ordered
2 sleeping with the head of the bed elevated to 45 degrees
3 using a humidifier to promote sinus drainage
4 maintaining a liberal fluid intake to help liquefy secretions

4 Which of the following nursing interventions for the person with posterior nasal packing is of highest priority?
1 Elevate the head of the bed.
2 Apply cold compresses to the nose.
3 Maintain oxygen therapy.
4 Provide frequent oral hygiene.

5 A person in the emergency department following facial trauma complains that his nose 'just keeps dripping'. The drainage appears like watery blood. The most appropriate nursing action would be to:

1 provide a box of tissues
2 reassure the person that this is expected with a nasal fracture
3 suction the nasopharynx
4 obtain a specimen for glucose testing

6 Expected findings in a person with obstructive sleep apnoea would include (select all that apply):
1 confusion and signs of dementia
2 enlarged tongue
3 complaints of daytime sleepiness
4 decreased oxygen saturation levels while awake
5 elevated blood pressure
6 complaints of morning headache

7 The nurse in a doctor's office notes that a person's voice is hoarse, a change from previous visits. The most appropriate question to ask the person would be:
1 'How long has your voice been hoarse?'
2 'Do you smoke?'
3 'Do you have a sore throat?'
4 'Would you like a prescription for throat lozenges?'

8 The nurse evaluates his teaching as effective when a person with stage I laryngeal cancer states:
1 'I'm glad I don't have to worry about treating this cancer now because it is so early.'
2 'I hate to think about eventually losing the ability to speak, but I'd rather treat it aggressively than lose my life to cancer.'
3 'I'm glad this was diagnosed early, when it can be treated with radiation, so I won't lose my voice.'
4 'Thank goodness this type of cancer usually doesn't spread anywhere else.'

9 Place the following nursing interventions for a person who has undergone total laryngectomy and radical neck dissection in order of priority.
1 Arrange consultation with speech therapist.
2 Provide small, frequent meals.
3 Encourage to express feelings regarding loss of voice.
4 Suction via tracheostomy as needed.
5 Instruct to support head when moving.

10 When providing tracheostomy care, the nurse:
1 cuts the dressing using sterile scissors
2 secures clean ties before removing soiled ones
3 uses clean technique to cleanse the outer cannula
4 cleanses the incision with an iodine-based antiseptic

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CHAPTER 35

NURSING CARE OF PEOPLE WITH VENTILATION DISORDERS

MAJELLA HALES

LEARNING OUTCOMES

- Relate the pathophysiology of lower respiratory tract infections and inflammatory disorders to a person's alteration in oxygenation.
- Discuss the development, consequences and management of a person experiencing selected pleural disorders.
- Explore the pathophysiology, clinical considerations and care of an individual experiencing a thoracic or inhalation injury.
- Review the causes and consequences of lung cancer and responsibilities of caring for individuals requiring surgery or other invasive procedures.

CLINICAL COMPETENCIES

- Assess the lower respiratory tract and chest wall disorders on ventilation and gas exchange.
- Use assessment data and knowledge of the effects of lower respiratory tract disorders to identify priority nursing care.
- Use the nursing process and evidence-based nursing research to plan and implement individualised nursing care.
- Develop measures to promote ventilation and gas exchange.
- Plan and provide appropriate teaching for health promotion in vulnerable populations.
- Evaluate the effectiveness of nursing interventions and teaching, revising strategies and teaching plans as needed.
- Coordinate interprofessional care and administer prescribed medications and treatments for people with lower respiratory tract disorders.

KEY TERMS

Disorders affecting the lower respiratory tract (below the larynx), pleural cavity and chest wall can affect the ability to effectively move air into and out of the lungs (ventilation). The exchange of oxygen and carbon dioxide across the alveolar–capillary membrane (respiration) is also affected. The disorders discussed in this chapter—respiratory infections and inflammation, trauma and disorders of the chest wall or pleural cavity, and neoplasms of the lung—all affect the ability to maintain clear and patent airways and ventilate the lungs. Nursing care for individuals with these disorders generally focuses on maintaining airway patency and an effective breathing pattern. Disorders that primarily affect gas exchange are discussed in Chapter 36.

The lower respiratory tract and chest wall disorders discussed in this chapter and Chapter 36 both have local and systemic effects. Local effects include cough, excess mucus production, **dyspnoea** (shortness of breath), **haemoptysis** (bloody sputum) and chest pain. Systemic effects may include fever, anorexia and malaise, **cyanosis** (grey/blue skin colour caused by oxygen-poor haemoglobin) and other manifestations of impaired gas exchange. Respiratory disease is very common. Figure 35.1 demonstrates the incidence and disparity of respiratory illness in Australians between 2011 and 2013, by Indigenous status, sex and jurisdiction. Before continuing, review the anatomy, physiology and assessment of the lower respiratory tract in Chapter 33.

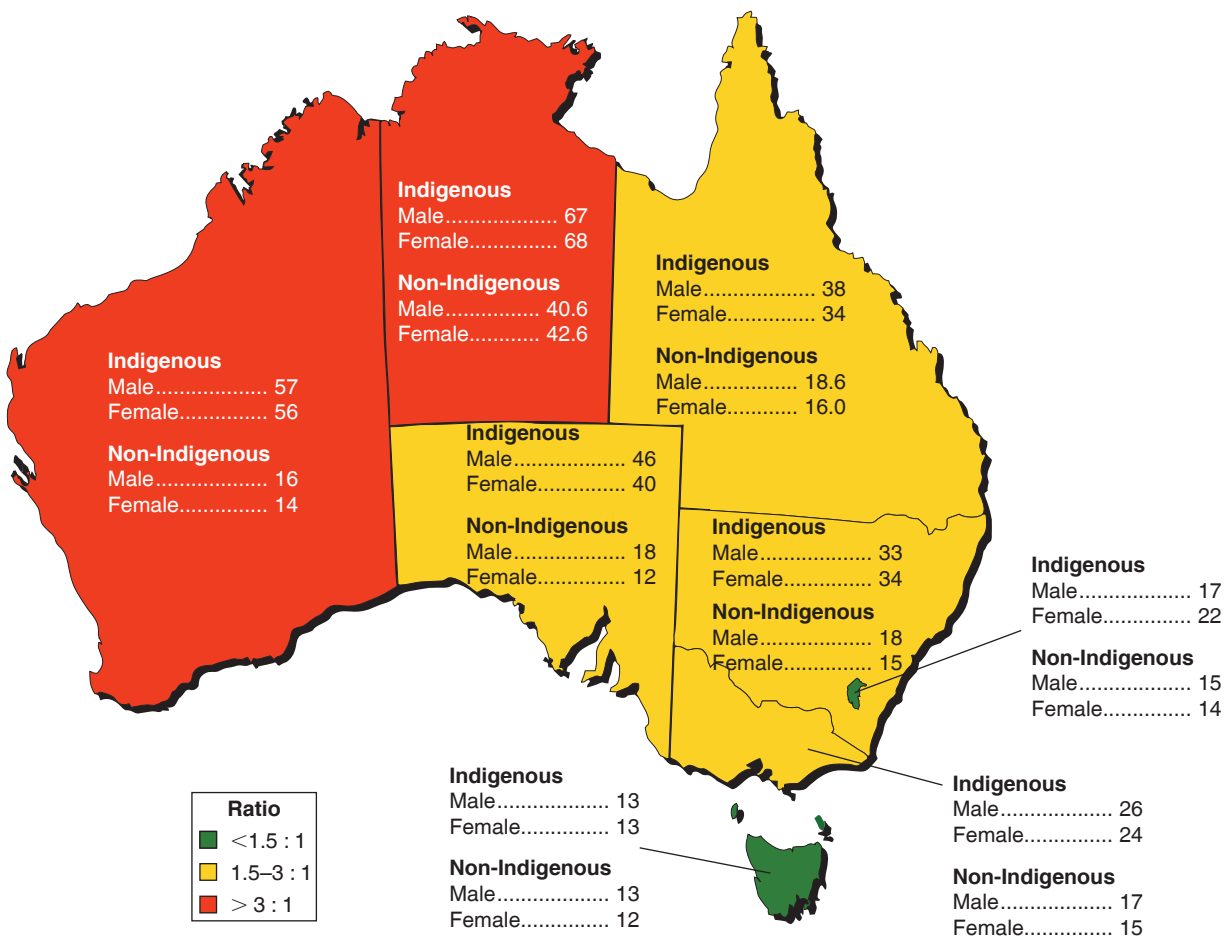


FIGURE 35.1 ■ Age-standardised hospitalisation rates for respiratory disease (per 1000), by Indigenous status, gender and jurisdiction, 2011–2013 (text) and ratios comparing Indigenous and non-Indigenous respiratory disease rates (colour)

Source: Generated using data from the Australian Institute of Health and Welfare (AIHW) (2015b). *Aboriginal and Torres Strait Islander Health Performance Framework data*. Retrieved from www.aihw.gov.au/indigenous-data/health-performance-framework/.

INFECTIONS AND INFLAMMATORY DISORDERS

Infections and inflammation of the lower respiratory tract are common and vary in type and severity. The respiratory tree is constantly exposed to the environment as air moves into and out of the lower respiratory tract. In addition, the oropharynx is colonised by large numbers of microorganisms that may be aspirated into the bronchial tree. Both anatomical and

physiological defences help maintain the sterility of the lower respiratory tract. When these defences are impaired, the risk of infection increases. Drugs, alcohol or neuromuscular disease may suppress the cough reflex and the influenza virus can leave the respiratory epithelium vulnerable to bacterial infection. Even in healthy people, microorganisms and other foreign material occasionally enter the bronchial tree and lung parenchyma.

THE PERSON WITH ACUTE BRONCHITIS

Bronchitis, inflammation of the bronchi, may be either an acute or a chronic condition. Acute bronchitis is relatively common in adults. In Australia in 2013–2014, 2.2% of all visits to the general practitioner (GP) for new problems were for management of acute bronchitis/bronchiolitis (Britt et al., 2014). Impaired immune defences and cigarette smoking increase the risk of acute bronchitis. In otherwise healthy adults, it typically follows a viral upper respiratory tract infection (URTI). Chronic bronchitis is a component of chronic obstructive pulmonary disease (COPD) and is discussed in Chapter 36.

FAST FACTS

Chronic obstructive pulmonary disease (COPD) is sometimes referred to by other names:

- COAD—chronic obstructive airways disease
- COLD—chronic obstructive lung disease
- CORD—chronic obstructive respiratory disease
- CAL—chronic airways limitation

Pathophysiology and manifestations

Infectious bronchitis can be caused by any pathogen. However, it is most commonly caused by bacteria and viruses that damage the respiratory mucosa. In healthy adults, bacterial bronchitis generally only occurs as a complication of viral infection. Inhalation of toxic gases or chemicals can lead to inflammatory bronchitis (Bullock & Hales, 2012).

The inflammatory response to infection or tissue damage from inhaled substances causes capillary dilation and oedema of the mucosal lining of the bronchi. Inflammatory cells infiltrate the affected mucosa, leading to exudate formation and increased mucus production. Ciliated epithelium is damaged by the inflammatory response and ciliary function is impaired (Papadakis, McPhee & Rabow, 2013). The immune response of lymphocytes and tissue macrophages is inhibited by some viruses and mycobacteria, increasing the risk of bacterial infection. Mucosal irritation and increased mucus production initiate the cough reflex. The respiratory tract may become hyperirritable for an extended period of time, leading to paroxysms of coughing and bronchospasm (Carolan, 2015).

Acute bronchitis is typically evidenced by a non-productive cough that becomes productive. The cough often occurs in paroxysms and may be aggravated by cold, dry or dusty air. Pleuritic pain (chest pain related to lung issues), often substernal, is common. Other manifestations include moderate fever and general malaise.

INTERPROFESSIONAL CARE

The diagnosis of acute bronchitis typically is based on the history and clinical presentation. A chest x-ray may be ordered to rule out pneumonia because the presenting manifestations can be similar. Other diagnostic testing is rarely indicated. Management focuses on symptom relief and includes rest, increased fluid intake and the use of paracetamol to relieve fever and malaise. Bronchodilators may also be beneficial. Antibiotics and corticosteroids are of limited value in the management of an individual with bronchitis. The Australian Lung Foundation (2014) suggests that acute bronchitis is viral in more than 95% of cases. However, antibiotics may still be used excessively; for example, a document produced by the AIHW (2012) reported that in Australia in 2008, 75% of people with a concession card who were dispensed any respiratory medication were also dispensed an oral antibiotic.

In order to encourage more judicious use of antibiotics, the National Prescribing Service (2014) recommended a creed for prescribing principles that minimises antibiotic resistance. The antibiotic creed uses the acronym MINDME (see Box 35.1).

Other medications may include a codeine-based preparation to ease the coughing paroxysms. However, care must be taken when using medication to suppress the cough reflex, as the infection will have affected the mucociliary escalator and therefore the cough will be the last line of defence.

BOX 35.1 The antibiotic creed

- **M**icrobiology guides therapy wherever possible
- **I**ndications should be evidence based
- **N**arrowest spectrum required
- **D**osage appropriate to the site and type of infection
- **M**inimise duration of therapy
- **E**nsure monotherapy in most situations

Source: National Prescribing Service (2014). *Therapeutic guidelines: Antibiotic* (15th ed.), p. 1, Box 1. Melbourne: Therapeutic Guidelines Ltd.

Nursing care

Nursing interventions for people with acute bronchitis are primarily educational and should include the following teaching topics:

- Increase fluid intake to keep mucus thin and meet increased fluid loss related to fever.
- Use over-the-counter (OTC) analgesics and cough preparations containing pseudoephedrine for symptom relief.

- Be aware of the ingredients of each of the preparations so as not to overdose on a drug that may be in several of the combination medications taken.
- Stress the importance of smoking cessation (as appropriate).

THE PERSON WITH PNEUMONIA

Inflammation of the lung **parenchyma** (the respiratory bronchioles and alveoli) is known as **pneumonia**. Despite significant advances in medical management of respiratory disorders, in 2013 influenza and pneumonia were ranked as the fifteenth leading causes of death in Australia for Aboriginal and Torres Strait Islander people and the sixteenth leading causes of death for non-Indigenous Australians (Australian Bureau of Statistics (ABS), 2015a). In 2013, almost 2500 deaths in Australia were attributed to pneumonia and influenza (ABS, 2015b). Rates of hospitalisation for pneumonia vary depending on age, gender and the state in which an individual lives. Incidence and mortality rates are highest in older adults and people with debilitating diseases. It is estimated that the financial burden of pneumonia is approximately $20 million per year (Li et al., 2012).

Pneumonia may be either infectious or non-infectious. Bacteria, viruses, fungi, protozoa and other microbes can lead to infectious pneumonia. Non-infectious causes include aspiration of gastric contents and inhalation of toxic or irritating gases. Pneumonias are often classified as community acquired, healthcare associated, nosocomial (hospital acquired) or opportunistic. Currently, as no universal nomenclature for the classification of pneumonia exists, there is some disparity in the use of these terms. Some authors use the terms 'nosocomial' and 'healthcare associated' interchangeably. Some authors consider exposure to a pathogen from an aged care facility, dialysis unit or outpatient clinic as a type of community-acquired pathogen. However, within this textbook these are considered healthcare associated. They often differ from both hospital-acquired and community-acquired pneumonia, not only in transmission but also in causative pathogens.

Different organisms are implicated in each of these classifications (see Table 35.1). The most common causative organisms for community-acquired pneumonia are the respiratory viruses *Streptococcus pneumoniae* (also called pneumococcus) and *Mycoplasma pneumonia. Haemophilus influenza, Legionella species* and *Chlamydia* species are also common community-acquired pneumonia pathogens (Centre for Healthcare Related Infection Surveillance and Prevention (CHRISP), 2015; Cunha, 2014). *Staphylococcus aureus* and Gram-negative bacteria such as *Pseudomonas aeruginosa*, *Klebsiella pneumoniae* and enteric bacilli, including *Escherichia coli*, are often implicated as nosocomial causes of pneumonia. Organisms such as *Pneumocystis jiroveci* generally cause infections more often in immunocompromised people (opportunistic infections).

FOCUS ON CULTURAL DIVERSITY

- Aboriginal and Torres Strait Islander children are five times more likely to die from pneumonia than non-Indigenous Australians (Ley, 2015).
- In Victoria, a group evaluating the primary health needs of the local refugee population within its area determined that, although the local refugees were 23% more likely to present to an emergency department and 47% more likely to be admitted for healthcare issues (than non-refugee locals), the refugees were actually no more likely to require admission for pneumonia or influenza than non-refugees within the area (Cheng et al., 2011).

Physiology review

In health, the lower respiratory tract is sterile. A number of defence mechanisms help maintain this sterile environment. Infectious particles trapped by the mucous membranes of the nose are removed by sneezing, while those deposited in the nasopharynx usually are swallowed or expectorated. Reflex closure of the epiglottis and the branching bronchial tree present anatomical barriers to entry of microorganisms and other possible contaminants. The cilia and mucus that line the respiratory tract and the cough reflex serve to trap and eliminate foreign matter that enters the lower respiratory tract. Organisms that make it past these barriers are usually rapidly phagocytised in the alveolus by resident macrophages, then attacked by the inflammatory and immune defences of the body. Ageing impairs these immune responses, increasing the risk of pneumonia.

TABLE 35.1 Common organisms causing pneumonia in adults

HOSPITAL ACQUIRED	HEALTHCARE ASSOCIATED	COMMUNITY ACQUIRED	OPPORTUNISTIC
Staphylococcus aureus	*Streptococcus pneumoniae*	*Streptococcus pneumoniae*	*Pneumocystis jiroveci*
Pseudomonas aeruginosa	*Staphylococcus aureus*	*Mycoplasma pneumoniae*	*Mycobacterium tuberculosis*
Klebsiella pneumoniae	*Haemophilus influenzae*	*Haemophilus influenzae*	Cytomegalovirus (CMV)
Legionella spp.	Influenza virus	*Chlamydia pneumoniae*	Atypical mycobacteria
Escherichia coli	*Enterobacter* spp.	*Legionella* spp.	Fungi
		Influenza virus	

Pathophysiology

The most common portal of entry for pathogens into the lung is aspiration of oropharyngeal secretions containing microbes. Microorganisms also may be inhaled after having been released when an infected person coughs, sneezes or talks. Finally, bacteria may spread to the lungs through the bloodstream from infection elsewhere in the body. Host defences must be overwhelmed either by the number of organisms or their *virulence* (disease-causing ability) in order for an infection to develop.

When the invading microorganisms colonise the alveoli, an inflammatory and immune response is initiated. The body's antigen–antibody response and endotoxins released by some organisms damage bronchial and alveolar mucous membranes, causing inflammation with vascular congestion and oedema. Infectious debris and exudate can fill alveoli, interfering with ventilation and gas exchange (see Figure 35.2). Pneumonia may develop in four distinct patterns: lobar pneumonia, bronchopneumonia, interstitial pneumonia and miliary pneumonia (see Table 35.2). The pathological process, anatomical location and manifestations of pneumonias vary according to the infective organism.

Lifespan considerations

When compared with an adult, the anatomical development and physiological maturation of an infant's respiratory tract increases the child's risk of developing respiratory tract infections. At birth, only 10% of alveoli are present, resulting in a greater surface area to volume ratio. Alveolar multiplication occurs until approximately 8 years of age. Airway lumens in children are smaller and chest wall recoil and underdeveloped respiratory musculature can result in fatigue sooner than in an adult. The breathing zone of a child is lower and therefore heavier particles can travel deeper into the respiratory system.

However, in advancing age, several changes associated with ageing and disease also affect respiratory function and airway clearance. The number of cilia decreases and the cough weakens. Gag and cough reflexes diminish. The older adult is at greater risk of dehydration, leading to thick, viscous mucus that is difficult to expectorate. Immune function declines with ageing. These factors increase the risk of pulmonary infection and reduce the older adult's ability to respond effectively to infectious processes (Bullock & Hales, 2012). Other factors also may increase the risk for and severity of lower respiratory tract infections in the older adult: immobility, smoking history, surgical procedures, use of multiple medications, malnutrition and such diseases as COPD and heart disease.

Acute bacterial pneumonia

Of the bacterial pneumonias, the pathogenesis of pneumococcal (*Streptococcus pneumoniae*) pneumonia is best understood (see Figure 35.3). These bacteria reside in the upper respiratory tract of up to 70% of adults. They may be spread by droplet contamination. In many cases, infection results from aspiration of resident bacteria. In the lower respiratory tract, the inflammatory response initiated by these organisms causes alveolar oedema and the formation of exudate. As alveoli and respiratory bronchioles fill with serous exudate, blood cells, fibrin and bacteria, *consolidation* (swelling) of lung tissue occurs. The lower lobes of the lungs are usually affected because of gravity. Consolidation of a large portion of an entire lung lobe is known as *lobar pneumonia*. This is the typical pattern for pneumococcal pneumonia. *Bronchopneumonia* is patchy consolidation involving several lobules. Other bacterial pneumonias often present with the patchy involvement of bronchopneumonia; pneumococcal pneumonia may also follow this pattern (see Figure 35.4). The process resolves when macrophages predominate, digesting and removing inflammatory exudate from the infected lung.

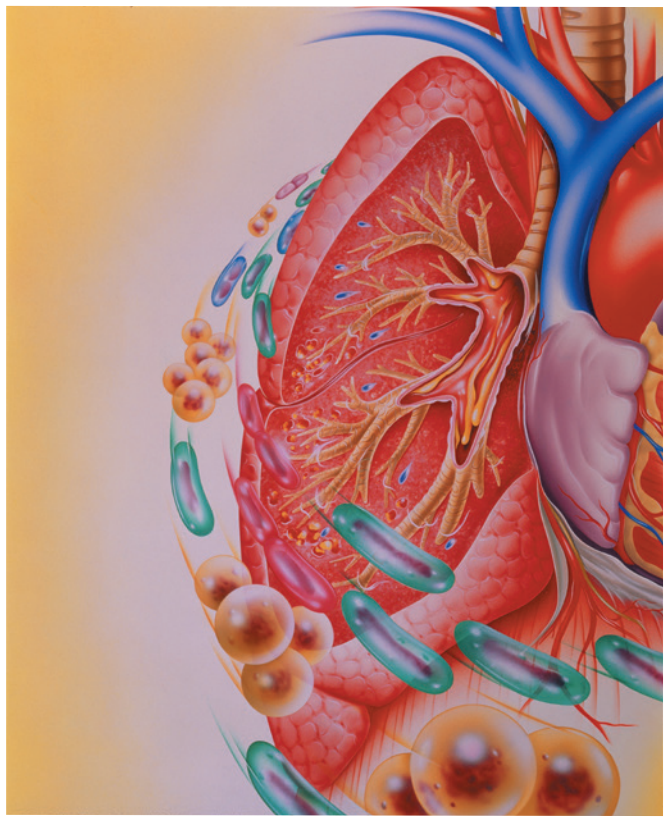


FIGURE 35.2 ■ In pneumonia, the inflammatory response causes fluid to accumulate in the alveoli and oedema to form as alveolar capillaries dilate and allow fluid to leak into interstitial tissues

Source: SPL/Custom Medical Stock Photo.

MANIFESTATIONS The presentation of bacterial pneumonia is usually acute, with rapid onset of chills, fever and cough productive of rust-coloured or purulent sputum. Chest aching or *pleuritic pain* (sharp, localised chest pain that increases with breathing and coughing) is common. Limited breath sounds and fine crackles are heard over the affected area of lung. A pleural friction rub may be audible. If the involved area is large and gas exchange is impaired, dyspnoea and cyanosis may be noted.

A more insidious onset with low-grade fever, cough and scattered crackles is more typical of bronchopneumonia. Dyspnoea is less commonly seen. The older adult or debilitated person may have atypical manifestations of pneumonia, with little cough,

TABLE 35.2 Patterns of lung involvement in pneumonia

PATTERN OF INVOLVEMENT	DESCRIPTION
Lobar pneumonia	Typically involves an entire lobe of a lung. Early in the process, when the immune response is minimal, bacteria spread throughout the affected lobe by rapid accumulation of fluid exudate. As the immune and inflammatory responses develop, RBCs and neutrophils, damaged epithelial cells and fibrin accumulate in the alveoli. Purulent exudate containing neutrophils and macrophages forms. As alveoli and respiratory bronchioles fill with exudate, blood cells, fibrin and bacteria, *consolidation* (solidification) of lung tissue occurs. Finally, the process resolves as enzymes destroy the exudate and residual debris is reabsorbed, phagocytised or coughed out.
Bronchopneumonia	Usually involves dependent portions of lung tissue, characterised by patchy consolidation. Exudate tends to remain primarily in the bronchi and bronchioles, with less oedema and congestion of the alveoli than in lobar pneumonia.
Interstitial pneumonia	The inflammatory process primarily involves the interstitium: the alveolar walls and connective tissue supporting the bronchial tree. Involvement may be patchy or diffuse as lymphocytes, macrophages and plasma cells infiltrate the alveolar septa. While alveoli typically do not contain significant exudates, protein-rich hyaline membranes may line the alveoli, interfering with gas exchange.
Miliary pneumonia	In miliary pneumonia, numerous discrete inflammatory lesions develop as a result of spread of the pathogen to the lungs via the bloodstream. Miliary pneumonia is primarily seen in people who are severely immunocompromised. As a result, the immune response is poor and damage to pleural tissue may be significant.

scant sputum and minimal evidence of respiratory distress. Fever, tachypnoea and altered mentation or agitation may be the primary presenting symptoms.

COMPLICATIONS Pneumonia typically resolves uneventfully; normal lung structure is restored on completion of the process. Local extension of the infection to involve the pleura (*pleuritis*) is the most common complication. Pneumonias caused by *Staphylococcus aureus* and some Gram-negative bacteria often cause extensive parenchymal damage with necrosis, lung abscess and empyema or pleural effusion. Progressive destruction of lung tissue and functional impairment is a possible consequence of *Klebsiella* pneumonia.

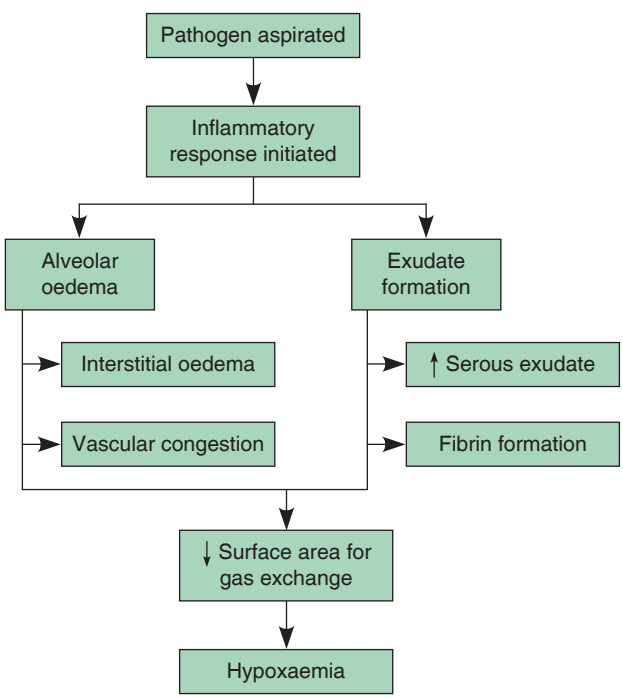


FIGURE 35.3 ■ The pathogenesis of pneumonia

A *lung abscess* is a local area of necrosis and pus formation within the lung itself. It is relatively uncommon. The manifestations of lung abscess develop slowly and include weight loss, malaise, night sweats, fever and a productive cough. Sputum is foul smelling and tasting. Rupture of the abscess into a larger airway is evidenced by production of copious amounts of purulent sputum.

Empyema is accumulation of purulent exudate in the pleural cavity. It is identified by chest x-ray or computed tomography (CT) scan. Thoracentesis may be performed or a chest tube inserted to remove purulent exudates. (Nursing care of the person undergoing thoracentesis or with a chest tube is discussed later in this chapter.)

Bacteraemia can spread the infection to other tissues, leading to meningitis, endocarditis or peritonitis, and increasing the risk of mortality.

Legionnaires' disease

Legionnaires' disease (legionellosis) is a form of atypical bronchopneumonia caused by *Legionella* bacteria. Although there are over 40 strains, not all cause disease in humans. The most common strain is *Legionella pneumophila* (Whiley et al., 2014). This Gram-negative bacterium is found in warm water or warm damp places. Legionnaires' disease occurs sporadically and in outbreaks, such as that which occurred at an American Legion convention in 1976 when the disease was first recognised. Contaminated water-cooled air-conditioning systems and other water sources have been implicated in its spread. In Australia in 2014, there were 425 notifications of legionellosis, which equates to 1.8 for every 100 000 people, down from 20.2 in 2013 (Department of Health, 2015a). See Figure 35.5 for Australian legionellosis statistics by year.

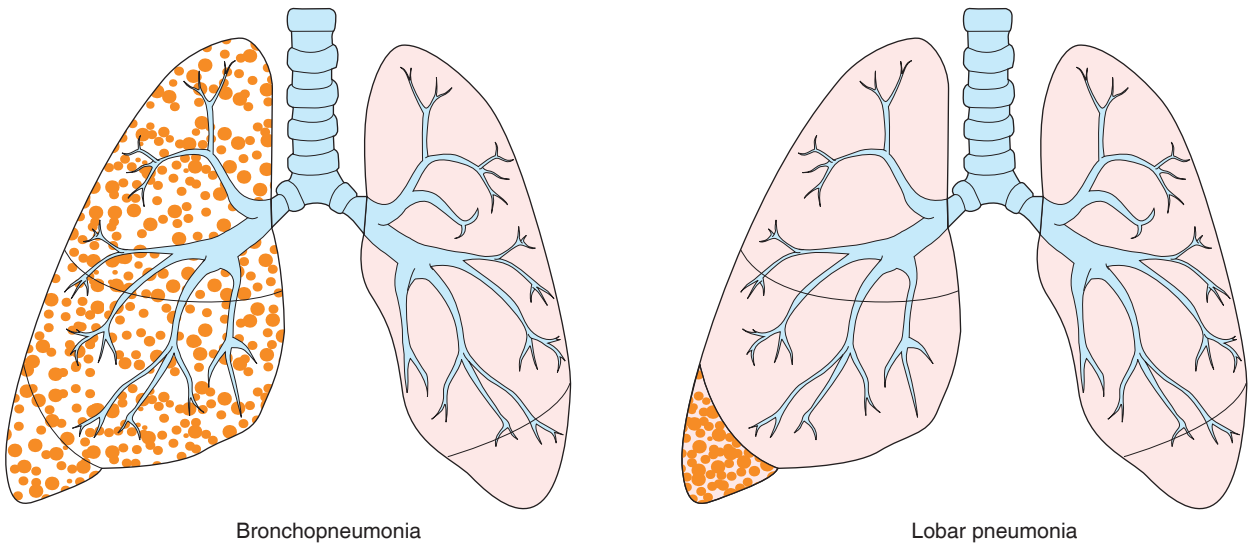


FIGURE 35.4 ■ Comparison of bronchopneumonia and lobar pneumonia

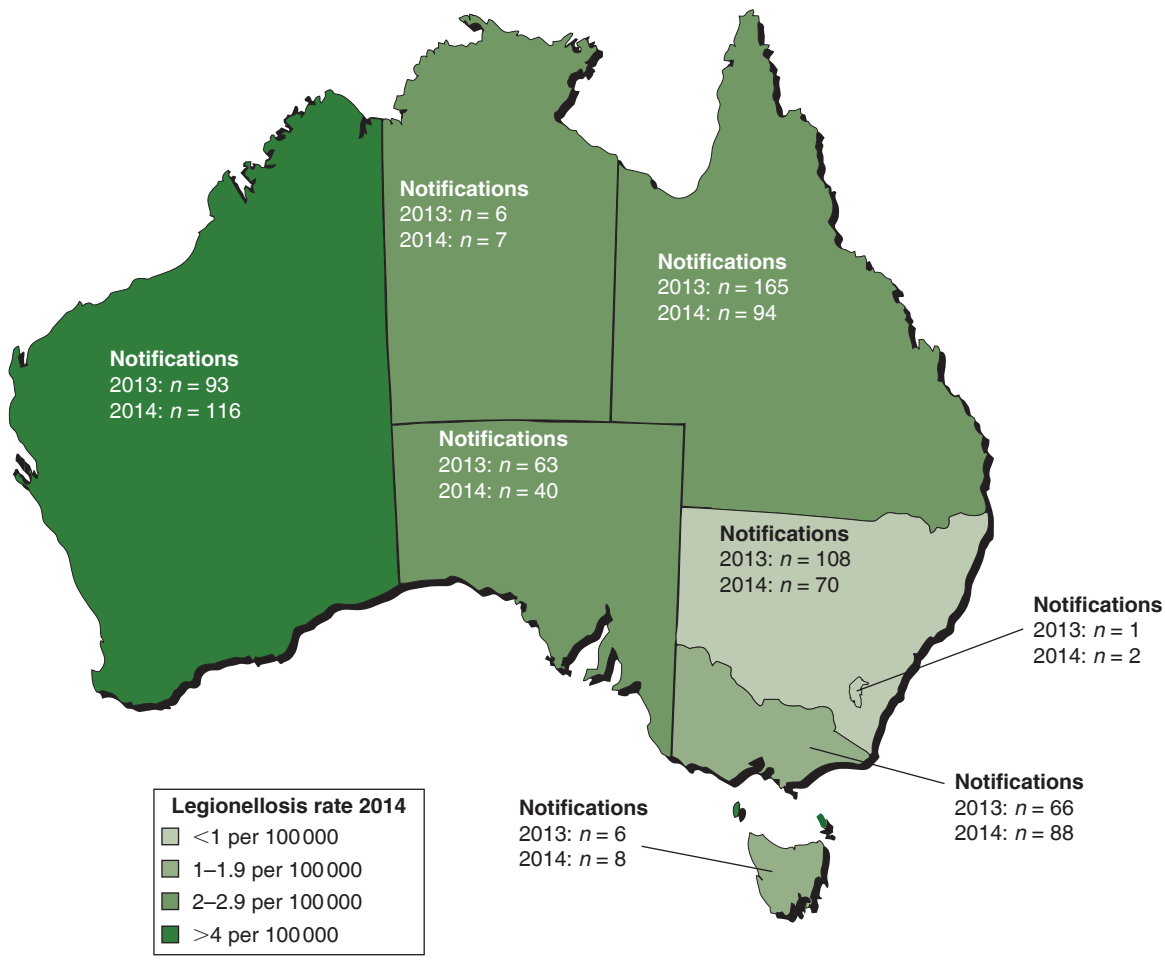


FIGURE 35.5 ■ Number of notifications for legionellosis in Australia for 2013 and 2014 (text) and notification rates per 100 000 for 2014 (colour)

Source: Generated using data from Department of Health (2015a). *National Notifiable Diseases Surveillance System. Number of notifications for legionellosis by year, Australia, 1991 to 2015.* Retrieved from www9.health.gov.au/cda/source/rpt_4_sel.cfm.

FAST FACTS

The largest outbreak of legionellosis in Australia occurred in 2000 at the Melbourne Aquarium.

- 125 cases were confirmed.
- 76% of the infected individuals required hospitalisation.
- Four people died.
- *Legionella pneumophila* was the organism isolated in the cooling towers.
- Cigarette smoking (>70/week) was a critical risk factor for the development of Legionnaires' disease in the individuals visiting the aquarium during the relevant 16 days in April 2000 (Greig et al., 2000).

More recently, in 2013 in a major private hospital in Queensland, two people were found to be infected with *Legionella pneumophila.* Subsequently one of the individuals died. As a result, all of Queensland's public, private and day hospitals were tested for *Legionella* spp. Although 159 facilities were free from contamination, 106 facilities returned positive samples for *Legionella* bacteria (Springborg, 2013; Queensland Government, 2014).

Smokers, older adults and people with chronic diseases or impaired immune defences are most susceptible to Legionnaires' disease. Symptoms develop gradually, beginning 2 to 10 days after exposure. Dry cough, dyspnoea, general malaise, chills and fever, headache, confusion, anorexia and diarrhoea, myalgias and arthralgias are common manifestations. Consolidation of lung tissue is patchy or lobar.

Viral pneumonia

Viral pneumonia is more common in children (Mosenifar, 2015). However, it is relatively common to have a secondary bacterial pneumonia as a direct result of a primary viral infection. Other viruses such as herpes viruses and measles virus also may cause viral pneumonia. As in primary atypical pneumonia, lung involvement in viral pneumonia is limited to the alveolar septum and interstitial spaces.

Viral pneumonia is typically a mild disease that often affects older adults and people with chronic conditions. It usually occurs in community epidemics. Flu-like symptoms of headache, fever, fatigue, malaise and muscle aching are common, along with a dry cough.

See Box 35.2 for examples of emerging zoonotic viral pneumonias.

Aspiration pneumonia

Aspiration of gastric contents into the lungs results in a chemical and bacterial pneumonia known as *aspiration pneumonia*. Major risk factors for aspiration pneumonia include emergency surgery or obstetric procedures, depressed cough and gag reflexes, and impaired swallowing. Older adults are at significant risk. Enteral nutrition by either nasogastric or gastric tube also increases the risk of aspiration pneumonia. Vomiting is not always apparent; silent regurgitation of gastric contents may occur when the level of consciousness is decreased. Measures to reduce the risk of aspiration pneumonia include minimising the use of preoperative medications, promoting anaesthetic elimination from the body and preventing nausea and gastric distension.

The low pH of gastric contents causes a severe inflammatory response when aspirated into the respiratory tract. Pulmonary oedema and respiratory failure may result. Common complications of aspiration pneumonia include abscesses, bronchiectasis (chronic dilation of the bronchi and bronchioles) and gangrene of pulmonary tissue.

BOX 35.2 Emerging zoonotic viral pneumonias

Severe acute respiratory syndrome (SARS) is a lower respiratory tract illness caused by the SARS coronavirus (SARS Co-V). The pathogen is zoonotic (causing disease that can cross the species barrier). Originally described in Asia in 2002, it quickly became a global concern and was identified as an epidemic in Asia, Europe and North America. However, the World Health Organization (WHO) reported there have only been six probable cases of SARS in Australia, and no deaths (WHO, 2014a). Following the declaration by the WHO that the cumulative total of global SARS cases exceeded 8000 people, it is believed that there have been no cases anywhere in the world since 2004 (WHO, 2015a).

Middle East respiratory syndrome (MERS) is a lower respiratory tract illness caused by the MERS coronavirus (MERS Co-V). It is thought to have originated in Saudi Arabia in 2012. Unlike SARS, which demonstrated relatively efficient transmission yet low mortality rates (~11%), MERS has relatively inefficient transmission but a higher mortality rate (~36%). Currently, there are no reports of MERS cases in Australia (WHO, 2015b).

Typical symptoms for both these viral illnesses include shortness of breath and cough. The person often has a fever and pneumonia is common. Transmission is most likely droplet. There are currently no vaccines and treatment is supportive, managing the person's signs and symptoms.

INTERPROFESSIONAL CARE

Prevention is a key component in managing pneumonia. Identifying vulnerable populations and instituting preventive strategies are measures to reduce the mortality and morbidity associated with pneumonia. With early identification of the infecting organism, appropriate treatment and support of respiratory function, most people recover uneventfully. However, pneumonia remains a serious disease with significant mortality, especially in aged and debilitated populations.

Diagnosis

The history and physical examination, along with diagnostic testing, are used to establish the diagnosis, determine the extent of lung involvement and identify the causative organism. See Chapter 33 for more information about the following tests and their nursing implications:

- *Chest x-ray* is obtained to determine the extent and pattern of lung involvement. Fluid, infiltrates, consolidated lung tissue and atelectasis (areas of alveolar collapse) appear as densities on the film. The *CT scan* provides a more detailed image of pulmonary tissue and may be used when the chest x-ray is not diagnostic.
- *Sputum Gram stain* rapidly identifies the infecting organisms as Gram-positive or Gram-negative bacteria. Antibiotic therapy can then be directed at the predominant type of organism until culture and sensitivity results are obtained.
- *Sputum culture and sensitivity* is ordered to identify the infecting organism and determine the most effective antibiotic therapy. When obtaining sputum for culture, it is important to obtain secretions from the lower respiratory tract, not the mouth and nasal passages (see Procedure 33.1).
- *Full blood count (FBC) with white blood cell (WBC) differential* shows an elevated WBC (>10 × 10^9/L) with increased circulating immature leucocytes in response to the infectious process. White blood cell changes are minimal in viral and other pneumonias.
- *Serology testing*, blood tests to detect antibodies to respiratory pathogens, may be used to identify the infecting organism when blood and sputum cultures are negative.
- *Pulse oximetry*, a non-invasive method of measuring peripheral oxygen saturation, is ordered to continuously monitor gas exchange. The saturation of peripheral oxygen (SpO_2) is normally 95% or higher. An SpO_2 of less than 95% may indicate impaired alveolar gas exchange.
- *Arterial blood gases (ABGs)* may be ordered to evaluate gas exchange. Respiratory secretions or pleuritic pain can interfere with alveolar ventilation. Alveolar inflammation can interfere with gas exchange across the alveolar–capillary membrane, especially if exudate or consolidation is present. An arterial oxygen tension (PaO_2) of less than 75 to 80 mmHg indicates impaired gas exchange or alveolar ventilation. See Chapters 9 and 33 for more information about gas transport, ABGs and normal or expected values.
- *Fibre-optic bronchoscopy* may be done to obtain a sputum specimen or remove secretions from the bronchial tree (see Figure 33.9). Nursing care related to bronchoscopy is summarised in the 'Diagnostic tests' box in Chapter 33.

Immunisation

Vaccines offer some degree of protection against the most common bacterial and viral pneumonias.

In Australia, a few pneumococcal vaccines are available (see Table 35.3). The critical difference is the number of serotypes contained within the vaccine. Recently a 7-valent pneumococcal vaccine has been replaced by a 13-valent one. There is also a pneumococcal vaccine with 10 serotypes and one with 23 serotypes. The individual's medical conditions, age, Indigenous status, overall immunisation status and geographical area can influence the choice of vaccine.

TABLE 35.3 Serotypes in current Australian pneumococcal vaccines

10-VALENT PNEUMOCOCCAL CONJUGATE VACCINE (10vPCV)	13-VALENT PNEUMOCOCCAL CONJUGATE VACCINE (13vPCV)	23-VALENT PNEUMOCOCCAL POLYSACCHARIDE VACCINE (23vPPV)
1, 4, 5, 6B, 7F, 9V, 14, 18C, 19F and 23F	1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F	1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19F, 19A, 20, 22F, 23F and 33F

Source: Department of Health (2015b). Pneumococcal disease. *Australian immunisation handbook* (10th ed.). Retrieved from www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home~handbook10part4~handbook10-4-13.

The predominant strain of influenza virus varies from year to year. A new vaccine formulation is prepared yearly, incorporating antigens of the influenza strains predicted to be the most prevalent for the upcoming flu season (typically the winter months). Vulnerable populations for whom yearly vaccine is recommended include individuals with chronic conditions involving lung, heart, diabetes or kidney disease, healthcare workers and residents of long-term care facilities. The vaccine contains egg protein and is not recommended for people who have a severe allergy to eggs or who have previously experienced a severe hypersensitivity response to the vaccine.

Medications

Medications used to treat pneumonia may include antibiotics to eradicate bacterial infection and bronchodilators to reduce bronchospasm and improve ventilation.

Broad-spectrum antibiotic therapy is initially used; then, based on the results of sputum microscopy/culture and sensitivity results, more specific antibiotic therapy may be commenced.

When an inflammatory response to the infection causes bronchospasm and constriction, bronchodilators may be ordered to improve ventilation and reduce hypoxia. Bronchodilators generally belong to one of two main groups: the sympathomimetic drugs, such as salbutamol (Ventolin) and the anticholinergic drugs, such as ipratropium bromide (Atrovent). Use of these drugs and related nursing implications are discussed in detail in the section on asthma in Chapter 36. Figure 35.6 demonstrates the actions of common respiratory drugs.

A mucolytic agent 'breaks up' mucus or reduces its viscosity. Acetylcysteine (Mucomyst) helps to liquefy mucus, making it easier to expectorate. For many people, however, increasing fluid intake is an effective means of liquefying mucus.

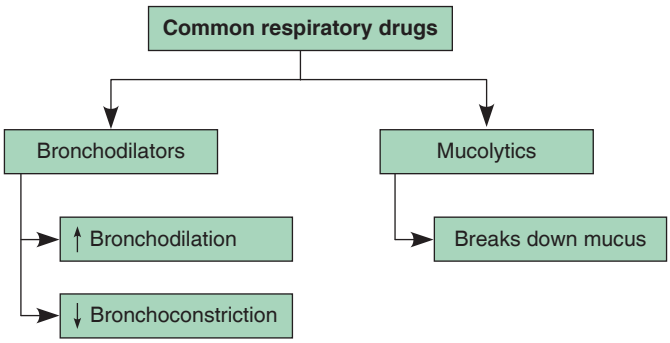


FIGURE 35.6 ■ Common drugs used in respiratory disorders

Treatments

When mucus secretions are thick and viscous, increasing fluid intake to 2500 to 3000 mL per day helps liquefy secretions, making them easier to cough up and expectorate. If the person is unable to maintain an adequate oral intake, intravenous fluids and nutrition may be required.

Incentive spirometry may be used to promote deep breathing, coughing and clearance of respiratory secretions. Endotracheal suctioning may be required if the cough is ineffective. This invasive technique is discussed in Chapter 36 in Procedure 36.1, describing nursing care for the person undergoing endotracheal suctioning.

OXYGEN THERAPY Oxygen therapy may be indicated for the person who is tachypnoeic or hypoxaemic.

Inflammation of the alveolar–capillary membrane interferes with diffusion of gases across the membrane. Diffusion is affected by several other factors, including the partial pressure of gases on each side of the membrane. Increasing the percentage of inspired oxygen above that of room air (21%) increases the partial pressure of oxygen in the alveoli and enhances its diffusion into the capillaries. Supplemental oxygen improves oxygenation of the blood and tissues in people with pneumonia.

Depending on the degree of hypoxia, oxygen may be administered by either a low-flow or a high-flow system. Low-flow systems include nasal prongs, simple face mask, partial rebreathing mask and non-rebreathing mask (see Figure 35.7). Nasal prongs can deliver 24–45% oxygen concentrations with flow rates of 2 to 6 L/min. The nasal prongs are comfortable and do not interfere with eating or talking. A simple face mask delivers 40–60% oxygen concentrations with flow rates of 5 to 8 L/min. Up to 100% oxygen can be delivered by the non-rebreather mask, the highest concentration possible without mechanical ventilation. When the amount of oxygen delivered must be precisely regulated, a high-flow system such as a Venturi mask is used (see Figure 35.8). The Venturi mask regulates the ratio of oxygen to room air, allowing precise regulation of the oxygen percentage delivered, from 24% to 50%. Severe hypoxia may necessitate intubation and mechanical ventilation. Endotracheal intubation and methods of mechanical ventilation are discussed in Chapter 36.

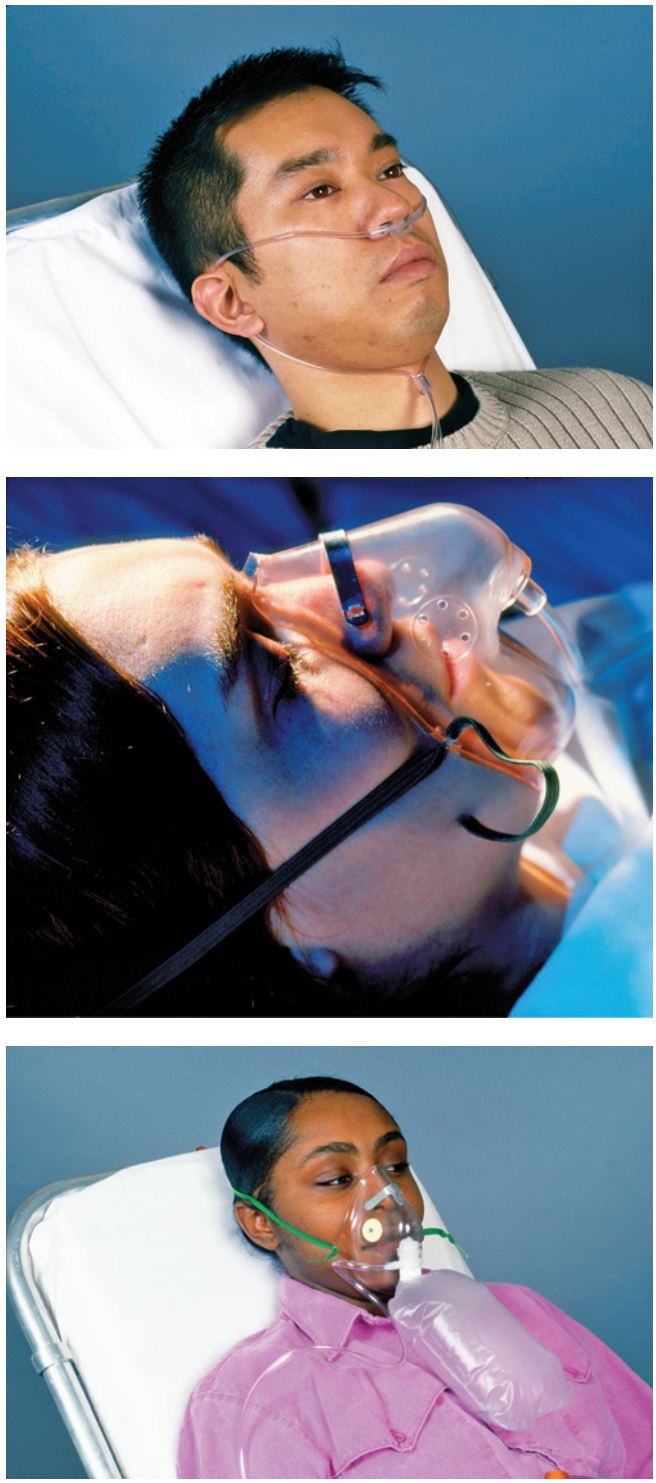


FIGURE 35.7 ■ Oxygen delivery devices: *A*, nasal prongs; *B*, simple face mask; *C*, non-rebreather mask

Sources: A and *C*, Michal Heron/Pearson Education; *B*, Tony McConnell/Science Source.

CHEST PHYSIOTHERAPY Chest physiotherapy, including percussion, vibration and postural drainage, may be prescribed to reduce lung consolidation and prevent atelectasis. *Percussion* is performed by rhythmically striking the chest wall with

FIGURE 35.8 ■ Venturi mask, a high-flow oxygen delivery system

Source: Michal Heron, Pearson Education/Ph College.

cupped hands (see Figure 35.9A), using rapid wrist flexion and extension. Cupping traps air between the palm and the person's skin, setting up vibrations through the chest wall that loosen respiratory secretions. The trapped air also provides a cushion, preventing injury. When performed correctly, percussion produces a hollow, popping sound. The breasts, sternum, spinal column and kidney regions are avoided during percussion.

Vibration facilitates secretion movement into larger airways. It usually is combined with percussion, although it may be used when percussion is contraindicated or poorly tolerated. Vibration is performed by repeatedly tensing the arm and hand muscles while maintaining firm but gentle pressure over the affected area with the flat of the hand (see Figure 35.9B).

Percussion and vibration are done in conjunction with *postural drainage*, which uses gravity to facilitate removal of secretions from a particular lung segment. The person is positioned with the segment to be drained superior to or above the trachea or mainstem bronchus. Drainage of all lung segments requires a variety of positions (see Figure 35.10); rarely do all segments require drainage. Bronchodilators or nebuliser treatments are administered as ordered prior to postural drainage. It is best to perform postural drainage before meals to avoid nausea and vomiting.

Nursing care

Health promotion

Health promotion activities focus on pneumonia prevention. Make sure individuals in high-risk groups are aware of the benefits of immunisations against influenza and pneumococcal pneumonia. A single dose of pneumococcus vaccine usually produces immunity to most strains of pneumococcal pneumonia, although repeat doses may be needed for older adults and people who are immunosuppressed. (Pneumococcus vaccine is contraindicated for people receiving immunosuppressive therapy.) Annual influenza vaccine helps prevent pneumonia because pneumonia often occurs as a sequelae to influenza.

CONSIDERATION FOR PRACTICE

Inquire about allergic responses to eggs or previous influenza vaccinations prior to administering influenza vaccine. A significant hypersensitivity response may occur in people who are allergic to eggs as the vaccine contains egg protein.

FIGURE 35.9 ■ *A*, Percussing (cupping) the upper posterior chest. Notice the cupped position of the nurse's hands. *B*, Vibrating the upper posterior chest

Source: © Beau Lark/Corbis.

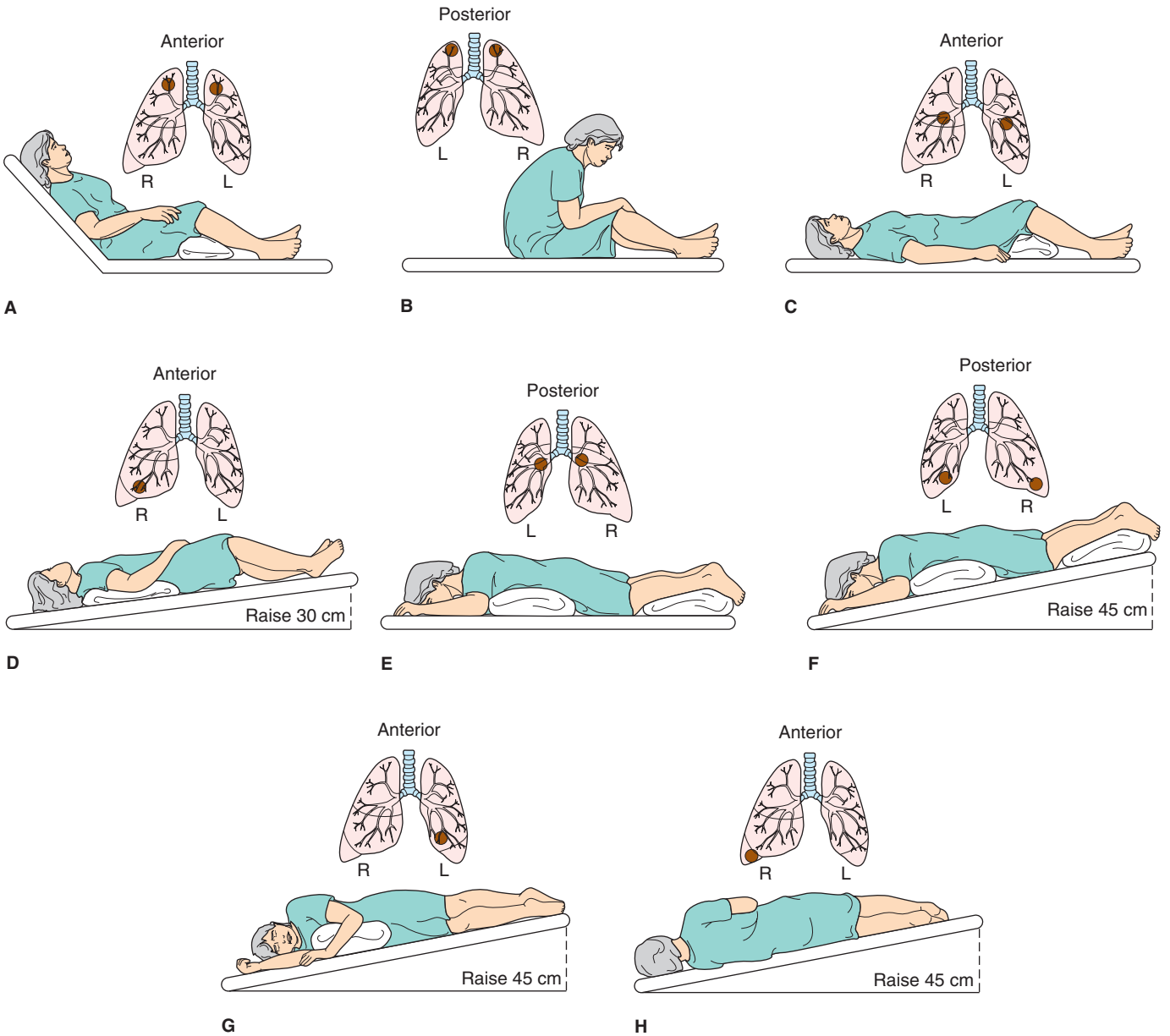


FIGURE 35.10 ■ Positions for postural drainage. *A*, Left and right anterior apical. *B*, Left and right posterior apical. *C*, Left and right anterior upper. *D*, Right middle lobe. *E*, Left and right superior lower lobes. *F*, Left and right lower posterior. *G*, Left lower lateral. *H*, Right lower lateral

Assessment

Focused assessment of the person with pneumonia includes the following:

- *Health history:* current symptoms and their duration; presence of shortness of breath, chest pain and its relationship to breathing; cough, productive or non-productive, colour, consistency of sputum; other symptoms; recent upper respiratory or other acute illness; chronic diseases such as diabetes, chronic lung disease or heart disease; current medications; medication allergies.
- *Physical examination:* presentation, apparent distress; level of consciousness; vital signs, including temperature; skin colour, temperature; respiratory excursion, use of accessory muscles of respiration; lung sounds.
- *Diagnostic tests:* WBC with differential, sputum Gram stain, culture and sensitivity, chest x-ray or CT scan.

Nursing diagnoses and interventions

People with lower respiratory tract disorders such as pneumonia may have multiple nursing care needs, depending on the severity of the illness. Alveolar ventilation and the process of

alveolar respiration can be affected by inflammation and secretions. **Hypoxaemia**, low levels of oxygen in the blood, and tissue hypoxia may result. Nursing care focuses on supporting optimal respiratory function and promoting rest to reduce metabolic and oxygen needs.

CONSIDERATION FOR PRACTICE

Promptly report signs of respiratory distress, including tachypnoea, tachycardia, nasal flaring, use of accessory muscles, intercostal retractions, cyanosis, increasing restlessness, anxiety or decreased level of consciousness (LOC). These may be early manifestations of respiratory failure and inability to maintain ventilatory effort.

Ineffective airway clearance

The inflammatory response to infection causes tissue oedema and exudate formation. In the lungs, the inflammatory response can narrow and potentially obstruct bronchial passages and alveoli. Assessment findings supporting this nursing diagnosis include adventitious breath sounds such as crackles (rales) and wheezes; dyspnoea and tachypnoea; coughing; and indicators of hypoxia such as cyanosis, reduced SpO_2 levels, anxiety and apprehension.

- Assess respiratory status, including vital signs, breath sounds, SpO_2 and skin colour, at least every 4 hours. *Early identification of respiratory compromise allows intervention before tissue hypoxia is significant.*
- Assess cough and sputum (amount, colour, consistency and possible odour). *Assessment of the cough and the nature of sputum produced allows evaluation of the effectiveness of respiratory clearance and the response to therapy.*
- Monitor ABG results; report increasing hypoxaemia and other abnormal results to the doctor. *Blood gas changes may be an early indicator of impaired gas exchange due to airway narrowing or obstruction.*
- Place in Fowler's or high-Fowler's position. Encourage frequent position changes and ambulation as allowed. *The upright position promotes lung expansion; position changes and ambulation facilitate the movement of secretions.*
- Assist to cough, deep breathe and use assistive devices. Provide endotracheal suctioning using aseptic technique as required if the person is intubated. *Coughing, deep breathing and suctioning help clear airways.*
- Provide a fluid intake of at least 2500 to 3000 mL per day. *A liberal fluid intake helps liquefy secretions, facilitating their clearance.*
- Work with the doctor and physiotherapist to provide pulmonary hygiene measures, such as postural drainage, percussion and vibration. *These techniques help mobilise and clear secretions.*
- Administer prescribed medications as ordered and monitor their effects. *If the infecting organism is resistant to the prescribed antibiotic, little improvement may be seen with treatment. Bronchodilators help maintain open airways but may have adverse effects such as anxiety and restlessness.*

Ineffective breathing pattern

Pleural inflammation often accompanies pneumonia, causing sharp localised pain that increases with deep breathing, coughing and movement, which can lead to rapid and shallow breathing. Distal airways and alveoli may not expand optimally with each breath, increasing the risk of atelectasis and decreasing gas exchange. Fatigue from the increased work of breathing is an additional problem in pneumonia. This, too, can lead to decreased lung inflation and an ineffective breathing pattern.

- Provide for rest periods. *Rest reduces metabolic demands, fatigue and the work of breathing, promoting a more effective breathing pattern.*
- Assess for pleuritic discomfort. *Provide analgesics as ordered. Adequate pain relief minimises splinting and promotes adequate ventilation.*
- Provide reassurance during periods of respiratory distress. *Hypoxia and respiratory distress produce high levels of anxiety, which tends to further increase tachypnoea and fatigue and decrease ventilation.*
- Administer oxygen as ordered. *Oxygen therapy increases the alveolar oxygen concentration and facilitates its diffusion across the alveolar–capillary membrane, reducing hypoxia and anxiety.*
- Teach slow abdominal breathing. *This breathing pattern promotes lung expansion.*

Activity intolerance

Impaired airway clearance and gas exchange interfere with oxygen delivery to body cells and tissues. At the same time, the infectious process and the body's response to it increase metabolic demands on the cells. The net result of this imbalance between oxygen delivery and oxygen demand is a lack of physiological energy to maintain normal daily activities.

- Assess activity tolerance, noting any increase in pulse, respirations, dyspnoea, diaphoresis or cyanosis. *These assessment findings may indicate limited or impaired activity tolerance.*
- Schedule activities, planning for rest periods. *Rest periods minimise fatigue and improve activity tolerance.*
- Provide assistive devices. *Assistive devices facilitate movement and reduce energy demands.*
- Enlist the family's help to minimise stress and anxiety levels. *Stress and anxiety increase metabolic demands and can decrease activity tolerance.*
- Perform active or passive range-of-motion (ROM) exercises. *Exercises help maintain muscle tone and joint mobility and prevent contractures if bed rest is prolonged.*
- Provide emotional support and reassurance that strength and energy will return to normal when the infectious process has resolved and the balance of oxygen supply and demand is restored. *The person may be concerned that activity intolerance will continue to be a problem after the acute infection is resolved.*

CONSIDERATION FOR PRACTICE

Activity intolerance may be an early sign of cardiorespiratory compromise, particularly in the older adult or person with pre-existing heart disease. New or worsening manifestations of activity intolerance should be reported to the doctor.

Community-based care

Individuals with pneumonia usually are treated in the community, unless their respiratory status is significantly compromised (e.g. altered mental status, tachypnoea, tachycardia, hypotension, hypo- or hyperthermia, and altered blood gases) or if risk

NURSING CARE PLAN A person with pneumonia

Mary O'Neal is a 35-year-old personal assistant and part-time university student. On returning home from class one evening, she begins to experience a chill. She alternates between chills and sweats all night. Staying home from work, she remains in bed most of the next day. Her fever continues and she develops a cough and dull aching chest pain. When the cough becomes productive of rust-coloured sputum the following day, she seeks medical treatment from her family doctor.

ASSESSMENT

Mrs O'Neal is admitted. She denies any previous history of respiratory diseases 'other than the usual colds, flu and such'. She also denies any history of smoking or medication allergies. She says her symptoms began abruptly with the onset of the chills. She describes her chest pain as a dull ache that was initially substernal but now is localised in her lower lateral right chest. The pain increases with deep breathing, coughing and moving. Her cough is increasing in frequency and severity and her sputum appears rusty brown. Her vital signs are BP 116/74, P 104 and regular, R 26, T 38.7°C. Skin warm and flushed, with no evidence of cyanosis. Respirations shallow, unlaboured; respiratory excursion equal. Diminished breath sounds in bases bilaterally, crackles noted in right posterior and lateral base. Faint pleural rub heard at right midaxillary line.

A FBC shows a WBC of 18.4 x 10^9/L; differential shows increased numbers of neutrophils and immature WBCs (bands). Mrs O'Neal provides a sputum specimen for culture and Gram stain prior to seeing the doctor.

The doctor orders a chest x-ray after examining Mrs O'Neal. Based on her history, examination and the chest x-ray, he makes the diagnosis of acute bacterial pneumonia, probably pneumococcal. He prescribes the current antibiotic medication for pneumonia.

DIAGNOSES

- *Ineffective breathing pattern* related to pleuritic chest pain manifested by tachypnoea and shallow breaths.
- *Hyperthermia* related to inflammatory process manifested by febrile temperature.

PLANNING

- Assess knowledge and understanding of pneumonia and its effects.
- Assist to develop a medication schedule that coordinates with normal daily routine.

Expected outcomes

- Maintain normal pulmonary function.
- Describe measures to minimise elevations in body temperature.
- Identify a schedule for taking her medication that will facilitate compliance with the regimen.
- Describe manifestations that should be reported to the doctor.

IMPLEMENTATION

- Teach about the following:
  a. importance of avoiding use of a cough suppressant except at night to facilitate rest
  b. ways to increase fluid intake to reduce fever and maintain thin mucus for easy expectoration
  c. beneficial effects of rest, especially during the acute phase of her illness
  d. safe use of aspirin and paracetamol to reduce fever
  e. importance of taking all prescribed medication doses as scheduled
  f. common side effects of medications and their management
  g. early manifestations of penicillin allergy that necessitate stopping the medication and notifying the doctor
  h. signs of complications of pneumonia or worsening pneumonia to report.

EVALUATION

The sputum culture confirms *S. pneumoniae* as the cause of Mrs O'Neal's pneumonia. When she returns for her follow-up appointment, she reports that she began to feel better after 2 days on the penicillin and returned to work the following Monday. Her examination reveals good breath sounds throughout with no adventitious sounds. The follow-up sputum culture is free of pathogens.

CRITICAL THINKING IN THE NURSING PROCESS

1. Do any of the factors identified in the case study increase Mrs O'Neal's risk of acute bacterial pneumonia?
2. Even though Mrs O'Neal has no history of medication allergies, an anaphylactic reaction remains a potential risk. Describe the sequence of events leading to anaphylactic shock, its initial symptoms and immediate nursing interventions.
3. Had Mrs O'Neal required hospitalisation to treat her acute pneumonia, interruption of her usual activities and responsibilities could have led to anxiety. Develop a care plan for this situation, using the nursing diagnosis of *Ineffective role performance* related to hospitalisation.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. You developed and undertook an eight-point teaching plan for Mrs O'Neal (see implementation above). Which evaluation data will demonstrate that this comprehensive teaching plan has been understood and will be successful?

factors such as advanced age and/or coexisting heart, kidney or liver disease are present.

Discuss the following topics when preparing the person and family for home care:

- the importance of completing the prescribed medication regimen as ordered
- recommendations for limiting activities and increasing rest
- maintaining adequate fluid intake to keep mucus thin for easier expectoration
- ways to maintain adequate nutritional intake, such as small, frequent, well-balanced meals
- the importance of avoiding smoking or exposure to second-hand smoke to prevent further irritation of the lungs
- manifestations to report to the doctor, such as increasing shortness of breath, difficulty breathing, increased fever, fatigue, headache, sleepiness or confusion.

CONSIDERATION FOR PRACTICE

Beware of any person with cardiac co-morbidities who develops a respiratory illness. Individuals with hypertension or cardiac failure may be inadvertently ordered both a sympathomimetic and a beta-blocker concomitantly (e.g. salbutamol and metoprolol). These two classes of drugs are contraindicated—sympathetic agonist and sympathetic antagonist.

The accompanying nursing care plan provides further nursing interventions for people treated in the community.

CONSIDERATION FOR PRACTICE

Epidemic: when there are more cases in a region than normal.
Pandemic: a worldwide epidemic.

THE PERSON WITH LUNG ABSCESS

A **lung abscess** is a localised area of lung destruction or necrosis and pus formation. The most common cause of lung abscess is aspiration and resulting pneumonia. Risk factors, therefore, are those for aspiration: decreased LOC due to anaesthesia, injury or disease of the central nervous system (CNS), seizure, excessive sedation or alcohol abuse; swallowing disorders; dental caries; and debilitation secondary to cancer or chronic disease. Lung abscess may also occur as a complication of some types of pneumonia, including those due to *S. aureus*, *Klebsiella* and *Legionella*. Many organisms can cause a lung abscess (see Figure 35.11).

Pathophysiology and manifestations

A lung abscess forms after lung tissue becomes consolidated (i.e. after alveoli become filled with fluid, pus and microorganisms). Consolidated tissue may become necrotic. This necrotic process can spread to involve the entire bronchopulmonary segment and progress proximally until it ruptures into a bronchus. With rupture, the contents of the abscess empty into the bronchus, leaving a cavity filled with air and fluid, a process known as *cavitation*. If purulent material from the abscess is not expectorated, the infection may spread, leading to diffuse pneumonia or a syndrome similar to acute respiratory distress syndrome.

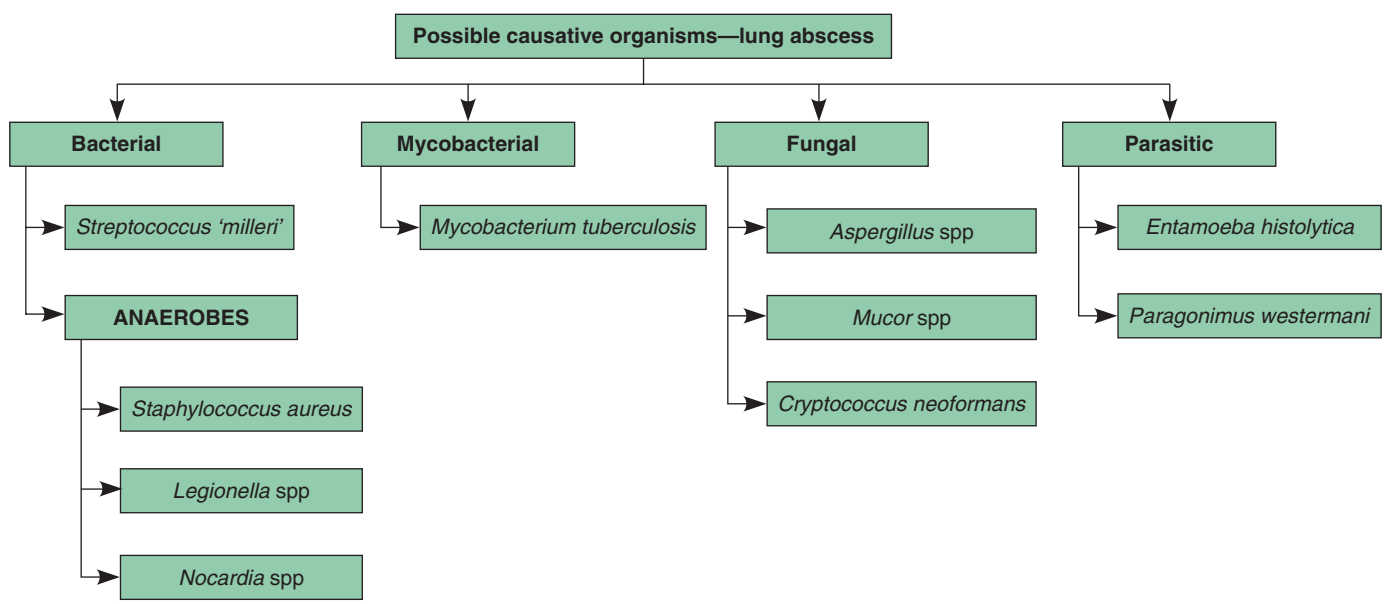


FIGURE 35.11 ■ Possible causative organisms—lung abscess

Source: Generated from information from the Royal College of Pathologists Australasia (RCPA) (2015). Retrieved from www.rcpa.edu.au/Library/Practising-Pathology/RCPA-Manual/Items/Clinical-Problems/L/Lung-absces.

Manifestations of lung abscess typically develop about 2 weeks after the precipitating event (aspiration, pneumonia and so on). Their onset may be either acute or insidious. Early symptoms are those of pneumonia: productive cough, chills and fever, pleuritic chest pain, malaise and anorexia. The temperature may be significantly elevated, 39.4°C or higher. When the abscess ruptures, the person may expectorate large amounts of foul-smelling, purulent and possibly blood-streaked sputum. Breath sounds are diminished and crackles may be noted in the region of the abscess. A dull percussion tone is also present.

INTERPROFESSIONAL CARE

The diagnosis of lung abscess usually is based on the history and presentation. The FBC may indicate leucocytosis. Sputum culture may not show the organism involved unless rupture occurs. Chest x-ray shows a thick-walled, solitary cavity with surrounding consolidation, although differentiating lung abscess from consolidation can be difficult until cavitation occurs.

Lung abscess is treated with antibiotic therapy. Postural drainage may be ordered to relieve obstruction and promote drainage. In some cases, bronchoscopy is used to drain the abscess. If the pleural space becomes involved, a chest tube (*tube thoracostomy*) may be used to drain the abscess. See the section on pneumothorax later in this chapter for further discussion of chest tubes.

Nursing care

Although most persons with lung abscess recover fully with appropriate antibiotic treatment, rupture and drainage of the abscess into a bronchus is a frightening experience. Nursing care needs of the individual relate primarily to maintaining a patent airway and adequate gas exchange.

Health education for the person and family focuses on the importance of completing the prescribed antibiotic therapy. Most lung abscesses are successfully treated with antibiotics; however, treatment may last up to 1 month or more. Emphasise the importance of completing the entire course of therapy to eliminate the infecting organisms. Infection from lung abscess can spread not only to lung and pleural tissue but also systemically, causing sepsis. If postural drainage is ordered, teach the person and family how to perform this procedure. When procedures such as bronchoscopy or thoracostomy are performed to drain the abscess, provide preoperative teaching and instruction on postoperative care.

THE PERSON WITH TUBERCULOSIS

Tuberculosis (TB) is a chronic, recurrent infectious disease that usually affects the lungs, although any organ can be affected. This disease, caused by *Mycobacterium tuberculosis*, is relatively uncommon in Australia. Although only 49 people in Australia died of tuberculosis in 2013 (ABS, 2015b), since 1999 an average of over 1100 people per year have been diagnosed with tuberculosis (see Figure 35.12). Many hospitalisations for tuberculosis are for migrants from the Philippines, India, Vietnam and China (King, Douglas & Beath, 2011).

Mycobacterium tuberculosis is a relatively slow-growing, slender, rod-shaped, acid-fast organism with a waxy outer capsule, which increases its resistance to destruction. Although the lungs are usually infected, tuberculosis can involve other organs as well. It is transmitted by *droplet nuclei*—airborne droplets produced when an infected person coughs, sneezes, speaks or sings. The tiny droplets can remain suspended in the air for several hours. Infection may develop when a susceptible host breathes in air containing droplet nuclei and the contaminated particle eludes the normal defences of the upper respiratory tract to reach the alveoli.

Incidence and prevalence

The incidence of tuberculosis in Australia is low in comparison with many other developing and developed countries, with a rate of approximately 5.7 per 100 000 people (Department of Health, 2015c). These results compare well with other countries; for example, South Africa records incidence at approximately 860 per 100 000 people and both Mozambique and Zimbabwe at approximately 552 per 100 000 people (WHO, 2014b). See Figure 35.13 for the WHO's estimated 2014 incidence globally by region.

In many other countries, including the US, TB is primarily attributed to the resurgence of HIV/AIDS; however, in Australia, only approximately 1% of individuals with TB have HIV/AIDS (Australian Government Department of Health, 2012). The highest incidence rates occur in the age group 20 to 44 years. The Northern Territory reports the highest incidence rates and Tasmania the lowest (see Figure 35.14).

The emergence of multidrug-resistant (MDR) strains of TB has complicated the treatment and control of this infectious respiratory disease. Nevertheless, mechanisms to reduce the development of drug resistance have been implemented to assist with this problem. Directly observed therapy (DOT), in which individuals are observed taking each and every medication dose, is recommended by the WHO. In Australia, the Department of Health has devised a 4-year strategic plan for the control of tuberculosis with priorities including improving surveillance, reducing disparities within subgroups and managing the risk of increasing drug resistance.

Risk factors

The risk of infection by *M. tuberculosis* is affected by characteristics of the infectious person, the extent of air contamination, duration of exposure and susceptibility of the host. The number of microbes in the sputum, frequency and force of coughing, and behaviours such as covering the mouth when coughing, affect

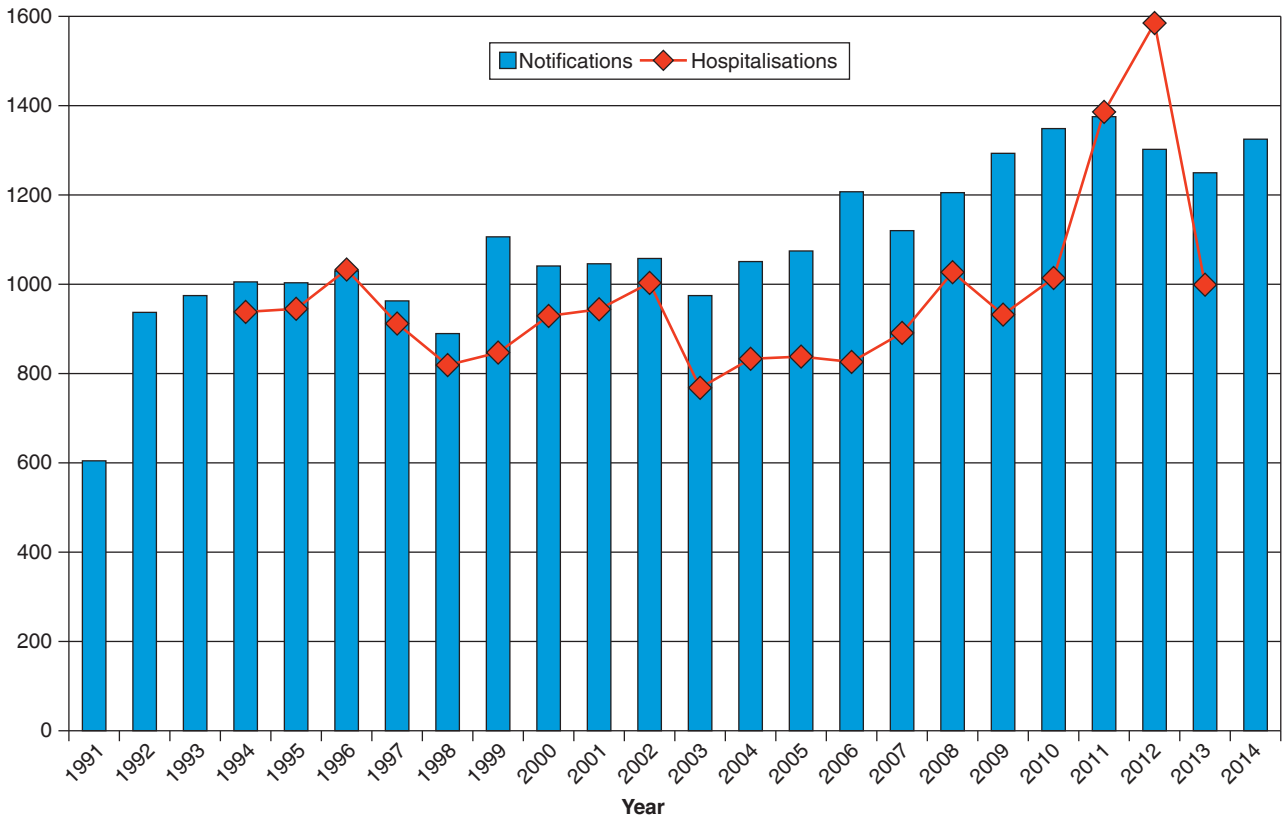


FIGURE 35.12 ■ Australian tuberculosis incidence 1991–2014, and hospitalisation admissions 1994–2013

Sources: Generated from Department of Health (2015c). *National Notifiable Diseases Surveillance System. Number of notifications for tuberculosis by year, Australia, 1991 to 2014.* Retrieved from www9.health.gov.au/cda/source/rpt_4.cfm; Australian Institute of Health and Welfare (AIHW) (2015a). *Principal diagnosis cubes for 1993–94 to 1997–98, 1998–99 to 2009–10, 2010–11, and 2011–12 to 2012–13.* Retrieved from www.aihw.gov.au/principal-diagnosis-data-cubes/.

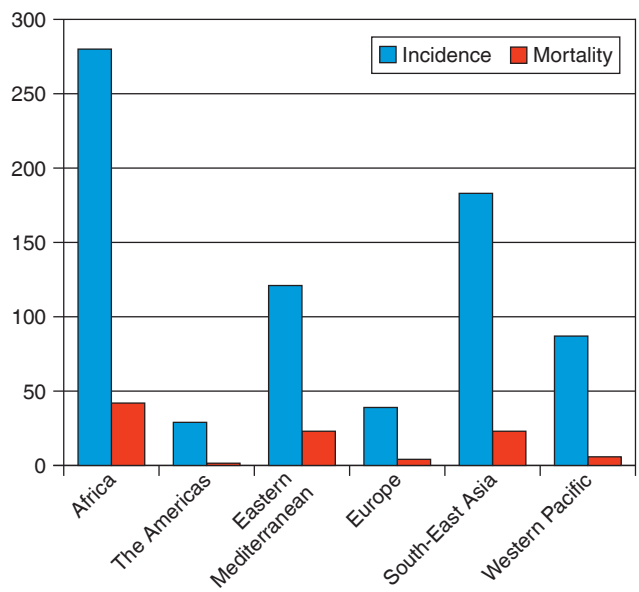


FIGURE 35.13 ■ World Health Organization estimated incidence and mortality of the burden of tuberculosis, 2014, globally by region (per 100 000 population)

Source: Generated from information from the World Health Organization (2014b). *Global tuberculosis report 2014.* Retrieved from www.who.int/tb/publications/global_report/en/.

the production of droplet nuclei. In a small, closed or poorly ventilated space, droplet nuclei become more concentrated, increasing the risk of exposure. Prolonged contact, such as living in the same household, increases the risk. Less-than-optimal immune function—a problem for people in lower socioeconomic groups, injecting drug users, the homeless, alcoholics and people with HIV infection—increases the susceptibility of the host.

Pathophysiology

Pulmonary tuberculosis

Minute droplet nuclei containing one to three bacilli that elude upper airway defence systems to enter the lungs implant in an alveolus or respiratory bronchiole, usually in an upper lobe. As the bacteria multiply, they cause a local inflammatory response. The inflammatory response brings neutrophils and macrophages to the site. These phagocytic cells surround and engulf the bacilli, isolating them and preventing their spread. *M. tuberculosis* continues to slowly multiply; some bacilli enter the lymphatic system to stimulate a cellular-mediated immune response. (See Chapter 11 for a review of immune responses.) Neutrophils and macrophages isolate the bacteria but cannot destroy them. A granulomatous lesion called a *tubercle*, a sealed-off colony of bacilli, is formed (Bullock & Hales, 2012). Within the tubercle, infected tissue dies, forming a cheese-like centre, a process called *caseation necrosis*.

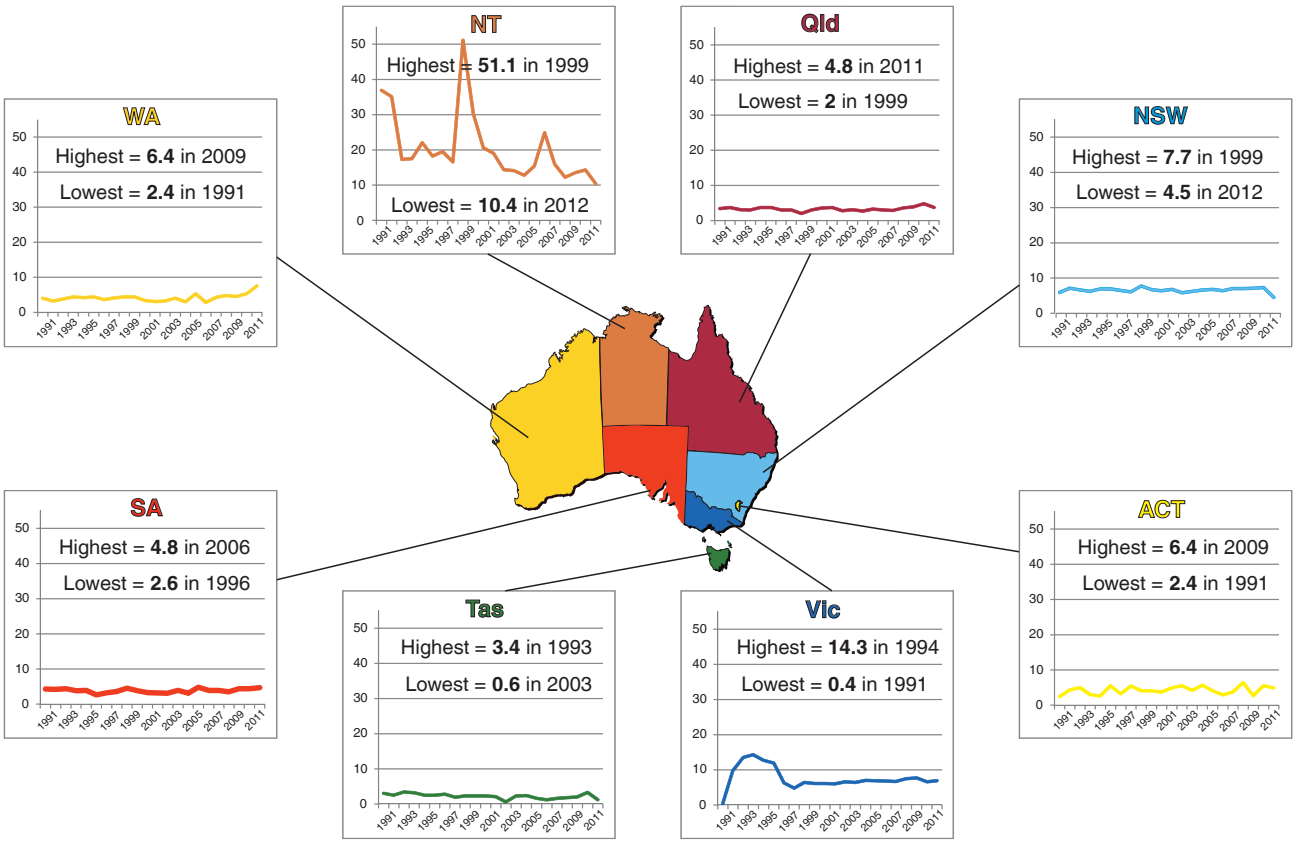


FIGURE 35.14 ■ Incidence of tuberculosis per state 1991–2012 (per 100 000 population)

Source: Generated from Department of Health (2015c). *National Notifiable Diseases Surveillance System. Number of notifications for tuberculosis by year, Australia, 1991 to 2015*. Retrieved from www9.health.gov.au/cda/source/rpt_4.cfm.

If the immune response is adequate, scar tissue develops around the tubercle and the bacilli remain encapsulated. These lesions eventually calcify and are visible on x-ray. The person, although infected by *M. tuberculosis*, does not develop TB, disease. If the immune response is inadequate to contain the bacilli, the disease of TB can develop. Occasionally, the infection can progress, leading to extensive destruction of lung tissue. In *primary tuberculosis*, granulomatous tissue may erode into a bronchus or into a blood vessel, allowing the disease to spread throughout the lung or other organs. This severe form of TB is uncommon in adults.

A previously healed TB lesion may be reactivated. *Reactivation tuberculosis* occurs when the immune system is suppressed due to age, disease or use of immunosuppressive drugs. The extent of lung disease can vary from small lesions to extensive cavitation of lung tissue. Tubercles rupture, spreading bacilli into the airways to form satellite lesions and produce tuberculosis pneumonia. Without treatment, massive lung involvement can lead to death or a more chronic process of tubercle formation and cavitation may result (Herchline, 2014). People with chronic disease continue to spread *M. tuberculosis* into the environment, potentially infecting others. 'Pathophysiology illustrated: tuberculosis' illustrates the pathogenesis of TB.

Individuals with HIV are at high risk of developing active TB, due to primary infection or reactivation. HIV infection suppresses cellular immunity, which is vital to limiting the replication and spread of *M. tuberculosis*.

MANIFESTATIONS AND COMPLICATIONS The initial infection causes few symptoms and typically goes unnoticed until the tuberculin test becomes positive or calcified lesions are seen on chest x-ray. Manifestations of primary progressive or reactivation TB often develop insidiously and are initially non-specific (see the 'Manifestations' box below). Fatigue, weight loss, anorexia, low-grade afternoon fever and night sweats are common. A dry cough develops, which later becomes productive of purulent and/or blood-tinged sputum. It is often at this stage that the person seeks medical attention.

MANIFESTATIONS Pulmonary tuberculosis

- Fatigue
- Weight loss
- Anorexia
- Low-grade afternoon fever and night sweats
- Cough: initially dry, later productive of purulent and/or blood-tinged sputum

NURSING CARE OF THE OLDER ADULT Tuberculosis

Presenting symptoms of tuberculosis in the older adult are often vague, including coughing, weight loss, anorexia, night sweats or periodic fevers. These signs and symptoms should not be dismissed as a normal part of ageing.

Tuberculin skin testing with purified protein derivative (PPD) is required when diagnosis is considered. A chest x-ray and sputum culture for acid-fast bacilli are obtained if the PPD is positive.

Successful treatment for TB generally requires four drugs for at least 6 months to totally eradicate the organism. Individuals are most often required to take the medication under the direct supervision of a nurse or other healthcare worker to improve adherence to the management regimen and decrease the risk of multidrug-resistant strains forming.

ASSESSING FOR HOME CARE

Community-dwelling older adults are susceptible to tuberculosis as well as those in care facilities.

Assess risk factors for tuberculosis:

- general health and nutritional status, including intake of specific nutrients such as vitamin D (lack of vitamin D is associated with a higher risk of developing active TB)
- presence of a chronic disease such as silicosis, diabetes, alcoholism or HIV infection; past history of a gastrectomy
- past history of a positive tuberculin test that now has converted to negative
- medications such as corticosteroids or other immunosuppressive drugs.

Assess living and social situation:

- natural light and ventilation in the home
- access to clean water, cooking facilities, supermarkets and other services
- possible exposure to infected people; for example, sharing a household with someone with active TB, crowded living facilities, homelessness, frequent participation in senior activities, volunteer work in residential care facilities or other institutional settings
- access to healthcare.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

Teaching focuses on improving the older adult's ability to self-manage the disease and treatment. Teach about tuberculosis and how it is spread. Emphasise the importance of taking all medications as prescribed and complying with follow-up appointments and testing. Discuss the importance of:

- using disposable tissues to contain respiratory secretions, especially during the first 2 weeks of treatment when the disease may be transmitted to others
- avoiding exposure to crowds or people with infectious diseases
- eating a well-balanced diet with adequate nutrients
- getting adequate rest, sleep and exercise to maintain good general health
- ensuring that housemates or others having frequent contact with the person are tested and receive prophylactic treatment if indicated.

Teach about possible side effects of the prescribed medications and the importance of reporting these to healthcare providers (see Table 35.4).

TABLE 35.4 Antitubercular medications

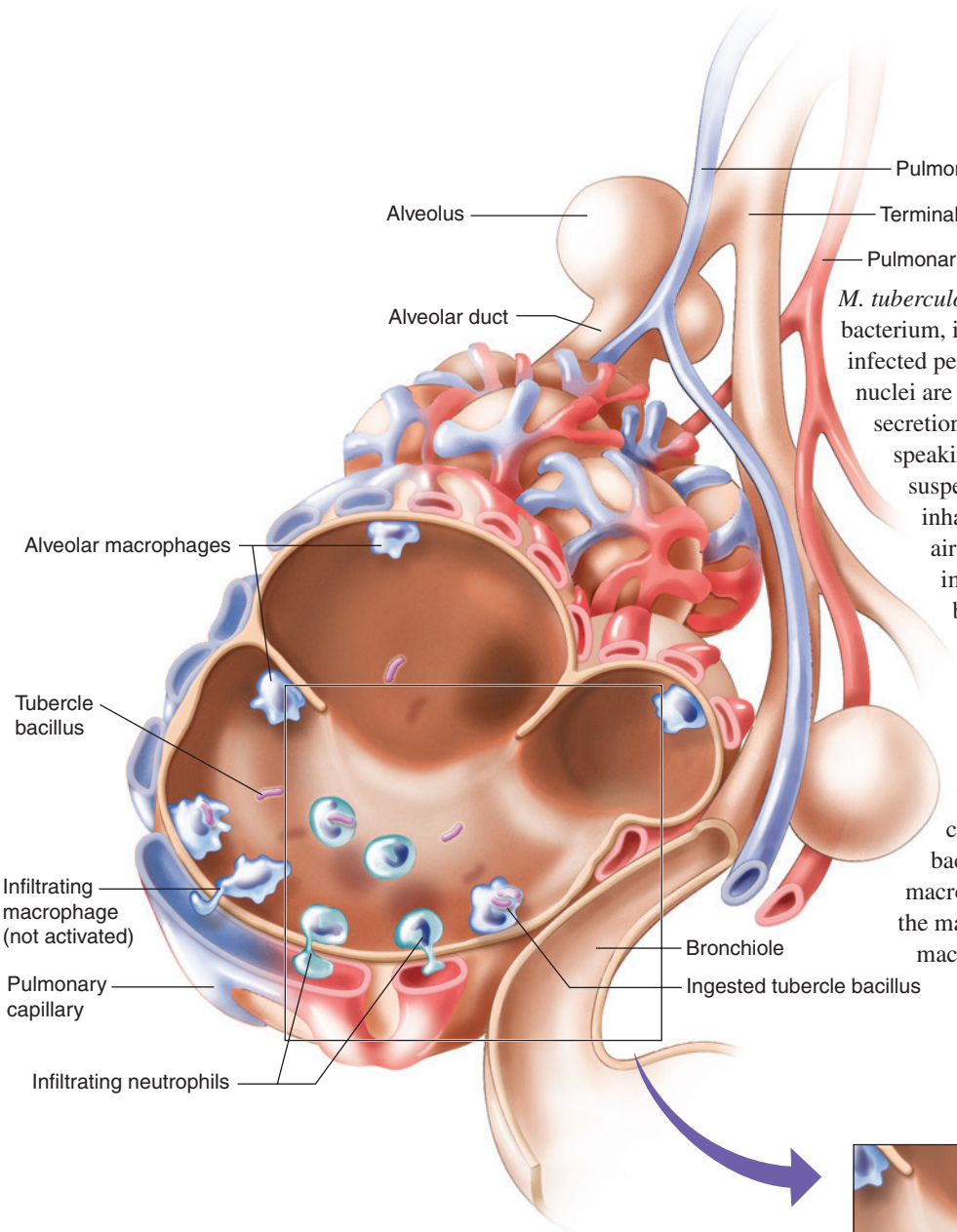
DRUG AND DOSAGE	ADVERSE EFFECTS	NURSING IMPLICATIONS
Isoniazid (INH), oral: 300 mg daily or 900 mg 1, 2 or 3 times weekly	Peripheral neuropathy Hepatitis	Administer pyridoxine (vitamin B_6) concurrently. Monitor liver function studies (AST and ALT); avoid other hepatotoxins.
Rifampicin (RMP), oral: 600 mg daily or 2 or 3 times weekly	Hepatitis Flu-like syndrome; fever Colours body fluids—including sweat, urine, saliva, tears and cerebrospinal fluid (CSF)—orange–red	As for INH. Do not miss or skip doses; flu-like syndrome and fever occur when drug is resumed. Contact lenses may become discoloured and should not be worn.
Pyrazinamide (PZA), oral: 1 to 2 g daily; or 2 to 4 g twice weekly	Hyperuricaemia Hepatotoxicity	Monitor uric acid levels. Monitor AST and ALT; avoid other hepatotoxins.
Ethambutol (EMB), oral: 800 mg to 1600 mg daily; or 2 to 4 g twice weekly	Optic neuritis	Monitor red–green colour discrimination and visual acuity.

Tuberculosis empyema and bronchopleural fistula are the most serious complications of pulmonary tuberculosis. When a tuberculosis lesion ruptures, bacilli may contaminate the pleural space. Rupture also may allow air to enter the pleural space from the lung, causing pneumothorax.

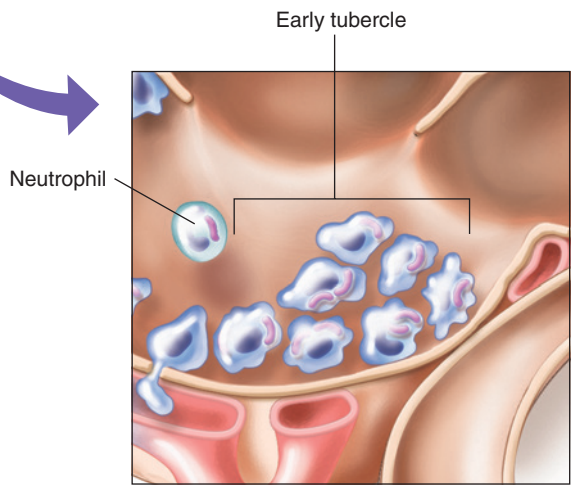
Extrapulmonary tuberculosis

When primary disease or reactivation allows live bacilli to enter the bronchi, the disease may spread through the blood and lymph system to other organs. These distant disease metastases may produce an active lesion or may become dormant

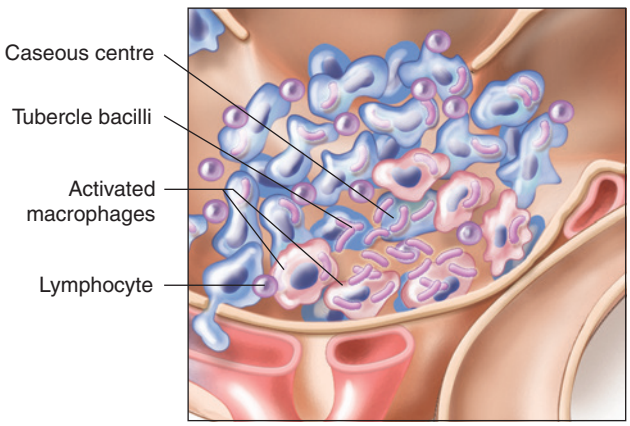
Tuberculosis



M. tuberculosis, a rod-shaped aerobic bacterium, is spread via droplet nuclei from an infected person to a susceptible host. Droplet nuclei are tiny droplets of respiratory secretions spread via coughing, sneezing or speaking. When dried, they can remain suspended in air for several hours. Most inhaled bacilli are trapped in the upper airways; those reaching distal airways implant in the respiratory bronchioles and alveoli. Rarely, these tubercle bacilli multiply unchecked to cause primary tuberculosis. In most cases, activated alveolar macrophages ingest the bacilli. The bacilli may be destroyed; however, unique characteristics of the tuberculosis bacillus resist its destruction by the macrophage. The bacilli multiply within the macrophage, eventually killing the macrophage.

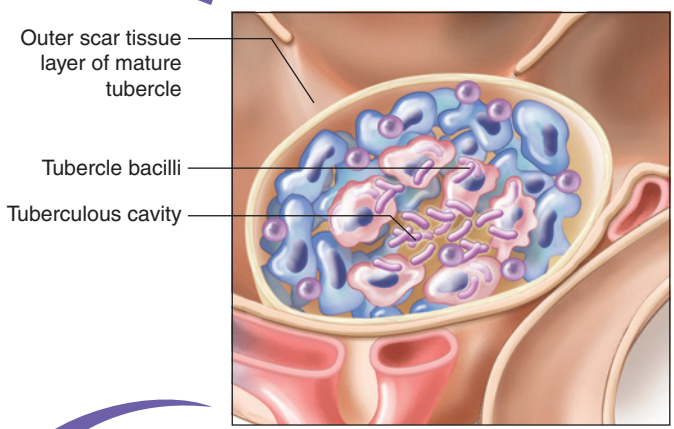


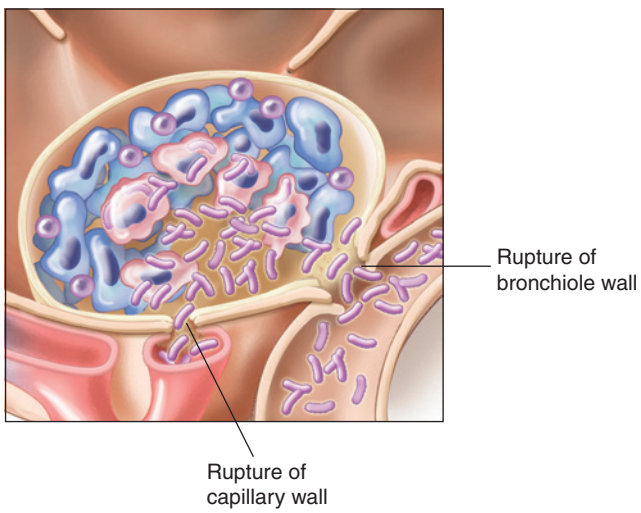
The dead macrophages lyse, releasing various chemotaxic factors into the bloodstream. Neutrophils and non-activated macrophages are attracted to the site. These phagocytic cells ingest the tubercle bacilli released from the lysed macrophages.



After several weeks, a delayed hypersensitivity response to bacterial antigens destroys many of the macrophages. Concurrently, a cell-mediated immune response activates additional macrophages, which ingest and destroy the bacilli. The lysed macrophages and bacilli are surrounded by a mass of live, activated macrophages and lymphocytes. Scar (granulomatous) tissue forms, encapsulating the primary lesion. Most lesions calcify and are visible on x-ray. These lesions may remain dormant for a year or more (in some cases, many years) before being reactivated to produce secondary or reactivation tuberculosis.

When the immune and macrophage-activating responses are weakened by age or disease (e.g. HIV disease), the tuberculosis bacilli continue to multiply within the lesion. The caseous material at the centre of the lesion liquefies, and the lesion grows.





The enlarging lesion damages surrounding bronchial walls and blood vessels. Granulomatous tissue surrounding the lesion can erode into a bronchus, forming an air-filling cavity. Within this cavity, the bacilli multiply, spreading into the airways and the environment via infected sputum. Bacilli also spread via the blood and within macrophages to regional lymph nodes, and from there to many organs and tissues. Resulting extrapulmonary lesions evolve in the same sequence as pulmonary lesions.

and reactivate at a later time. Extrapulmonary tuberculosis is especially prevalent in people with HIV.

FAST FACTS

- Immunisation with bacillus Calmette–Guérin (BCG) vaccine is not part of the standard vaccination regimen in Australia. However, individuals with high risk are encouraged to receive it.
- High-risk individuals include:
  - Indigenous Australian neonates in high TB incidence regions
  - infants born in Australia to migrant parents
  - children travelling (for an extended period) to countries of high TB prevalence
  - healthcare workers in frequent contact with people with TB.

Source: Department of Health (2015b). *Australian immunisation handbook* (10th ed.). Retrieved from www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home~handbook10part4~handbook10-4-13.

FAST FACTS

- The organism causing tuberculosis, *Mycobacterium tuberculosis*, is spread through droplet nuclei that remain suspended in air for several hours.
- A tubercle is a sealed-off colony of bacilli; if it ruptures, organisms spread, leading to tuberculosis pneumonia.
- Primary or secondary TB lesions may affect other body systems such as the kidneys, genitalia, bone and brain.

INTERPROFESSIONAL CARE

Tuberculosis was a major public health concern early in the last century, before the development of effective sanitation measures and drug treatment. The development of drug-resistant strains, susceptibility of people with HIV disease and inadequate access to healthcare for high-risk populations contribute to TB continuing to be a significant public health threat. Interprofessional care, therefore, focuses on the following:

- early detection
- accurate diagnosis
- effective disease treatment
- preventing TB spread to others.

With appropriate treatment, individuals become non-infective to others fairly rapidly. Nurses and other healthcare workers are at risk of exposure if the disease has not yet been diagnosed. When a person with TB is hospitalised, maintain respiratory isolation to minimise the risk of infection to other people and the healthcare workers.

Non-adherence with prescribed treatment is a major problem in treating active TB: the person can continue transmitting the disease to others and drug-resistant strains of bacteria can develop when treatment is incomplete. Tuberculosis is a communicable disease and state and territory health authorities must be notified. Contact tracking will commence and individuals may be identified and examined. People who share living or work environments with the person are tested and may receive prophylactic treatment. Local health authorities or services will often continue contact with people who have active TB to facilitate effective containment and infection control.

Screening

The tuberculin test is used to screen for TB infection. A cellular or delayed hypersensitivity response to *M. tuberculosis* develops within 3 to 10 weeks after the infection. Injecting a small amount of *purified protein derivative (PPD)* of tuberculin any time thereafter activates this response, attracting macrophages to the area and causing a pronounced local inflammatory response. The amount of induration surrounding the injection site is used to determine infection (see Figure 35.15).

It is important to remember that a positive response indicates that infection and a cellular (T-cell) response have

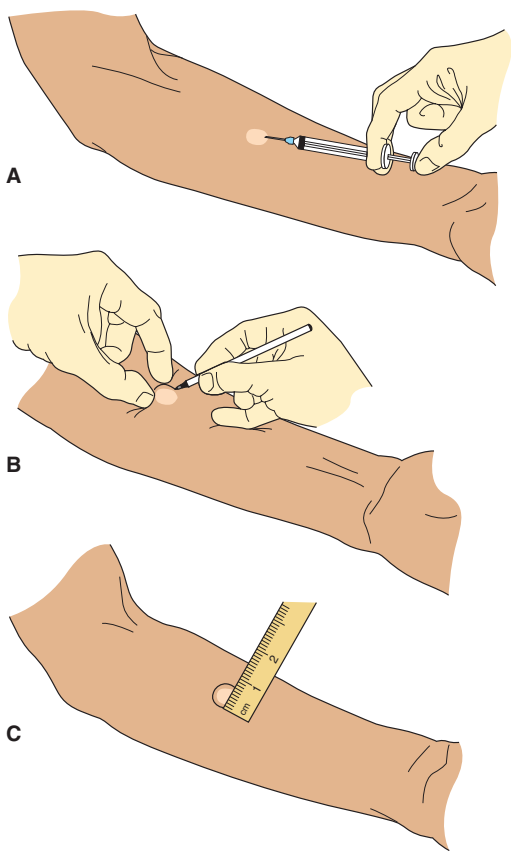


FIGURE 35.15 ■ *A*, Intradermal injection for tuberculin testing. *B*, The injection causes a local inflammatory response (wheal). *C*, Measurement of induration following tuberculin testing

developed; however, it does not mean that active disease is present or that the person is infectious to others.

Tuberculin testing should only be performed by individuals competent in the administration and assessment of the Mantoux test.

An intradermal injection of 5 units of PPD is administered into the inside forearm of the individual. The reaction is read at 48–72 hours. A negative test is defined as induration < 5 mm. All other results should be considered in the context of the clinical picture (RCPA, 2015).

Diagnosis

A positive tuberculin test alone does not indicate active disease. Sputum tests for the bacillus and chest x-rays are routinely used to diagnose and evaluate active disease. A series of three consecutive early morning sputum specimens is typically examined for bacilli (see Procedure 33.1).

See the 'Diagnostic tests' box in Chapter 33 for nursing care related to bronchoscopy.

- *Sputum smear* is microscopically examined for acid-fast bacilli. *M. tuberculosis* resists decolour chemicals after staining. This property is called *acid fast*. The acid-fast smear provides a rapid indicator of the tubercle bacillus.
- *Sputum culture* positive for *M. tuberculosis* provides the definitive diagnosis. However, *M. tuberculosis* is slow growing, requiring 4 to 8 weeks before it can be detected using traditional culture techniques. Automated radiometric culture systems (such as Bactec) allow detection of *M. tuberculosis* in several days.
- Once the organism is detected, *sensitivity testing* is performed to identify appropriate drug therapy.
- *Polymerase chain reaction (PCR)* permits rapid detection of DNA from *M. tuberculosis*.
- *Chest x-ray* is ordered to diagnose and evaluate TB. Typical findings in pulmonary TB include dense lesions in the apical and posterior segments of the upper lobe and possible cavity formation.

Nursing care

Health promotion

Tuberculosis today presents a greater threat to public health than it does to individuals. Nurses play a key role in maintaining public health. Education and tuberculosis screening are major nursing strategies to prevent TB.

Public health teaching includes increasing awareness of TB as a re-emerging threat. Teach people in all settings how to reduce the spread of TB by covering their mouths when coughing or sneezing and disposing of sputum appropriately. The benefit of screening programs to identify infected (though not necessarily infective) people also needs to be included in public health education.

The best TB prevention is early diagnosis of infections and appropriate treatment to achieve cure. BCG vaccine is recommended for infants born in countries where TB is prevalent.

Assessment

Focused assessment for the person with suspected TB includes the following:

- *Health history:* complaints of fatigue, weight loss, night sweats, difficulty breathing, cough (productive or nonproductive), bloody sputum or chest pain; known exposure to TB; most recent tuberculin test and results; living circumstances; alcohol and other recreational drug use.
- *Physical examination:* vital signs, including temperature; general appearance; respiratory rate and lung sounds.
- *Diagnostic tests:* tuberculin test results, presence of acid-fast bacilli in sputum, chest x-ray.

Nursing diagnoses and interventions

Nursing care related to tuberculosis focuses primarily on infection control and compliance with prescribed treatment.

Deficient knowledge

Adequate knowledge and information are necessary to manage the disease and prevent its transmission to others. The person needs to understand reasons for prolonged drug therapy and the importance of complying with treatment and follow up. Antitubercular agents are relatively toxic. The person needs to know how to minimise toxicity.

- Teach about TB and the prescribed treatment, including:
  a. nature of the disease and its spread
  b. purpose of treatment and follow-up procedures
  c. measures to prevent spreading the disease to others
  d. importance of maintaining good general health by eating a well-balanced, high-protein, high-carbohydrate diet; balancing exercise with rest; and avoiding crowds and people with upper respiratory infections
  e. names, doses, purposes and adverse effects of prescribed medications
  f. importance of avoiding alcohol and other substances that may damage the liver while taking chemotherapeutic drugs
  g. fluid intake needs of 2 to 3 L per day
  h. manifestations to report to the doctor: chest pain, haemoptysis, difficulty breathing; anorexia, nausea or vomiting; yellow tint to skin or sclera; sudden weight gain, swollen feet, ankles, legs or hands; hearing loss, tinnitus or vertigo; change in vision or difficulty discriminating colour.

  Tuberculosis is a chronic disease requiring lengthy treatment with antitubercular medications. A good understanding of the disease, its treatment and the potential adverse effects of therapy prepares the person to manage care.
- Document teaching and level of understanding. Reinforce teaching and learning as needed. *Teaching is not complete until the person can demonstrate learning of the information.*

NURSING CARE PLAN A person with tuberculosis

Harry Flanders, aged 53, arrives at a metropolitan public health clinic complaining of aching chest pain that has lasted for the past few days. He says that his sputum also is bloody. He is afraid he might have lung cancer, so he came in to see a doctor.

ASSESSMENT

The public health nurse at the clinic obtains an admission history and physical examination of Mr Flanders. The nurse notes that Mr Flanders is a homeless person who has lived on the streets and in various shelters for the past '10 years or so'. He usually prefers to sleep outdoors, taking refuge in shelters only during very cold or very wet weather. He has a small disability income, but usually scrounges for food or eats with other homeless people at soup kitchens. Mr Flanders states that he has had a cough for a long time, which has become worse recently. It is now productive, especially in the mornings. He also admits that he has recently been waking up drenched with sweat in the middle of the night and is more tired than usual.

Although Mr Flanders' clothes are tattered, he is fairly clean. He answers questions appropriately and intelligently. The nurse does not detect any odour of alcohol on his breath. He is very thin, almost emaciated. Mr Flanders' vital signs are BP 152/86, P 92, R 20 and T 37.8°C.

Suspecting tuberculosis, the nurse obtains a sputum specimen for Gram stain and culture, administers a tuberculin test and sends Mr Flanders for a chest x-ray before he sees the clinic doctor. Although the chest x-ray is inconclusive, the Gram stain is positive for acid-fast bacilli. The diagnosis of probable active pulmonary TB is made. The doctor prescribes isoniazid, 300 mg orally; rifampicin, 600 mg orally; and pyrazinamide, 1500 mg orally daily for 2 months, to be followed by twice-weekly isoniazid 900 mg orally and rifampicin 600 mg orally. The doctor also orders weekly sputum cultures for the first month.

DIAGNOSES

- *Ineffective health maintenance* related to homelessness.
- *Risk of non-adherance with prescribed treatment* related to lack of understanding and resources manifested by failure to attend clinic for direct observation therapy.
- *Inadequate nutrition* related to increased metabolic needs associated with infection manifested by very low body mass index.
- *Risk of altered sensory perception* related to effects of isoniazid therapy manifested by reports of altered sensory deficits.

PLANNING

- Utilise social work services to facilitate securing accommodation.
- Organise for directly observed medical therapy (DOT) to increase medication compliance and reduce multidrug-resistant TB.
- Identify verbally and in writing manifestations to report to the doctor.

Expected outcomes

- Keep all follow-up appointments as scheduled.
- Verbalise an understanding of his disease and its treatment.
- Follow the prescribed plan of care.
- Demonstrate measures to prevent spread of the organism to others.
- Gain 0.5–1 kg of weight per week.
- Promptly report symptoms of peripheral neuropathy, including numbness, tingling or burning sensations.

IMPLEMENTATION

- Teach about TB and provide an education pamphlet about the disease.
- Instruct about the prescribed medications, potential adverse effects and the importance of completing the entire prescribed regimen.
- Emphasise the importance of continued follow up.
- Teach and demonstrate sputum and droplet control measures.

EVALUATION

The healthcare team secures Mr Flanders accommodation and community assistance for DOT. He often still sleeps outside when the weather permits but he adheres to the requirement for supervised medication administration because he 'likes the food there'. Always a clean person, Mr Flanders is able to demonstrate appropriate sputum control measures and practises them faithfully. The sputum culture done after 2 months of treatment is negative for tubercle bacilli and his chest x-ray indicates no disease progression.

CRITICAL THINKING IN THE NURSING PROCESS

1. Many homeless people have schizophrenia or other mental diseases. How would you adapt the care plan for a homeless person with schizophrenia with active tuberculosis?
2. The public health nurse was fortunate in having access to an incentive shelter with healthcare workers to supervise medication compliance. Identify available resources in your area for homeless people infected with tuberculosis.
3. Develop a care plan for the nursing diagnosis of *Ineffective airway clearance* related to mucopurulent sputum and weak cough.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. In this case study accommodation was found for Mr Flanders. Consider the situation where an individual chooses not to accept accommodation. Given that tuberculosis is a public health issue, which next steps would be necessary?

Risk of infection

- Place a mask on the person during transport to other parts of the facility for diagnostic or treatment procedures. *Covering the person's nose and mouth during transport minimises air contamination and the risk to visitors and personnel.*
- Inform all personnel having contact with the person of the diagnosis. *This allows personnel to take appropriate precautions.*
- Assist visitors to mask prior to entering the room. *Providing visitors with appropriate masks or respirators reduces their risk of infection.*
- Teach the person how to limit transmitting the disease to others:
  a. Always cough and expectorate into tissues.
  b. Dispose of tissues properly, placing them in a closed bag.
  c. Wear a mask if you are sneezing or unable to control respiratory secretions.
  d. The disease is not spread by touching inanimate objects, so no special precautions are required for eating utensils, clothing, books or other objects used.

Community-based care

Most people with TB are managed in community settings; few require institutionalisation. In addition to the teaching topics and strategies identified above, discuss the following topics when preparing the person and significant others for home care:

- importance of screening close contacts for infection and possibly prophylactic treatment
- effect, dose and timing for all medications, and potential side effects and their management
- importance of long-term therapy in eradicating the disease
- principles of good nutrition, dietary guidelines for a person with TB and other measures to help maintain good health, such as balancing rest with exercise
- signs and symptoms of complications to report to the doctor or healthcare provider.

Provide referrals as appropriate:

- smoking cessation clinics or support groups
- alcohol treatment facilities, Alcoholics Anonymous, other treatment programs or support groups
- drug treatment facilities, Narcotics Anonymous, other outpatient or inpatient treatment programs or support groups
- community clinics and incentive programs for people with TB
- counselling, support groups and other community resources that provide additional assistance and support.

THE PERSON WITH A FUNGAL INFECTION

Fungal spores are endemic and present in the air everyone breathes. Normal respiratory defence mechanisms allow few of these spores to reach the lungs. If they reach the lungs, pulmonary macrophages and neutrophils efficiently remove them in most people. When they do cause infection, it is typically mild and self-limiting. Most fungi are opportunistic, able to cause infection only in people who are immunocompromised. For this reason, people with AIDS, kidney failure, leukaemia, burns or chronic diseases, as well as people receiving corticosteroids or immunosuppressants, are particularly susceptible to fungal diseases.

The course and manifestations of fungal lung diseases resemble those of tuberculosis. Lung lesions are slow to develop and symptoms are mild. The fungus can disseminate from the lung to other organs.

Pneumocystis carinii pneumonia

Pneumocystis carinii pneumonia (PCP) is caused by a fungus, recently renamed *Pneumocystis jiroveci*. PCP is common in people with AIDS, and others with significant immunocompromise are at risk of developing an opportunistic pneumonia. Opportunistic infection may develop in people treated with immunosuppressive or cytotoxic drugs for cancer or organ transplant and in people with genetic or acquired immunodeficiency.

Infection with PCP produces patchy involvement throughout the lungs, causing affected alveoli to thicken, become oedematous and fill with foamy, protein-rich fluid. Gas exchange is severely impaired as the disease progresses.

PCP has an abrupt onset with fever, tachypnoea and shortness of breath, and a dry, non-productive cough. Respiratory distress can be significant, with intercostal retractions and cyanosis. Table 35.5 compares the manifestations of infectious pneumonias.

Aspergillosis

Aspergillus spores are common in the environment, but rarely cause disease except in the immunocompromised. When they do cause infection, *Aspergillus* species invade blood vessels and produce hyphae that branch at acute angles, frequently causing venous or arterial thrombosis. In the lungs, aspergillosis can cause an acute, diffuse, self-limited pneumonitis. The manifestations of pulmonary aspergillosis include dyspnoea, non-productive cough, pleuritic chest pain, chills and fever. If the organism invades a pulmonary blood vessel, haemoptysis or massive pulmonary haemorrhage can occur (Harman, 2015). In individuals with underlying lung disease, balls of *Aspergillus* hyphae may form within cysts or cavities, usually in the upper lobes of the lung. When this occurs, symptoms often are milder and more insidious in onset, with fever, weight loss, night sweats and cough.

INTERPROFESSIONAL CARE

Most fungal lung infections can be diagnosed by microscopic examination of a sputum specimen for the fungus. Blood cultures also may be taken, as well as cultures of cerebrospinal

TABLE 35.5 Manifestations of infectious pneumonias

TYPE	ONSET	RESPIRATORY MANIFESTATIONS	SYSTEMIC MANIFESTATIONS
Pneumococcal or lobar pneumonia	Abrupt	Cough productive of purulent or rust-coloured sputum; pleuritic or aching chest pain; decreased breath sounds and crackles over affected area; possible dyspnoea and cyanosis	Chills and fever
Bronchopneumonia	Gradual	Cough, scattered crackles; minimal dyspnoea and respiratory distress	Low-grade fever
Legionnaires' disease	Gradual	Dry cough; dyspnoea	Chills and fever; general malaise; headache; confusion; anorexia and diarrhoea; myalgias and arthralgias
Primary atypical pneumonia	Gradual	Dry, hacking, non-productive cough	Fever, headache, myalgias and arthralgias predominate
Viral pneumonia	Sudden or gradual	Dry cough	Flu-like symptoms
Pneumocystis pneumonia	Abrupt	Dry cough; tachypnoea and dyspnoea; significant respiratory distress	Fever

fluid if indicated. Chest x-ray may show typical changes in lung tissue or widening of the mediastinum, depending on the infecting organism.

A broad-spectrum antifungal agent is commonly prescribed to treat most fungal infections. Some fungal lung diseases and individuals who are immunocompromised are often treated with intravenous amphotericin B. Surgery (lobectomy) may be indicated for individuals with severe haemoptysis associated with aspergillosis.

Nursing care

People with fungal lung infections have different nursing care needs, depending on the disease and their immune status. For most people, nursing care focuses on education. People living in high-prevalence areas or who have specific risk factors such as exposure to bird droppings (e.g. by cleaning chicken coops, pigeon lofts or barns where birds roost), decomposed vegetation, rotting wood or stored grain need to be aware of the risk, common symptoms and measures to reduce the risk. Teach people receiving antifungal drugs about the specific drug, its intended and adverse effects, the duration of therapy and symptoms to report to the doctor. Include teaching about any specific precautions such as drug or food interactions. Amphotericin B is a toxic drug. Administer the initial intravenous dose slowly. Occasionally, premedication with an antihistamine and anti-emetic may be indicated to manage its adverse effects. Monitor carefully during infusion and therapy for changes in vital signs, hydration, nutrition, weight or urine output.

DISORDERS OF THE PLEURA

The *pleura* is a thin membrane with two layers: the visceral pleura, which overlies the lung surface; and the parietal pleura, which lines the inner chest wall. Between the layers of pleura is a potential space, the *pleural cavity*, which contains a thin layer of serous fluid. As the thoracic cavity expands during inspiration, the pressure in this space becomes negative in relation to atmospheric and alveolar pressure. The expansible lung is drawn out and air rushes into the alveoli. When the pleura is inflamed or affected by disease or injury, air or fluid can collect in the pleural cavity, restricting lung expansion, air movement and ventilation.

THE PERSON WITH PLEURITIS

Pleuritis (*pleurisy*), inflammation of the pleura, irritates sensory fibres of the parietal pleura, causing characteristic pain. Pleural inflammation usually occurs secondarily to another process, such as a viral respiratory illness, pneumonia or rib injury.

The onset of pleuritis is typically abrupt. The pain is unilateral and well localised; it is usually sharp or stabbing in nature. Pain may be referred to the neck or the shoulder. Deep breathing, coughing and movement aggravate the pain. Respirations

are rapid and shallow, and chest wall movement is limited on the affected side. Breath sounds are diminished and a pleural friction rub may be heard over the site.

The diagnosis of pleuritis is based on its manifestations. Chest x-ray and ECG may be ordered to rule out other causes of chest pain. Treatment for pleuritis is symptomatic. Analgesics and non-steroidal anti-inflammatory drugs (NSAIDs)—indomethacin (Indocin), in particular—help relieve the pain. Codeine may be ordered, both to relieve pain and to suppress the cough.

Nursing care for the person with pleuritis is directed towards promoting comfort, including administration of NSAIDs and analgesics. Positioning and splinting the chest while coughing also are helpful.

Teach the individual and family that pleuritis is generally self-limited and of short duration. Discuss symptoms to report to the doctor: increased fever, productive cough or shortness of breath. Provide information about prescription and non-prescription NSAIDs and analgesics, including the drug ordered, how to use it and its desired and possible adverse effects.

THE PERSON WITH A PLEURAL EFFUSION

The pleural space normally contains only about 10 to 20 mL of serous fluid.

Pleural effusion is collection of excess fluid in the pleural space. Pleural effusions result from either systemic or local disease. Systemic disorders that may lead to pleural effusion include heart failure, liver or kidney disease, and connective tissue disorders, such as rheumatoid arthritis and systemic lupus erythematosus (SLE). Pneumonia, atelectasis, tuberculosis, lung cancer and trauma are local conditions that may cause pleural effusion.

Pathophysiology and manifestations

Excess pleural fluid may be either *transudate*, formed when capillary pressure is high or plasma proteins are low, or *exudate*, the result of increased capillary permeability. Heart failure is the most common precipitating factor in transudate formation; it may also accompany kidney failure, nephrosis, liver failure and malignancy. Exudate, a protein-rich fluid, is seen with inflammatory processes such as infections, systemic inflammation (e.g. rheumatoid arthritis or SLE), pulmonary infarction (leading to tissue necrosis and an inflammatory response) and malignancy (Bullock & Hales, 2012). Other pleural fluid collections include *empyema*, pus in the pleural cavity; *haemothorax*, the presence of blood in the cavity; *haemorrhagic pleural effusion*, a mixture of blood and pleural fluid; and *chylothorax*, a collection of lymph in the pleural space. In adults, chylothorax may be iatrogenic resulting from thoracic surgery or placement of a central line in one of the great veins (Adams, 2014).

A large pleural effusion compresses adjacent lung tissue. This causes the characteristic manifestation of dyspnoea. Pain may develop, although with inflammatory processes pleuritic pain often is relieved by formation of an effusion, as the fluid reduces friction between inflamed visceral and parietal pleura. Breath sounds are diminished or absent and a dull percussion tone is heard over the affected area. Chest wall movement may be limited.

INTERPROFESSIONAL CARE

Chest x-ray often provides the first evidence of a pleural effusion. Because fluid typically collects in dependent regions, it is seen at the base of the affected lung on an upright chest x-ray and along the lateral wall when the person is positioned on the affected side. CT scans and ultrasonography also are used to localise and differentiate pleural effusions.

Thoracentesis

If the cause of pleural effusion is not apparent, a thoracentesis is undertaken. **Thoracentesis** is an invasive procedure in which fluid (or occasionally air) is removed from the pleural space with a needle. Aspirated fluid is analysed for appearance, cell counts, protein and glucose content, the presence of enzymes such as LDH and amylase, abnormal cells and culture.

When pleural effusion is significant and interferes with respirations, thoracentesis is the treatment of choice to remove the fluid (see Figure 35.16). Thoracentesis may be performed at the bedside, in a procedure room or in an outpatient setting. Local anaesthesia is used and the procedure can be performed in less than 30 minutes. Percussion, auscultation, radiography or ultrasonography are used to locate the effusion and needle insertion site. The amount of fluid removed is limited to 1200 to 1500 mL at one time to reduce the risk of cardiovascular collapse from rapid removal of too much fluid. Pneumothorax is a possible complication of thoracentesis if the visceral pleura is punctured or a closed drainage system is not maintained during the procedure. Nursing care for the person undergoing a thoracentesis is outlined in the box below.

Treatments

Because pleural effusion usually occurs secondarily to another disease or disorder, medical management also focuses on treating the underlying condition to prevent further fluid accumulation. An empyema may require repeated drainage, as well as high doses of parenteral antibiotics. Occasionally, thoracotomy and surgical excision may be necessary. See the box later in this chapter for nursing care of the person having lung surgery. Recurrent pleural effusions, often due to cancer, may be prevented by instilling an irritant, such as talc, into the pleural space to cause adhesion of the parietal and visceral pleura (*pleurodesis*). Water-seal chest tube drainage is often employed for haemothorax.

Nursing care

Nursing care for the person with a pleural effusion is directed towards supporting respiratory function and assisting with procedures to evacuate collected fluid. With a large pleural effusion and partial lung collapse, *Impaired gas exchange* and

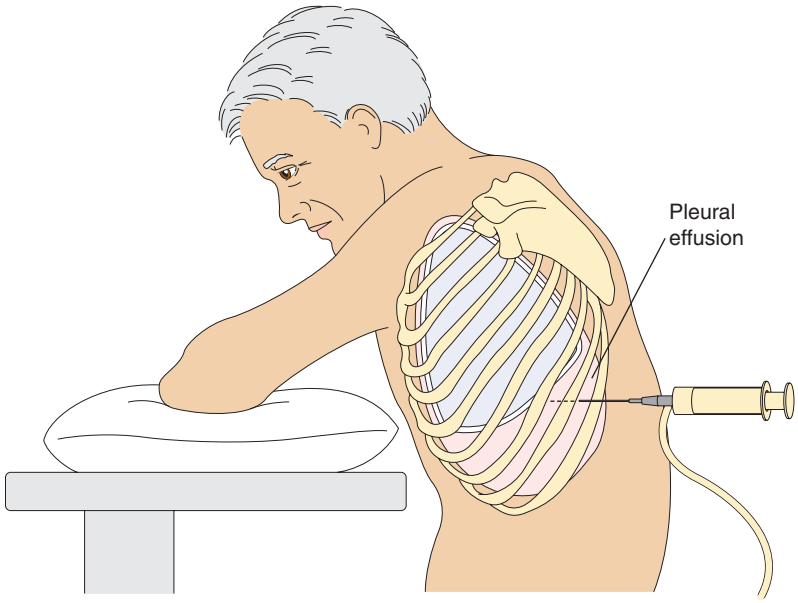


FIGURE 35.16 ■ Thoracentesis. With the person seated, a needle is inserted between the ribs into the pleural space to withdraw accumulated fluid

Activity intolerance are high-priority nursing problems. *Risk of impaired gas exchange* is also a priority problem during the initial period following thoracentesis.

Teaching for home care focuses on symptoms of recurrent effusion or complications following a thoracentesis to report to the doctor: increasing dyspnoea, cough and haemoptysis. Pleuritic pain may be an early sign of effusion and should also be reported. Further teaching about an underlying condition also may be necessary; for example, the person with heart failure may need teaching about a salt-restricted diet.

NURSING CARE OF THE PERSON having a thoracentesis

BEFORE THE PROCEDURE

- Verify a signed informed consent for the procedure. *This invasive procedure requires informed consent.*
- Assess knowledge and understanding of the procedure and its purpose; provide additional information as needed. *A person who is fully informed will be less apprehensive and more able to cooperate during the thoracentesis.*
- Pre-procedure fasting or sedation is not required. *Only local anaesthesia is used in this procedure and the gag and cough reflexes remain intact.*
- Administer a cough suppressant if indicated. *Movement and coughing during the procedure may cause inadvertent damage to the lung or pleura.*
- Obtain a thoracentesis tray, sterile gloves, injectable lignocaine, povidone-iodine, dressing supplies and an extra over-bed table or Mayo stand. *These supplies are used by the doctor performing the procedure.*
- Position the person upright, leaning forward with arms and head supported on an anchored over-bed table. *This position spreads the ribs, enlarging the intercostal space for needle insertion.*
- Inform the person that, although local anaesthesia prevents pain as the needle is inserted, a sensation of pressure may be felt. *A pressure sensation occurs as the needle punctures the parietal pleura to enter the pleural space.*

DURING AND AFTER THE PROCEDURE

- Monitor pulse, colour, oxygen saturation and other signs during thoracentesis. *These are indicators of physiological tolerance of the procedure.*
- Apply a dressing over the puncture site and position on the unaffected side for 1 hour. *This allows the pleural puncture to heal.*
- Label obtained specimen with name, date, time, source; send specimen to the laboratory for analysis. *Fluid obtained during thoracentesis may be examined for abnormal cells, bacteria and other substances to determine the cause of the pleural effusion.*
- During the first several hours after thoracentesis, frequently assess and document vital signs; oxygen saturation; respiratory status, including respiratory excursion, lung sounds, cough or haemoptysis; and puncture site for bleeding or crepitus. *Frequent assessment is important to detect possible complications of thoracentesis, such as pneumothorax.*
- Obtain a chest x-ray. *Chest x-ray is ordered to detect possible pneumothorax.*
- Normal activities generally can be resumed after 1 hour if no evidence of pneumothorax or other complication is present. *The puncture wound of thoracentesis heals rapidly.*

THE PERSON WITH PNEUMOTHORAX

Accumulation of air in the pleural space is called **pneumothorax**. Pneumothorax can occur spontaneously, without apparent cause, as a complication of pre-existing lung disease, as a result of blunt or penetrating trauma to the chest, or from an iatrogenic cause (e.g. following thoracentesis).

Pathophysiology

Pressure in the pleural space is normally negative in relation to atmospheric pressure. This negative pressure is vital to the process of breathing. Contraction of the diaphragm and the intercostal muscles enlarges the thoracic space. Negative intrapleural pressure draws the lung outwards, increasing its volume so air rushes in to fill the expanded lung space.

When either the visceral or the parietal pleura is breached, air enters the pleural space, equalising this pressure. Lung expansion is impaired and the natural recoil tendency of the lung causes it to collapse to a greater or lesser extent, depending on the size and rapidity of air accumulation. Table 35.6 illustrates the classifications of pneumothorax.

Spontaneous pneumothorax

Spontaneous pneumothorax develops when an air-filled bleb, or blister, on the lung surface ruptures. Rupture allows air from the airways to enter the pleural space. Air accumulates until pressures are equalised or until collapse of the involved lung section seals the leak. Spontaneous pneumothorax may be either *primary* (*simple*) or *secondary* (*complicated*).

Primary pneumothorax affects men who are relatively well, most often smokers, usually tall, slender and between ages 18 and 40 (Daley, 2015). The cause of primary pneumothorax is unknown. Air-filled blebs tend to form in the apices of the lungs. This is considered to be a benign condition, although recurrences are common. Certain activities also increase the risk of spontaneous pneumothorax, such as high-altitude flying and rapid decompression during scuba diving.

Secondary pneumothorax, generally caused by overdistension and rupture of an alveolus, is more serious and potentially life threatening. It develops in people with underlying lung disease, usually COPD. Middle-aged and older adults are primarily affected. Secondary pneumothorax also may be associated with asthma, cystic fibrosis, pulmonary fibrosis, tuberculosis, acute respiratory distress syndrome (ARDS) and other lung diseases. Rarely, a form of secondary pneumothorax called *catamenial pneumothorax* can develop in affected women within 24 to 48 hours of the onset of menstrual flow.

MANIFESTATIONS The manifestations of spontaneous pneumothorax depend on size of the pneumothorax, extent of lung collapse and any underlying lung disease. Typically, pleuritic chest pain and shortness of breath begin abruptly, often while at rest. The respiratory and heart rates increase as gas exchange is affected. Chest wall movement may be asymmetrical, with less movement on the affected side than the unaffected side. The affected side is hyperresonant to percussion and breath sounds may be diminished or absent. Hypoxaemia may develop, although normal mechanisms that shunt blood flow to the unaffected lung often maintain normal oxygen saturation levels. Hypoxaemia is more pronounced in secondary pneumothorax.

Traumatic pneumothorax

Blunt or penetrating trauma of the chest wall and pleura can cause pneumothorax. Blunt trauma—for example, due to a motor vehicle crash, fall or during cardiopulmonary resuscitation (CPR)—can lead to a *closed pneumothorax*. Fractured ribs penetrating the pleura are the leading cause of pneumothorax due to blunt trauma (Daley, 2015). Fracture of the trachea and a ruptured bronchus or oesophagus also may result from blunt trauma, leading to closed pneumothorax.

Open pneumothorax (*sucking chest wound*) results from penetrating chest trauma such as a stab wound, gunshot wound or impalement injury. With open pneumothorax, air moves freely between the pleural space and the atmosphere through the wound. Pressure on the affected side equalises with the atmosphere and the lung collapses rapidly. The result is significant hypoventilation.

Iatrogenic pneumothorax may result from puncture or laceration of the visceral pleura during central-line placement, thoracentesis or lung biopsy. During bronchoscopy, bronchi or lung tissue can be disrupted. Alveoli can become over distended and rupture during anaesthesia, resuscitation procedures or mechanical ventilation.

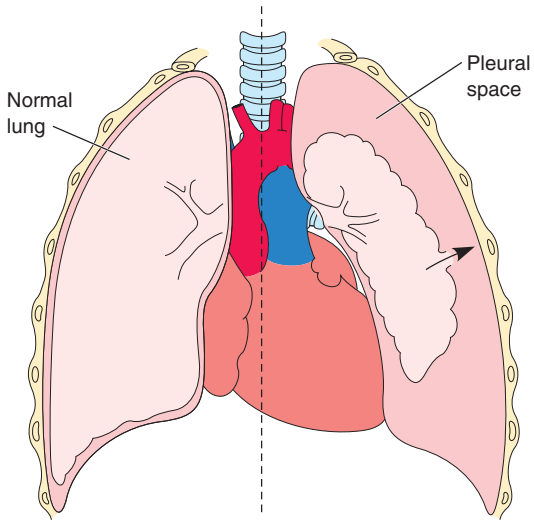
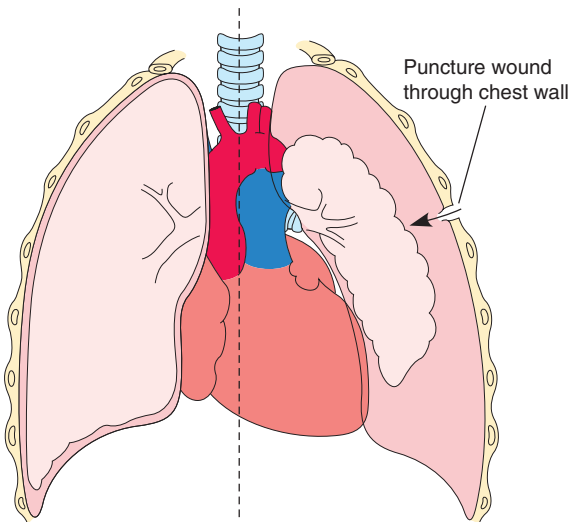
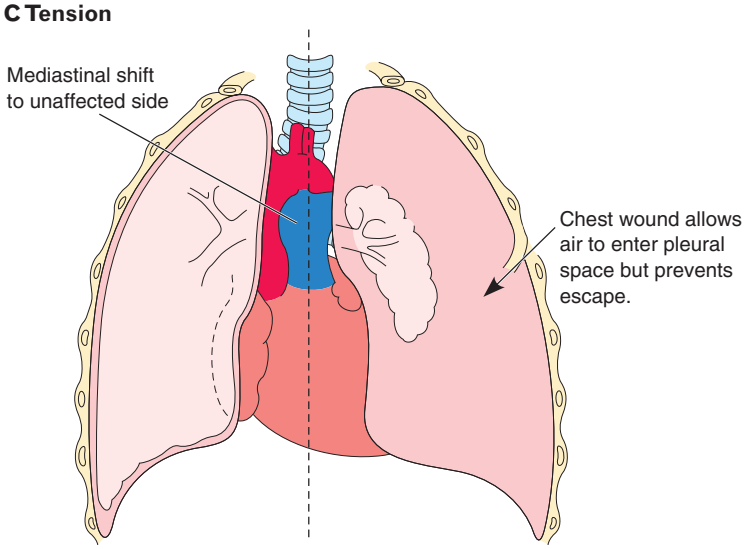
MANIFESTATIONS With traumatic pneumothorax, manifestations of pain and dyspnoea may be masked or missed due to other injuries. Tachypnoea and tachycardia may be attributed to the primary injury. Focused assessment for evidence of pneumothorax is vital. Chest wall movement on the affected side is diminished and breath sounds are absent. If a penetrating wound is present, air may be heard and felt moving through it with respiratory efforts. Haemothorax frequently accompanies traumatic pneumothorax. The manifestations of iatrogenic pneumothorax are similar to those of spontaneous pneumothorax.

Tension pneumothorax

Tension pneumothorax develops when injury to the chest wall or lungs allows air to enter the pleural space but prevents it from escaping. Pressure within the pleural space becomes positive in relation to atmospheric pressure as air rapidly accumulates with each breath. The lung on the affected side collapses and pressure on the mediastinum shifts thoracic organs to the unaffected side of the chest, placing pressure on the opposite lung as well. Ventilation is severely compromised and venous return to the heart is impaired. Tension pneumothorax is a medical emergency requiring immediate intervention to preserve respiration and cardiac output.

MANIFESTATIONS In addition to manifestations of pneumothorax, hypotension and distended neck veins are evident as venous return and cardiac output are affected. The trachea is displaced towards the unaffected side as a result of the mediastinal shift. Signs of shock may be present. See Chapter 10 for the manifestations and treatment of shock.

TABLE 35.6 Types of pneumothorax

TYPE	PATHOPHYSIOLOGY	MANIFESTATIONS
A Spontaneous 	Rupture of a bleb on the lung surface allows air to enter pleural space from airways. • *Primary pneumothorax* affects previously healthy people. • *Secondary pneumothorax* affects people with pre-existing lung disease (e.g. COPD).	• Abrupt onset • Pleuritic chest pain • Dyspnoea, shortness of breath • Tachypnoea, tachycardia • Unequal lung excursion • Decreased breath sounds and hyperresonant percussion tone on affected side
B Traumatic 	Trauma to the chest wall or pleura disrupts the pleural membrane. • *Open* occurs with penetrating chest trauma that allows air from the environment to enter the pleural space. • *Closed* occurs with blunt trauma that allows air from the lung to enter the pleural space. • *Iatrogenic* involves laceration of visceral pleura during a procedure such as thoracentesis or central-line insertion.	• Pain • Dyspnoea • Tachypnoea, tachycardia • Decreased respiratory excursion • Absent breath sounds in affected area • Air movement through an open wound
C Tension 	Air enters pleural space through chest wall or from airways but is unable to escape, resulting in rapid accumulation. Lung on affected side collapses. As intrapleural pressure increases, heart, great vessels, trachea and oesophagus shift towards the unaffected side.	• Hypotension, shock • Distended neck veins • Severe dyspnoea • Tachypnoea, tachycardia • Decreased respiratory excursion • Absent breath sounds on affected side • Tracheal deviation towards unaffected side

INTERPROFESSIONAL CARE

Treatment for pneumothorax depends on the severity of the problem. A small simple pneumothorax may require no treatment other than monitoring with serial x-rays. Air is absorbed from the pleural space, allowing most small pneumothoraces to resolve spontaneously. A large pneumothorax with significant symptoms usually requires treatment with *thoracostomy* or the placement of chest tubes. Surgical intervention may be necessary to prevent recurrent spontaneous pneumothorax.

Diagnosis

Oxygen saturation measurements are obtained to evaluate the effect of pneumothorax on gas exchange. ABGs may be obtained to further assess gas exchange.

The chest x-ray is an effective diagnostic tool for pneumothorax. In tension pneumothorax, air is evident on the affected side and mediastinal structures are shifted towards the opposite or unaffected side.

Treatments

CHEST TUBES The treatment of choice for significant pneumothorax is placement of a closed-chest catheter to allow the lung to re-expand. When a tube is placed in the pleural cavity to remove air or fluid, it must be sealed to prevent air from also entering the tube and, in essence, creating an open pneumothorax. Chest tubes are sealed with a Heimlich (one-way) valve (see Figure 35.17) or connected to a closed drainage system with a 'water seal'. The valve or water seal prevents air from entering the chest cavity during inspiration and allows air to escape during expiration. Applying a low level of suction to the system helps to re-establish negative pressure in the pleural space, allowing the lung to re-expand.

A number of closed-drainage chest tube systems are available. Most are self-contained disposable systems (see Figure 35.18). Drainage from the chest tube is collected in the first collection chamber. This sealed chamber is connected to a water seal chamber, which is in turn connected to the suction control chamber. Nursing care of the person with chest tubes is discussed in the box below.

A large-bore needle or plastic intravenous catheter may be inserted through the chest wall as emergency treatment of a tension pneumothorax. This allows air to escape from the affected side, relieving pressure on mediastinal structures and the opposite lung.

FIGURE 35.17 ■ The Heimlich one-way valve allows air to escape from the pleural space, helping to re-establish negative pressure and allowing the lung to re-expand

Source: George Draper/Pearson Education.

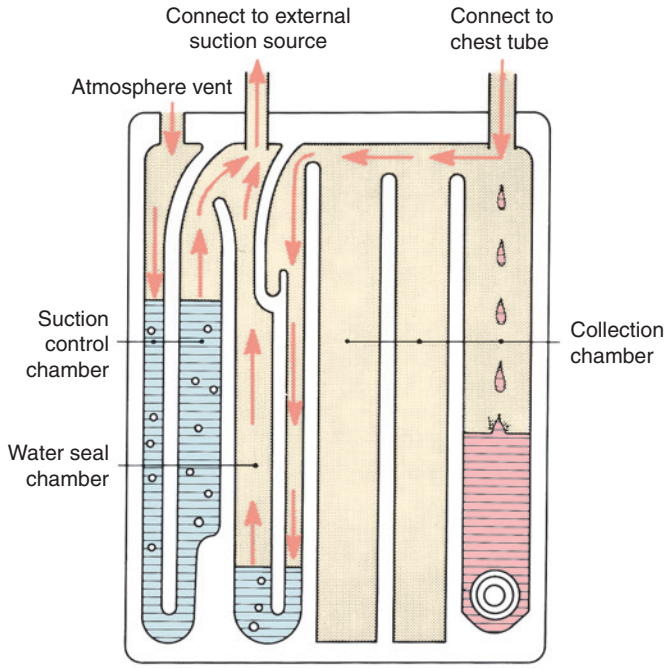


FIGURE 35.18 ■ A closed-chest drainage system

PLEURODESIS Although controversial, *pleurodesis*, or creation of adhesions between the parietal and visceral pleura, may be used to prevent recurrent pneumothorax. This procedure involves instilling a chemical agent such as talc into the pleural space. The subsequent inflammatory response creates scar tissue and adhesions between the pleural layers. This procedure reduces the recurrence rate to as low as 2% but can make subsequent surgery more difficult (Daley, 2015).

Surgery

The risk of recurrence of spontaneous pneumothorax increases with each attack. People at high risk of recurrent pneumothorax may have surgery to reduce the risk of future ruptures. A thoracotomy is done to excise or oversew blebs (usually at the apices of the lungs). The overlying pleura is then roughened or irritated to induce scarring and adhesion to the surface of the lung. In some cases, the parietal pleura may be partially excised. These procedures can be done using video-assisted thoracoscopic surgery (VATS), a minimally invasive surgical technique (Daley, 2015).

Nursing care

Health promotion

Health promotion activities to prevent spontaneous and traumatic pneumothorax primarily involve health teaching. Initiate and participate in programs to prevent smoking by children and teenagers.

NURSING CARE OF THE PERSON with chest tubes

BEFORE THE PROCEDURE

- Ensure a signed informed consent for chest tube insertion. *This invasive procedure requires informed consent.*
- Provide additional information as indicated. Explain that local anaesthesia will be used but that pressure may be felt as the trocar is inserted. Reassure that breathing will be easier once the chest tube is in place and the lung re-expands. *The person may be extremely anxious and may need reassurance that this invasive procedure will provide relief.*
- Gather all needed supplies, including thoracostomy tray, injectable lignocaine, sterile gloves, chest tube drainage system, sterile water and a large, sterile, catheter-tipped syringe to use as a funnel for filling water-seal and suction chambers. *These supplies are used during the insertion procedure to establish a water-seal drainage system.*
- Position as indicated for the procedure. *Either an upright position (as for thoracentesis) or a side-lying position may be used, depending on the site of the pneumothorax.*
- Assist with chest tube insertion as needed. The procedure may be performed in a procedure room, in the surgical suite or at the bedside. *Although chest tube insertion is a relatively simple procedure, nursing assistance is necessary to support the person and rapidly establish a closed drainage system.*

AFTER THE PROCEDURE

- Assess respiratory status at least every 4 hours. *Frequent assessment is necessary to monitor respiratory status and the effect of the chest tube.*
- Maintain a closed system. Tape all connections and secure the chest tube to the chest wall. *These measures are important to prevent inadvertent tube removal or disruption of system integrity.*
- Keep the intercostal catheter's underwater-seal drain system below the level of the chest. *Pleural fluid drains into the collection apparatus by gravity flow.*
- Check tubes frequently for kinks or loops. *These could interfere with drainage.*
- Check the water seal frequently. The water level should fluctuate with respiratory effort. If it does not, the system may not be patent or intact. Periodic air bubbles in the water-seal chamber are normal and indicate that trapped air is being removed from the chest. *Frequent assessment of the system is important to ensure appropriate functioning.*
- Measure swinging, bubbling and drainage every hour, marking the level on the drainage chamber. Report drainage that is cloudy, in excess of 70 mL per hour or red, warm and free flowing. *Red, free-flowing drainage indicates haemorrhage; cloudiness may indicate an infection.*
- Periodically assess water level in the suction control chamber, adding water as necessary. *Adequate water in the suction control chamber prevents excess suction from being placed on delicate pleural tissue.*
- Assist with frequent position changes and sitting and ambulation as allowed. Chest tubes should not prevent performance of allowed activities. *Care is needed to prevent inadvertent disconnection or removal of the tubes.*
- When the chest tube is removed, immediately apply a sterile occlusive dressing. *An occlusive dressing prevents air from re-entering the pleural space through the chest wound.*

Teach safe behaviours such as always wearing a seat belt in a motor vehicle, driving safely and using precautions to prevent falls when working or recreating in high places.

Assessment

The person with pneumothorax may be in acute respiratory distress, necessitating rapid and focused assessment.

- *Health history:* current symptoms and their duration; precipitating factors or activities if known; previous episodes of pneumothorax; smoking history; chronic pulmonary diseases such as COPD.
- *Physical assessment:* general appearance and degree of apparent respiratory distress; evidence of chest trauma; vital signs, oxygen saturation, skin colour, LOC; respiratory excursion, percussion tone and breath sounds anterior and posterior chest; neck vein inspection, position of trachea; peripheral pulses.
- *Diagnostic tests:* chest x-ray, ABGs.

Nursing diagnoses and interventions

Maintaining or restoring adequate alveolar ventilation and gas exchange is of highest priority for the person with a pneumothorax. Chest tubes may interfere with physical mobility, contributing to a high risk of injury.

Impaired gas exchange

Loss of negative pressure in the pleural cavity and the resulting collapse of lung tissue can cause poor chest expansion and loss of alveolar ventilation. As the pneumothorax is removed or reabsorbed, ventilation and gas exchange improve.

- Assess and document vital signs and respiratory status, including rate, depth, lung sounds and oxygen saturation at least every 4 hours. *Frequent assessment is important to monitor the adequacy of respirations and lung expansion.*
- Place in Fowler's or high-Fowler's position. *This position facilitates lung expansion.*
- Administer oxygen as ordered. *Supplemental oxygen is given to improve oxygenation of the blood and tissues.*
- Assess chest tube, system function and drainage every hour. The system must remain patent and intact to function effectively. Assess swinging, bubbling and draining.
- Provide rest. *Adequate rest is important to conserve energy and reduce oxygen demand.*

CONSIDERATION FOR PRACTICE

Provide emotional support, particularly in early stages and during chest tube insertion. Dyspnoea and hypoxaemia can cause extreme anxiety and apprehension, impairing the ability to cooperate with procedures.

Risk of injury

Pain and the presence of chest tubes can reduce the perceived ability to ambulate and provide self-care. Moderate activity is encouraged unless respiratory function is significantly impaired. Caution is taken to maintain integrity of the chest tube system. If the tube is inadvertently pulled out or system integrity is disrupted, the pneumothorax may increase or infection may develop.

CONSIDERATION FOR PRACTICE

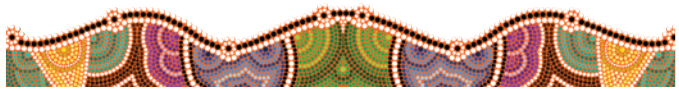
Avoid placing tension on chest tubes during positioning, ambulation and care activities. The chest tubes are minimally secured to the chest wall and can be dislodged if tension is placed on them.

- Secure a loop of drainage tubing to the gown. *Looping the drainage tubing prevents direct pressure on the chest tube itself.* Be sure not to dislodge tube when removing gown.
- When turning to the affected side, ensure that neither the chest tube nor the drainage tubing is kinked or occluded under the person. *This maintains patency of the system.*
- Teach the person how to ambulate with the drainage system, keeping the system lower than the chest. In most cases, suction can be discontinued during ambulation. *Ambulation facilitates lung ventilation and expansion. Drainage systems are portable to allow ambulation while chest tubes are in place. Keeping the drainage system lower than the chest promotes drainage and prevents reflux.*
- Observe insertion site for redness, swelling, pain or drainage. Report any signs of infection, including fever, to the doctor. *Interruption of skin integrity by chest tube insertion increases the risk of infection.*
- If a connection comes loose, reconnect it as soon as possible. *A closed, sealed system is vital to prevent air from entering the pleural space and an open pneumothorax.*

Community-based care

People who have experienced spontaneous pneumothorax need education about their future risk. After a single episode of spontaneous pneumothorax, the risk of recurrence is 15–30%. This risk increases with subsequent episodes (Daley, 2015). Stress the importance of quitting smoking to reduce the risk. Other activities that can precipitate recurrent episodes include mountain climbing or those involving exposure to high altitudes, flying in unpressurised aircraft and scuba diving (Daley, 2015). The person may be advised to avoid contact sports.

Following a pneumothorax, instruct the person to gradually increase exercise and activity to previous levels. Stress the importance of follow-up care and monitoring. Discuss manifestations to report to the doctor: upper respiratory infections; fever, cough or difficulty breathing; sudden, sharp chest pain; or redness, pain, swelling, tenderness or drainage from the chest tube puncture wound.



THE PERSON WITH HAEMOTHORAX

Haemothorax, or blood in the pleural space, usually occurs as a result of chest trauma, surgery or diagnostic procedures. Haemothorax develops in about 30–60% of people with chest trauma, usually due to laceration of the lung, an intercostal vessel or the internal mammary artery. If a major thoracic vessel is disrupted, haemorrhage can be massive (Mancini, 2014; Papadakis et al., 2013). Tumour, pulmonary infarction and infections such as tuberculosis also can cause haemothorax. When blood collects in the pleural space, pressure on the affected lung impairs ventilation and gas exchange. With significant haemorrhage, a risk of shock exists.

Haemothorax causes symptoms similar to those of pneumothorax or pleural effusion. Lung sounds are diminished and a dull percussion tone is noted over the collected blood, typically at the base of the lung. Chest x-ray is used to confirm the diagnosis of haemothorax.

Thoracentesis or thoracostomy with chest tube drainage is used to remove blood from the pleural space. With significant haemorrhage (e.g. due to trauma or surgery), the blood may be collected for subsequent autotransfusion; this blood should be collected and reinfused within 4 hours. Strict aseptic technique is used in collecting the blood. It is collected through a gross particulate filter into a container primed with anticoagulant and reinfused when the container is full or when transfusion is necessary. Air is removed from the blood container prior to reinfusion and a filter is used to eliminate debris such as degenerating blood cells, fat particles and fibrin.

Priority nursing care for the person with haemothorax focuses on assessing and maintaining adequate respiratory function and cardiac output. The priority of care depends on the rate and extent of haemothorax. In a large, slow-developing haemothorax, ventilatory status may be affected significantly. In this instance, *Impaired gas exchange* and *Ineffective breathing pattern* are priority nursing diagnoses. When haemothorax develops rapidly and haemorrhage is significant, additional priority nursing diagnoses include *Decreased cardiac output* and *Risk of deficient fluid volume*.

When preparing the person for home care following a haemothorax, discuss the importance of avoiding smoking and preventing respiratory infection. Include symptoms to report to the doctor. If trauma or infection caused the haemothorax, discuss measures to prevent future trauma and continuing treatment for the infection as indicated.

TRAUMA OF THE CHEST OR LUNG

Chest injury is a leading cause of death from trauma. It is commonly associated with motor vehicle crashes, violent crime and falls. Chest injuries can range from mild, such as a simple rib fracture, to severe and fatal. Traumatic injury to the chest may involve both the chest wall and underlying thoracic structures, including the lungs, heart, great vessels and oesophagus. Chest and lung injury can result from several different mechanisms: penetrating trauma, such as a stab or gunshot wound; blunt trauma, such as a fall, motor vehicle accident (MVA), vehicle–pedestrian impact or crush injury; or inhalation injury, such as smoke inhalation or near drowning.

Rapid and continuing primary survey assessing airway, breathing and circulation (ABC) is vital in chest or lung injuries. Chest trauma can disrupt any or all of these functions. Chest injuries that may be life threatening include airway obstruction, tension pneumothorax, open pneumothorax, massive haemothorax and flail chest with pulmonary contusion.

THE PERSON WITH A THORACIC INJURY

Thoracic injuries may be minor and have little effect on respiratory status; for example, simple rib fracture in a previously healthy person. When pain or chest wall instability impairs breathing or the underlying lung tissue is damaged, the risk is more significant. Thoracic trauma usually is caused by motor vehicle crashes or falls.

Pathophysiology and manifestations

Acceleration–deceleration injury and direct mechanisms of injury (e.g. crush injuries) are the most common mechanisms of thoracic injuries. Acceleration–deceleration injuries are caused by a rapid change in velocity such as occurs in an MVA or fall. The body stops suddenly, but the tissues and organs within the chest cavity continue to move forward until they impact with the chest wall. Injuries sustained can be significant, depending on the velocity (speed) of the vehicle or body at the point of impact, the surface with which the body impacts and individual characteristics (e.g. size and bone structure).

Rib fracture

Simple rib fracture, usually involving a single rib, is the most common chest wall injury. Rib fracture generally is tolerated well and heals rapidly in a young, previously healthy person. In an older adult or person with pre-existing lung disease, however, a fractured rib may lead to significant complications, such as pneumonia, atelectasis and, potentially, respiratory failure. Displaced fractured ribs can penetrate the pleura, leading to pneumothorax and possible haemothorax. Fractures of certain ribs are more frequently associated with underlying tissue damage. Intrathoracic vessels may be damaged or torn with fractures of the first and second ribs. Fractures of the seventh to tenth ribs may cause liver or spleen injuries.

Rib fracture causes pain on inspiration and coughing. This leads to voluntary splinting, with rapid, shallow respirations and inhibited cough. Bruising may be seen over the fracture and crepitus may be palpated with respiratory movement. Breath sounds are diminished, especially in the bases, due to splinting. If pneumothorax develops, chest wall movement on the affected side may be reduced and breath sounds absent or significantly diminished. A hyper-resonant percussion tone usually is noted. Haemothorax also causes diminished or absent breath sounds on the affected side, with a dull percussion note.

Flail chest

Multiple rib fractures may impair chest wall stability and normal chest wall function. When two or more consecutive ribs are fractured in multiple places, a free-floating segment of the chest wall, or **flail chest**, results. Physiological function of the chest wall is impaired as the flail segment is sucked inwards during inhalation and moves outwards with exhalation. This is known as *paradoxical movement* (see Figure 35.19).

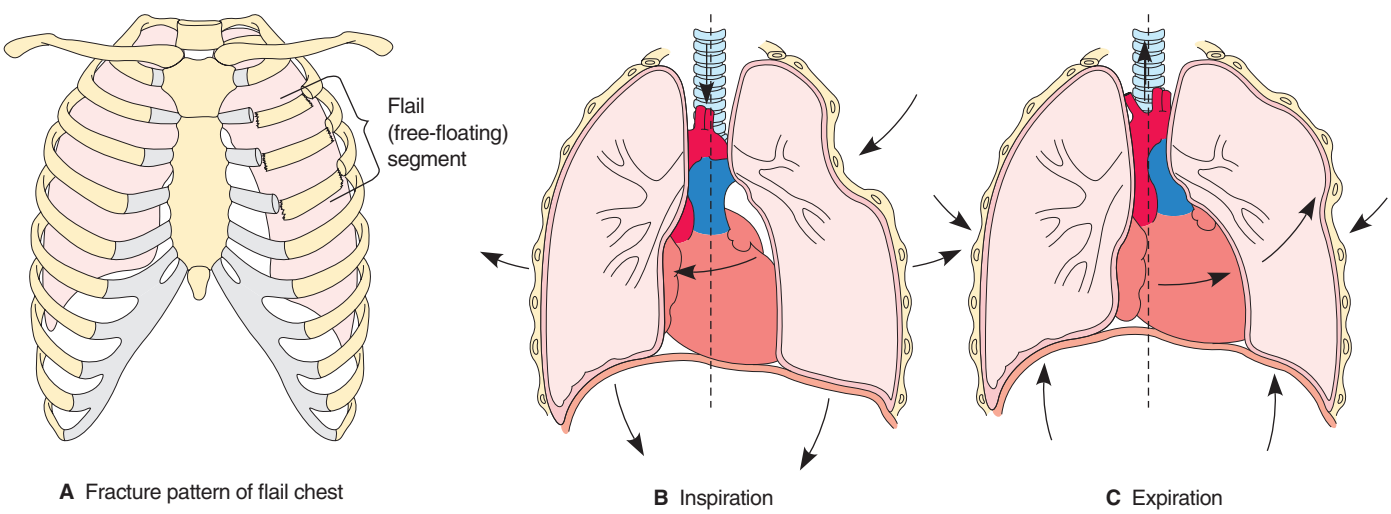


FIGURE 35.19 ■ Flail chest with paradoxical movement

Flail chest can significantly affect ventilation and, consequently, gas exchange. Lung expansion is impaired and the work of breathing increases. Flail chest is frequently associated with underlying pulmonary contusion, which may lead to respiratory failure.

Flail chest causes dyspnoea and pain, especially on inspiration. Paradoxical chest movement is evident with inspection. Chest expansion is unequal and palpable crepitus is present. Breath sounds are diminished and crackles may be heard on auscultation.

Pulmonary contusion

Pulmonary contusion, or lung tissue injury, is frequently associated with flail chest and other blunt chest trauma. It may occur unilaterally or bilaterally. Pulmonary contusion often results from abrupt chest compression followed by sudden decompression, as can occur with MVA, significant fall or crush injury. Alveoli and pulmonary arterioles rupture, causing intra-alveolar haemorrhage and interstitial and bronchial oedema. The resulting inflammatory response increases capillary permeability, leading to oedema, which may be localised to the damaged lung tissue or more generalised. Inflammation and oedema impair the production of surfactant within the alveoli, decreasing compliance. Pulmonary vascular resistance increases and blood flow decreases. Airway obstruction, atelectasis and impaired gas diffusion result. Associated chest wall injury impairs the ability to clear secretions effectively and the work of breathing is significantly increased.

Manifestations of pulmonary contusion may not be apparent until 12 to 24 hours after the injury. Increasing shortness of breath, restlessness, apprehension and chest pain are early signs. Copious sputum, which may be blood tinged, is present. Later manifestations include tachycardia, tachypnoea, dyspnoea and cyanosis. Even with appropriate treatment, pulmonary contusion can lead to acute respiratory distress and potential death.

INTERPROFESSIONAL CARE

Diagnosis

Chest x-ray is used to identify most chest wall injuries. Rib fractures are evident on x-ray. Pulmonary contusion may show as initial patchy opacifications progressing to diffuse opacification or ‘white out’. Changes in oxygen saturation and ABGs depend on the degree to which ventilation and gas exchange are affected by the injury.

Management

Simple rib fractures typically heal uneventfully. Providing adequate analgesia to promote breathing, coughing and movement is the primary intervention. With multiple rib fractures, an intercostal nerve block may be used to ensure adequate ventilation. Rib belts, binders and taping to stabilise the rib cage are not recommended, because they may interfere with ventilation and lead to atelectasis. Even with simple rib fracture, older people and individuals with pre-existing lung disease require close monitoring to prevent and detect atelectasis, pneumonia and other complications.

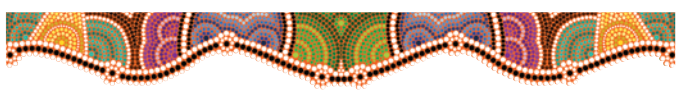
Intercostal nerve blocks or continuous epidural analgesia may be employed to manage the pain associated with flail chest. For a small flail chest, analgesia combined with supplemental oxygen therapy may be adequate. In some cases, internal or external fixation of the flail segment may be done.

Mechanical ventilation

The preferred treatment for flail chest is intubation and mechanical ventilation. Positive-pressure ventilation provides support and stabilisation of the flail segment and improves ventilation and gas exchange. The work of breathing is decreased and healing improved.

People with pulmonary contusion often are critically ill, requiring intensive care management. Treatment is supportive, directed at maintaining adequate ventilation and alveolar gas exchange. Endotracheal intubation and mechanical ventilation are necessary in most cases. Repeated bronchoscopy may be done to remove secretions and cellular debris, preventing atelectasis. Although adequate hydration is necessary to prevent shock, overhydration can increase pulmonary oedema. Pulmonary arterial pressure monitoring with a Swan–Ganz catheter and frequent ABG measurement is sometimes used to assist in the assessment of optimal fluid replacement and management of ventilatory support. See Chapter 36 for nursing care of the person who is intubated and ventilated.

Unilateral pulmonary contusion may present a unique management problem. Mechanical ventilation with positive end-expiratory pressure (PEEP) to maintain open alveoli and adequate gas exchange can damage the unaffected lung. Intubation with a double-lumen endotracheal tube that permits independent ventilation of each lung may be used.



Nursing care

Health promotion

Encourage the use of seat belts, shoulder harnesses and supplemental restraint systems such as air bags to significantly reduce the incidence of thoracic injury associated with motor vehicle crashes. Discuss the importance of appropriate protective equipment and gear for people engaging in potentially hazardous activities such as contact sports or mountain climbing and occupations such as roofing or house painting.

Assessment

The nursing assessment of the person with a thoracic injury may need to be rapid and focused.

- *Health history:* pain, difficulty breathing; circumstances of the injury, including position in the motor vehicle, use of restraints, speed and type of impact; distance of a fall, surface and position on impact; history of chronic lung or heart disease; smoking history.
- *Physical examination:* airway, breathing, circulation; LOC; colour, vital signs; respiratory rate, depth, ease; symmetry of chest movement; lung sounds and percussion tone; presence of bruising, crepitus or paradoxical chest movement.

Nursing diagnoses and interventions

Chest wall trauma can interfere with adequate chest expansion and alveolar ventilation. When a pulmonary contusion is also present, gas exchange is affected as well. Priorities for nursing management include controlling pain, ensuring adequate ventilation and promoting gas exchange.

Acute pain

With many thoracic injuries, pain interferes with lung expansion and coughing, leading to such complications as pneumonia and atelectasis. Adequate pain management is a key component of medical and nursing management for these individuals.

- Frequently assess pain, using a standard pain scale and objective data. *Increased respiratory rate, shallow respirations, diminished breath sounds and reluctance to move and cough may indicate inadequate pain control in a thoracic injury.*
- Administer analgesics by patient-controlled analgesia or on a schedule to maintain pain control. *Analgesics are more effective when pain is not allowed to become intense.*

CONSIDERATION FOR PRACTICE

Assess for possible respiratory depression due to narcotic analgesia. Respiratory depression can further compromise ventilation in the person with thoracic injury.

- Notify the doctor if pain relief is inadequate or excess sedation and respiratory depression occur. An intercostal nerve block may be done to reduce the need for narcotic analgesia. *Assess for bleeding and adequate ventilation following a nerve block.*

Ineffective airway clearance

Aggressive respiratory hygiene may be necessary to maintain open airways and adequate ventilation.

- Assess lung sounds and respiratory rate, depth and effort frequently. Encourage to cough, deep breathe and change position every 1 to 2 hours, and to use the incentive spirometer. *Frequent assessment and measures to maintain airway patency are vital to prevent complications in the person with thoracic injury.*
- Teach how to splint the affected area with a blanket or pillow when coughing. *Splinting reduces movement and discomfort of the affected area.*
- Suction airway as indicated. Work with respiratory therapy to maintain optimal mechanical ventilation. Secure the endotracheal tube to maintain appropriate position and lung ventilation. *Endotracheal tube security is* particularly *important when a double-lumen endotracheal tube is in place because malposition can occlude one main bronchus and prevent ventilation of the affected lung.*
- Elevate the head of the bed. *Elevating the head of the bed facilitates lung expansion and reduces the work of breathing.*

Impaired gas exchange

Impaired gas exchange is of particular concern in pulmonary contusion. Alveolar damage and pulmonary oedema can significantly impair oxygenation of the blood and removal of carbon dioxide.

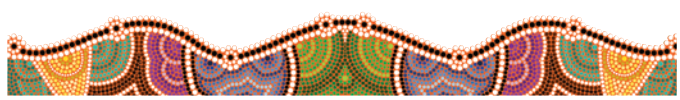
- Monitor vital signs, colour, oxygen saturation and ABGs. Assess for manifestations such as anxiety or apprehension, restlessness, confusion, lethargy or complaints of headache. *These assessment data alert the nurse and care providers to potential hypoxaemia or hypercapnia due to impaired gas exchange.*
- Maintain oxygen therapy and mechanical ventilation as ordered. Hyperoxygenate prior to suctioning. *Oxygen and mechanical ventilation support alveolar gas exchange. Hyperoxygenation prior to suctioning reduces the degree of hypoxaemia that occurs during suctioning.*
- Monitor intake and output, weigh daily and monitor central venous pressure and pulmonary artery pressure as ordered. Maintain any ordered fluid restriction. *Fluid volume status is monitored to reduce the effects of pulmonary oedema on lung tissues.*
- Maintain bed rest or activity restriction as ordered. Space activities to allow periods of uninterrupted rest. *Rest reduces the metabolic rate and oxygen consumption.*

Community-based care

Simple rib fracture and minor chest wall injuries often are managed on an outpatient basis. Include the following topics when teaching for home care:

- pain management and its importance in preventing respiratory complications
- importance of coughing and deep breathing; how to splint the rib cage during coughing
- reasons for not taping or wrapping the chest
- symptoms to report to the doctor: chills and fever, productive cough, purulent or bloody sputum, shortness of breath and increasing chest pain
- importance of avoiding respiratory irritants, such as cigarette smoke and occupational or environmental pollutants.

Significant pulmonary contusion can result in long-term respiratory insufficiency. Discuss activity modifications and occupational changes with the person and family as indicated. Refer to home care services if needed.



THE PERSON WITH INHALATION INJURY

The internal environment of the lungs normally is protected from noxious substances by respiratory defence mechanisms. If these defences are breached, inhaled agents, such as gases, fumes, toxins and water, can cause internal trauma to the lungs.

Pathophysiology and manifestations

Smoke inhalation

Pulmonary injury due to inhalation of hot air, toxic gases or particulate matter is the leading cause of death in burn injury (Lafferty, 2014). Smoke inhalation can significantly affect normal respiratory function through three different mechanisms:

1. thermal damage to the airways, leading to impaired ventilation
2. carbon monoxide or cyanide poisoning, resulting in tissue hypoxia
3. chemical damage to the lung from noxious gases, which can impair gas exchange.

Smoke inhalation is suspected whenever a burn occurs in a closed space; if there are burns to the face or upper torso or singed nasal hairs; if sputum contains ash-like material; and when manifestations such as dyspnoea, wheezing or rales develop.

The lower airways of the lungs typically are protected from thermal damage by cooling of the inhaled gases in the upper airway and laryngeal spasm. Upper airway obstruction due to tissue oedema and laryngeal spasm can occur quickly, however, resulting in **asphyxiation** or oxygen deprivation, without lung damage. Steam inhalation can cause thermal damage to tissues of the lower respiratory tract.

Inhalation of carbon monoxide or cyanide gas poses an immediate threat to life. Carbon monoxide is a colourless, odourless gas produced in a fire. It binds readily with haemoglobin. The affinity of carbon monoxide for haemoglobin is 200 to 250 times stronger than that of oxygen. Haemoglobin bound to carbon monoxide reduces the oxygen-carrying capacity of blood and oxygen delivery to cells of the body. Carbon monoxide poisoning is suspected if the burn occurred in a closed space, if there is evidence of inhalation injury or if dyspnoea develops.

The manifestations of carbon monoxide poisoning depend on the level of carboxyhaemoglobin saturation. When haemoglobin is 10–20% saturated with carbon monoxide, symptoms include headache, dizziness, dyspnoea and nausea. A characteristic cherry-red colour of the skin and mucous membranes may be seen. With increasing levels, confusion, visual disturbances, irritability, hallucinations, hypotension, seizures and coma develop. Permanent neurological deficit can occur in survivors of severe, acute carbon monoxide poisoning.

Many other toxic chemicals may be present in smoke, especially in a house fire or industrial plant fire. Hydrogen cyanide can be lethal when inhaled. Inhalation of toxic chemicals causes bronchospasm and oedema of the airways and alveoli. Acute respiratory distress syndrome may develop within 1 to 2 days. Sloughing of damaged mucosa leads to airway obstruction and atelectasis. Pneumonia is common following smoke inhalation.

Near drowning

Drowning is a leading cause of preventable accidental death in Australia. Between July 2013 and June 2014, 266 people died of drowning (Royal Life Saving Society, 2014). In 2012–2013, 536 people were admitted to hospital for a near drowning or submersion (AIHW, 2015a).

In a small percentage of drowning victims, laryngeal spasm causes asphyxiation, not the aspiration of water. This is known as ‘dry drowning’. However, in most cases, asphyxiation and hypoxaemia result from aspiration of fluid. Loss of consciousness can occur within 3 to 5 minutes after total immersion. Circulatory impairment, brain injury and brain death can occur within 5 to 10 minutes. Immersion in very cold water and the *dive reflex*—a protective mechanism that slows the heartbeat, constricts peripheral vessels and shunts blood to the brain and heart—may prolong survival.

When aspirated, the type of water will directly affect the outcome. Freshwater is hypotonic; when aspirated, it is rapidly absorbed from the alveoli, leading to hypervolaemia and haemodilution. Haemolysis occurs as blood cells are subjected to a hypotonic environment and serum electrolytes are diluted. Electrolyte imbalances can cause cardiac arrhythmias and death. Haemolysis can lead to acute tubular necrosis and acute kidney failure. Aspiration of freshwater impairs pulmonary surfactant and damages the alveolar–capillary membrane. Respiratory failure can result. Nearly the opposite effects occur with saltwater aspiration. As a hypertonic fluid, saltwater draws fluid into the alveoli, resulting in hypovolaemia. Haemolysis is insignificant and small elevations in serum sodium and chloride levels rarely cause life-threatening effects. With either type of near drowning episode, inhaled microorganisms and debris can lead to pneumonia. The pathophysiological changes associated with freshwater and saltwater near drowning are illustrated in Figure 35.20.

Manifestations of near drowning may include altered LOC, restlessness and apprehension. The person may complain of headache or chest pain. Other signs include vomiting, possible cyanosis, apnoea, tachypnoea and wheezing. If pulmonary oedema is present, pink froth may be visible in the mouth and nose. Other manifestations include tachycardia, arrhythmias, hypotension, shock and cardiac arrest. Hypothermia may be present.

INTERPROFESSIONAL CARE

With inhalation injuries, the most effective treatment is prevention. A working smoke detector (with functioning batteries) could prevent the majority of deaths from smoke inhalation occurring in the home. The statement, ‘A smoke detector was found, but the batteries had been removed’ is all too familiar in news reports of fire-related deaths.

The second most important line of defence against death or permanent injury from inhalation injuries is removing the victim from the area of the fire or water and administering effective CPR. In many cases, immediate restoration of effective breathing and circulation is key to preserving life. Hypoxaemia progresses rapidly until breathing is restored; reversal of tissue hypoxia depends on adequate circulation. In both smoke inhalation and near drowning, intubation may be necessary to establish an airway. Oxygen is administered as soon as possible. Attempts to drain water from the lungs of the near

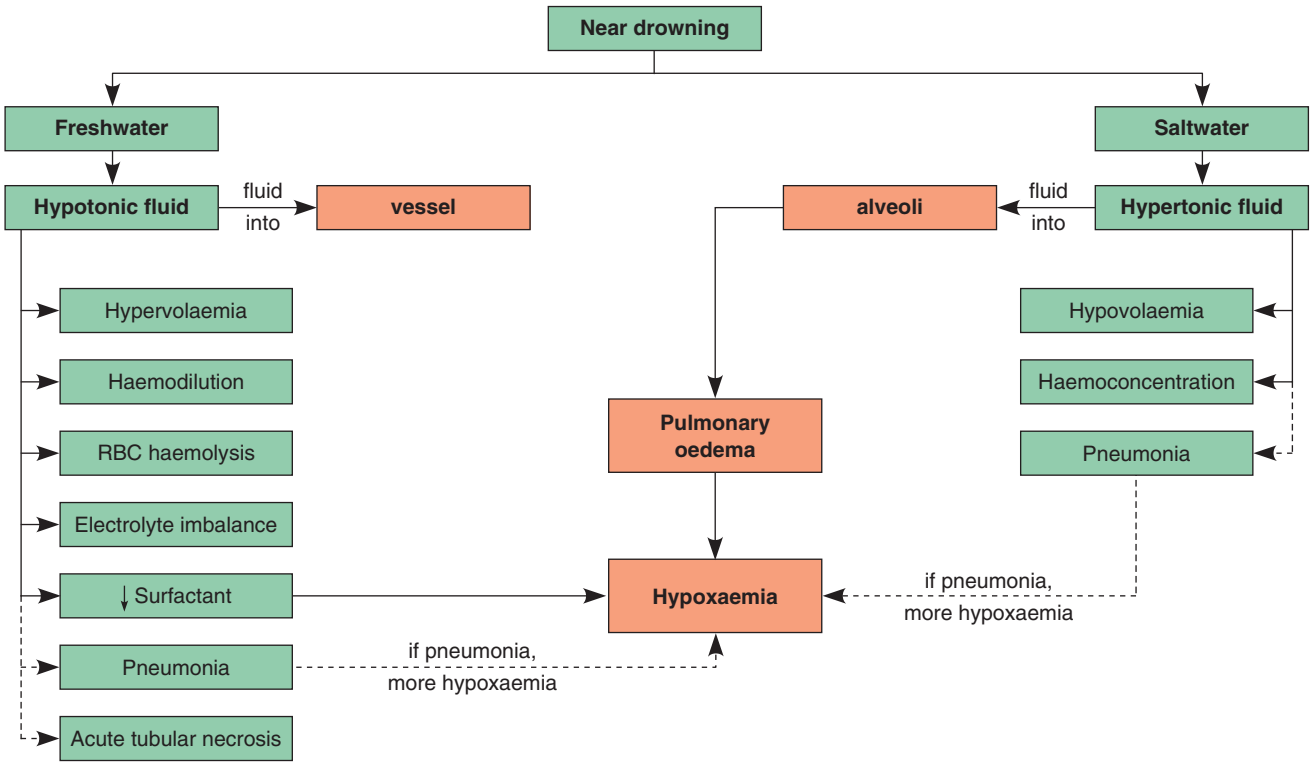


FIGURE 35.20 ■ The pathogenesis of near drowning: freshwater and saltwater

drowning victim waste time and are generally ineffective in restoring alveolar ventilation. External cardiac defibrillation may be necessary to re-establish an effective cardiac rhythm and circulation. When the victim is hypothermic, resuscitation measures are continued until the core body temperature reaches approximately 32°C. The basic rule in hypothermia is that the person is not declared dead until the body has been rewarmed and life signs remain absent.

To prevent drowning, life preservers and flotation vests or jackets should be worn on the body, not stored in the hold of the boat. These devices are designed to keep the head above water. Even accomplished swimmers should never enter the water alone in unguarded areas. Just as alcohol and driving do not mix, neither do alcohol and boating nor other water sports.

Diagnosis

When inhalation injury is known or suspected, the following diagnostic tests may be done:

- *ABGs* are drawn to evaluate gas exchange and the degree of hypoxaemia. Combined respiratory and metabolic acidosis may be apparent. With effective ventilation and supplemental oxygen, acidosis may reverse quickly. With carbon monoxide poisoning, arterial PO_2 may be normal, but oxyhaemoglobin saturation is less than normal.
- *Carboxyhaemoglobin levels* are drawn in suspected carbon monoxide poisoning. Normal levels are less than 5% in non-smokers and less than 10% in smokers. Higher levels indicate carbon monoxide poisoning. Levels less than 20% are considered mild poisoning; between 20% and 40% is moderate poisoning; and 40% to 60% is severe poisoning. Levels higher than 60% are generally fatal.
- *Serum electrolytes* and *osmolality levels* vary in near drowning, depending on the type of water aspirated. In freshwater drowning, serum electrolyte levels and osmolality may be significantly reduced. With saltwater drowning, serum sodium and chloride may be somewhat high, and osmolality is increased because of hypovolaemia.
- *Chest x-ray* is done, but may not show changes until 12 or more hours after the insult. Evidence of ARDS may be seen 24 to 48 hours after inhalation injury.
- *Bronchoscopy* may be ordered to inspect damaged lung tissue, particularly with smoke inhalation and possible thermal injury.

Treatments

Treatment of inhalation injury is generally supportive. Endotracheal intubation and mechanical ventilation often are required to maintain the airway and provide adequate alveolar ventilation and oxygenation. All people with inhalation injury require supplemental oxygen, even when intubation and ventilation are not required. *Hyperbaric oxygen therapy*, the delivery of 100% oxygen at increased atmospheric pressure, may be used to treat carbon monoxide poisoning. This treatment carries some risks, such as oxygen toxicity and potential trauma to lung tissues, sinuses and ears due to the increased pressures.

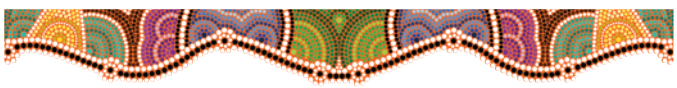
Other treatment measures may include bronchodilator therapy to manage bronchospasm. Bronchodilators can be administered by aerosol inhalation or intravenous infusion.

Coughing and suctioning are important to remove secretions and debris. Chest physiotherapy with percussion and postural drainage may be performed.

Intravenous fluids may be ordered; if significant haemolysis has occurred, packed red blood cells may be given to improve the oxygen-carrying capacity of the blood. Fluid therapy is monitored carefully, using pulmonary artery or central venous pressures to reduce the risk of pulmonary oedema.

With near drowning victims, measures such as inducing hypothermia or barbiturate-induced coma and administering corticosteroids and osmotic diuretics may be employed to help prevent neurological damage.

Careful monitoring for complications such as pneumonia and ARDS is vital throughout the course of treatment. Respiratory status, vital signs and other data are frequently assessed to identify complications and allow early intervention.



Nursing care

Health promotion

Prevention of inhalation injuries is an important nursing responsibility. Teach everyone the value of a working smoke detector, especially in the sleeping areas of the house. Encourage families to develop an escape plan in case of fire and to use fire drills to rehearse getting out of the house. Smouldering cigarettes are a leading cause of house fires; help people develop a plan to stop smoking. Teach people to drop and roll should clothing catch fire. (Flames rise, increasing the risk of respiratory injury when upright.)

Learning to swim safely is important to prevent drowning. Teach individuals never to swim alone, when fatigued or immediately following a meal. Remind people that knowing how to swim will not prevent drowning in very cold water or in large bodies of water, such as lakes, rivers or the ocean. Instruct to always wear flotation devices while boating, water skiing, surfing or windsurfing. Wetsuits help prevent hypothermia during activities in very cold water. Advise covering or fencing swimming pools and ponds to prevent inadvertent entry and drowning.

A population well trained in effective, safe CPR provides the best second line of defence against inhalation injury. Rapid restoration of breathing is essential to prevent hypoxia and brain damage. Encourage all people to be trained and regularly update CPR skills. Work with communities to increase the number of trained people. Refer individuals to Australian Red Cross, Surf Life Saving, St John's Ambulance or any other accredited CPR training facility.

Assessment

Inhalation injuries may be a medical emergency, necessitating focused and timely nursing assessment.

- *Health history:* circumstances of the injury, including duration of exposure to smoke or time under water, explosion or fire in a closed area, type and temperature of water immersed in; resuscitation measures used; allergies and current medical problems.
- *Physical examination:* airway, breathing, circulation; LOC; colour, oxygen saturation level; vital signs; heart and lung sounds; urine output; evidence of burns or soot around nares or mouth.
- *Diagnostic tests:* carboxyhaemoglobin levels, serum electrolytes and osmolality; ABGs; chest x-ray.

Nursing diagnoses and interventions

Nursing care priorities for the person with an inhalation injury are determined by the type of injury or tissue damage. Airway clearance is a major concern in all inhalation injuries, as is impaired gas exchange. Tissue hypoxia also can be a significant problem.

Ineffective airway clearance

Nursing measures to maintain an adequate airway begin with careful and frequent assessment of respiratory status, including rate, depth and effort, as well as breath sounds. Note amount, colour and consistency of sputum. Assist to cough frequently; suction the intubated person as needed to remove secretions. Elevate the head of the bed to facilitate alveolar ventilation unless otherwise ordered. Stabilise the endotracheal tube to prevent displacement into a mainstem bronchus, which could lead to ventilation of only one lung. Report changes in the character of secretions that may indicate complications: pink, frothy sputum suggesting pulmonary oedema or purulent sputum suggestive of pneumonia. Administer bronchodilators as ordered. Perform percussion and postural drainage as ordered.

Impaired gas exchange

Support gas exchange by administering supplemental oxygen, with or without mechanical ventilation, as required. Frequently assess oxygen saturation, skin colour and mental status. Decreasing level of consciousness may be an early sign of hypoxaemia. Monitor exhaled carbon dioxide, ABGs and pulmonary artery pressures as ordered and indicated. Report changes to the doctor. Maintain oxygen flow rates as ordered. Provide frequent mouth care to reduce the discomfort of dry mucous membranes and prevent tissue breakdown. Work with physiotherapists to maintain effective oxygen delivery with mechanical ventilation. Administer sedation as required. Maintain fluid restriction if ordered.

Ineffective cerebral tissue perfusion

Impaired cerebral tissue perfusion is a priority problem, especially with near drowning. Hypoxia and possible hypervolaemia can lead to cerebral oedema and increased intracranial pressure (IICP), further impairing blood flow. Monitor vital signs and neurological status frequently. A change in level of consciousness or behaviour is typically the earliest sign of IICP. Changes noted on an intracranial pressure monitor also provide early evidence of IICP. Increasing systolic blood pressure and pulse pressure and slowed heart rate are late signs. Other manifestations may include pupillary changes and decreasing muscle strength. Report changes promptly to the

doctor. Elevate the head of the bed and keep the head in neutral position to promote drainage from the cranial vault. Maintain effective ventilation and oxygenation; hypercapnia and hypoxaemia increase cerebral oedema. Administer sedation, osmotic diuretics or corticosteroids as ordered to reduce cerebral oedema. Maintain fluid restriction. Space activities and promote rest to reduce metabolic demands.

Community-based care

Teach individuals who do not require hospitalisation for inhalation injury about symptoms that may indicate a complication and should be reported to the doctor: increasing dyspnoea, cough productive of purulent or pink frothy mucus, confusion or other changes. Manifestations of respiratory damage may not be apparent for 24 to 48 hours following the injury.

Significant hypoxia due to near drowning or carbon monoxide poisoning may cause permanent neurological effects. Work with the family to develop communication techniques and identify remaining strengths. Help the family identify future care needs and means for meeting them, such as community-based care, personal care aides or long-term care facilities. Provide social services and support group referrals.

LUNG CANCER

THE PERSON WITH LUNG CANCER

Incidence and risk factors

Lung cancer is the second leading underlying cause of death for Australian males and the sixth for females. Death rates for lung cancer have risen gradually since the 1940s (see Figure 35.21).

In Australia, the estimated incidence of lung cancer in 2014 was well over 11000 people. It is the fourth most common cancer for both males and females; however, of all the cancers, lung cancer is the most common cause of death in both male and females. Between 1991 and 2009, lung cancer incidence fell by 26% in males but rose by 37% in females (AIHW, 2014).

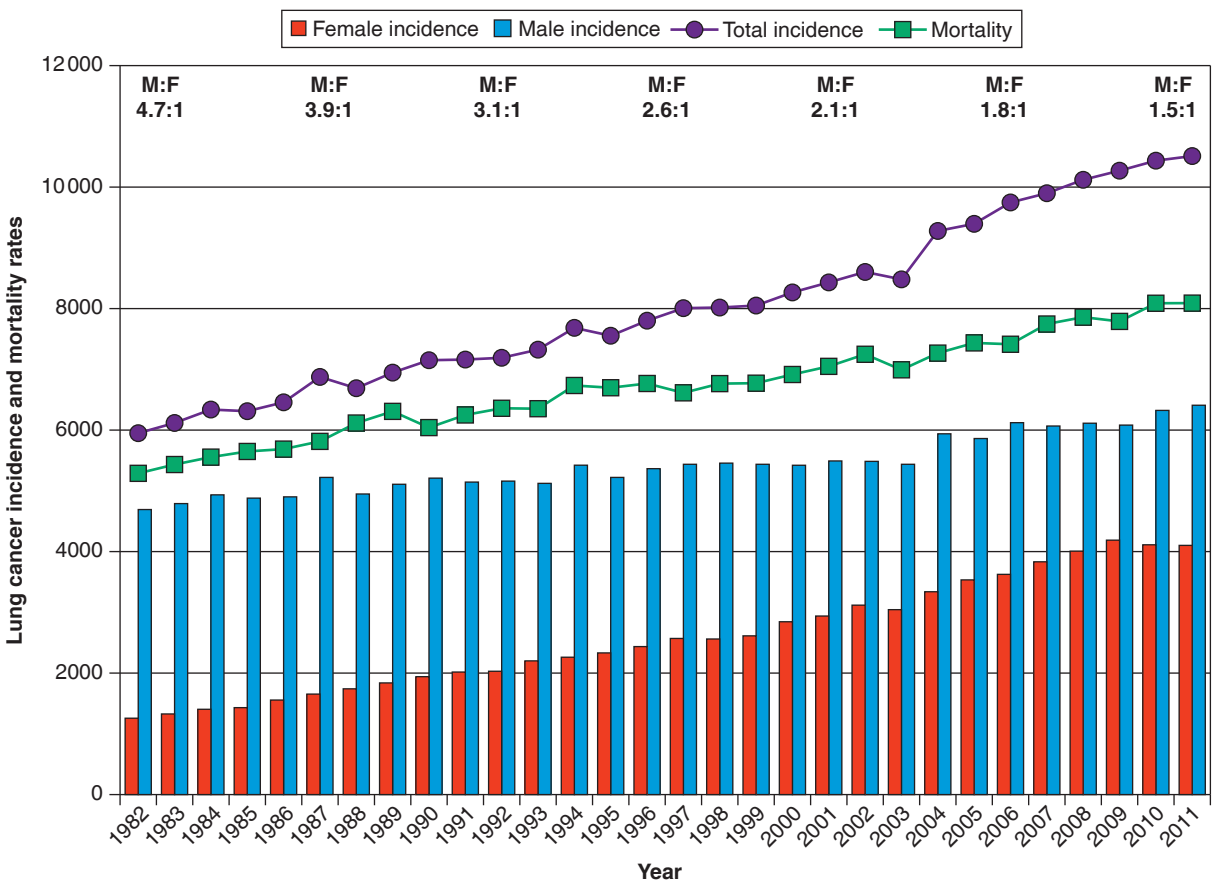


FIGURE 35.21 ■ Lung cancer incidence in Australia, 1982–2011; mortality rates, 1982–2011; and male:female incidence ratio for selected dates

Source: Generated from AIHW (2015c). *Australian cancer incidence and mortality books: Lung cancer*. Retrieved from www.aihw.gov.au/acim-books/.

Nearly 90% of lung cancers are caused by tobacco smoking, and smokers have 16 times more chance of developing lung cancer than a non-smoker (Cancer Council, 2015). Cigarette smoke, which contains over 40 known chemical carcinogens and cancer promoters, is clearly the most significant cause of lung cancer. Even former smokers who have abstained for a number of years have a higher risk of developing lung cancer than non-smokers.

Pathophysiology

Lung cancer develops as damaged bronchial epithelial cells mutate over time to become neoplastic. The genetic abnormality commonly seen is on chromosome 3, with loss of genetic material. Alterations of suppressor genes are also seen in some types of lung cancer.

The vast majority of primary lung lesions are bronchogenic carcinoma tumours of the airway epithelium. These tumours are further differentiated by cell type: small-cell carcinoma, adenocarcinoma, squamous cell carcinoma and large-cell carcinoma. For clinical purposes, the latter three cell types frequently are classified together as non-small-cell carcinomas. *Small-cell carcinomas*, which account for approximately 18% of lung cancers, grow rapidly and spread early. These tumours have paraneoplastic properties; that is, they produce manifestations at sites that are not directly affected by the tumour. Small-cell lung carcinomas can synthesise bioactive products and hormones such as adrenocorticotropic hormone (ACTH), antidiuretic hormone (ADH), a parathormone-like hormone and gastrin-releasing peptide. *Non-small-cell carcinoma* accounts for about 70% of lung cancers. Each cell type differs in its incidence, presentation and manner of spread.

Table 35.7 outlines the incidence and unique characteristics of each cell type.

Bronchogenic cancer, regardless of cell type, tends to be aggressive and locally invasive, and to have widespread metastatic lesions. Tumours begin as mucosal lesions that grow to form masses that obstruct the bronchi or invade adjacent lung tissue. All types frequently spread via the lymph system to nodes and other organs such as the brain, bones and liver.

Manifestations

The manifestations of lung cancer are related to the location and spread of the tumour. People may present with symptoms related to the primary tumour, manifestations of metastatic disease or with systemic symptoms. Initial symptoms often are attributed to smoking or chronic bronchitis. Chronic cough is common, as is haemoptysis. Wheezing and shortness of breath occur as a result of airway obstruction. Dull, aching chest pain occurs as the tumour spreads to the mediastinum; pleuritic pain occurs when the pleura is invaded. Hoarseness and/or dysphagia indicate pressure of the tumour on the trachea or oesophagus.

Systemic and paraneoplastic manifestations of lung cancer include weight loss, anorexia, fatigue and weakness; bone pain, tenderness and swelling; clubbing of the fingers and toes; and various endocrine, neuromuscular, cardiovascular and haematological symptoms.

Confusion, impaired gait and balance, headache and personality changes may indicate brain metastasis. Bone metastases cause bone pain, pathological fractures and possible spinal cord compression, as well as thrombocytopenia and anaemia if bone marrow is invaded. When the liver is affected, symptoms of liver dysfunction and biliary obstruction—including jaundice, anorexia and upper right quadrant pain—are evident.

See 'Multisystem effects of lung cancer' on page 1299.

Complications and course

Superior vena cava syndrome, partial or complete obstruction of the superior vena cava, is a potential complication of lung cancer, particularly when the tumour involves the superior mediastinum or the mediastinal lymph nodes. Obstructed venous flow from the head and neck produces the symptoms of superior vena cava syndrome (oedema of the neck and face, headache, dizziness, vision disturbances and syncope) and may develop acutely or more gradually. Veins of the upper chest and neck are dilated; flushing occurs, followed by cyanosis. Cerebral oedema may affect the level of consciousness; laryngeal oedema may impair respirations.

Paraneoplastic syndromes commonly associated with lung cancer include syndrome of inappropriate ADH secretion (SIADH) with fluid retention, hyponatraemia and oedema, Cushing's syndrome (see Chapter 18) related to abnormal ACTH production, and hypercalcaemia. Lung tumours may also produce procoagulation factors, increasing the risk of venous thrombosis, pulmonary embolism and thrombotic endocarditis (Bullock & Hales, 2012). In lung cancer, neuromuscular symptoms such as muscle weakness and wasting of the limbs may be the first indication of the disease.

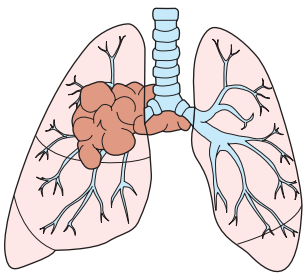
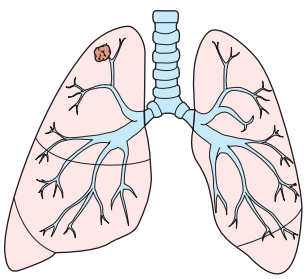
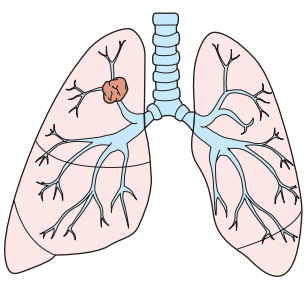
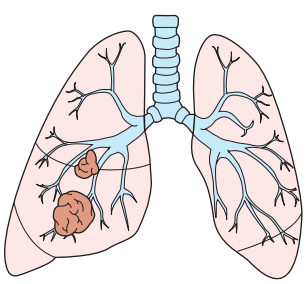
At the time of diagnosis, cancer of the lung is typically well advanced, with distant metastasis present in 55% of people and regional lymph node involvement in another 25%. The prognosis is generally poor: the overall 5-year survival rate is only 13% for males and 17% for females (Cancer Council, 2015).

INTERPROFESSIONAL CARE

Because lung cancer typically is advanced when diagnosed and the prognosis generally is poor, prevention of the disease must be a primary goal for all healthcare providers. Reducing cigarette smoking can have a significant impact on the death rate from lung cancer—a far greater impact than advances in treatment.

Establishing an accurate diagnosis is the first step in treating lung cancer. Treatment decisions are based on the tumour location, type of cancer cell, staging of the tumour and the person's ability to tolerate treatment. Lung cancer is staged by the tumour size, location, degree of invasion of the primary tumour and the presence of metastatic disease. Lung cancer staging is summarised in Table 35.8. Surgery is the treatment of choice for most forms of lung cancer.

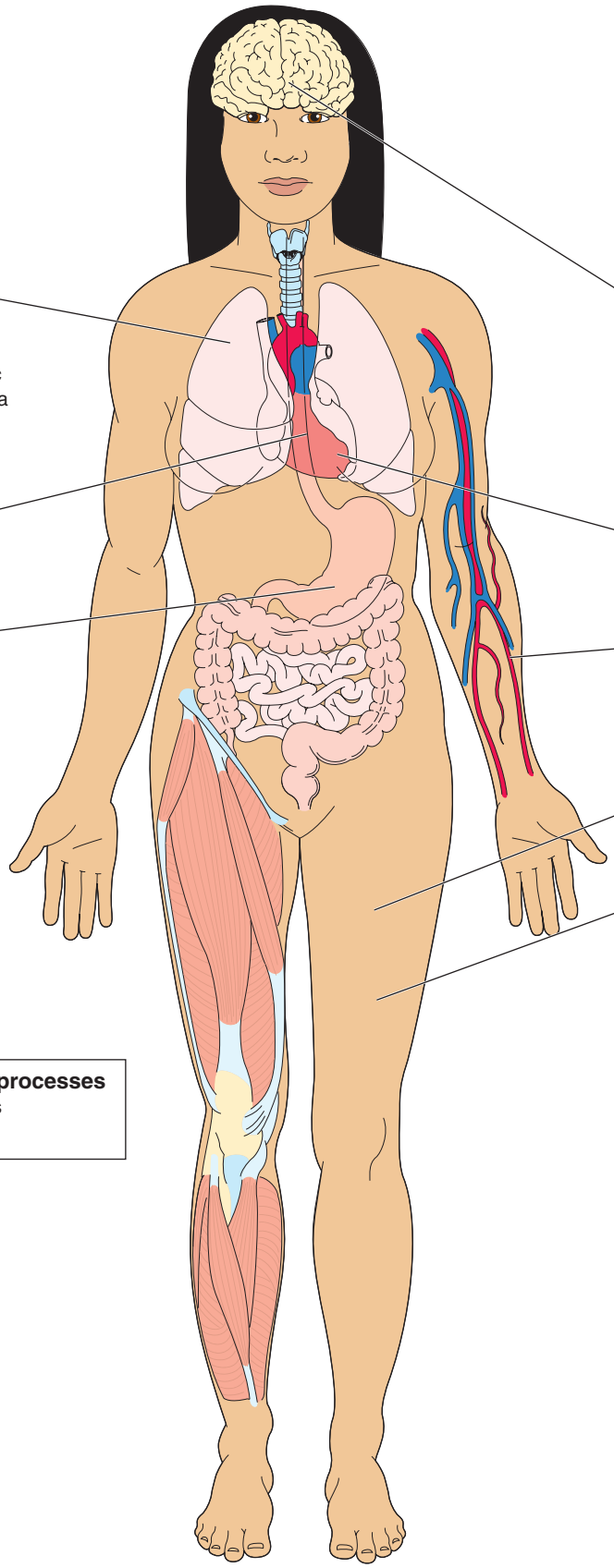
TABLE 35.7 Comparison of lung cancer cell types

	CELL TYPE AND PREVALENCE	PRESENTATION AND ASSOCIATED MANIFESTATIONS	SPREAD
	Small-cell (oat-cell) carcinoma: 18% of all lung cancers	Central lesion with hilar mass common, early mediastinal involvement, no cavitation; SIADH, Cushing's syndrome, thrombophlebitis	Aggressive tumour; more than 40% of people have distant metastasis at time of presentation
	Adenocarcinoma: 32% of all lung cancers	Peripheral mass involving bronchi; few local symptoms; hypertrophic pulmonary osteoarthropathy	Early metastasis to CNS, skeleton and adrenal glands
	Squamous cell carcinoma: 29% of all lung cancers	Central lesion located in large bronchi; person presents with cough, dyspnoea, atelectasis and wheezing; hypocalcaemia common	Spreads by local invasion
	Large-cell carcinoma: 9% of all lung cancers	Usually, peripheral lesion that is larger than that associated with adenocarcinoma and tends to cavitate; gynaecomastia, thrombophlebitis	Early metastasis

Diagnosis

- *Chest x-ray* usually provides the first evidence of lung cancer. It is particularly reliable as a diagnostic tool when compared with a previous chest x-ray. In high-risk populations, the chest x-ray may be used as a screening tool for lung cancer.
- *Sputum specimen* is sent for *cytological examination* to establish the diagnosis of lung cancer. The sputum sample is collected on arising in the morning. If malignant cells are found in the sputum, more expensive and invasive examinations may be unnecessary. However, a sputum sample negative for malignant cells does not rule out lung cancer; it may simply indicate that the tumour is not shedding cells into mucus secretions.
- *Bronchoscopy* is frequently done to visualise and obtain tissue for biopsy from the tumour. When a tumour mass or suspicious tissue is identified visually, a cable-activated instrument is used to obtain a biopsy specimen. If the tumour cannot be seen, the airways may be flushed with a saline solution (bronchial washing) to obtain cells for cytological examination. Nursing care of the person undergoing a bronchoscopy is included in the 'Diagnostic tests' box in Chapter 33.
- *CT scan* is used to evaluate and localise tumours, particularly tumours in the lung parenchyma and pleura. It also is completed prior to needle biopsy to localise the tumour. CT scanning can also detect distant tumour metastasis and evaluate tumour response to treatment.

MULTISYSTEM EFFECTS OF LUNG CANCER



Respiratory
- Cough
- Haemoptysis
- Wheezing and dyspnoea
- Chest pain, dull or pleuritic
- Hoarseness and dysphagia
- Pleural effusion

Cardiovascular
- Compression of the superior vena cava

Gastrointestinal
- Anorexia

Metabolic processes
- Weight loss
- Fever

Paraneoplastic syndromes

Endocrine system
- Hypercalcaemia
- Hyperphosphataemia
- Cushing's syndrome
- Syndrome of inappropriate antidiuretic hormone (SIADH) with water retention and hyponatraemia

Cardiovascular system
- Thrombophlebitis
- Endocarditis

Haematological effects
- Anaemia
- Disseminated intravascular coagulation (DIC)
- Eosinophilia

Connective tissue
- Osteoarthropathy with clubbing and periosteal inflammation

Neuromuscular effects
- Peripheral neuropathy
- Cerebellar degeneration
- Myasthenia-like muscle weakness

TABLE 35.8 Lung cancer staging

	PRIMARY TUMOUR (T STAGE)	REGIONAL LYMPH NODES (N)	DISTANT METASTASIS (M)
	T_0—No evidence of primary tumour		
Stage 0	T_X—Malignant cells in bronchopulmonary secretions, but no tumour visualised		M_X—Presence of distant metastasis cannot be assessed
Stage I	T_1S—Carcinoma in situ	N_0—No regional lymph node metastasis	M_0—No distant metastasis
	T_1—Tumour that is 3 cm in diameter or less, with no evidence of invasion		
Stage II	T_2—Tumour that is greater than 3 cm in diameter or invades visceral pleura or has associated atelectasis or pneumonitis	N_1—Metastasis or direct extension to peribronchial or ipsilateral hilar nodes	
Stage III	T_3—Tumour with direct extension into an adjacent structure or any tumour with associated pleural effusion or atelectasis or pneumonitis of entire lung	N_2—Metastasis to ipsilateral mediastinal or subcarinal nodes	
Stage IV	T_4—Tumour that invades mediastinum or involves the heart, great vessels, trachea, oesophagus, vertebral body or carina; presence of malignant pleural effusion	N_3—Metastasis to contralateral mediastinal, scalene or supraclavicular nodes	M_1—Distant metastasis present

- *Cells or tissue for cytological examination and biopsy* may be obtained by aspirating fluid from a pleural effusion, percutaneous needle biopsy and lymph node biopsy. These procedures may be done in an outpatient or a surgical setting.
- *FBC, liver function studies* and *serum electrolytes* including calcium are obtained to evaluate for evidence of metastatic disease or paraneoplastic syndromes.
- *Tuberculin test (PPD)* is performed to rule out tuberculosis as the cause of symptoms and abnormalities seen on chest x-ray.
- *Respiratory function tests (RFTs)* and *ABGs* may be performed prior to the initiation of treatment if the person has manifestations of respiratory insufficiency (e.g. dyspnoea, activity intolerance, low oxygen saturation levels).

See Chapter 33 for nursing care related to commonly used diagnostic tests for lung cancer.

Medications

Combination chemotherapy (often combined with radiation therapy and/or surgery) is the treatment of choice for small-cell lung cancer because of its rapid growth, dissemination and sensitivity to cytotoxic drugs. Used in combination, chemotherapeutic drugs allow tumour cells to be attacked at different parts of the cell cycle and in different ways, increasing the effectiveness of therapy. Fifty per cent of individuals with tumours at early stages achieve complete tumour remission with combination chemotherapy. When a complete tumour response is achieved in the first few cycles of chemotherapy, the chances for long-term survival are much greater.

Combination chemotherapy is used also as an adjunct to surgery or radiation therapy for other types of lung cancer. It may be used to reduce the size of advanced local tumours prior to surgery and to lengthen survival when distant metastases are present. See Chapter 13 for further discussion of chemotherapy.

Bronchodilators may be prescribed to reduce airway obstruction. Analgesics and pain management strategies are vital when the cancer is advanced. See Chapter 8 for more information about postoperative and cancer pain management.

Surgery

Surgery offers the only real chance for a cure in non-small-cell lung cancer. Unfortunately, most tumours are inoperable or only partially resectable at the time of diagnosis. The type of surgery performed depends on the location and size of the tumour, as well as the person's pulmonary and general health. The goal of surgery is to remove all involved tissue while preserving as much functional lung as possible. Table 35.9 outlines various surgical procedures used to treat lung cancer. Nursing care for the person having lung surgery is outlined in the box below.

Radiation therapy

Radiation therapy is used alone or in combination with surgery or chemotherapy for lung cancer (Tan, 2015). The treatment goal may be either cure or symptom relief (palliative). Prior to surgery, radiation therapy is used to 'debulk' tumours. When cancer has spread by direct extension to other thoracic structures and surgery is not feasible, radiation therapy may be the treatment of choice. It also may be used to relieve manifestations such as cough, haemoptysis, pain due to bone metastasis and dyspnoea from bronchial obstruction. Complications of lung cancer, such as superior vena cava syndrome, may be treated with radiation.

Radiation therapy may be delivered by external beam to the primary tumour site or by intraluminal radiation or

TABLE 35.9 Types of surgery for lung cancer

PROCEDURE	DESCRIPTION	USED FOR
Laser bronchoscopy	Bronchoscopy-guided laser used to resect tumour	Tumours localised in a main bronchus
Mediastinoscopy	Visualisation of the mediastinum using an endoscope passed through a suprasternal incision	Evaluation and biopsy of a mediastinal tumour and lymph nodes
Thoracotomy	Incision into the chest wall	Access the lung and thoracic cavity for surgery
Wedge resection	Removal of a small section (wedge) of peripheral lung tissue	Small, peripheral lung tumours
Segmental resection	Removal of an individual bronchovascular segment of a lobe	Peripheral lung tumour with no evidence of extension to the chest wall or metastasis
Sleeve resection (bronchoplastic reconstruction)	Resection of a section of a major bronchus with reconstruction of remaining normal bronchus	Small lesion of a major bronchus
Lobectomy	Removal of a single lung lobe	Tumours confined to a single lobe
Pneumonectomy	Removal of an entire lung	Tumour widespread throughout the lung, involving the main bronchus or fixed to the hilum

brachytherapy. Radiation therapy and related nursing care are discussed further in Chapter 13. Specific nursing measures for the person undergoing radiation therapy for lung cancer are outlined in the 'Nursing care' box below.

Nursing care

Health promotion

Teach people of all ages, particularly children and teenagers, about the link between cigarette smoking and lung cancer. Not smoking, and avoiding exposure to second-hand smoke, is the primary preventive measure for lung cancer. In addition, explain the risk of lung cancer to people with occupational risk factors—exposure to asbestos products, in particular.

Assessment

Nursing assessment related to lung cancer focuses on identifying risk factors for the disease, early manifestations of lung cancer and respiratory function in the person undergoing treatment.

- *Health history:* current symptoms, including chronic cough, shortness of breath, blood-tinged sputum; systemic manifestations such as recent weight loss, fatigue, anorexia, bone pain; smoking history; occupational exposure to carcinogens; chronic diseases such as COPD.
- *Physical examination:* general appearance; skin colour, evidence of clubbing; weight and height; vital signs; respiratory rate, depth, excursion; lung sounds to percussion and auscultation.
- *Diagnostic tests:* FBC and coagulation studies, serum electrolytes and osmolality, liver and renal function studies; chest x-ray and CT scan results; ABGs and oxygen saturation levels.

NURSING CARE OF THE PERSON having lung surgery

PREOPERATIVE CARE

- Provide routine preoperative nursing care as outlined in Chapter 3.
- Note any history of smoking, respiratory and cardiac diseases, and other chronic conditions in the nursing history. *These factors may affect the response to surgery and the risk of postoperative complications.*
- Provide emotional and psychological support for the person and family. *In addition to facing surgery, the individual may be adjusting to a new diagnosis of cancer and the possibility that surgical intervention will be only partially successful.*
- Instruct about postoperative procedures, including respiratory therapy, breathing exercises and coughing techniques. *Allow practice time. Learning will be easier in the preoperative period, when pain and analgesia are not affecting mental function.*
- If the person will return from surgery with an endotracheal tube and mechanical ventilation, establish a means of communication using hand or eye signals or a magic slate. *Establishing a means of communication prior to surgery reduces postoperative anxiety at being unable to speak.*
- If the person will return to the critical care unit, introduce the person and family to the unit and any machines, such as ventilators and monitors, that will be used. *The knowledge that this is an expected part of surgical recovery reduces the person's and family's postoperative anxiety.*

(continued)

NURSING CARE OF THE PERSON having lung surgery (continued)

POSTOPERATIVE CARE

- Assess and provide routine postoperative care as outlined in Chapter 3.
- Assess for adequate pain control and provide analgesics as needed. *Incisional pain commonly causes altered breathing patterns in the person who has undergone lung surgery.*
- Frequently assess respiratory status, including colour, oxygen saturation, respiratory rate and depth, chest expansion, lung sounds, percussion tone and ABGs. *Maintaining adequate ventilation and gas exchange postoperatively is vital to reduce mortality and morbidity. Gas exchange may be impaired by complications of lung surgery, including pneumothorax, atelectasis, bronchospasm, pulmonary embolus, bronchopleural fistula and ARDS.*
- Assist with effective coughing techniques, postural drainage and incentive spirometry. Perform endotracheal suctioning as needed while intubated. *Surgical manipulation and anaesthesia can increase mucus production, leading to airway obstruction. Aggressive respiratory hygiene is important to prevent this complication.*
- Monitor and maintain effective mechanical ventilation. *This is vital to ensure adequate ventilation and gas exchange in the early postoperative period.*
- Maintain patent chest tubes and a closed drainage system. Monitor chest tube output every hour initially and then every 2 to 4 or 8 hours as indicated. Notify the doctor if chest tube output exceeds 70 mL per hour and/or is bright red, warm and free flowing. *Maintaining a patent, intact chest drainage system is vital for re-establishing negative pressure within the chest cavity and re-expansion of the lungs. Increased amounts of warm, free-flowing blood indicate intrathoracic haemorrhage that may necessitate surgical intervention.*
- Assess for signs of infection involving the incision or chest tube site(s). Use strict aseptic technique in caring for incisions and invasive monitoring devices. *The postoperative period poses risks for individuals from incisional infections, empyema in the chest cavity and pneumonia.*
- Assist with turning and encourage the person to ambulate as soon as possible. *Early mobility is important to prevent possible complications, such as pneumonia or pulmonary embolus.*
- Assess and maintain nutritional status. Initiate enteral or parenteral nutrition early if intubation and mechanical ventilation will be required for an extended period. Provide frequent small feedings once extubated. *Maintaining nutritional status promotes wound healing and prevents negative nitrogen balance. Giving frequent small feedings reduces the fatigue associated with eating.*

NURSING CARE OF THE PERSON receiving radiation therapy

Although radiation therapy is well controlled and specifically directed towards the tumour cells, some normal cells are also damaged in the process of treatment. Nursing care and teaching help the person cope with uncomfortable side effects associated with radiation therapy.

NURSING RESPONSIBILITIES

- Monitor for potential complications:
  a. radiation pneumonitis—dyspnoea on exertion, dry cough, fever
  b. pericarditis—chest pain, pericardial friction rub; muffled heart sounds, paradoxical pulse, ECG abnormalities (notify the doctor if symptoms develop)
  c. oesophagitis—pain, sore throat, difficulty swallowing.
- Encourage adequate fluid intake to liquefy respiratory secretions.
- Provide local analgesics and local anaesthetics such as viscous lignocaine as ordered to relieve dysphagia and sore throat.
- Offer small frequent meals of soft, cool foods and liquids to maintain nutritional status.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- If dyspnoea or pneumonitis develops, teach positioning, pursed-lip techniques and relaxation exercises to facilitate breathing.
- Reassure that pneumonitis is generally a self-limiting process and should resolve when the course of radiotherapy is completed.
- Teach the manifestations of pericarditis, which may develop during treatment or up to 1 year after its completion. Chest pain or pressure, rapid heartbeat and fever may signal pericarditis; increasing fatigue, dyspnoea and light headedness can indicate a chronic process with pericardial effusion and possible cardiac tamponade.
- Instruct to eliminate hot, spicy or acidic foods from the diet if oesophagitis is a problem. Alcohol and tobacco should also be avoided.
- Adequate rest and nutrition are important to alleviate the symptoms of radiation fatigue, which is common in people receiving radiation therapy for lung cancer. The fatigue is generally temporary.

Nursing diagnoses and interventions

The person with lung cancer is facing invasive treatments with undesirable side effects, possibly surgery and typically a poor prognosis for long-term survival. Nursing care needs are diverse, related to respiratory status, the cancer itself and possible metastases, and the treatment plan. Priority nursing diagnoses related to respiratory function include *Ineffective breathing pattern* and *Activity intolerance*. *Pain* and *Anticipatory grieving* also are likely to be high-priority problems. See the accompanying nursing care plan.

Ineffective breathing pattern

Breathing pattern and ventilation may be affected by the tumour itself or by treatment of the tumour. Thoracic surgery increases the risk due to the incision and disruption of the muscles of respiration. Maintaining effective lung ventilation is particularly important postoperatively to re-expand remaining lung tissue and prevent surgical complications.

- Assess and document respiratory rate, depth and lung sounds at least every 4 hours; evaluate more frequently in the immediate postoperative period or as indicated by condition. *Early detection of signs of respiratory compromise or adventitious lung sounds is vital for effective intervention.*

CONSIDERATION FOR PRACTICE

Monitor oxygen saturation and/or blood gas results, reporting changes from normal. Changes in levels of blood oxygen may be early indications of respiratory compromise.

- Frequently assess and document pain level (using a standard pain scale); provide analgesics as needed. *Pain and attempting to avoid chest movement to prevent additional pain can lead to rapid, shallow respirations and ineffective ventilation.*
- Elevate the head of the bed to 60 degrees. *Elevating the head of the bed reduces pressure on the diaphragm from the abdominal contents and permits optimal lung expansion.*
- Assist to turn, cough and deep breathe, and to use incentive spirometry. Help splint the chest with a pillow or blanket when coughing. *These measures promote airway clearance.*
- Suction airway as needed. *Suctioning may be required to remove secretions that the person is unable to cough up and expectorate.*

CONSIDERATION FOR PRACTICE

Maintain chest tube integrity and patency by ensuring uninterrupted gravity flow. Chest tubes help re-establish negative pressure in the thoracic cavity, allowing the lung to fully re-expand.

- Provide chest physiotherapy with percussion and postural drainage as needed or ordered. *Percussion and postural drainage help maintain airway patency and effective respirations.*
- If mechanical ventilation is instituted, work with respiratory therapy and use analgesia or sedation as needed to synchronise respirations with the ventilator. *Coordination of the person's respiratory effort with ventilator-delivered breaths is important for fully effective mechanical ventilation.*
- Provide reassurance and emotional support. *These measures help relieve anxiety and promote an effective breathing pattern.*

Activity intolerance

Both resectional lung surgery and inoperable lung cancer reduce the amount of functional lung tissue and surface area for gas diffusion. This can lead to activity intolerance if the oxygen supply is insufficient to meet the body's oxygen demand.

CONSIDERATION FOR PRACTICE

Assess and document physiological responses to activity, including pulse, respiratory rate, dyspnoea and fatigue. These assessments are good indicators of activity tolerance.

- Plan rest periods between activities and procedures. *Rest periods reduce oxygen demands and fatigue.*
- Assist the person to increase activities gradually in the postoperative period. *Increasing activity levels gradually improves exercise tolerance.*
- Teach measures to conserve energy while performing ADLs, such as sitting while showering and dressing, and wearing slip-on shoes. *These energy-conserving measures reduce oxygen demand and allow the person to remain independent as long as possible.*
- Keep frequently used objects within easy reach. *This helps conserve energy.*
- Administer oxygen as prescribed. Teach the person and family about home oxygen use if appropriate. *Supplemental oxygen can help improve activity and exercise tolerance.*
- Encourage maintenance of physical activity to tolerance. *Maintaining activity levels to the degree possible improves physical and emotional wellbeing.*
- Allow family members to provide assistance as needed. *This helps the person conserve energy and allows the family to retain a sense of usefulness.*

Pain

Pain is a priority problem in both the postoperative period and the terminal stages of cancer. Poorly managed pain prolongs recovery from surgery. In the person with terminal cancer,

chronic and acute pain must be managed effectively to allow a peaceful death.

- Assess and document pain using a standardised pain scale and objective data. Pain is a subjective experience, best evaluated by the person. *Changes in vital signs, guarded movement or unwillingness to move may indicate unreported pain.*
- Provide analgesics as needed to maintain comfort. *Postoperative recovery and restoration of function is facilitated by adequate pain management.*
- For cancer pain, maintain an around-the-clock medication schedule using narcotic, non-steroidal anti-inflammatory drugs and other medications as ordered. *Addiction is not a concern in terminal cancer; providing adequate pain relief that does not allow 'breakthrough' pain is important.*
- Provide or assist with comfort measures, such as massage, positioning, distraction and relaxation techniques. *These techniques promote relaxation and enhance pain relief.*
- Assist the person and family to plan and engage in activities that distract from pain, such as reading, watching television and engaging in social interactions. *Distraction helps the individual focus away from the pain.*
- Spend as much time with the person as possible; allow family members to remain with the person. *Physical presence of the nurse and family provides emotional support for the individual.*

NURSING CARE PLAN A person with lung cancer

After coughing up bloody sputum one morning, James Mueller, a 68-year-old retired mill worker, sees his doctor. A chest x-ray shows a suspicious density in the central portion of his right lung. Mr Mueller is admitted to the hospital the following Monday for diagnostic tests.

ASSESSMENT

Mr Mueller is admitted to the oncology unit and the nurse obtains a nursing history. Mr Mueller is married and has three grown children. He worked in a local paper mill for 35 years before retiring at age 62. He describes himself as 'pretty healthy', except for a chronic smoker's cough. He started smoking as a young man in the army. He has a 50-pack-a-year smoking history, having smoked a packet a day for 50 years since age 18. Mr Mueller says he briefly quit smoking following a small heart attack 3 years ago, but started again after 4 months. On further questioning, Mr Mueller says his cough has been productive for the past few months, especially in the morning, and that he is more short of breath than usual with activity.

Mr Mueller's examination data include BP 162/86, P 78 and regular, R 20 and T 36.9°C. Colour good, skin warm and dry. Inspiratory and expiratory wheezes noted in right chest but good breath sounds throughout. No other abnormal findings are noted on examination. The doctor orders early-morning sputum specimens for 3 days for cytological examination and schedules a CT scan of the chest the morning after admission.

Mr Mueller's FBC shows mild anaemia, but the remaining routine laboratory tests are essentially normal. Sputum cytology is positive for small-cell bronchogenic cancer. The CT scan shows a central mass approximately 4 cm in diameter with involved mediastinal and subclavicular lymph nodes. A small mass is also noted on the lumbar spine. After conferring with his doctor and an oncologist, Mr Mueller decides to undergo a trial course of chemotherapy.

DIAGNOSES

- *Impaired gas exchange* related to tumour mass and effects of chronic cigarette smoking manifested by increased shortness of breath.
- *Risk of inadequate nutrition* related to effects of chemotherapy manifested by weight loss and/or anorexia.
- *Risk of increased stress and anxiety* related to new diagnosis of lung cancer manifested by behavioural changes, sleep disturbances and/or changes in eating habits.
- *Knowledge deficit* about lung cancer and aids to smoking cessation manifested by continued smoking habits despite significant health challenges.

PLANNING

- Discuss symptoms to report to the doctor: increased dyspnoea or haemoptysis, severe stridor or wheezing, chest pain.
- Discuss measures to relieve nausea associated with chemotherapy, including premedication with a prescribed anti-emetic.
- Discuss possible effects of lung cancer with Mr and Mrs Mueller.
- Encourage Mr and Mrs Mueller to call a family conference to discuss the disease with their children and grandchildren.
- Refer to local cancer support group.
- Refer to community services for follow up and further teaching.
- Ask the doctor for a prescription for nicotine patches or gum for Mr Mueller.

Expected outcomes

- Maintain a patent airway.
- Maintain current weight.
- Express feelings and concerns about the effect of cancer on the family unit.
- Participate in care.
- Contact appropriate support groups.
- Verbalise an understanding of the disease, its treatment and prognosis.
- Develop a plan to stop smoking.

IMPLEMENTATION

- Evaluate family members' knowledge and understanding of lung cancer, correcting misinformation and teaching as needed.
- Teach coughing, deep breathing and hydration measures to facilitate airway clearance.
- Have a dietitian consult with Mr and Mrs Mueller to develop a diet plan for maintaining ideal weight.

NURSING CARE PLAN A person with lung cancer (continued)

- Have an Australian Lung Foundation volunteer contact the family.
- Work with Mr Mueller to develop a plan to stop smoking.

EVALUATION

Mr Mueller had his first chemotherapy treatment in the hospital and was discharged 4 days after admission. After 3 months of chemotherapy, his tumour shows little regression and a liver scan reveals further metastasis. He and his wife decide to stop chemotherapy, a decision with which the children reluctantly agree. Mr and Mrs Mueller are referred to hospice services. With the help of hospice nurses and volunteers, Mr Mueller is able to remain at home. His pain is managed initially with oral MS Contin, a sustained-release form of morphine sulfate, and later with an intravenous morphine infusion. Mr Mueller dies at home with his family at his side 9 months after his diagnosis of lung cancer.

CRITICAL THINKING IN THE NURSING PROCESS

1. The oncologist prescribed a chemotherapy regimen of cyclophosphamide, doxorubicin and vincristine. Describe how each of these drugs works against cancer cells and discuss the rationale for using this combination.
2. Develop a care plan to deal with the specific side effects for the above treatment regimen.
3. Mr Mueller had small-cell (oat-cell) cancer. How would his presentation and treatment differ if the diagnosis had been non-small-cell adenocarcinoma, stage $T_2N_2M_0$?

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Given that Mr Mueller has continued to smoke even after experiencing a myocardial infarction, which communication and education strategies could you use to assist him with success in quitting smoking now he has developed lung cancer?

Anticipatory grieving

Because lung cancer is often advanced when diagnosed, the person faces the very real prospect of dying from the disease. Grieving for the anticipated loss of life is a normal response as the person and family begin to adapt to the diagnosis. Nursing care goals are to promote expression of feelings and thoughts about the loss and to help the person and family initiate grief work, make decisions and use appropriate resources and coping mechanisms to deal with the loss.

- Spend time with the person and family. *Time is necessary to develop a trusting, therapeutic relationship.*
- Answer questions honestly; do not deny the probable outcome of the disease. *Honesty reinforces reality and provides a sense of control over decisions to be made.*

TRANSLATION TO PRACTICE How can communication practices in a multidisciplinary lung cancer care team influence a person's care?

Qualitative research by Rowlands and Callen (2012) was part of a bigger mixed-methods study exploring the methods and efficacy of communication within and between the multidisciplinary, hospital-based, lung cancer care team. Twenty-two members were interviewed using semi-structured in-depth interviews. A thematic grounded theory approach was used to determine that there were two key themes emerging. First, that the characteristics of communication were influenced by roles within the team, where doctors dominated; and second, that current mediums for communication and traditional influences on role delineation impacted on communication. It was noted that face-to-face verbal communication was preferred and that existing cancer care guidelines did not address team communication barriers.

IMPLICATIONS FOR NURSING

Specific attention must be placed on introducing and developing communication skills between nurses and other members of the healthcare team to benefit the management not only of individuals with lung cancer but also of all people in need of quality healthcare. It is critical for healthcare professionals to work effectively together. When staff work within professional 'silos', the concept of a functioning team fails to exist. Also, when face-to-face communication is preferred, there is a risk that failure to document observations or changes to care can compromise the integrity and completeness of the person's medical record. Further investigation and professional development is required to ensure that a person's care is not compromised by failures in communication.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. In this study, the findings indicated that although some literature suggests that multidisciplinary communication is well developed within healthcare, more work is required to ensure that a person's care is not compromised. Which characteristics and interventions should be undertaken to improve a team's capacity to communicate well? Make a table outlining both individual characteristics and team characteristics that will promote improved communication.
2. How will the findings of this study impact on how you will approach communication with other healthcare colleagues? Which further education requirements would be needed to improve multidisciplinary team communication?

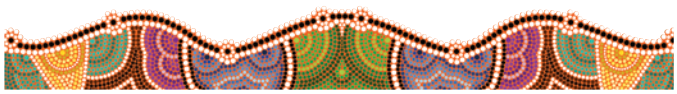
- Encourage the person and family to express their feelings, fears and concerns. *Open expression of feelings helps to promote understanding and acceptance.*
- Assist with understanding the grieving process and acceptance of feelings as normal. *Feelings of guilt, anger or depression may cause the person to withdraw from others. Explanation of the grieving process enhances understanding and ability to cope.*
- Help identify strengths and coping measures that have been used effectively in the past. Provide positive reinforcement for effective coping behaviour. *Past effective coping measures can help the person and family deal with the present situation and regain a sense of control.*
- Help the person and family make decisions regarding treatment and care. *This is also important to give them a sense of control.*
- Encourage use of other support systems, such as spiritual and social groups. Refer the individual and family to support groups, social support services and hospice care as indicated. Provide Cancer Council Australia literature and information as appropriate. *These support systems provide emotional support and help the person and family cope with the diagnosis.*
- Discuss advance directives (living wills) and power of attorney for healthcare with the individual and family. *These documents give the person and family a sense of control over the medical care provided if the person is no longer able to express their own wishes.*

Community-based care

A primary teaching need to prepare the person and family affected by lung cancer for home care is information about the disease itself, expected prognosis and planned treatment strategies. Provide honest information; do not promote false hope. Include the following additional topics in teaching for home care:

- importance of quitting smoking, especially if surgery has been performed. (The person with lung cancer may have difficulty recognising the need to stop smoking. Include information about the effects of nicotine and the tars in cigarette smoke on healing and already compromised lung tissue.)
- planned treatments such as chemotherapy or radiation therapy, including expected effects and usual side effects of each
- strategies to cope with noxious effects of radiation or chemotherapy
- activities and exercises to improve strength and regain function for the individual in the postoperative period
- the need to continue coughing and deep-breathing exercises at home
- symptoms to report to the doctor: fever, increasing or continued shortness of breath, cough, increased or purulent sputum, redness, pain, swelling or incisional drainage
- use of prescribed medications, including desired and potential side effects and interactions with other drugs or foods
- use of analgesics and other pain-relief measures for postoperative or cancer pain
- information about hospice services, community support, local cancer support groups for individuals and caregivers, and Cancer Council Australia services.

Refer the person and family for home health services, including nursing care, assistance with ADLs, respiratory care and respite care as needed.



CHAPTER HIGHLIGHTS

- Pneumonia, inflammation of the respiratory bronchioles and alveoli, usually is bacterial in origin. Different organisms are usually found in hospital-acquired pneumonia than in community-acquired pneumonia. Nursing care focuses on promoting airway clearance, supporting effective gas exchange and promoting rest.
- Infection control measures, including standard, airborne and contact precautions, are vital to prevent the spread of viral severe acute respiratory syndrome.
- Tuberculosis affects many people worldwide; in Australia, the primary affected populations are migrants, people with compromised immunity and people living in crowded or unsanitary conditions.
- The tuberculin test (PPD) detects a cellular immune response to *M. tuberculosis*, indicating infection but not necessarily active disease.
- Effective tuberculosis treatment is a public health concern, requiring therapy and compliance monitoring, contact follow up and assessment for adverse treatment effects.
- Disorders of the pleura, such as pleural effusion and pneumothorax, can affect lung expansion, ventilation and gas exchange when significant.
- Tension pneumothorax develops when air enters the pleural space but is unable to escape, collapsing the lung on the affected side and placing pressure on the unaffected lung and mediastinum. Ventilation, gas exchange, venous return and cardiac output can be significantly affected.
- Trauma may affect the chest wall (rib fracture, flail chest), the surface of the lungs (pulmonary contusion) or the airways and alveoli (smoke inhalation and near drowning). Flail chest and pulmonary contusion often occur concurrently; haemothorax also frequently develops with chest trauma. Chest trauma (chest wall or airways) can endanger effective ventilation and gas exchange.
- Lung cancer, the leading cause of cancer deaths, typically is advanced when diagnosed. Surgery, radiation therapy and chemotherapy are used to treat lung cancer, often in combination.

CONCEPT CHECK

1 Admitting orders for a person with acute bacterial pneumonia include an intravenous antibiotic every 8 hours, oxygen per nasal prongs at 5 L/min, continuous pulse oximetry monitoring, bed rest with bathroom privileges and chair at bedside as desired, diet as tolerated, sputum specimen for culture and sensitivity, FBC, urinalysis and electrolytes. Which order should the nurse carry out first?
1 Start the oxygen per nasal prongs.
2 Insert an intravenous catheter and start the prescribed antibiotic.
3 Provide a dinner tray to the person.
4 Obtain the sputum specimen.

2 When assessing a person with bacterial pneumonia, the nurse notes that the person's overall skin tone is somewhat grey and there is a bluish tinge around the person's finger tips. The nurse should (place the following in the correct order of priority):
1 start oxygen
2 assess breath sounds
3 notify the doctor
4 raise the head of the bed
5 obtain oxygen saturation level

3 The nurse evaluating a tuberculin test result 72 hours after it was administered notes an area of induration 9 mm in diameter. What additional information would indicate to the nurse that this is a positive result? The person:
1 resides in a long-term care facility
2 was born in South-East Asia
3 has HIV disease
4 is an injecting drug user

4 The nurse teaching a person taking prophylactic daily isoniazid (INH) following tuberculin test conversion includes which of the following in the instructions?
1 This drug turns your urine red-orange. This is harmless.
2 Report numbness and tingling of your extremities to your doctor.
3 You will need to have periodic eye examinations during treatment.
4 Do not use aspirin while taking this drug because abnormal bleeding may occur.

5 Which of the following statements made by a person with a new diagnosis of lung cancer would indicate that the nurse's teaching has been effective?
1 'Well, since I'm going to die anyway, I may as well go home, put my affairs in order and spend the rest of my time in the easy chair.'
2 'I understand that because the cancer has already spread, I will be undergoing aggressive cancer treatment for the next several years to beat this thing.'
3 'Even though I can't undo the damage caused by cigarette smoking, I will try to quit to prevent further damage to my lungs.'
4 'Having the "big C" is very scary; I'm just glad it is one of the more curable forms of cancer.'

6 The nurse caring for a person following a lobectomy notes 100 mL of red drainage in the chest drainage container since checking it 30 minutes previously. The nurse should (select all that apply):
1 empty the chest-tube drainage system
2 note the finding and re-evaluate drainage in 30 minutes
3 notify the surgeon
4 assess vital signs and level of consciousness
5 apply pressure to the chest tube insertion site

7 The nurse caring for a person having a thoracentesis appropriately assists the person to:
1 sit upright leaning forward during the procedure
2 breathe deeply as the needle is inserted
3 remain on quiet bed rest for 4 hours following the procedure
4 cough as the fluid is withdrawn

8 The nurse teaches a person being discharged from the emergency department with a diagnosis of fractured rib to:
1 avoid using pain medications to prevent respiratory depression
2 heavily tape the chest wall and promote comfort
3 remain on bed rest for a week to allow the fracture to stabilise
4 use a small pillow to splint the area when coughing

9 Which of the following assessment findings of a person with smoke inhalation does the nurse find of greatest concern?
1 Ash-like material in the sputum
2 Respiratory rate of 36
3 Skin and mucous membranes pink
4 Fine crackles in bilateral bases

10 Which of the following nursing diagnoses does the nurse identify as of highest priority for a person with tension pneumothorax?
1 *Decreased cardiac output*
2 *Ineffective breathing pattern*
3 *Acute pain*
4 *Risk of aspiration*

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CHAPTER 36

NURSING CARE OF PEOPLE WITH GAS EXCHANGE DISORDERS

MAJELLA HALES

LEARNING OUTCOMES

- Describe the epidemiology, pathophysiology, manifestations and management of a person with asthma.
- Differentiate between the types, pathophysiology and management of an individual experiencing a chronic obstruction pulmonary disease.
- Compare the progression and potential outcomes for a person with a lung disease such as cystic fibrosis, atelectasis or bronchiectasis.
- Describe the influence on gas exchange for a person experiencing an occupational lung disease or sarcoidosis.
- Differentiate between the types of pulmonary vascular disorders in relation to aetiology, manifestations and management.
- Identify the types, pathophysiology, manifestations and management options for a person with respiratory failure.

CLINICAL COMPETENCIES

- Assess functional health status of a person with a disorder affecting ventilation and gas exchange.
- Use data and knowledge of the effects of the disorder and prescribed treatment to identify priority nursing diagnoses and to plan care for a person with a disorder affecting ventilation and gas exchange.
- Use the nursing process and evidence-based nursing research to plan and implement individualised nursing care for individuals, including measures to promote ventilation and gas exchange.
- Plan and provide appropriate teaching for health promotion in vulnerable populations and to prepare individuals and families for community-based care.
- Evaluate the effectiveness of nursing interventions and teaching, revising strategies and teaching plans as needed.
- Coordinate safe interprofessional care and administer prescribed medications and treatments for people with disorders affecting ventilation and gas exchange.

KEY TERMS

Normal function of the lower respiratory tract depends on several organ systems: the central nervous system, which stimulates and controls breathing; chemoreceptors in the brain, aortic arch and carotid bodies, which monitor the pH and oxygen content of blood; the heart and circulatory system, which provide for blood supply and gas exchange; the musculoskeletal system, which provides an intact thoracic cavity capable of expanding and contracting; and the lungs and bronchial tree, which allow air movement and gas exchange. Impaired function of any of these systems affects ventilation. As a result, tissues may become *hypoxic*, with inadequate oxygen to support metabolic activity.

Although some of the disorders discussed in this chapter can affect ventilation (air movement into and out of the airways and alveoli), all can have significant effects on gas exchange. The mechanisms by which they affect gas exchange differ:

- In reactive airway disease (asthma) and obstructive disorders, air trapping reduces the amount of oxygen available to drive gas exchange.
- Interstitial lung disorders affect the ability of the lungs to expand and the work of breathing—again, reducing alveolar oxygenation and gas exchange.
- Pulmonary vascular disorders affect blood flow to the lungs or a portion of the lungs, reducing gas exchange through their effects on perfusion of the lungs.
- Respiratory failure is the ultimate consequence of impaired gas exchange; the lungs cannot adequately oxygenate the blood or eliminate sufficient carbon dioxide.

With a few exceptions, the disorders discussed in this chapter are relatively common, chronic lung diseases.

Disorders of other body systems, such as neurological disorders (e.g. head injury, spinal cord trauma or disorders, multiple sclerosis and myasthenia gravis) can also affect gas exchange through their effects on the central or peripheral nervous systems. These disorders and their effects on the respiratory system are discussed in subsequent chapters of this text.

FAST FACTS

- Approximately 6.3 million Australians live with a chronic respiratory condition.
- Approximately 10% of Australians have asthma.
- Approximately 5.7% of Australians over 55 have a chronic obstructive pulmonary disease (COPD).
- In 2013, mortality from COPD in males had reduced by two-thirds of that in 1970.
- In 2014, over 6400 people died of COPD (54% males, 45% females).

Sources: Australian Institute of Health and Welfare (AIHW) (2014a); Australian Bureau of Statistics (ABS) (2015a).

Ageing affects pulmonary ventilation and gas exchange as well. The number of alveoli decrease and emphysematous changes reduce the surface area for gas exchange. Alveoli become less elastic, causing increased air trapping and dead space. For most older adults who remain active, these changes have minimal effect on exercise tolerance and activities of daily living (ADLs). When combined with lung disease, however, age-related pulmonary changes increase the person's risk of developing respiratory failure.

In 2013, chronic lower respiratory diseases ranked fourth overall as the leading cause of death for non-Indigenous Australians, and third for Aboriginal and Torres Strait Islander Australians (ABS, 2012; 2015c).

REACTIVE AIRWAY DISEASE

In reactive airway disease, the airways narrow in response to a stimulus. Airway narrowing limits airflow both into and out of the alveoli. Limited airflow increases the work of breathing and the residual volume of the lungs as air is trapped behind narrowed airways. Inspired air mixes with an abnormally large volume of residual air, effectively reducing the amount of oxygen available in the alveoli. Decreased alveolar ventilation further reduces oxygen available for exchange.

THE PERSON WITH ASTHMA

Asthma is a chronic inflammatory disorder of the airways characterised by recurrent episodes of wheezing, breathlessness, chest tightness and coughing. Inflammation causes increased responsiveness of the airways to multiple stimuli. The widespread airflow obstruction that occurs during acute episodes usually reverses either spontaneously or with treatment. While most episodes or asthma 'attacks' are relatively brief, some individuals with asthma may experience longer episodes with some degree of airway impairment daily. In rare cases, an acute episode of asthma is so severe that respiratory failure and death result.

Prevalence and risk factors

In Australia, approximately 10% of the population had asthma in 2013. Rates are decreasing in children and young adults, but have remained stable in adults 35 years and older (ABS, 2015a). Prevalence is similar in males and females aged 5–34, but higher in females aged 35 years and over. Admissions for asthma vary across the lifespan with the significant majority of admissions occurring between 1 and 4 years of age (see Figure 36.1). Although mortality rates have reduced by 45% since 1997, 0.3% of all deaths annually are asthma related (ABS, 2015b; AIHW, 2014b). In comparison with international standards this figure is high. Asthma remains a significant problem and burden on healthcare resources (AIHW, 2014b).

A number of risk factors can be identified for asthma, although many people develop the disease in the absence of known risk factors. Allergies play a strong role in childhood asthma; a lesser role in adults. There is a strong genetic component to the disease, although a specific pattern of inheritance has not been identified. More than 50 different genes have been identified as being involved in a variety of processes contributing to the development of asthma (Weiss, 2015). Environmental factors, including dust

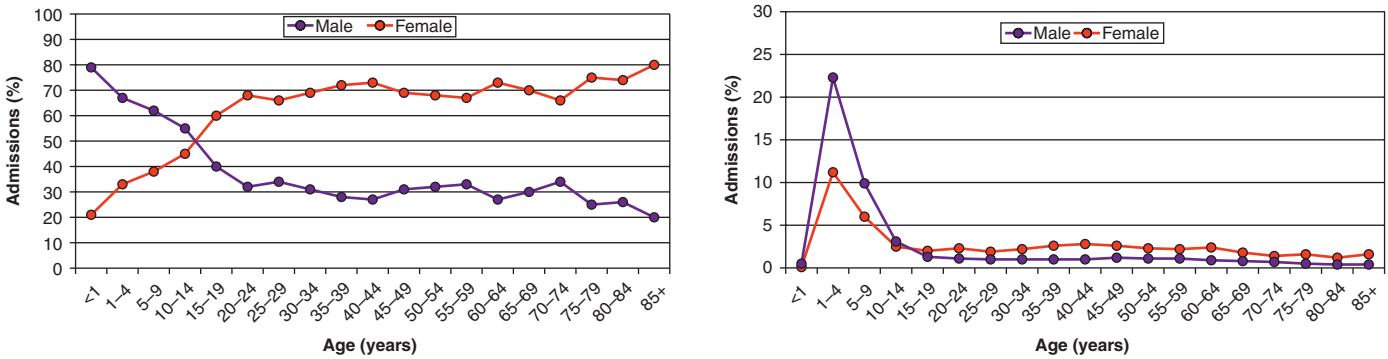


FIGURE 36.1 ■ Asthma admissions. *A*, As a percentage by gender across each age group, 2012–2013, Australia. *B*, As a percentage of all asthma admissions by age and gender, 2012–2013, Australia

Source: Generated using data from AIHW (2015a). *Principal diagnosis data cubes: Separation statistics by principal diagnosis in ICD-10-AM, Australia, 2011–12 to 2012–13—ICD-10 J45-Asthma & J46-Status asthmaticus*. Retrieved from www.aihw.gov.au/hospitals-data/principal-diagnosis-data-cubes/.

mites, animal and fungal allergens, pollution and occupational exposure to industrial compounds, may contribute. Respiratory viruses such as rhinovirus and influenza can precipitate asthma attacks. Other contributory factors include exercise (particularly in cold air) and emotional stress (Morris, 2015).

FAST FACTS

- Only 20% of people with asthma 15 years and over and only 41% of children between 0 and 14 years of age have a written asthma action plan.
- Respiratory tract infections are frequently identified as an exacerbating factor in admission to hospital.
- In 2008–2009*, $665 million was spent on asthma, equating to approximately 0.9% of the healthcare budget.
- Fifty per cent of the healthcare budget is related to the prevision/subsidy of prescription pharmaceuticals.

*Most recent data available.

Sources: Data extracted from AIHW (2015b; 2015c); National Asthma Council Australia (NACA) (2015).

Physiology review

Airways within the lungs contain criss-crossing strips of smooth muscle that control their diameter. This muscle is innervated by the autonomic nervous system. Parasympathetic (cholinergic) stimulation leads to bronchoconstriction or narrowing of the airways. Sympathetic stimulation through β_2-adrenergic receptors causes bronchodilation or expansion of the airways (Martini, Nath & Bartholomew, 2014) and slight bronchoconstriction normally predominates. However, when increased airflow is necessary (e.g. during exercise), the parasympathetic system is inhibited and stimulation of the sympathetic system causes bronchodilation. Inflammatory mediators (such as histamine) released during an antigen–antibody response act directly on bronchial smooth muscle to produce bronchoconstriction.

Pathophysiology

In asthma, the airways are in a persistent state of inflammation. During symptom-free periods, airway inflammation in asthma is subacute or quiet. Even during these periods, however, inflammatory cells such as eosinophils, neutrophils and lymphocytes

FOCUS ON CULTURAL DIVERSITY Prevalence of asthma in certain populations

- Asthma prevalence is higher in Aboriginal and Torres Strait Islander peoples in every age group, as shown in the table below:

AGE IN YEARS	ABORIGINAL AND TORRES STRAIT ISLANDER AUSTRALIANS	NON-INDIGENOUS AUSTRALIANS
0–14	14.5	9.3
15–24	17.1	10.7
45–54	22.0	9.6
> 55	22.2	10.5

- Prevalence of asthma is lower in people who are living in Australia but were born overseas, as represented by the following table:

COUNTRY OF BIRTH	RELATIVE PERCENTAGE OF POPULATION
Australia	11.2 %
North Africa and the Middle East	10.8 %
United Kingdom	10.0 %
Other Oceania	9.0 %
Other North-West Europe	7.5 %
Southern and Eastern Europe	5.9 %
South-East Asia	4.7 %

Sources: Data extracted from ABS (2012; 2013).

may be found in airway tissues, and oedema may be present. An acute inflammatory response, during which resident inflammatory cells interact with inflammatory mediators, cytokines and additional infiltrating inflammatory cells, may be triggered by a variety of factors (Bullock & Hales, 2012). Common triggers for an acute asthma attack include exposure to allergens, respiratory tract infection, exercise, inhaled irritants and emotional upsets.

Attack triggers

Childhood asthma (which may continue into adulthood) is most often linked to inhalation of allergens such as pollen, animal dander (shed skin flakes) or household dust. Individuals with allergic asthma often have a history of other allergies. Environmental pollutants, such as tobacco smoke and irritant gases (e.g. sulfur dioxide, nitrogen dioxide and ozone), can provoke asthma. Exposure to second-hand smoke as a child is associated with a higher risk and increased severity of asthma. Agents found in the workplace, such as noxious fumes and gases, chemicals and dusts, may cause occupational asthma.

Respiratory infections—viral, in particular—are a common internal stimulus for an asthmatic attack. Exercise-induced asthma attacks are also relatively common. Loss of heat or water from the bronchial surface may contribute to exercise-induced asthma. Exercising in cold, dry air increases the risk of an asthma attack in susceptible people.

Emotional stress is a significant aetiological factor for attacks in almost half of people with asthma. Common pharmacological triggers include aspirin and other non-steroidal anti-inflammatory drugs, sulfites (which are used as preservatives in wine, beer, fresh fruits and salad) and beta-blockers.

See Box 36.1 for triggers of asthma.

Responses

When a trigger such as inhalation of an allergen or irritant occurs, an acute or *early response* develops in the hyper-reactive airways predisposed to bronchospasm. Sensitised mast cells in the bronchial mucosa release inflammatory mediators such as histamine, prostaglandins and leukotrienes. Resident and infiltrating inflammatory cells also produce inflammatory mediators such as cytokines, bradykinin and growth factors. These mediators stimulate parasympathetic receptors and bronchial smooth muscle to produce bronchoconstriction. They also increase capillary permeability, which allows plasma to escape and leads to mucosal oedema. Mucus production is stimulated; excess mucus collects in the narrowed airways (Bullock & Hales, 2012).

The attack is prolonged by the *late phase response*, which develops 4 to 12 hours after exposure to the trigger. Inflammatory cells such as basophils and eosinophils are activated, which damage airway epithelium, produce mucosal oedema, impair mucociliary clearance and produce or prolong bronchoconstriction. The degree of hyper-reactivity depends on the extent of inflammation. Together, bronchoconstriction, oedema and inflammation, and mucus secretion narrow the airway. Airway resistance increases, limiting airflow and increasing the work of breathing (see Figure 36.2).

Limited expiratory airflow traps air distal to the spastic, narrowed airways. Trapped air mixes with inspired air in the alveoli, reducing its oxygen tension and gas exchange across the alveolar–capillary membrane. Distended alveoli compress alveolar capillaries, reducing blood flow and further affecting gas exchange. As a result, hypoxaemia develops. Hypoxaemia and increased lung volume due to trapping stimulate the respiratory rate. Hyperventilation causes the $PaCO_2$ to fall, leading to respiratory alkalosis. (See Chapter 9 for more information about acid–base imbalances.)

To summarise, in an acute asthma attack inflammatory mediators are released from sensitised airways followed by activation of inflammatory cells. These events lead to bronchoconstriction, airway oedema and impaired mucociliary clearance. Airway narrowing limits airflow and increases the work of breathing; trapped air mixes with inhaled air, impairing gas exchange.

Manifestations and complications

An asthma attack is characterised by a subjective sensation of chest tightness, cough, dyspnoea and wheezing (see 'Manifestations' box below). The onset of symptoms may be either abrupt or insidious, and an attack may subside rapidly or persist for hours or days. A sense of chest constriction and non-productive cough are common early manifestations of an attack. During an attack, tachycardia, tachypnoea and prolonged expiration are common. Diffuse wheezing is heard on auscultation. With more severe attacks, use of the accessory muscles of respiration, intercostal retractions, loud wheezing and distant breath sounds may be noted. Fatigue, anxiety, apprehension and severe dyspnoea that allows speaking only one or two words between breaths may occur with persistent severe episodes. The onset of respiratory failure is marked by inaudible breath sounds with reduced wheezing and an ineffective cough. Without careful assessment, this apparent relief of symptoms can be misinterpreted as an improvement.

BOX 36.1 Triggers of asthma

- Environmental allergens (often small glycoproteins, e.g. dust mites, animal dander, pollen, fungi)
- Cigarette smoking
- Pollution
- Irritants (fumes from volatile compounds, e.g. cleaning agents, glues, paints)
- Respiratory tract infections
- Medications (aspirin, non-steroidal anti-inflammatory drugs, beta-blockers)
- Physical factors (exercise, changes in temperature)
- Gastro-oesophageal reflux
- Emotional stress
- Occupational exposure to organic compounds
- Food additives

MANIFESTATIONS Acute asthma

- Dyspnoea
- Tachypnoea
- Tachycardia
- Chest tightness
- Wheezing
- Cough
- Anxiety

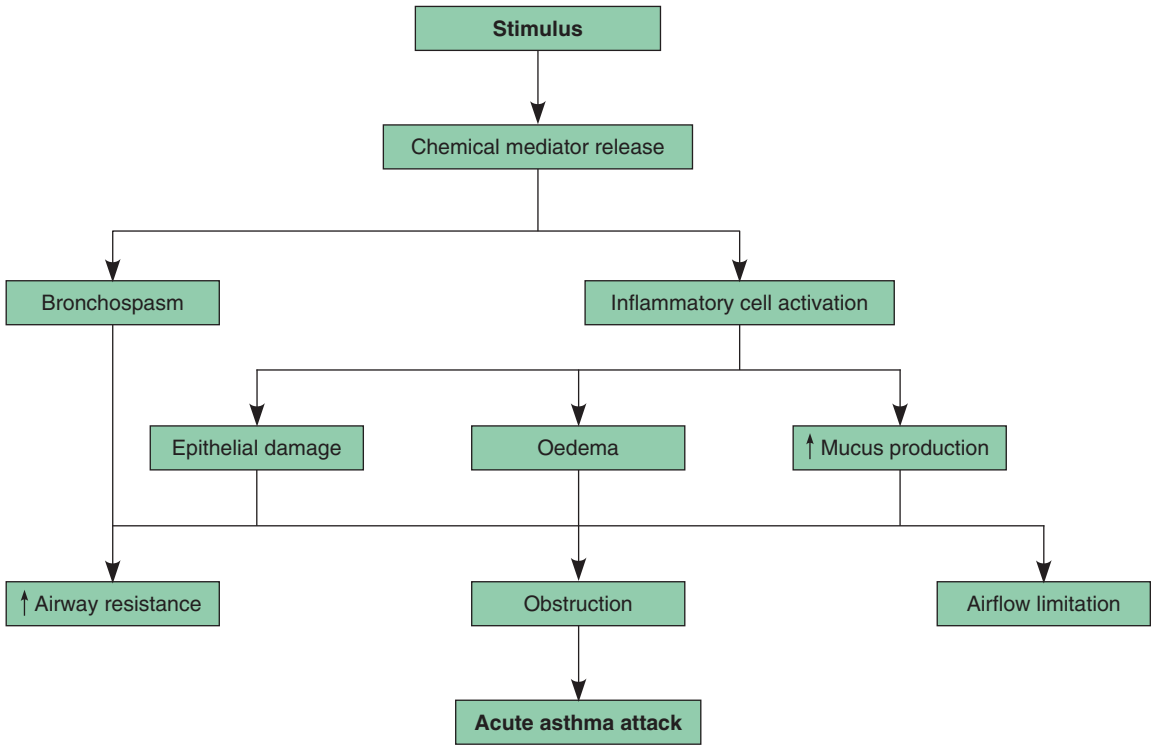


FIGURE 36.2 ■ Pathogenesis of an acute episode of asthma

The frequency of attacks and severity of symptoms vary greatly from person to person. Although some people have infrequent, mild episodes, others have nearly continuous manifestations of cough, dyspnoea on exertion and wheezing, with periodic severe exacerbations (see Table 36.1). **Status asthmaticus** is severe, prolonged asthma that does not respond to routine treatment. Without aggressive therapy, status asthmaticus can lead to respiratory failure with hypoxaemia, hypercapnia and acidosis. Endotracheal intubation, mechanical ventilation and aggressive drug treatment may be necessary to sustain life.

In addition to acute respiratory failure, other complications associated with acute asthma include dehydration, respiratory infection, atelectasis, pneumothorax and cor pulmonale.

Cough-variant asthma

A common symptom of asthma is a cough which is usually non-productive and non-paroxysmal (Morris, 2015). Cough can be initiated by either upper airway irritants (e.g. post nasal drip or gastro-oesophageal reflux disease) or by inflammation or constriction of the lower airways. Most commonly, cough associated with asthma is accompanied by classic asthma symptoms

TABLE 36.1 GINA assessment of asthma control in adults, adolescents and children 6–11 years

ASTHMA SYMPTOM CONTROL	CONTROLLED	PARTLY CONTROLLED	UNCONTROLLED
Daytime asthma symptoms more than twice a week? Any night waking due to asthma? Reliever needed for symptoms more than twice a week? Any activity limitation due to asthma?	None of these	1–2 of these	3–4 of these
Risk factors for poor asthma outcomes Assess risk factors at diagnosis and periodically, particularly for individuals experiencing exacerbations. Measure FEV_1 at start of treatment, after 3–6 months of controller treatment to record the individual's personal best lung function, then periodically for ongoing risk assessment.			Having one or more of the risk factors increases the risk of exacerbations even if symptoms are well controlled.

Source: Global Initiative for Asthma (GINA) (2015). *Global strategy for asthma management and prevention*. Retrieved from www.ginasthma.org/local/uploads/files/GINA_Report_2015_May19.pdf.

such as chest constriction, dyspnoea and wheezing. Individuals with *cough-variant asthma*, however, have persistent cough without wheezing or dyspnoea, often delaying diagnosis. These individuals do have significant airway inflammation and demonstrate the pathophysiological features of asthma.

INTERPROFESSIONAL CARE

The diagnosis of asthma is based primarily on the history and manifestations. Treatment goals are twofold. Daily management focuses on controlling symptoms and preventing acute attacks. During an acute attack, therapy is directed towards restoring airway patency and alveolar ventilation.

Diagnosis

Diagnostic tests are used to determine the degree of airway involvement during and between acute episodes, and to identify causative factors such as allergens. See Chapter 33 for more information about and the nursing care related to these diagnostic tests.

- *Respiratory function tests (RFTs)* are used to evaluate the degree of airway obstruction. Respiratory function testing done before and after use of an aerosolised bronchodilator helps determine the reversibility of airway obstruction. The residual volume of the lungs may be increased and the vital capacity decreased or normal even during periods of remission. The forced expiratory volume and peak expiratory flow rate are the most valuable respiratory function studies to evaluate the severity of an asthma attack and the effectiveness of treatment measures.
- *Challenge* or *bronchial provocation testing* uses an inhaled substance such as methacholine or histamine with RFTs to confirm the diagnosis of asthma by detecting airway hyperresponsiveness.
- *Arterial blood gases (ABGs)* are drawn during an acute attack to evaluate oxygenation, carbon dioxide elimination and acid–base status. ABGs initially show hypoxaemia with a low PaO_2 and mild respiratory alkalosis with an elevated pH and low $PaCO_2$ due to tachypnoea. Severe airflow obstruction causes significant hypoxaemia and respiratory acidosis ($pH < 7.35$ and $PaCO_2 > 42$ mmHg), indicative of respiratory failure and the need for mechanical ventilation. See Chapter 9 for more information about arterial blood gases and their interpretation.
- *Skin testing* may be done to identify specific allergens if an allergic trigger is suspected for asthma attacks.

Disease monitoring

Peak expiratory flow rate (PEFR) is used on a day-to-day basis to evaluate the severity of bronchial hyperresponsiveness. Small, inexpensive meters to measure PEFR are available. Readings taken at varying times of day over several weeks are used to establish the person's personal best or normal PEFR. This value is then used to evaluate the severity of airway obstruction. The National Asthma Council of Australia recommends that some people with asthma may benefit from regular peak flow monitoring, which enables not only the individual but also asthma healthcare professionals to monitor and recognise exacerbations early (NACA, 2015).

Preventive measures

Asthma attacks often can be prevented by avoiding allergens and environmental triggers. Modifying the home environment by controlling dust, removing carpets, covering mattresses and pillows to reduce dust mite populations and installing air-filtering systems may be useful. Pets may need to be removed from the household. Eliminating all tobacco smoke in the home is vital. Wearing a mask that retains humidity and warm air while exercising in cold weather may help prevent attacks of exercise-induced asthma. Early treatment of respiratory infections is vital to prevent asthma exacerbations. National guidelines recommend that all individuals with asthma have a written asthma action plan. Despite the recommendation being in place for over 20 years, the majority of people with asthma in Australia do not have a plan. Healthcare professionals should work towards ensuring that all individuals in their care have a written asthma action plan.

Asthma first aid

Friends and family should be aware of how to manage an individual who is having an asthma attack. The National Asthma Council Australia advocates a four-step plan for acute asthma management. The First Aid for Asthma chart (see Figure 36.3) clearly identifies steps to assist an individual who is having an asthma attack.

Medications

Medications are used to prevent and control asthma symptoms, reduce the frequency and severity of exacerbations, and reverse airway obstruction. Drugs used for long-term control of asthma are taken daily to maintain control of the disease. The primary drugs in this group are anti-inflammatory agents, long-acting bronchodilators and leukotriene receptor antagonists. Quick-relief medications provide prompt relief of bronchoconstriction and airflow obstruction with associated wheezing, cough and chest tightness. Short-acting adrenergic stimulants (rapid-acting bronchodilators), anticholinergic drugs and methylxanthines fall into this category.

A step-wise approach for managing asthma is recommended (see Figure 36.4). For all individuals, a short-acting inhaled β_2-agonist is recommended for quick relief of acute symptoms. Up to three treatments at 20-minute intervals or a single nebuliser treatment may be used as needed. Strategies for long-term control may need to be modified if a short-acting bronchodilator is needed more than twice a week (NACA, 2015).

Many of the drugs used for continued asthma management and relief of an acute attack can be administered by a metered-dose inhaler (MDI), dry powder inhaler (DPI) or nebuliser. The advantages of administering medications locally by inhalation include rapid onset and reduced systemic effects of the drugs. In an MDI, a chemical propellant is used to deliver the medication when the canister is depressed. In contrast, DPI contains no propellant. Instead, the medication is released by inhaling rapidly through the mouthpiece.

Nursing implications for medications used to treat asthma are outlined in the 'Medication administration' box below.

First Aid for Asthma

1

Sit the person comfortably upright.
Be calm and reassuring.
Don't leave the person alone.

2

Give 4 puffs of a blue/grey reliever
(e.g. Ventolin, Asmol or Airomir)
Use a spacer, if available.
Give 1 puff at a time with 4 breaths after each puff
Use the person's own inhaler if possible.
If not, use first aid kit inhaler or borrow one.

OR

Give 2 separate doses of a Bricanyl or Symbicort inhaler
If a puffer is not available, you can use Symbicort **(people over 12)** or Bricanyl, even if the person does not normally use these.

3

Wait 4 minutes.
If the person still cannot breathe normally, **give 4 more puffs.**

Wait 4 minutes.
If the person still cannot breathe normally, **give 1 more dose.**

4

If the person still cannot breathe normally,
CALL AN AMBULANCE IMMEDIATELY (DIAL 000)
Say that someone is having an asthma attack.

Keep giving reliever.
Give 4 puffs every 4 minutes until the ambulance arrives.
Children: 4 puffs each time is a safe dose.
Adults: For a severe attack you can give up to 6–8 puffs every 4 minutes

If the person still cannot breathe normally, **CALL AN AMBULANCE IMMEDIATELY (DIAL 000)** Say that someone is having an asthma attack.

Keep giving reliever while waiting for the ambulance:
For Bricanyl, give 1 dose every 4 minutes
For Symbicort, give 1 dose every 4 minutes (up to 3 more doses)

HOW TO USE INHALER

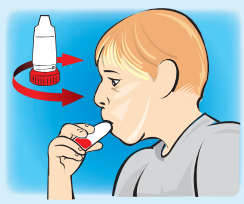
WITH SPACER

- **Assemble spacer**
- **Remove puffer cap** and **shake well**
- Insert puffer upright into spacer
- Place mouthpiece between teeth and seal lips around it
- **Press once firmly** on puffer to fire one puff into spacer
- **Take 4 breaths** in and out of spacer
- Slip spacer out of mouth
- **Repeat** 1 puff at a time until 4 puffs taken – remember to shake the puffer before each puff
- **Replace cap**

WITHOUT SPACER

- **Remove cap** and **shake well**
- **Breathe out** away from puffer
- Place mouthpiece between teeth and seal lips around it
- **Press once firmly** on puffer while breathing in slowly and deeply
- Slip puffer out of mouth
- **Hold breath** for 4 seconds or as long as comfortable
- Breathe out slowly away from puffer
- **Repeat** 1 puff at a time until 4 puffs taken – remember to shake the puffer before each puff
- **Replace cap**

BRICANYL OR SYMBICORT



- **Unscrew cover** and remove
- **Hold inhaler upright and twist grip** around and then back
- **Breathe out** away from inhaler
- Place mouthpiece between teeth and seal lips around it
- **Breathe in forcefully and deeply**
- Slip inhaler out of mouth
- Breathe out slowly away from inhaler
- **Repeat** to take a second dose – remember to twist the grip both ways to reload before each dose
- **Replace cover**

Not Sure if it's Asthma?
CALL AMBULANCE IMMEDIATELY (DIAL 000)
If a person stays conscious and their main problem seems to be breathing, follow the asthma first aid steps. Asthma reliever medicine is unlikely to harm them even if they do not have asthma.

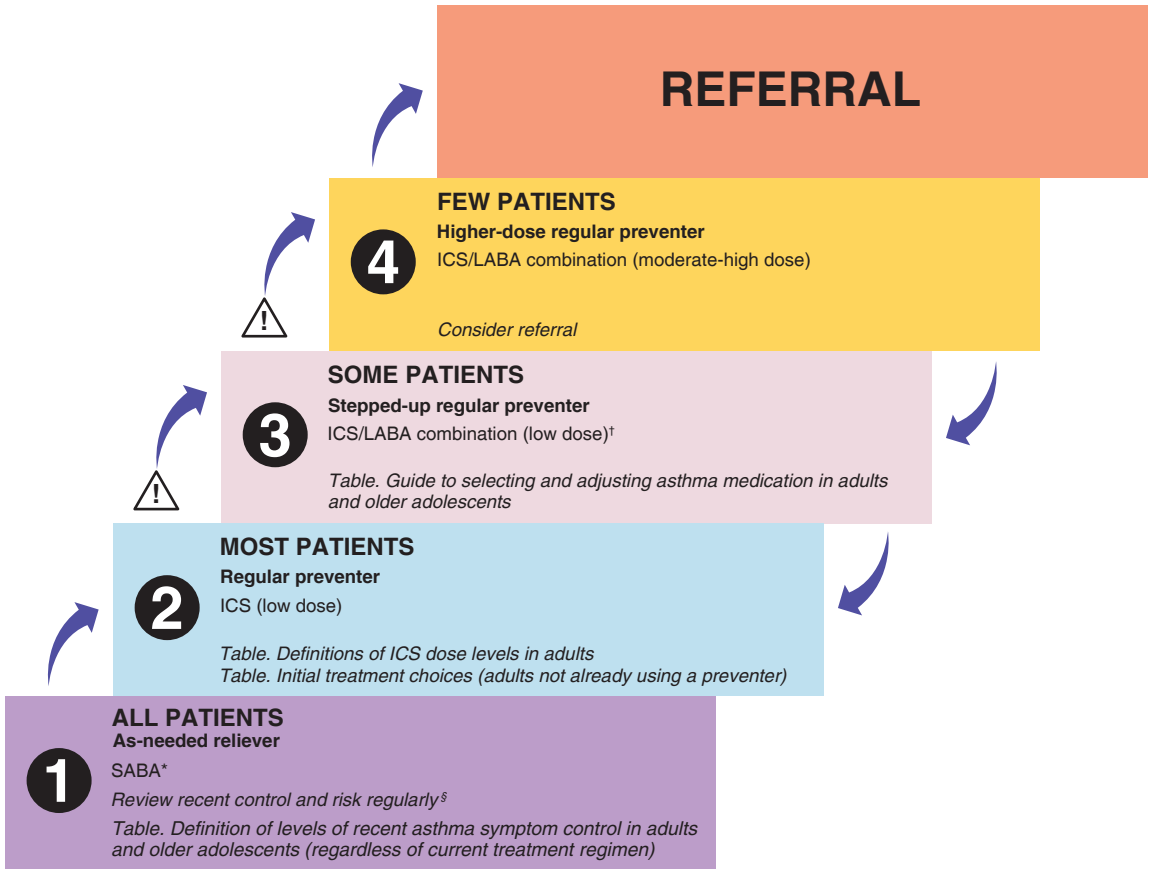
Severe Allergic Reactions
CALL AMBULANCE IMMEDIATELY (DIAL 000)
Follow the person's Action Plan for Anaphylaxis if available. If the person has known severe allergies and seems to be having a severe allergic reaction, use their adrenaline autoinjector (e.g. EpiPen, Anapen) before giving asthma reliever medicine.

For more information on asthma visit:
Asthma Foundations – www.asthmaaustralia.org.au
National Asthma Council Australia – www.nationalasthma.org.au

National Asthma Council Australia
leading the attack against asthma

FIGURE 36.3 ■ First Aid for Asthma chart

Source: NACA (2011). *First Aid for Asthma.* Retrieved from www.nationalasthma.org.au/uploads/content/22-First-Aid-Asthma-Chart.pdf.



ICS: inhaled corticosteroid; SABA: short-acting $beta_2$ agonist; LABA: long-acting $beta_2$ agonist

* Reliever: Short-acting $beta_2$ agonist (or low-dose budesonide/eformoterol combination for patients using this combination as both maintenance and reliever).

§ In addition, manage flare-ups with extra treatment when they occur, and manage exercise-related asthma symptoms as indicated.

† Montelukast can be added to inhaled corticosteroid as an alternative to switching to ICS/LABA, but is less effective.

Note: PBS status as at April 2015: Montelukast treatment is not subsidised by the PBS for people aged 15 years or over. Special Authority is available for DVA gold card holders, or white card holders with approval for asthma treatments.

FIGURE 36.4 ■ Stepped approach to adjusting asthma medication in adults

Source: Adapted from National Asthma Council Australia (2015). *Asthma management handbook—2014*. Retrieved from www.asthmahandbook.org.au/figure/show/31. Reproduced with permission.

BRONCHODILATORS Most asthmatics need bronchodilator therapy to control their symptoms (see Table 36.2). Inhalation of nebulised medication is the preferred means of administration. The primary bronchodilators used include adrenergic stimulants and anticholinergic agents. These drugs often are administered in combination with an anti-inflammatory agent.

Adrenergic stimulants (β_2-agonists) affect receptors on smooth muscle cells of the respiratory tract, causing smooth muscle relaxation and bronchodilation. Long-acting adrenergic stimulants such as inhaled salmeterol and oral sustained-release salbutamol are used in conjunction with anti-inflammatory drugs to control symptoms, but are not appropriate to treat an acute episode of asthma. Inhaled short-acting beta-adrenergic agonists such as salbutamol and terbutaline, administered by MDI or DPI, are the treatment of choice for quick relief. They act within minutes, but their duration generally is short, lasting only 4 to 6 hours. Tachycardia and muscle tremors, common side effects of adrenergic agonists, are minimal with inhalation therapy.

Anticholinergic medications prevent bronchoconstriction by blocking parasympathetic input to bronchial smooth muscle. Ipratropium bromide, an anticholinergic drug administered by MDI, is useful when asthma symptoms are poorly controlled by adrenergic stimulants alone. Anticholinergic drugs act more slowly than adrenergic stimulants, requiring up to 60 to 90 minutes to achieve maximal effect.

ANTI-INFLAMMATORY AGENTS Corticosteroids and two non-steroidal anti-inflammatory agents, cromolyn sodium and

TABLE 36.2 Asthma medications and delivery devices

GENERIC NAME	COMMON TRADE NAMES	DEVICE OR PRESENTATION	ROUTE OF DELIVERY	CLASS
RELIEVERS				
Salbutamol	Airomir Asmol Butamol Epaq Ventolin	Autohaler MDI Nebulising solution Syrup Injection *(Often for obstetric purposes)*	Inhaled Oral Subcutaneous injection Intramuscular injection Intravenous injection	SAßa
Terbutaline	Bricanyl	Turbuhaler Elixir Solution for injection	Inhaled Oral Subcutaneous injection	
PREVENTERS				
Ciclesonide	Alvesco	MDI	Inhaled	ICS
Fluticasone propionate	Flixotide	Accuhaler	Inhaled	ICS
Budesonide	Pulmicort	Turbuhaler Nebulising suspension	Inhaled	ICS
Beclomethasone dipropionate	Qvar	MDI Autohaler	Inhaled	OCS
Sodium cromoglycate	Intal Forte	Nebulising solution	Inhaled	Mast cell stabiliser
Nedocromil sodium	Tilade	MDI	Inhaled	Mast cell stabiliser
Montelukast	Singulair	Tablet	Oral	LTRA
SYMPTOM CONTROLLERS				
Eformoterol fumarate dehydrate	Oxis Foradile	Turbuhaler	Inhaled	LAßa
Salmeterol xinafoate	Serevent	Accuhaler	Inhaled	LAßa
COMBINATION MEDICATIONS				
Fluticasone and salmeterol	Seretide	Accuhaler MDI	Inhaled	LAßa + ICS
Budesonide and eformoterol	Symbicort	Turbuhaler	Inhaled	LAßa + ICS

ICS = inhaled corticosteroids; LAßa = long-acting, selective beta$_2$-adrenoreceptor agonist; MDI = metered dose inhaler; SAßa = short-acting beta agonist; LTRA = leukotriene receptor antagonist.

nedocromil, are used to suppress airway inflammation and reduce asthma symptoms.

Corticosteroids block the late response to inhaled allergens and reduce bronchial hyper-responsiveness. The preferred route of administration is by MDI or DPI to minimise systemic absorption and reduce the adverse effects of prolonged steroid use (cushingoid effects). For a severe acute attack, corticosteroids may be given systemically to alleviate symptoms and induce remission.

Cromolyn sodium and nedocromil are used to prevent acute episodes of asthma. They reduce airway hyper-reactivity and inhibit the release of mediator substances. These drugs are used for long-term control of asthma, not quick relief. They have a wide margin of safety and few side effects.

LEUKOTRIENE ANTAGONISTS Leukotriene modifiers, montelukast (Singulair) and zafirlukast (Accolate), are oral medications that reduce the inflammatory response in asthma. They appear to improve lung function, diminish symptoms and reduce the need for short-acting bronchodilators. These drugs affect the metabolism and excretion of other medications such as warfarin and theophylline and may cause liver toxicity.

METHYLXANTHINE Theophylline is an old drug of the methylxanthine class, which is used less commonly nowadays

MEDICATION ADMINISTRATION Asthma

ADRENERGIC STIMULANTS

Adrenergic stimulants affect sympathetic receptors in the respiratory tract. Administered by metered-dose inhalers or dry powder inhalers, these drugs are the treatment of choice for acute bronchial asthma. Nearly all of the drugs in this class (adrenaline and isoprenaline being the exceptions) selectively activate β_2-receptors at the doses typically used to treat asthma. β_2-receptor activation results in smooth muscle relaxation and bronchodilation. Salmeterol and eformoterol are highly selective to β_2-receptors, resulting in fewer adverse effects. However, they have been shown to increase the risk of serious asthma exacerbations and death by masking the symptoms of worsening inflammation. Salmeterol (or eformoterol) should be used with concomitant corticosteroid therapy to prevent rapid deterioration from serious bronchospasm (MIMS, 2015a).

Oral forms of adrenergic agonists may be used for prophylaxis but are not effective in treating an acute attack because of their slow onset. When administered orally or parenterally, their effect on sympathetic nervous system receptors can produce undesirable side effects, such as nervousness, irritability, tachycardia and cardiac arrhythmias.

Nursing responsibilities

- Use with caution in individuals with hypertension, cardiovascular disease or arrhythmias, hyperthyroidism or diabetes.
- When given to a person who is hypoxaemic and acidotic, these drugs may cause potentially dangerous cardiac stimulation.
- Observe for desired effect of reduced dyspnoea and wheezing. Central nervous system stimulation (anxiety, irritability and insomnia) and tremor are common side effects.

Health education for the person and family

- Follow the First Aid for Asthma chart (see Figure 36.3).
- Use the prescribed inhaler or nebuliser as directed.
- If taking a bronchodilator along with another medication by inhalation, use the bronchodilator first to open airways and enhance the effectiveness of the second medication.
- Rinse mouth after using inhalers to reduce systemic absorption of the medication.
- Keep a log to track bronchodilator use. If the drug becomes less effective or if higher doses are needed, contact a doctor.
- Report palpitations, irregular pulse and other side effects to the doctor.

ANTICHOLINERGICS

Anticholinergics are potent bronchodilators, blocking muscarinic receptors of the parasympathetic nervous system. Activation of muscarinic receptors produces smooth muscle contraction and bronchoconstriction; blockade of these receptors facilitates smooth muscle relaxation and bronchodilation. Atropine is used infrequently because of its tendency to dry secretions of the mucous membranes and other side effects. Ipratropium and tiotropium bromide are available as inhalers and have fewer side effects than atropine.

Nursing responsibilities

- Assess for possible contraindications to the drug, including hypersensitivity, glaucoma, prostatic hypertrophy or bladder-neck obstruction.
- Assess for desired and/or adverse effects: improving or worsening symptoms; nausea, vomiting, abdominal cramping, anxiety, dizziness; headache.
- Provide ice chips or sips of clear fluid to relieve dry mouth.

Health education for the person and family

- To prevent overdose, take no more than the prescribed number of doses per day.
- If the drug becomes less effective over time, notify the doctor; an adjustment in dosage may be needed.

CORTICOSTEROIDS

The anti-inflammatory effect of corticosteroids helps both prevent and treat acute episodes. Corticosteroids are used to reduce the frequency and severity of asthma attacks and allow reduced dosages of other drugs. The beneficial effects of corticosteroids for asthma result from their ability to decrease the synthesis and release of inflammatory mediators (such as histamine and leukotrienes), reduce inflammatory cell activation and infiltration and decrease airway oedema. Corticosteroids also increase the number and receptivity of β_2-receptors (Chetta & Olivieri, 2012). The cushingoid side effects of corticosteroids, always a major concern with their use, are minimised when they are inhaled. Note that the combination product salmeterol/fluticasone is associated with an increased risk of serious asthma exacerbations and death. It is a second-line drug, recommended for use only when asthma is inadequately controlled using other preparations (MIMS, 2015b).

Nursing responsibilities

- Administer inhaler doses after bronchodilators to facilitate transit of the medication to distal airways.
- Assess for common side effects: sore throat; hoarseness; and oropharyngeal or laryngeal *Candida albicans* infection.
- Administer antifungal medications or gargles as ordered.

Health education for the person and family

- Rinse the mouth after using the inhaler and maintain good oral hygiene to reduce the risk of fungal infections.
- These medications should not be used to alleviate the symptoms of an acute attack.
- Several weeks of continued therapy may be required before a beneficial effect is noticed.
- Notify the doctor if you develop weight gain, fluid retention, muscle weakness, redistribution of fat or mood changes.

MAST CELL STABILISERS

Cromolyn sodium and nedocromil inhibit inflammatory cells in the airway, blocking early and late responses to inhaled antigens. Both drugs also prevent bronchoconstriction in response to inhaling cold air. These drugs act primarily by stabilising the cytoplasmic membrane of mast cells, preventing the cells from releasing inflammatory mediators

MEDICATION ADMINISTRATION Asthma (continued)

such as histamine. These drugs are used only for preventing asthma attacks, not to treat an acute attack. They are administered by metered-dose inhaler and have a wide margin of safety. Individuals using nedocromil may complain of an unpleasant taste.

Nursing responsibilities

- Evaluate for potential adverse effects of wheezing and bronchoconstriction.

Health education for the person and family

- Gargling or sipping water can decrease the throat irritation associated with nebuliser treatment.
- Use appropriate technique. Inhale deeply with head tipped back to open airways, hold breath and then exhale. Repeat until all the drug dose has been inhaled.
- These drugs are used only to prevent asthma attacks; they are not effective in treating an acute attack.
- Several weeks may be required before a beneficial effect is noted.

LEUKOTRIENE RECEPTOR ANTAGONISTS

Leukotriene receptor antagonists interfere with the inflammatory process in the airways by suppressing the effects of leukotrienes, a group of inflammatory mediators. Leukotrienes are powerful bronchoconstrictors and vasodilators; blocking their synthesis or binding to their receptors improves airflow, decreases symptoms and reduces the need for short-acting bronchodilators. They are used for maintenance therapy in adults and children over the age of 12 as an alternative to inhaled corticosteroid therapy. They are not used to treat an acute attack.

Nursing responsibilities

- Administer at least 1 hour before or 2 hours after meals.
- These drugs inhibit some liver enzymes, affecting the metabolism of warfarin and possibly terfenadine and theophylline. Monitor prothrombin times and theophylline blood levels.
- Monitor liver enzymes, because these drugs may be hepatotoxic.

Health education for the person and family

- Take the drugs as prescribed on an empty stomach.
- Notify the doctor if a change in colour of stools or urine is noted or if jaundice develops.

METHYLXANTHINES

The methylxanthines are central nervous system (CNS) stimulants chemically related to caffeine. These drugs produce bronchodilation through relaxation of bronchial smooth muscle. As CNS stimulants, they produce adverse effects such as nervousness, insomnia and tremors. When administered in large doses, convulsions may result.

Once the drugs of choice for preventing and treating asthma attacks, they are now used primarily to prevent nocturnal asthma. Theophylline has a narrow margin of safety and high potential for toxicity. Because the metabolism and excretion of theophylline vary significantly from person to person—affected by such factors as age, smoking, genetic factors, alcoholism and other chronic diseases—monitoring of serum levels is vital.

Nursing responsibilities

- The therapeutic blood level for theophylline is 10 to 20 mg/L.
- Monitor for manifestations of toxicity. Anorexia, nausea, vomiting, restlessness, insomnia, cardiac arrhythmias and seizures are early manifestations. Other manifestations include epigastric pain, haematemesis, diarrhoea, headache, irritability, muscle twitching, palpitations, tachycardia, flushing and circulatory failure.
- Administer with meals or a full glass of water or milk to minimise gastric irritation.
- Monitor effect closely when administering concurrently with medications such as barbiturates, anticonvulsants, thyroid hormone, β-blockers, bronchodilators and others.

Health education for the person and family

- Oral methylxanthines are ineffective to treat an acute asthma attack; do not delay other treatment by using these drugs.
- Check with the doctor before taking any over-the-counter (OTC) medications or other prescription drugs while on theophylline.
- Do not smoke while using this drug.
- Report adverse effects to the doctor.

CONSIDERATION FOR PRACTICE

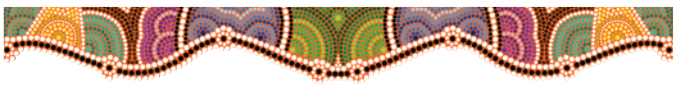
The treatment of chronic health conditions becomes complicated in women of child-bearing age. Before administering *any* medication, the pregnancy category should be assessed (see table below).

PREGNANCY CATEGORY FOR COMMON MEDICATIONS USED IN ASTHMA

Drug	Category	Drug	Category
acetylcysteine	B2	methylprednisolone (IV)	A
beclomethasone	B3	montelukast	B1
budesonide	A	nedocromil	B1
ciclesonide	B3	omalizumab	B1
dexamethasone	A	prednisolone	A
dornase alfa	B1	prednisone	A
eformoterol	B3	salbutamol (MDI)	A
fluticasone	B3	salmeterol	B3
hydrocortisone	A	sodium cromoglycate	A
ipratropium bromide	B1	terbutaline	A
isoprenaline	A	zafirlukast	B1

Sources: Extracted from information in MIMS (2015c). *Full product information—Various drugs.* Retrieved from www-mimsonline-com-au. Reproduced with permission from MIMS Australia, please visit www.tga.gov.au to view its copyright and disclaimer policies; Prescribing medicines in pregnancy database (2015), Therapeutic Goods Administration, used by permission of the Australian Government www.tga.gov.au/hp/medicines-pregnancy.htm.

because of its frequent adverse effects. Many other drugs available have more efficiency and fewer adverse effects attributed to their use. Theophylline should never be used as a first-line drug for the treatment of asthma, but is occasionally used as adjunctive treatment. It relaxes bronchial smooth muscle, inhibits the release of chemical mediators of the inflammatory response and increases diaphragmatic contractility. Theophylline has significantly more adverse effects associated with its use (NACA, 2015).



Nursing care

Nurses encounter individuals with asthma both in the acute care setting during an acute exacerbation and as outpatients or in homes. The priority nursing care needs differ with each setting.

Health promotion

Although specific measures to prevent asthma have not yet been identified, the link between parental smoking and childhood asthma is strong. Discuss this link with young people and families with children. Encourage all individuals to avoid ever starting smoking and, if they already do smoke, to quit. Provide referrals to smoking cessation clinics, help groups or a care provider for nicotine patches as needed to facilitate quitting.

Assessment

Assessment of the person experiencing an acute asthma attack must be very focused and timely.

- *Health history:* current symptoms, including chest tightness, dyspnoea; duration of current attack; measures used to relieve symptoms and their effect; identification of precipitating factors for the attack; frequency of attacks; current medications; known allergies.
- *Physical examination:* apparent level of distress; colour; vital signs; respiratory rate and excursion, breath sounds throughout lung fields; apical pulse.
- *Diagnostic tests:* forced expiratory volume, peak expiratory flow rate; arterial blood gases.

Nursing diagnoses and interventions

An acute asthma attack causes fear as breathing becomes increasingly difficult and hypoxaemia develops. Anxiety in turn tends to increase the severity and manifestations of the attack. Priority nursing care needs during an acute attack focus on improving airway clearance and reducing fear and anxiety. Teaching about prevention of future attacks and home management must be postponed until adequate ventilation is restored.

Ineffective airway clearance

Bronchospasm and bronchoconstriction, increased mucus secretion and airway oedema narrow the airways and impair airflow during an acute attack of asthma. Both inspiratory and expiratory volumes are affected, decreasing the oxygen available at the alveolus for the process of respiration. Narrowed air passages increase the work of breathing, increasing the metabolic rate and tissue demand for oxygen.

- Monitor skin colour and temperature and level of consciousness (LOC). *Cyanosis, cool clammy skin and changes in LOC (agitation, lethargy or confusion) indicate worsening hypoxia.*

CONSIDERATION FOR PRACTICE

Assess respiratory status at least every 1 to 2 hours. Observe respiratory rate and depth, chest movement or excursion, breath sounds and peak expiratory flow rate. Respiratory status can change rapidly during an acute asthma attack and its treatment. Decreasing PEFRs indicate worsening airflow restriction. Slowed, shallow respirations with significantly diminished breath sounds and decreased wheezing may indicate exhaustion and impending respiratory failure. Immediate intervention is necessary.

- Assess ABG results and pulse oximetry readings; notify the doctor of abnormal values or changes in status. *These values provide information about gas exchange and the adequacy of alveolar ventilation. A fall in oxygen saturation levels is an early indicator of impaired gas exchange.*

CONSIDERATION FOR PRACTICE

Assess cough effort and sputum for colour, consistency and amount. Ineffective cough may also signal impending respiratory failure.

- Place in Fowler's, high-Fowler's or orthopnoeic (with head and arms supported on the over-bed table) position to facilitate breathing and lung expansion. *These positions reduce the work of breathing and increase lung expansion, especially of basilar areas.*
- Administer oxygen as ordered. If a mask is used, monitor closely for feelings of claustrophobia or suffocation. *Supplemental oxygen reduces hypoxaemia. Although the mask is a very effective oxygen delivery system, it may increase anxiety.*
- Administer nebuliser treatments and provide humidification as ordered. *Nebuliser treatments are used to administer bronchodilators and other medications; humidity helps loosen secretions.*
- Initiate or assist with chest physiotherapy, including percussion and postural drainage. *Percussion and postural drainage facilitate the movement of secretions and airway clearance.*
- Increase fluid intake. *Increasing fluids helps reduce the viscosity of the secretions.*
- Provide endotracheal suctioning as needed. *Endotracheal suctioning may be necessary to remove secretions and improve ventilation if the person is unable to clear secretions by coughing.*

Ineffective breathing pattern

The physiological changes in lung ventilation that occur during an acute asthma attack impair both lung expansion

and emptying. Anxiety caused by hypoxia and dyspnoea compounds the problem by increasing the respiratory rate. Collaborative and nursing interventions can help restore a more normal breathing pattern and adequate lung ventilation.

- Monitor vital signs and laboratory results. *Tachypnoea, tachycardia, an elevated blood pressure and increasing hypoxaemia and hypercapnia are signs of compromised respiratory status.*

CONSIDERATION FOR PRACTICE

Frequently assess respiratory rate, pattern and breath sounds. Note manifestations of ineffective breathing, including rapid rate, shallow respirations, nasal flaring, use of accessory muscles, intercostal retractions and diminished or absent breath sounds. Early identification of ineffective respirations allows timely initiation of interventions.

- Assist with ADLs as needed. *This conserves energy and reduces fatigue.*
- Provide rest periods between scheduled activities and treatments. *Scheduled rest is important to prevent fatigue and reduce oxygen demands.*
- Teach and assist to use techniques to control breathing pattern:
  a. pursed-lip breathing
  b. abdominal breathing
  c. relaxation techniques, including visualisation and meditation.

  Pursed-lip breathing helps keep airways open by maintaining positive pressure and abdominal breathing improves lung expansion. Relaxation techniques reduce anxiety and its effect on the respiratory rate.
- Administer medications, including bronchodilators and anti-inflammatory drugs, as ordered. Monitor for desired and possible adverse effects. *Medications are used to improve airway status and facilitate breathing.*

Anxiety

Acute exacerbations of asthma can produce significant anxiety. Fear of being unable to breathe and feelings of suffocation associated with acute asthma are significant. Financial or other concerns may cause the person to want to avoid hospitalisation. Increasingly frequent and severe episodes may cause fear for the future. Hypoxia contributes to anxiety as well, stimulating the sympathetic nervous system and the fight-or-flight response.

- Assess level of anxiety. *Interventions for severe anxiety or panic differ from those for mild or moderate anxiety.*
- Assist to identify coping skills that have been successful in the past. *Successful coping helps the person to regain control of the situation, reducing anxiety.*
- Listen actively to concerns; do not deny or negate the fear of dying or of being unable to breathe. *Active listening promotes trust and helps the person express concerns.*
- Include the person in care planning and decisions as appropriate, without making excessive demands. *Participating in decision making increases the person's sense of control. Because high levels of anxiety interfere with the ability to make decisions, it is important to avoid placing demands on the person that may further increase the level of anxiety.*
- Reduce excessive environmental stimuli and maintain a calm demeanour. *This promotes rest.*

CONSIDERATION FOR PRACTICE

Provide physical and emotional support. Remain with the person during episodes of severe anxiety; schedule time every 1 to 2 hours to be with the mildly or moderately anxious person. Answer call lights promptly. The severely anxious person may fear being alone or believe that they will die if someone is not on hand. Knowing that the nurse is readily available and will return quickly reduces anxiety.

CONSIDERATION FOR PRACTICE

Provide clear, concise directions and explanations about procedures. Avoid presenting more information than the person is able to assimilate. Anxiety interferes with the ability to learn. Explanations may need to be repeated frequently.

- Allow supportive family members to remain with the person. *Significant others provide additional support and can help reduce anxiety.*
- Assist to use relaxation techniques, such as guided imagery, muscle relaxation and meditation. *These techniques help restore psychological balance and reduce sympathetic stimulation and responses.*

Ineffective therapeutic regimen management

Once acute asthma is under control and effective respirations have been re-established, it is important to help the person identify contributing factors to the attack. This helps the person prevent future episodes.

- Assess level of understanding about asthma and the prescribed treatment regimen. Provide additional information and teaching as indicated. *Assessment helps to identify and clarify misperceptions and difficulties with disease management.*
- Discuss the person's perception of the illness and its effect on their lifestyle. *Open discussion can help identify conflicts between lifestyle and the treatment regimen.*
- Assist the person and significant others to identify problems or difficulties integrating the treatment regimen into their lifestyle. *Asthma and its management may necessitate lifestyle modifications to prevent acute exacerbations. This can significantly impact on family members—for example, eliminating cigarette smoking or pets from the household, removing carpets or daily damp dusting to remove dust mites.*
- Assess knowledge and understanding of prescribed medications and use of OTC preparations. *This is important to determine misperceptions or possible misuse of medications.*
- Provide verbal and written instructions. *Written instructions reinforce teaching and allow future reference.*

CONSIDERATION FOR PRACTICE

Assist to identify factors that contributed to the acute episode. Identifying contributing factors increases the person's awareness of the disease and of strategies to prevent future exacerbations.

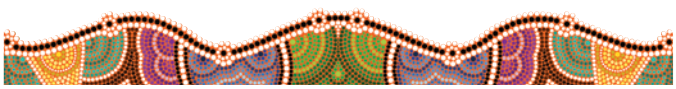
- Refer to counselling, support groups or self-help organisations. *These can help the person and family adapt to living with asthma and the treatment regimen.*

Community-based care

Asthma is a chronic disease that is best managed by the person with assistance from medical personnel. Teaching for home care focuses on promoting the highest level of wellness and preventing and managing acute episodes and exacerbations of the disease. Topics to include in teaching are as follows:

- Suggestions for lifestyle changes to avoid specific triggers for asthma attacks—for example:
  - Warm up slowly before exercising in cold weather; wear a special mask or scarf to retain air warmth and humidity while exercising.
  - Substitute indoor exercises during cold, dry weather.
  - Reduce the risk of respiratory infections (e.g. adequate rest, good nutrition and stress management to maintain immune function, yearly influenza vaccines and immunisation against pneumococcal pneumonia).
  - Use techniques to reduce or manage physical and psychological stress.
- Using peak expiratory flow rate (PEFR) meter to monitor airway status; how to manage the disease based on results.
- Using prescribed medications, including:
  - name, frequency, dose and desired effect
  - potential adverse effects and their management, including effects to report to the doctor
  - potential interactions with other drugs (including OTC herbal preparations) or foods
  - if tolerance is a potential risk, how to identify it and steps to take.

Provide referrals to local or regional resources for further teaching and support as needed. Consider the need for community-based services, home respiratory care services and others as needed.



CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Individuals with chronic airflow obstruction due to chronic bronchitis and/or emphysema are said to have **chronic obstructive pulmonary disease (COPD)**.

Incidence and risk factors

In 2013, approximately 6434 Australians died of COPD (4% of all deaths). Males accounted for 55% of deaths and females 45% (ABS, 2015b). More than one in 20 Australians over 55 years of age has COPD and the financial burden is calculated to cost Australians approximately $929 million annually (AIHW, 2015d). Prevalence data in Australia is under-reported and COPD is thought to be under-diagnosed.

FAST FACTS

- In 2012 in Australia, COPD was the fifth leading cause of death.
- In 2013 in Australia, 52% of all respiratory deaths were from COPD.
- The average length of stay in hospital for COPD-associated issues is 5 days for people aged 55–69 years and 6 days for people over 85 years of age, compared with 3 days for individuals with respiratory tract infections without complications and/or co-morbidities.

Sources: ABS (2015b); AIHW (2015d); Lung Foundation Australia (2015).

Obstructive lung disease typically affects middle-aged and older adults. Cigarette smoking is clearly implicated as the primary cause of COPD. It is estimated that over 70% of deaths in people with COPD are attributed to smoking (Scollo & Winstanley, 2012). Cigarette smoke and the irritants it contains impair ciliary movement, inhibit the function of alveolar macrophages and cause mucus-secreting glands to hypertrophy. Smoke also produces emphysema or airway destruction and constricts smooth muscle, increasing airway resistance. Other contributing factors include air pollution, occupational exposure to noxious dusts and gases, airway infection and familial and genetic factors (see the accompanying 'Genetic considerations' box).

Pathophysiology

COPD is characterised by slowly progressive obstruction of the airways. The disease is one of periodic exacerbations, often related to respiratory infection, with increased symptoms of dyspnoea and sputum production. Unlike acute processes in which lung tissues recover, airways and lung parenchyma do not return to normal following an exacerbation; instead, they demonstrate progressive destructive changes.

Although one or the other may predominate, COPD typically includes components of both chronic bronchitis and emphysema, two distinctly different processes. Small airways disease, narrowing of small bronchioles, is also part of the COPD complex. Through different mechanisms, these processes cause airways to narrow, resistance to airflow to increase and expiration to become slow or difficult (see Figure 36.5). The result is a mismatch between alveolar ventilation and blood flow or perfusion, leading to impaired gas exchange.

Chronic bronchitis

Chronic bronchitis is a disorder of excessive bronchial mucus secretion. It is characterised by a productive cough lasting 3 or more months in 2 consecutive years (Bullock & Hales, 2012). Cigarette smoke is the main factor implicated in the development of chronic bronchitis.

Inhaled irritants lead to a chronic inflammatory process with vasodilation, congestion and oedema of the bronchial mucosa. Goblet cells increase in size and number, and mucous glands enlarge. Thick, tenacious mucus is produced in increased amounts (Mosenifar, 2015). Changes in bronchial squamous cells impair the ability to clear mucus. Narrowed airways and excess secretions

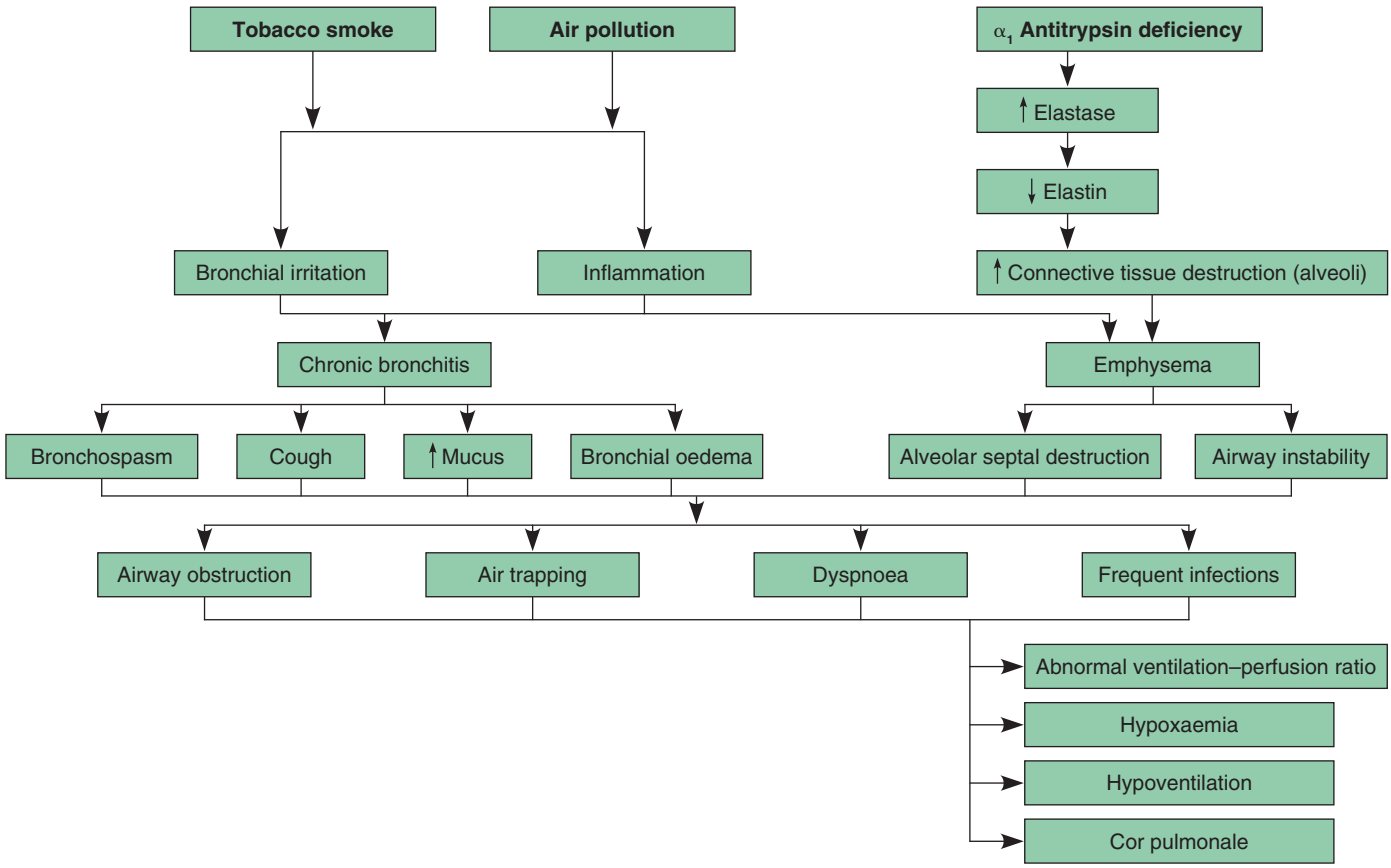


FIGURE 36.5 ■ Pathogenesis of chronic obstructive pulmonary disease

obstruct airflow; expiration is affected first, then inspiration. Because ciliary function is impaired, normal defence mechanisms are unable to clear the mucus and any inhaled pathogens. Recurrent infection is common in chronic bronchitis (Mosenifar, 2015). An imbalance between ventilation and perfusion leads to hypoxaemia, hypercapnia and pulmonary hypertension. Pulmonary hypertension often leads to right-sided heart failure.

Emphysema

Emphysema is characterised by destruction of the walls of the alveoli, with resulting enlargement of abnormal air spaces. As in chronic bronchitis, cigarette smoking is strongly implicated as a causative factor in most cases of emphysema. Deficiency of α_1-antitrypsin, an enzyme that normally inhibits the activity of proteolytic enzymes and tissue destruction in the lungs, contributes to the development of emphysema, especially when combined with exposure to cigarette smoke.

Inflammatory cells that collect in distal airway tissues appear to lead to destruction of elastic fibres in the respiratory bronchioles and alveolar ducts. Alveolar wall destruction causes alveoli and air spaces to enlarge, with loss of corresponding portions of the pulmonary capillary bed. As a result, the surface area for alveolar–capillary diffusion is reduced, affecting gas exchange. Elastic recoil is lost, reducing the volume of air that is passively expired. The loss

GENETIC CONSIDERATIONS Emphysema

Severe α_1-antitrypsin (alpha-1 antitrypsin or ATT) deficiency, present in about 2% of people with COPD, is a proven risk factor for COPD. Normal alpha-1 antitrypsin levels are associated with the common M allele. Two other alleles, the S allele and the Z allele, lead to reduced alpha-1 antitrypsin levels. An estimated one in 2500 Australians carries a single gene associated with alpha-1 antitrypsin deficiency and can pass that gene on to their offspring (Lung Foundation Australia, 2014a). Different concentrations of alpha-1 antitrypsin can be produced, depending on the inherited phenotype (observable characteristic or trait). Approximately 1 in 2500 people in Australia inherits severe alpha-1 antitrypsin deficiency (see Table 36.3).

TABLE 36.3 Concentration of alpha-1 antitrypsin related to phenotype inherited

PHENOTYPE INHERITED	ALPHA-1 ANTITRYPSIN CONCENTRATION
MM	normal
MS	80% of normal
MZ	60% of normal
SZ	40% of normal
ZZ	10% of normal

Source: Lung Foundation Australia (2012a). *Alpha-1 antitrypsin deficiency and lung disease*. Retrieved from http://lungfoundation.com.au/wp-content/uploads/2013/12/Alpha-1-Antitrypsin-Deficiency-and-Lung-Disease1.pdf. Reproduced with permission from Lung Foundation Australia.

of support tissue also affects airways, increasing the risk of expiratory collapse and further air trapping. Anatomically, either respiratory bronchioles or alveoli may be the primary tissue involved.

To summarise, COPD is a progressive, non-reversible process of airway narrowing and loss of supporting tissue. Three separate processes typically are involved:

- chronic bronchitis with persistent airway oedema, excessive mucus production and impaired airway clearance
- emphysema with loss of interstitial membranes and airway support tissue, resulting in airway collapse and loss of alveolar surface area for gas exchange
- small airways disease with bronchoconstriction.

The result of these processes and their combined effects is increased work of breathing, impaired expiration with air trapping and impaired gas exchange.

Manifestations

The clinical presentation of COPD varies from simple chronic bronchitis without disability to chronic respiratory failure and severe disability. Table 36.4 outlines the classifications of COPD severity.

Manifestations are typically absent or minor early in the disease. When the person finally seeks care, productive cough, dyspnoea and exercise intolerance often have been present for as long as 10 years. The cough typically occurs in the mornings and often is attributed to 'smoker's cough'. Initially, dyspnoea occurs only on extreme exertion; as the disease progresses, dyspnoea becomes more severe and accompanies mild activity. Manifestations characteristic of chronic bronchitis and emphysema develop. The clinical features and manifestations of COPD are summarised in Table 36.5.

TABLE 36.4 Classification of COPD by severity

		FEV_1 (% PREDICTED)	SYMPTOMS	HISTORY OF EXACERBATIONS	CO-MORBID CONDITIONS*
COPD SEVERITY	MILD	60–80	Breathlessness on moderate exertion Recurrent chest infections Little or no effect on daily activities	Frequency may increase with severity	Present across all severity groups
	MODERATE	40–59	Increasing dyspnoea Breathlessness walking on level ground Increasing limitation of daily activities Cough and sputum production Exacerbations requiring corticosteroids and/or antibiotics		
	SEVERE	40	Dyspnoea on minimal exertion Daily activities severely curtailed Experiencing regular sputum production Chronic cough		

FEV_1 = forced expiratory volume in 1 second.
*Common co-morbid conditions include cardiovascular disease, skeletal muscle dysfunction, metabolic syndrome, osteoporosis, anxiety or depression, lung cancer, peripheral vascular disease and sleep apnoea.
Source: Lung Foundation Australia (2014b). *The COPD-X: Concise Guide for Primary Care*. Retrieved from http://lungfoundation.com.au/wp-content/uploads/2015/06/LFA-COPD-X-Concise-Guide-for-Primary-Care.pdf. Reproduced with permission from Lung Foundation Australia.

TABLE 36.5 Clinical features and manifestations of COPD

	FEATURE	CHRONIC BRONCHITIS	EMPHYSEMA
History	Onset	After age 35; recurrent respiratory infections	After age 50; insidious progressive dyspnoea
	Smoking	Usual	Usual
	Cough	Persistent, productive of copious mucopurulent sputum	Absent or mild with scant clear sputum, if any
Physical examination	Appearance	Often obese; oedematous and cyanotic; distended neck veins and other symptoms of right-sided heart failure	Usually thin and cachectic; barrel chest; prominent accessory muscles of respiration
	Chest	Adventitious sounds with wheezing and rhonchi; normal percussion note	Distant or diminished breath sounds; hyper-resonant percussion note
Other features	Blood gases	Hypercapnia and hypoxaemia; respiratory acidosis	Normal or mild hypoxaemia; normal pH
	Pulmonary function studies	Normal or decreased total lung capacity; moderately increased residual volume	Increased total lung capacity; markedly increased residual volume
	Pulmonary hypertension	May be severe	Only when advanced

Manifestations of chronic bronchitis are a productive cough with copious amounts of thick, tenacious sputum, cyanosis and evidence of right-sided heart failure, including distended neck veins, oedema, liver engorgement and an enlarged heart. Adventitious lung sounds, including loud rhonchi and possible wheezes, are prominent on auscultation.

Emphysema is insidious in onset. Dyspnoea is the initial symptom. Initially occurring only with exertion, dyspnoea may progress to become severe even at rest. Cough is minimal or absent. Air trapping and hyperinflation increase the anterior–posterior chest diameter, causing *barrel chest*. The person often is thin, tachypnoeic, uses accessory muscles of respiration and often assumes a position of sitting and leaning forward (see Figure 36.6). The expiratory phase of the respiratory cycle is prolonged. On auscultation, breath sounds are diminished and the percussion tone is hyper-resonant.

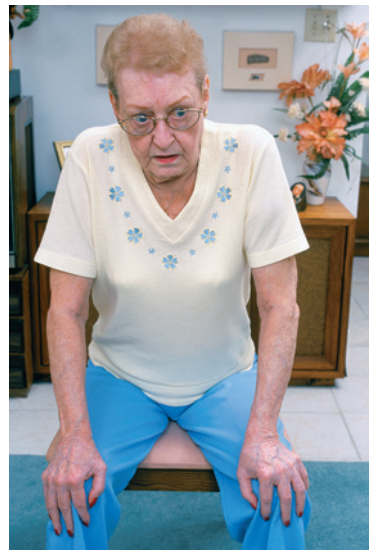


FIGURE 36.6 ■ Typical appearance of a person with emphysema. Note the person's anxious expression and assumption of the tripod position, leaning forward with the hands on the knees

Source: Courtesy of Michal Heron/Pearson Education/PH College.

INTERPROFESSIONAL CARE

Although COPD can be prevented in most people, it cannot be cured. Smoking abstinence is the only certain way to prevent COPD and to slow its progression. To a certain extent, airway obstruction can be reversed and disability minimised early in the disease. Treatment generally focuses on relieving symptoms, minimising obstruction and slowing disability.

Diagnosis

Diagnostic tests are used to help establish the diagnosis of chronic obstructive pulmonary disease and identify the predominant component, emphysema or chronic bronchitis. These procedures also are used to assess respiratory status and monitor treatment effectiveness. See Chapter 33 for more information about these diagnostic procedures and related nursing care.

- *Respiratory function testing* is performed to establish the diagnosis and evaluate the extent and progress of COPD (see Box 33.1). Results are based on calculated norms for each person by age, height, sex and weight; note these as well as all current medications on the requisition. In COPD, the total lung capacity and residual volume typically are increased. The forced expiratory volume (FEV_1) and forced vital capacity (FVC) are decreased due to narrowed airways and resistance to airflow (see Figure 36.7).
- *Ventilation–perfusion scanning* may be performed to determine the extent of ventilation–perfusion mismatch—that is,

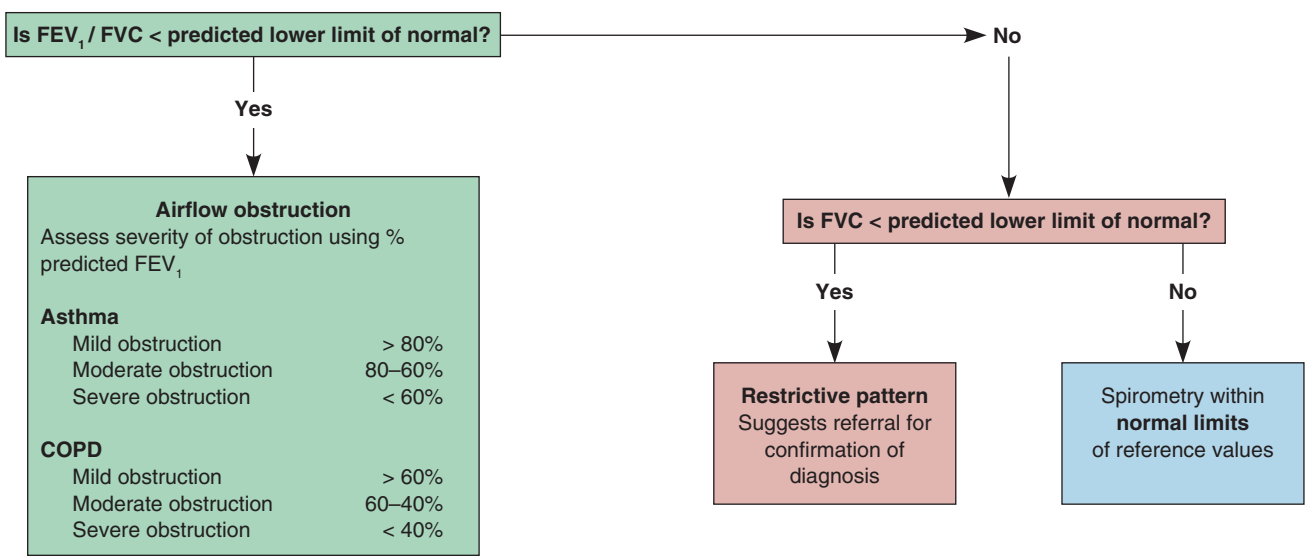


FIGURE 36.7 ■ Guideline for spirometry interpretation

Source: Johns, D., Burton, D. & Swanney, M. (2015). *Spirometer users' and buyers' guide.* Melbourne: National Asthma Council Australia. © National Asthma Council Australia. Retrieved from www.nationalasthma.org.au/uploads/content/209-Spirometer-Users-and-Buyers-Guide-2015-updated.pdf.

the extent to which lung tissue is ventilated but not perfused (dead space) or perfused but inadequately ventilated (physiological shunting) (see Figure 36.8). A radioisotope is injected or inhaled to illustrate areas of shunting and absent capillaries.

- *Serum α_1-antitrypsin (alpha-1 antitrypsin) levels* may be drawn to screen for deficiency, particularly in people with a family history of obstructive airway disease, those with an early onset, women and non-smokers. Normal adult serum alpha-1 antitrypsin levels range from 90 to 200 mg/dL. Fasting is not required prior to this test.
- ABGs are drawn to evaluate gas exchange, particularly during acute exacerbations of COPD. Individuals with predominant emphysema often have mild hypoxaemia and normal or low carbon dioxide tension. Respiratory alkalosis may be present due to an increased respiratory rate. Predominant chronic bronchitis and airway obstruction may cause marked hypoxaemia and hypercapnia with respiratory acidosis. Oxygen saturation levels are low due to marked hypoxaemia.
- *Pulse oximetry* is used to monitor oxygen saturation of the blood. Marked airway obstruction and hypoxaemia often cause oxygen saturation levels of less than 95%. Pulse oximetry may be continuously monitored to assess the need for supplemental oxygen.
- *Exhaled carbon dioxide (capnogram* or $ETCO_2$) may be measured to evaluate alveolar ventilation. The normal $ETCO_2$ reading is 35 to 45 mmHg; it is elevated when ventilation is inadequate and decreased when pulmonary perfusion is impaired. $ETCO_2$ monitoring can reduce the frequency of ABG determinations.
- *Full blood count (FBC) with white blood cell (WBC) differential* often shows increased red blood cells (RBCs) and haematocrit (erythrocytosis) as chronic hypoxia stimulates increased erythropoiesis to improve the oxygen-carrying capacity of the blood. *Polycythaemia*, increased numbers of all blood cells, may be evident. Increased WBC count and a higher percentage of immature WBCs (bands) are often indicative of bacterial infection.
- *Chest x-ray* may show longer lung fields and flattening of the diaphragm due to hyperinflation and evidence of pulmonary infection if present. The anterior–posterior (AP) diameter is wider (see Figure 36.9).

CONSIDERATION FOR PRACTICE

Hypercapnia (elevated $PaCO_2$ levels) is often chronic in individuals with COPD (CO_2 retainers). In these individuals, administering excessive oxygen flows can actually reduce respiratory rate which results in increased $PaCO_2$, potentially leading to somnolence and acute respiratory failure. While oxygen is the drug of choice for treating individuals with COPD, close monitoring is necessary during oxygen therapy.

Quitting smoking

Smoking cessation can not only prevent COPD from developing, but can also improve lung function once the disease has

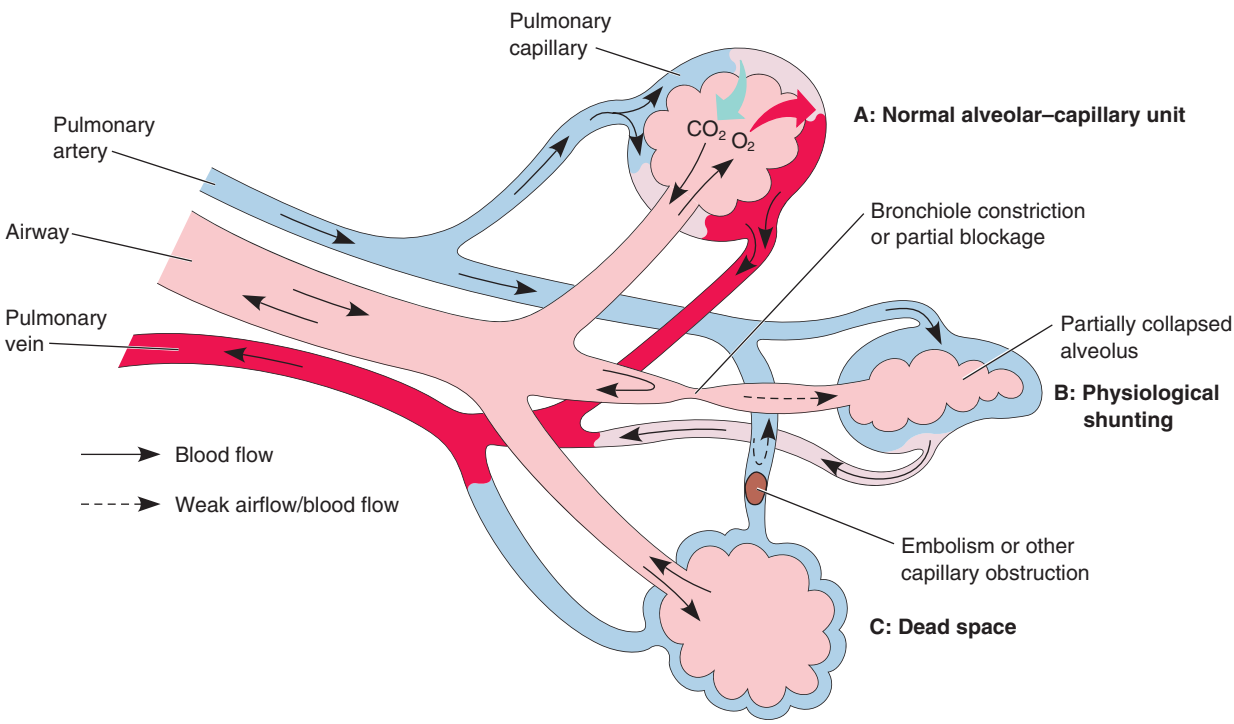


FIGURE 36.8 ■ Ventilation–perfusion relationships. *A*, Normal alveolar–capillary unit with an ideal match of ventilation and blood flow. Maximal gas exchange occurs between alveolus and blood. *B*, Physiological shunting: a unit with adequate perfusion but inadequate ventilation. *C*, Dead space: a unit with adequate ventilation but inadequate perfusion. In the latter two cases, gas exchange is impaired

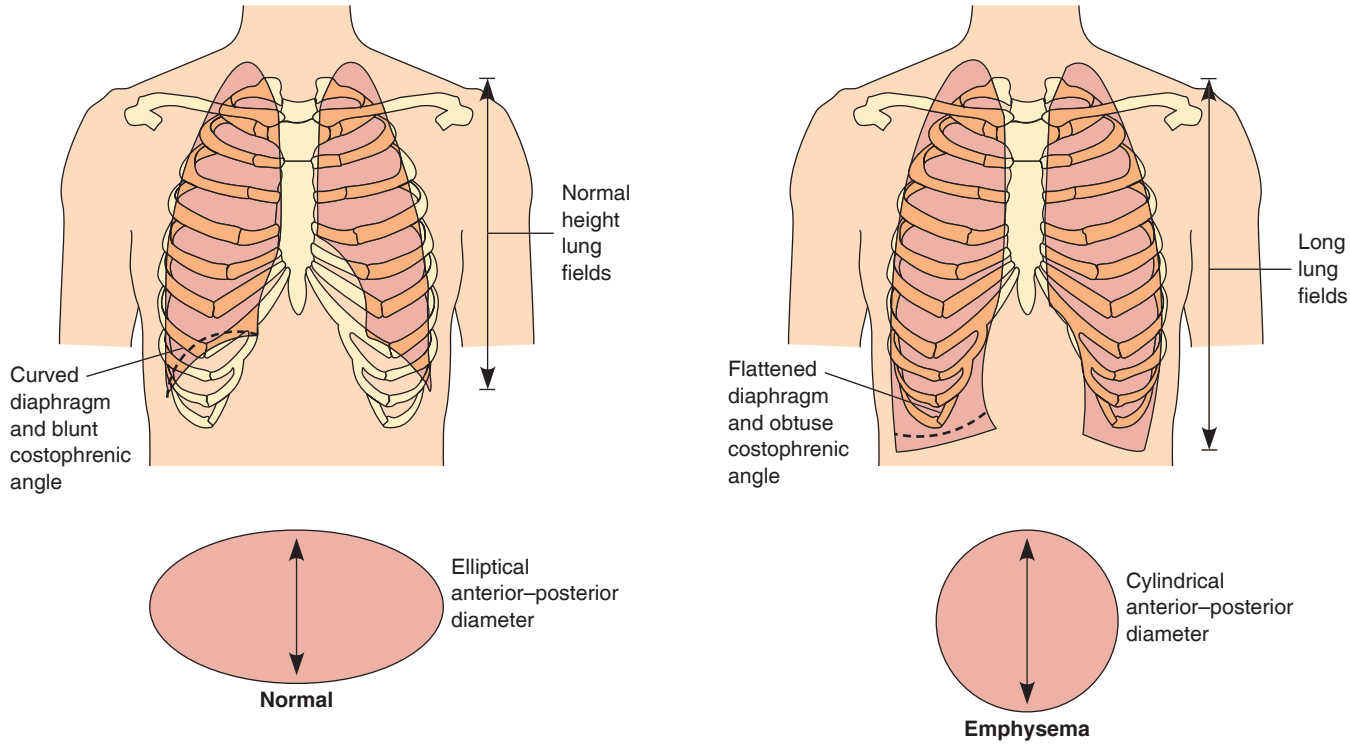


FIGURE 36.9 ■ Anatomical changes related to emphysema

been diagnosed. FEV_1 improves and survival is prolonged, largely due to lower rates of lung cancer and heart disease. Sustained quitting is difficult; however, various methods may assist people.

- Nicotine replacement therapy can increase the rate of quitting by up to 70%.
- Partial nicotine agonists such as varenicline can increase the rate of quitting by up to 50%.
- The non-nicotine oral therapy bupropion can improve the rate of quitting more than a placebo but less successfully than varenicline.
- Group counselling can improve quitting success above minimal support.
- Brief motivational advice from healthcare professionals has a success rate of 3–6%.
- Interventions such as hypnotherapy, acupuncture, naltrexone, aversive smoking, biomedical feedback, electronic cigarettes, physical activity, the Alan Carr method or the consumption of St John's wort (*Hypericum perforatum*) herb extract do not currently demonstrate sufficient good-quality empirical evidence to support their success (Zwar et al., 2014).

Medications

Immunisation against pneumococcal pneumonia and influenza is recommended yearly to reduce the risk of respiratory infections. If the infection is considered to be bacterial, a broad-spectrum antibiotic is prescribed. Antibiotics should be administered in times of exacerbation. However, prophylactic antibiotics, although they have a statistically significant reduction in number of sick days experienced, do not have a place in routine medication regimens as the risk of increasing antibiotic resistance is too great (Abramson et al., 2014).

Bronchodilators improve airflow and reduce air trapping in COPD, resulting in improved dyspnoea and exercise tolerance. Bronchodilators may be given by MDI, DPI, nebuliser or orally. Oral administration may promote adherence, but is associated with much higher rates of adverse effects. A spacer may facilitate effective use of an MDI. Ipratropium bromide, an anticholinergic agent administered by MDI, is frequently prescribed. It has a longer duration of action than the short-acting β_2-adrenergic stimulant bronchodilators and few side effects. Salmeterol, a longer-acting β_2-agonist, may be used in combination therapy. Bronchodilators are discussed in further detail in the section on asthma and their nursing implications are outlined in the 'Medication administration' box earlier in this chapter.

Corticosteroid therapy may be used when asthma is a major component of COPD. It also improves symptoms and exercise tolerance and may reduce the severity of exacerbations and the need for hospitalisation. Oral corticosteroids, such as prednisone, are used initially. If a beneficial response occurs, the amount is reduced to the lowest effective dose. Every-other-day dosing or administration by inhaler is preferred to minimise steroid side effects, such as cushingoid effects and an increased risk of osteoporosis and vertebral fractures.

Alpha-1 antitrypsin replacement therapy is not common practice in Australia.

Treatments

In addition to refraining from smoking, exposure to other airway irritants and allergens should be avoided. The person should remain indoors during periods of significant air pollution to prevent exacerbations of the disease. Air-filtering systems or air conditioning may be useful.

Respiratory hygiene measures, including hydration, effective coughing, percussion and postural drainage, are used to improve clearance of airway secretions. Maintaining adequate systemic hydration is essential to keep secretions thin. Forceful coughing is often less effective than leaning forward and repeatedly 'huffing', with relaxed breathing between huffs. Percussion and postural drainage may be necessary if the person is unable to clear secretions by usual means. Cough suppressants and sedatives generally are avoided because they may cause retention of secretions.

Unless disabling cardiac disease is present, a regular exercise program is beneficial for:

- improving exercise tolerance
- enhancing ability to perform ADLs
- preventing deterioration of physical condition.

A program of regular aerobic exercise designed to gradually increase exercise tolerance is recommended. Activities that strengthen the muscles used for breathing and ADLs, such as swimming and golf, also are beneficial.

Breathing exercises are used to slow the respiratory rate and relieve accessory muscle fatigue. Pursed-lip breathing slows the respiratory rate and helps maintain open airways during exhalation by keeping positive pressure in the airways. Abdominal breathing relieves the work of accessory muscles of respiration.

OXYGEN Long-term oxygen therapy is used for severe and progressive hypoxaemia. Oxygen therapy improves exercise tolerance, mental functioning and quality of life in advanced COPD. It also reduces the rate of hospitalisation and increases length of survival. Oxygen may be used intermittently, at night or continuously. For severely hypoxaemic individuals, the greatest benefit is seen with continuous oxygen. Home oxygen may be supplied as liquid oxygen, compressed gas cylinders or oxygen concentrators.

An acute exacerbation of COPD may necessitate oxygenation and inspiratory positive-pressure assistance with a face mask or intubation and mechanical ventilation. Oxygen administered without intubation and mechanical ventilation requires caution: administering oxygen to individuals with chronic elevated carbon dioxide levels in the blood can actually increase the $PaCO_2$, leading to increased somnolence and even respiratory failure. Close monitoring of LOC and ABGs during oxygen therapy is vital (Mosenifar, 2015).

Surgery

When medical therapy is no longer effective, lung transplantation may be an option; however, it has been shown to have limited survival benefit (Abramson et al., 2014). Lung reduction surgery is an experimental surgical intervention for advanced diffuse emphysema and lung hyperinflation. The procedure reduces the overall volume of the lung, reshapes it and improves elastic recoil. As a result, respiratory function and exercise tolerance improve and dyspnoea is reduced. See the box in Chapter 35 for nursing care of the person undergoing lung surgery. Special nursing care considerations related to lung or heart–lung transplant are summarised in Box 36.2.

Nursing care

Health promotion

Not smoking—never starting or quitting—is the best preventive measure for chronic obstructive pulmonary disease. Even in individuals with COPD, smoking cessation improves lung function and increases survival. Educate all individuals, including preschool and school-aged children, about the risks of smoking (see Box 36.3).

Assessment

Focused assessment for the person with chronic obstructive pulmonary disease includes:

- *Health history:* current symptoms, including cough, sputum production, dyspnoea, activity tolerance; frequency of respiratory infections and most recent episode; previous diagnosis of emphysema, chronic bronchitis or asthma; current medications; smoking history (in pack years—packets per day times number of years smoked), history of exposure to second-hand smoke, occupational or other pollutants.
- *Physical examination:* general appearance, weight for height, mental status; vital signs, including temperature; skin colour and temperature; anterioposterior and lateral chest diameter, use of accessory muscles, nasal flaring or pursed-lip breathing; respiratory excursion and diaphragmatic excursion; percussion tone; breath sounds throughout; neck veins, apical pulse and heart sounds, peripheral pulses, oedema.
- *Diagnostic tests:* FVC and FEV_1, ABGs, haematocrit.

Nursing diagnoses and interventions

Individuals with chronic obstructive pulmonary disease, whether hospitalised or in the community, have multiple nursing care needs. Because of the obstructive nature of the disease, airway clearance is a high priority. Nutritional deficit is common, particularly when emphysema is predominant. Because this chronic disease affects all functional health patterns, psychosocial issues are also of concern in planning nursing care. In addition to the nursing diagnoses presented here, see the nursing care plan that follows.

Ineffective airway clearance

Both chronic bronchitis and emphysema affect the ability to maintain open airways. In chronic bronchitis, copious amounts of thick, tenacious mucus are produced. Ciliary action is impaired, making it difficult to clear mucus from the airways.

BOX 36.2 Nursing considerations related to lung transplant

While immediate postoperative care for individuals undergoing lung or heart–lung transplant is provided by specially trained interprofessional teams in transplant centres, increased survival following transplant means that these individuals are more commonly seen in community-based settings, non-transplant units and on general nursing care wards. An understanding of common post-transplant complications and care needs of the person following transplant facilitates appropriate nursing care.

Common post-transplant complications

In the early post-transplant period, the most common complications relate to the surgical procedure itself or to rejection of the transplanted organ(s).

- *Rejection*. Acute organ rejection can occur at any time following the transplant. An acute change in FEV_1 and FVC on home spirometry is often the first indication of acute rejection. Other manifestations of rejection include fever, dyspnoea and an elevated WBC count. Because these manifestations are similar to those of infection, the person is instructed to contact their doctor or transplant coordinator for further investigation. Acute rejection is treated with increased corticosteroids and adjustment of the immunosuppressive regimen (see Chapter 12). Chronic rejection is less amenable to therapy, ultimately necessitating retransplant.
- *Infection*. Prevention of infection is vital in a person following lung transplantation. Individuals are encouraged to reduce their risk of infection by avoiding contact with people who have an infectious disease (e.g. upper respiratory tract infection (URTI), shingles, diseases of childhood). The transplant recipient may not have typical manifestations of infection due to immunosuppression. Any vague symptoms with or without fever or leucocytosis are investigated. Recurrent viral infections such as cytomegalovirus have been associated with chronic rejection; hence, they are aggressively treated with antiviral therapy. Treatment of other infections is targeted to the infectious organism.

Nursing considerations for the person following transplant

Reverse isolation procedures are not necessary unless the neutrophil count is very low ($< 500/mm^3$). Use good handwashing and standard precautions at all times and aseptic technique for dressing changes, IV starts and site care, and other invasive procedures (such as urinary catheterisation). Do not allow caregivers or visitors with URTI to have contact with the person; a mask may be provided for short visits if contact is unavoidable. Skin surveillance and care is vital following transplant. Intact skin reduces the risk of infection; however, corticosteroid therapy increases the risk of skin tears and breakdown.

The effect of all medications on immunosuppressive therapy and the transplanted organ(s) should be carefully investigated prior to administration. Some antibiotics and other drugs can affect blood levels of immunosuppressants.

Particular attention must be paid to respiratory hygiene. Denervation of the transplanted lung eliminates the usual cough stimuli (Whitson, 2015). Regularly scheduled coughing and deep breathing, and the use of vibration, percussion and postural drainage, are important to prevent accumulation of secretions (Goodman & Fuller, 2015).

BOX 36.3 Cigarette smoking and tobacco use

The use of tobacco reaches back to early civilisations when it was used in religious ceremonies and as an offering of friendship. At one time tobacco was thought to have medicinal qualities effective against all common diseases. Widespread use of tobacco in the male population of the industrialised world began during World War I.

Tobacco is now recognised as the leading cause of preventable illness in the world. Diseases directly related to tobacco use are responsible for the deaths of more than 19 000 Australians every year (Department of Health and Ageing, 2012). In spite of this knowledge, aggressive marketing of the product continues and its worldwide use is increasing, especially in developing countries.

The link between tobacco use and lung cancer was reported as early as 1912. Today, 90% of lung cancer in men and 65% in women is considered to be caused by smoking tobacco (Cancer Australia, 2015). The World Health Organization (WHO) reported that tobacco smoke contributes to the death of 6 million people a year worldwide (WHO, 2015).

Cigarette smoke contains over 4000 chemicals (more than 50 of which are known to cause cancer), including nicotine (WHO, 2015). Nicotine is a highly addictive psychoactive substance that is relatively cheap and readily available. It produces euphoria, which acts as a positive reinforcer for continued use.

Tar is the particulate matter in cigarette smoke that is responsible for most of its carcinogenic and pathological effects on the lungs. Smoke also paralyses the cilia, reducing their ability to remove the tar. The risk of cancer and other lung diseases is dose related, affected by the age at which smoking began, the number of cigarettes smoked per day and the number of years smoked. Smoking cessation reduces the risks associated with tobacco use. For some, such as those at risk of coronary heart disease, quitting smoking yields rapid benefits. For others, the degree of risk reduction is less immediate, but still significant.

Nurses need to do more than simply advise individuals to quit smoking and talk about the risks of smoking. Nurses can take an active role in smoking cessation. Identify smoking habits, smoking-related illnesses and previous efforts to

(continued)

BOX 36.3 Cigarette smoking and tobacco use (continued)

quit. Work with the person to identify barriers and obstacles to quitting. Educate about the addictive nature of nicotine and explain the manifestations of nicotine withdrawal (anxiety, irritability, headache and disturbed sleep). Develop a plan with the individual that specifies a target date to quit and includes ways to deal with obstacles to quitting, withdrawal symptoms and the temptation to resume smoking. Offer self-help material at an appropriate reading level. Refer to a counsellor, doctor, self-help group or smoking cessation clinic. If a relapse occurs, accept it as a normal part of rehabilitation from any addictive substance. Continue to provide support and encouragement, helping the person to avoid further relapses.

Nurses can be especially effective in primary prevention of cigarette smoking and the diseases associated with it. Just as tobacco companies direct advertising at women and teens, nurses can target these populations and younger children for programs to prevent smoking. In addition, nurses need to become active in reducing minors' access to tobacco products, especially cigarettes.

Nursing diagnoses that may be appropriate related to smoking include the following:

- *Ineffective maintenance of health* related to tobacco use evidenced by cough, haemoptysis, or shortness of breath.
- *Decisional conflict* related to tobacco use evidenced by inability to determine a 'quit smoking date'.
- *Ineffective denial* related to substance abuse and dependence evidenced by statements discounting health concerns related to tobacco smoking.

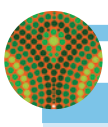
The loss of supporting tissue caused by emphysema increases the risk of airway collapse. In both cases, air is trapped distally and less oxygen is available to the alveoli for diffusion. Normal respiratory defence mechanisms are impaired and mucus-plugged airways provide an ideal environment for bacterial growth. Respiratory infection further impairs airway clearance and is often the cause of an acute exacerbation.

- Assess respiratory status every 1 to 2 hours or as indicated. Assess rate and pattern; cough and secretions (colour, amount, consistency and odour); and breath sounds, both normal and adventitious. *Frequent assessment is vital to monitor current status and response to treatment. Adventitious sounds should decrease with effective intervention. Diminished or absent breath sounds may indicate increasing airway obstruction and possible atelectasis.*

CONSIDERATION FOR PRACTICE

Promptly report changes in oxygen saturation, skin colour or mental status. A drop in oxygen saturation levels, increasing cyanosis or altered LOC indicate hypoxaemia, which may be related to airway obstruction.

- Monitor ABG results. *Increasing hypoxaemia, hypercapnia and respiratory acidosis may indicate increasing airway obstruction.*
- Weigh daily, monitor intake and output, and assess mucous membranes and skin turgor. *Dehydration causes respiratory secretions to become thicker, more tenacious and difficult to expectorate; fluid overload can further compromise respiratory status.*



TRANSLATION TO PRACTICE Evidence-based practice: community nurse mentoring improves self-management in people with COPD

A Tasmanian controlled study by Wood-Baker et al. in 2012 demonstrated that a community nurse can facilitate improved quality of life and early healthcare utilisation to reduce the risk of exacerbation in people with chronic obstructive pulmonary disease (COPD). The group investigated the effectiveness of a program including home visits, the development of a behaviour-specific, reasonable and measureable action plan, and telephone follow ups. People with COPD over 45 years of age without other lung diseases or unstable cardiac, renal or liver disease, and with an anticipated survival rate of greater than 12 months, were included. Results demonstrated that increased quality of life and reduced time to death and readmission can be achieved through well-designed community nurse mentoring programs.

IMPLICATIONS FOR NURSING

Years of life lost, readmission for exacerbation and reduced quality of life are serious social and financial costs of COPD. Programs which directly facilitate better outcomes in any of these factors should be considered beneficial in the management of individuals with COPD. It would be reasonable to consider that financial gains from reducing exacerbation and improving a person's quality of life would easily offset costs of such a program. Among other things, this study reaffirmed the fact that community nursing support appears to be an important factor in the risk of readmission for exacerbation.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Identify factors that may be important in such a program. Consider the practicalities of offering such a program including necessary content, volume and type of support, and logistical issues such as provision of support for individuals in rural and remote regions.
2. Which factors would be important to measure in order to determine an improvement in a person with COPD?
3. People with COPD generally have co-morbid issues. What implications would cardiac, renal or hepatic disease have on an individual with COPD?

- Encourage a fluid intake of at least 2000 to 2500 mL per day unless contraindicated. *Adequate fluid intake helps keep mucus secretions thin.*
- Place in Fowler's, high-Fowler's or orthopnoeic position; encourage movement and activity to tolerance. *Upright positions improve ventilation and reduce the work of breathing. Activity helps mobilise secretions and prevent them from pooling.*
- Assist with coughing and deep breathing at least every 2 hours while awake. Position seated upright, leaning forward during coughing. *The upright position promotes chest expansion, increasing the effectiveness of coughing and reducing the work involved.*
- Provide tissues and a paper bag to dispose of expectorated sputum. *This important infection control measure reduces the spread of respiratory organisms to other people.*
- Refer to a respiratory therapist and assist with or perform percussion and postural drainage as needed. *Percussion helps loosen secretions in airways; postural drainage facilitates movement of these secretions out of the respiratory tract.*
- Provide rest periods between treatments and procedures. *The person with COPD fatigues easily; adequate rest is important to conserve energy and reduce fatigue.*
- Administer expectorant and bronchodilator medications as ordered. Correlate timing with respiratory treatments. *Using expectorants and bronchodilators prior to coughing, percussion and postural drainage increases their effectiveness in clearing airways.*

CONSIDERATION FOR PRACTICE

Provide endotracheal, oral or nasopharyngeal suctioning as necessary. Suctioning may be necessary to stimulate cough and help clear secretions.

- Provide supplemental oxygen as ordered. *Supplemental oxygen helps maintain adequate blood and tissue oxygenation.*

CONSIDERATION FOR PRACTICE

Prepare for intubation and mechanical ventilation if respiratory status deteriorates (increasing hypoxaemia and hypercapnia, decreased LOC, cyanosis or worsening airway obstruction). Respiratory failure is a possible complication of an acute exacerbation of COPD and requires immediate intervention to preserve life.

NURSING CARE PLAN A person with COPD

Anna Mercurio is an 83-year-old widow who lives with her two adult sons. During the past 15 years, Mrs Mercurio has become increasingly short of breath while gardening and walking, two favourite activities. She also has developed a chronic cough that is particularly bad in the mornings. Ten years ago her family doctor told her that she had emphysema. She is admitted to the hospital with possible pneumonia and acute exacerbation of COPD.

ASSESSMENT

Mrs Mercurio was admitted to the medical unit. In the nursing history, the nurse notes that she denies ever smoking, but says that her husband and two sons have been smokers 'for practically their whole lives'. She says she lived an active life before developing lung disease, but her breathing difficulties and cough have progressed so that she now must rest after just a few minutes of housework or other activity. Her cough is productive of moderate to large amounts of sputum, particularly in the mornings. She developed increasing shortness of breath and sputum 2 days ago; this morning, she could not complete her morning activities without resting, so she contacted her doctor.

On physical examination, Mrs Mercurio demonstrated the following: skin very warm and dry, colour dusky. Pauses frequently while speaking to breathe. Respiratory rate 36, fairly shallow; coughs frequently, producing large amounts of thick, tenacious green sputum. Other vital signs: P 115 and irregular, BP 186/60, T 39°C. Appears very thin; weight 43.6 kg, height 160 cm. Anteroposterior and lateral chest diameter increased; moderate kyphosis noted. Chest hyperresonant to percussion. Auscultation reveals distant breath sounds with scattered wheezes and rhonchi throughout lung fields. Chest x-ray shows flattening of diaphragm, long lung fields, slight cardiac enlargement, prominent vascular and bronchial markings, and patchy infiltrates. Initial laboratory work reveals moderate erythrocytosis, leucocytosis and low serum albumin. Arterial blood gas results: pH 7.19; PaO_2 54 mmHg; $PaCO_2$ 59 mmHg; HCO_3^- 30 mg/dL and O_2 saturation 88%. Admitting orders include sputum specimen for culture; intravenous penicillin G, 2 million units every 4 hours; salbutamol/ipratropium inhaler, two puffs every 6 hours; salmeterol/fluticasone dry powder inhaler, twice a day; bed rest with bathroom privileges; oxygen per nasal prongs at 2 L/min continuously; and regular diet.

DIAGNOSES

- *Ineffective airway clearance* related to pneumonia and COPD evidenced by increased shortness of breath and respiration rate.
- *Impaired gas exchange* related to acute and chronic lung disease evidenced by low oxygen saturations.
- *Risk of impaired ventilation* related to loss of hypoxaemic respiratory drive and respiratory muscle fatigue evidenced by decreasing respiration rate and oxygen saturations.
- *Impaired home maintenance* related to activity intolerance evidenced by inability to undertake activities of daily living.

PLANNING

- Closely monitor response to oxygen therapy, including skin colour, oxygen saturation, sputum consistency and respiratory drive.

(continued)

NURSING CARE PLAN A person with COPD (continued)

- Assess respiratory status and LOC every 1 to 2 hours until stable, then at least every 4 hours.
- Contact respiratory therapy for percussion and postural drainage following inhaler treatments.
- Meet with Mrs Mercurio and her sons to develop a post-discharge care plan.
- Refer to home health department for nursing follow up.
- Refer to social services for possible assistance with home maintenance.

Expected outcomes

- Expectorate secretions effectively.
- Return to level of respiratory function prior to acute exacerbation.
- Demonstrate improved ABG and oxygen saturation values.
- Maintain spontaneous respirations without excess fatigue.
- Verbalise willingness to allow sons or a housekeeper to assist with daily household tasks.

IMPLEMENTATION

- Increase fluid intake to at least 2500 mL per day and provide bedside humidifier.
- Elevate head of bed to at least 30 degrees at all times.
- Teach 'huff' coughing technique.
- Administer medications as ordered, providing ipratropium inhaler before beclomethasone inhaler. Provide mouth care after inhalers.
- Provide for uninterrupted rest periods following treatments and procedures.

EVALUATION

After the first day in the hospital Mrs Mercurio's condition begins to improve slowly. On discharge 6 days later she is able to provide self-care with less fatigue and dyspnoea. She is using oxygen at night only, admitting that it is only for security. Although a few scattered wheezes and rhonchi are still present in her lungs, Mrs Mercurio's sputum is thinner, white and easily expectorated. She will continue taking oral penicillin V for an additional 10 days at home. She will also continue using the inhalers as prescribed. Although Mrs Mercurio's sons admit they will probably never be able to quit smoking, they have agreed to smoke only in the garage or outside. A community health nurse will initially evaluate Mrs Mercurio's progress three times weekly. Arrangements have been made for a housekeeper to come twice a week for cleaning and laundry. Mrs Mercurio is glad to be returning home and grateful for the arrangements that have been made.

CRITICAL THINKING IN THE NURSING PROCESS

1. Mrs Mercurio has never been a smoker but has had long-term exposure to second-hand smoke. How does second-hand smoke contribute to lung diseases in adults and children?
2. The nursing care plan included the nursing diagnosis *Risk of impaired ventilation* related to loss of hypoxaemic respiratory drive and respiratory muscle fatigue. Identify the normal physiological events that stimulate breathing and describe how these differ for the person with chronic hypoxaemia and hypercapnia.
3. The person with an acute exacerbation of COPD is at risk of respiratory failure. Which changes in Mrs Mercurio's assessment findings could indicate this complication?
4. Develop a nursing care plan for Mrs Mercurio for the nursing diagnosis *Deficient diversional activities* related to inability to continue preferred activities.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will take into your future practice.
2. In individuals experiencing acute exacerbations, extreme shortness of breath can result in an inability to speak in sentences of more than a couple of words. How might this impact on communication and the ability to collect an adequate detailed health history? How should you prioritise the information required at that point in time? Which techniques would you use to facilitate adequate communication without further compromising a person's oxygenation?

Inadequate nutrition

With advanced COPD, minimal activity, including eating, can cause fatigue and dyspnoea. The person may be unable to consume a full meal without resting. At the same time, the increased work of breathing increases metabolic demands and more kilojoules are required. The person may appear cachectic (thin and wasted). Poor nutritional status further impairs immune function and increases the risk of a complicating infection.

- Assess nutritional status, including diet history, weight for height (use reference tables of desired weights) and anthropometric (skinfold) measurements. *It is important to differentiate nutritional status from body type rather than assume a nutritional impairment.*
- Observe and document food intake, including types, amounts and kilojoule intake. *This information can provide direction for supplementation, if needed.*
- Monitor laboratory values, including serum albumin and electrolyte levels. *These values provide information about the adequacy of nutritional intake, including protein.*
- Consult with a dietitian to plan meals and nutritional supplements that meet kilojoule needs. More concentrated sources of high-energy foods may be required to maintain kilojoule intake without excess fatigue. *A diet high in proteins and fats without excess carbohydrates is recommended to minimise carbon dioxide production during metabolism. (Carbohydrates are metabolised to form CO_2 and water.)*
- Provide frequent, small feedings with between-meal supplements. *Frequent, small meals help maintain intake and reduce fatigue associated with eating.*

- Place seated or in high-Fowler's position for meals. *An upright position promotes lung expansion and reduces dyspnoea.*
- Assist to choose preferred foods from the menu; encourage family members to bring food from home if allowed. *Providing preferred foods encourages eating.*
- Keep snacks at the bedside. *Snacks provide additional kilojoule intake.*
- Provide mouth care prior to meals. *This helps enhance the appetite.*
- If unable to maintain oral intake, consult with the doctor about enteral or parenteral feedings. *Maintenance of kilojoule and nutrient intake is vital to prevent catabolism.*

Compromised family coping

Chronic illness affects the entire family structure. Roles and relationships change; additional demands are placed on the family. Family members may blame the person for causing the illness and may have distorted perceptions about it, even denying its existence. They may refuse to assist or participate in care. The person may develop an attitude of helplessness or dependence, or may demonstrate anger, hostility or aggression.

- Assess interactions between individuals and their family. *Assessment helps identify desired and potential destructive behaviours.*
- Assess the effect of the illness on the family. *Assessment of family interactions, roles and relationships assists in planning appropriate interventions.*
- Help the person and family identify strengths for coping with the situation. *Identifying personal and family strengths helps the family regain a sense of control.*
- Provide information and teaching about COPD. *Education helps the family gain an understanding of the individual's condition and needs.*
- Encourage expression of feelings. *Avoid judging feelings expressed by family members as 'good' or 'bad', 'right' or 'wrong'. It is important that the nurse remain objective to maintain the therapeutic relationship.*
- Help family members recognise behaviours and attitudes that may hinder effective treatment, such as continuing to smoke in the house. *Family members may be unaware of the effect of their behaviour on the person's ability to change habits and cope with a disabling disease.*
- Encourage family members to participate in care. *This helps develop skills for use at home.*
- Initiate a care conference involving the person, family and healthcare team members from a variety of disciplines. *A wide range of perspectives and areas of expertise aids in problem solving and facilitates communication.*
- If dysfunctional family relationships interfere with measures to enhance coping, advocate for the person, reaffirming his or her right to make decisions. *Dysfunctional family relationships are not likely to change simply because of illness. The nurse can better meet the person's needs by accepting their limitations in dealing with family members.*
- Refer the person and family to support groups and respiratory rehabilitation programs, as available. *Support groups and structured rehabilitation programs enhance coping abilities.*
- Arrange a social services consultation. *This can help the person and their family identify care and support service needs.*
- Refer community agencies or home services such as Meals on Wheels as appropriate. *Agencies or community services can provide additional support beyond the family's means or capability.*

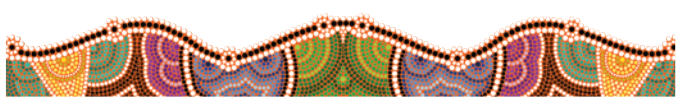
Conflict: smoking

Smoking is more than a habit; it is an addiction. The person who must quit is facing a significant loss, not only of nicotine but also of a lifestyle. Although the person may fully comprehend the consequences of continuing to smoke, the decision to give up a part of their life is not easy. This fear may be expressed in such concerns as 'I'll gain weight' or 'What will I do with my hands?' In addition to providing practical information, a plan and assistance with nicotine withdrawal, the nurse must support the person's decision-making process to comply with an order to stop smoking.

- Assess knowledge and understanding of the choices involved and possible consequences of each. *The decision to quit smoking ultimately belongs to the individual. They need a full understanding of the consequences of quitting or continuing to smoke.*
- Acknowledge concerns, values and beliefs; listen non-judgmentally. *The nurse needs to avoid imposing their own values and beliefs about smoking on the person.*
- Spend time with the person, encouraging expression of feelings. *This demonstrates acceptance of the person and their right to make the decision.*
- Help plan a course of action for quitting smoking and adapt it as necessary. *When the person develops the plan, they have more ownership in it and interest in making it work.*
- Demonstrate respect for decisions and the right to choose. *Respect supports self-esteem and the ability to cope.*
- Provide referral to a counsellor or other professional as needed. *Counsellors or other people trained to assist with smoking cessation can help with decision making.*

Community-based care

As with any chronic disease, the person and family will have primary responsibility for disease management. Teaching is vital to promote optimal health and slow disease progression. Teaching for home care focuses on effective coughing and breathing techniques (see Box 36.4), preventing exacerbations and managing prescribed therapies.



BOX 36.4 Person-centred teaching: effective coughing and breathing techniques

Pursed-lip and diaphragmatic breathing techniques help minimise air trapping and fatigue. Pursed-lip breathing helps maintain open airways by maintaining positive pressures longer during exhalation. Teach the person to:

1. Inhale through the nose with the mouth closed.
2. Exhale slowly through pursed lips, as though whistling or blowing out a candle, making exhalation twice as long as inhalation.

Diaphragmatic or abdominal breathing helps conserve energy by using the larger and more efficient muscles of respiration. Teach the person to:

1. Place one hand on the abdomen, the other on the chest.
2. Inhale, concentrating on pushing the abdominal hand outwards while the chest hand remains still.
3. Exhale slowly, while the abdominal hand moves inwards and the chest hand remains still.

Repeat these exercises as often as necessary until the techniques become incorporated into normal breathing.

Several different coughing techniques may be useful. For controlled cough technique, teach the person to:

1. Following prescribed bronchodilator treatment, inhale deeply and hold breath briefly.
2. Cough twice, the first time to loosen mucus, the second to expel secretions.
3. Inhale by sniffing to prevent mucus from moving back into deep airways.
4. Rest. Avoid prolonged coughing to prevent fatigue and hypoxaemia.

For 'huff' coughing, teach the person to:

1. Inhale deeply while leaning forwards.
2. Exhale sharply with a 'huff' sound to help keep airways open while mobilising secretions.

In addition, include the following topics when teaching for home care:

- maintaining adequate fluid intake (at least 2 L of fluid daily)
- avoiding respiratory irritants, including cigarette smoke, both primary and secondary, other smoke sources, dust, aerosol sprays, air pollution and very cold, dry air
- preventing exposure to infection, especially upper respiratory infections
- importance of pneumococcal vaccine and annual influenza immunisation
- prescribed exercise program, maintaining ADLs and balancing rest and exercise
- maintaining nutrient intake (e.g. eating small frequent meals and using nutritional supplements to provide adequate kilojoules)
- ways of reducing sodium intake if prescribed
- identifying early signs of an infection or exacerbation and the importance of seeking medical attention for the following: fever, increased sputum production, purulent (green or yellow) sputum, upper respiratory infection, increased shortness of breath or difficulty breathing, decreased activity tolerance or appetite, increased need for oxygen
- prescribed medications, including purpose, proper use and expected effects
- avoiding use of OTC medications unless approved by the doctor
- other prescribed therapies, such as use of home oxygen, percussion, postural drainage and nebuliser treatments
- use, cleaning and maintenance of any required special equipment
- importance of wearing an identification band and carrying a list of medications at all times in case of an emergency.

Provide referrals to home care services such as home health, assistance with ADLs as needed, home maintenance services, respiratory therapy and home oxygen services, and other agencies such as Meals on Wheels as indicated.

OTHER SELECTED LUNG DISEASES AFFECTING THE AIRWAY

THE PERSON WITH CYSTIC FIBROSIS

Cystic fibrosis (CF) is an autosomal recessive disorder that affects epithelial cells of the respiratory, gastrointestinal and reproductive tracts and leads to abnormal exocrine gland secretions. Although it can affect many organ systems, CF is particularly damaging to the lungs, leading to COPD in childhood and early adulthood. Respiratory manifestations of CF are the usual cause of morbidity and death from this disease. The gastrointestinal tract also is affected significantly; exocrine pancreatic insufficiency is characteristic of CF. Abnormally high sweat electrolytes also occur in CF.

Incidence and prevalence

In Australia, 34 people died from cystic fibrosis in 2013 (ABS, 2015c). As at 31 December 2013, 3235 people were recorded to have cystic fibrosis on the Australian Cystic Fibrosis Data Registry. Of the 92 new Australian CF diagnoses in 2013, 86% were diagnosed before 1 year of age. In 2001, the number of adults on the register was 35%; in 2013, 49.9% of the people on the CF register were adults. Due to advances in medical management, 22% of the people on the register are over 30 and 4% are over 60 years of age (Australian Cystic Fibrosis Data Registry (ACFDR), 2015).

Pathophysiology

The CFTR protein is involved in membrane transport of chloride and sodium in cells lining the ducts of exocrine glands (sweat glands, pancreas, liver and reproductive systems). The genetic abnormality of CF leads to a lack or abnormality of this protein, with resulting abnormal electrolyte transport across epithelial cell membranes (see the accompanying box).

GENETIC CONSIDERATIONS
Cystic fibrosis

The gene responsible for cystic fibrosis is at a single locus on the long arm of chromosome 7. This gene codes for a protein known as the *cystic fibrosis transmembrane conductance regulator (CFTR)*. More than 1000 mutations of this gene have been identified. The most common mutation, identified as ΔF508, accounts for about 66% of cystic fibrosis (Sharma, 2014). Cystic fibrosis is an autosomal recessive disorder: it is not transmitted as a sex-linked trait and the normal gene is dominant. People with one abnormal gene do not have the disorder but can transmit this abnormal gene to their offspring. When a child inherits an abnormal gene from both parents, the disorder is seen. Genetic screening of family members can detect 70–75% of carriers of the CF gene. Screening for the CF gene is not recommended for the general population.

Defective chloride transport causes more water and sodium reabsorption than normal. Secretions in affected organs become thick and viscous, obstructing glands and ducts. This obstruction causes dilation of secretory glands and damage to exocrine tissue. The hallmark pathophysiological effects of CF include:

- excess mucus production in the respiratory tract with impaired ability to clear secretions and progressive COPD
- pancreatic enzyme deficiency and impaired digestion
- abnormal elevation of sodium and chloride concentrations in sweat.

In the lungs, viscous mucus plugs small airways and impairs mucociliary clearance, leading to atelectasis, infection, bronchiectasis and dilation of distal airways. Lower respiratory infections with *Staphylococcus aureus* and *Pseudomonas, Haemophilus influenza and Escherichia coli* are common (ACFDR, 2015). A multidrug-resistant bacteria *Burkholderia cepacia* (previously *Pseudomonas cepacia*) is clinically significant as it may also hamper the potential for lung transplantation (Lubamba et al., 2012) Acute and chronic damage to lung parenchyma causes tissue loss and extensive scarring and fibrosis. The upper lobes are involved to a greater extent than the lower lobes. Severe airway obstruction and chronic hypoxaemia lead to pulmonary hypertension, right ventricular hypertrophy and eventual cor pulmonale. Death usually results from a combination of cardiovascular changes and respiratory failure.

Pancreatic insufficiency is a frequent component of CF. It can range from slight pancreatic dysfunction to complete absence of function due to obstruction of pancreatic ducts with thick mucus and degenerative and fibrotic changes. Pancreatic insufficiency and impaired enzyme secretion lead to impaired digestion and absorption of proteins, carbohydrates and fats.

About 8–12% of individuals with CF over 25 years of age develop diabetes mellitus. Liver failure is another potential complication of the disease (Sharma, 2014). Because the genetic defect also affects cells of the reproductive tract, males with CF usually are sterile. Although females may have difficulty conceiving, pregnancies are usually carried to term.

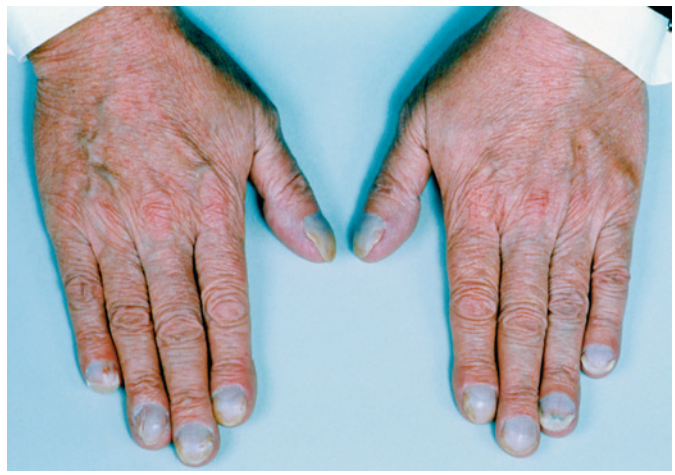


FIGURE 36.10 ■ Clubbing of fingers caused by chronic hypoxaemia

Source: © John Radcliffe Hospital/Science Source.

Depending on a women's pre-pregnancy health, pregnancy can be tolerated well despite at least a moderate decrease in lung function (Lau et al., 2011).

Manifestations

Manifestations of CF in a young adult include a history of chronic lung disease. Recurrent pneumonia, exercise intolerance and chronic cough are typical. Other pulmonary manifestations include *clubbing* of the fingers and toes (see Figure 36.10), increased anteroposterior chest diameter (barrel chest), hyperresonant percussion tone and basilar crackles on auscultation. Distended neck veins, ascites and peripheral oedema accompany right-sided heart failure. Abdominal pain and *steatorrhoea* (excess fat in the stools, causing frequent, bulky, foul-smelling stool) commonly result from associated pancreatic insufficiency. Growth and development are often retarded, resulting in small stature.

INTERPROFESSIONAL CARE

The treatment plan for cystic fibrosis is multidisciplinary, with the goals of preventing or treating respiratory complications and maintaining adequate nutrition. Psychosocial care is vital, as is genetic and occupational counselling.

Diagnosis

Although evidence of lung disease and pancreatic insufficiency suggests CF, analysis of Cl^- concentration in sweat is used to confirm the diagnosis. In CF, the Cl^- concentration is > 70 mEq/L. Pilocarpine (a parasympathomimetic agent) and a small electric current are used to increase sweat production on the forearm. Absorbent paper or gauze is used to collect the sweat for analysis.

ABGs and oxygen saturation levels show hypoxaemia. Pulmonary function studies reveal reduced airflow, reduced forced vital capacity (see Box 33.1) and reduced total lung capacity. Alveolar–capillary diffusion also is typically reduced.

Medications

Immunisation against respiratory infections is vital to promote optimal health. Yearly influenza vaccine is recommended, along with measles and pertussis boosters as needed.

Bronchodilator inhalers may be used to control airway constriction. Acute pulmonary infections are treated with appropriate antibiotic therapy as determined by sputum culture and sensitivity tests. A prolonged treatment course or multiple antibiotics may be required to eradicate pulmonary infections. Antibiotics may be administered by several routes, including inhalation, to achieve the desired concentration in large airways (Sharma, 2014). Dornase alfa, recombinant human DNase, breaks down the excess DNA in the sputum of individuals with CF, decreasing its viscosity and making it easier to clear. Dornase alfa, administered by aerosol, reduces the frequency of hospitalisations and the need for antibiotics for some individuals.

Treatments

Chest physiotherapy with percussion and postural drainage is used to promote airway clearance. Newer airway clearance techniques include the use of the 'huff' cough technique with specified breathing cycles or patterns. In one technique, a valved mask or mouthpiece is used to maintain positive expiratory pressure (PEP) for approximately 20 breaths, followed by three to five 'huff' coughs. This cycle is repeated for a total of 20 minutes. The autogenic drainage technique, a form of biofeedback, involves controlled breathing at specific lung volumes and patterns to facilitate the movement of mucus into larger airways, where it can be cleared with the 'huff' cough. A flutter valve device, which looks like a fat pipe, contains a steel ball within an inner cone. The weight of the ball provides intermittent PEP, which vibrates airway walls to loosen secretions.

Oxygen therapy may be required for hypoxaemia. A liberal fluid intake helps reduce the viscosity of mucus secretions. A diet high in protein, fat and kilojoules may be necessary to maintain weight. Vitamins and minerals are supplemented to counteract excess losses in the sweat and stools. Enteral or parenteral nutrition may be required during acute exacerbations of the disease.

Surgery

Lung transplantation currently offers the only definitive treatment for CF. Lung transplantation lengthens lifespan and improves quality of life. Single-lung, double-lung and heart–lung transplants have been successfully completed. Because the donor lungs do not have the CF gene, they do not develop the pathophysiological changes of CF. Although the other defects characteristic of CF remain, these can be managed with pharmacological therapy.

Nursing care

Nursing care for the person with cystic fibrosis is much the same as that for any chronic obstructive lung disease. Promoting airway clearance is the priority of nursing care. The genetic component of the disease and the person's age are important considerations. Adults with CF are just entering their productive years and face a lifespan that is likely to be shortened significantly. Females who do conceive face the prospect of transmitting the defective gene to their offspring.

Nursing diagnoses and interventions

Ineffective airway clearance

Bronchial hygiene measures, including vibration, percussion and postural drainage, are the mainstay of treatment for person with CF.

- Assess respiratory status, including vital signs, breath sounds, SpO_2 and skin colour, at least every 4 hours. *Early identification of respiratory compromise allows intervention before tissue hypoxia is significant.*
- Assess cough and sputum (amount, colour, consistency and possible odour). *Assessment of the cough and nature of sputum produced allows evaluation of the effectiveness of respiratory clearance and the response to therapy.*
- Monitor ABG results; report increasing hypoxaemia and other abnormal results to the doctor. *Blood gas changes may be an early indicator of impaired gas exchange due to airway obstruction.*
- Place in Fowler's or high-Fowler's position. Encourage frequent position changes and ambulation as allowed. *The upright position promotes lung expansion; position changes and ambulation facilitate the movement of secretions.*
- Assist to cough, deep breathe and use assistive devices. *Coughing and deep breathing help clear airways.*
- Provide a fluid intake of at least 2500 to 3000 mL per day. *A liberal fluid intake helps liquefy secretions, facilitating their clearance.*
- Work with the doctor and respiratory therapist to provide pulmonary hygiene measures, such as postural drainage, percussion and vibration. *These techniques help mobilise and clear secretions.*
- Administer prescribed medications as ordered and monitor their effects. If the infecting organism is resistant to the prescribed antibiotic, little improvement may be seen with treatment. *Bronchodilators help maintain open airways but may have adverse effects such as anxiety and restlessness.*

Anticipatory grieving

The person with CF and family members face the knowledge that their lifespan is likely to be short.

- Spend time with the person and family. *Time is necessary to develop a trusting, therapeutic relationship.*
- Answer questions honestly; do not deny the probable outcome of the disease. *Honesty reinforces reality and provides a sense of control over decisions to be made.*
- Encourage the person and family to express their feelings, fears and concerns. *Open expression of feelings helps to promote understanding and acceptance.*
- Assist with understanding the grieving process and acceptance of feelings as normal. *Feelings of guilt, anger or depression may cause the person to withdraw from others. Explanation of the grieving process enhances understanding and ability to cope.*

- Help the individual and their family make decisions regarding treatment and care. *This also is important to give them a sense of control.*
- Encourage use of other support systems, such as spiritual and social groups. Refer the person and their family to support groups, social support services and hospice care as indicated. *These support systems provide emotional support and help the person and family cope with the diagnosis.*
- Discuss advance directives (living wills) and power of attorney for healthcare with the person and their family. *These documents give the person and their family a sense of control over the medical care provided if the person is no longer able to express their own wishes.*

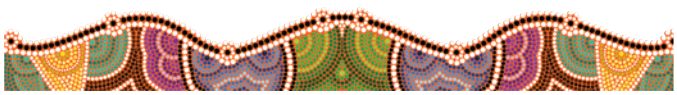
Community-based care

Education of the person and family affected by CF is essential to maintaining optimal health. The adult whose disease was diagnosed in infancy or childhood has grown up with the disease and often has a much greater knowledge level than many caregivers. However, when the initial diagnosis is made as an adolescent or young adult, teaching needs are significant. Include the following topics when teaching for home care:

- respiratory care techniques, including percussion, postural drainage and controlled cough techniques
- specific breathing and coughing exercises and procedures
- the importance of avoiding respiratory irritants, such as cigarette smoke, air pollution and occupational dusts and gases
- measures to prevent respiratory infection, such as maintaining immunisations and optimal general health and avoiding exposure to large crowds and infected people.

Refer to a dietitian for planning and teaching to maintain adequate nutrition and minimise gastrointestinal symptoms. Referral to community agencies and support groups is also helpful.

Discuss the genetic transmission of cystic fibrosis and refer for counselling and possible genetic testing. Help the individual and their family sort through the impact of the disease on future pregnancies and generations. Remember that the possibility of CF may present an ethical dilemma regarding future pregnancies. Provide support as needed.



THE PERSON WITH ATELECTASIS

Atelectasis is not a disease but a condition associated with many respiratory disorders. It is a state of partial or total lung collapse and airlessness. It may be acute or chronic. The most common cause of atelectasis is obstruction of the bronchus ventilating a segment of lung tissue. The affected segment may be small or an entire lobe. Other causes include compression of the lung by pneumothorax, pleural effusion or tumour; or loss of pulmonary surfactant and inability to maintain open alveoli.

The manifestations of atelectasis depend on its size. Diminished breath sounds over the affected area may be the only sign of a small atelectasis. If a large lung segment is affected, manifestations may include tachycardia, tachypnoea, dyspnoea, cyanosis and other signs of hypoxaemia. Chest expansion may be reduced and breath sounds absent on the affected side. Fever and other manifestations of infection may be present.

Chest x-ray shows an area of airless lung. Computed tomography (CT) scan may help determine the cause of atelectasis.

The primary therapy for atelectasis is prevention. Individuals with COPD, smokers undergoing surgery and people on prolonged bed rest or mechanical ventilation should have vigorous chest physiotherapy to maintain open airways as they are at high risk of atelectasis. Frequently assess respiratory status, including rate, breath sounds and spirometry readings, for early detection and treatment.

When atelectasis develops, treatment focuses on the underlying cause. Vigorous coughing and chest therapy may relieve obstruction by a mucus plug. Bronchoscopy may be necessary to remove the obstruction. Antibiotic therapy is ordered to treat infectious causes.

Nursing care to prevent and treat atelectasis is directed towards airway clearance. Position the person with atelectasis on the unaffected side to promote gravity drainage of the affected segment. Encourage frequent position changes, ambulation, coughing and deep breathing. Unless contraindicated, encourage fluids to help liquefy secretions. Teach the person at high risk of developing atelectasis about respiratory care measures, fluid intake and preventing respiratory infections.

THE PERSON WITH BRONCHIECTASIS

Bronchiectasis is characterised by permanent abnormal dilation of one or more large bronchi and destruction of bronchial walls. Infection often is present. The destructive process of bronchiectasis is initiated by inflammation, usually due to recurrent airway infections. About half of all cases of bronchiectasis are related to cystic fibrosis. Other causes include infections, such as severe pneumonia, tuberculosis or fungal infections; lung abscess; exposure to toxic gases; abnormal lung or immunological defences; and localised airway obstruction due to a foreign body or tumour. Inflammation and airway obstruction are common to all of these processes. Bronchial walls become weakened and dilated as a result, leading to pooling of secretions and further infection and inflammation.

A chronic cough productive of large amounts of mucopurulent sputum is characteristic. Other manifestations of bronchiectasis include haemoptysis, recurrent pneumonia, wheezing and shortness of breath, malnutrition, right-sided heart failure and cor pulmonale.

Collaborative care for bronchiectasis focuses on maintaining optimal pulmonary function and preventing progression of the disorder. The diagnosis is typically based on the history and physical examination. Chest x-ray and CT scan may be ordered to help confirm the diagnosis and determine the extent of lung damage.

Antibiotics are prescribed at the first indication of infection and may also be used prophylactically. Inhaled bronchodilators may be ordered. Chest physiotherapy is a vital component of continuing care for bronchiectasis. Percussion and postural

drainage help mobilise secretions. Oxygen may be prescribed. Bronchoscopy may be used to clear retained secretions or obstruction, or to evaluate haemoptysis. If lung destruction is localised and unresponsive to conservative management, surgical lung resection may be necessary.

Nursing care of the person with bronchiectasis is similar to that for individuals with other obstructive lung diseases. Airway clearance is a primary problem, as is ineffective breathing pattern. Other applicable nursing diagnoses may include *Impaired gas exchange*, *Inadequate nutrition* and *Self-care deficit*.

INTERSTITIAL PULMONARY DISORDERS

Many lung diseases damage the interstitial or connective tissue of the lung. Occupational lung diseases and sarcoidosis are interstitial lung diseases. Toxic drugs and radiation also cause interstitial damage. Table 36.6 identifies common causes of interstitial lung disorders. These disorders may be acute or insidious. Their rate of progression varies from person to person, as does the degree of disability they produce. Statistics are difficult to locate. However, in Australia, in 2013, 807 people died from lung diseases due to external agents, such as pneumoconiosis from asbestos or other mineral fibres, or pneumonitis due to organic substances, solids or liquids (ABS, 2015b).

THE PERSON WITH AN OCCUPATIONAL LUNG DISEASE

Occupational lung diseases are a diverse group of disorders directly related to inhalation of noxious substances in the work environment. There are two main classifications of occupational lung diseases:

- *Pneumoconiosis:* chronic fibrotic lung diseases caused by inhalation of inorganic dusts and particulate matter.
- *Hypersensitivity pneumonitis:* allergic pulmonary diseases caused by exposure to inhaled organic dusts.

Physiology review

Lung tissue contains elastin and collagen fibres. Elastin fibres are easily stretched, facilitating lung expansion. Collagen fibres, in contrast, resist stretching. This increases the work of breathing. Both elastin and collagen affect lung compliance or the ease with which the lungs are inflated (Caronia, 2014). Other factors affecting compliance include the water content of lung tissue and surface tension.

Pathophysiology and manifestations

When a noxious substance is inhaled, the response to that substance depends on:

- the size of particulates
- its nature (organic or inorganic)
- where it deposits in the respiratory tract
- the susceptibility of the individual.

Relatively large particles, larger than 6 μm, are too big to reach lower airways and often are deposited in the nose. Smaller particles can be carried with inspired air into the alveoli. Normal lung defences, including alveolar macrophages, lymph channels and the mucociliary escalator, attempt to remove particulate matter from the alveoli. Cigarette smoking, alcohol ingestion or hypersensitivity reactions can impair these defences.

The inhaled substance damages alveolar epithelium, leading to an inflammatory process of the alveoli and interstitial tissue of the lung. The inflammatory response produces further damage and abnormal fibrotic (scar) tissue replaces the elastin fibres of normal lung tissue. As a result, the lungs become stiff and non-compliant. Lung volumes decrease, the work of breathing increases and alveolar–capillary diffusion is impaired, leading to hypoxaemia.

Asbestosis

Inhalation of asbestos fibres is a common cause of occupational lung disease. *Asbestosis* is a diffuse interstitial fibrotic disease involving the terminal airways, alveoli and pleurae. Exposure to asbestos fibres occurs during mining, milling, manufacturing and application of asbestos products. Although symptoms may not become apparent until 20 years after exposure, they tend to progress, even when further exposure has been halted.

TABLE 36.6 Selected causes of interstitial lung disorders

CAUSE	EXAMPLES
Inorganic dusts	Silica (silicosis), asbestos (asbestosis), coal (coal worker's pneumoconiosis), talc (talcosis)
Organic dusts	Cotton (byssinosis), sugar cane (bagassosis), mouldy hay (farmer's lung)
Drugs	Antineoplastic agents, antibiotics, gold salts, phenytoin
Radiation	External radiation or inhaled radioactive materials
Infections	Widespread tuberculosis or fungal infections, viral or *Pneumocystis carinii* pneumonia
Poisons and noxious gases	Paraquat, nitrogen dioxide, chlorine, ammonia, sulfur dioxide
Systemic diseases	Uraemia, pulmonary oedema
Unknown causes	Sarcoidosis, idiopathic pulmonary fibrosis, connective tissue disorders

Asbestosis is also associated with an increased risk of bronchogenic carcinoma, especially in cigarette smokers. Mesothelioma, a rare cancer of the pleural membrane, is also associated with asbestos exposure. The period between asbestos exposure and tumour development in mesothelioma is long, ranging from 20 to 30 years. People exposed to asbestos prior to the imposition of strict environmental controls may only now be developing manifestations of this disease.

The manifestations of asbestosis include exertional dyspnoea, exercise intolerance and inspiratory crackles. Diffuse, small, irregular or linear opacities appear on chest x-ray, primarily in the lower lobes. As the disease progresses, respiratory failure and marked hypoxaemia may develop.

Silicosis

Inhalation of silica dust by hard-rock miners, foundry workers, sandblasters, pottery makers and granite cutters can lead to *silicosis*, a nodular pulmonary fibrosis (Varkey, 2013). In silicosis, macrophages are destroyed as they engulf silica particles, releasing substances that damage lung tissue and lead to fibrosis and scarring.

Simple silicosis is asymptomatic with no demonstrable respiratory impairment. Complicated silicosis, in contrast, is characterised by large conglomerate densities in the upper lungs. These individuals may be severely dyspnoeic and have a productive cough. Respiratory function testing shows both restrictive and obstructive changes. The increasing size of conglomerate masses can lead to severe disability, cor pulmonale and death.

Coal worker's pneumoconiosis

In the US, this occupational lung disease affects approximately 30% of all miners with 16% ultimately developing interstitial fibrosis (Khan, 2014). In 2012–2013 in Australia, 27 people were admitted for healthcare related to the effects of coal worker's pneumoconiosis (CWP) (AIHW, 2015a). Ingestion of coal dust by alveolar macrophages causes 'coal macules' to form, leading to *coal worker's pneumoconiosis* or *black lung disease*. Coal macules appear on chest x-ray as diffuse, small opacities primarily affecting the upper lungs.

Simple CWP generally is asymptomatic. A small percentage of individuals (1–2%) develop progressive massive fibrosis, which destroys the pulmonary vascular bed and airways of the upper lungs. This progressive form of the disease causes symptoms similar to those of complicated silicosis.

Hypersensitivity pneumonitis

Workers exposed to organic dusts and gases may develop *hypersensitivity pneumonitis*, an allergic pulmonary disease affecting the airways and alveoli. Byssinosis, resulting from cotton dust exposure; bagassosis, due to exposure to mouldy sugar cane fibre; farmer's lung and bird-fancier's lung are examples of hypersensitivity pneumonitis.

Either acute or subacute illness can occur. Acute illness occurs 4 to 8 hours after exposure and is heralded by sudden onset of malaise, chills and fever, dyspnoea, cough and nausea. The subacute syndrome is characterised by an insidious onset of chronic cough, progressive dyspnoea, anorexia and weight loss. Diffuse fibrosis occurs after repeated exposure to the organic material, leading to respiratory insufficiency.

INTERPROFESSIONAL CARE

Prevention is a key strategy for all occupational lung diseases. Containing dust and wearing personal protective devices that limit the amount of inhaled particles are essential for people who work in industries with known risks.

Investigations

Chest x-ray, pulmonary function studies, bronchoscopy and possibly lung biopsy are used to establish the diagnosis of pneumoconiosis. Characteristic patterns are seen for each disorder on x-ray. Pulmonary function testing shows restrictive impairment of lung ventilation, with reduced vital capacity and reduced total lung capacity. The diffusing capacity of the lungs is also decreased. Blood gas analysis reveals hypoxaemia, especially with exercise. Bronchoscopy may be performed to obtain tissue for biopsy. Specialised lung scans may be used to determine the extent of fibrosis.

Management

Eliminating further exposure to the offending agent is an important part of disease management. There is no specific therapy. Anti-inflammatory drugs, such as corticosteroids, may reduce the inflammatory response and slow the progression of the disease. Preventing exposure to other damaging substances such as cigarette smoke and pollution is vital. Pneumococcal vaccine and annual influenza immunisations are recommended to reduce the risk of lower respiratory infections. Other care is supportive, similar to that for COPD.

Nursing care

Health promotion

Teaching about the dangers of occupational lung diseases and ways to reduce their risks needs to begin early, before the disease develops. Nurses in industrial and public health settings can begin by recognising potential dangers and teaching workers about measures to reduce dust in their work area and the use of personal protective devices such as masks. Nurses working with affected families have an excellent opportunity to begin educating children about the risks associated with the occupation.

Nursing diagnoses and interventions

Nursing care for the person with occupational lung diseases is similar to that for those with COPD. Activity intolerance is a high-priority problem for many individuals. Severe dyspnoea can significantly interfere with ADLs. Nursing measures to reduce energy expenditures and provide for rest are essential. Caregiver role strain, either actual or potential, must be considered when the person with severe disability is being cared for at home.

Both the individual and their family coping may be compromised. Many of these diseases develop after 20 to 30 years of exposure to the hazardous material. Individuals who entered

the industry following high school may develop evidence of disease in their forties and face the possibility of changing their occupation or developing significant disability. The resulting role strain affects all members of the family.

Other nursing diagnoses to consider for the person with an occupational lung disease follow:

- *Ineffective oxygenation* related to restrictive lung disease evidenced by low oxygen saturations.
- *Situational low self-esteem* related to effects of ill heath, evidenced by reduced ability to maintain self-care.

Community-based care

The affected person and their family need teaching in preparation for home care, including:

- prevention of further lung damage—for example, avoiding cigarette smoke and heavy air pollution
- recommendations for pneumococcal and annual influenza immunisations; yearly tuberculin testing for individuals with silicosis
- respiratory hygiene measures, such as liberal fluid intake, coughing and deep-breathing exercises
- use and care of oxygen therapy equipment if required
- use and effects of any prescribed or recommended OTC medications.

THE PERSON WITH SARCOIDOSIS

Sarcoidosis is a chronic, multisystem disease characterised by an exaggerated cellular immune response in involved tissues. This abnormal immune response leads to granuloma formation in the lungs, lymph nodes, liver, eyes, skin and other organs. Its cause is unknown. Sarcoidosis primarily affects young adults between the ages of 20 and 40. In Australia in 2013, 35 people died of sarcoidosis (ABS, 2015c). The disease is unusual in Indigenous Australians and in individuals from an Asian background. Women are affected at a slightly higher rate than men. Approximately 1 in 10000 people in Australia have sarcoidosis (Allen, 2015).

In sarcoidosis, multiple granulomas form; these lesions may resolve spontaneously or proceed to fibrosis. The lungs are affected in most individuals with sarcoidosis. Sarcoidosis has a low mortality rate—less than 5% (Kamangar, 2015). Pulmonary haemorrhage and cardiac and respiratory failure from pulmonary fibrosis are the leading causes of death from sarcoidosis.

The manifestations of sarcoidosis vary depending on the organ system affected. It may be asymptomatic, diagnosed by characteristic findings on routine chest x-ray. Symptoms may be insidious, with anorexia, fatigue, weight loss, fever, dyspnoea, arthralgias and myalgias. Skin lesions, uveitis, lymphadenopathy, hepatomegaly or other manifestations may also develop.

Leucopenia, eosinophilia and an elevated erythrocyte sedimentation rate (ESR) typically are noted in sarcoidosis. The chest x-ray helps to determine the extent of pulmonary involvement. Biopsy of a granulomatous lesion may be required to confirm the diagnosis. Pulmonary function tests reveal decreased compliance and impaired diffusing capacity.

Sarcoidosis often resolves spontaneously; therefore, treatment is indicated only when symptoms are severe or disabling. Corticosteroid therapy is prescribed to suppress the inflammatory process when indicated. Relapse frequently occurs when corticosteroids are discontinued. Other anti-inflammatory or immune-modifier medications may also be used.

Nursing care for individuals with sarcoidosis is directed by involved organ systems and related manifestations. Respiratory care is supportive and includes avoiding respiratory irritants and maintaining adequate ventilation. Refer for smoking cessation assistance as needed.

Teach individuals about the disease and symptoms to report to a healthcare provider, including shortness of breath, tearing and eye inflammation, chest pain or irregular pulse, skin lesions and swollen and painful joints. If corticosteroid therapy is prescribed, teach the importance of taking the drug as prescribed and not stopping it abruptly. Include information about managing the side effects of corticosteroids by limiting sodium and increasing potassium in the diet, taking the medication with food or milk to minimise gastric irritation and identifying early signs of infection.

PULMONARY VASCULAR DISORDERS

The cardiovascular and respiratory systems are closely interrelated. As blood flows through the capillary network of the pulmonary vascular system, oxygen diffuses into it and carbon dioxide diffuses out. An effective match of alveolar ventilation and capillary perfusion is essential to maintain this process and, ultimately, tissue oxygenation and function of all organ systems. Both vascular and alveolar changes can alter gas exchange. Arteriosclerotic changes in pulmonary vasculature reduce blood flow to the alveolus. Nearly all lower respiratory system disorders potentially can affect ventilation. Many also have a secondary effect on lung perfusion, because breakdown or fibrosis of alveolar walls destroys the capillary network as well. This section focuses on primary disorders of the pulmonary vascular system.

THE PERSON WITH PULMONARY EMBOLISM

A **pulmonary embolism** (or *thromboembolism*) is obstruction of blood flow in part of the pulmonary vascular system by an embolus. *Thromboemboli*, or blood clots, that develop in the venous system (deep venous thrombosis, or DVT) or right side of the heart are the most frequent cause of pulmonary embolism. Other sources of emboli include tumours that

have invaded venous circulation, fat or bone marrow entering the circulation due to fracture or other trauma, amniotic fluid released into the circulation during childbirth and intravenous injection of air or other foreign substances.

Pulmonary embolism (PE) is a medical emergency. Most people who die from PE will generally die within the first few hours (Ouellette, 2015). In many cases, DVT has not been recognised or treated; often embolisation also goes undetected. Prevention is the most effective treatment strategy for PE.

Incidence and risk factors

In Australia, approximately 12 445 people were hospitalised for the treatment of PE in 2012–2013 (AIHW, 2015a). In 2013, pulmonary embolism caused 392 deaths in Australia (ABS, 2015c). Although many substances can become emboli, thrombus arising from the deep veins of the legs is the leading cause of PE. The risk factors for a pulmonary embolus are those for DVT: stasis of venous blood flow, vessel wall damage and altered blood coagulation.

Prolonged immobility; trauma, including hip and femur fractures; surgery (orthopaedic, pelvic and gynaecological surgery, in particular); myocardial infarction and heart failure; obesity; and advanced age are risk factors for DVT. Women who use oral contraceptives or oestrogen therapy are at risk, as are women during pregnancy and childbirth. See Chapter 31 for more information about DVT.

Physiology review

The right heart receives deoxygenated blood from the systemic venous circulation. The entire output of the right ventricle enters the pulmonary circulation via the pulmonary artery. This artery branches into successively smaller arteries, arterioles and capillaries of the pulmonary vascular system. Each alveolus of the lungs is surrounded by a meshwork of capillaries. Oxygen and carbon dioxide readily diffuse across the alveolar–capillary membrane, driven by a concentration gradient. The partial pressure of oxygen in the alveolus is greater than in the capillary; therefore, it diffuses into the blood. Carbon dioxide diffuses from the capillaries into the alveoli, driven by the higher pressure of dissolved carbon dioxide in venous blood.

A match between blood flow through the pulmonary vascular system (perfusion) and lung ventilation is necessary for effective *respiration* (gas exchange). Local factors regulate ventilation and perfusion to maintain this match. A low alveolar PO_2 constricts alveolar capillaries, directing blood flow to better-ventilated areas of the lung. High alveolar PCO_2 levels cause local bronchodilation, increasing airflow and eliminating excess carbon dioxide.

Pathophysiology

Thrombi affecting only the deep veins of the calf rarely embolise to the pulmonary circulation. However, thrombi often propagate proximally to the popliteal and ileofemoral veins. From there, they may break loose to become an embolus. As vessels of the venous system become progressively larger, the embolus moves freely until it enters the pulmonary arterial system, with its progressively smaller vessels leading to the pulmonary capillary beds (see Figure 36.11).

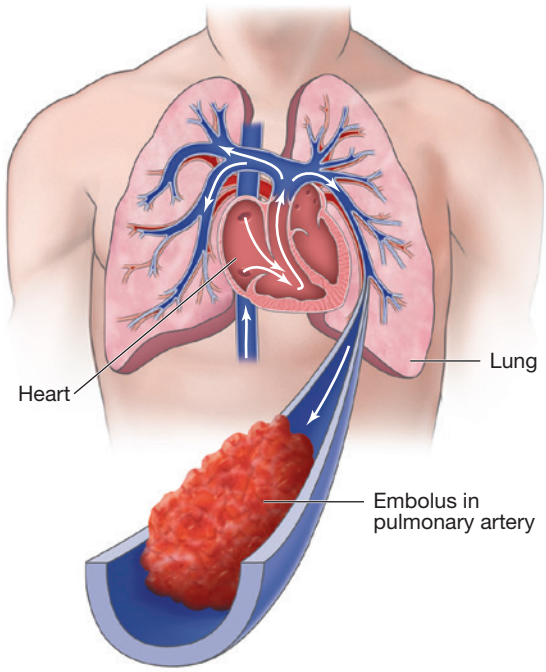


FIGURE 36.11 ■ A thromboembolism lodged in a pulmonary vessel

The impact of a pulmonary embolus depends on the extent to which pulmonary blood flow is obstructed, the size of the embolus, its nature and secondary effects of the obstruction. The effects can range widely:

- Occlusion of a large pulmonary artery with sudden death. Gas exchange is significantly reduced or prevented, and cardiac output falls dramatically as blood fails to move through the pulmonary vascular system and return to the left heart.
- Lung tissue infarction due to occlusion of a significant portion of pulmonary blood flow. Fewer than 10% of pulmonary emboli result in pulmonary infarction.
- Obstruction of a small segment of the pulmonary circulation with no permanent lung injury.
- Chronic or recurrent, possibly multiple, small emboli with recurring symptoms.

Obstruction of pulmonary blood flow by an embolus affects both perfusion and ventilation. Neurohumoral reflexes triggered by obstruction cause vasoconstriction, increasing pulmonary vascular resistance. In severe cases, this can lead to pulmonary hypertension and right ventricular heart failure. Systemically, hypotension and a drop in cardiac output may develop. Bronchoconstriction occurs in the affected area of lung. Dead space (areas of the lung that are ventilated but not perfused) increases. Alveolar surfactant decreases, increasing the risk of atelectasis.

If infarction does not occur, the thrombolytic system (see Chapter 28) ultimately dissolves the clot and pulmonary function returns to normal. Infarcted tissue becomes scarred and fibrotic.

Fat emboli are the most common non-thrombotic pulmonary emboli. A fat embolism usually occurs after fracture of long bone

(typically the femur) releases bone marrow fat into the circulation. Adipose tissue or liver trauma may also lead to fat emboli.

Manifestations

The manifestations of PE depend on its size and location. Small emboli may be asymptomatic. Manifestations usually develop abruptly, over a period of minutes. The most common symptoms are dyspnoea and pleuritic chest pain. Anxiety, a sense of impending doom and cough are also common (see box below). Diaphoresis and haemoptysis may develop. Massive pulmonary embolus can cause syncope and cyanosis. On examination, tachycardia and tachypnoea are noted. Crackles may be heard on auscultation of the chest and a cardiac gallop (S_3 and possibly S_4) may be noted. A low-grade fever may develop. It is difficult to differentiate pulmonary embolism from myocardial infarction or pneumonia by manifestations.

Characteristic manifestations of fat emboli include sudden onset of cardiopulmonary and neurological symptoms: dyspnoea, tachypnoea, tachycardia, confusion, delirium and decreased LOC. Petechiae often develop on the chest and arms.

MANIFESTATIONS Pulmonary embolism

COMMON
- Dyspnoea
- Chest pain
- Anxiety
- Cough
- Tachycardia and tachypnoea
- Crackles (rales)
- Low-grade fever

LESS COMMON
- Diaphoresis
- Haemoptysis
- Syncope
- Cyanosis
- S_3 and/or S_4 gallop

INTERPROFESSIONAL CARE

Because deep venous thrombosis may not be identified until pulmonary embolism occurs, prevention is the primary goal in treating PE.

Early ambulation is an effective means of preventing venous stasis and reducing the incidence of PE. External pneumatic compression of the legs is also effective for individuals undergoing neurosurgery, urological surgery or major surgery of the hip or knee or when anticoagulant therapy is contraindicated. Other preventive measures include elevating the legs and active and passive leg exercises.

When PE occurs, treatment is supportive. Oxygen therapy is initiated and analgesics may be ordered to relieve severe pleuritic pain and anxiety. Pulmonary artery and wedge pressures are monitored with a balloon (Swan–Ganz) catheter. Cardiac outputs also may be assessed. Cardiac rhythm is monitored to detect arrhythmias.

Diagnosis

The studies performed to identify DVT differ from those used to diagnose a PE. See Chapter 31 for diagnostic studies for venous thrombosis.

- *Plasma d-dimer levels* detect inflammation. A d-dimer is a fragment of fibrin formed during lysis of a blood clot; elevated blood levels indicate thrombus formation and lysis (e.g. DVT and PE).
- *Chest CT with contrast* is the principal test used to diagnose PE. Chest CT effectively shows large, central PE; newer-generation scanners also can detect peripheral emboli.
- *Lung scans*, including perfusion and ventilation scans, may be used. In a perfusion lung scan, radio-tagged albumin is injected intravenously and distributed in the lungs by the pulmonary blood flow. The lungs are then scanned for distribution of the isotope. An area of lung in which the isotope is undetectable is suggestive of occluded blood flow and PE. For a ventilation scan, a radio-tagged gas is inhaled and the lungs are scanned for gas distribution. Combined perfusion and ventilation scans allow identification of areas of the lungs that are ventilated but not perfused, a characteristic of PE.
- *Pulmonary angiography* is the definitive test for PE when other, less invasive tests are inconclusive. It is possible to detect very small emboli with angiography. A contrast medium injected into the pulmonary arteries illustrates the pulmonary vascular system on x-ray.
- *Chest x-ray* often shows pulmonary infiltration and occasionally pleural effusion.
- *Electrocardiogram (ECG)* is ordered to rule out acute myocardial infarction as the cause of symptoms. ECG findings commonly associated with PE include tachycardia and non-specific T-wave changes.
- *ABGs* usually show hypoxaemia (PaO_2 less than 80 mmHg) and often respiratory alkalosis (pH >7.45, $PaCO_2$ <38 mmHg) due to tachypnoea and hyperventilation.
- *$ETCO_2$* may be measured to evaluate alveolar perfusion. The normal $ETCO_2$ reading is 35 to 45 mmHg; it is decreased when pulmonary perfusion is impaired.
- *Coagulation studies* are ordered to monitor the response to therapy. The *activated partial thromboplastin time (aPTT* or *PTT*) is used to assess the intrinsic clotting pathway and the response to heparin therapy. Desired levels with anticoagulant therapy are 1.5 to 2 times the control value. The risk of recurrent thromboembolism is high at lower levels; the risk of bleeding increases at higher levels. The *International Normalized Ratio (INR)* is used to assess the extrinsic clotting system and oral anticoagulation with warfarin (Coumadin). The goal of anticoagulant therapy is to achieve a therapeutic range of 2.0 to 3.0.

Medications

Anticoagulant therapy is the standard treatment to prevent pulmonary emboli. It is often instituted in high-risk individuals who have no evidence of PE to prevent possible devastating effects. In the person with DVT or a pulmonary embolus, anticoagulants are administered to prevent further clotting and embolisation. See Chapter 31 for more information about anticoagulant therapy to prevent and treat DVT. See the ‘Medication

administration' box on anticoagulant therapy in Chapter 31 for the nursing implications for such therapy.

For PE, heparin therapy is initiated with an intravenous bolus of 5000 to 10 000 units of heparin, followed by continuous infusion at the rate of 1000 to 1500 units per hour. The aPTT or PTT is monitored frequently until stabilised. Heparin therapy is typically continued for about 5 days or until oral anticoagulant therapy has become fully effective.

Oral anticoagulant therapy with warfarin sodium is initiated at the same time as heparin. Warfarin alters the synthesis of vitamin-K-dependent clotting factors and requires 5 to 7 days to be fully effective. Anticoagulant therapy is continued for 2 to 3 months when few risk factors for thromboemboli exist; long-term therapy is used when chronic disorders that increase the risk of thromboemboli are present.

Bleeding is a risk associated with anticoagulant therapy. Although major haemorrhage is uncommon, it occurs in approximately 5% of individuals receiving intravenous heparin. Cardiac, hepatic and renal disease increase the risk of significant bleeding, as does age over 60 years. Protamine, a protein that combines with heparin to inactivate it, is used to stop its anticoagulant effect if major bleeding occurs. Vitamin K is given to treat bleeding associated with warfarin therapy.

Thrombolytic therapy may be used to treat massive pulmonary embolus and hypotension. Streptokinase, urokinase or tissue plasminogen activator (tPA) are used to *lyse* (disintegrate) the embolus, restore pulmonary blood flow and reduce pulmonary artery and right heart pressures. Although thrombolytic therapy may not reduce mortality associated with pulmonary embolus, it may reduce the incidence of pulmonary hypertension, which develops 3 to 5 years after an embolism. Thrombolysis significantly increases the risk of bleeding, particularly cerebral bleeding. Contraindications to thrombolysis include intracranial disease, recent stroke, active bleeding or a bleeding disorder, pregnancy, severe hypertension and recent surgery or trauma. Because of the increased risk of haemorrhage, invasive procedures are avoided after thrombolysis. See Chapter 29 for further discussion of thrombolytic therapy and its nursing implications.

Surgery

When anticoagulant therapy fails to prevent recurrent emboli or is contraindicated, an umbrella-like filter may be inserted into the inferior vena cava to trap large emboli while allowing continued blood flow. The filter usually is inserted percutaneously, via either the femoral or the jugular vein.

Nursing care

Health promotion

Nurses are pivotal in preventing pulmonary embolism. Encouraging individuals to ambulate after surgery or illness, applying compression stockings or pneumatic compression devices, teaching and encouraging leg exercises, discouraging the use of pillows under the knees—all of these measures help prevent DVT and subsequent PE.

Teach individuals to reduce the risks associated with long periods of immobility, stopping every 1 to 2 hours during long automobile trips for a brief stretch and walk, getting up every hour or so and doing leg exercises while seated during long flights and avoiding crossing the legs to prevent venous stasis and pooling. Regular exercise such as walking also reduces the risk of DVT. Instruct individuals who stand for long periods to use well-fitted elastic stockings, being careful to avoid hose that bind around the knee or thigh.

Assessment

Because pulmonary embolus can be a medical emergency, assessment may be very focused. In other instances, when emboli are small and not life threatening, a more extensive nursing assessment may be done.

- *Health history:* chest pain, dyspnoea, other symptoms, including onset, severity, precipitating factors; history of recent surgery, venous thrombosis or other risk factor such as childbirth or malignancy; current medications.
- *Physical examination:* level of consciousness, presence of respirations and pulse; colour, skin temperature and moisture; vital signs, including apical pulse and temperature; breath sounds and heart sounds; oxygen saturation level; neck vein distension, peripheral oedema.
- *Diagnostic tests:* plasma d-dimer levels, coagulation studies; chest x-ray and other imaging studies; oxygen saturation and ABGs; ECG.

Nursing diagnoses and interventions

A large pulmonary embolus can cause a significant mismatch between pulmonary ventilation and circulation. Impaired gas exchange is a priority problem and focus for interventions. Cardiac output may be significantly affected by obstructed pulmonary blood flow. Thrombolytic and anticoagulant therapy affect the clotting process, increasing the risk of bleeding. Anxiety accompanies PE almost universally.

Impaired gas exchange

Pulmonary embolism results in areas of the lung that are ventilated but not perfused; they receive no capillary blood flow. If the embolus is large and a major segment of the lung is not perfused, gas exchange is significantly affected. Nursing interventions are directed towards compensating for impaired gas exchange.

- Frequently assess respiratory status, including rate, depth, effort, lung sounds and oxygen saturation. *Impaired ventilation will further compromise gas exchange and worsen hypoxaemia. Oxygen saturation can be monitored continuously and non-invasively to evaluate gas exchange.*

CONSIDERATION FOR PRACTICE

Monitor and record LOC, mental status and skin colour. Hypoxaemia often causes confusion and agitation; hypercapnia may reduce LOC. Cyanosis indicates significant hypoxaemia.

- Place in Fowler's or high-Fowler's position, with the lower extremities dependent. *This position facilitates maximal lung expansion and reduces venous return to the right side of the heart, lowering pressures in the pulmonary vascular system.*

CONSIDERATION FOR PRACTICE

Start oxygen per nasal prongs or mask. Obtain a doctor's order if one has not been written. Supplemental oxygen increases alveolar and arterial oxygenation. Oxygen is a drug and must be prescribed by the doctor. It may, however, be initiated by the nurse in an emergency to prevent tissue hypoxia.

- Monitor ABG results, reporting abnormal findings as indicated. An arterial line may be inserted for monitoring arterial pressure and arterial blood sampling. *ABGs are used to assess gas exchange and tissue oxygenation.*
- Maintain bed rest. *Bed rest reduces metabolic demands and tissue needs for oxygen.*

Decreased cardiac output

The impact of a large pulmonary embolus on haemodynamic status can be significant. Pressures in the pulmonary vascular system and right heart increase; blood return to the left heart and cardiac output may significantly decrease. Nursing interventions focus on preserving an adequate blood pressure and organ function until cardiopulmonary status stabilises. A central line for haemodynamic monitoring may be instituted. (See Chapter 30 for nursing care related to haemodynamic monitoring.)

- Auscultate heart sounds every 2 to 4 hours, reporting any abnormalities. *Sounds such as an S3 or S4 gallop may indicate cardiac compromise.*

CONSIDERATION FOR PRACTICE

Assess and record vital signs and cardiopulmonary status every 15 to 30 minutes initially, then every 2 to 4 hours as condition stabilises. Frequent assessment facilitates timely interventions to maintain cardiovascular status and preserve organ function.

CONSIDERATION FOR PRACTICE

Record intake and output hourly. Decreased urinary output often is an early indicator of decreased cardiac output. Maintaining renal perfusion is vital to preserve renal function and prevent acute renal failure.

- Assess skin colour and temperature. *These assessments monitor tissue perfusion.*
- Monitor cardiac rhythm. *A drop in cardiac output and other haemodynamic alterations resulting from pulmonary embolism can precipitate arrhythmias. Arrhythmias, in turn, can further impair cardiac output.*
- Administer vasopressors and other medications as ordered. Carefully monitor the response to prescribed medications. *Drugs may be prescribed to maintain adequate arterial pressure and tissue perfusion. Potent drugs such as vasopressors require careful monitoring for desired and adverse effects.*
- Monitor pulmonary artery pressures, neck vein distension and peripheral oedema. Report findings as indicated. *Right-sided heart failure is a potential complication of PE because of increased pulmonary artery pressures.*
- Maintain intravenous and arterial access sites as well as central lines. *The person may be in an unstable and critical condition, potentially needing immediate interventions to maintain life.*
- Instruct to report chest pain or other symptoms. *Decreased cardiac output and an increased workload due to pulmonary hypertension may cause anginal pain.*

CONSIDERATION FOR PRACTICE

Provide frequent skin care. Impaired tissue perfusion and oxygenation increase the risk of skin and tissue breakdown.

Ineffective protection

Thrombolytics and anticoagulant therapy impair normal clotting mechanisms, increasing the risk of bleeding and haemorrhage. This risk is particularly acute during the first 24 to 48 hours following thrombolytic drug administration.

- Assess frequently for overt and covert signs of bleeding: bleeding gums; haematuria; obvious or occult blood in stool or vomitus; incisional bleeding, bleeding or bruising of injection sites or with minor trauma; joint pain or immobility; abdominal or flank pain. *Careful monitoring is necessary to identify early signs of abnormal bleeding and prevent potential haemorrhage.*
- Report coagulation study results outside the desired range for anticoagulant therapy. *Levels less than the target range may indicate an increased risk of further clot development and pulmonary emboli; levels above the target range indicate an increased risk of bleeding.*

CONSIDERATION FOR PRACTICE

Promptly report changes in neurological status. Although cerebral bleeding is not evident externally, changes in LOC and other neurological signs suggest it and should be reported immediately.

- Keep protamine sulfate available for heparin therapy and vitamin K available for warfarin therapy. *Bleeding or haemorrhage due to excess anticoagulant may require antidote administration to rapidly reverse anticoagulant effects.*
- Assess medication regimen for possible drug interactions that could potentiate or inhibit anticoagulant effects. *Drug interactions can increase the risk of haemorrhage or further embolus formation.*
- Avoid invasive procedures, injections and venipunctures when possible, particularly during and following thrombolytic therapy. *Invasive procedures increase the risk of tissue trauma and bleeding.*

- Maintain firm pressure on injection and venipuncture sites. Maintain pressure for 30 minutes following arterial puncture. *Firm pressure reduces the risk of bleeding into the tissues.*
- Maintain adequate fluid intake. Administer stool softeners as ordered. *These measures help prevent constipation and straining, which may precipitate bleeding of haemorrhoids.*

CONSIDERATION FOR PRACTICE

Use an infusion device to administer heparin infusion. Using an infusion pump or device helps prevent administration of excess medication.

Anxiety

Pulmonary embolism is a physiological and psychological threat to safety and integrity. It is a major physiological stressor, eliciting a strong neuroendocrine stress response. The feeling of suffocation and inability to catch one's breath that accompanies a pulmonary embolus is also a strong psychological stressor. Fear and anxiety are common responses.

- Assess anxiety level. *Appropriate interventions are determined by the level of anxiety.*

CONSIDERATION FOR PRACTICE

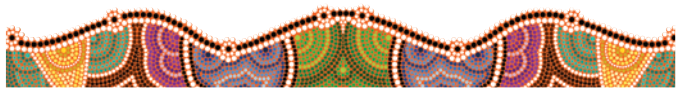
Provide reassurance and emotional support, listening to fears. Do not negate the fear of dying, but reassure that treatment usually restores effective respiratory function. The fear of death is very real and must not be discounted; however, it is important to provide reassurance to alleviate excess anxiety.

- Remain with the person as much as possible. *The presence of a caring nurse helps reduce fear.*
- Explain procedures and treatments, using short, simple sentences. *Providing clearly understood, simple instructions reduces fear of the unknown.*
- Reduce environmental stimuli and use a calm, reassuring manner. *These measures help reduce anxiety (for both the nurse and the individual).*
- Allow supportive family members to remain with the person as much as possible. *Calm, supportive family members provide further reassurance.*
- Administer morphine sulfate as ordered. *Morphine is given to reduce pain and anxiety.*

Community-based care

Discuss the following topics when preparing the person with pulmonary embolism and family members for home care:

- use of prescribed anticoagulant, including drug interactions, scheduled laboratory testing and manifestations of bleeding to report to the primary care provider
- using a soft toothbrush and electric razor to reduce the risk of bleeding
- avoiding aspirin (unless prescribed) and other OTC medications unless approved by the doctor
- importance of wearing a MedicAlert® tag for anticoagulant use
- health-promotion measures to reduce the risk of recurrent PE
- symptoms of recurrent PE, such as sudden chest pain, shortness of breath and, possibly, bloody sputum.



THE PERSON WITH PULMONARY HYPERTENSION

The pulmonary vascular system is normally a high-flow, low-pressure, low-resistance system that can accommodate large increases in blood flow when necessary (e.g. during exercise). The normal mean arterial pressure in the pulmonary system is 12 to 15 mmHg (25 to 28 systolic/8 diastolic). **Pulmonary hypertension** is abnormal elevation of the pulmonary arterial pressure. In Australia, approximately 3 to 10 people per million have idiopathic pulmonary hypertension. A family history is present in 10–12% of cases (PHA Australia, 2015). In 2012–2013 in Australia, 1581 people were admitted to hospital for management of either primary (idiopathic) or secondary hypertension (AIHW, 2015a).

Pathophysiology

Pulmonary hypertension can develop as a primary disorder, but usually occurs secondarily to another condition. In both instances, changes in the pulmonary artery lead to abnormal growth and remodelling of pulmonary vessels. Smooth muscle cells and fibroblasts proliferate, leading to abnormal vasoconstriction and fibrosis of pulmonary vessels. Once initiated, pulmonary vascular changes are progressive and non-reversible. Vasoconstrictive substances such as endothelin 1 and thromboxane A_2 are produced in excess, while the production of vasodilating substances such as nitric oxide is reduced. This further contributes to vasoconstriction and increased pulmonary artery pressures. Thromboxane A_2 also stimulates platelet aggregation, promoting clot formation in pulmonary vessels. Inflammation may contribute to progression of the disease. Vasoconstriction and increased pressures in the pulmonary system increase the workload of the right ventricle, ultimately leading to right ventricular failure (Oudiz, 2014; Kamangar, 2014).

Primary pulmonary hypertension

Primary pulmonary hypertension is an uncommon disorder without identified cause. It occurs in both familial and sporadic patterns. In the familial form, a gene transmitted in an autosomal dominant pattern affects a protein receptor in the walls of pulmonary arteries, leading to abnormal vessel growth and remodelling. Primary pulmonary hypertension mainly affects women in their thirties or forties.

Secondary pulmonary hypertension

Secondary pulmonary hypertension is more common than primary. HIV infection and collagen diseases (e.g. scleroderma and lupus) may lead to secondary pulmonary hypertension. However, its usual cause is the reduced size of the pulmonary vascular bed, which may be due to vasoconstriction or widespread vessel destruction or obstruction. Hypoxaemia is a potent pulmonary vasoconstrictor and common initiating

factor in pulmonary hypertension. Chronic lung diseases, sleep apnoea and hypoventilation due to obesity or neuromuscular disease can lead to hypoxaemia. Alveolar wall destruction associated with emphysema leads to loss of pulmonary capillaries. Large or multiple pulmonary emboli may cause vessel obstruction. Left ventricular failure or mitral stenosis also can lead to elevated pulmonary pressures. Once initiated, pulmonary hypertension becomes self-sustaining, as pulmonary vessels undergo changes that further narrow the pulmonary bed.

Manifestations

The manifestations of pulmonary hypertension are progressive dyspnoea, fatigue, angina and syncope with exertion. In secondary pulmonary hypertension, the signs and symptoms often are masked by those of the underlying disease. Dull, retrosternal chest pain may occur in addition to the manifestations of the primary disease. Primary pulmonary hypertension is a progressive disorder that generally causes a steady decline to death within 3 to 4 years.

Complications

Cor pulmonale is a condition of right ventricular hypertrophy and failure resulting from long-standing pulmonary hypertension. Chronic obstructive pulmonary disease is the most common cause of cor pulmonale.

The manifestations of cor pulmonale are those of the underlying pulmonary disorder and right-sided heart failure. Chronic productive cough, progressive dyspnoea and wheezing are common. With right-sided heart failure, peripheral oedema and distended neck veins are seen. Skin is warm, moist and cyanotic because of increased numbers of RBCs and hypoxaemia.

INTERPROFESSIONAL CARE

An FBC commonly shows *polycythaemia*, increased numbers of red blood cells. ABGs and oxygen saturation measurements reveal hypoxaemia. A chest x-ray shows right heart enlargement and dilation of central pulmonary arteries. Typical ECG changes are those of right ventricular hypertrophy. An echocardiogram may be done to identify cardiac changes occurring either as a cause or a result of pulmonary hypertension. Doppler ultrasonography is a non-invasive means of estimating pulmonary artery pressure, but cardiac catheterisation may be required for definitive diagnosis. See Chapter 28 for nursing care of the person undergoing cardiac catheterisation.

Treatment for pulmonary hypertension focuses on slowing the course of the disease, preventing thrombus formation and reducing pulmonary vasoconstriction. Oxygen is administered to reduce hypoxaemia and improve activity tolerance. If polycythaemia is present, venesection is performed to reduce the viscosity of the blood.

The calcium channel blockers nifedipine or diltiazem may be given to reduce pulmonary vascular resistance and improve cardiac output. Short-acting direct vasodilators such as intravenous epoprostenol or oral bosentan may be used for individuals who do not respond to calcium channel blockers. An oral anticoagulant (warfarin) is given to prevent clotting (Oudiz, 2014; Kamangar, 2014).

Bilateral lung or heart–lung transplant is the most effective long-term treatment for primary pulmonary hypertension.

When cor pulmonale is present, salt and water restrictions as well as diuretic therapy are added to the above regimen to manage the right-sided heart failure.

Nursing care

Nursing care for the person with pulmonary hypertension or cor pulmonale is largely supportive. The focus is on the underlying lung disease. Impaired gas exchange due to contraction of the pulmonary vascular system is a significant problem that causes many secondary problems, such as activity intolerance, anxiety and fatigue. Nursing interventions for impaired gas exchange are directed towards maintaining adequate alveolar ventilation, oxygenation and perfusion. The following measures may be included:

- monitoring breath sounds, respiratory rate, skin colour and use of accessory muscles
- positioning for optimal lung expansion
- coughing, deep breathing and chest physiotherapy
- administering prescribed vasodilators.

It is important to assess fatigue and dyspnoea with activities and to plan frequent rest periods. Assist with self-care as needed to conserve energy.

With primary pulmonary hypertension, *Anticipatory grieving* and *Hopelessness* are additional potential nursing diagnoses. When cor pulmonale is present, *Decreased cardiac output*, *Excess fluid volume* and *Ineffective individual coping* must be considered.

Community-based care

Most care for these chronic conditions is provided in the home and community settings. Teaching is directed both at the underlying lung disease, if present, and the resulting hypertensive process. Refer to the section on COPD for teaching related to this disease, the most frequent underlying cause of cor pulmonale. In addition, provide teaching about the following topics for the person and their family:

- disease process, its management and the prognosis
- manifestations or changes in condition to report to the doctor, such as a change in activity tolerance, increased oedema and signs of respiratory infection or exacerbation
- importance of planned rest periods between activities and measures to conserve energy, such as using a shower chair
- importance of not smoking due to its irritant and vasoconstrictive effects
- prescribed medications, including their use and effects.

RESPIRATORY FAILURE

Many of the conditions discussed in this chapter and in Chapter 35, from pneumonia to acute respiratory distress syndrome, can lead to respiratory failure. In **respiratory failure**, the lungs are unable to oxygenate the blood and remove carbon dioxide adequately to meet the body's needs, even at rest.

THE PERSON WITH ACUTE RESPIRATORY FAILURE

Respiratory failure is not a disease but a consequence of severe respiratory dysfunction. It is often defined by ABG values. An arterial oxygen level (PaO_2) of less than 50 to 60 mmHg and an arterial carbon dioxide level ($PaCO_2$) of greater than 50 mmHg are generally accepted as indicators of respiratory failure. However, individuals with advanced COPD may be alert and functional with blood gas values that would indicate respiratory failure in someone whose respiratory function was previously normal. In people with COPD, respiratory failure is indicated by an acute drop in blood oxygen levels along with increased carbon dioxide levels.

Respiratory failure can result from inadequate alveolar ventilation (hypoventilation), impaired gas exchange or a significant ventilation–perfusion mismatch. COPD is the most common cause of respiratory failure. Other lung diseases, chest injury, inhalation trauma, neuromuscular disorders and cardiac conditions can also lead to respiratory failure. Selected causes of acute respiratory failure are identified in Table 36.7.

Pathophysiology

Respiratory failure may be characterised by primary hypoxaemia or a combination of hypoxaemia and hypercapnia (see Figure 36.12). In hypoxaemic respiratory failure, PaO_2 is significantly reduced, whereas $PaCO_2$ remains normal or is low due to stimulation of the respiratory centre and tachypnoea. Impaired diffusion across the alveolar–capillary membrane and a ventilation–perfusion

TABLE 36.7 Selected causes of respiratory failure

TYPE OF DYSFUNCTION	EXAMPLES
Impaired ventilation	
• Airway obstruction	Laryngospasm, foreign body aspiration, airway oedema
• Respiratory disease	Asthma, COPD
• Neurological causes	Spinal cord injury, poliomyelitis, Guillain–Barré syndrome, drug overdose, stroke
• Chest wall injury	Flail chest, pneumothorax
Impaired diffusion	
• Alveolar disorders	Pneumonia, pneumonitis, COPD
• Pulmonary oedema	Heart failure, ARDS, near-drowning
Ventilation–perfusion mismatch	Pulmonary embolism

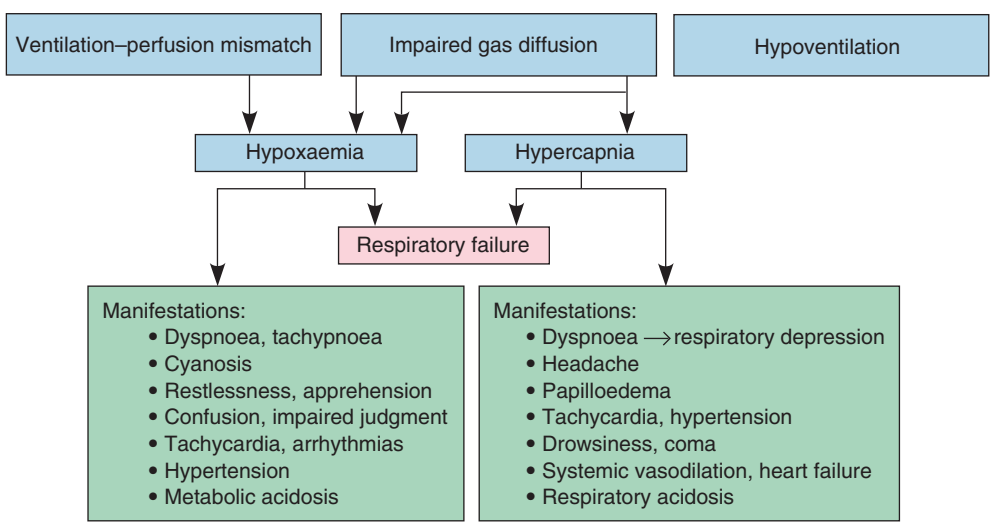


FIGURE 36.12 ■ Causes and manifestations of respiratory failure

mismatch can cause a drop in arterial oxygen levels that is more rapid than the rise in carbon dioxide. Metabolic acidosis results from tissue hypoxia. The increased work of breathing can eventually lead to respiratory muscle fatigue and hypoventilation.

Hypoventilation, or reduced movement of air into and out of the lung, causes carbon dioxide retention. With significant hypoventilation, the carbon dioxide level in the blood rises rapidly, leading to respiratory acidosis. Hypoxaemia develops more slowly and responds readily to administration of oxygen unless gas exchange also is impaired.

In summary, hypoxaemia without a corresponding rise in carbon dioxide levels indicates a failure of oxygenation; hypoxaemia with hypercapnia is the result of lung hypoventilation.

See the accompanying 'Pathophysiology illustrated' feature.

Manifestations and course

The manifestations of respiratory failure are caused by hypoxaemia and hypercapnia, as well as the underlying disease process. Hypoxaemia causes dyspnoea and neurological symptoms such as restlessness, apprehension, impaired judgment and motor impairment. Tachycardia and hypertension develop as the cardiac output increases in an effort to bring more oxygen to the tissues. Cyanosis is present. As hypoxaemia progresses, arrhythmias, hypotension and decreased cardiac output may develop.

Increased carbon dioxide levels depress CNS function and cause vasodilation. Dyspnoea and headache are early signs. Other manifestations include peripheral and conjunctival vasodilation, papilloedema, neuromuscular irritability and decreased LOC. As hypercapnia worsens, the respiratory centre may be depressed, reducing dyspnoea and slowing respirations. Increased carbon dioxide and hydrogen ion concentrations no longer stimulate the respiratory centre; hypoxaemia provides the primary active breathing stimulus. Administering oxygen without ventilatory support may further reduce the drive to breathe, leading to respiratory arrest.

The prognosis for acute respiratory failure varies, depending on the underlying disease process. Respiratory failure resulting from uncomplicated drug overdose generally resolves quickly without long-term effects. When respiratory failure results from underlying lung disease, the course may be prolonged and the outcome less favourable.

INTERPROFESSIONAL CARE

Treatment of respiratory failure focuses on correcting the underlying cause or disease, supporting ventilation and correcting hypoxaemia and hypercapnia. Care related to disorders that can precipitate respiratory failure is discussed in the sections specific to each disorder.

Diagnosis

Exhaled carbon dioxide and ABGs are used to diagnose and monitor treatment of respiratory failure.

- *$ETCO_2$* is used to evaluate alveolar ventilation. The normal $ETCO_2$ is 35 to 45 mmHg; it is elevated when ventilation is inadequate and decreased when pulmonary perfusion is impaired.
- *ABGs* also are used to evaluate alveolar ventilation and gas exchange. With hypoxaemic respiratory failure, the $PaCO_2$ may be normal, 38 to 42 mmHg, or even low due to tachypnoea. A pH of less than 7.35 and low bicarbonate levels indicate metabolic acidosis, typical of hypoxaemic respiratory failure.

  In respiratory failure due to hypoventilation, the $PaCO_2$ is elevated, usually greater than 50 mmHg. The pH is low due to respiratory acidosis. Acidosis develops rapidly in hypoxaemia and hypercapnia because of increased acid production (metabolic) and decreased acid elimination (respiratory).

Medications

Drugs used in treating respiratory failure depend on the underlying cause of the failure and the need for intubation and mechanical ventilation.

Beta-adrenergic (sympathomimetic) or anticholinergic medications may be administered by inhalation to promote bronchodilation. If mechanical ventilation is required, the drugs may be given by nebuliser attached to the ventilator. See 'Medication administration: Asthma' on pages 1318–1319 and the asthma section of this chapter for more information about bronchodilators and their nursing implications. Corticosteroids, administered by inhalation or intravenously, may be ordered to reduce airway oedema. Antibiotics are given to treat any underlying infection.

Sedation and analgesia often are required during mechanical ventilation to decrease pain and anxiety. Benzodiazepines such as diazepam, lorazepam or midazolam may be used for sedation and to inhibit the respiratory drive. Intravenous morphine or fentanyl provides analgesia and also inhibits the respiratory drive, allowing more effective mechanical ventilation. Occasionally, the person's respiratory drive competes with the ventilator despite sedation, decreasing its effectiveness and increasing the work of breathing. A neuromuscular blocking agent may be necessary to induce paralysis and suppress the ability to breathe. Nursing implications of neuromuscular blockers are described in the 'Medication administration' box below.

Oxygen therapy

Oxygen is administered to reverse hypoxaemia in acute respiratory failure. In general, the goal is to achieve an oxygen saturation of 90% or greater without oxygen toxicity. A PaO_2 of about 60 mmHg usually is adequate to meet the oxygen needs of body tissues. Higher levels do not significantly increase oxygen saturation and may lead to hypoventilation in individuals with chronic hypercapnia. As little as 1 to 3 L of oxygen per nasal prongs or 28% oxygen per Venturi mask may correct hypoxaemia in advanced COPD. Oxygen concentrations of 40–60% may be required when diffusion is impaired (e.g. in pneumonia or ARDS). High concentrations are used only for short periods to avoid oxygen toxicity. Both the oxygen concentration and duration of therapy contribute to oxygen toxicity. Continued high oxygen concentrations impair the synthesis of surfactant, reducing lung compliance (ease of inflation). ARDS or absorption atelectasis may develop.

When respiratory failure is caused by hypoventilation or usual oxygen delivery systems do not correct hypoxaemia, a tight-fitting mask to maintain *continuous positive airway pressure (CPAP)* may be used. CPAP increases lung volume,

MEDICATION ADMINISTRATION Neuromuscular blockers

NON-DEPOLARISING NEUROMUSCULAR BLOCKERS

Non-depolarising neuromuscular blockers competitively block the action of acetylcholine (ACh) at skeletal muscle receptors, preventing muscle depolarisation and contraction. Complete muscle paralysis is achieved within minutes. Facial muscles are affected first, followed by muscles of the limbs, neck and trunk. The muscles of respiration (the diaphragm and intercostal muscles) are least sensitive to the effects of neuromuscular blockers and are paralysed last. When the drug is discontinued or an antagonist is given, muscles recover in reverse order; respiratory function is recovered first.

Nursing responsibilities

- Prior to administering, assess endotracheal tube placement and ensure effective mechanical ventilator function. The risk of hypoxaemia and organ damage is significant if respiratory muscles are paralysed without adequate ventilatory support in place.
- Administer the drug by slow intravenous injection and/or intravenous infusion as prescribed.
- Keep an acetylcholinesterase (AChE) inhibitor such as neostigmine available at the bedside to rapidly reverse neuromuscular effects if needed.
- Administer morphine sulfate, diazepam, midazolam or other anti-anxiety agent or sedative as ordered. Neuromuscular blockers provide no sedation or pain relief; muscle paralysis in a conscious person produces extreme anxiety.
- Instil artificial tears every 2 to 4 hours.
- Suction oral cavity as needed to remove saliva.
- Ensure ventilator alarms are always on and appropriate for the person especially when administering neuromuscular blockers. Should the tubing become disconnected or plugged, the person is unable to breathe independently. Begin manual intermittent positive pressure ventilation with an air viva bag or bag/valve system and/or call for assistance.
- Treat the person as though awake and alert. Although unable to respond, mental function is unaffected.

Health education for the person and family

- Reassure that the ability to move and communicate will return when the drug is discontinued.
- Teach the family about the effects of the drug and the reason for its use. Explain that the person can hear and understand what is going on.

opening previously closed alveoli, improving ventilation of underventilated alveoli and ventilation–perfusion relationships.

Airway management

If the upper airway is obstructed or positive-pressure mechanical ventilation is necessary to correct hypoxaemia and hypercapnia, an endotracheal tube that extends from the mouth or nose into the trachea is inserted (see Figure 36.13). To maintain positive-pressure ventilation, the tube is cuffed with an air-filled balloon just above the end of the tube. When the cuff is inflated it obstructs the upper airway, preventing air from escaping back into the nose or mouth. Excess pressure from the cuff can cause tissue ischaemia and necrosis of the trachea. To minimise this risk, high-volume, low-pressure ('floppy') cuffs are used. Tubes with low-pressure cuffs may be left in place for 3 to 4 weeks.

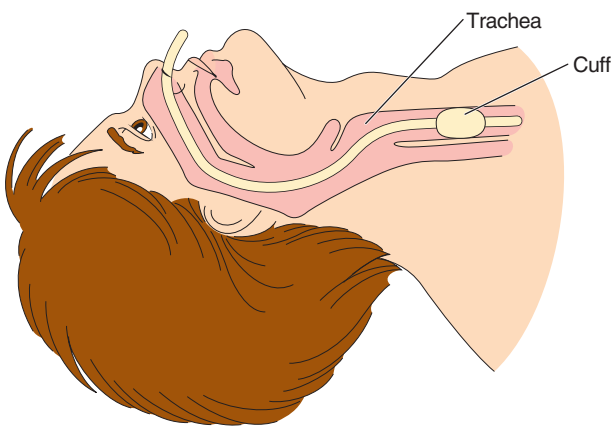


FIGURE 36.13 ■ Nasal endotracheal (nasotracheal) intubation

A tracheostomy may be performed if long-term ventilatory support is required. Although a tracheostomy is more comfortable and easier to secure in place, complications such as cuff necrosis and increased risk of infection are associated with tracheostomy as well as endotracheal intubation. Table 36.8 compares the advantages, disadvantages and possible complications of endotracheal tubes and tracheostomy.

When the person is able to maintain effective respirations and ventilatory support is no longer required, the endotracheal tube is removed (*extubation*). Gag, cough and swallow reflexes must be intact to prevent aspiration. After oxygenation and suctioning, the cuff is deflated and the tube removed. Humidified oxygen is provided immediately following removal. Close observation for respiratory distress is vital following extubation. Inspiratory stridor within the first 24 hours indicates laryngeal oedema, which may necessitate re-intubation. Sore throat and a hoarse voice are common after extubation. Oral intake is reinitiated slowly, with careful assessment of swallowing.

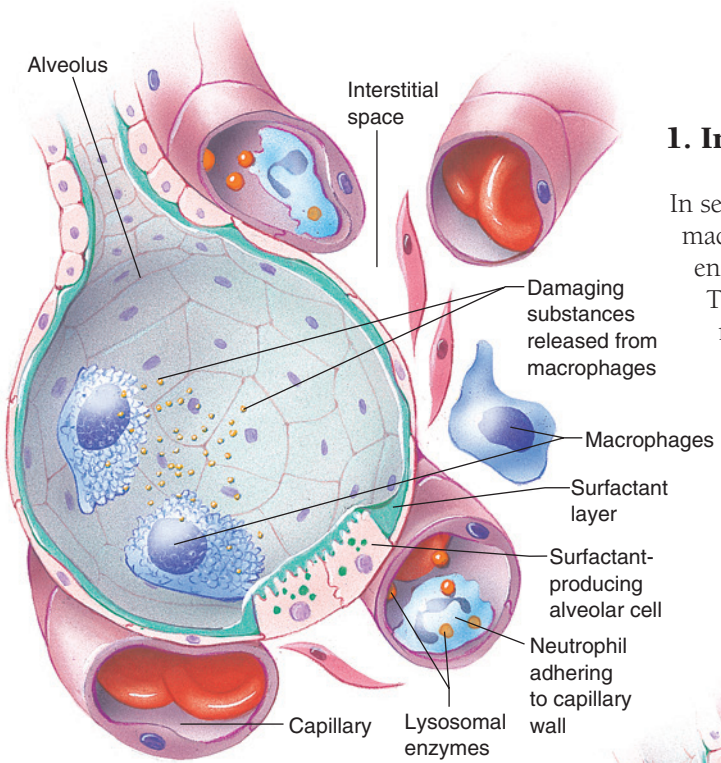
Mechanical ventilation

Mechanical ventilation is indicated when alveolar ventilation is inadequate to maintain blood oxygen and carbon dioxide levels. Specific indications for mechanical ventilation include:

- apnoea or acute ventilatory failure
- hypoxaemia unresponsive to oxygen therapy alone
- increased work of breathing with progressive fatigue.

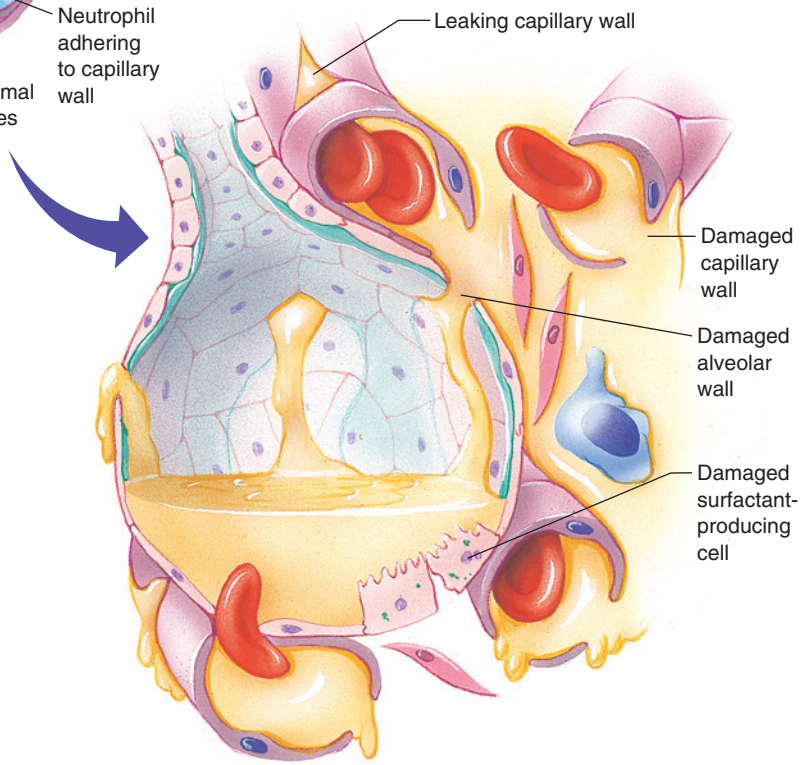
Acute respiratory distress syndrome

Acute respiratory distress syndrome (ARDS) is a severe form of acute respiratory failure that occurs in response to pulmonary or systemic insults. ARDS is characterised by non-cardiogenic pulmonary oedema caused by inflammatory damage to alveolar and capillary walls. Many disorders may precipitate ARDS, although sepsis is the most common.



1. Initiation of ARDS

In sepsis-induced ARDS, bacterial toxins cause macrophages and neutrophils to adhere to endothelial surfaces of the alveoli and capillaries. The macrophages release oxidants, inflammatory mediators, enzymes and peptides that damage the capillary and alveolar walls. In response, neutrophils release lysosomal enzymes, causing further damage.

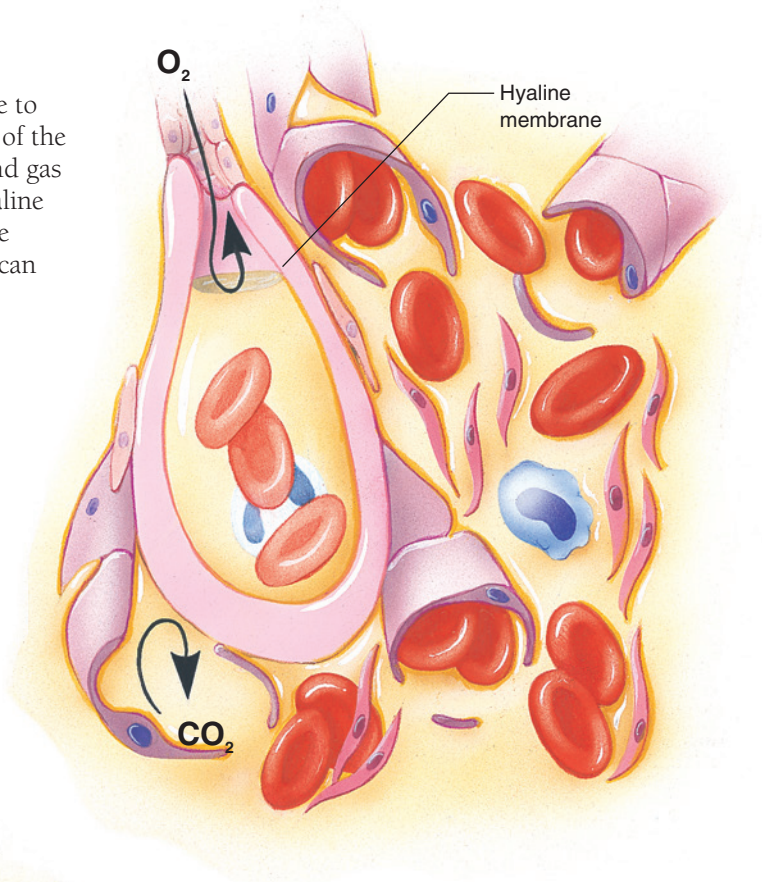


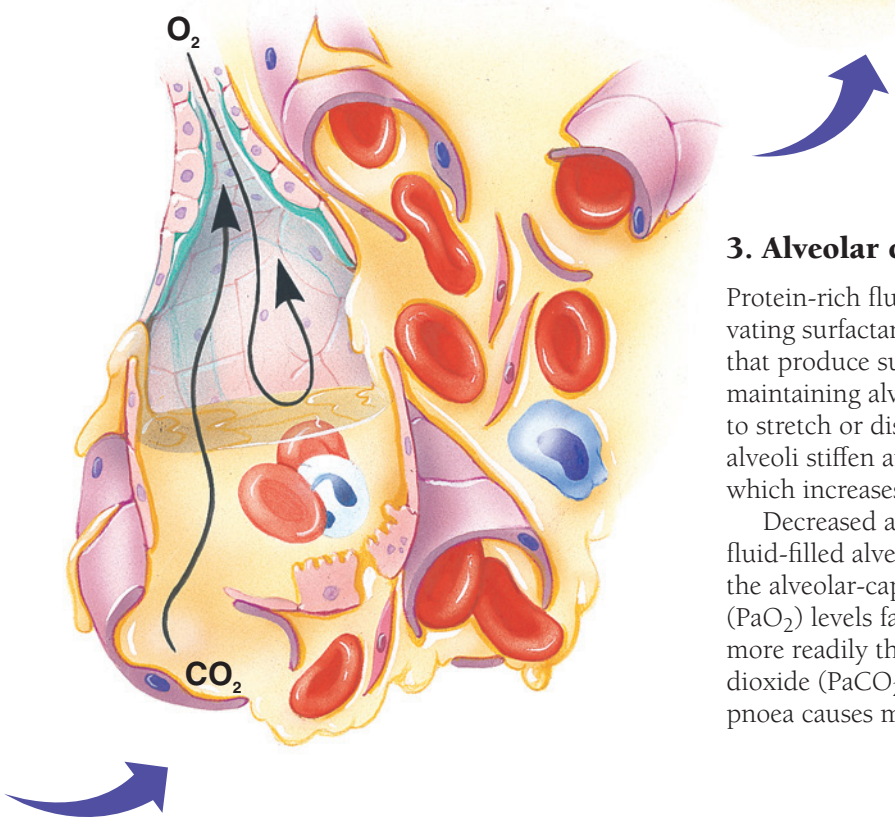
2. Onset of pulmonary oedema

The damaged capillary and alveolar walls become more permeable, allowing plasma, proteins and erythrocytes to enter the interstitial space. As interstitial oedema increases, pressure in the interstitial space rises and fluid leaks into alveoli. Plasma proteins accumulating in the interstitial space lower the osmotic gradient between the capillary and interstitial compartments. As a result, the balance is disrupted between the osmotic force that pulls fluid from the interstitial space into the capillaries and the normal hydrostatic pressure that pushes fluid out of the capillaries. This imbalance causes even more fluid to enter alveoli.

4. End-stage ARDS

Fibrin and cell debris from necrotic cells combine to form hyaline membranes, which line the interior of the alveoli and further reduce alveolar compliance and gas exchange. Because CO_2 cannot diffuse across hyaline membranes, $PaCO_2$ levels now begin to rise while PaO_2 levels continue to fall. Rising $PaCO_2$ levels can lead to respiratory acidosis. Without respiratory support, respiratory failure will develop.
Even with aggressive treatment, almost 50% of people with ARDS die.





3. Alveolar collapse

Protein-rich fluid accumulates in the alveoli, inactivating surfactant and damaging type II alveolar cells that produce surfactant. (Surfactant is important in maintaining alveolar compliance—the ability of tissue to stretch or distend.) As active surfactant is lost, the alveoli stiffen and collapse, leading to atelectasis, which increases breathing effort.

Decreased alveolar compliance, atelectasis and fluid-filled alveoli interfere with gas exchange across the alveolar-capillary membrane. Blood oxygen (PaO_2) levels fall. Because carbon dioxide diffuses more readily than oxygen, however, blood carbon dioxide ($PaCO_2$) levels also fall initially as tachypnoea causes more CO_2 to be expired.

TABLE 36.8 A comparison of endotracheal tubes and tracheostomy

	ADVANTAGES	DISADVANTAGES	POTENTIAL COMPLICATIONS
Oral endotracheal tube	• More easily inserted • Larger tube can be used, facilitating work of breathing, suctioning	• More difficult to secure • Can be obstructed by biting • Communication and mouth care more difficult • Increased risk of lower respiratory infection	• Obstruction or displacement • Pressure necrosis of lip • Tracheo-oesophageal fistula
Nasal endotracheal tube	• More easily secured and stabilised • Well tolerated • Facilitates communication and oral hygiene	• Necessitates smaller tube, which may impede removal of secretions • Increased risk of lower respiratory infection	• Obstruction or displacement • Pressure necrosis of nares • Obstruction of sinus drainage, possible sinusitis • Tracheo-oesophageal fistula
Tracheostomy	• Easily secured and stabilised • Enables swallowing, speech and oral hygiene • Avoids upper airway complications	• Requires surgical incision • Increased risk of lower respiratory infection	• Haemorrhage due to incision or vessel erosion by tube • Wound infection • Subcutaneous emphysema • Tracheo-oesophageal fistula • Tracheal infarction and stenosis

One of the most common indicators for ventilatory support is respiratory muscle fatigue or its potential. Drug overdose, neural disorders, chest wall injury and airway problems such as severe asthma or COPD can lead to acute ventilatory failure. Disorders that affect alveolar–capillary diffusion, such as pulmonary contusion, pneumonia and ARDS, may necessitate mechanical ventilation to attain adequate oxygenation. Positive-pressure ventilation increases lung volume, helps redistribute fluid from the alveolar to the interstitial space and helps reduce the oxygen demand caused by increased work of breathing.

TYPES OF VENTILATORS Two broad general classifications of mechanical ventilators are available. Negative-pressure ventilators create negative (subatmospheric) pressure externally to draw the chest outwards and air into the lungs, mimicking spontaneous breathing. The iron lung and cuirass ventilator are examples of negative-pressure ventilators. Negative-pressure ventilators are rarely used nowadays.

Positive-pressure ventilators are used more often than negative-pressure ones, especially in treating acute respiratory failure (see Figure 36.14). These ventilators push air into the lungs, rather than drawing it in like negative-pressure ventilators. Either invasive ventilation using an endotracheal tube or tracheostomy or non-invasive positive-pressure ventilation may be used. Increasingly, non-invasive techniques, which use a nasal or face mask, nasal plugs or an oral mouthpiece, are being used with various levels of success (Byrd, 2015).

Non-invasive ventilation (NIV) provides ventilator support using a tight-fitting face mask, thus avoiding intubation. Its primary use is to support individuals with obstructive sleep apnoea, neuromuscular disease or impending respiratory failure (e.g. advanced COPD). NIV also may be used for individuals in respiratory failure who refuse intubation. The degree of success varies, primarily limited by the person's intolerance due to the physical and psychological discomfort of wearing a mask when dyspnoeic (Soo Hoo, 2014). NIV tends to be more successful in individuals without significant underlying lung disease (e.g. respiratory failure related to neuromuscular disease).

Several variables are used to trigger, cycle and limit airflow with positive-pressure ventilators. The *trigger* prompts the ventilator to deliver a breath. The individual's inspiratory effort triggers ventilator-assisted breaths. *Ventilator-controlled breaths* usually are triggered by a preset time interval (e.g. a breath is delivered every 5 seconds for a rate of 12 breaths per minute). The *ventilator cycle*, or duration of inspiration, can be limited by volume, pressure, flow or time. *Volume-cycled ventilators* deliver air until a preset volume is delivered. *Pressure-cycled ventilators* cycle off when a preset pressure is achieved within the airways. *Flow-cycled ventilators* are cycled by a preset inspiratory flow rate, and *time-cycled ventilators* deliver air for a set time interval. Airflow delivered by the ventilator also can be limited by factors such as airway pressure (e.g. a volume-cycled ventilator can be set to immediately stop inspiratory flow if airway pressure exceeds a preset value).

MODES OF VENTILATION A number of different *modes* or patterns of ventilation may be used with positive-pressure ventilators. The mode determines whether a breath is initiated by the person or the ventilator and the pattern of airway support provided by the ventilator. CPAP, bi-level airway pressure support, assist-control mode ventilation, synchronised intermittent mandatory ventilation, positive end-expiratory pressure, pressure support ventilation and pressure-control ventilation are common modes of ventilation in use today (see Table 36.9).

Continuous positive airway pressure applies positive pressure to the airways of a person who is spontaneously breathing. CPAP may be used with either endotracheal intubation or a tight-fitting face mask. All breathing is spontaneous (triggered by the person) and pressure controlled. CPAP is used to help maintain open airways and alveoli, decreasing the work of breathing. *Bi-level ventilators (BiPAP)* provide inspiratory positive airway pressure as well as airway support during expiration. Bi-level ventilation is primarily used at night with a tight-fitting mask (nasal, facial

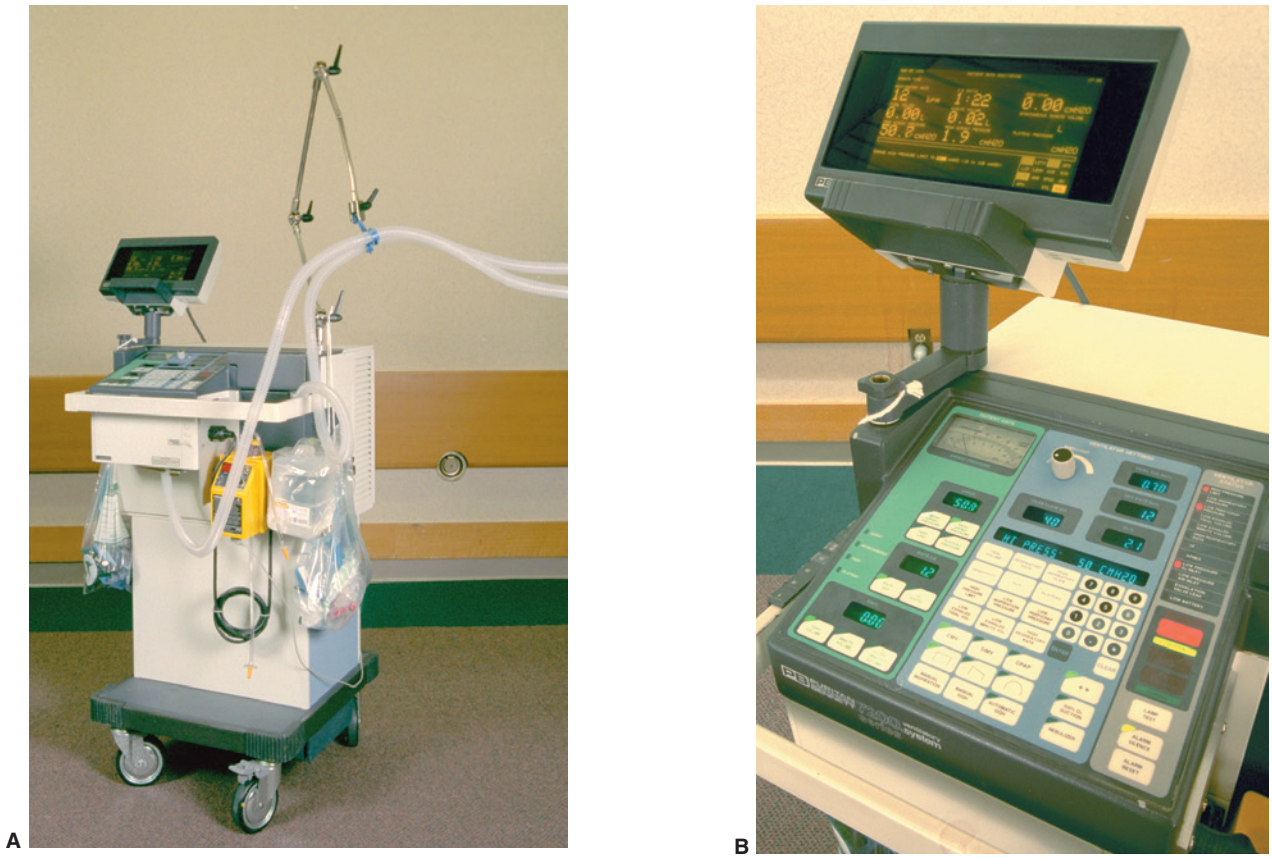


FIGURE 36.14 ■ *A*, Positive-pressure ventilator. *B*, Control panel used to set the mode, rate, limits and percentage of oxygen delivered

or oral). Three modes of ventilation can be used with BiPAP: spontaneous breathing (S); timed mode (T), in which pressure-supported breaths are delivered at a predetermined rate; and spontaneous/timed (S/T), in which the ventilator switches to timed mode if spontaneous breathing falls below a preset rate (International Ventilator Users Network, 2015).

Assist-control mode ventilation (ACMV or AC) is frequently used to initiate mechanical ventilation and when the person is at risk of respiratory arrest (e.g. overdose or head injury). Assisted breaths are triggered by inspiratory effort; however, if the respiratory rate falls below a preset number (e.g. 14 per minute), ventilator-controlled breaths are delivered. All breaths, assisted and controlled, are delivered at a specific tidal volume or pressure and inspiratory flow rate.

Synchronised intermittent mandatory ventilation (SIMV) allows the person to breathe spontaneously, without ventilator assistance, between delivered ventilator breaths. Mandatory or ventilator-controlled breaths are delivered at a preset rate, volume and/or pressure, coordinated with the individual's inspiratory efforts. New evidence appearing suggests that respiratory muscles may not be able to rest between mandatory breaths and, therefore, this mode may result in fatigue which actually prolongs the need for ventilation (Byrd, 2015).

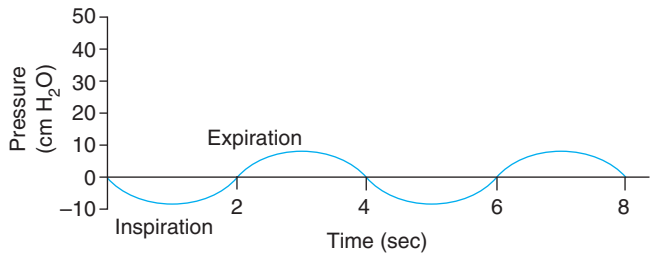
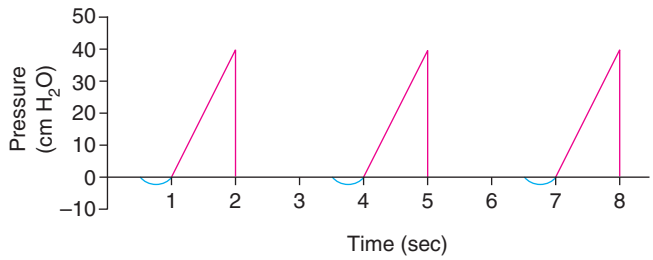
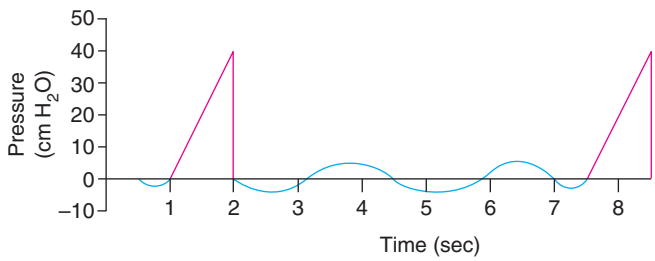
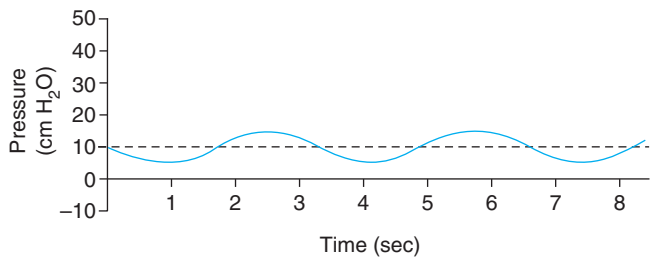
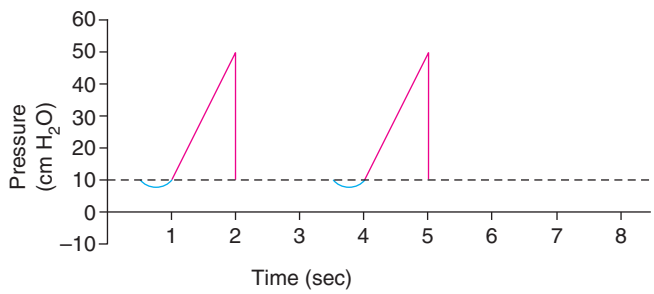
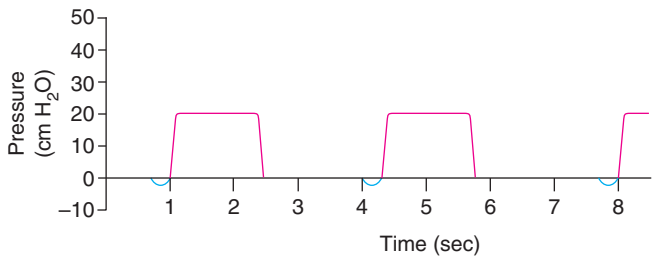
Positive end-expiratory pressure (PEEP) requires intubation and can be applied to any of the previously described ventilator modes. With PEEP, a positive pressure is maintained in the airways during exhalation and between breaths. Keeping alveoli open between breaths improves ventilation–perfusion relationships and diffusion across the alveolar–capillary membrane. This reduces hypoxaemia and allows use of lower percentages of inspired oxygen. PEEP is particularly useful for treating ARDS.

In *pressure support ventilation (PSV)*, ventilator-assisted breaths are delivered when the person initiates an inspiratory effort. The cycle is flow limited; inspiration is terminated when inspiratory airflow falls below a preset rate. This mode decreases the work of breathing. It can be used in combination with SIMV when the respiratory drive is depressed. Ventilator support can be gradually withdrawn during weaning.

Pressure-control ventilation (PCV), in contrast, controls pressure within the airways to reduce the risk of airway trauma (e.g. following thoracic surgery). Ventilation is time triggered and time cycled, but pressure is limited. The ventilator maintains a preset airway pressure throughout inspiration. Because all breaths are controlled by the ventilator, heavy sedation may be required to prevent competition between inspiratory effort and ventilator control.

Pressure-regulated volume control (PRVC) is a newer mode. It enables the desired tidal volume to be preset and permits the ventilator to deliver a pressure-limited (controlled) breath. Some benefits include automatic breath-to-breath

TABLE 36.9 Modes of positive-pressure ventilator operation

MODE	DESCRIPTION	PATTERN
Spontaneous breathing	Person has full control of rate, tidal volume, pressures.	
Assist-control mode ventilation (ACMV)	Person can trigger ventilator to deliver breaths at preset volume or pressure and inspiratory flow rate; breaths will be delivered at preset rate if person does not initiate.	
Synchronised intermittent mandatory ventilation (SIMV)	Mandatory breaths delivered by ventilator are synchronised with person's inspiratory effort.	
Continuous positive airway pressure (CPAP)	Positive pressure is maintained in airways; all breaths are spontaneous.	
Positive end-expiratory pressure (PEEP)	Used in conjunction with other ventilator modes; positive airway pressure is maintained throughout respiratory cycle.	
Pressure support ventilation (PSV)	Pressurised inspiratory flow supports the person's inspiratory effort, decreasing the work of breathing.	

adjusting according to the individual's lung compliance, resulting in the lowest peak inspiratory pressure needed to achieve a preset tidal volume.

VENTILATOR SETTINGS In addition to choosing the mode of ventilation, other parameters are set to meet individual's needs when positive-pressure ventilation is used (see Table 36.10).

For most adults, the rate is initially set between 12 and 15 breaths per minute. With ACMV or SIMV, the person's respiratory rate often is higher than the ventilator setting due to spontaneous breathing. Exhaled carbon dioxide ($ETCO_2$) or the $PaCO_2$ may be used to determine the rate. A $PaCO_2$ of less than 38 mmHg indicates hyperventilation and respiratory alkalosis; the set rate is reduced. A $PaCO_2$ above 42 mmHg or an $ETCO_2$ greater than 45 mmHg indicates hypoventilation and a need to increase the rate.

The tidal volume setting controls the amount of gas delivered with each ventilator breath. Depending on various factors, an average normal adult tidal volume at rest may range between 5 and 10 mL/kg of body weight. The tidal volume delivered by mechanical ventilation is slightly higher to compensate for tubing dead space. Higher tidal volumes can cause lung tissue trauma.

The percentage of oxygen delivered with ventilator breaths is adjusted to maintain the oxygen saturation and PaO_2 within acceptable ranges. Because prolonged delivery of high oxygen concentrations increases the risk of oxygen toxicity and pulmonary fibrosis, the FiO_2 is set at the lowest possible level for adequate tissue oxygenation. For most individuals, the goal is to maintain an oxygen saturation level of greater than 90%. Lower oxygen saturation levels may be appropriate for individuals with longstanding COPD.

COMPLICATIONS Although endotracheal intubation and mechanical ventilation can be lifesaving in respiratory failure, they are not without risk. Improper endotracheal tube placement or advancement of the tube into a mainstem bronchus can result in ventilation of one lung only. The inflated lung becomes over-distended and traumatised and the uninflated lung develops atelectasis. In non-invasive ventilation, associated complications include gastric dilation, aspiration, facial skin necrosis, drying of the eyes and mucous membranes, stress and claustrophobia (Soo Hoo, 2014).

Healthcare-associated pneumonia Infection is a significant risk associated with intubation and mechanical ventilation. Normal upper respiratory tract defence mechanisms are bypassed, with loss of air humidification and trapping of pathogens. Oral secretions and gastric contents can enter the respiratory tree through the open epiglottis. Frequent, meticulous oral hygiene, upright positioning with the head of the bed elevated >30 degrees, gastric ulcer prophylaxis, minimised ventilator time, removal of condensate from circuits and frequent and thorough hand hygiene from the healthcare professionals are vital interventions in reducing the risk of ventilator-associated pneumonia (Elliot, Aitken & Chaboyer, 2012). Often the cough reflex is inhibited or impaired by the underlying disease process and the continued presence of the endotracheal tube. Secretions often become thick and tenacious, increasing the risk of atelectasis.

Barotrauma *Barotrauma* (also called *volutrauma*) is lung injury due to alveolar over-distension. Both the volume of delivered gas and the pressures under which it is delivered can contribute to barotraumas. As a result, over-distended alveoli rupture, allowing air to escape into the pulmonary interstitial spaces and the mediastinum, pleural space and other tissues. Subcutaneous emphysema, pneumothorax and pneumomediastinum are possible results of barotrauma.

Subcutaneous emphysema, or air in the subcutaneous tissue, causes tissue swelling of the chest, neck and face. A 'crackling' or air-bubble-popping sensation is felt on palpation of subcutaneous emphysema. Swelling may be massive. Once the cause is corrected, the air is gradually reabsorbed.

Pneumothorax is identified by signs of unequal chest expansion, a sudden loss or significant decrease in breath sounds on the affected side and a hyper-resonant percussion tone. Rapid chest tube insertion is necessary to prevent tension pneumothorax and cardiovascular compromise.

Pneumomediastinum is the presence of air in the mediastinum, the space between the lungs that contains the heart, great vessels, trachea and oesophagus. Air in the mediastinal space can interfere with the function of all of these organs and lead to such complications as pneumopericardium (air in the pericardial sac). Pneumomediastinum may have few manifestations, but the chest x-ray shows widening of the mediastinal space.

Cardiovascular effects Positive-pressure ventilation increases intrathoracic pressure, which can interfere with venous return

TABLE 36.10 Ventilator settings

PARAMETER	DESCRIPTION
Rate (*f*)	Number of ventilator-delivered breaths per minute: usually 10 to 15 in adults using ACMV; may be lower in SIMV
Tidal volume (*VT*)	Amount of gas delivered with each ventilator breath: usually 8 to 10 mL/kg of body weight
Oxygen concentration (FiO_2)	Percentage of oxygen delivered with ventilator breaths; can be set between 21% (room air) and 100%
I:E ratio	Duration of inspiration to expiration: usually 1:2 to 1:1.5
Flow rate	Speed at which air is delivered
Sensitivity	Effort required to initiate a ventilator-assisted breath
Pressure limit	Maximal pressure within airways that will terminate a ventilator breath

to the heart and ventricular filling. As a result, cardiac output falls. Use of PEEP increases the effects of mechanical ventilation on cardiac output. The decreased cardiac output can affect liver and kidney function secondarily.

Gastrointestinal effects Gastrointestinal complications are commonly associated with prolonged mechanical ventilation. Stress ulcers (erosive gastritis) may develop, leading to painless gastrointestinal haemorrhage. Histamine H_2-receptor blockers are often used to prevent stress ulcers. Air leaks around the endotracheal tube can cause gastric distension; a nasogastric tube is inserted to prevent vomiting. Sedation and other medications used during mechanical ventilation can slow intestinal motility, leading to constipation.

WEANING The process of removing ventilator support and re-establishing spontaneous, independent respirations is called **weaning**. Weaning begins only after the underlying process causing respiratory failure has been corrected or stabilised. The process and time required for weaning depend on factors such as pre-existing lung condition, duration of mechanical ventilation and the person's general condition, both physical and psychological. In all cases, the vital signs, respiratory rate, extent of dyspnoea, blood gases and clinical status are used to evaluate weaning and its progress. More rapid weaning protocols are common in people with no underlying pulmonary condition who have only been ventilated for a short period of time. In this situation, care must be taken to avoid failed extubation (Elliot et al., 2012).

Following a brief period of mechanical ventilation, a T-piece or CPAP may be used for weaning. In T-piece weaning, the ventilator is removed for brief periods during which oxygen is delivered using a T-piece (see Figure 36.15). The duration of periods off the ventilator is gradually increased until the person can maintain adequate independent respirations for several hours. Vital signs, oxygen saturation, $ETCO_2$ and PaO_2 are carefully monitored during the process. The person is placed back on the ventilator at previous settings if signs of respiratory distress develop. When mechanical ventilation is no longer needed, the endotracheal tube is removed. CPAP weaning follows a similar process, with trials of spontaneous breathing supported by the ventilator in CPAP mode.

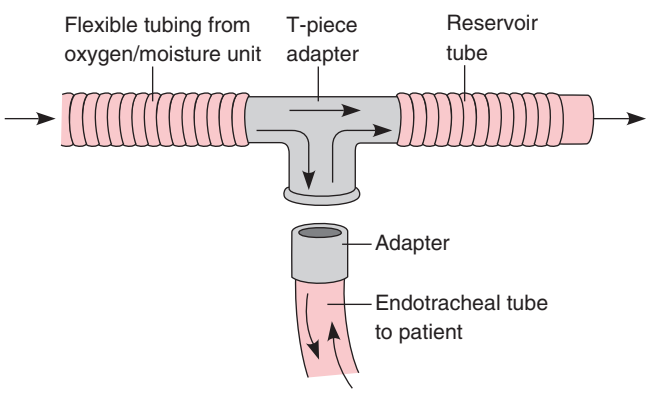


FIGURE 36.15 ■ A T-piece for weaning from mechanical ventilation

SIMV and PSV are used for weaning when the duration of mechanical ventilation has been longer and reconditioning of respiratory muscles is needed. When SIMV is used, the number of mandatory ventilator-assisted breaths is gradually decreased as ABGs, $ETCO_2$ and the respiratory rate are monitored.

Weaning is the primary use for pressure-support ventilation. Initially, PSV is set slightly below the peak inspiratory pressures required during volume-cycled ventilation. Pressure support levels are gradually decreased, often in a cyclical pattern of periods of minimal support alternating with higher support to recondition respiratory muscles. Various weaning protocols exist; however, some parameters used identify an individual's readiness for extubation including a respiratory rate of less than 25 breaths per minute, a tidal volume of greater than 5 mL/kg and a PaO_2 of greater than 80 mmHg (Byrd, 2015).

Terminal weaning When an illness is terminal or irreversible with a poor prognosis, terminal weaning may be requested by the person or their family. *Terminal weaning* is the gradual withdrawal of mechanical ventilation when survival without assisted ventilation is not expected. Unlike weaning when recovery is expected, which usually occurs in a critical care unit, the person is moved to a quiet critical care or hospice room or even home prior to initiating terminal weaning. Family members are encouraged to remain with the person throughout the process. If possible, decisions about sedation and analgesia prior to and during weaning are made with the person, as are decisions about hydration and nutritional support following weaning. Ventilator support is gradually withdrawn using the same modes described earlier (SIMV, PSV). Analgesia and sedation are given to promote comfort during weaning.

Nutrition and fluids

Attention also must be paid to fluid and electrolyte status and adequate nutrition. Mechanical ventilation promotes sodium and water retention due to its effects on cardiac output. Renal perfusion is decreased, stimulating the renin–angiotensin–aldosterone system to retain sodium and water. A Swan–Ganz catheter is often inserted to monitor pulmonary artery pressures and cardiac output. An arterial line allows repeated blood gas analysis and continuous arterial pressure monitoring. Serum electrolytes are drawn frequently and intake, output and daily weight are carefully monitored.

Enteral or parenteral nutrition are provided during mechanical ventilation because the endotracheal tube prohibits eating. A nasogastric, gastrostomy or jejunostomy feeding tube is placed for enteral nutrition. A jejunostomy tube may be used to reduce the risk of regurgitation and aspiration.

Nursing care

Health promotion

Education is a primary strategy to prevent respiratory failure. Teach all individuals and the public about the risks of smoking,

water safety, the value of a working smoke detector and measures to prevent smoke inhalation in a fire. Discuss the importance of pneumococcal vaccine and annual influenza immunisations for people who are at high risk, including those over age 65 and people with chronic diseases. Teach individuals with spinal cord injury or neuromuscular disease to use effective breathing and coughing techniques to maintain airway patency. Work with individuals addicted to narcotic drugs to attain and maintain drug-free status. Teach individuals with COPD about measures to reduce their risk of respiratory infection and symptoms to report to the doctor.

Assessment

Focused assessment data related to respiratory failure include the following:

- *Health history:* current manifestations, their duration and identified precipitating factors (may need to be obtained from family members if mental status is affected); history of previous episodes; chronic diseases such as COPD, occupational lung disease; current medications.
- *Physical examination:* LOC, mental status; vital signs; colour and oxygen saturation; respiratory assessment, including rate and depth, use of accessory muscles, respiratory excursion, auscultation; cardiovascular assessment, including heart rate and sounds, neck vein distension, peripheral pulses, evidence of clubbing.
- *Diagnostic tests*: ABGs, chest x-ray, pulmonary artery pressure and wedge pressure readings, cardiac output.

Nursing diagnoses and interventions

Individuals in respiratory failure are often unstable and critically ill. They require both intensive medical care and intensive nursing care. Priority nursing needs relate to maintaining ventilation and a patent airway. Perhaps less obvious, but no less critical, nursing care needs to relate to preventing injury and managing anxiety.

Impaired spontaneous ventilation

In acute respiratory failure, fatigue from the work of breathing may impair the ability to maintain adequate ventilation. This is a concern both prior to initiation of mechanical ventilation and during the weaning process.

- Assess and document respiratory rate, vital signs and oxygen saturation every 15 to 30 minutes. *Close monitoring is vital to detect early signs of increasing respiratory distress and inability to sustain adequate breathing.*

CONSIDERATION FOR PRACTICE

Promptly report signs of respiratory distress, including tachypnoea, tachycardia, nasal flaring, use of accessory muscles, intercostal retractions, cyanosis, increasing restlessness, anxiety or decreased LOC. These may be early manifestations of respiratory failure and inability to maintain ventilatory effort.

- Promptly report worsening ABGs and oxygen saturation levels. *Close assessment of these values allows timely intervention as needed.*
- Administer oxygen as ordered, monitoring response. Observe closely for respiratory depression, especially in the person with COPD. *Oxygen administration reduces the hypoxic respiratory drive. Chronically high $PaCO_2$ levels depress the respiratory centre; hypoxaemia may provide the only respiratory drive.*
- Place in Fowler's or high-Fowler's position. *Sitting positions decrease pressure on the diaphragm and chest, improving lung ventilation and decreasing the work of breathing.*
- Minimise activities and energy expenditures by assisting with ADLs, spacing procedures and activities, and by allowing uninterrupted rest periods. *Rest is vital to reduce oxygen and energy demands.*

CONSIDERATION FOR PRACTICE

Avoid sedatives and respiratory depressant drugs. These medications can further depress the respiratory drive, worsening respiratory failure.

- Prepare for endotracheal intubation and mechanical ventilation:
  a. Obtain an intubation tray with a selection of sterile endotracheal tubes and laryngoscope with a variety of adult blades.
  b. Check laryngoscope bulb; replace battery or bulb as needed.
  c. Set up for endotracheal suction, bringing suction equipment and gloves to the bedside.
  d. Set up the ventilator.
  e. Notify radiology that a portable chest x-ray will be needed on completion of intubation to verify correct placement of the endotracheal tube.

  Intubation and mechanical ventilation may be required to maintain ventilation and gas exchange.
- Explain the procedure and its purpose to the person and their family, providing reassurance that this is a temporary measure to reduce the work of breathing and allow rest. Inform the person and their significant others that talking is not possible while the endotracheal tube is in place and establish a means of communication. *Thorough explanation is important to relieve anxiety.*

Ineffective airway clearance

Ineffective airway clearance may either cause respiratory failure or occur as a result of interventions. Impaired ventilation frequently leads to acute respiratory failure, particularly in a person with COPD or asthma. Chest trauma also can impair airway patency as a result of pulmonary contusion and ineffective cough. Although intubation and mechanical ventilation can be lifesaving measures, they also increase the risk of respiratory infection and ineffective secretion management.

- Suction as needed to maintain a patent airway. Indicators for suctioning include crackles and rhonchi on auscultation, frequent coughing or setting off of the high-pressure alarm, and increasing restlessness or anxiety. Procedure 36.1 outlines endotracheal suctioning. *Although individuals with a tracheostomy can usually cough up secretions, the length and diameter of endotracheal tubes*

PROCEDURE 36.1 Endotracheal suctioning

GATHER SUPPLIES

- Suction unit with connecting tubing and connector at the bedside
- If an in-line suction catheter is not present:
  a. sterile suction catheter (a simple formula for sizing is:
     if ETT < 6 mm (Fr = ETT size (mm) x 2)
     if ETT ≥ 6 mm (Fr = ETT size (mm) – 2 x 2)
  b. a solution (e.g. water) to clean suction tubing following intervention
- Personal protective devices as indicated: goggles, mask, gown

BEFORE THE PROCEDURE

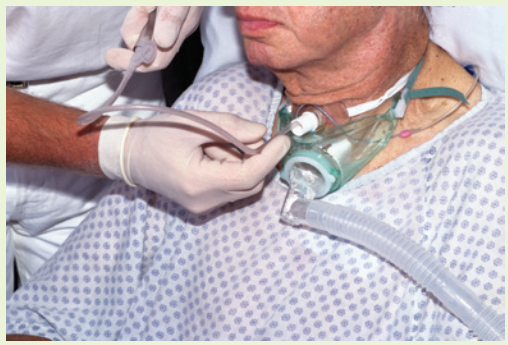
Explain the procedure and why it is being done. Tell the person that although suctioning is not painful, it is uncomfortable. While suction is being applied, breathing is difficult but these periods last only 10 seconds. Stress that suctioning allows removal of secretions and stimulates coughing, and helps clear secretions. Establish a means of communicating; for example, tell the person to raise a finger or rapidly blink if unable to tolerate suctioning.

DURING THE PROCEDURE

1. Use standard precautions.
2. Prepare the suction unit by turning it on and regulating it to no more than –80 to –120 mmHg.
3. Open sterile saline bottle and pour approximately 50 mL of saline into the opened sterile container.

With an in-line catheter

- Wearing exam gloves, attach the catheter to the suction tubing.
- Adjust the oxygen (FiO_2) to 100%; allow three breaths.
- Manipulating the catheter through the plastic shield (to maintain its sterility), insert the catheter without applying suction until resistance is met; apply suction while slowly withdrawing the catheter with a twirling motion (see photograph).
- Suction for no longer than 10 seconds (count the seconds or watch the clock—the time passes more quickly than you think. Holding your own breath during this time also gives you an indication of how quickly the technique needs to be completed), then allow to rest for three to five breaths. Repeat the procedure as needed for a total of no more than three times.
- Remove suction tubing from the catheter, clear the tubing, turn off suction and remove and discard gloves.



Endotracheal suctioning

Source: © Ansell Horn/Phototake.

As an open suction technique

- Open suction catheter. Ensure that there is water to clean the catheter between attempts.
- With a non-touch technique, attach catheter to suction tubing, keeping dominant hand clean; remove the outside package with the non-dominant hand and take the sterile catheter with the dominant hand.
- Use the non-dominant hand to adjust oxygen (FIO_2) to 100%; allow three breaths.
- Using the non-dominant hand, disconnect ventilator tubing from the endotracheal tube. Manipulating the suction catheter with the dominant hand and the suction control valve with the non-dominant hand, insert the catheter, without applying suction, until resistance is met. Then, apply suction intermittently while slowly withdrawing the catheter, using a twirling motion.
- Suction for no longer than 10 seconds. Reconnect the ventilator and allow the person to rest for three to five breaths; clear suction tubing as per protocol.
- Repeat the preceding two steps as needed for a total of three times.
- Reconnect ventilator tubing to the endotracheal tube.
- Clear suction tubing, turn off suction and remove the catheter, discarding it with the gloves.

4. Provide three additional breaths at 100% oxygen and then readjust to the previous ordered level.
5. Note colour, quantity, consistency and odour of sputum.
6. Assess lung sounds and tolerance of the procedure.
7. Wash hands.

AFTER THE PROCEDURE

Document results of suctioning procedure, along with the character of the sputum and the person's tolerance of the procedure. Report changes in sputum character, such as purulence or an odour that may indicate infection.

make this extremely difficult. Even with humidification, secretions often become thick and tenacious, further inhibiting their removal.

CONSIDERATION FOR PRACTICE

Frequently assess respiratory rate, chest movement, lung sounds, oxygen saturation, $ETCO_2$ and ABGs. Intubation and mechanical ventilation do not ensure adequate oxygenation and ventilation. Displacement of the endotracheal tube or obstruction by respiratory secretions impairs ventilation.

- Obtain sputum for culture if it appears purulent or is odorous. *Culture is necessary to identify pathogens and guide antibiotic therapy.*
- Perform percussion, vibration and postural drainage as required. *These techniques help loosen secretions and move them into larger airways for removal by coughing or suctioning.*

CONSIDERATION FOR PRACTICE

Evaluate endotracheal tube cuff pressure by measurement (no more than 20 to 25 mmHg of pressure). The minimum effective cuff pressure to maintain alveolar ventilation is used to reduce the risk of tracheal ischaemia and necrosis.

- Firmly secure endotracheal or tracheostomy tube. Provide adequate slack on ventilator tubing to prevent tension on the tube when turning, positioning or transferring to chair or stretcher. *These measures are important to ensure proper airway placement and prevent its inadvertent removal.*
- Assess fluid balance and maintain adequate hydration. *Adequate hydration helps liquefy secretions.*

Risk of injury

Many factors increase the risk of injury in acute respiratory failure. Hypoxaemia and hypercapnia affect the level of consciousness and may impair mental status. Endotracheal intubation and mechanical ventilation carry risks of tracheal damage and trauma to the lungs. Neuromuscular blockade, if used, presents a significant risk of injury as the person is unable to breathe spontaneously, communicate and move.

- Assess frequently, noting the following:
  a. LOC, orientation and awareness
  b. condition of mucosa of mouth and nose
  c. respiratory: lung sounds, chest excursion and ventilator pressures
  d. cardiovascular: vital signs, skin colour, capillary refill and peripheral pulses
  e. gastrointestinal: bowel sounds; test gastric secretions and faeces for occult blood
  f. genitourinary: urine output, daily weight
  g. skin and extremities.

  Complications associated with respiratory failure and mechanical ventilation can affect many body systems. Frequent assessment allows early detection and intervention.
- Report condition changes such as increasing air leak around the cuff and decreased breath sounds or chest movement. *These may be manifestations of a complication of intubation and ventilation, such as tracheal necrosis, displacement of the endotracheal tube into the right mainstem bronchus, pneumothorax or atelectasis.*

CONSIDERATION FOR PRACTICE

Do not bypass or turn off any ventilator alarms. A person who is intubated is unable to communicate verbally and cannot call for help. If neuromuscular blockers are used, the person is also unable to breathe without ventilator support.

- Turn and reposition frequently, taking care to stabilise endotracheal tube during movement. *Repositioning helps maintain tissue perfusion and prevent skin and tissue breakdown.*
- Keep skin and bed linen clean, dry and wrinkle-free. Protect pressure areas with padding, egg crate or heel and elbow protectors. *The person may not be able to perceive and report pain and move voluntarily to reduce pressure, necessitating excellent skin care.*
- Perform passive range-of-motion exercises every 4 to 8 hours. *These exercises maintain joint flexibility and help prevent contractures associated with long-term immobility.*
- Keep side rails up and use soft restraints as needed. *These safety measures are important to prevent falls, inadvertent disconnection of the ventilator or dislodging of the endotracheal tube.*
- Administer histamine H_2-blockers as ordered. *Stress gastritis and possible gastrointestinal haemorrhage are common, preventable complications of mechanical ventilation.*

Anxiety

Critical illness creates anxiety for any person. In acute respiratory failure, this anxiety is compounded by the presence of an endotracheal tube or tracheostomy, mechanical ventilator, numerous monitors and equipment, and, potentially, neuromuscular blockade and paralysis of voluntary muscles. Fear of continued dependence on the mechanical ventilator and inability to return to a normal life may compound this anxiety.

CONSIDERATION FOR PRACTICE

Frequently monitor anxiety level. High levels of anxiety increase oxygen use and often interfere with the ability to work with the respirator. This can increase hypoxaemia and further increase anxiety; intervention is necessary to break this cycle.

TRANSLATION TO PRACTICE Evidence-based practice for the person who is intubated

Individuals who are intubated cannot communicate verbally. In a cross-sectional, correlational study by Khalaila et al. (2011), 65 critically ill people who were intubated for at least 24 hours and extubated in the preceding 72 hours were investigated. The study measured communication difficulties and psychoemotional distress (involving fear and anger) related to endotracheal intubation. The researchers found that communication difficulties were a positive predictor for psychological distress. Stressful experiences were positively related to feelings of anger, and the number of different communication methods was negatively associated with feelings of fear and anger.

IMPLICATIONS FOR NURSING

People who are intubated and receiving mechanical ventilation are critically ill. Stress can contribute to physical deterioration; therefore, identifying and attempting to remove stressors can have a positive effect on a person's recovery. This research reinforces the need to ensure that methods to reduce frustration and fear related to communication difficulties from endotracheal intubation should be instigated.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Why is a person who is mechanically ventilated unable to communicate verbally?
2. What tools are available to assist the nurse in facilitating improved communication skills with people who are unable to communicate verbally?
3. Is there any way that a person who is undergoing neuromuscular blockade and mechanical ventilation can communicate with the nurse? Which factors need to be considered in this circumstance?
4. How can a nurse be sure that the information the person is trying to express is the same information that is being received by the nurse?

See the accompanying 'Translation to practice' box for information about assessing and managing pain in individuals who are intubated.

- Remain with the person as much as possible. *The frequent and continuing presence of a caregiver provides reassurance that help is readily available.*
- Explain all monitors, procedures, unusual sounds and machinery. *Understanding of the environment and various sounds and alarms reduces anxiety.*
- Provide a simple means of communication, such as a slate, picture board or alphabet board. Reassure that endotracheal tube removal restores the ability to speak. *The inability to speak and call out for help is frightening for the person. Providing an alternate means of communication helps reduce anxiety.*
- Encourage frequent family visits, especially if the time of visitations is being limited. Encourage family participation in care. *Family visits help reduce anxiety and feelings of abandonment. Allowing family members to participate in care helps reduce their anxiety as well.*
- Explain to the family that the person can hear and understand. Emphasise the importance of talking to the person, not over or about the person. *The family may not understand that the person may be mentally alert although unable to respond. Talking to the person about everyday things reduces the person's sense of isolation and fear.*
- Provide distraction with radio or television if available. *Distraction helps reduce the focus on machines and unusual sounds of monitors and alarms.*
- Attend to physical needs promptly and completely. *This provides reassurance that needs will be met even though the person is unable to ask for assistance.*
- Reassure that intubation and mechanical ventilation are temporary measures to allow the lungs to rest and heal. Reinforce that the person will be able to breathe independently again. *The person may fear continued dependence on mechanical ventilation.*

CONSIDERATION FOR PRACTICE

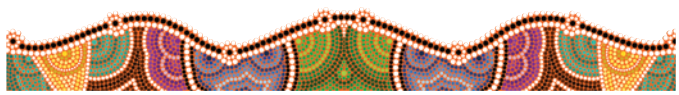
Provide sedation and anxiolytic medications as needed, especially when neuromuscular blockade is used. Although neuromuscular blockade paralyses voluntary muscles, the level of consciousness is unimpaired.

Community-based care

Prior to hospital discharge, teach the person and their family about the following topics:

- factors that precipitated respiratory failure and measures to prevent it in the future (e.g. the impact of respiratory irritants on compromised lungs)
- measures to prevent future episodes, such as remaining indoors with an air filter or air conditioning when pollution levels are high, obtaining influenza and pneumonia immunisations, and avoiding exposure to cigarette smoke
- effective coughing and pulmonary hygiene measures such as percussion, vibration and postural drainage.

Acute respiratory failure resulting from an acute insult such as pneumonia or near-drowning often resolves with few long-term sequelae. When respiratory failure results from an underlying disease such as COPD, the prognosis is less optimistic. Individuals with end-stage COPD may have repeated episodes of respiratory failure, with a gradual loss of respiratory function and reserve. These individuals may choose terminal weaning rather than a future of increasing disability. Discuss what to expect during the terminal weaning process with the person and their family. Discuss use of sedation prior to and during the weaning process. Explain that medications are used to reduce respiratory distress and dyspnoea during weaning. Assure the person and family that nursing support is continuously available during the weaning process and that family and other supporters such as clergy are allowed to remain with the person.



THE PERSON WITH ACUTE RESPIRATORY DISTRESS SYNDROME

Acute respiratory distress syndrome (ARDS) is characterised by non-cardiac pulmonary oedema and progressive refractory hypoxaemia. First identified in 1967, ARDS has been known by various names, such as shock lung and adult hyaline membrane disease. It is widely recognised as a severe form of acute respiratory failure. In Australia, 12 people died of ARDS in 2013 (ABS, 2015c).

Although the exact cause of ARDS is unclear, it is known that ARDS does not occur as a primary process but may follow a number of diverse conditions producing direct or indirect lung injury (see Table 36.11). Individuals who develop ARDS as a complication of an acute lung injury or condition are more likely to fully recover than individuals with chronic conditions.

Pathophysiology

The underlying pathology in ARDS is acute lung injury resulting from an unregulated systemic inflammatory response to acute injury or inflammation. Inflammatory cellular responses and biochemical mediators damage the alveolar–capillary membrane. This damage develops rapidly, often within 90 minutes of the systemic inflammatory response and within 24 hours of the initial insult. Damaged capillary membranes allow plasma and blood cells to escape into the interstitial space. Increased interstitial pressure and damage to the alveolar membrane allow fluid to enter the alveoli. Within the alveolus, the fluid dilutes and inactivates surfactant. Surfactant-producing cells are damaged by the inflammatory process, leading to a deficit of surfactant, increased alveolar surface tension and alveolar collapse with atelectasis. The lungs become less compliant and gas exchange is impaired. As the syndrome progresses, hyaline membranes form, further reducing gas exchange and compliance. Finally, fibrotic changes occur in the lungs. Intra-alveolar septa thicken and alveolar surface area for gas exchange is reduced. Hypoxaemia becomes refractory or resistant to improvement with supplemental oxygen and the $PaCO_2$ rises as diffusion is further impaired. Figure 36.16 illustrates the pathogenesis of ARDS.

TABLE 36.11 Conditions associated with the development of ARDS

CONDITIONS	EXAMPLES
Shock	Haemorrhagic shock, septic shock
Inhalation injuries	Aspiration of gastric contents, smoke and toxic gases, near-drowning, oxygen toxicity
Infections	Gram-negative sepsis, viral pneumonias, *Pneumocystis carinii* pneumonia
Drug overdose	Heroin, methadone, aspirin
Trauma	Burns, head injury, lung contusion, fat emboli
Other	Disseminated intravascular coagulation, pancreatitis, uraemia, amniotic fluid and air emboli, multiple transfusions, open heart surgery with cardiopulmonary bypass

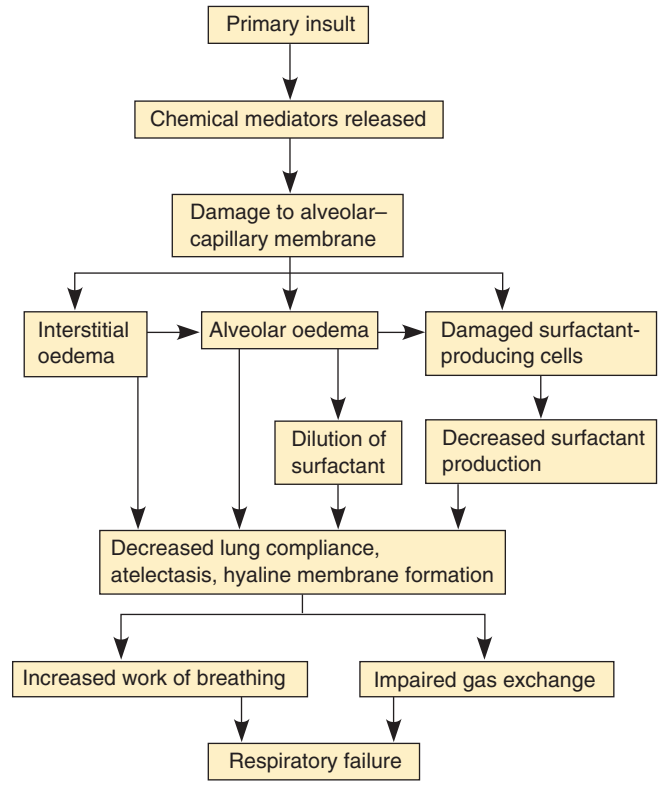


FIGURE 36.16 ■ The pathogenesis of ARDS

As ARDS progresses, tissue hypoxia becomes significant and metabolic acidosis develops. Carbon dioxide exchange is impaired as well as oxygen exchange, leading to combined respiratory and metabolic acidosis. Sepsis and multiple-organ-system dysfunction of the kidneys, liver, gastrointestinal tract, CNS and cardiovascular system are the leading causes of death in ARDS. If the process is halted before this occurs, the long-term prognosis for recovery is good.

Manifestations

Initial manifestations of ARDS typically develop 24 to 48 hours after the initial insult. Dyspnoea, tachypnoea and anxiety are early manifestations. Progressive respiratory distress develops, with increasing respiratory rate, intercostal retractions and use of accessory muscles of respiration. Cyanosis develops that may not improve with oxygen administration. Breath sounds are initially clear, but crackles (rales) and rhonchi develop later. As respiratory failure progresses, mental status changes such as agitation, confusion and lethargy occur.

INTERPROFESSIONAL CARE

ARDS management is directed towards identifying and treating its underlying cause and providing aggressive respiratory support.

- *ABGs* initially show hypoxaemia with a PaO_2 of less than 60 mmHg and respiratory alkalosis due to tachypnoea.

- *Chest x-ray* changes may not be evident for as long as 24 hours after the onset of ARDS. Diffuse infiltrates are seen initially, progressing to a 'white out' pattern. Chest CT scan provides a better illustration of the pattern of alveolar consolidation and atelectasis in ARDS (Horlander, 2014).
- *Pulmonary function testing* shows decreased lung compliance with reduced vital capacity, minute volume and functional vital capacity (see Box 33.1).
- *Pulmonary artery pressure monitoring* shows normal pressures in ARDS, helping distinguish ARDS from cardiogenic pulmonary oedema.

Medications

Although there is no definitive drug therapy for ARDS, a number of medications may be used. Inhaled nitric oxide reduces intrapulmonary shunting and improves oxygenation by dilating blood vessels in better ventilated areas of the lungs. Surfactant therapy may be prescribed. Surfactant is a complex mixture of phospholipids, neutral lipids and proteins that forms a thin layer atop a thin layer of water on the inner surface of the alveolus, reducing the surface tension within the alveoli. Surface tension tends to pull the walls of the alveoli together, increasing the likelihood of collapse during exhalation. Surfactant, by reducing surface tension, helps maintain open alveoli, decreasing the work of breathing, improving compliance and gas exchange, and preventing atelectasis.

Diagnosis

Refractory hypoxaemia (hypoxaemia that does not improve with oxygen administration) is the hallmark of ARDS.

Interventions to block the inflammatory response are under investigation, such as using non-steroidal anti-inflammatory agents and corticosteroids. Corticosteroids may be used late in the course of ARDS to improve oxygenation and lung mechanics when fibrotic changes occur.

Mechanical ventilation

The mainstay of ARDS management is endotracheal intubation and mechanical ventilation. With ARDS, it is rarely possible to maintain adequate tissue oxygenation with oxygen therapy alone.

With mechanical ventilation, the FiO_2 is set at the lowest possible level to maintain a PaO_2 higher than 60 mmHg and oxygen saturation of approximately 90%. When the PaO_2 cannot be maintained with less than 50% inspired oxygen, there is a risk that oxygen toxicity will accentuate ARDS. Often it is necessary to add CPAP, BiPAP or PEEP to mechanical ventilation settings to maintain blood and tissue oxygenation. Maintaining open airways and alveoli enhances gas diffusion and reduces ventilation–perfusion mismatch. PEEP decreases cardiac output and increases the risk of barotrauma, necessitating close monitoring. Either assist-control or SIMV may be used along with PEEP or CPAP in treating ARDS.

It is important to remember that mechanical ventilation does not cure ARDS; it simply supports respiratory function while the underlying problem is being identified and treated.

Treatments

Atelectasis frequently occurs in dependent lung regions in ARDS. Prone positioning in conjunction with mechanical ventilation reduces the pressure of surrounding tissue on dependent regions and improves oxygenation.

Other management strategies include careful fluid replacement, attention to nutrition, treatment of any infection and correction of the underlying condition. A Swan–Ganz line may be placed to monitor pulmonary artery pressures and cardiac output. Fluid replacement is carefully tailored to these measurements to avoid fluid imbalances, which may worsen hypoxia and ARDS. Enteral or parenteral feeding is necessary to maintain nutritional status and prevent tissue catabolism. Infections are treated with intravenous antibiotic therapy tailored to the causative organism. Low-molecular-weight heparin may be ordered to prevent thrombophlebitis and possible pulmonary embolus or disseminated intravascular coagulation, a possible complication of ARDS.

Nursing care

The nursing care needs of the person with ARDS are very similar to those of any individual with acute respiratory failure. Maintaining adequate ventilation and respirations is of highest priority, along with preventing injury and managing anxiety. See the section on acute respiratory failure for nursing care related to these diagnoses. Additional high-priority nursing care concerns for the person with ARDS are related to the effects of PEEP on cardiac output and potential problems of weaning ventilatory support. See the nursing care plan that follows for additional nursing interventions for the person with ARDS.

Nursing diagnoses and intervention

Decreased cardiac output

With positive-pressure ventilation, increased intrathoracic pressure decreases cardiac output. When PEEP is applied, intrathoracic pressure increases further; this can significantly decrease venous return, ventricular filling, stroke volume and cardiac output. Manifestations of decreased cardiac output include hypotension and compensatory tachycardia as the heart attempts to maintain cardiac output despite decreased stroke volume. In the person who is already hypoxic because of ARDS, this drop in cardiac output can increase tissue damage. Urine output falls and arrhythmias may develop.

- Monitor and record vital signs, including apical pulse, at least every hour; more frequently immediately following initiation of mechanical ventilation or the addition of PEEP. *Frequent assessment is vital to detect early signs of decreased cardiac output.*
- Assess level of consciousness at least every 4 hours. *Altered LOC, confusion and restlessness are early signs of cerebral hypoxia due to decreased cardiac output.*

- Monitor pulmonary artery pressures, central venous pressure and cardiac output readings every 1 to 4 hours. *Changes in these measurements may indicate worsening cardiac status.*
- Assess heart and lung sounds frequently. *Increasing crackles or abnormal heart sounds may indicate heart failure.*
- Weigh daily at the same time. *Accurate daily weights are the best indicator of fluid volume status.*
- Frequently provide good skin care, keeping skin clean and dry and protecting pressure points. *Tissue hypoxia increases the risk of skin breakdown, which in turn increases the risk of infection and sepsis.*
- Maintain intravenous fluids as ordered. *Intravenous fluids are given to maintain vascular volume and prevent dehydration.*
- Administer analgesics, sedatives and neuromuscular blockers as needed. *These medications may be prescribed to decrease cardiac workload.*

Dysfunctional ventilatory weaning response

The person with dysfunctional ventilatory weaning response has difficulty adjusting to reduced mechanical ventilator support, prolonging the weaning process. Airway congestion, inadequate rest or nutrition, pain, anxiety and a non-supportive environment are factors that can contribute to difficulty weaning. With ARDS, the pathological processes of the disease and its effects on gas exchange may be responsible for a prolonged or ineffective weaning process.

Assessment findings indicative of dysfunctional weaning include:

- dyspnoea, apprehension or agitation
- decreasing oxygen saturation level
- cyanosis or pallor, diaphoresis
- increased blood pressure, pulse and respiratory rate
- diminished or adventitious breath sounds, use of accessory muscles
- decreased LOC
- deteriorating ABG values
- shallow, gasping breaths or paradoxical abdominal breathing.

Nursing interventions for dysfunctional weaning include the following:

- Observe the person closely following changes in ventilator settings and during T-piece trials. *Vital signs—heart and respiratory rates, in particular—can provide early signs of hypoxaemia and poor tolerance of the weaning process.*

CONSIDERATION FOR PRACTICE

Frequently monitor oxygen saturation, $ETCO_2$ and ABGs following changes in ventilator settings. These values are used to assess the adequacy of ventilation and gas exchange during the weaning process.

- Place in Fowler's or high-Fowler's position. *Fowler's position facilitates lung expansion and reduces the work of breathing.*
- Fully explain all weaning procedures, along with expected changes in breathing. *Adequate explanations help reduce anxiety and improve the ability to cooperate.*
- Remain with the person during initial periods following changes of ventilator settings or T-piece trials. *This provides reassurance and allows close monitoring of the response.*
- Limit procedures and activities during weaning periods. *Reducing energy expenditures and cardiac work facilitates the weaning process.*
- Provide diversion, such as television or radio. *Diversion helps distract the focus from breathing.*
- Begin weaning procedures in the morning, when the person is well rested and alert; weaning may be discontinued overnight to provide rest. *The work of breathing increases during the weaning process; adequate rest is important.*
- Avoid administering drugs that may depress respirations during the weaning process (except as ordered at night to facilitate rest when ventilator support is provided). *Sedatives or analgesics that depress respirations can impair the weaning process.*
- Keep oxygen at the bedside following weaning and extubation. *Supplemental oxygen may be necessary to maintain adequate blood and tissue oxygenation.*

CONSIDERATION FOR PRACTICE

Frequently assess respiratory status following weaning and extubation. Keep an intubation kit readily available following extubation; be prepared for emergency re-intubation.

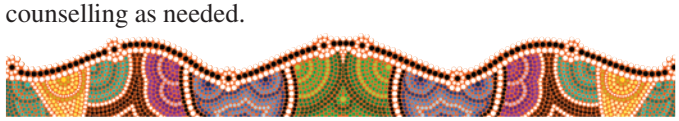
- Provide pulmonary hygiene with percussion and postural drainage. *Maintaining patent airways and adequate alveolar ventilation is vital during the weaning process.*

Community-based care

When preparing the person who has recovered from ARDS and the family for home care, discuss the following topics:

- ARDS did not result from something they did or did not do, but developed as a consequence of serious illness. Provide factual information about ARDS.
- Maximal respiratory function following ARDS is usually achieved within 6 months; respiratory function may remain significantly impaired. This may necessitate changes in occupation, lifestyle and family roles.
- Avoiding smoking and exposure to second-hand smoke and environmental pollutants is vital to prevent further lung damage.
- Obtain immunisation for pneumococcal pneumonia and annual influenza immunisations to prevent further episodes of serious respiratory disease.

Provide referrals to community health and respiratory care services as indicated, as well as for occupational therapy and counselling as needed.



NURSING CARE PLAN A person with ARDS

Peggy Adamson is a 36-year-old single woman (75 kg) admitted to the hospital following a near drowning at the local lake. On admission to the emergency department, Ms Adamson is alert and oriented, having been rescued and resuscitated within 2 minutes of the accident. Rescuers report that she seemed to have aspirated 'a lot' of water as she was water-skiing when the accident occurred. She is admitted to the critical care unit for observation. Oxygen is started per nasal prongs at 6 L/min, intravenous fluids are administered to correct electrolyte imbalances and 40 mg of frusemide is given intravenously for hypervolaemia.

ASSESSMENT

Throughout her stay, Ms Adamson has remained alert and oriented with stable vital signs. Her respiratory rate has been 20 to 24 per minute, with scattered crackles, oxygen saturations of around 94%, and a PaO_2 of 75 to 80 mmHg on 6 L/min of oxygen. Her pulse has been 96 to 100 and regular. On her initial assessment, the nurse notes that Ms Adamson seems apprehensive and anxious. Although her blood pressure is 116/74, unchanged from previous levels, her heart rate is up to 106 and respiratory rate is 28 per minute. Her lungs have scattered crackles but good breath sounds throughout, unchanged from previous assessments. Ms Adamson's oxygen saturation has dropped to 84%, her oxygen is increased to 8 L/min. ABG results show PaO_2, 65 mmHg; respiratory alkalosis, pH, 7.48; and $PaCO_2$, 32 mmHg.

Portable chest x-rays were ordered. The doctor orders a non-rebreather mask at 8 L/min and repeat ABGs in 1 hour. The chest x-ray reveals scattered infiltrates and a normal heart size.

Ms Adamson's oxygen saturation continues to fall and subsequent blood gases show a PaO_2 of 55 mmHg. The attending doctor diagnoses probable ARDS. Ms Adamson is intubated via endotracheal tube and is commenced on mechanical ventilation.

DIAGNOSES

- *Impaired gas exchange* related to effects of near drowning evidenced by increased respiration rate and decreasing oxygen saturations.
- *Anxiety* related to hypoxaemia evidenced by decreasing oxygen saturations.
- *Risk of decreased cardiac output* related to mechanical ventilation evidenced by decreasing blood pressure and increasing heart rate.
- *Risk of injury* related to endotracheal intubation evidenced by haemoptysis.

PLANNING

- Obtain all necessary supplies and radiology in preparation for intubation and mechanical ventilation.
- Explain the purpose and procedure of intubation.
- Provide an opportunity to express fears related to intubation and mechanical ventilation; answer questions and provide reassurance.
- Discuss communication strategies while intubated; obtain a magic slate.

Expected outcomes

- Breathe effectively with the mechanical ventilator.
- Demonstrate improved oxygen saturation, $ETCO_2$ and ABG values.
- Express fears related to intubation and mechanical ventilation.
- Demonstrate reduced anxiety levels (relaxed facial expression, ability to rest).
- Maintain adequate cardiac output and tissue perfusion.
- Tolerate endotracheal intubation and mechanical ventilation without evidence of infection or barotrauma.

IMPLEMENTATION

- Administer analgesics and/or sedatives as ordered.
- Monitor oxygen saturation and $ETCO_2$ levels every 30 to 60 minutes initially after mechanical ventilation is commenced; report changes to the doctor.
- Obtain ABGs as ordered or indicated; monitor and report results.
- Suction via endotracheal tube as needed to maintain clear airways.
- Provide periods of uninterrupted rest.
- Monitor vital signs every 1 to 2 hours.
- Assess skin colour, capillary refill and the presence of oedema every 4 hours.
- Monitor urine output hourly; report output of less than 30 mL per hour.
- Assess lung sounds and chest excursion every 1 to 2 hours.

EVALUATION

Ms Adamson is intubated and placed on a volume-cycled ventilator at 50% FiO_2 and a tidal volume of 730 mL in the assist-control mode at 16 breaths per minute. She has difficulty working with the ventilator initially, so morphine and midazolam are ordered to reduce her anxiety. Ms Adamson's oxygen saturation, $ETCO_2$ and ABG results do not begin to improve until 5 mmHg of PEEP is added to ventilator settings. After 3 days of mechanical ventilation with PEEP and aggressive fluid and diuretic therapy, Ms Adamson begins to improve. She is placed on SIMV and, over the course of another 3 days, is gradually weaned off the ventilator to a face mask with CPAP. She eventually recovers fully, with minimal apparent long-term effects.

CRITICAL THINKING IN THE NURSING PROCESS

1. Endotracheal intubation and mechanical ventilation were effective in supporting Ms Adamson's respiratory status as she recovered from ARDS. Discuss a possible sequence of events had it not been possible to wean her from the ventilator.
2. How might the presentation and management of an acute episode of respiratory failure due to ARDS differ from respiratory failure related to COPD?
3. What measures can nurses take to prevent the development of ARDS?
4. Develop a nursing care plan for Ms Adamson for the nursing diagnosis *Powerlessness* related to endotracheal intubation and mechanical ventilation.

REFLECTING ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future practice.
2. Communication from the nurse to the individual and their significant others is important. Given Ms Adamson's diagnosis of ARDS, what information and education should be communicated to both Ms Adamson and her significant others? Create a different list for both.

CHAPTER HIGHLIGHTS

- Obstructive disorders of the lower respiratory system, including asthma, COPD and cystic fibrosis, impair airflow into and out of the lungs, often affecting outflow of air to a greater extent than inflow. As a result, air trapped in the alveoli increases the residual volume of the lungs and reduces functional residual capacity. Alveolar ventilation is reduced as well. The net result is less available oxygen in the alveoli and impaired gas exchange.
- In many instances, acute episodes of asthma can be avoided through the use of inhaled steroids to reduce airway inflammation, inhaled long-acting bronchodilators and frequent self-monitoring of expiratory flow rate. Nursing care focuses on teaching for self-management and providing care during acute episodes of airway constriction.
- Chronic obstructive pulmonary disease (COPD) is a long-term process of progressive lung dysfunction. COPD involves two different disease processes: chronic bronchitis, characterised by airway oedema; and excessive mucus production and emphysema, characterised by destruction of supporting tissue with enlargement of respiratory bronchioles and alveolar spaces and loss of surface area for gas exchange.
- Smoking and exposure to tobacco smoke is the single greatest risk factor for COPD. A small percentage of cases result from an inherited deficiency of α_1-antitripsin, an enzyme that inhibits lung tissue destruction. Although smoking cessation does not reverse COPD, it does slow the progress of the disease.
- Cystic fibrosis, inherited as an autosomal recessive disorder, causes thick, viscous secretions in affected organs, primarily the lungs, pancreas, sweat glands and reproductive tract. In the lungs, small airway clearance is impaired, leading to atelectasis, bronchiectasis, infection and dilation of distal airways with air trapping and impaired gas exchange. Chest physiotherapy and early treatment of respiratory infections are key components of disease management. Ultimately, lung or heart–lung transplant may be required.
- Occupational lung diseases, pneumoconiosis and hypersensitivity pneumonitis damage interstitial tissues of the lungs, leading to fibrosis and scarring that causes the lungs to become stiff and non-compliant. Lung volumes decrease, the work of breathing increases and gas diffusion is impaired. Most occupational lung diseases are progressive and non-reversible. Interprofessional care is similar to that provided for individuals with COPD.
- Pulmonary vascular disorders affect blood flow through the pulmonary vascular system and gas exchange. Pulmonary embolism, obstruction of pulmonary blood flow, is a potentially critical condition usually resulting from deep venous thrombosis. Sudden onset of chest pain and dyspnoea with changes in haemodynamic status are possible manifestations of pulmonary embolism. Prevention through early ambulation, lower extremity exercises and sequential compression devices is the most effective treatment for pulmonary embolism.
- In primary and secondary forms of pulmonary hypertension, constriction of pulmonary vessels and remodelling of the pulmonary vascular bed increase pressure in the pulmonary system and right heart, ultimately leading to right-sided heart failure (cor pulmonale). Treatment focuses on slowing disease progression through oxygen therapy, administration of vasodilators and anticoagulants, and supporting function.
- Hypoventilation, impaired gas exchange and significant ventilation–perfusion mismatch (e.g. pulmonary embolism) can lead to respiratory failure. Hypoventilation leads to hypoxaemia and hypercapnia, whereas in impaired gas exchange or ventilation–perfusion mismatch, hypoxaemia predominates.
- The manifestations of respiratory failure relate directly to the effects of hypoxaemia (dyspnoea, restlessness, apprehension, impaired judgment, motor impairment, tachycardia, hyper- or hypotension, and cyanosis) and hypercapnia (dyspnoea, headache, peripheral and conjunctival vasodilation, papilloedema, neuromuscular irritability and decreased LOC).
- Respiratory support often is required, using positive-pressure ventilators. Variables of mechanical ventilation include the mode or cycle of ventilation, the flow rate and amount, pressures delivered and the oxygen concentration. Either invasive or non-invasive techniques may be used.
- Complications of mechanical ventilation include lung and mucous membrane trauma and infection, reduced cardiac output, gastric dilation, impaired communication and stress.
- ARDS is non-cardiac pulmonary oedema caused by a diffuse inflammatory response with increased pulmonary capillary permeability leading to interstitial and alveolar oedema and impaired gas exchange. As the process continues, lung compliance decreases, increasing the work of breathing, and atelectasis and consolidation of lung tissue develop. Respiratory failure with refractory hypoxaemia results.
- Mechanical ventilation and measures to support physiological function are the primary treatments for ARDS. The mortality rate, however, remains high at about 50%.

CONCEPT CHECK

1 All of the following nursing diagnoses are appropriate for a person with an acute asthma attack. Which is of highest priority?

1 *Anxiety* related to difficulty breathing
2 *Ineffective airway clearance* related to bronchoconstriction and increased mucus production
3 *Ineffective breathing pattern* related to anxiety
4 *Ineffective health maintenance* related to lack of knowledge about attack triggers and appropriate use of medications

2 The nurse caring for a person with asthma notices that the individual's respirations have slowed and he is no longer coughing. Breath sounds are diminished throughout his lung fields and absent in the bases. The nurse should:

1 notify the doctor
2 allow the person to rest undisturbed
3 obtain a chest x-ray
4 ask family members to leave

3 When teaching use of a metered-dose inhaler (MDI), the nurse's instruction should be:

1 take quick shallow breaths in rapid succession while holding the canister down
2 use the inhaler containing the anti-inflammatory drug first, then the bronchodilator
3 use the anti-inflammatory drug as needed to treat acute episodes of wheezing
4 rinse the mouth after using the inhaler to reduce systemic absorption of the drug

4 Which of the following would be an expected assessment finding in a person admitted with chronic obstructive airway disease?
1 AP chest diameter equal to or greater than lateral chest diameter
2 mental confusion and lethargy
3 3+ pitting oedema of ankles and lower legs
4 oxygen saturation readings of 85% or less

5 An appropriate goal for a person admitted with an acute exacerbation of COPD would be:
1 will verbalise self-care measures to regain lost lung function
2 arterial blood gases will be within normal limits by discharge
3 will maintain SpO_2 (saturation or peripheral oxygen) of 90% or higher
4 will identify strategies to help reduce number of cigarettes smoked per day

6 Which of the following statements best represents a nurse's understanding of use of supplemental oxygen in individuals with COPD?
1 The individual should not smoke because oxygen supports combustion.
2 Oxygen is used only at night for individuals with COPD.
3 Oxygen is never used for individuals with COPD because they may become dependent on it.
4 The person needs to be closely monitored for signs of respiratory depression.

7 The community health nurse working with a person who has cystic fibrosis specifically directs the home care aide to report which of the following? (Select all that apply.)
1 thick, tenacious milky white sputum
2 fever
3 bulky, fatty stools
4 difficulty clearing mucus secretions
5 increasing shortness of breath and fatigue

8 A person in skeletal traction suddenly develops right-sided chest pain and shortness of breath. The nurse should:
1 check for Homans' sign
2 start oxygen per nasal prongs
3 administer the prescribed analgesic
4 elevate the head of the bed to 45 degrees

9 The nurse caring for a person with COPD recognises which of the following as an early sign of possible respiratory failure?
1 restlessness and tachypnoea
2 deep coma
3 hypotension and tachycardia
4 decreased urine output

10 The nurse caring for a person undergoing mechanical ventilation for acute respiratory failure plans and implements which of the following measures to help maintain effective alveolar ventilation?
1 keeps the person in supine position
2 increases the tidal volume on the ventilator
3 maintains ordered oxygen concentration
4 performs endotracheal suctioning as indicated

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UNIT 9 BUILDING CLINICAL COMPETENCE

Responses to altered respiratory function

CLINICAL SCENARIO

You have been assigned to work with the following individuals on a respiratory unit. Significant data obtained during report are as follows:

- Jack Holt, a 65-year-old male, has bacterial pneumonia. Vital signs are T 38.3°C, P 94, R 30, BP 146/88. He is complaining of chest pain with breathing and has a productive cough of rusty-coloured sputum. His SpO_2 is 90% in room air.
- Maggie Sawyer, an 82-year-old female, is being discharged today following treatment for a deep venous thrombosis. She has congestive obstructive pulmonary disease. Suddenly she complains of difficulty breathing, chest pain, coughing, restlessness and a feeling that she is going to die.
- James Mohr, a 25-year-old male, sustained head, neck and chest injuries, and has a tracheostomy, from a motor vehicle crash. His vital signs were stable at the last assessment. He begins coughing and puts on his call light.
- Amy Campbell, a 30-year-old female, is being treated after having a severe asthma attack. Her current vital signs are T 37.2°C, P 64, R 26, BP 124/84. She has inspiratory and expiratory wheezing. Her SpO_2 is 94% on 4 L/min O_2 via nasal prongs.

Critical thinking questions

1 In what order would you visit these individuals after report?

1 _____
2 _____
3 _____
4 _____

2 Which top two priorities would you choose for each of the individuals presented above? Can you explain, if asked, the rationale for your choices?

	Priority #1	Priority #2
Jack Holt		
Maggie Sawyer		
James Mohr		
Amy Campbell		

3 The older adult is prone to respiratory problems due to which age-related changes in the respiratory system? (Select all that apply.)

1. loss of skeletal muscle strength in the thorax
2. increased elastic recoil of lungs during expiration
3. alveoli that are less elastic and more fibrotic
4. decreased residual volume of lung
5. decreased effectiveness of coughing

4 Bacterial pneumonia is spread by droplets. When using standard precautions, which equipment is necessary to prevent its spread?

1. Wear a gown when providing hygiene care for the person.
2. Wear a gown and gloves when touching the person.
3. Wear a mask and gloves when suctioning the person.
4. Wear a cap to keep hair from touching the individual.

5 For the individual receiving percussion and vibration with postural drainage for left lower lobe pneumonia, which position most facilitates removal of secretions?

1. semi-Fowler's position with arms elevated
2. right Sims' position with head in Trendelenberg
3. high-Fowler's position leaning on a bedside tray
4. left Sims' position with head flat

6 On discharge, the nurse teaches Mrs Sawyer ways to prevent pulmonary embolism. Which instruction is appropriate?

1. Use pillows under the knees when in bed.
2. Apply knee-high elastic stockings when ambulating.
3. Exercise the legs vigorously to encourage blood flow.
4. Stop every 1 to 2 hours to stretch legs when travelling.

7 Mr Mohr will be discharged with a tracheostomy. What should the nurse teach him about tracheostomies?

1. The tracheostomy will not interfere with lifting when returning to work.
2. Water-skiing is allowed, but swimming in a pool or lake is not allowed.
3. Showering is allowed as long as the tracheostomy is covered with a washcloth.
4. A small amount of alcohol is allowed but smoking is not allowed.

8 When admitted to the emergency department, which laboratory studies would you expect to obtain on a person with an inhalation injury?

1. arterial blood gases, carboxyhaemoglobin levels, electrolytes
2. sputum cultures and sensitivity, serology testing, sputum Gram stain
3. methaemoglobin levels, venous blood gases, white blood cell count
4. full blood cell count, oxygen saturation, sputum specimen

9 Arterial blood gases results are ordered on a person with acute respiratory distress. The results are: pH, 7.22; PaO_2, 50 mmHg; $PaCO_2$, 58 mmHg; HCO_3^-, 29 mEq/L. How would a nurse interpret these results?

1. respiratory acidosis
2. respiratory alkalosis
3. metabolic acidosis
4. metabolic alkalosis

10 Corticosteroids are ordered to decrease the inflammatory process of asthma. Which comment indicates the person understands how to take corticosteroids?

1. 'Corticosteroids have very few side effects to worry about.'
2. 'I understand that I cannot stop taking the medication abruptly.'
3. 'I can stop taking the corticosteroids as soon as I feel better.'
4. 'I can take an over-the-counter medication if I develop a cold.'

11 Which are early manifestations of pulmonary tuberculosis?

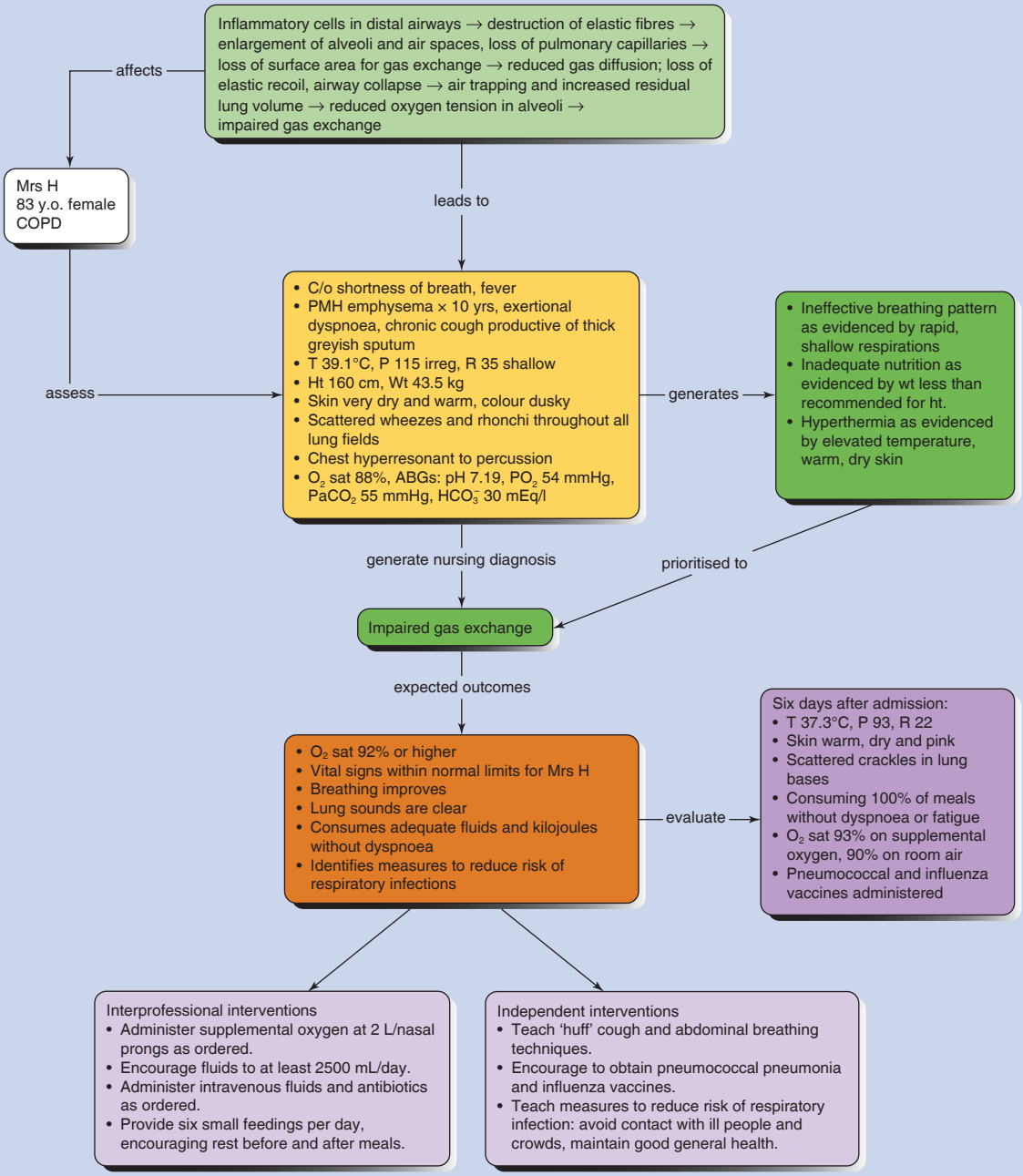
1. tachypnoea, tachycardia, activity intolerance
2. bradypnoea, foul-smelling sputum, weight gain
3. blood-tinged sputum, high-grade fever, fatigue
4. low-grade fever, night sweats, dry cough

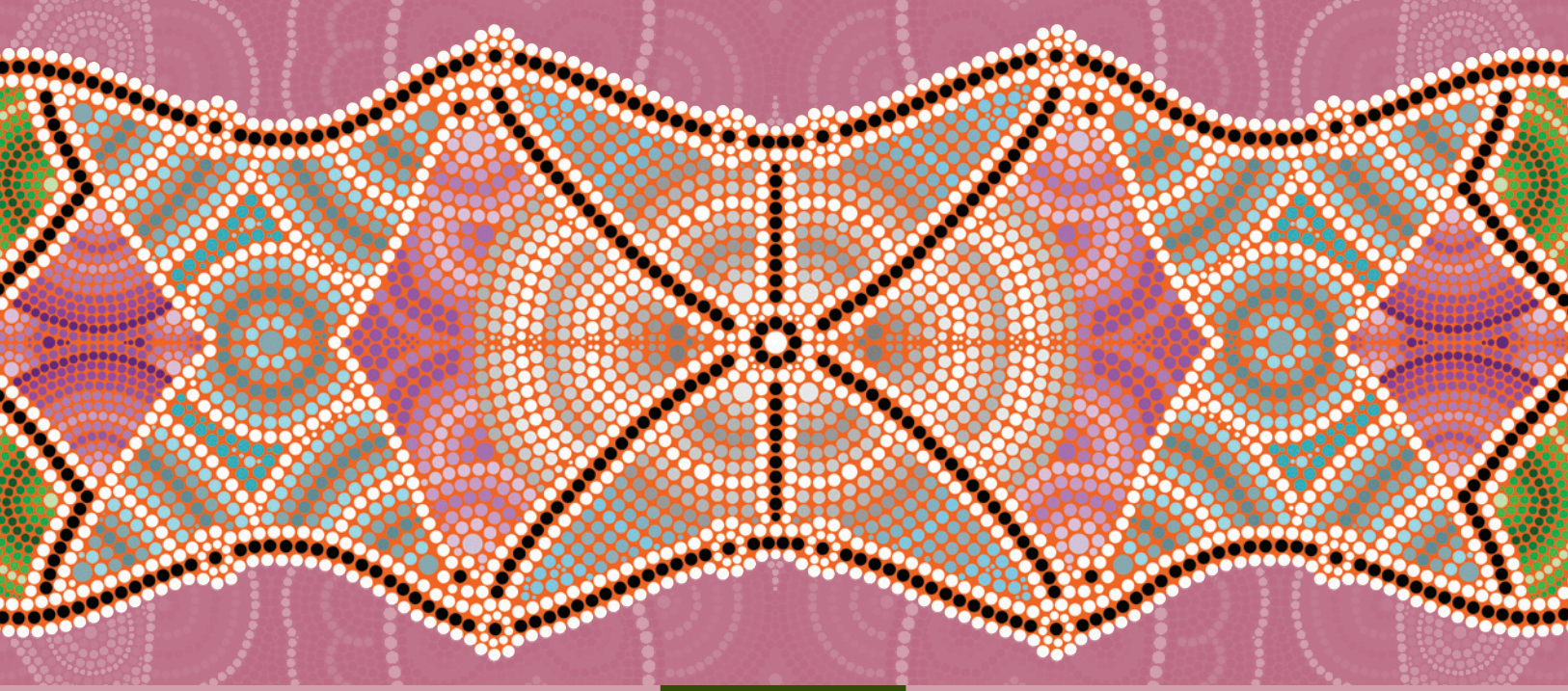
CASE STUDY

Gladys Hamer is an 83-year-old female who is seen in the emergency department with complaints of shortness of breath and fever. Her vital signs are T 39.1°C, P 115 and irregular, R 35 and shallow, BP 168/66. Her height is 160 cm and weight is 43.5 kg. On assessment, her skin is very dry and warm. Her colour is dusky. Scattered wheezes and rhonchi are heard throughout all lung fields. Her chest is hyper-resonant to percussion. A pulse oximeter is applied and the O_2 sat reading is 88%. Mrs Hamer has a past medical history of emphysema for 10 years, complaints of shortness of breath on exertion and has a chronic cough productive of thick, greyish sputum. Her spouse died 5 years ago and she has lived with her two adult sons since then.

Blood is drawn for arterial blood gases and results are pH, 7.19; PaO_2, 54 mmHg, $PaCO_2$, 55 mmHg; HCO_3^-, 30 mEq/L. These results indicate respiratory acidosis. Based on her current assessment, arterial blood gas results and past medical history, a medical diagnosis of chronic obstructive pulmonary disease (COPD) is determined.

When planning nursing care for Mrs Hamer, the nursing diagnosis of *Impaired gas exchange* related to acute and chronic lung disease is appropriate for implementing nursing interventions.





UNIT 10

RESPONSES TO ALTERED MUSCULOSKELETAL FUNCTION

CHAPTER 37

A PERSON-CENTRED APPROACH TO ASSESSING THE MUSCULOSKELETAL SYSTEM

DEBRA RAYMOND

LEARNING OUTCOMES

- Describe the anatomy, physiology and functions of the musculoskeletal system.
- Explain the normal movements allowed by synovial joints.
- Identify specific topics for consideration during a health history interview of the person with health problems involving the musculoskeletal system.
- Describe normal variations in assessment findings for the older adult.
- Identify manifestations of impairment of the musculoskeletal system.

CLINICAL COMPETENCIES

- Conduct and document a health history for people who have or are at risk of alterations in the musculoskeletal system.
- Conduct and document a physical assessment of musculoskeletal structures and functions.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Tape measure
- Goniometer

KEY TERMS

The tissues and structures of the musculoskeletal system perform many functions, including support, protection and movement. The musculoskeletal system has two subsystems: the bones and joints of the skeleton, and the skeletal muscles. These subsystems work together to allow the body to perform both gross and simple movements, such as closing a door, and fine, complex movements, such as repairing a watch.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE MUSCULOSKELETAL SYSTEM

The musculoskeletal system is composed of bones of the skeletal system; ligaments, tendons and muscles of the muscular system; and joints. The bones serve as the framework for the body and for the attachment of muscles, tendons and ligaments. Innervated by the nervous system, contraction and relaxation of muscles permit movement at joints.

The skeleton

Bones form the body's structure and provide support for soft tissues. They also protect vital organs from injury and serve to move body parts by providing points of attachment for muscles. Bones also store minerals and serve as a site for **haematopoiesis** (blood cell formation).

The human skeleton is made up of 206 bones (see Figure 37.1). Bones of the skeletal system are divided into the axial skeleton and the appendicular skeleton. The axial skeleton includes the bones of the skull, ribs, sternum and vertebral column. The appendicular skeleton consists of all the bones of the upper and lower limbs, shoulder girdles and pelvic girdle.

Bone structure

Bone cells include osteoblasts (cells that form bone), osteocytes (cells that maintain bone matrix) and osteoclasts (cells that resorb bone). Bone matrix is the extracellular element of bone tissue; it consists of collagen fibres, minerals (primarily calcium and phosphate), proteins, carbohydrates and ground substance. Ground substance is a gelatinous material that facilitates diffusion of nutrients, wastes and gases between the blood vessels and bone tissue. Bones are covered with periosteum, a double-layered connective tissue. The outer layer of the periosteum contains blood vessels and nerves; the inner layer is anchored to the bone.

Bones consist of a rigid connective tissue called osseous tissue of which there are two types: compact bone is smooth and dense; spongy bone contains spaces between meshworks of bone. Both types contain the same elements and are found in almost all bones of the body.

The basic structural unit of compact bone is the Haversian system (also called an osteon). The Haversian system consists of a central canal, called the Haversian canal; concentric layers of bone matrix, called lamellae; spaces between the lamellae, called lacunae; osteocytes within the lacunae; and small channels, called canaliculi (see Figure 37.2).

Spongy bone has no Haversian systems. Instead, the lamellae are arranged in concentric layers called trabeculae that branch and join to form meshworks. The spongy sections of long bones and flat bones contain tissue for haematopoiesis. In the adult, these sections, called red marrow cavities, are present in the spongy centre of flat bones (especially the sternum) and in only two long

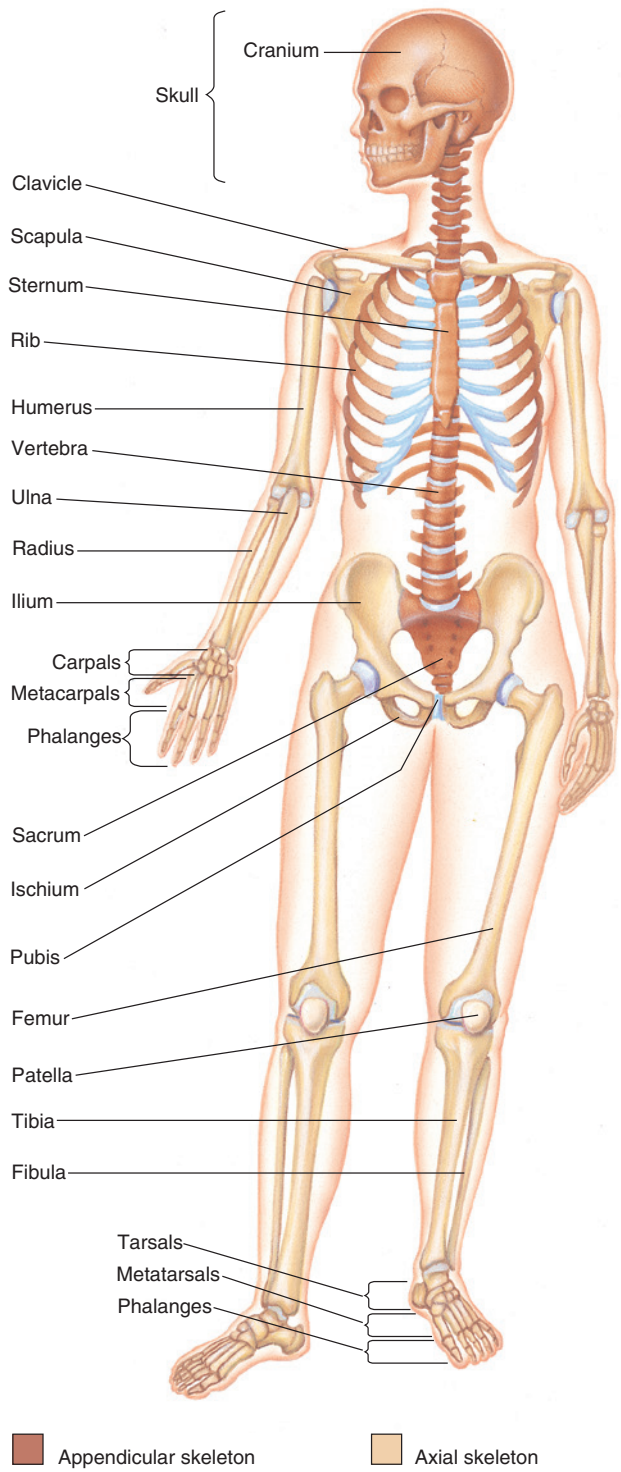


FIGURE 37.1 ■ Bones of the human skeleton

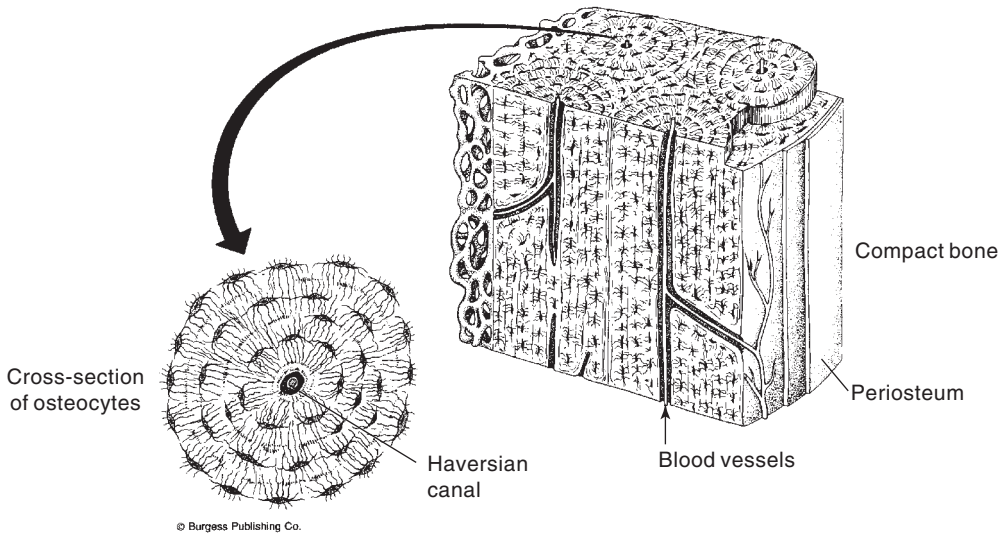


FIGURE 37.2 ■ The microscopic structure (Haversian system) of compact bone

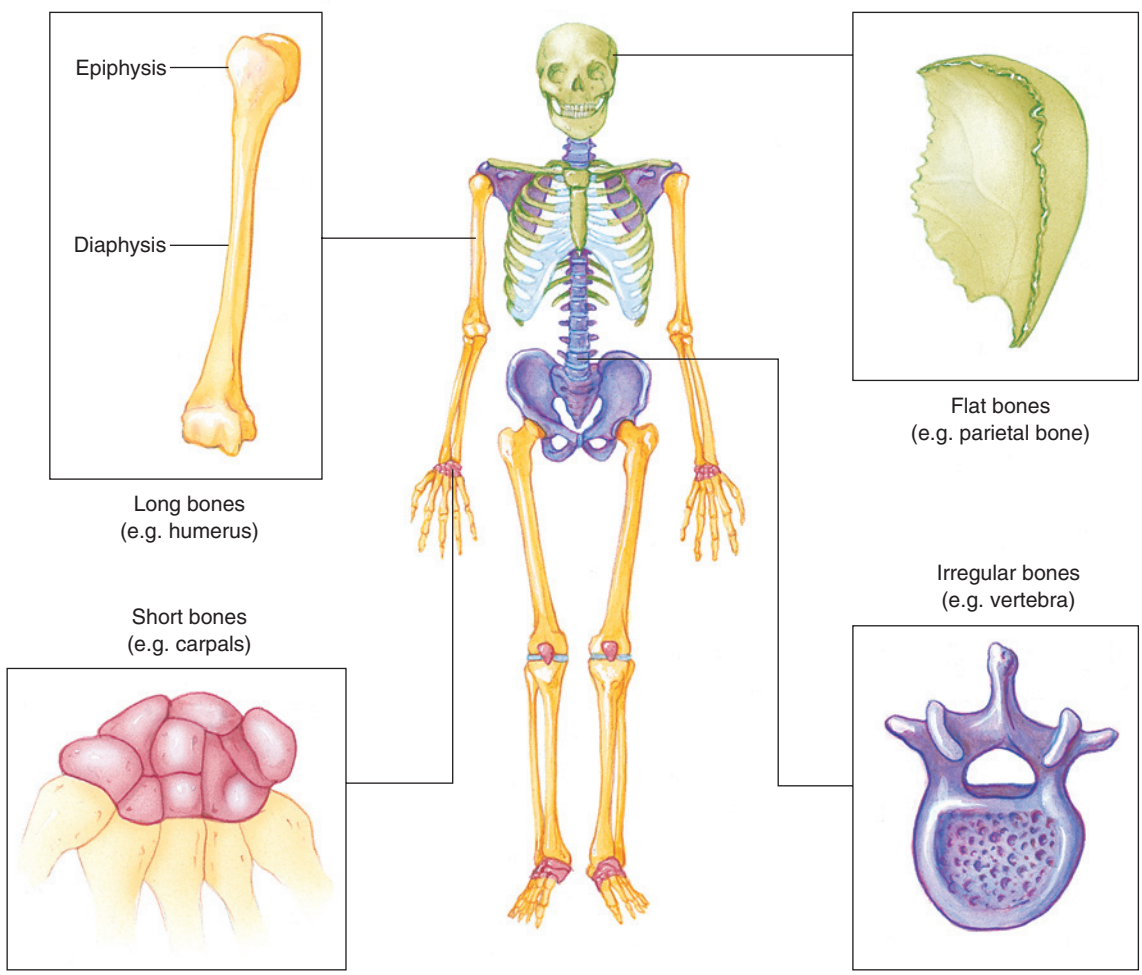


FIGURE 37.3 ■ Classification of bones according to shape

bones: the humerus and the head of the femur. This red marrow is active in haematopoiesis in adults.

Bone shapes

Bones are classified by shape (see Figure 37.3).

- *Long bones* are longer than they are wide. They have a midportion or shaft, called a diaphysis, and two broad ends, called epiphyses (see Figure 37.4). The diaphysis is compact bone and contains the marrow cavity, which is lined with endosteum. Each epiphysis is spongy bone covered by a thin layer of compact bone. Long bones include the bones of the arms, legs, fingers and toes.
- *Short bones*, also called cuboid bones, are spongy bone covered by compact bone. They include the bones of the wrist and ankle.
- *Flat bones* are thin and flat and most are curved. Their disc-like structure consists of a layer of spongy bone between two thin layers of compact bone. Flat bones include most bones of the skull, the sternum and the ribs.
- *Irregular bones* are of various shapes and sizes and, like flat bones, are plates of compact bone with spongy bone between. Irregular bones include the vertebrae, the scapulae and the bones of the pelvic girdle.

Bone remodelling in adults

Although the bones of adults do not normally increase in length and size, constant remodelling of bones, as well as repair of damaged bone tissue, occurs throughout life. In the bone remodelling process, bone resorption and bone deposit occur at all periosteal and endosteal surfaces. Hormones and forces that put stress on the bones regulate this process, which involves a combined action of the osteocytes, osteoclasts and osteoblasts. Bones that are in use, and are therefore subjected to stress, increase their osteoblastic activity to increase **ossification** (the development of bone). Bones that are inactive undergo increased osteoclast activity and bone resorption.

The hormonal stimulus for bone remodelling is controlled by a negative feedback mechanism that regulates blood calcium levels. This stimulus involves the interaction of parathyroid hormone (PTH) from the parathyroid glands and calcitonin from the thyroid gland. When blood levels of calcium decrease, PTH is released; PTH then stimulates osteoclast activity and bone resorption so that calcium is released from the bone matrix. As a result, blood levels of calcium rise and the stimulus for PTH release ends. Rising blood calcium levels stimulate the secretion of calcitonin, inhibit bone resorption and cause the deposit of calcium salts in the bone matrix. Thus, bones are necessary to regulate blood calcium levels.

Calcium ions are necessary for the transmission of nerve impulses, the release of neurotransmitters, muscle contraction, blood clotting, glandular secretion and cell division. Of the body's 1200 to 1400 g of calcium, over 99% is present as bone minerals.

Bone remodelling is also regulated by the response of bones to gravitational pull and to mechanical stress from the pull of muscles. Although the exact mechanism is not fully understood, it is known that bones that undergo increased stress are heavier and larger. This finding supports Wolff's law, which states that bone develops and remodels itself to resist the stresses placed on it.

The process of bone repair following a fracture is discussed in Chapter 38.

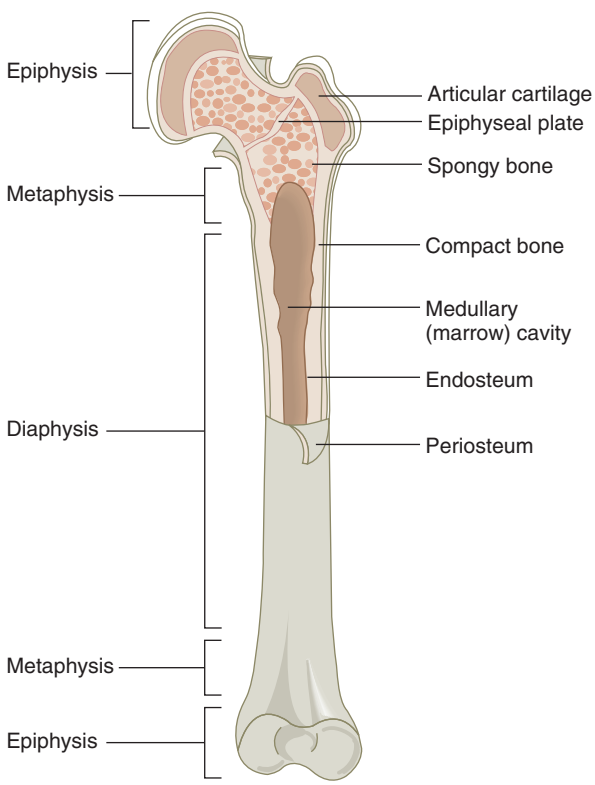


FIGURE 37.4 ■ Parts of a long bone

Muscles

The three types of muscle tissue in the body are skeletal muscle, smooth muscle and cardiac muscle (see Table 37.1). This discussion focuses on skeletal muscle, the only muscle that allows musculoskeletal function. Skeletal muscles attach to and cover the bones of the skeleton. Skeletal muscles promote body movement, help maintain posture and produce body heat. They may be moved by conscious, voluntary control or by reflex activity. The body has approximately 600 skeletal muscles (see Figure 37.5).

Skeletal muscles are thick bundles of parallel multinucleated contractile cells called fibres. Each single muscle fibre is itself a bundle of smaller structures called myofibrils. The myofibrils have alternating light and dark bands that give skeletal muscle

TABLE 37.1 Types of body muscle

TYPE	DESCRIPTION	EXAMPLES
Skeletal	Striated, voluntary muscle (can consciously move) can contract rapidly and vigorously, and is adaptable	Biceps, triceps, deltoid, gluteus maximus
Smooth	Non-striated, involuntary muscle (cannot consciously move)	Muscles in the walls of the bladder, stomach and bronchi
Cardiac	Striated, involuntary muscle	Heart muscle

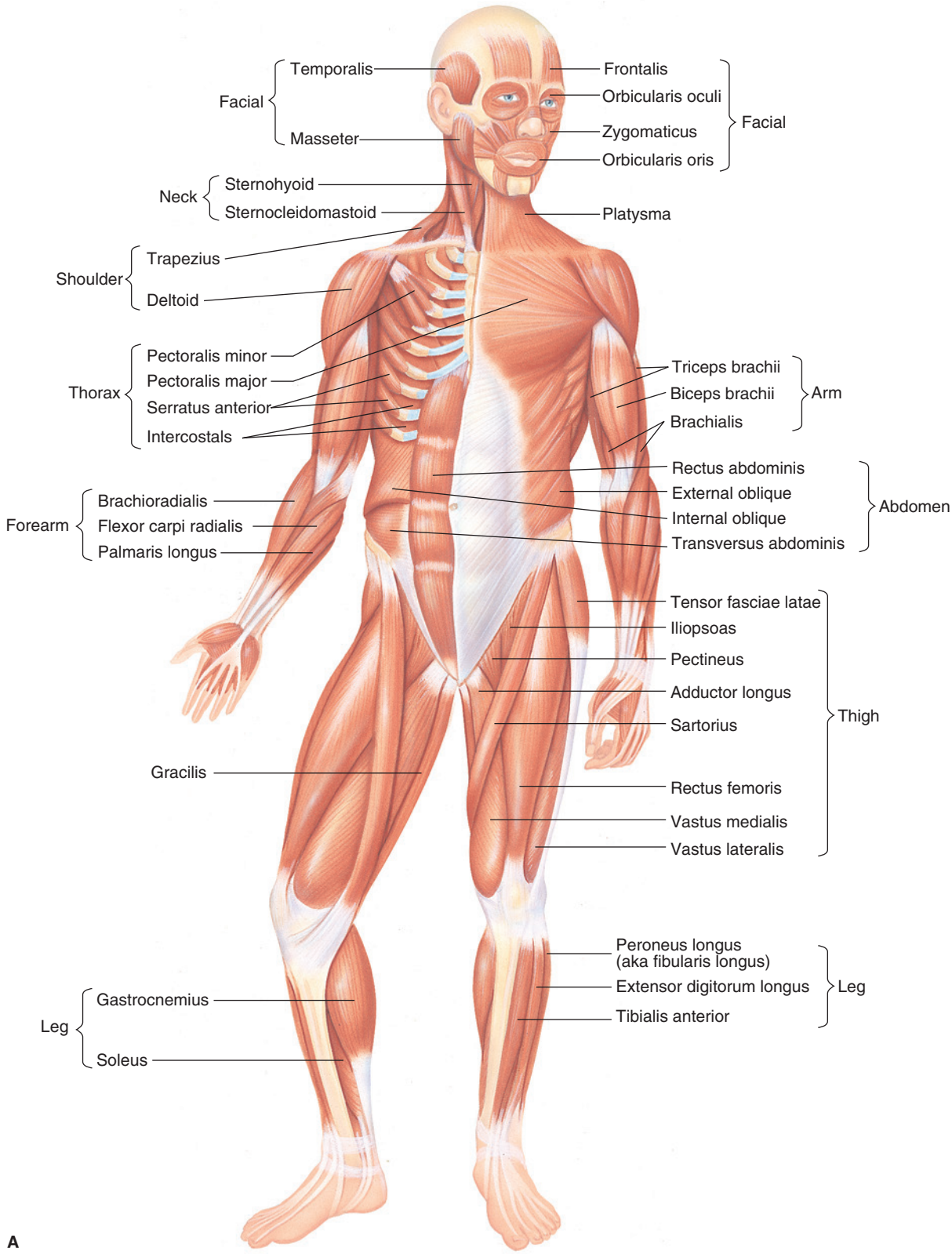


FIGURE 37.5 ■ *A*, Muscles of the anterior body

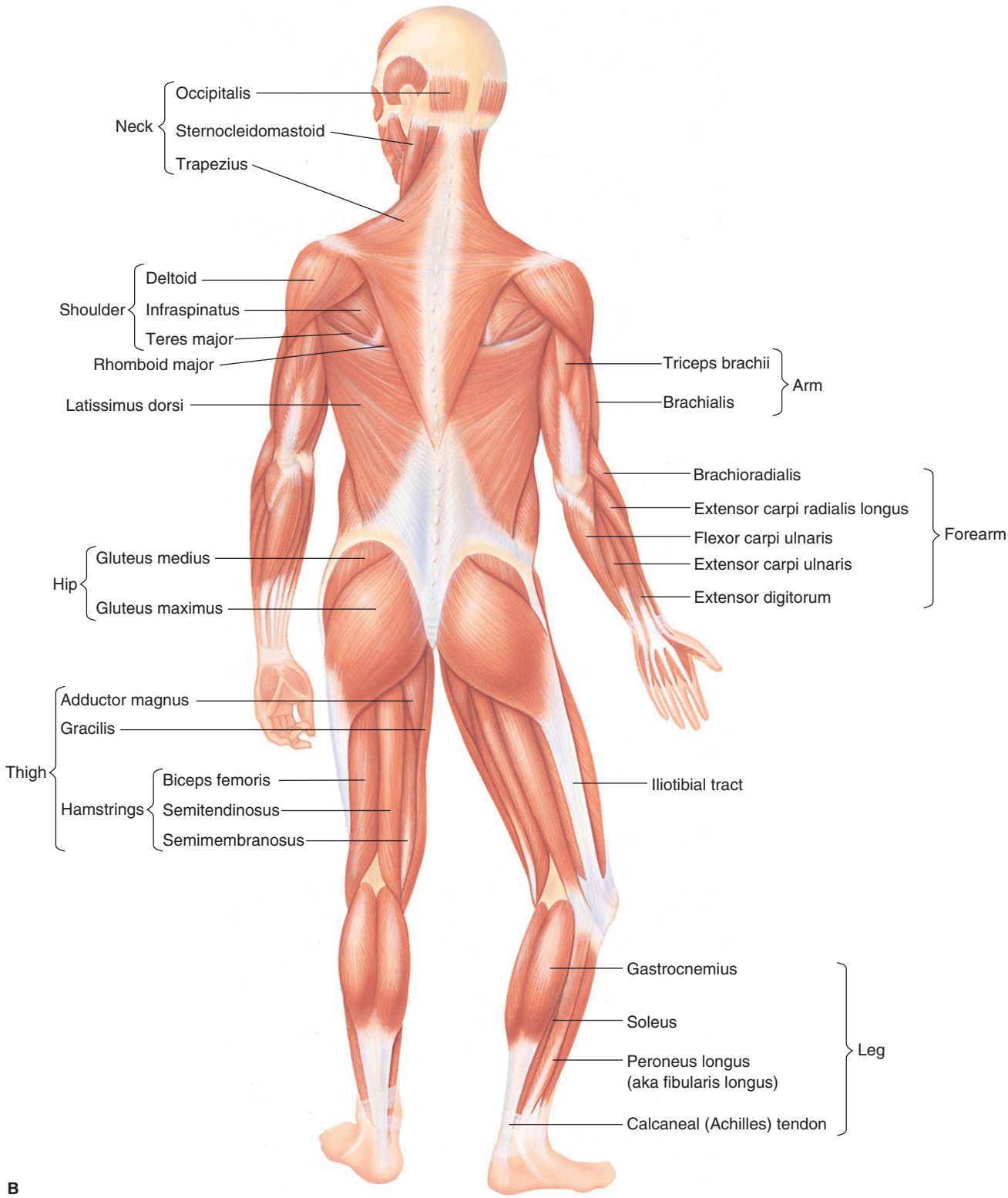


FIGURE 37.5 ■ *B*, Muscles of the posterior body

its striated (striped) appearance under an electron microscope. Myofibrils are strands of smaller repeating units called sarcomeres, which consist of thick filaments of myosin and thin filaments of actin, proteins that contribute to muscle contraction.

Skeletal muscle cells have typical functional properties:

- *Excitability:* the ability to receive and respond to a stimulus. The stimulus is usually a neurotransmitter released by a neuron and the response is the generation and transmission of an action potential along the plasma membrane of the muscle cell.
- *Contractibility:* the ability to respond to a stimulus by forcibly shortening.
- *Extensibility:* the ability to respond to a stimulus by extending and relaxing; muscle fibres shorten when they contract and extend when they relax.
- *Elasticity:* the ability to resume its resting length after it has shortened or lengthened.

Skeletal muscle movement is triggered when motor neurons release acetylcholine, a neurotransmitter that crosses the neuromuscular junction and alters the permeability of the muscle fibre. Sodium ions enter the fibre, producing an action potential that causes muscle contraction. The more fibres that contract, the stronger the contraction of the entire muscle.

Prolonged strenuous activity causes continuous nerve impulses and eventually results in a build-up of lactic acid and reduced energy in the muscle, or muscle fatigue. However, continuous nerve impulses are also responsible for maintaining muscle tone. Lack of use results in muscle atrophy, whereas regular exercise increases the size and strength of muscles.

Joints, ligaments and tendons

Joints, or articulations, are regions where two or more bones meet. Joints hold the bones of the skeleton together while allowing the body to move. Joints may be classified by function as synarthroses, amphiarthroses or diarthroses. Table 37.2 describes each of these types. Joints are also classified by structure as fibrous, cartilaginous or synovial.

Fibrous joints

Fibrous joints permit little or no movement because the articulating bones are joined either by short connective tissue fibres that bind the bones together, as with the sutures of the skull, or by short cords of fibrous tissue called ligaments, which permit slight give but no true movement.

Cartilaginous joints

Some cartilaginous joints, such as the sternocostal joints of the rib cage, are composed of hyaline cartilage growths that fuse together the articulating bone ends. These joints are immobile. In other cartilaginous joints, such as the intervertebral discs, the hyaline cartilage fuses to an intervening plate of flexible fibrocartilage. This structural feature accounts for the flexibility of the vertebral column.

Synovial joints

Bones in synovial joints are enclosed by a cavity that is filled with synovial fluid, a filtrate of blood plasma (see Figure 37.6). Synovial joints are freely movable, allowing many kinds of movements, as listed and described in Table 37.3. Synovial joints are found at all articulations of the limbs.

They have several characteristics:

- The articular surfaces are covered with articular cartilage.
- The joint cavity is enclosed by a tough, fibrous, double-layered articular capsule; internally, the cavity is lined with a synovial membrane that covers all surfaces not covered by the articular cartilage.
- Synovial fluid fills the free spaces of the joint capsule, enhancing the smooth movement of the articulating bones.

Bursae are small sacs of synovial fluid that cushion and protect bony areas that are at high risk of friction, such as the knee and the shoulder. Tendon sheaths are a form of bursae, but they are wrapped around tendons in high-friction areas.

The fibrous capsules that surround synovial joints are supported by ligaments, dense bands of connective tissue that connect bones

TABLE 37.2 Functional classification of joints

TYPE	DESCRIPTION	EXAMPLES
Synarthrosis	Immovable joint	Skull sutures Epiphyseal plates Joint between first rib and manubrium of sternum
Amphiarthrosis	Slightly movable joint	Vertebral joints Joint of the pubic symphysis
Diarthrosis	Freely movable joint	Joints of the limbs Shoulder joints Hip joints

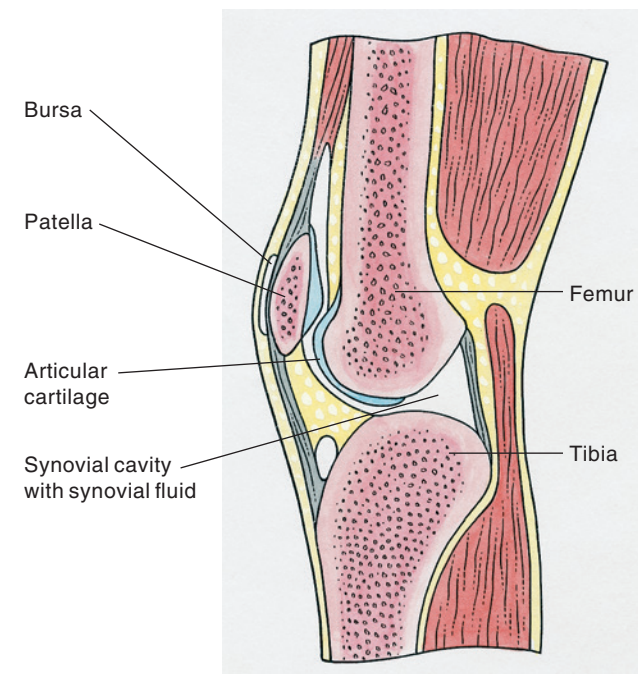


FIGURE 37.6 ■ Structure of a synovial joint (knee)

Source: DK Images.

TABLE 37.3 Movements allowed by synovial joints

TYPE	DESCRIPTION
Abduction	Move limb away from body midline
Adduction	Move limb towards body midline
Extension	Straighten limbs at joint
Flexion	Bend limbs at joint
Dorsiflexion	Bend ankle to bring top of foot towards shin
Plantar flexion	Straighten ankle to point toes down
Pronation	Turn forearm to place palm down
Supination	Turn forearm to place palm up
Eversion	Turn out
Inversion	Turn in
Circumduction	Move in circle
Internal rotation	Move inwards on a central axis
External rotation	Move outwards on a central axis
Protraction	Move forwards and parallel to ground
Retraction	Move backwards and parallel to ground

to bones. Ligaments limit or enhance movement, provide joint stability and enhance joint strength. Tendons are fibrous connective tissue bands that connect muscles to the periosteum of bones and enable the bones to move when skeletal muscles contract. When muscles contract, increased pressure causes the tendon to pull, push or rotate the bone to which it is connected.

ASSESSING MUSCULOSKELETAL FUNCTION

Structures and functions of the musculoskeletal system are assessed by a health assessment interview to collect subjective data, a physical assessment to collect objective data and findings from diagnostic tests. Sample documentation of an assessment of the musculoskeletal system is given in the accompanying box.

Health assessment interview

A health assessment interview to determine problems with musculoskeletal structure and/or function may be conducted during a health screening, and may focus on a chief complaint (such as joint pain) or may be part of a total health assessment. Health problems affecting the neurological system may manifest as problems with musculoskeletal function, and an assessment of both systems may be necessary. (See Chapter 40 for assessment of the neurological system.) If the person has problems with musculoskeletal structure or function, analyse its onset, characteristics, course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, ask the person:

- Describe the pain you have had in your elbow. Does the pain increase with movement? Have you noticed any redness or swelling?
- Did you injure your ankle before you began to experience difficulty walking?
- Is your pain worse in the morning or does it get worse throughout the day?

The primary manifestations of altered function of the musculoskeletal system are pain and limited mobility. Specific descriptors of the pain, its location and its nature are important. Other significant information includes associated manifestations, such as fever, fatigue, changes in weight, rash and/or swelling. Also collect information about the person's lifestyle: type of employment, ability to carry out activities of daily living (ADLs) and provide self-care, exercise or participation in sports, use of alcohol or drugs, and nutrition. Explore past injuries and measures to self-treat pain (such as over-the-counter (OTC) medications, prescribed medications, application of heat or cold, splinting, wrapping or rest).

Interview questions categorised by functional health patterns are listed in the Functional health pattern interview box below.

SAMPLE DOCUMENTATION

Assessment of the musculoskeletal system

7/4/2015
NURS
0900

58-year-old Caucasian male, employed as a roofer, comes to the orthopaedic clinic for evaluation of chronic knee pain. The person states, 'The pain in my knees is worse when I get up in the morning and when I carry something heavy at work.' Posture erect, gait even without obvious limp. Bones of lower extremities appear equal in size and shape bilaterally. No swelling noted, bulge test negative for fluid around knee. Crepitus heard in both knees during flexion and extension. Range of motion (ROM) in both knees slightly decreased. No obvious decrease in muscle mass. The person states that his knee pain during ROM is a 3 on a 1 to 10 scale. Referred to clinic physician for further evaluation, including x-rays of both knees.

———— R. Coleman

R. COLEMAN RN

Physical assessment

Physical assessment of the musculoskeletal system may be performed either as part of a total assessment or alone for a person with known or suspected problems. The techniques used to assess the musculoskeletal system are inspection, palpation and measurement of muscle mass and range of motion (ROM). The person should be comfortably dressed in clothing that lets you see the movement of all joints clearly. The person may be standing, sitting or lying down; the sequence of the examination should be such that the person does not have frequent position changes. An assessment of the older adult, the person in pain or the person who is weak may take extra time. Normal age-related findings for the older adult are summarised in Table 37.4.

FUNCTIONAL HEALTH PATTERN INTERVIEW Musculoskeletal system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Have you ever had any muscle or bone diseases or injuries? If so, describe them. ■ Describe any surgery, physical therapy, heat or other treatments you have received for problems with your muscles or bones. ■ List any medications, such as muscle relaxants or prescribed or over-the-counter (OTC) medications and ointments you use for musculoskeletal problems. ■ Do you take any herbal or nutritional supplements for musculoskeletal problems? If so, what and how often?
Nutritional–Metabolic	■ Describe your dietary intake in a typical 24-hour period. Does your diet include milk, cheese, cottage cheese and vegetables? If so, how often? ■ Do you take vitamins and/or additional calcium supplements? If so, what type and how often? ■ Have you had a recent weight gain or loss? What do you see as your ideal weight? ■ Have you had any redness or swelling in your joints?
Elimination	■ Does your musculoskeletal problem make it difficult for you to get to the bathroom?
Activity–Exercise	■ Describe your usual activities for a 24-hour period. ■ Describe any musculoskeletal problems (such as weakness, stiffness, pain) that limit your activities of daily living (ADLs), such as driving, gardening, dressing, bathing, walking, climbing stairs, cooking or cleaning. ■ Has there been a change in your usual ability to move around? Describe. ■ Do you regularly exercise or take part in strenuous activities such as heavy lifting? Describe. If you have to lift heavy objects at work, do you use any type of special equipment? Describe. ■ Do you use any assistive devices (such as a cane or walker) to help move around?
Sleep–Rest	■ Does having this problem with your musculoskeletal system interfere with your ability to rest and sleep? If so, how, and what do you do?
Cognitive–Perceptual	■ Describe any muscle, bone or joint pain that you have. What relieves it or makes it worse? ■ Describe any changes in the colour, temperature or sensations in your extremities. ■ Describe any muscle weakness you are experiencing. ■ Do you have stiffness in your joints when you wake up? Does it get better with movement? ■ Do you ever have muscle cramps?
Self-perception–Self-concept	■ How does having this condition make you feel about yourself?
Role–Relationships	■ How has having this condition affected your relationships with others? ■ Has having this condition interfered with your ability to work? Explain. ■ Has anyone in your family had problems with bone, joint or muscle disease? Explain.
Sexuality–Reproductive	■ Has this condition interfered with your usual sexual activity?
Coping–Stress–Tolerance	■ Has having this condition created stress for you? ■ Have you experienced any kind of stress that makes the condition worse? Explain. ■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments that you would not use to treat this problem?

TABLE 37.4 Age-related changes in the musculoskeletal system

AGE-RELATED CHANGE	SIGNIFICANCE
Bones and joints: • ↓ bone mass and minerals. • ↓ calcium reabsorption, a slow resorption of the interior of long bones and slower production of new bone on the outside surface of bones. • Vertebrae shorten and intervertebral discs thin, and kyphosis often occurs. • Cartilage on bone surfaces in joints deteriorates and bone spurs may occur.	Decreased bone mass as well as decreased calcium absorption contributes to bones that are often thinner and weaker, with an increased risk of fractures with trauma. As the spinal column shortens, height decreases. Loss of joint cartilage and formation of bone spurs makes movement more painful and may even limit mobility. Especially common in older adults.
Muscles: • Muscle fibres atrophy and fibrous tissue slowly replaces muscle tissue. • ↓ muscle mass and strength. • ↓ muscle movements, especially in the arms and legs. • Range of motion decreases. • Tendons shrink and harden. • Muscle cramping is common.	Regular exercise is very important in decreasing the loss associated with ageing in terms of maintaining muscle mass, strength and agility.

Prior to the examination, collect all equipment and explain the techniques to decrease the person's anxiety. The sequence for a musculoskeletal examination follows:

1. Begin the examination with an assessment of gait and posture. Observe how the person walks, sits and/or moves about in bed.
2. Inspect and palpate the bones for any obvious deformity or changes in size or shape. Palpation also will elicit tenderness or pain.
3. Measure the extremities for length and circumference. Before taking measurements, make sure the person is lying in a comfortable position. Remember to compare limbs bilaterally.
4. Assess muscle mass by first inspecting for obvious increase or decrease in size. Assess and document muscle strength on a scale of 0 to 5 (see Table 37.5). Box 37.1 provides instructions for testing the strength of various muscles.
5. Assess joints for swelling, pain, redness, warmth, crepitus and ROM. Only assess the ROM of every joint if the person has a specific musculoskeletal problem; however, assessing one or more joints is a common part of nursing care. Use a goniometer for precise measurements of joint ROM (see Figure 37.7). This device has a pointer joined to a protractor at 0 degrees. The two arms are placed along articulating bones and the angle of joint movement is recorded in degrees.

BOX 37.1 Guidelines for assessing muscle strength

In adults, muscles are usually strong and equally strong bilaterally. However, neuromuscular diseases, disuse, metabolic disorders or infections can cause muscle weakness. Muscle strength is expected to be greater in the dominant arm and leg. In most instances (and especially when moving digits and extremities), the nurse provides resistance by pushing in the opposite direction.

The muscles listed below are routinely tested. Instructions for the person are also provided.

MUSCLE	INSTRUCTIONS FOR THE PERSON
Ocular muscles and lids	Close eyes tightly.
Finger muscles	Shake hands.
	Make a fist.
	Spread fingers.
Facial muscles	Blow out cheeks.
	Stick out tongue.
Hip muscles	Raise straight leg while supine.
Neck muscles	Bend head forwards and backwards.
Gluteal and leg muscles	Alternately cross legs while sitting.
Deltoid muscles	Hold arms up.
Biceps muscle	Bend the arm.
Quadriceps muscle	Straighten leg.
Triceps muscle	Straighten the arm.
Wrist muscles	Bend hand forwards and backwards.
Ankle and foot muscles	Bend foot up and down.

TABLE 37.5 Muscle grading scale

GRADE	ASSESSMENT DESCRIPTION
0	(No visible) contraction; paralysis
1	Can feel contraction of muscle but there is no movement of limb
2	Passive ROM
3	Full ROM against gravity
4	Full ROM against some resistance
5	Full ROM against full resistance

Diagnostic tests

The results of diagnostic tests of musculoskeletal structure and function are used to support the diagnosis of a specific injury or disease, to provide information to identify or modify the appropriate medications or therapy used to treat the disease, and to help nurses monitor the person's responses to treatment and nursing care interventions. Diagnostic tests to assess the structures and functions of the musculoskeletal system are described in the table below and summarised in the following

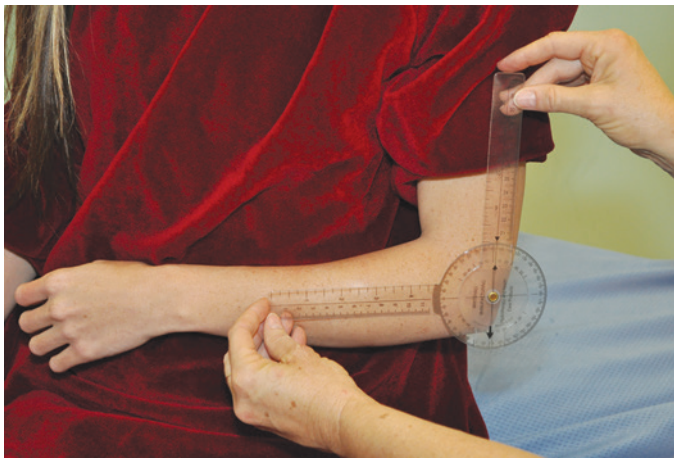


FIGURE 37.7 ■ Using a goniometer to measure joint ROM

bulleted list. More information is included in the discussion of specific injuries or diseases in Chapters 38 and 39.

- Blood tests are used to monitor levels of alkaline phosphatase, calcium, uric acid and creatine kinase, which are commonly increased in bone and joint diseases and muscle trauma (see Table 37.6).
- Radiological examinations, including x-rays, CT scans, MRIs and bone scans, are done to identify and evaluate bone density and structure in conditions such as arthritis, intervertebral disc disease, musculoskeletal trauma, muscle tears, osteomyelitis and bone tumours.
- Bone density examinations (dual energy x-ray absorptiometry (DEXA), quantitative ultrasound (QUS) and bone mineral density (BMD)) are done to evaluate bone mineral density and evaluate the degree of osteoporosis.
- An arthroscopy uses a fibre-optic endoscope to examine the joint interior, to diagnose diseases and to perform surgery. An arthrocentesis is done to withdraw fluid from a joint by needle aspiration.
- Both an electromyogram (EMG) and a somatosensory evoked potential (SSEP) are tests of the electrical activity of skeletal muscle.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, for assessing for medication use that may affect the outcome of the tests, for supporting the person during the examination as necessary, for documenting the procedures as appropriate, and for monitoring the results of the tests.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of the adult. During the health assessment interview, ask about family members with health problems affecting musculoskeletal structure or function. In addition, ask about a family history of arthritis, abnormally long bones, muscular dystrophy and motor neurone disease. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the box below). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

TABLE 37.6 Blood tests with purposes specific to the musculoskeletal system

NAME OF TEST	PURPOSE	NORMAL VALUE
Alkaline phosphatase (ALP)	To identify bone diseases. Increased in bone cancer, Paget's disease, healing fractures, rheumatoid arthritis and osteoporosis	Neonate: 50–300 U/L Growing child: 70–350 U/L Adult, non-pregnant: 25–100 U/L Higher levels are seen in the third trimester of pregnancy and in individuals over 50 years of age
Calcium (Ca)	To monitor calcium levels and detect calcium imbalances. Decreased with lack of calcium and vitamin D intake and malabsorption from the gastrointestinal tract. Increased in bone cancer and multiple fractures	4.5–5.5 mEq/L or 9–11 mg/dL (serum) Total calcium: 2.10–2.60 mmol/L Corrected calcium: 2.15–2.60 mmol/L Ionised calcium: 1.16–1.30 mmol/L
Phosphorus (P), phosphate (PO_4)	To assess phosphorus levels. Increased with bone tumours and healing fractures	1.7–2.6 mEq/L or 2.5–4.5 mg/dL Adult: 0.8–1.5 mmol/L Levels are slightly higher in children
Rheumatoid factor (RF)	To diagnose rheumatoid arthritis (RA) (positive for RA at > 1:80 titre). Also increased in lupus erythematosus and scleroderma	< 1:20 titre < 30 IU/L
Uric acid	To diagnose and monitor the treatment of gout. Panic level considered > 12 mg/dL	Male: 3.5–8.0 mg/dL Female: 2.8–6.8 mg/dL
Human leucocyte antigen (HLA)	To diagnose diseases such as juvenile rheumatoid arthritis or ankylosing spondylitis	Match or no match; no normal values
Creatine kinase (CK)	To diagnose muscle trauma or disease. Increased in muscular dystrophy and traumatic injuries (specifically, CPK-MM isoenzyme)	Neonate: 70–380 U/L Adult female: 30–180 U/L Adult male: 60–220 U/L

DIAGNOSTIC TESTS The musculoskeletal system

NAME OF TEST Blood chemistry

PURPOSE AND DESCRIPTION See Table 37.6

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST X-ray

PURPOSE AND DESCRIPTION X-rays are done to identify and evaluate bone density and structure. Injection of contrast medium with an accompanying x-ray may be done to visualise joint structures, intervertebral discs and wounds deep in muscle.

RELATED NURSING CARE No special preparation needed for standard x-rays. If contrast medium is used, assess for allergy to shellfish, iodine or contrast medium used in previous tests. If allergy is present, test will not be performed.

NAME OF TEST Computed tomography (CT) scan

PURPOSE AND DESCRIPTION Provides a three-dimensional picture used to evaluate musculoskeletal trauma and bony abnormalities.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Magnetic resonance imaging (MRI)

PURPOSE AND DESCRIPTION Used in diagnosis and evaluation of avascular necrosis, osteomyelitis, tumours, disc abnormalities and tears in ligament or cartilage. Uses radiowaves and magnetic fields; gadolinium may be injected to increase visualisation of bony or muscular structures.

RELATED NURSING CARE Assess for metallic implants or metal on clothing. (Metallic implants, such as clips on aneurysms, pacemakers or shrapnel, will prohibit having an MRI.)

NAME OF TEST Bone scan

PURPOSE AND DESCRIPTION Degree of uptake of a radioisotope (based on blood supply to bone) is measured with a Geiger counter and recorded on paper. Uptake is increased in osteomyelitis, osteoporosis, cancers of the bone and in some fractures. Uptake is decreased in avascular necrosis.

RELATED NURSING CARE No special preparation is needed; tell the person to increase oral fluids after the test to aid in excretion of the radioisotope.

NAME OF TEST Bone density (BD)

- Dual energy x-ray absorptiometry (DEXA)
- Quantitative ultrasound (QUS)
- Bone mineral density (BMD)
- Bone absorptiometry

PURPOSE AND DESCRIPTION Bone density examinations are done to evaluate bone mineral density and to evaluate degree of osteoporosis. DEXA can calculate the size and thickness of bone and detect even a 1% loss of bone mass. Osteoporosis is diagnosed if the peak bone mass level is below > 2.5 standard deviations.

Normal value: 1 standard deviation below peak bone mass.

RELATED NURSING CARE No special preparation is needed. Assess for previous fractures, which may increase bone density.

NAME OF TEST Arthroscopy

PURPOSE AND DESCRIPTION An endoscopic examination of the interior surfaces of a joint, used to perform surgery and diagnose diseases of the patella, meniscus and synovial and extrasynovial membranes. In addition, fluid may be drained from the joint and tissue removed for biopsy. A fibre-optic endoscope is inserted into the joint, with either local or general anaesthesia.

RELATED NURSING CARE If general anaesthesia is used, the person is nil by mouth at least 6–8 hours prior to surgery. Following the procedure, assess for bleeding and swelling, apply ice to the area if prescribed and teach the person to avoid excessive use of the joint for 2 to 3 days.

NAME OF TEST Arthrocentesis

PURPOSE AND DESCRIPTION Done to obtain synovial fluid from a joint for diagnosis (such as infections) or to remove excess fluid. A needle is inserted through the joint capsule and fluid is aspirated.

RELATED NURSING CARE No special preparation is needed. Apply compression dressing and assess for bleeding and leakage of fluid following the procedure.

DIAGNOSTIC TESTS The musculoskeletal system (continued)

NAME OF TEST Electromyogram (EMG)

PURPOSE AND DESCRIPTION Measures the electrical activity of skeletal muscles at rest and during contraction; useful in diagnosing neuromuscular diseases. Needle electrodes are inserted into skeletal muscle (as on the legs) and electrical activity can be heard, viewed on an oscilloscope and recorded on graph paper. Normally, there is no electrical activity at rest.

RELATED NURSING CARE Tell the person not to drink fluids containing caffeine or to smoke for 3 hours before the test and not to take medications such as muscle relaxants, anticholinergics or cholinergics.

NAME OF TEST Somatosensory evoked potential (SSEP)

PURPOSE AND DESCRIPTION Measures nerve conduction along pathways to evaluate evoked potential of muscle contractions. Used to identify dysfunction of lower motor neurons as well as muscle disease. Transcutaneous or percutaneous electrodes are applied to the skin and provide recordings.

RELATED NURSING CARE No special preparation is needed.

GENETIC CONSIDERATIONS Musculoskeletal disorders

- Myotonic dystrophy is an inherited disorder in which the muscles become weak, have a decreased ability to relax and eventually waste away. Other effects are mental deficiency, hair loss and cataracts. Although rare, the disease does increase in severity with each successive generation.
- Marfan syndrome, an autosomal dominant disorder of connective tissue, affects the bones, lungs, eyes, heart and blood vessels. It is characterised by abnormally long extremities and is believed to have affected Abraham Lincoln. The aspect of the disease that is most life threatening is the effect on the cardiovascular system. With advances in the diagnosis, evaluation and management of the organ abnormalities associated with Marfan syndrome, the life expectancy for a person with the disease has nearly doubled in the past 25 years. Today, individuals with Marfan syndrome can expect to live about 70 years (National Marfan Foundation, 2013).
- Ellis–van Creveld syndrome is a rare genetic disorder characterised by a variety of physical alterations, including short-limb dwarfism, additional fingers or toes, malformed wrists, cardiac abnormalities and partial tooth eruption.
- Duchenne muscular dystrophy, an X-linked disorder, primarily affects males. It is one of the most common muscular dystrophies and is characterised by rapid muscle degeneration early in life.
- Motor neurone disease is a neurological disease that affects the motor neurons in the spinal cord and brain, eventually resulting in paralysis and death.
- Other musculoskeletal diseases believed to have a genetic component include rheumatoid arthritis, osteoarthritis, gout, muscular dystrophy, ankylosing spondylitis, lupus erythematosus and scleroderma.

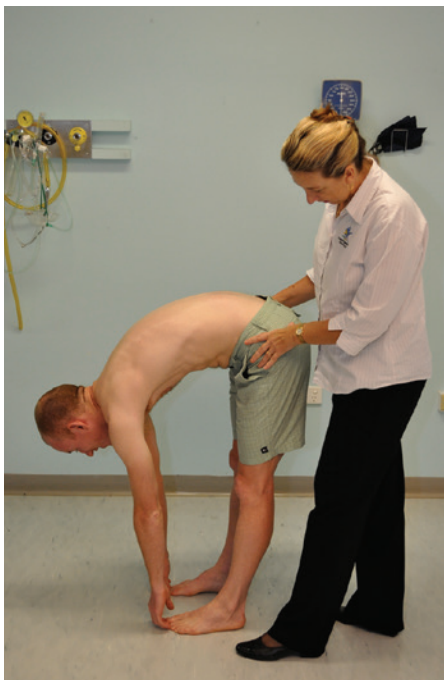
MUSCULOSKELETAL ASSESSMENTS

Technique/normal findings	Abnormal findings
Gait and body posture	
Inspect body posture and gait. *Body posture should be upright; gait should be smooth and steady.*	■ Joint stiffness, pain, deformities and muscle weakness can cause changes in gait and posture.
Inspect the spine for curvature. Ask the person to stand and bend back slowly as far as possible, bend slowly to the right and then to the left as far as possible, turn slowly to the right and left in a circular motion, and bend forwards slowly and try to touch fingers to toes. *When viewed from the back, the cervical and lumbar spine are concave, the thoracic spine is convex and the spine is straight.*	■ With herniated lumbar discs, the lumbar curve flattens and spinal mobility is decreased. ■ An increased lumbar curve, called **lordosis**, may be seen in obesity or pregnancy. ■ A lateral, S-shaped curvature of the spine is called **scoliosis**. Functional scoliosis usually is a compensatory response to painful paravertebral muscles, herniated discs or discrepancy in leg length. It disappears with forward flexion. Structural scoliosis is often congenital and tends to appear during adolescence. It is accentuated with forward bending. ■ **Kyphosis** is an exaggerated thoracic curvature of the spine common in older adults.

Technique/normal findings	Abnormal findings
Joints	
Inspect the joints for deformity, swelling and redness. *There should be no visible deformity, swelling or redness of joints.*	■ Diseases of the joints may be manifested by such deformities as tissue loss, tissue overgrowth or contractures or irreversible shortenings of muscles and tendons. ■ Disease in a joint may cause obvious bulging. ■ Redness, swelling and pain are evidence of an inflammation or infection in the joint. ■ Inflammation and injury cause joint pain. ■ Arthritis, bursitis, tendonitis and osteomyelitis (infection of a bone) result in painful, hot joints. ■ **Crepitation** (a grating sound) is present in a joint when the articulating surfaces have lost their cartilage, such as in arthritis.
Palpate the joints for tenderness, warmth, crepitation, consistency and muscle mass. *Joints should be non-tender and consistent bilaterally and without visible or palpable excess warmth, crepitation or masses.*	
Range-of-motion	
Assess joint ROM by asking the person to perform activities specific to each joint, as described below. *All bilateral joints should move through full range of motion.*	
Temporomandibular joint: 'Open your mouth wide and then close your mouth.' (As the person opens and closes the mouth, palpate the temporomandibular joints with your index and middle fingers, as shown in Figure 37.8.)	■ Clicking or popping noises, decreased ROM, pain and swelling may indicate temporomandibular joint syndrome or, in rare cases, osteoarthritis.
Cervical spine: 45-degree flexion: 'Touch your chin to your chest.' 55-degree extension: 'Look at the ceiling.' 40-degree lateral bending: 'Try to touch your right ear to your right shoulder.' Repeat with the left side. 70-degree rotation: 'Try to touch your chin to each shoulder.'	■ Neck pain and limited extension with lateral bending are seen with herniated cervical discs and in cervical spondylosis. ■ An immobile neck with head and neck thrust forwards is seen with ankylosing spondylitis.
Lumbar spine: 75- to 90-degree flexion: 'Touch your toes with your fingers' (see Figure 37.9A). 30-degree extension: 'Bend backwards slowly.' 35-degree lateral bending: 'Bend right and left' (see Figure 37.9B). 30-degree rotation: 'Twist your shoulders right and left' (see Figure 37.9C).	■ Decreased movement or pain with movement may indicate an abnormal spinal curvature, arthritis, herniated disc or spasm of paravertebral muscles.

FIGURE 37.8 ■ Palpating the temporomandibular joints

Technique/normal findings	Abnormal findings



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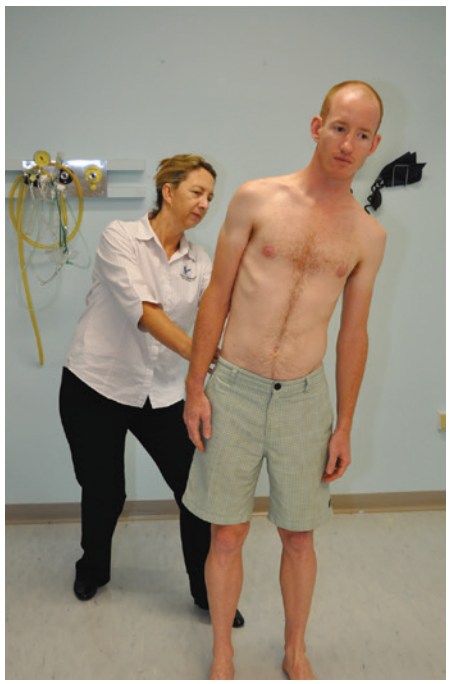

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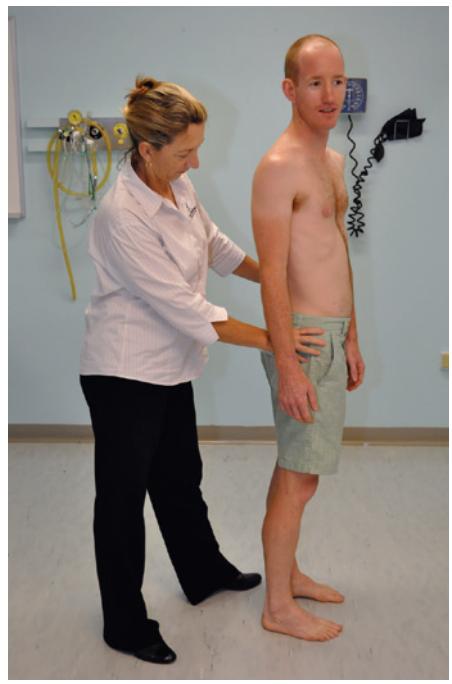

C

FIGURE 37.9 ■ *A*, Forward flexion of spine. *B*, Lateral flexion of spine. *C*, Rotation of spine

Fingers: Flexion: 'Make a fist.' Extension: 'Open your hand.' Abduction: 'Spread your fingers.' Adduction: 'Close your fingers.'	■ Flexion and extension of fingers are decreased in arthritis. ■ Heberden's nodes and Bouchard's nodes are hard, non-tender nodules on the dorsolateral parts of the distal and proximal interphalangeal joints, respectively. They are common in osteoarthritis. ■ Stiff, painful, swollen finger joints are seen in acute rheumatoid arthritis. ■ Boutonnière and swan-neck deformities are seen in chronic rheumatoid arthritis. ■ Swollen finger joints with a white chalky discharge may be seen in chronic gout.
Wrists: 90-degree flexion: 'Bend wrist down.' 70-degree extension: 'Bend wrist up.' 55-degree ulnar deviation: 'Bend wrist towards little finger.' 20-degree radial deviation: 'Bend wrist towards thumb.'	■ Bilateral chronic swelling in the wrist is seen in arthritis.
Elbows: 160-degree flexion: 'Touch your hands to your shoulders.' 180-degree extension: 'Straighten your elbows.' 90-degree supination: 'Bend your elbows 90 degrees and turn hands palm up.' 90-degree pronation: 'Bend your elbows 90 degrees and turn fists down.'	■ Swollen, tender, inflamed elbows are apparent in gouty arthritis and rheumatoid arthritis. ■ Pain and tenderness at the lateral epicondyle occur in tennis elbow.

Technique/normal findings	Abnormal findings
Shoulders: 180-degree flexion: 'Hold your arms straight up and out.' 50-degree hyperextension: 'Put your straight arm behind your back.' 90-degree internal rotation: 'Put your forearm behind your lower back.' 180-degree abduction: 'Raise your straight arm up and out to your side.' 50-degree adduction: 'Put your straight arm across your chest.'	■ Pain and tenderness over the biceps tendon occurs with **tendonitis** (inflammation of a tendon). ■ The arm cannot be abducted fully when the supraspinatus tendon of the shoulder is ruptured. ■ Pain and limited abduction is also seen with **bursitis** (inflammation of a bursa) and calcium deposits in this area.
Toes: 90-degree flexion: 'Walk on your toes.'	■ The great toe is excessively abducted in hallux valgus. ■ The joint above the great toe is swollen, inflamed and painful in gouty arthritis. ■ There is hyperextension of the metatarsophalangeal joint and flexion of the proximal interphalangeal joint with hammer toes.
Ankles: 20-degree dorsiflexion: 'Point your foot to the ceiling.' 45-degree plantar flexion: 'Point your foot to the floor.' 30-degree inversion: 'Walk on the outside of your feet.' 20-degree eversion: 'Walk on the inside of your feet.'	■ Contractures of the Achilles tendon may occur in people with rheumatoid arthritis or following prolonged bed rest.
Knees: 130-degree flexion: 'Do a deep knee bend.' 180-degree extension: 'Sit down and hold your legs straight out in front of you.'	■ Swelling over the suprapatellar pouch is seen with inflammation and fluid in the articular capsule of the knee. **Synovitis** is inflammation of the synovial membrane lining the articular capsule of a joint. It is common with knee trauma. ■ Swelling over the patella is seen in bursitis.
Hips: (The person is lying down.) 120-degree flexion: 'Bring bent knee up to your chest.' 30-degree hyperextension: 'Lie on your abdomen and lift up one leg at a time.' 45-degree abduction: 'Hold your leg straight and move it out to the side.' 40-degree internal rotation: 'Bend your knee and swing it towards your other leg.' 45-degree external rotation: 'Bend your knee and swing it out to the side.'	■ Movement of the hip is limited and/or painful in arthritis.

Technique/normal findings	Abnormal findings
Special assessments	
Perform Phalen's test. Ask the person to hold the wrist in acute flexion for 60 seconds (see Figure 37.10). *There should be no tingling, numbness or pain.*	■ Numbness and burning in the fingers during Phalen's test may indicate carpal tunnel syndrome.

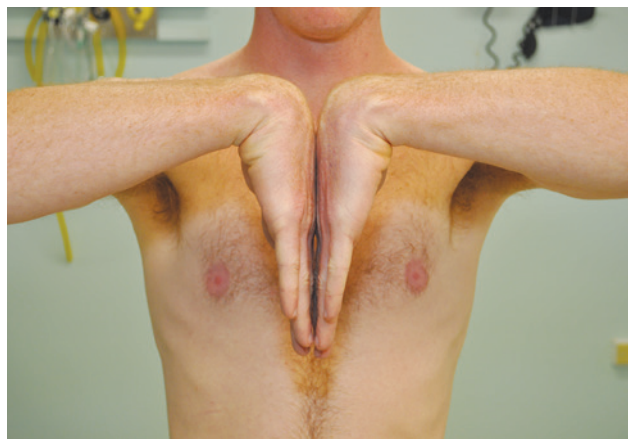

FIGURE 37.10 ■ Phalen's test

Check for small amounts of fluid on the knee by conducting the bulge test. Milk upwards on the medial side of the knee and then tap the lateral side of the patella (see Figure 37.11). *No bulge of fluid should appear on the medial side of the knee.*	■ A fluid bulge indicates increased fluid in the knee joint rather than soft tissue swelling.

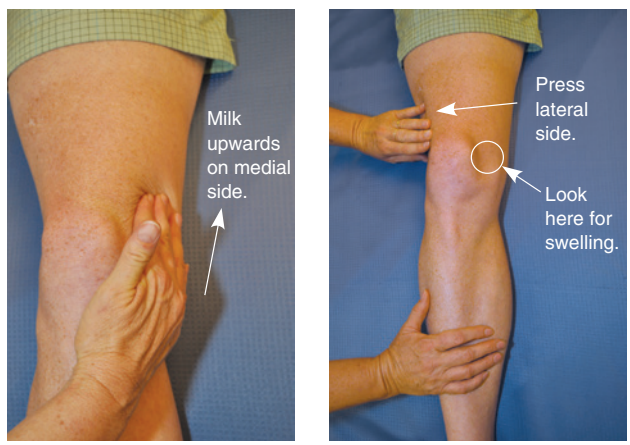

FIGURE 37.11 ■ Checking for the bulge sign

Conduct the ballottement test to detect large amounts of fluid in the knee. Apply downward pressure on the knee with one hand while pushing the patella backwards against the femur with the other hand (see Figure 37.12). *There should be no movement of the patella. The patella should rest firmly over the femur.*	■ Increased fluid will cause a tapping sound as the patella displaces the fluid and hits the femur.

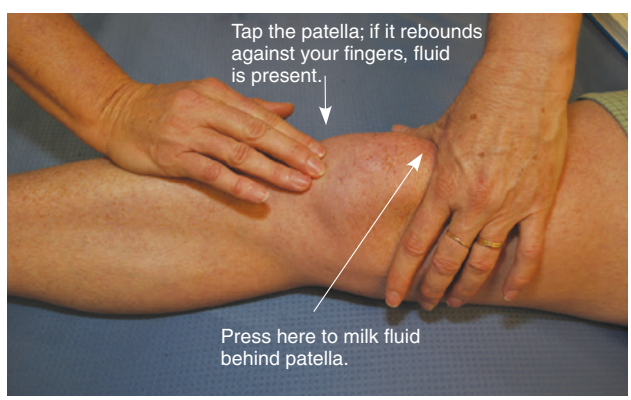

FIGURE 37.12 ■ Checking for ballottement

Technique/normal findings	Abnormal findings
Perform McMurray's test. While reclining, ask the person to turn the flexed knee towards the centre of the body. Stabilise the knee with one hand and apply pressure on the lower leg with the other hand (see Figure 37.13). *There should be no pain or clicking.*	■ Pain, locking (inability to fully extend the knee) or a popping sound may indicate an injury to a meniscus, a disc of cartilaginous tissue in the knee.

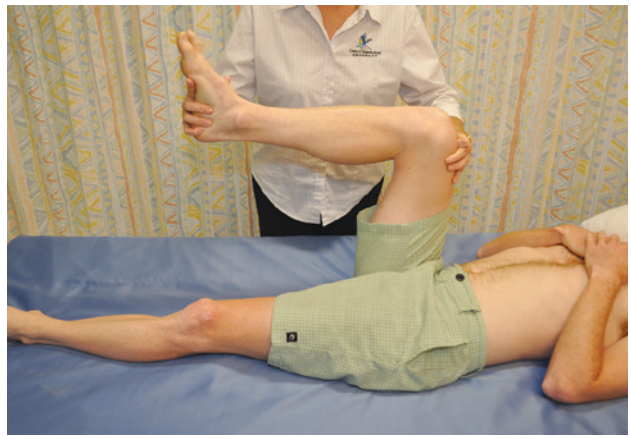

FIGURE 37.13 ■ McMurray's test

Perform the Thomas test. Ask the person to lie down and extend one leg while bringing the knee of the opposite leg to the chest (see Figure 37.14). *The extended leg should not rise off the table.*	■ A hip flexion contracture will cause the extended leg to rise off the table.

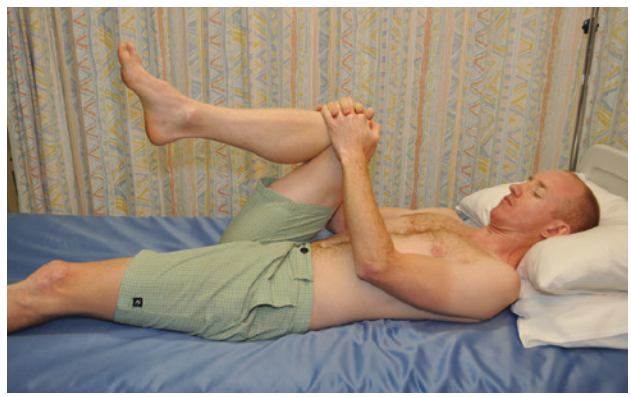

FIGURE 37.14 ■ Thomas test for hip contracture

CONCEPT CHECK

1 A person you are caring for has an epiphyseal fracture. Based on this information, which classification of bone is involved?
1 irregular
2 flat
3 long
4 short

2 When asking a person to move an extremity away from the body midline, you are assessing:
1 abduction
2 adduction
3 extension
4 flexion

3 A person you are caring for asks you, 'Why is blood being examined for uric acid?' What would be your most accurate response?
1 'A uric acid test is done to see if your gout medication is effective.'
2 'A uric acid test is done to diagnose rheumatoid arthritis.'
3 'Do you have a family history of muscle or bone disease?'
4 'Tell me how you got that big bruise on your hip.'

4 At what age would a woman be most likely to have a bone density examination?
1 as a teenager
2 in her twenties
3 in her forties
4 in her sixties

5 With ageing, bone mass and calcium absorption decrease. Which risk is increased as a result?
1 obesity
2 weakness
3 fractures
4 deformity

6 What would you ask the person to do in order to assess facial muscle strength?
1 'Close your eyes tightly.'
2 'Stick out your tongue.'
3 'Bend your head forwards.'
4 'Open your eyes wide.'

7 Which term is used to document a grating sound when a joint is moved?
1 crackles
2 arthritis
3 synovitis
4 crepitation

8 While conducting the ballottement test, you note the patella rebounds against your fingers. What does this finding indicate?
1 deformity of the elbow
2 infection of the metatarsals
3 fluid in the knee joint
4 crepitus in the hip joint

9 During the physical assessment of a young adult, you note a lateral, S-shaped curve of the spine. What is the name of this condition?
1 lordosis
2 scoliosis
3 kyphosis
4 musclosis

10 What are the most common manifestations of musculoskeletal disorders?
1 pain and limited mobility
2 swelling and exaggerated reflexes
3 cyanosis and decreased pulses
4 pallor and decreased ROM

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CHAPTER 38

NURSING CARE OF PEOPLE WITH MUSCULOSKELETAL TRAUMA

ANN-MARIE BROWN

KEY TERMS

LEARNING OUTCOMES

- Compare and contrast the causes, risk factors, pathophysiology, manifestations, interprofessional care and nursing care of contusions, strains, sprains, joint dislocations and fractures.
- Describe the stages of bone healing.
- Explain the pathophysiology, manifestations and related treatment for complications of bone fractures: compartment syndrome, fat embolism syndrome, deep venous thrombosis, infection, delayed union and non-union, and reflex sympathetic dystrophy.
- Discuss the purposes and related nursing interventions for casts, traction and stump care.
- Explain the causes, levels, types and potential complications (infection, delayed healing, chronic stump pain, phantom pain and contractures) of an amputation.
- Describe the pathophysiology, interprofessional care and nursing care for repetitive use injuries: carpal tunnel syndrome, bursitis and epicondylitis.

CLINICAL COMPETENCIES

- Assess functional health status of people with musculoskeletal injuries and monitor, document and report abnormal manifestations.
- Use evidence-based research to plan and implement nursing care for people with skeletal pin sites.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with musculoskeletal injuries.
- Provide skilled cast care, traction care and stump care.
- Integrate interprofessional care into care of people with musculoskeletal trauma.
- Provide teaching appropriate for prevention and self-care of traumatic injuries of the musculoskeletal system.
- Revise the plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to people with traumatic injuries of the musculoskeletal system.

Musculoskeletal trauma is an injury to muscle, bone or soft tissue that results from excessive external force. The external force transmits more kinetic energy than the tissue can absorb and injury results. The severity of the trauma depends not only on the amount of force but also on the location of the impact, because different parts of the body can withstand different amounts of force. A wide variety of external forces can cause trauma and the force involved can vary in severity (e.g. a step off the curb, a fall, being tackled in a football game and a motor vehicle crash). See Chapter 10 for a detailed discussion of the results of different forces and types of injury from trauma.

Traumatic musculoskeletal injuries include blunt tissue trauma, alterations in tendons and ligaments, and fractures of bones. Various forces that cause musculoskeletal trauma are typical for a specific environment, activity or age group. For example, motorcycle accidents resulting in fractures of the distal tibia, midshaft femur and radius are common in young men. Sports injuries, resulting from either overuse or acute trauma, are seen more often in adolescents and young adults. Falls are the most common cause of injury in people age 65 or older, with fractures of the vertebrae, proximal humerus and hip seen most often (Porth & Matfin, 2014). Regardless of the cause, the injury may require rehabilitation and temporary or permanent changes in lifestyle.

Musculoskeletal trauma can result in mild or severe injuries. A person may experience a soft tissue injury, a fracture and/or a complete amputation. In addition, trauma to one part of the musculoskeletal system often produces dysfunction in adjacent structures. For example, a fracture of the femur prevents the adjacent muscles from abducting and adducting. Nursing care helps minimise the effects of trauma, prevents complications and hastens restoration of function. This chapter discusses fractures, amputations, soft tissue injuries, dislocations and repetitive use injuries.

TRAUMATIC INJURIES OF THE MUSCLES, LIGAMENTS AND JOINTS

THE PERSON WITH A CONTUSION, STRAIN OR SPRAIN

Contusions, strains and sprains are among the most commonly reported injuries. They account for about 50% of work-related injuries, with lower back injuries being the most commonly reported occupational injury. However, many sprains and strains are not work related and often are not reported. The lower back and cervical region of the spine are the most common sites for muscle strains; the ankle is the most commonly sprained joint, usually caused by forced inversion of the foot.

Pathophysiology and manifestations

A contusion, the least serious form of musculoskeletal injury, is bleeding into soft tissue that results from a blunt force, such as a kick or striking a body part against a hard object. The skin remains intact, but small blood vessels rupture and bleed into soft tissues. A contusion with a large amount of bleeding is referred to as a *haematoma*. The manifestations of a contusion include swelling and discolouration of the skin. The blood in the soft tissue initially results in a purple-blue colour commonly referred to as a *bruise*. As the blood begins to reabsorb, the area involved becomes brown and then yellow until it disappears.

A **strain** is a stretching injury to a muscle or a muscle–tendon unit caused by mechanical overloading. A muscle that is forced to extend past its elasticity will become strained. Lifting heavy objects without bending the knees or a sudden acceleration–deceleration, as in a motor vehicle crash, can cause strains. The most common sites for a muscle strain are the lower back and cervical regions of the spine. The manifestations of a strain include pain, limited motion, muscle spasms, swelling and possible muscle weakness. Severe strains that partially or completely tear the muscle or tendon are painful and disabling.

A **sprain** is a stretch and/or tear of one or more ligaments surrounding a joint. Forces going in opposite directions cause the ligament to overstretch and/or tear. The ligaments may be partially or completely torn. Although any joint may be involved, sprains of the ankle and knee are most common. Manifestations include loss of the ability to move or use the joint, a feeling of a 'pop' or tear, discolouration, pain and rapid swelling. Motion increases the joint pain. The intensity of the manifestations depends on the severity of the sprain. A comparison of sprains and strains is presented in Box 38.1.

INTERPROFESSIONAL CARE

The goal of the initial stage of treating soft tissue trauma is to reduce swelling and pain. People should follow a regimen of rest, ice, compression and elevation (RICE) for the first 24 to 48 hours (see Table 38.1 for RICE therapy). Severe sprains may require surgical repair. Ankle sprains may be immobilised with an air cast, with no limitations on weight bearing. A knee injury often requires a knee immobiliser, such as a knee brace (e.g. Zimmer brace). If an upper extremity is injured, a sling is provided. Physical therapy may be recommended for rehabilitation. Time required for healing depends on the severity of the injury; for example, a mild ankle sprain may require up to 3 to 6 weeks

BOX 38.1 Comparison of sprains and strains

Sprain
- Defined as an injury to a ligament that results from a twisting motion.
- Can cause joint instability.
- Pain, oedema and swelling are present.
- Motion increases the joint pain.

Strain
- Defined as a microscopic tear in the muscle.
- Sharp or dull pain is present.
- Pain increases with isometric contraction of the muscle.
- Swelling and local tenderness are present.

TABLE 38.1 RICE therapy for musculoskeletal injuries

ACTION	HEALTH EDUCATION FOR THE PERSON AND FAMILY
Rest	• Decrease regular activities of daily living and exercise as needed. • If advised by your healthcare provider, do not put weight on the injured area for 48 hours. • Crutches may help if you can't put weight on an ankle or knee. • If you use a cane or crutch for an ankle injury, use it on the uninjured side so you can lean away from and relieve weight on the injured ankle.
Ice	• Apply an ice pack to the injured area for 20 minutes at a time, four to eight times a day. • An ice bag, cold pack, plastic bag filled with ice or a bag of frozen peas may be used. • Do not apply the ice pack for longer than 20 minutes to avoid cold injury and frostbite.
Compression	• Compression often helps reduce swelling. The kind you use will depend on the recommendation of your healthcare provider. • Examples of compression bandages include special boots, air casts and splints.
Elevation	• Keep the injured extremity elevated on a pillow above heart level, to help reduce swelling and pain.

of rehabilitation, whereas a severe sprain may require up to 8 to 12 months to return to full activities (National Institute of Arthritis and Musculoskeletal and Skin Diseases, 2015).

When soft tissue trauma is suspected, x-rays are taken to rule out soft tissue injury and magnetic resonance imaging (MRI) may be done if further assessment is necessary. Medications used to treat soft tissue trauma include non-steroidal anti-inflammatory drugs (NSAIDs) and analgesics.

Nursing care

The nursing care of each person is individualised. A strain or sprain may not be as devastating to a person with a desk job as it would be to a professional athlete; therefore, the nurse should determine what the injury means to the particular person.

Nursing diagnoses and interventions

Nursing diagnoses focus on providing information about self-care to decrease pain and return physical mobility to pre-injury levels.

Acute pain

The pain that results from soft tissue trauma is due primarily to the injury to the muscle or ligament and secondarily to bleeding and oedema at the injury site.

- Teach the person to use RICE (rest, ice, compression, elevation) therapy to care for the injury. *The interventions included in RICE therapy allow the injured muscle, ligament or tendon to heal (rest), cause vasoconstriction and reduce pain (ice), decrease oedema formation and pain (compression), and promote venous return to decrease oedema and pain (elevation).*

Impaired physical mobility

Pain causes the person to avoid using or bearing weight on the injured extremity. Always observe the person's use of assistive devices; if the device is inappropriate, the person can face a greater risk of falling. Also consider that the device may be appropriate but the person may not be using it correctly or safely. As a person ages, muscle mass in the upper extremities declines. As a result, the older person with a sprained ankle may not be able to use crutches because crutches require that the person distribute body weight along the upper extremities. Older people may therefore find a walker more useful.

- Teach the correct use of crutches, walkers, canes or slings if prescribed. *Use of the correct technique increases safety and encourages use of these devices.*
- Encourage follow-up care. *Severe sprains may require further evaluation to determine if surgical intervention is indicated.*

THE PERSON WITH A JOINT DISLOCATION

A **dislocation** is an injury of a joint in which the ends of bones are forced from their normal position. Dislocations usually follow trauma such as a fall or blow, with the bone ends displaced or separated from their normal position in the joint capsule. They commonly are seen in people who take part in contact sports such as football or from falls during activities such as skiing. Although dislocations may occur in any joint, they occur most frequently in the shoulder and acromioclavicular joints. Dislocations may also result from a disease such as rheumatoid arthritis. A **subluxation** is a partial dislocation in which the bone ends are still partially in contact with each other.

Pathophysiology

Dislocations may be congenital, traumatic or pathological. Congenital dislocations are present at birth and are seen in the hip and knee. Traumatic dislocations result from falls, blows or rotational injuries. Pathological dislocations result from disease of the joint, including infection, rheumatoid arthritis, paralysis and neuromuscular diseases.

Manifestations

The manifestations of a dislocation include pain, deformity and limited motion.

INTERPROFESSIONAL CARE

Care of the person with a dislocation focuses on relieving pain, correcting the dislocation and preventing complications. The dislocation is diagnosed by physical examination and x-rays. The joint is most often reduced (bone ends realigned) by means of manual traction.

Treatment of a shoulder joint dislocation depends on the severity of the dislocation. Reduction of shoulder dislocations may be undertaken in the emergency department or on occasions in the operating theatre. Immobilisation is no longer recommended and only the most severe dislocations are surgically reduced. A dislocated hip requires immediate reduction in the emergency room under sedation to prevent necrosis of the femoral head and injury to the sciatic and femoral nerves. After reduction, the person is placed on bed rest. In some cases, traction is needed for several weeks. If a hip dislocation is accompanied by a fracture, the person will undergo surgery to increase mobility, decrease complications and rapidly stabilise the joint.

Children, generally between the ages of 1 and 4 years, often have common injuries such as a pulled elbow. This is a result of the radius slipping out of its normal position at the elbow joint. It is usually caused by a sudden yank or pull on the child's lower arm or wrist (e.g. when a child is being lifted up). The child presents as distressed and is unable to use the arm, with the elbow in the extension and the forearm in the pronation position. This can be reduced with manipulation of the elbow (Royal Children's Hospital Melbourne, 2015a).

Nursing care

Nursing care of the person with a dislocation or subluxation is individualised to the cause of injury, the type of dislocation and the age of the person. It is important to teach people to seek immediate medical attention, to splint the joint to prevent further damage and to put ice on the injured joint (Mayo Clinic, 2015). Assessment of the affected limb is required to observe for changes in movement, sensation, colour, warmth and increased pain. These changes need to be reported and documented.

Nursing diagnoses and interventions

Nursing diagnoses focus on relieving pain and preventing complications.

Risk of injury

The person with a dislocation requires frequent assessments to ensure that neurovascular compromise does not develop.

- Monitor neurovascular status by assessing the '5 Ps': pain, pulses, pallor, paralysis and paraesthesia. *Neurovascular compromise is indicated by increased pain, decreased or absent pulses, pale skin, inability to move a body part or extremity, and changes in sensation (such as 'pins and needles' sensations or loss of sense of sharp/dull touch).*
- Maintain immobilisation after reduction. *Immobilisation prevents the joint from dislocating again.*

Community-based care

Joint dislocations often tend to be recurring injuries for people actively participating in contact sports and other vigorous physical activities. Prolonged immobilisation (for several weeks after the injury) and aggressive rehabilitation following the initial dislocation can reduce the risk of recurrent dislocation. The following topics should be addressed in preparing the person for community-based care:

- importance of complying with the prescribed length of immobilisation
- skin care and ways to prevent skin-to-skin contact, particularly in the axillary area
- prescribed rehabilitation exercises that will strengthen muscles and other supportive structures in the shoulder, decreasing the risk of future dislocations
- alternatives to activities that precipitate recurrent dislocations
- instructions or referrals to physical therapy if needed for further teaching about using assistive devices
- referrals to physical and occupational therapy and community-based services as needed.

TRAUMATIC INJURIES OF BONES

THE PERSON WITH A FRACTURE

A **fracture** is any break in the continuity of a bone. Fractures vary in severity according to the location and the type of fracture. Although fractures occur in all age groups, they are more common in people who have sustained trauma and in older people.

Pathophysiology

Any of the 206 bones in the body can be fractured. A fracture occurs when the bone is subjected to more kinetic energy than it can absorb. Fractures may result from a direct blow, a crushing force (compression), a sudden twisting motion (torsion), a severe muscle contraction or disease that has weakened the bone (called a *stress* or *pathological fracture*). Two basic mechanisms produce fractures: direct force and indirect force. With direct force, the kinetic energy is applied at or near the site of the fracture. The bone cannot withstand the force. With indirect force, the kinetic energy is transmitted from the point of impact to a site where the bone is weaker. The fracture occurs at the weaker point.

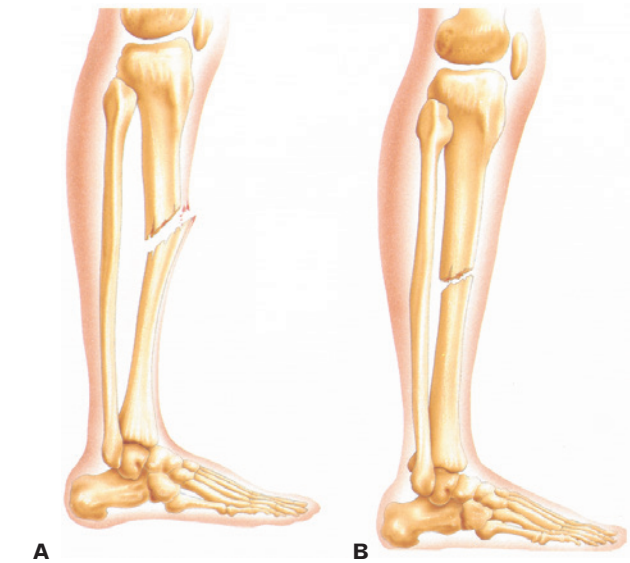


FIGURE 38.1 ■ *A*, An open fracture. *B*, A closed fracture

Fractures in adults are classified in the following ways:

- If the skin is intact, the fracture is considered a *closed* (*simple*) *fracture*. If the skin integrity is interrupted, the fracture is considered an *open* (*compound*) *fracture* (see Figure 38.1). An open fracture allows bacteria to enter the injured area and increases the risk of complications.
- The fracture line may be *oblique* (at an angle to the bone) or *spiral* (curved around the bone). An *avulsed* fracture occurs when the fracture pulls bone and other tissues away from the point of attachment. It may also be described as *comminuted* (the bone breaks in many pieces), *compressed* (the bone is crushed), *impacted* (the broken bone ends are forced into each other) or *depressed* (the broken bone is forced inwards) (see Figure 38.2).
- *Complete fractures* involve the entire width of the bone, whereas incomplete fractures involve only a part of the width of the bone.
- A *stable* (non-displaced) *fracture* is one in which the bones maintain their anatomical alignment. An *unstable* (displaced) *fracture* occurs when the bones move out of correct anatomical alignment. If a fracture is displaced, immediate interventions are required to prevent further damage to soft tissue, muscle and bone.

Fractures may also be classified by point of reference on the bone, such as midshaft, middle third and distal third. The point of reference may also be specific, such as intra-articular or diaphyseal.

Fracture healing

Regardless of classification or type, fracture healing progresses over three phases: the inflammatory phase, the reparative phase and the remodelling phase (see 'Pathophysiology illustrated' below). The bleeding and inflammation that develop at the site of the fracture initiate the inflammatory phase. A haematoma forms between the fractured bone ends and around the bone surfaces. The osteocytes at the bone ends die as the haematoma clots, obstructing blood flow and depriving them of oxygen and nutrients. Necrosis of the cells heightens the inflammatory response, which in turn leads to vasodilation and oedema. In addition, fibroblasts, lymphocytes, macrophages and even osteoblasts from the bone migrate to the fracture site. Fibroblasts form a fibrin meshwork and promote the growth of granulation tissue and capillary buds. The lymphocytes and macrophages wall off the area, localising and containing the inflammation. The capillary buds invade the fracture site and supply a source of nutrients to promote the formation of collagen. The collagen allows calcium to be deposited.

Once calcium is deposited, a callus begins to form. In this reparative phase, osteoblasts promote the formation of new bone and osteoclasts destroy dead bone and assist in the

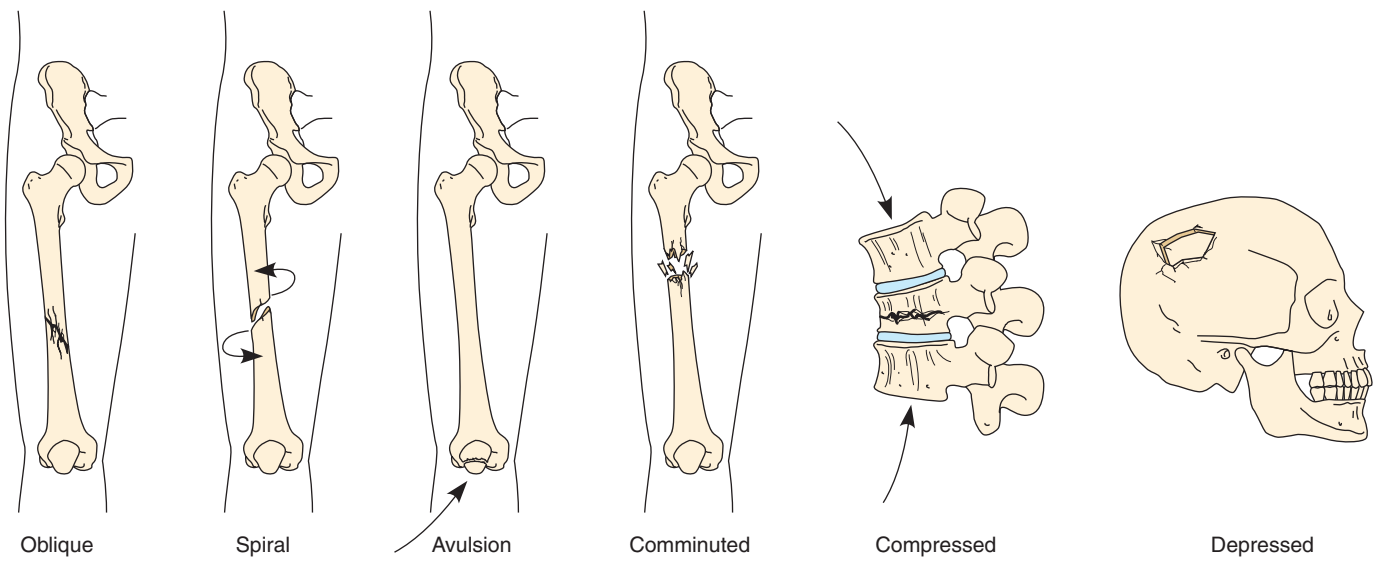


FIGURE 38.2 ■ Types of fractures

synthesis of new bone. Collagen formation and calcium deposition continue. During the remodelling phase, excess callus is removed and new bone is laid down along the fracture line. Eventually, the fracture site is calcified and the bone is reunited.

The age, physical condition of the person and the type of fracture sustained influence the healing of fractures. Other factors influence bone healing either positively or negatively and may be grouped according to their local or systemic influence (see Box 38.2). Healing time varies with the individual. An uncomplicated fracture of the arm or foot can heal in 6 to 8 weeks. A fractured vertebra will take at least 12 weeks to heal. Healing of a fractured hip may take from 12 to 16 weeks.

Manifestations

Fractures are often accompanied by soft tissue injuries that involve muscles, arteries, veins, nerves or skin. The degree of soft tissue involvement depends on the amount of energy or force transmitted to the area. Fracture manifestations and their causes are outlined in the 'Manifestations' box below.

Complications

Complications of musculoskeletal trauma are associated with pressure from oedema and haemorrhage, development of fat emboli, deep venous thrombosis, infection, loss of skeletal integrity or involvement of nerve fibres. Bone fragments may also result in further injury or complications.

BOX 38.2 Factors influencing bone healing

Positive factors

Local:

- Immobilisation
- Timely correction of displacement
- Application of ice
- Electrical stimulation

Systemic:

- Adequate amounts of growth hormone, vitamin D and calcium
- Adequate blood supply
- Absence of infection or diseases
- Younger age
- Moderate activity level prior to injury

Negative factors

Local:

- Delay in correction of displacement
- Open fracture (increases risk of infection)
- Presence of foreign body at fracture site

Systemic:

- Immunocompromised status
- Decreased circulation (as in diabetes or peripheral vascular disease)
- Malnutrition
- Osteoporosis
- Advanced age

Compartment syndrome

A compartment is a space enclosed by a fibrous membrane, or fascia. The fascia lines the compartment within the limbs and is non-expandable. Compartments within the limbs may enclose and support bones, nerves and blood vessels. **Compartment syndrome** occurs when excess pressure in a limited space constricts the structures within a compartment, reducing circulation to muscles and nerves. Acute compartment syndrome may result from haemorrhage and oedema within the compartment following a fracture or from a crush injury, or from external compression of the limb by a cast that is too tight. Increased pressure within the confined space of the compartment results in entrapment of nerves, blood vessels and muscles.

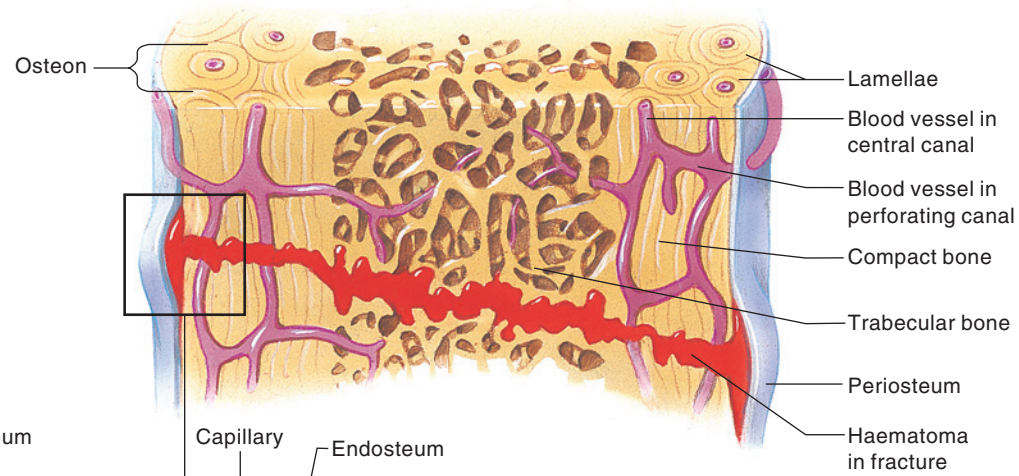
Entrapment of blood vessels limits tissue perfusion, beginning a cycle of events that may result in the loss of the limb. Inadequate oxygen supply causes cellular acidosis, which intensifies as cellular energy requirements are met through anaerobic metabolism. The capillaries inside the compartment dilate in an attempt to increase the supply of blood and oxygen. Additional blood and oxygen are not available and plasma proteins leak out into the interstitial tissues. The interstitial tissue then pulls fluid in to balance the protein load. As a result, oedema within the compartment increases. The oedema causes further compression of the vascular network and the cycle continues. Uninterrupted, this cycle threatens the person's limb and increases the risk of sepsis. Compartment syndrome usually develops within the first 48 hours of injury, when oedema is at its peak. Manifestations of compartment syndrome are listed in the box below. It is important to note that arterial pulses may remain normal, even when pressure within the compartment is high enough to significantly impair tissue perfusion.

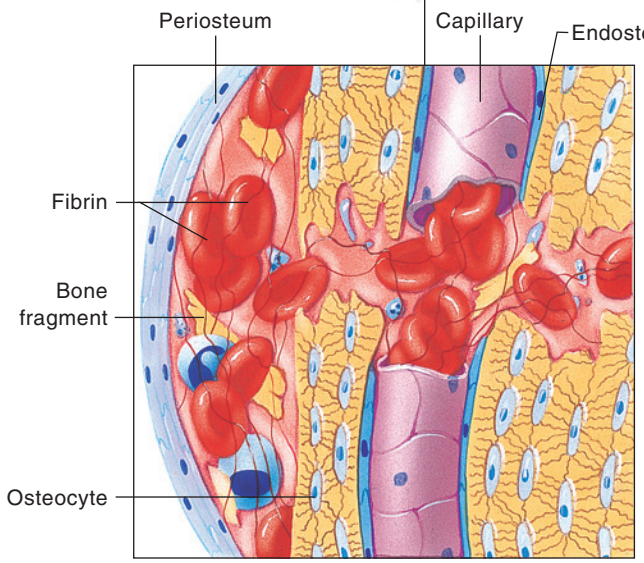
If compartment syndrome develops, interventions to alleviate pressure will be implemented; these may include removal of a tightly fitting cast. If the pressure is internal, a *fasciotomy*,

MANIFESTATIONS Fracture

MANIFESTATION	CAUSE
Deformity	Abnormal position of bones secondary to fracture and muscles pulling on fractured bone
Swelling	Oedema from localisation of serous fluid and bleeding
Pain/tenderness	Muscle spasm, direct tissue trauma, nerve pressure, movement of fractured bone
Numbness	Nerve damage or nerve entrapment
Guarding	Pain
Crepitus	Grating of bones or entrance of air in an open fracture. *Note:* Do not manipulate the extremity to elicit crepitus; doing so may cause additional damage.
Hypovolaemic shock	Blood loss or associated injuries
Muscle spasms	Muscle contraction near the fracture
Ecchymosis	Extravasation of blood into the subcutaneous tissue

Bone healing





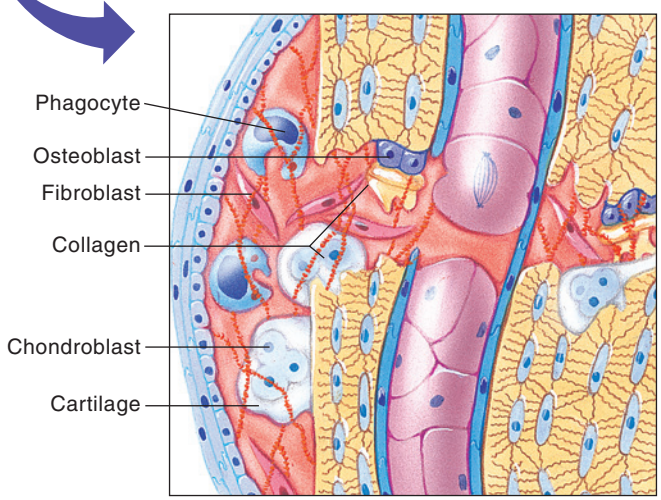
1. Bone injury

When a bone fractures, blood vessels within the bone and surrounding soft tissues tear and begin to bleed, forming a haematoma. Necrotic bone tissue adjacent to the fracture causes an intense inflammatory response characterised by vasodilation, exudate formation and white cell migration to the fracture site.

2. Fibrocartilaginous callus formation

Clotting factors within the haematoma form a fibrin meshwork. Within 48 hours, fibroblasts and new capillaries growing into the fracture form granulation tissue that gradually replaces the haematoma. Phagocytes begin to remove cell debris.

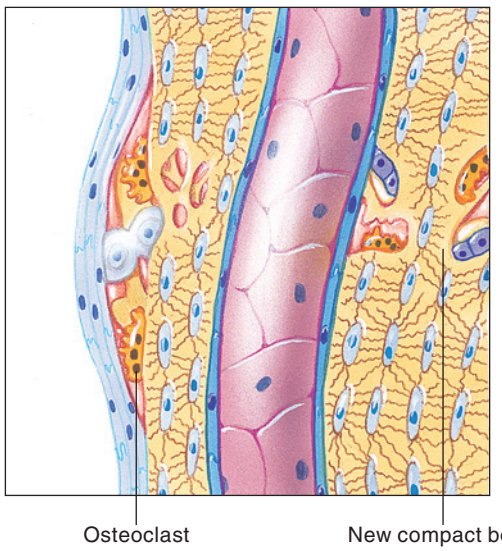
Osteoblasts, bone-forming cells, proliferate and migrate into the fracture site, forming a fibrocartilaginous callus. The osteoblasts build a web of collagen fibres from both sides of the fracture site that eventually unites to connect bone fragments, thus splinting the bone. Chondroblasts lay down patches of cartilage that provide a base for bone growth.

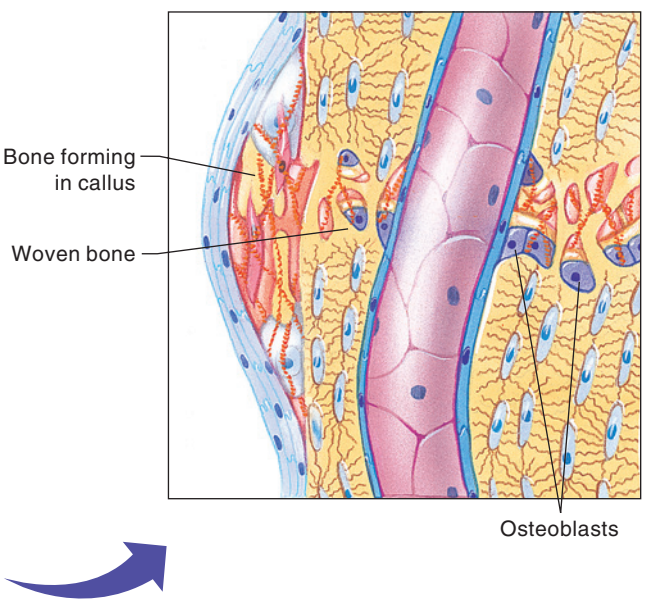


4. Bone remodelling

Osteoblasts continue to form new woven bone, which is in turn organised into the lamellar structures of compact bone. Osteoclasts resorb excess callus as it is replaced by mature bone.

As the bone heals and is subjected to the mechanical stress of everyday use, osteoblasts and osteoclasts respond by remodelling the repair site along the lines of force. This ensures that the repaired section of bone eventually resembles the structure of the uninjured part.





3. Bony callus formation

Osteoblasts continue to proliferate and synthesise collagen fibres and bone matrix, which are gradually mineralised with calcium and mineral salts to form a spongy mass of woven bone. The trabeculae of woven bone bridge the fracture. Osteoclasts migrate to the repair site and begin removing excess bone in the callus. Bony callus formation usually continues for 2 to 3 months.

a surgical intervention in which muscle fascia is cut to relieve pressure within the compartment, may be necessary. After a fasciotomy, the incision is left open and passive ROM exercises are performed on the extremity.

Volkmann's contracture, a common complication of elbow fractures, can result from unresolved compartment syndrome. Arterial blood flow decreases, leading to ischaemia, degeneration and contracture of the muscle. Arm mobility is impaired and the person is unable to completely extend the arm.

Fat embolism syndrome

Fat emboli occur when fat globules lodge in the pulmonary vascular bed or peripheral circulation. **Fat embolism syndrome (FES)** is characterised by neurological dysfunction, pulmonary insufficiency and a petechial rash on the chest, axilla and upper arms. Long bone fractures and other major trauma are the principal risk factors for fat emboli; hip replacement surgery also poses a risk of FES.

When a bone is fractured, pressure within the bone marrow rises and exceeds capillary pressure; as a result, fat globules leave the bone marrow and enter the bloodstream. Another contributing factor may be the stress-induced release of catecholamine, which causes the rapid mobilisation of fatty acids. Once the fat globules are released, they combine with platelets and travel to the brain, lungs, kidneys and other organs, occluding small blood vessels and causing tissue ischaemia.

Manifestations usually develop within a few hours to a week after injury. The manifestations result from the occlusion of the blood supply and the presence of fatty acids. Altered cerebral blood flow causes confusion and changes in level of consciousness. Pulmonary circulation may be disrupted and free fatty acids damage the alveolar–capillary membrane. Pulmonary oedema, impaired surfactant production and atelectasis can result in significant respiratory insufficiency and manifestations of acute respiratory distress syndrome (see Chapter 36). Fat droplets activate the clotting cascade, causing thrombocytopenia. Petechiae (pin-sized purplish areas from bleeding under the skin) appearing on the skin, buccal membranes and conjunctival sacs are thought to result from either microvascular clotting or the accompanying thrombocytopenia.

MANIFESTATIONS Compartment syndrome

EARLY MANIFESTATIONS
- Pain
- Normal or decreased peripheral pulse

LATER MANIFESTATIONS
- Cyanosis
- Tingling, loss of sensation (paraesthesias)
- Weakness (paresis)
- Severe pain, especially when the extremity is passively flexed
- Eventual renal failure (due to release of myoglobin into the bloodstream; myoglobin molecule is too large for effective filtration and excretion by kidney, and renal failure results)

Early stabilisation of long bone fractures is preventive for FES. Prompt identification and treatment of the syndrome are necessary to maintain adequate pulmonary function. In severe cases, the person may require intubation and mechanical ventilation to prevent hypoxaemia. Fluid balance is closely monitored. Corticosteroids may be administered to decrease the inflammatory response of lung tissues, stabilise lipid membranes and reduce bronchospasm (Porth & Matfin, 2014).

Deep venous thrombosis

A *deep venous thrombosis (DVT)* is a blood clot that forms along the intimal lining of a large vein. Three precursors linked to DVT formation are: (1) venous stasis or decreased blood flow; (2) injury to blood vessel walls; and (3) altered blood coagulation (see Table 38.2). Any or all of these precursors can cause a DVT to form. Damage to the lining of the vein causes the platelets to aggregate or clump together, forming the thrombus. Fibrin, white blood cells (WBCs) and red blood cells (RBCs) begin to cling to the thrombus and a tail forms. This tail or the entire thrombus may dislodge and move to the brain, lungs or heart. Five per cent of DVTs dislodge and enter the pulmonary circulation to form a pulmonary embolus. If the thrombus remains in the vein, venous insufficiency may result from scarring and valve damage.

If a DVT is present, there may be swelling, leg pain, tenderness or cramping. Not all people experience manifestations, however. For this reason diagnostic tests, such as a venogram or Doppler ultrasound of lower extremities, may be required. A venogram requires intravenous administration of dye in the radiology department, whereas a Doppler ultrasound study is non-invasive and can be performed at the person's bedside. Doppler ultrasonography uses sound waves to form an image on a computer screen.

The best treatment for DVT is prevention. Early immobilisation of the fracture and early ambulation of the person are imperative. The extremity should be elevated above the level of the heart. Frequent assessments of the injured extremity may lead to early recognition of DVT and prevent the formation of pulmonary embolus. Prophylactic anticoagulant administration is beneficial. Anti-embolism stockings and compression boots increase venous return and prevent stasis of blood. Constrictive clothing should be avoided.

The diagnosis of DVT requires rapid intervention. Fibrinolytic agents, which dissolve the clot, may be administered.

TABLE 38.2 Precursors of deep venous thrombosis

PRECURSOR	IMPLICATIONS FOR FRACTURES
Decreased blood flow	Common in people with a fracture who are immobilised and less active. Bed rest alone can decrease venous flow by 50%.
Injury to blood vessel wall	May occur as a direct result of the force that caused the fracture or from surgical manipulation.
Altered blood coagulation	May result from active blood loss. The body's attempt to maintain homeostasis leads to increased production of platelets and clotting factor

Heparin may be administered intravenously or subcutaneously, to prevent more clots from forming. Prophylactic warfarin, rivaroxaban or low-molecular-weight heparin (e.g. enoxaparin (Clexane)) may also be administered to prevent DVT (Hirsch et al., 2008; NPS MedicineWise, 2013). A vena cava filter may be placed to prevent the existing clot from entering the pulmonary circulation and forming a pulmonary embolus. In extreme cases in which anticoagulation therapy is contraindicated, a thrombectomy (surgical removal of the clot) may be necessary. See Chapter 31 for further discussion of DVT.

Infection

Infection is more likely to occur in an open fracture than a closed fracture, but any complication that decreases blood supply increases the risk of infection. Infection may result from contamination at the time of injury or during surgery. *Pseudomonas*, *Staphylococcus* or *Clostridium* organisms may invade the wound or bone. *Clostridium* infection is particularly serious because it may lead to severe gas gangrene and cellulitis, but any infection may delay healing and result in osteomyelitis—infection within the bone that can lead to tissue death and necrosis. (See Chapter 39 for a discussion of osteomyelitis.)

Delayed union and non-union

Delayed union is the prolonged healing of bones beyond the usual period. Many factors may inhibit bone healing, including poor nutrition, inadequate immobilisation, prolonged reduction time, infection, necrosis, age, immunosuppression and severe bone trauma resulting in multiple fragments. Delayed union is diagnosed by means of serial x-ray studies. It is important to note that x-ray findings may lag 1 to 2 weeks behind the healing process; for example, a person may be completely healed by week 13, but this fact may not be apparent on the x-ray until week 14.

Delayed union may lead to **non-union**, which can cause persistent pain and movement at the fracture site. Non-union may require surgical interventions, such as internal fixation and bone grafting. If infection is present, the bones are surgically debrided. Electrical stimulation of the fracture site may be as effective as bone grafting.

Reflex sympathetic dystrophy

Reflex sympathetic dystrophy may occur after musculoskeletal or nerve trauma. This term refers to a group of poorly understood post-traumatic conditions involving persistent pain, hyperaesthesias, swelling, changes in skin colour and texture, changes in temperature and decreased motion. Diagnosis is made by the person's history and physical examination. X-rays may demonstrate spotty osteoporosis and bone scans may reveal increased uptake of radionuclide. Treatment with a sympathetic nervous system blocking agent often alleviates the manifestations.

INTERPROFESSIONAL CARE

A fracture requires treatment to stabilise the fractured bone(s), maintain bone immobilisation, prevent complications and restore function. The diagnosis of a fracture is primarily based on physical assessments and x-rays.

Emergency care

Emergency care of the person with a fracture includes immobilising the fracture, maintaining tissue perfusion and preventing infection. In the case of serious trauma, normal body alignment must be maintained and may involve cervical immobilisation. Once the person is in a secure location, they are assessed for instability or deformity of the bone. If any deformity or instability is detected, the extremity is rapidly immobilised. Open wounds are covered with sterile dressings and bleeding may be controlled with a pressure dressing. The extremities are assessed for the presence of pulses, movement and sensation. The joints above and below the deformity are immobilised. Pulses, movement and sensation are re-evaluated after splinting.

The fracture is splinted to maintain normal anatomical alignment and prevent the fracture from dislocating. Splinting relieves pain and prevents further damage to the arteries, nerves and bones. Splinting can be accomplished with air splints. If equipment is not available, the limb may be secured to the body. For example, an arm may be secured to the torso with a sling or one leg may be strapped to the other leg.

Diagnosis

Diagnosis of a fracture begins with the history and initial assessment and usually is confirmed by radiographical tests. X-rays and bone scans are used to identify fractures (see Figure 38.3). Blood chemistry studies, full blood count (FBC), group and hold and coagulation studies may be used to assess blood loss, renal function, muscle breakdown and the risk of excessive bleeding or clotting. Diagnostic tests are described in Chapter 37.

Medications

Most people with a fracture require pharmacological interventions. The priority intervention focuses on relieving pain. In the case of multiple fractures or fractures of large bones, narcotics are administered initially. As healing progresses, the person begins to take oral medication for pain. Pain management for the person with a fracture is described in Box 38.3.

Stool softeners may be administered to decrease the risk of constipation secondary to narcotics and immobility. People who have sustained trauma are often placed on anti-ulcer medications or antacids. NSAIDs may be prescribed to decrease inflammation. Antibiotics may be administered prophylactically, particularly to people with open or complex fractures. Anticoagulants may be prescribed to prevent DVT.

Treatments

Fracture treatment may involve a closed reduction and the application of a cast, or may include one or more of the following: traction, casts, surgery and electrical bone stimulation.

TRACTION Muscle spasms usually accompany fractures and may pull bones out of alignment. Traction is the application of a straightening or pulling force to return the factured bones to or maintain them in normal anatomical position. Weights are applied to maintain the necessary force (see Figure 38.4). Traction is not used often in orthopaedic management due to the advancement of orthopaedic implant technologies and operative techniques. It may be used in the short term for the individual awaiting operative fixation.

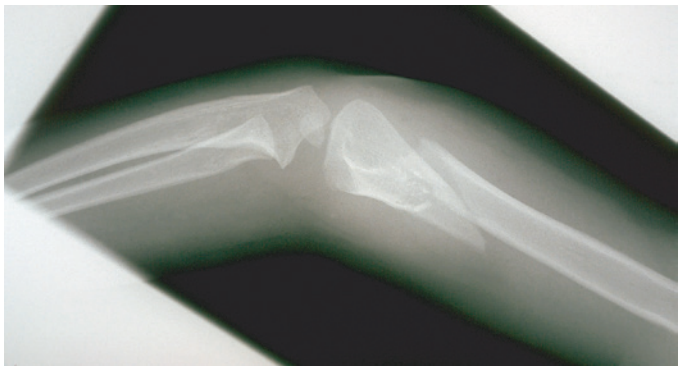


FIGURE 38.3 ■ X-ray of an oblique fracture of the femur

Source: © Charles Stewart and Associates.

Types of traction are as follows:

- In *manual traction*, the hand directly applies the pulling force.
- *Skin traction* (also called straight traction) is used to control muscle spasms and to immobilise a part of the body before surgery, with traction exerting its grabbing and pulling force through the person's skin. The most common type of skin traction is Buck's traction, in which traction tape or a foam boot is applied to the lower portion of a person's leg and a free-hanging weight is attached to the taped or booted area (see Figure 38.4A). Buck's traction is used to immobilise the leg before surgery to repair a fracture of the proximal femur. The advantage of skin traction is the relative ease of use and ability to maintain comfort. The disadvantage is that the weight required to maintain normal body alignment or fracture alignment cannot exceed the tolerance of the skin—about 13.2 kg per extremity. It is important to ensure that the weights remain hanging freely; they should never rest on the bed or the floor. The nurse may have to reposition the person or the weights if this occurs.
- *Balanced suspension traction* involves more than one force of pull. Several forces work in unison to raise and support the person's injured extremity off the bed and pull it in a straight line away from the body (see Figure 38.4B). The advantage of this type of traction is that it increases mobility without threatening joint continuity. The disadvantage is that the increased use of multiple weights makes the person more likely to slide down in the bed.
- *Skeletal traction* is the application of a pulling force through placement of pins into the bone (see Figure 38.4C). The person may receive a local, spinal or general anaesthetic, and the pins are inserted into the bone. This type of traction must be applied under sterile conditions because of the increased risk of infection. One or more pulling forces may be applied with skeletal traction. The advantage of this type of traction is that more weight can be used to maintain the proper anatomical alignment if necessary. The disadvantages include increased anxiety, increased risk of infection and increased discomfort. The weights used for skeletal traction are not removed by the nurse.

Nursing interventions for people in traction are described in Box 38.4.

CASTS

A cast is a rigid device applied to immobilise the injured bones and promote healing. The cast immobilises the joints above and below the fractured bone so that the bone will not move during healing. A fracture is first reduced manually (by hand) and a cast is then applied. Casts are applied on people who have relatively stable fractures.

BOX 38.3 Pain management in the person with a fracture

The person who has had musculoskeletal trauma experiences pain from many different causes:

- the interruption in the continuity of the bone itself
- damage to ligaments and tendons
- swelling of tissues around the trauma site
- muscle spasms
- tissue anoxia from swelling inside a cast, splint or the muscle fascia sheath
- haematoma formation
- pressure over bony prominences from casts or splints.

The pain is often severe and may be described as sharp, aching or burning. Carefully assess any complaint of pain; pain may be an indication of a serious complication, such as compartment syndrome, decreased tissue perfusion and neurovascular impairment, or pressure ulcers. Do not administer analgesics until the location, character and duration of pain have been carefully assessed. After the cause of the pain has been identified, the following nursing interventions may be implemented:

1. Administer prescribed analgesics, which may include NSAIDs and narcotic analgesics. For serious fractures or following orthopaedic surgery, patient-controlled analgesia (PCA) or epidural methods of providing pain relief may be used. If medications are used on an as-needed basis, tell the person to request the medication before the pain is severe; alternatively, offer the medications at regular intervals for the first 24 to 48 hours. Reassure the person that addiction does not result from taking medications to relieve fracture or surgical pain. Most people require only oral analgesics by the third or fourth day after orthopaedic surgery.
2. Elevate the involved extremity and apply cold (if prescribed) to help decrease swelling.
3. Monitor and drain the accumulated fluids in any drainage devices to ensure patency and to decrease the possibility of haematoma formation.
4. Encourage the person to wiggle fingers or toes on an extremity in a cast or traction to improve venous return and decrease oedema.
5. Assist the person to change positions to relieve pressure and use pillows to provide support.
6. Teach the person alternative methods of pain management, such as relaxation and guided imagery.
7. Notify the doctor of unrelieved pain, which may indicate a serious complication such as compartment syndrome or neurovascular impairment.

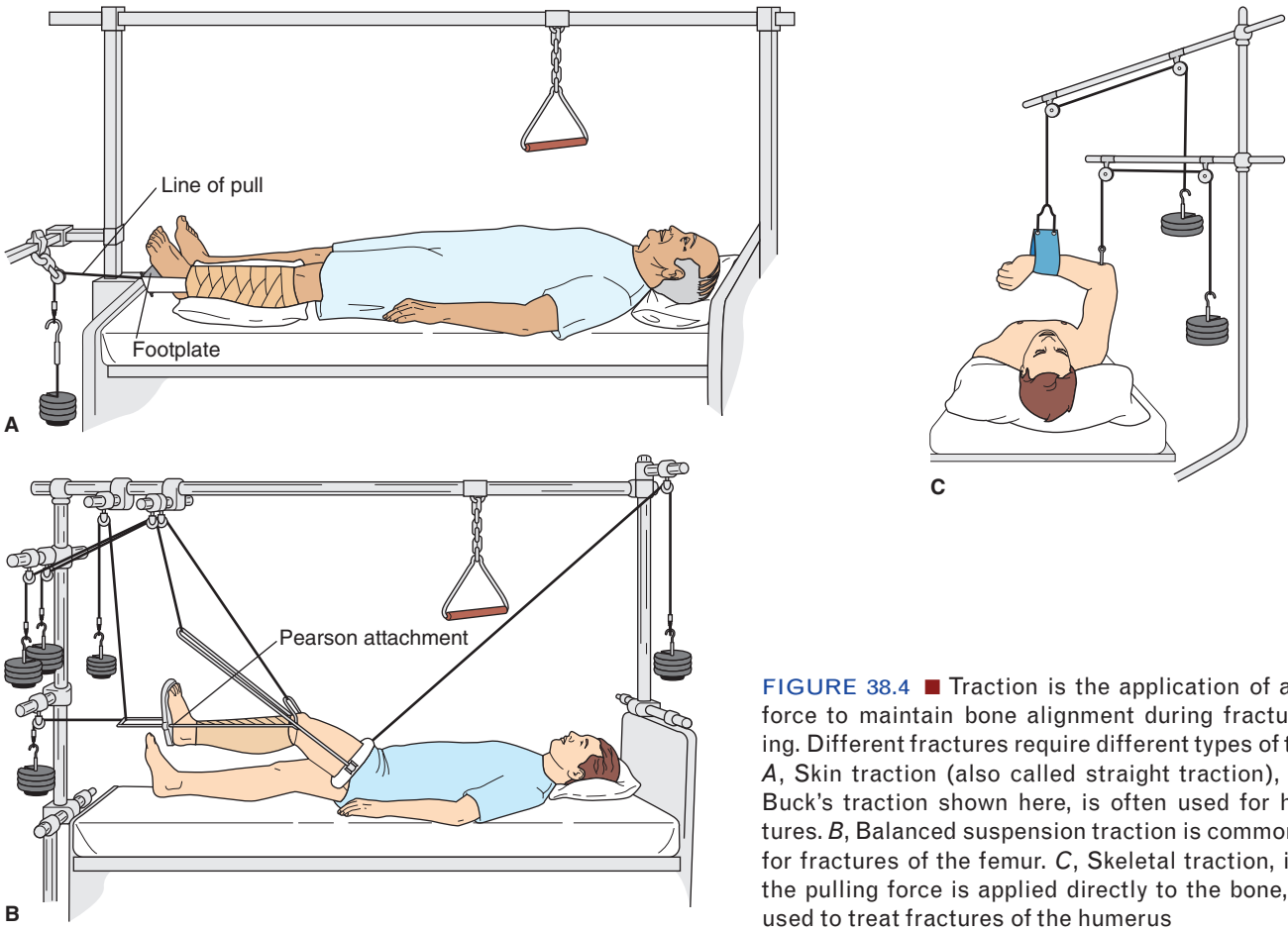


FIGURE 38.4 ■ Traction is the application of a pulling force to maintain bone alignment during fracture healing. Different fractures require different types of traction. *A*, Skin traction (also called straight traction), such as Buck's traction shown here, is often used for hip fractures. *B*, Balanced suspension traction is commonly used for fractures of the femur. *C*, Skeletal traction, in which the pulling force is applied directly to the bone, may be used to treat fractures of the humerus

The cast, which may be composed of plaster or fibreglass, is applied over a thin cushion of padding and moulded to the normal contour of the body. The cast must be allowed to dry before any pressure is applied to it; simply palpating a wet cast with the fingertips will leave dents that may cause pressure ulcers. A plaster cast may require up to 48 hours to dry, whereas a fibreglass cast dries in less than 1 hour. The type of cast applied is determined by the location of the fracture (see Figure 38.5). Nursing care of the person with a cast is discussed in the box below. During follow-up appointments,

BOX 38.4 Nursing interventions for people in traction

- In skeletal traction, never remove the weights.
- In skin traction, remove weights only when intermittent skin traction has been ordered to alleviate muscle spasm.
- For traction to be successful, countertraction is necessary. In most instances, the countertraction is the person's weight. Therefore, do not wedge the person's foot or place it flush with the footboard of the bed.
- Maintain the line of pull:
  a. Centre the person on the bed.
  b. Ensure that weights hang freely and do not touch the floor.
- Ensure that nothing is lying on or obstructing the ropes. Do not allow the knots at the end of the rope to come into contact with the pulley.
- If a problem is detected, assist in repositioning. The area of the fracture must be stabilised when the person is repositioned.
- In skin traction:
  a. Frequently assess skin for evidence of pressure, shearing or pending breakdown.
  b. Protect pressure sites with padding and protective dressings as indicated.
- In skeletal traction:
  a. Frequent skin assessments should include pin care per policy.
  b. Report signs of infection at pin sites, such as redness, drainage and increased tenderness.
  c. The person may require more frequent analgesic administration.
- Perform neurovascular assessments frequently.
- Assess for common complications of immobility, including formation of pressure ulcers, formation of renal calculi, deep venous thrombosis, pneumonia, paralytic ileus and loss of appetite.
- Teach the person and family about the type and purpose of the traction.

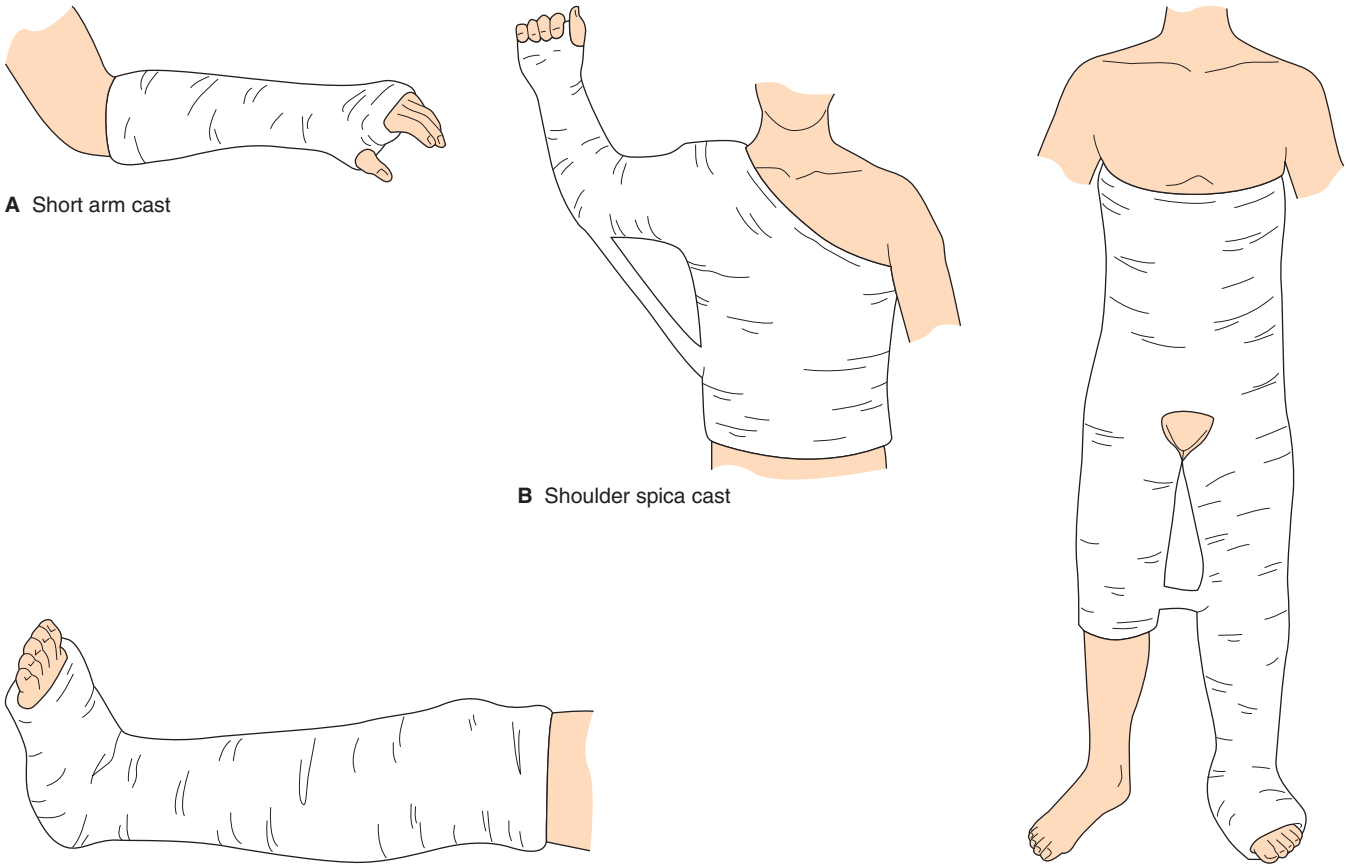


FIGURE 38.5 ■ Examples of types of casts used to immobilise fractures

the doctor may x-ray the bone to assess alignment and healing and possibly remove the cast for skin assessment.

SURGERY Surgery is indicated for a fracture that requires direct visualisation and repair, a fracture with common long-term complications or a fracture that is severely comminuted and threatens vascular supply.

The simplest form of surgery is done by external fixation with an external fixator device. An external fixator consists of a frame connected to pins that are inserted perpendicular to the long axis of the bone (see Figure 38.6). The number of pins inserted varies with the type and site of the fracture, but in all cases the same number of pins are inserted above and below the fracture line. The pins require care similar to that of skeletal traction pins. The person is monitored for infection, and frequent neurovascular assessment is performed. The fixator increases independence while maintaining immobilisation.

NURSING CARE OF THE PERSON with a cast

NURSING INTERVENTIONS

- Perform frequent neurovascular assessments.
- Palpate the cast for 'hot spots' that may indicate the presence of underlying infection.
- Report any drainage promptly.
- Assess the injured extremity for coolness, changes in colour, increased pain, increased swelling and/or loss of sensation.
- Use a blow dryer on the cool setting to relieve itching by blowing cool air into the cast.
- If a sling is used, it should distribute the weight of the cast evenly around the neck. Do not roll the sling; this can impair circulation to the neck.
- If crutches are used, arrange for a physiotherapist to teach correct crutch walking.
- When the cast is removed, an oscillating cast remover will be used. A guard prevents the cast remover from penetrating past the depth of the cast, so it will not cut the person. It is noisy and the person will feel vibration.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Do not place any objects in the cast.
- If the cast is made of plaster, keep it dry.
- If the cast is made of fibreglass, dry it with a blow dryer on the cool setting if it becomes wet. Be aware that some padding is not waterproof and therefore will need to be checked prior to the person showering or swimming.

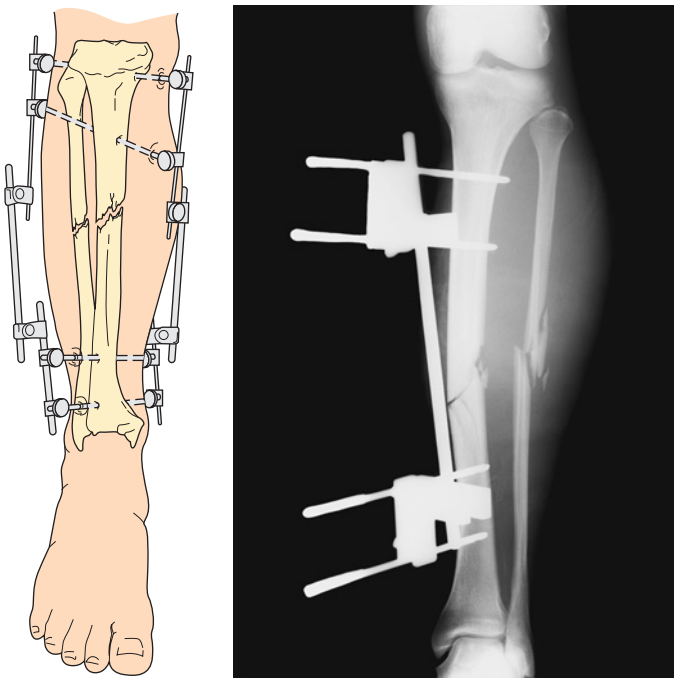


FIGURE 38.6 ■ In external fixation, pins are placed through the bone above and below the fracture site to immobilise the bone. External fixation rods hold the pins in place

Source: Image from SPL/Science Source.

Internal fixation can be accomplished through a surgical procedure called an *open reduction and internal fixation (ORIF)*. In this procedure, the fracture is reduced (placed in correct anatomical alignment) and nails, screws, plates or pins are inserted to hold the bones in place (see Figure 38.7). Open fractures of the arms and legs are most commonly repaired in this way. Hip fractures in older people are almost always repaired with ORIF to prevent complications and to allow early rehabilitation. Interventions for postoperative nursing care are presented in Box 38.5.

BOX 38.5 Nursing interventions for people with internal fixation

- Expect the person to have sutures and at least one drain (the type depends on the surgeon's choice).
- Perform neurovascular assessments frequently.
- Assess the following:
  a. wounds for drainage
  b. drainage device for the removal of serosanguineous fluid
  c. bowel sounds
  d. lung sounds.
- Administer medications, such as analgesics and antibiotics, per doctor's orders.
- In hip fractures, place an abductor pillow between the legs to prevent dislocation of the hip joint.
- Arrange for physical and occupational therapy, as ordered.
- Assist with weight-bearing program, if ordered.
- Encourage early mobilisation, coughing and deep breathing, as appropriate, to help prevent complications.

ELECTRICAL BONE STIMULATION Electrical bone stimulation is the application of an electrical current at the fracture site. It is a painless method of treating fractures that are not healing appropriately. The electrical stress increases the migration of osteoblasts and osteoclasts to the fracture site. Mineral deposition increases, promoting bone healing. Electrical bone stimulation can be accomplished invasively or non-invasively. In invasive stimulation, the surgeon inserts a cathode and a lead wire at the fracture site. The lead wire is attached to an internal or external generator, which delivers electricity through the lead wire to the cathode 24 hours a day. In non-invasive inductive stimulation, a treatment coil encircles the cast or skin directly over the fracture site. The coil is attached to an external generator that runs on batteries. The electricity goes through the skin to the fracture site. The time period for external stimulation can vary from 3 to 10 hours per day. The person may be taught to self-administer the non-invasive electrical stimulation. Electrical

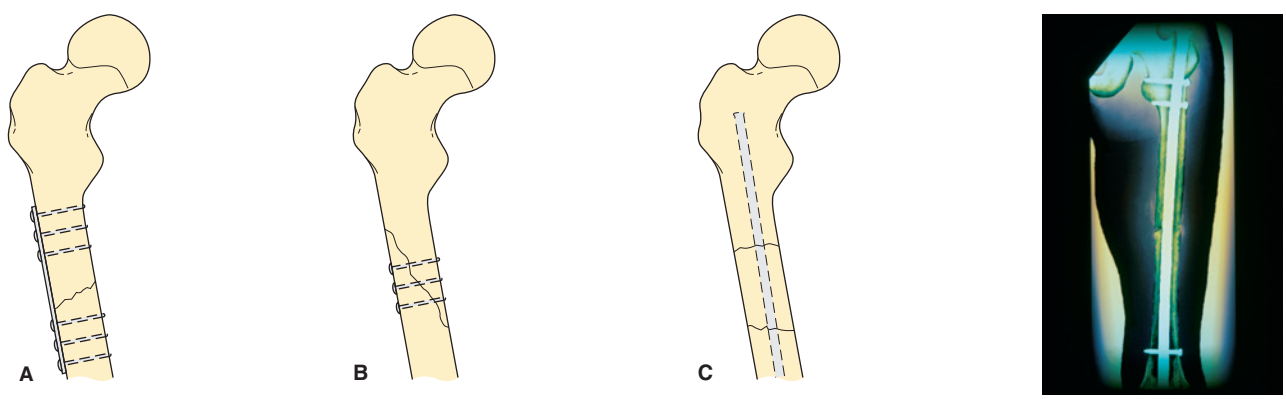


FIGURE 38.7 ■ Internal fixation hardware is entirely within the body. *A*, Fixation of a short oblique fracture using a plate and screws above and below the fracture. *B*, Fixation of a long oblique fracture using screws through the fracture site. *C*, Fixation of a segmental fracture using a medullary nail

Source: Image © ISM/Phototake. All rights reserved.

bone stimulation is contraindicated in the presence of infection and for upper extremities if the person has a pacemaker.

Fractures of specific bones or bony areas

Causes, manifestations, complications, treatment and selected nursing interventions are described for the following fractures: skull, face, spine, clavicle, humerus, elbow, radius/ulna, wrist/hand, ribs, pelvis, femur, hip, tibia/fibula and ankle/foot.

Fracture of the skull

The skull may be fractured as a result of either a fall or a direct blow. The person must be assessed for neurological damage and any loss of consciousness (LOC) must be documented. A complete neurological assessment is conducted: pupillary reaction to light; movement and strength of all extremities; complaints of nausea and vomiting; LOC and orientation to person, place and time are noted. A displaced skull fracture, which is referred to as depressed, may press on the brain and cause neurological damage. Brain injuries related to skull fractures are discussed in Chapter 41.

Fracture of the face

Fracture of the facial bones may result from a direct blow. The person presents with haematomas, pain, oedema and bony deformity. Non-displaced fractures are monitored to ensure the airway is not compromised. The person is observed for any neurological deficits. Severely displaced or multiple facial fractures are treated with ORIF with wires or plates.

Nursing care focuses on maintaining the airway by helping the person clear secretions from the oropharynx. The nurse monitors the person's breathing for increased effort or tachypnoea and notifies the doctor immediately if these findings are noted. Pain is treated with analgesics and body image disturbances are addressed. If the person asks to see their face, the nurse should plan to stay with the person and answer questions while the person looks in a mirror.

Fracture of the spine

The spine can be injured in many ways, including sports injuries, falls and motor vehicle accidents. The spine can be fractured in the cervical, thoracic, lumbar or sacral area. The most severe complication of spine fracture is injury to the spinal cord (discussed in Chapter 42). A fracture to the vertebrae may cause the bones to become displaced and apply pressure on the spinal cord which may in turn result in permanent paralysis.

A non-displaced cervical spinal fracture may be treated with a cervical collar or a halo immobilising brace. The displaced cervical fracture is reduced by manual or skeletal traction and, eventually, application of a brace and/or surgical stabilisation of the bones with plates and screws. Immobilisation after a spinal fracture may last as long as 6 months.

Fracture of the clavicle

A fracture of the clavicle commonly results from a direct blow or a fall. The most common location is midclavicular. A person with a midclavicular fracture typically assumes a protective slumping position to immobilise the arm and prevent shoulder movement. A less common fracture occurs along the distal third of the clavicle. This type of fracture may be associated with ligament damage. Injuries to the clavicle may be associated with skull or cervical fractures. The fractured bone, if displaced, may lacerate the subclavian vessels and result in haemorrhage. The fractured bone may also puncture the lung, resulting in a pneumothorax. Malunion may occur at the fracture site and result in asymmetry of the clavicles. Injury to the brachial plexus may result in numbness and decreased movement of the arm on the affected side.

A deformity may be observed or palpated along the clavicle. Treatment focuses on immobilising the fractured bone in normal anatomical position by applying a clavicular strap, or a surgical repair may be necessary.

Fracture of the humerus

The exact location of the fracture, the presence of displacement and the results of the neurovascular examination determine the severity of a fracture of the humerus and the appropriate interventions. Treatment focuses on immobilising the fractured bone in normal anatomical position. Common complications of humeral fracture include nerve and ligament damage, frozen or stiff joints, and malunion. Early interventions may prevent permanent damage.

Fractures of the proximal humerus are common in older adults. A simple non-displaced fracture of the proximal humerus (near the humeral head) with a normal neurovascular assessment can be safely treated with immobilisation. A more complicated displaced fracture of the proximal humerus with bone fragmentation requires surgical intervention. The more severe the fracture and damage to soft tissue, the more likely it is that the range of motion (ROM) of the shoulder will be impaired. Rehabilitative measures focus on increasing ROM.

The humerus may also fracture along the shaft, usually as a direct result of trauma. If the humeral shaft fracture is simple and non-displaced, a hanging arm cast is applied. This cast maintains alignment of the fracture by using the pulling force of gravity; therefore, the person must be instructed not to rest the cast on anything to alleviate the weight. If the person is on bed rest, a hanging arm cast is not applied because the arm would not be able to hang freely. Instead, the fracture is immobilised with external skeletal traction. This traction places the injured arm in an upright position over the face and weights are hung off the distal portion of the humerus (see Figure 38.4C). Nursing interventions for people with fractures of the humerus are presented in Box 38.6.

Fracture of the elbow

The most common location of an elbow fracture is the distal humerus. Elbow fractures usually result from a fall or a direct blow to the elbow. The person guards the injured extremity, holding the arm rigidly in a flexed position or an extended position. Because the radius, ulna or humerus may be involved in the elbow fracture, all three bones must be visualised by x-ray.

Complications of an elbow fracture include nerve or artery damage and haemarthrosis, a collection of blood in the elbow joint. The most serious complication of an elbow fracture is Volkmann's contracture, which results from arterial occlusion and muscle ischaemia. The person complains of forearm pain, impaired sensation and loss of motor function. Rapid

BOX 38.6 Nursing interventions for people with fractures of the humerus

- Perform neurovascular assessments frequently.
- Administer prescribed medications to alleviate pain.
- Encourage exercises for people with a hanging cast:
  a. finger exercises: move each finger of the affected arm through complete range of motion
  b. pendulum shoulder exercises: dangle the affected arm at the side and move it forwards and backwards about 30 degrees in each direction.
- If the person is discharged, instruct the person and family in cast care and sling application, neurovascular assessments, exercises, prescribed pain medications and manifestations of complications.
- If the person is admitted to the hospital, provide preoperative teaching.

interventions are aimed at relieving pressure on the brachial artery and nerve, and preventing muscle atrophy.

Non-displaced elbow fractures are treated by immobilising the fracture with a posterior splint or cast. The displaced fracture is first reduced and then immobilised. Nursing interventions focus on alleviating pain, maintaining immobilisation and educating people in neurovascular assessments.

Fracture of the radius and/or ulna

Fractures of the radius and ulna may occur as a result of either indirect injury, such as twisting or pulling on the arm, or direct injury, such as that resulting from a fall. The usual treatment of radius fractures depends on the location. The proximal radial head may be fractured from a fall on an outstretched hand. Blood commonly collects in the elbow joint and must be aspirated. If the fracture is non-displaced, a sling is applied. If the fracture is displaced, surgical intervention is required. After surgical repair of a displaced fracture, the arm is splinted with a posterior plaster splint. The person avoids movement for the first week and then initiates movement gradually.

When both bones are broken, the fracture is usually displaced. The person complains of pain and inability to turn up the palm of the hand. A non-displaced fracture is casted for about 6 weeks and either a shorter cast or a brace is then applied for 6 more weeks. If the fracture is displaced, surgical intervention is performed. The doctor reduces the fracture and may insert pins or screws to keep the bones in alignment. After the surgery, a cast is applied and the person is encouraged to exercise the fingers.

Complications after a radius and/or ulnar fracture include compartment syndrome, delayed healing and decreased wrist and finger movement. After surgery, the person also has an increased risk of infection. Nursing interventions focus on alleviating pain, maintaining immobilisation and educating people in neurovascular assessments, the importance of elevation and the need to inform the doctor of changes in sensation or an increase in pain.

Fractures in the wrist and hand

Wrist fractures often result from a fall onto an outstretched hand or onto the back of the hand. A common type of wrist fracture is *Colles' fracture*, in which the distal radius fractures after a fall onto an outstretched hand. The person with a wrist fracture presents with a bony deformity, pain, numbness, weakness and decreased ROM of the fingers. The capillary refill and sensation of the hand must be assessed.

The hand is composed of many bones. Most commonly, the metacarpals and phalanges are involved in a hand fracture. The injuring mechanism in a hand fracture varies from striking an object with a closed fist to closing a hand in a door. The person presents with complaints of pain, oedema and decreased ROM.

Comparative x-rays may be obtained to compare left and right wrists and hands. Complications of wrist and hand fractures are compartment syndrome, nerve damage, ligament damage and delayed union. A wrist fracture is commonly treated with closed reduction, cast application and elevation of the injured extremity. A hand fracture is splinted and elevated.

Nursing interventions focus on alleviating pain and educating the person in neurovascular assessments, the importance of elevation and how to exercise the fingers to prevent stiffness. If the dominant hand is injured, the person will require assistance in performing activities of daily living (ADLs).

Fracture of the ribs

Rib fractures commonly result from blunt chest trauma. The location of the fracture and involvement of underlying organs determine the severity of the injury. Fractures of the first through to third ribs may result in injury to the subclavian artery or vein. Fractures of the lower ribs may result in spleen and liver injuries.

The person presents with a history of recent chest trauma. Typically, the person complains of pain along the lateral portion of the rib. Palpation of the rib reveals a bony deformity and increases pain. Deep inspiration also increases pain. The skin over the fracture site may be ecchymotic (bruised).

A complication of rib fractures is a flail chest, which results from the fracture of two or more adjacent ribs in two or more places and the formation of a free-floating segment that moves in the opposite direction to the rib cage. The bony instability impairs respirations. Treatment is aimed at stabilising the flail segment and supporting respirations. Other complications of rib fractures include pneumothorax and/or haemothorax. The fractured rib may pierce and injure the lung. The lower ribs may pierce the liver or spleen, resulting in intra-abdominal bleeding. Pneumonia may also develop from ineffective clearing of respiratory secretions.

A simple rib fracture is treated with pain medication and instructions for coughing, deep breathing and splinting. The person is also instructed to return to the emergency room if shortness of breath develops. Nursing interventions focus on alleviating pain and teaching the person about splinting. Because deep inspiration increases pain, people frequently avoid it. The person may be instructed to splint the injured rib with the hand or a pillow, and to take deep breaths and cough to decrease the chance of developing pneumonia and/or atelectasis. Incentive spirometry is encouraged.

Fracture of the pelvis

Pelvic fractures are often caused by trauma, such as a fall or a motor vehicle crash. The person with a pelvic fracture presents with pain in the back or hip area. A single fracture in the pelvis

is treated conservatively with bed rest on a firm mattress. Log-rolling increases person comfort. A pelvic fracture with two fracture sites is considered unstable and treated with surgery. An external fixator may be applied to stabilise the pelvis. In the person who is not stable for surgery, a pelvic sling may be used. The pelvic sling stabilises the pelvis and allows the person to move in bed with less pain. Common complications include hypovolaemia, spinal injury, bladder injury, urethral injury, kidney damage and gastrointestinal trauma.

Nursing care focuses on alleviating discomfort, maintaining immobilisation and preparing the person for surgery if necessary. The nurse monitors the person for increased heart rate, decreased blood pressure and decreasing haemoglobin levels. These findings may indicate impending hypovolaemia due to bleeding into the pelvis. Any blood in the urine should be reported to the doctor; this may indicate kidney, bladder or urethral damage.

Fracture of the shaft of the femur

A large amount of force, such as from motor vehicle crashes, falls or acts of violence, is required to fracture the shaft of the femur. People with femoral shaft fractures often have associated multiple traumas. A fracture of the femoral shaft is manifested by an oedematous, deformed, painful thigh. The person is unable to move the hip or knee. Initial assessment focuses on the circulation and sensation present in the affected extremity. Pedal pulses and capillary refill in the affected extremity are compared to the unaffected extremity. Complications of a femoral shaft fracture include hypovolaemia due to blood loss (which may be as great as 1.0 to 1.5 L), fat embolism, dislocation of the hip or knee, muscle atrophy and ligament damage.

Treatment of fractures of the shaft of the femur initially includes skeletal traction to separate the bony fragments and reduce and immobilise the fracture. Depending on the location and severity of the fracture, traction may be followed by either external or internal fixation. Strength in the affected extremity is maintained through gluteal and quadricep exercises. ROM exercises for unaffected extremities are critical in preparation for ambulation. Although full weight bearing is usually restricted until x-rays demonstrate bone union, the person may be allowed to carry out non-weight-bearing activities with an assistive device.

The nurse assesses pulses in the extremity and compares them bilaterally. Sensation is evaluated by asking whether the person can feel touch and discriminate sharp from dull objects. Nursing interventions include providing pain medication, providing reassurance and decreasing anxiety, and assisting with exercises of the lower legs, feet and toes.

Fracture of the hip

A hip fracture refers to a fracture of the femur at the head, neck or trochanteric regions (see Figure 38.8). Hip fractures are classified as intracapsular or extracapsular. *Intracapsular fractures* involve the head or neck of the femur; *extracapsular fractures* involve the trochanteric region. The majority of hip fractures involve the neck or trochanteric regions. The femoral head and neck lie within the joint capsule and are not covered in periosteum; thus, they do not have a large blood supply. Fractures at this location usually fragment, further decreasing blood supply and increasing the risk of non-union and avascular necrosis. The trochanteric region is covered in periosteum and therefore has more blood supply than the head or neck.

Hip fractures are a common clinical issue in the older adult in both Australia and New Zealand. It is predicted that by the age of 90, 33% of all women and 19% of all men will have had a hip fracture (Agency for Clinical Innovation (ACI), 2014). In adults of both countries, hip fractures are more prevalent in women than in men, due to osteoporosis. The mortality rate for people with hip fractures also increases by 25% (Australian Institute of Health and Welfare (AIHW), 2015). Hip fractures cause a decrease in mobility and independence. Factors contributing to falls include problems with gait and balance,

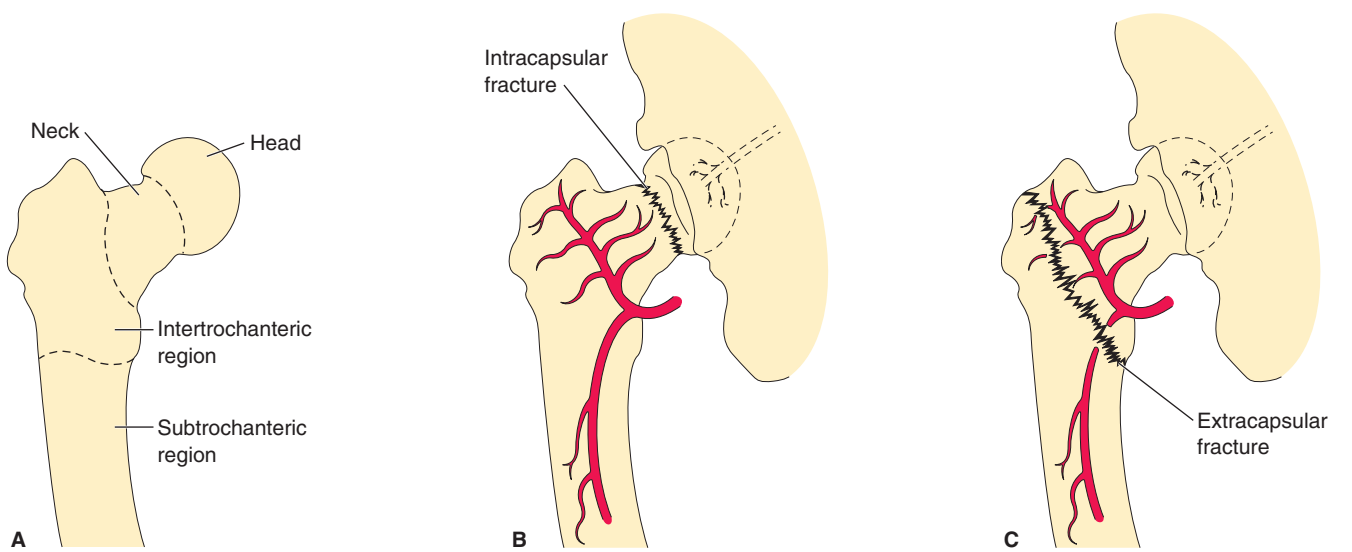


FIGURE 38.8 ■ Regions where hip fractures may occur: *A*, The head of the femur, the neck of the femur and the trochanteric regions of the femur. *B*, Intracapsular fractures occur across the head or neck of the femur. *C*, Extracapsular fractures occur across the trochanteric regions. Note how both intracapsular and extracapsular fractures disrupt the blood supply to the bone

neurological and musculoskeletal impairments, dementia, psychoactive medications and visual impairments. Modifiable risk factors, identified through research, include lower body weakness, problems with walking and balance, and taking four or more medications or any psychoactive medications.

Hip fractures occur in older adults as a result of decreases in bone mass and the increased tendency to fall. Whether the femur breaks spontaneously and causes the fall, or whether the fall causes the fracture, is not always clear; regardless of the cause of the fracture, rapid interventions are required to prevent bone necrosis. Assessment findings commonly associated with a hip fracture are pain, inability to walk and shortening and external rotation of the affected lower extremity. Rarely, the fracture dislocates posteriorly; if that occurs, the extremity may internally rotate. However, some people with a hip fracture have only vague pain in the buttocks, knees, thighs, groin or back, and their ability to walk is unaffected. If the fracture is not visible on x-ray, a bone scan or MRI may be done to confirm the presence of the fracture.

A hip fracture may be treated with traction to decrease muscle spasms, followed by surgery (however, this is being used less) (ACI, 2014); or surgery may be performed immediately or within the first 24 hours. The goal of surgery is to reduce and stabilise the fracture, thereby increasing mobility, decreasing pain and preventing complications. Surgery usually consists of ORIF of the fracture. Fixation is accomplished by securing the femur in place with pins, screws, nails or plates (see Figure 38.9A). An ORIF works well for fractures in the trochanteric area. Fractures of the femoral neck frequently disrupt blood supply to the femoral head. If blood supply is disrupted, the surgeon will replace the femoral head with a prosthesis (see Figure 38.9B). If the acetabulum has been damaged, the surgeon may insert a metal cup. Replacement of either the femoral head or the acetabulum with a prosthesis is called a *hemiarthroplasty*. Replacement of both the femoral head and the acetabulum is a *total hip arthroplasty (THA)*, discussed in Chapter 39.

Nursing care for a person with a hip fracture focuses on maintaining skin integrity, preventing infection, alleviating pain, maintaining circulation to the injured extremity and increasing mobility, and is discussed in more detail in the following 'Nursing care' section.

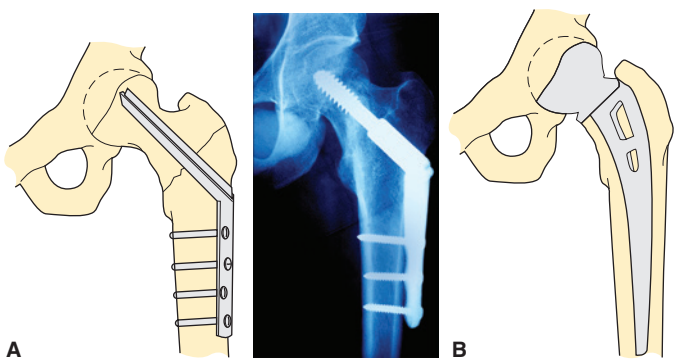


FIGURE 38.9 ■ Surgical fixation of hip fractures. *A*, A surgical nail or screw used to stabilise an intertrochanteric fracture. *B*, Use of a hip prosthesis (artificial hip) to replace a damaged femoral head

Source: Image from Cavallini James/BSIP/SuperStock.

FAST FACTS

Hip fracture in older adults

- Almost one in three older Australians has a fall each year.
- Over 40% have multiple falls.
- Over 30% require medical attention after a fall.
- Hip fracture in older adults is most often the result of a fall; in contrast, car crashes are the most common cause of a hip fracture in young and middle-aged adults.
- The risk of a fractured hip increases with each decade of life, especially in Caucasian postmenopausal women, who have the highest incidence of osteoporosis. Women have about 80% of all hip fractures. Women who smoke are at greater risk because smoking reduces bone density in menopausal women.
- Complications are related both to the fracture and to the resulting treatment. Only a small number of people retain their previous mobility, while about 20% require nursing home care.
- Half of all older adults hospitalised for a hip fracture cannot return home or live independently after the fracture.

Sources: Data from Australian Commission on Safety and Quality in Health Care (ACSQHC) (2015); Centers for Disease Control and Prevention (2012); Queensland Health (2015).

Fracture of the tibia and/or fibula

Fractures of the lower extremities often result from a fall on a flexed foot, a direct blow or a twisting motion. The person presents with oedema, pain, bony deformity and a haematoma at the level of injury.

Circulation and sensation are assessed to rule out common complications of the fracture, including damage to the peroneal nerve or tibial artery, compartment syndrome, haemarthroses and ligament damage. Peroneal nerve damage may be indicated by the person's inability to point the toe upwards on the affected side. Tibial artery damage may be the cause of an absent dorsalis pedis pulse on the affected side. Compartment syndrome may be present if the person develops pain on passive movement and paraesthesias. An oedematous knee may indicate a collection of blood in the knee joint. Ligament damage may be present if the person cannot move the knee and/or ankle.

If the fracture is closed, a closed reduction and casting are frequently performed. A long leg cast that allows for partial weight bearing is used. Partial weight bearing usually is prescribed by the doctor within 10 days of the fracture. A short leg cast will be applied in 3 to 4 weeks. If the fracture is open, either external fixation or ORIF will be performed. After surgery, a cast may be applied and weight bearing begins according to the doctor's orders, usually in about 6 weeks.

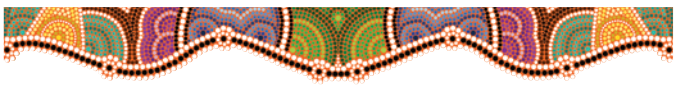
Nursing care is designed to increase comfort, monitor neurovascular status and prevent complications. The nurse instructs the person in cast care, on the use of assistive devices, how to perform neurovascular assessment and when to follow up with the doctor.

Fracture in the ankle and foot

The person with an ankle fracture presents with pain, limited ROM, haematoma, oedema and difficulty ambulating. Most ankle fractures are treated by closed reduction and casting. Open fractures are treated by surgical intervention and splinting.

The person with a foot fracture presents with similar symptoms; however, ROM of the ankle is not usually affected. Most foot fractures are non-displaced and treated with closed reduction and casting. More severe displaced foot fractures may require surgery and the placement of wires to maintain reduction of the fracture.

Nursing care focuses on increasing comfort, increasing mobility and educating the person. Analgesia is given for pain. The extremity should be elevated and ice can be applied. The person is taught cast care, neurovascular assessment and crutch walking.



Nursing care

In planning and implementing nursing care for the person with fractures, the nurse should consider the person's response to the traumatic experience. Although each person has individual needs, nursing care commonly focuses on the person's problems with pain, impaired physical mobility, impaired tissue perfusion and neurovascular compromise.

Health promotion

Trauma prevention can save lives. Many communities are educating people of all ages, from primary school students to older adults, in trauma prevention. Young adults face a high risk of sustaining trauma. They need to be taught the importance of safety equipment—such as automobile seat belts, bicycle and motorised vehicle helmets, shin guards, proper footwear, protective eyewear and hard hats—in preventing or decreasing the severity of injury from trauma. Older adults should have regular screenings for osteoporosis (with a bone density test), activity levels, cognitive and affective disorders, vision impairments and risk of falls. Older adults can reduce their risk of falling by increasing lower body strength and balance through regular physical activity and by asking their healthcare provider or pharmacist to review their medications. Educational programs about workplace and farm safety, including information about ergonomic principles, can also help prevent musculoskeletal injuries.

Having a regular exercise program and avoiding obesity are important factors in maintaining good bone health in all adults. An adequate intake of calcium is essential to ensure proper growth, development and maintenance of strong bones throughout life. It is important that women ensure good bone health prior to menopause, because the loss of oestrogen during and after menopause decreases calcium use and increases the risk of osteoporosis. Strong bones are formed by calcium intake and weight-bearing exercise, both of which are equally important in the postmenopausal woman.

Older people are at higher risk of musculoskeletal trauma due to falls. For these people, home assessments must be performed and potential hazards removed. Specific teaching topics for preventing falls in older adults are outlined in the 'Nursing care of the older adult' box below.

Assessment

Collect the following data through the health history and physical examination (see Chapter 37).

- *Health history:* age, history of traumatic event, history of chronic illnesses, history of prior musculoskeletal injuries, medications. (Ask the older adult specifically about anticoagulants and calcium supplements.)
- *Physical assessment:* pain with movement, pulses, oedema, skin colour and temperature, deformity, range of motion, touch. These assessments include the 5 Ps of neurovascular assessment, as follows, in both the initial assessment and ongoing focused assessments:
  1. *Pain*. Assess pain in the injured extremity by asking the person to grade it on a scale of 0 to 10, with 10 as the most severe pain.
  2. *Pulses*. Assess distal pulses, beginning with the unaffected extremity. Compare the quality of pulses in the affected extremity to those of the unaffected extremity.
  3. *Pallor*. Observe for pallor and skin colour in the injured extremity. Paleness and coolness may indicate arterial compromise, whereas warmth and a bluish tinge may indicate venous blood pooling.

NURSING CARE OF THE OLDER ADULT Teaching older adults to prevent falls

- Begin a regular exercise program; lack of exercise leads to weakness and an increased chance of falling. Exercises that improve balance and coordination (such as tai chi) are the most helpful.
- Make your home safer:
  - Remove any items in your pathway, including from stairs, to avoid tripping.
  - Remove small throw rugs or use double-sided tape to keep rugs from slipping.
  - Place frequently used items within easy reach to avoid use of a step-ladder.
  - Install grab bars next to your toilet and in the bath or shower.
  - Use non-slip mats in the bathtub and on shower floors.
  - Improve lighting, using lamp shades or frosted bulbs to reduce glare.
  - Install handrails and lights in all staircases.
  - Wear shoes that give good support and have thin, non-slip soles. Avoid wearing slippers and athletic shoes with deep treads.
- Ask your doctor to review your medications, including prescriptions and over-the-counter medications. Some medications, or a combination of medications, may cause dizziness or drowsiness, leading to falls.
- Have your vision checked by an eye doctor. Your glasses may no longer have the correct prescription or you may have developed an eye condition such as cataracts or glaucoma that limits your vision (ACI, 2014).

4 *Paralysis/Paresis*. Assess ability to move body parts distal to the fracture site. Inability to move indicates paralysis. Loss of muscle strength (weakness) when moving is paresis. A finding of limited range of motion may lead to early recognition of problems such as nerve damage and paralysis.
5 *Paraesthesia*. Ask the person if any change in sensation, such as burning, numbness, prickly feeling or stinging (all these are paraesthesias) has occurred.

Nursing diagnoses and interventions

Nursing care for people with fractures ranges from teaching for home care treatments provided in the emergency or urgent care department (such as manual reduction and cast application) to providing interventions to maintain health and decrease the risk of complications in people with complex or multiple fractures. Teaching is also necessary for caregivers of the older adult who is discharged home or to a long-term care or rehabilitation facility following a fractured hip. A nursing care plan is included below.

Acute pain

Pain is caused by soft tissue damage and is compounded by muscle spasms and swelling.

- Monitor vital signs. *Some analgesics decrease respiratory effort and blood pressure.*
- Ask the person to rate the pain on a scale of 0 to 10 (with 10 as the most severe pain) before and after any intervention. *This facilitates objective assessment of the effectiveness of the chosen pain relief strategy. Pain that increases in intensity or remains unrelieved with analgesics can indicate compartment syndrome.*
- For the person with a hip fracture, apply Buck's traction per doctor's orders. Keep the traction weights hanging freely. *Buck's traction immobilises the fracture and decreases pain and additional trauma.*
- Move the person gently and slowly. *Gentle moving helps prevent the development of severe muscle spasms.*

CONSIDERATION FOR PRACTICE
Do not let weights lie on the bed or the floor. The weights can be removed long enough to move the person up or down in bed to ensure freely hanging weights.

- Elevate the injured extremity above the level of the heart. *Elevating the extremity promotes venous return and decreases oedema, which decreases pain.*
- Encourage distraction or other non-invasive methods of pain relief, such as deep breathing and relaxation. *Distraction, deep breathing and relaxation help decrease the focus on the pain and may lessen the intensity of pain.*
- Administer pain medications as prescribed. For home care, explain the importance of taking pain medications before the pain is severe. *Analgesics alleviate pain by stimulating opiate receptor sites.*

CONSIDERATION FOR PRACTICE
In the case of fracture in an extremity, supporting the extremity above and below the fracture can also decrease pain and muscle spasms.

Risk of peripheral neurovascular dysfunction

In the person with a fracture, compartment syndrome or deep venous thrombosis can impair circulation and, in turn, tissue perfusion.

- Assess the 5 Ps every 1 to 2 hours. Report abnormal findings immediately. *Unrelenting pain, pallor, diminished distal pulses, paraesthesias and paresis are strong indicators of compartment syndrome.*

NURSING CARE PLAN A person with a hip fracture

Barbara Wallace, aged 74, has a history of osteoporosis. She is a widow and lives alone in a two-storey terrace house. Mrs Wallace is retired and depends on a pension and social security for her income. She takes pride in making all her own meals from scratch.

While walking to the shopping centre one day, Mrs Wallace trips and falls up the gutter and fractures her left hip. She is transported by ambulance to the nearest hospital emergency department.

ASSESSMENT

During the initial assessment at the ED, abnormal findings are that Mrs Wallace's left leg is shorter than her right leg and is externally rotated. Distal pulses are present and bilaterally strong; both legs are warm. Mrs Wallace complains of severe pain in her hip but states that no numbness or burning is present. She is able to wiggle the toes on her left leg and has full movement of her right leg. Initial vital signs are as follows: T 36.6°C, P 100 and regular, R 18, BP 120/58. Diagnostic tests include FBC, blood chemistry, cross-match and x-ray studies of the left hip and pelvis. The FBC reveals a haemoglobin of 11.0 g/dL and a normal WBC count. Blood chemistry findings are within normal limits. The x-ray reveals a fracture of the left femoral neck. Mrs Wallace is admitted to the hospital. She is given a femoral block and ordered regular analgesia. An open reduction and internal fixation (ORIF) is planned for the following day.

DIAGNOSES

- *Acute pain* related to fractured left femoral neck and muscle spasms.
- *Impaired physical mobility* related to bed rest and fractured left femoral neck.
- *Risk of ineffective tissue perfusion* related to unstable bones and swelling.
- *Risk of disturbed tactile sensory perception* related to the risk of nerve impairment.

PLANNING

- Identify and manage pain.
- Monitor changes in neurovascular status.
- Monitor postoperative complications.

(continued)

NURSING CARE PLAN A person with a hip fracture (continued)

Expected outcomes

- Mrs Wallace will verbalise a decrease in pain.
- Improved comfort for Mrs Wallace.
- Maintain normal neurovascular status.
- Prevention of postoperative complications including DVT, pressure areas, atelectasis, urinary stasis and constipation.

IMPLEMENTATION

- Assess pain on a scale of 0 to 10 before and after implementing measures to reduce pain.
- Administer narcotics per the doctor's order.
- Position Mrs Wallace so as to reduce pain.
- Perform neurovascular assessment every 2 to 4 hours and document findings.
- Encourage deep breathing and relaxation techniques.
- Demonstrate postoperative exercises.
- Teach the purpose of and the procedure for performing isometric and flexion/extension exercises.

EVALUATION

Three days after surgery, Mrs Wallace is out of bed and in a chair. She verbalises a decrease in pain. There have been no abnormal neurovascular assessments. She is able to independently perform isometric and flexion/extension exercises in both lower extremities. Safe discharge planning includes explaining the benefits to Mrs Wallace of going to a rehabilitation facility before going directly home, and referrals for home care. A community nurse will visit and the discharge planner at the hospital has organised review by the physiotherapist and the occupational therapist to order a trapeze for her bed, an elevated toilet seat, an elevated cushion for her chair and a walker.

CRITICAL THINKING IN THE NURSING PROCESS

1. What factors placed Mrs Wallace at risk of a hip fracture?
2. Identify the preoperative management and how you can explain this to Mrs Wallace. Explain the reason for the femoral block and why she needs to remain in bed.
3. Describe how each of the following, if manifested by Mrs Wallace, would increase her risk of postoperative complications: urinary incontinence; weight more than 20% under normal for her height; chronic constipation. Which nursing diagnoses and interventions would you include in her plan of care to decrease the risk?

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will take into your future practice.
2. Which education requirements would the nurse need to implement prior to Mrs Wallace being discharged?

CONSIDERATION FOR PRACTICE

Pulses may remain strong, even in the presence of compartment syndrome.

- Assess nail beds for capillary refill. If nails are too thick or discoloured, assess the skin around the nail. *Delayed capillary refill may indicate decreased tissue perfusion.*

CONSIDERATION FOR PRACTICE

It may not be possible to accurately assess capillary refill in older adults, who often have thickened, discoloured nails. If so, test nearby skin.

- Monitor the extremity for oedema and swelling. *Excessive swelling and haematoma formation can compromise circulation.*
- Assess for deep, throbbing, unrelenting pain. *Pain that is not relieved by analgesics may indicate neurovascular compromise.*
- Monitor the tightness of the cast. *Oedema can cause the cast to become tight; a tight-fitting cast may lead to compartment syndrome or paralysis.*
- If cast is tight, be prepared to assist the doctor with bivalving (see Figure 38.10). *Bivalving, the process of splitting the cast down both sides, alleviates pressure on the injured extremity.*
- If compartment syndrome is suspected, assist the doctor in measuring compartment pressure. Normal compartment pressure is 10 to 20 mmHg. *Compartment pressure greater than 30 mmHg indicates compartment syndrome.*
- Elevate the injured extremity above the level of the heart. *Elevating the extremity increases venous return and decreases oedema.*
- Administer anticoagulant per doctor's order. *Prophylactic anticoagulation decreases the risk of clot formation.*

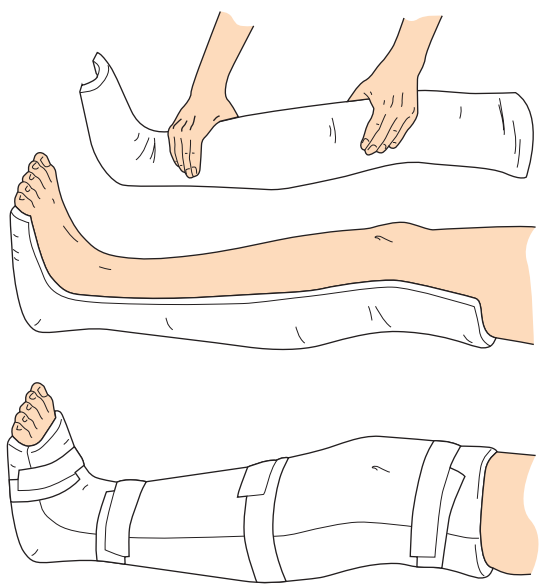


FIGURE 38.10 ■ Bivalving is the process of splitting the cast down both sides to alleviate pressure on or allow visualisation of the extremity

Risk of infection

The person who undergoes surgical repair will have a postoperative wound. Any break in skin integrity must be monitored for infection. Wound healing in an orthopaedic person is affected by the cause of the wound, as well as by the therapies used to repair musculoskeletal structures. It is important for nurses to understand normal wound healing processes; characteristics of musculoskeletal wounds, contamination and drainage; and potential complications, in order to plan for and implement appropriate interventions (Royal Children's Hospital Melbourne, 2015b).

- For people with skeletal pins, follow established guidelines for skeletal pin site care, as outlined in the 'Translation to practice' box below. *Pins or wires attached to traction, casts or external fixators stabilise a segment of bone so that optimal healing can occur. However, pin infections of varying severity do occur* (Holmes & Brown, 2005).
- Monitor vital signs and lab reports of WBCs. *Increases in pulse rate, respiratory rate, temperature and WBCs may indicate infection.*
- Use sterile technique for dressing changes. *The initial postoperative dressing will be changed by the surgeon. The nurse must change all subsequent dressings without introducing organisms into the operative site.*
- Assess the wound for size, colour and the presence of any drainage. *Redness, swelling and purulent drainage indicate infection.*
- Administer antibiotics per doctor's orders. Prophylactic antibiotic administration inhibits bacterial reproduction and thereby helps prevent skin flora from entering the wound. *In the case of 'dirty wounds', such as those occurring from vehicular crashes, antibiotics are routinely administered.*

Impaired physical mobility

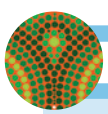
The person who has experienced a fracture requires immobilisation of the fractured bone(s). Immobilisation alters normal gait and mobility. The person will need to use assistive devices such as crutches, canes, slings or walkers.

- Teach or assist the person with ROM exercises for the unaffected limbs. *ROM exercises help prevent muscle atrophy and maintain strength and joint function. Flexion and extension exercises prevent the development of foot drop, wrist drop or frozen joints.*
- Teach isometric exercises and encourage the person to perform them every 4 hours. *Isometric exercises help prevent muscle atrophy and force synovial fluid and nutrients into the cartilage.*
- Encourage ambulation when able; provide assistance as necessary. *Ambulation maintains and improves circulation, helps prevent muscle atrophy and helps maintain bowel function.*
- Teach and observe the person's use of assistive devices (such as canes, crutches, walkers, slings) in conjunction with the physical therapist. *Proper use of devices is necessary for safe ambulation and helps prevent the loss of joint function secondary to complications and falls.*
- Turn the person on bed rest every 2 hours. If the person is in traction, teach the person to shift his or her weight every hour. *Turning and shifting weight increase circulation and help prevent skin breakdown.*

Risk of disturbed tactile sensory perception

The person who has sustained a fracture is at risk of nerve injury from the initial trauma, as well as from complications such as compartment syndrome.

- Assess the ability to differentiate between sharp and dull touch and the presence of paraesthesias and paralysis every



TRANSLATION TO PRACTICE Evidence-based practice for the person with skeletal pins

Clinical guidelines for specific person care interventions, such as skeletal pin care, should be based on research in order to provide the most appropriate evidence-based practice. Management of people with an external fixation device will depend on the surgeon. Each surgeon has their own protocol for pin care cleaning (Lethaby, Temple & Santy-Tomlinson, 2013).

- Pins located in areas with considerable soft tissue should be considered at greater risk of infection.
- People and their families should be taught pin site care before discharge from the hospital. They should be required to demonstrate whatever care needs to be done and should be provided with written instructions that include signs and symptoms of infection—such as exudate, erythema, tenderness and pain—which may require removal of the pins (Holmes & Brown, 2005).

IMPLICATIONS FOR NURSING

Evaluation of the literature by the systematic review of Lethaby et al. (2013) found that the preference of the surgeon determined the care required for external pin sites.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. List the factors that may increase the risk of infection of skeletal pin sites. Which nursing interventions may be used to reduce this risk?
2. You are caring for a person with skeletal pins for external fixation of a fracture of bones of a lower extremity. There is dried yellow drainage around the pin site. Based on clinical decision making without research to support your actions, would you remove the crusts? Why or why not?
3. You are teaching a person how to undertake pin care at home. Make a list of manifestations of infection the person may experience. What would you recommend if any of these manifestations occur?

Source: Data from Lethaby, A., Temple, J. & Santy-Tomlinson, J. (2013). Pin site care for preventing infections associated with external bone fixators and pins. *Cochrane Database of Systematic Reviews*, Issue 4.

1 to 2 hours. *Paraesthesias develop as a result of pressure on nerves and may indicate compartment syndrome.*

CONSIDERATION FOR PRACTICE

Paralysis is a late sign of nerve entrapment and requires that the healthcare provider be notified immediately.

- Elevate the injured extremity above the level of the heart. *Elevating the extremity decreases swelling and the risk of compartment syndrome and nerve entrapment.*
- Check the cast for fit. *A tightly fitting cast can decrease blood flow to distal tissues, compress nerves and cause compartment syndrome.*
- Support the injured extremity above and below the fracture site when moving the person. *Such support helps prevent displacement of bony fragments and decreases the risk of further nerve damage.*

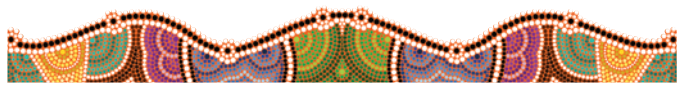
Community-based care

Person and family teaching focuses on individualised needs. The type of fracture and its location determine how much teaching the person and family will require. For example, a person who has a simple non-displaced tibial fracture may need to be taught only cast care and crutch walking. An older person who has sustained a hip fracture and requires surgical intervention, by contrast, has a wider array of teaching needs, including the use of an abduction pillow, proper bending and proper sitting. Address the following topics for home care of the person who has fractured a hip:

- Encourage independence in ADLs:
  - Explain that the person should sit only on high chairs to prevent excess flexion of the hip; a high toilet seat can be added to a regular toilet seat.
  - Encourage the person and family to equip the shower with a rail to aid stability and prevent falls.
  - If a walker is needed, teach the person its proper use: do not carry the walker, but lift it, advance it and then take two steps, or use a rolling walker.
  - If a walking stick is needed, instruct the person to use it on the affected side.
  - Stress the importance of well-balanced meals and explain all prescribed medications.

People who have experienced a fracture or have had orthopaedic surgery often have a cast and require an extended period of immobilisation or limited activities. Address the following topics for home care:

- Do not try to scratch under a cast with a sharp object.
- Do not get a plaster cast wet.
- Follow the doctor's order for weight bearing.
- Physiotherapy departments or offices can often evaluate the home environment for safety and suggest modifications as needed. Physiotherapists also teach crutch walking, limited weight bearing, transferring and other activities.
- Home care agencies can teach wound care and provide ongoing monitoring of wound healing.
- Local medical equipment and supply sources rent or sell durable equipment such as crutches, walkers, wheelchairs, overhead trapeze units, shower chairs, elevated toilet seats, grab bars and bedside commodes. Slings or braces may be purchased through medical equipment dealers.
- Local pharmacies are good resources for dressing supplies such as antiseptic solutions or ointments, dressings and tape.
- Fitness equipment suppliers may be able to provide rehabilitation equipment such as hand or ankle weights for strengthening exercises.



THE PERSON WITH AN AMPUTATION

An **amputation** is the partial or total removal of an extremity. Amputation may be the result of an acute process, such as a traumatic event, or a chronic condition, such as peripheral vascular disease or diabetes mellitus. Regardless of the cause, an amputation is devastating to the person.

The loss of all or part of an extremity has a significant physical and psychosocial effect on the person and family. Adaptation may take a long time and require much effort. Interprofessional healthcare is always important, but is especially necessary to meet the person's physical, spiritual, cultural and emotional needs after an unexpected or planned amputation.

Causes of amputation

Peripheral vascular disease (PVD) is the major cause of amputation of the lower extremities (see Chapter 31). Common risk factors for the development of PVD include hypertension, diabetes, smoking and hyperlipidaemia. Peripheral neuropathy also places the person with diabetes at risk of amputation. In peripheral neuropathy, loss of sensation frequently leads to unrecognised injury and infection. Untreated infection may lead to gangrene and the need for amputation. These risks are discussed in Chapter 19.

The incidence of traumatic amputations is highest in young men. Most amputations in this group result from motor vehicle crashes or accidents involving machinery at work. The person presents to the trauma centre with an injury that may be life threatening; significant loss of blood and tissue may have already occurred and shock may develop. (See Chapter 10 for a discussion of shock and trauma.) Other traumatic events that may necessitate an amputation are frostbite, burns or electrocution.

Amputations result from or are necessitated by interruption in blood flow, either acute or chronic. In acute trauma situations, the limb is partially or completely severed and tissue death ensues. However, replantation of fingers, small body parts and entire limbs has been successful. In chronic disease processes, circulation is impaired, venous pooling begins, proteins leak into the interstitium and oedema develops. Oedema increases the risk of injury and further decreases circulation. Stasis ulcers develop and readily become infected because impaired healing and altered immune processes allow bacteria to proliferate. The presence of progressive

FAST FACTS

- Males are at higher risk of amputations and are three times more likely to suffer an amputation than females.
- The middle and older age groups have the highest incidence of amputation. This is generally due to peripheral vascular disease, atherosclerosis and changes due to diabetes mellitus. In Australia, the most common causes of lower extremity amputations are disease, trauma and thermal injuries, congenital or birth defects, and tumours.
- Upper extremity amputation is usually due to trauma or birth defect.
- Demographics show that amputees represent 1 per 1000 individuals across Australia, and subsequently there are in excess of 20 000 amputees living in Australia.

Source: Australian Physiotherapists in Amputee Rehabilitation (2015). Retrieved from www.austpar.com/index.html.

infection further compromises circulation and ultimately leads to gangrene (tissue death), which requires amputation.

Levels of amputation

The level of amputation is determined by local and systemic factors. Local factors include ischaemia and gangrene; systemic factors include cardiovascular status, renal function and severity of diabetes mellitus. The goals are to alleviate symptoms, to maintain healthy tissue and to increase functional outcome. When possible, the joints are preserved because they allow greater function of the extremity. Figure 38.11 illustrates common sites of amputation.

Types of amputation

Amputations may be open (*guillotine*) or closed (*flap*). Open amputations are performed when infection is present. The wound is not closed but remains open to drain. When infection is no longer present, surgery is performed to close the wound. In closed amputations, the wound is closed with a flap of skin that is sutured in place over the stump. Terms used to refer to amputations are defined in Table 38.3.

Amputation site healing

For the prosthesis to fit well, the amputation site must heal properly. To promote healing, a rigid or compression dressing is applied to prevent infection and minimise oedema. A compression bandage is usually applied immediately after surgery to support the soft tissues. The bandage may be elastic and is applied to the residual limb. The compression bandage is worn at all times, except for showering and physiotherapy. Bandages are reapplied several times a day to ensure a snug fit, but not to interfere with circulation.

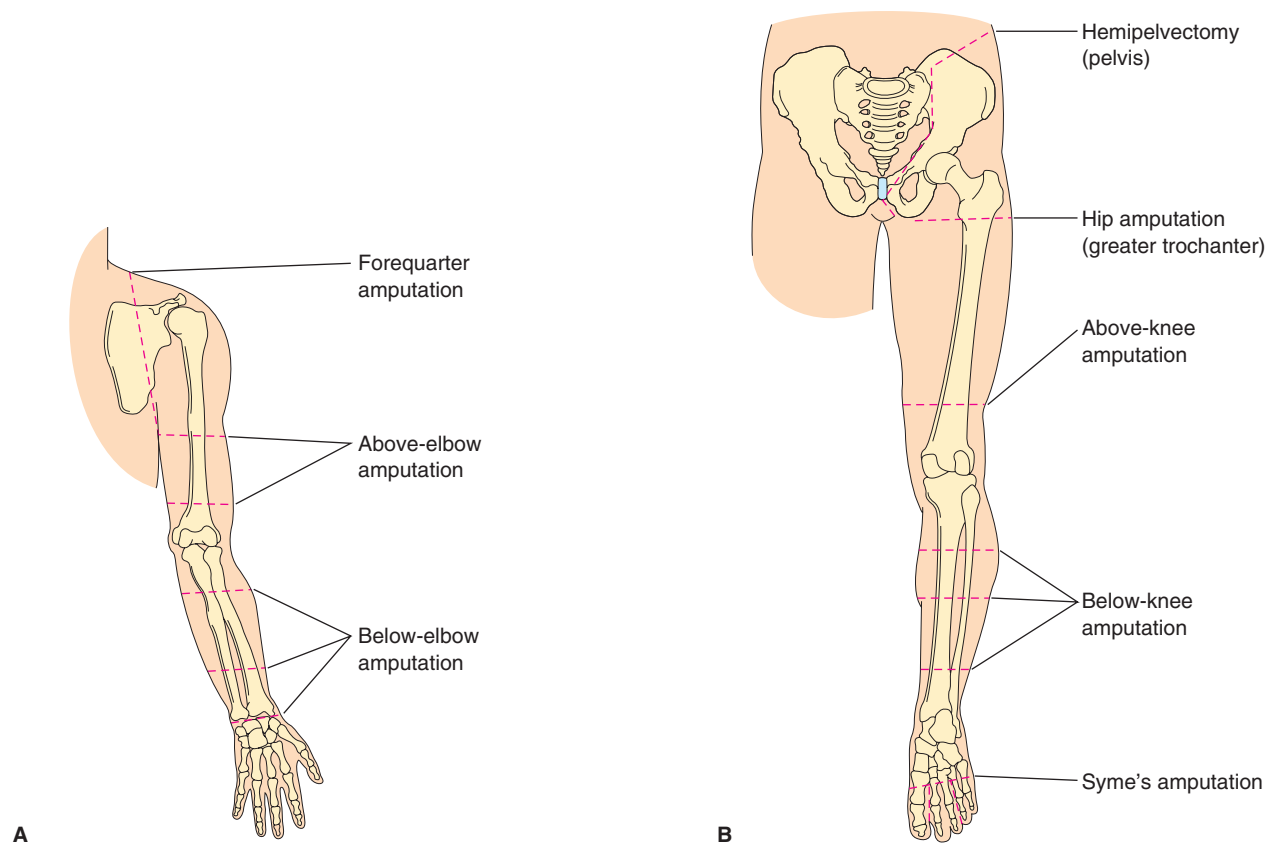


FIGURE 38.11 ■ Common sites of amputation. *A*, The upper extremities. *B*, The lower extremities. The surgeon determines the level of amputation based on blood supply and tissue condition

TABLE 38.3 Amputation terms

TERM	MEANING
Arm	Amputation of a portion of the arm, either above or below the elbow
Disarticulation	Amputation through a joint
Forequarter	Removal of the entire arm and disarticulation of the shoulder
Closed (flap)	Amputation in which a flap of skin is formed to cover the end of the wound
Open (guillotine)	Perpendicular cutting of the extremity in which the wound is left open; used when infection is present
Leg	Amputation below the knee (BK)
Thigh	Amputation above the knee (AK)
Finger or toe	Amputation of one or all of the fingers or toes
Syme	Modified disarticulation of the ankle
Foot	Amputation of part of the foot and toes

Once the wound has healed, the compression bandage need only be worn when the prosthesis is not worn. The bandage is applied from the distal to the proximal extremity (see Figure 38.12).

Complications

Complications that may occur after an amputation include infection, delayed healing, chronic stump pain and phantom pain, and contractures.

Infection

Generally, the person who suffers a traumatic amputation has a greater risk of infection than the person who has a planned amputation. However, even planned amputations carry a risk of infection. The person who is older, has diabetes mellitus or suffers peripheral neurovascular compromise is at a particularly high risk of infection. Infection may present itself locally or systemically. Local manifestations of infection include drainage, odour, redness and increased discomfort at the suture line. Systemic manifestations include fever, an increased heart rate, a decrease in blood pressure, chills and positive wound or blood cultures.

Delayed healing

If infection is present, or if the circulation remains compromised, delayed healing (occurring at a slower rate than expected) will result. In older people, other pre-existing conditions can increase the risk of delayed healing. In people of any age, electrolyte imbalances can contribute to delayed healing processes, as can a diet that lacks the proper nutrients to meet the body's increased metabolic demands during healing. Smoking compromises healing by causing vasoconstriction and decreasing blood flow to the stump. Deep venous thrombosis and compromised venous return, which may result from prolonged immobilisation, are other potential factors. Decreased cardiac output decreases blood flow and thus also delays healing.

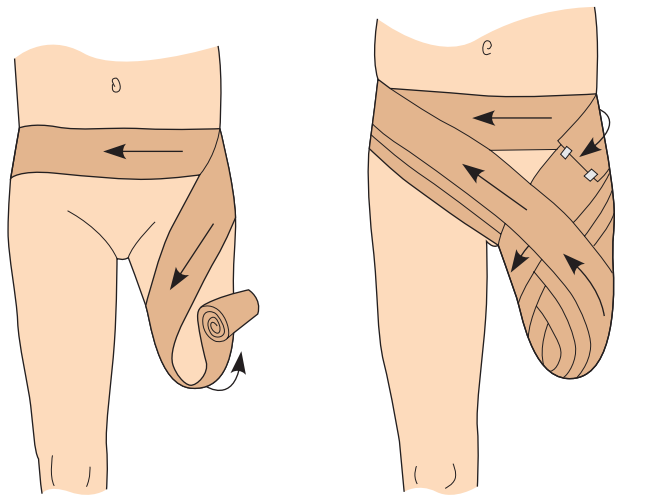


FIGURE 38.12 ■ Stump dressings increase venous return, decrease oedema and help shape the stump for a prosthesis. With an above-knee amputation, a figure-eight bandage is started by bringing the bandage down over the stump and back up around the hips

Chronic stump pain and phantom pain

Chronic stump pain is the result of neuroma formation, causing severe burning pain. Interventions to relieve this pain include medications, nerve blocks, transcutaneous electrical nerve stimulation (TENS) and surgical stump reconstruction. Phantom limb pain is not the same as phantom limb sensation. A majority of amputees experience phantom limb sensation (sensations such as tingling, numbness, cramping or itching in the phantom foot or hand) early in the postoperative period. It is often self-limiting, but may last for decades in some people. When phantom limb sensation is painful, it is referred to as **phantom limb pain**. Although various theories have been proposed, the exact cause of this experience is unknown. Treatments include pain management, TENS and a variety of surgical procedures. The management of phantom limb pain is difficult for both people and healthcare professionals. People with phantom limb pain often benefit from referral to a pain clinic for a comprehensive pain management program.

Contractures

A contracture is an abnormal flexion and fixation of a joint caused by muscle atrophy and shortening. Contracture of the joint above the amputation is a common complication. The person needs to be taught to extend the joint. The person with an above-the-knee amputation should lie prone for periods throughout the day. The person with a below-the-knee amputation should elevate the stump, keeping the knee extended. The same principles apply to the upper extremities. All joints should receive either active or passive ROM exercises every 2 to 4 hours. A trapeze frame should be added to the bed to encourage the person to change position every 2 hours. The person who has an upper extremity amputation should exercise both shoulders. Postural exercises can help prevent the person from hunching over secondary to the loss of weight on the affected side. The person with an above-the-knee amputation should not sit for prolonged periods of time; prolonged sitting can lead to hip contracture.

INTERPROFESSIONAL CARE

Interprofessional care is essential for the person who has sustained an amputation. Physiotherapy and occupational retraining are necessary and the person may also benefit from the presence of clergy. The entire healthcare team must view both the positive and the negative effects of amputation; that is, they must see amputation as a means to increase the person's independence and to relieve symptoms. The person should be able to become familiar with the members of the healthcare team and their roles; this allows the person greater control over their care and rehabilitation and promotes independence.

Diagnosis

Preoperatively, the person has routine laboratory and diagnostic tests (see Chapter 3 for care of the person having surgery). Preoperative tests, including Doppler flowmetry, segmental blood pressure determination, transcutaneous partial pressure oxygen readings and angiography, are performed to assess the circulation present in the limb at different levels and to determine the level of viable tissue. Postoperative tests include FBC to monitor for haemorrhage, WBC count to monitor for infection, blood chemistries to evaluate electrolytes and fluid balance, and a vascular Doppler ultrasonography if a DVT is suspected.

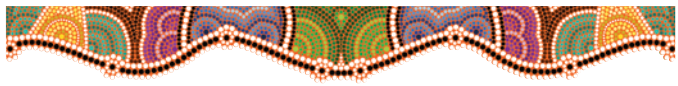
Medications

The person receives medications preoperatively, intraoperatively and postoperatively. Preoperatively, the doctor may prescribe intravenous antibiotics. Intraoperatively, anaesthetic agents are administered. It also may be necessary to administer agents to control blood pressure during the surgery. Postoperatively, the person resumes any routinely prescribed medications and in addition may receive antibiotics and analgesics. Steroids may be administered to decrease swelling. A protein pump inhibitor (PPI) antagonist may also be ordered to decrease the risk of peptic ulcer formation. Stool softeners may be administered to prevent constipation.

Prosthesis

The type of prosthesis selected for the person with an amputation depends on the level of the amputation as well as the person's occupation and lifestyle. Each prosthesis is based on a detailed prosthetic prescription and is custom made for the person based on the specific characteristics of the stump. Most are made of plastic and foam materials. Many factors influence the person's use of the prosthesis, including the status of the remaining limb, cognitive status, cardiovascular status, preoperative activity level and motivation to use the prosthesis.

People with a lower extremity amputation are often fitted with early walking aids. Pneumatic devices that fit over the stump are used in the immediate postoperative period to allow early ambulation, decreased postoperative swelling and improved morale. People may begin weight bearing as soon as 2 weeks after surgery. People with upper extremity amputations may be fitted for a prosthesis immediately after surgery. Rehabilitation of the person with an amputation is a team effort, involving the person, nurse, doctor, physical therapist, occupational therapist, social worker, prosthetist and vocational counsellor.



Nursing care

Health promotion

The goals of health promotion activities focus on preventing the progression of chronic diseases such as peripheral vascular disease and diabetes mellitus and on safety. People with PVD from any cause need education about foot care and early recognition of decreased circulation. Education within both urban and rural populations should provide knowledge about working safely with lawn care equipment as well as farm and occupational machinery.

In addition, it is important that the public know what to do if a traumatic amputation occurs in the home, community or workplace. The following guidelines may help preserve the amputated part until it can be surgically reattached:

- Keep the person in a prone position with the legs elevated.
- Apply firm pressure to the bleeding area, using a towel or article of clothing.
- Wrap the amputated part in a clean cloth. If possible, soak the cloth in saline (such as contact lens solution).
- Put the amputated part in a plastic bag and put the bag on ice. Do not let the amputated part come into direct contact with the ice or water.
- Send the amputated part to the emergency department with the injured person and be sure the emergency personnel know what it is.

Assessment

Collect the following data through the health history and physical examination. Further focused assessments are described in the following nursing interventions sections.

- *Health history:* mechanism of injury, current and past health problems, pain, occupation, ADLs, changes in sensation in the feet, cultural and/or religious guidelines for handling the amputated part.
- *Physical examination:* bilateral neurovascular status of the extremities, bilateral capillary refill time, skin over the lower extremities (discolouration, oedema, ulcerations, hair, gangrene).

Nursing diagnoses and interventions

The goals of nursing care for a person with an amputation are to relieve pain, promote healing, prevent complications, support the person and family during the process of grieving and adaptation to alterations in body image, and restore mobility. Care is individualised and the circumstances that led to the amputation (e.g. traumatic injury or disease) must also be addressed. (See the accompanying nursing care plan.) Applying rehabilitation principles to nursing care is also important.

Acute pain

Pain from the surgical procedure can be compounded by muscle spasms, swelling and phantom limb pain.

- Ask the person to rate the pain on a scale of 0 to 10 (with 10 as the most severe pain) before and after any intervention. *This facilitates objective assessment of the effectiveness of the chosen pain relief strategy. Pain that increases in intensity or remains unrelieved with analgesics can indicate compartment syndrome.*
- Splint and support the injured area. *Splinting prevents additional injury by immobilising the stump and decreasing oedema while moulding the stump for a good prosthetic fit.*
- Unless contraindicated, elevate the stump on a pillow for the first 24 hours after surgery. *Elevating the stump promotes venous return and decreases oedema, which will decrease pain.*
- Move and turn the person gently and slowly. *Gentle moving and turning prevents the development of severe muscle spasms.*
- Administer pain medications as prescribed. A PCA pump may be ordered by the doctor. *Analgesics alleviate pain by stimulating opiate receptor sites. PCA pumps increase person control over and allow early relief of pain before it intensifies.*
- Encourage deep breathing and relaxation exercises. *These techniques increase the effectiveness of analgesics and modify the pain experience.*
- Reposition the person every 2 hours; turn from side to side and onto abdomen. *Repositioning alleviates pressure from one area and distributes it throughout the body, and helps prevent cramping of muscles.*

CONSIDERATION FOR PRACTICE

Lying prone prevents hip contracture.

Risk of infection

The person who has an amputation is at risk of wound infection. Early recognition of infection can lead to early treatment and prevent wound dehiscence.

- Assess the wound for redness, drainage, temperature, oedema and suture line approximation. *Redness is normal in the immediate postoperative period; if it persists, however, it can indicate infection. A hot area that is palpated over the incision or increased drainage may also indicate infection.*
- Take the person's temperature every 4 hours. *Increased body temperature may indicate infection.*
- Monitor WBC count. *The WBC count rises in the presence of infection.*
- Use aseptic technique to change the wound dressing. *Aseptic technique prevents contamination of the wound with bacteria.*
- Administer antibiotics as ordered. *Antibiotics inhibit bacterial cell replication and help prevent or eradicate infection.*
- Teach the person stump-wrapping techniques. *Correctly wrapping the stump from the distal to the proximal extremity increases venous return and prevents pooling of fluid, thereby reducing the chance of infection.*

Risk of impaired skin integrity

Stump care is essential, not only in the postoperative healing period but also throughout life with a prosthesis. A variety of skin problems may be caused by a prosthesis, including epidermoid cysts, abrasions, blisters and hair follicle infections. The person must be taught stump care prior to discharge.

- Each day, preferably at night, wash the stump with soap and warm water, and dry thoroughly. *Inspect the stump for redness, irritation or abrasions. It is essential to maintain intact skin to ensure successful use of the prosthesis.*
- Massage the end of the stump, beginning 3 weeks after surgery. *Massage helps desensitise the remaining part of the limb and prevents scar tissue formation. If the skin adheres to the underlying tissue, it will tear when stressed from wearing a prosthesis.*
- Expose any open areas of skin on the remaining part of the limb for 1 hour four times a day. *Air exposure promotes healing.*
- Change stump socks and elastic wraps each day. Wash these in mild soap and water and allow to completely dry before using again. *Stump socks and elastic wraps must be kept clean and dry to prevent skin breakdown.*

Risk of dysfunctional grieving

The person who has lost an extremity is at risk of dysfunctional grieving. Denial of the need for surgery and the inability to discuss feelings compound this risk.

- Encourage verbalisation of feelings, using open-ended questions. *Asking open-ended questions allows the person to discuss feelings and communicates the listener's willingness to listen.*
- Actively listen and maintain eye contact. *Active listening and eye contact communicate respect for what the person is expressing.*
- Reflect on the person's feelings. *Reflection statements such as 'You seem angry' allow the person to recognise feelings and perhaps develop a plan for resolution.*

NURSING CARE PLAN A person with a below-the-knee amputation

John Rocke is a 45-year-old divorcee with no children. He has a history of type 2 diabetes mellitus and poor control of blood glucose levels. Mr Rocke is unemployed and currently receives unemployment benefits. He lives alone in a second-floor unit. Mr Rocke had developed gangrene in the foot and failed to seek prompt medical attention; as a result, a left below-the-knee amputation was necessary.

Mr Rocke is in his second postoperative day and his vital signs are stable. The stump is splinted and has a soft dressing. The wound is approximating well without signs of infection. He has not performed ROM exercises or turning since his surgery, complaining of severe pain. When the nurse goes into the room, he yells, 'Get out! I don't want anyone to see me like this.' No one has visited him since

NURSING CARE PLAN A person with a below-the-knee amputation (continued)

his hospitalisation. He is tolerating a diabetic diet and is using a urinal independently. He has an order for morphine, 5 mg IM/SC every 4 hours prn for pain and cephazolin, 1 g IV every 8 hours. He is on blood glucose coverage with regular insulin subcutaneously.

ASSESSMENT

Jane Simmons, RN, has just come on duty. She notes that Mr Rocke is upset and angry. He will not let anyone enter the room to give him medication or assess his vital signs.

DIAGNOSES

- *Disturbed body image* related to amputation of left lower leg.
- *Dysfunctional grieving* related to anger and loss of left lower leg.
- *Situational low self-esteem* related to appearance.
- *Risk of injury from infection and contractures* related to refusal of care.
- *Acute pain* related to surgery.

PLANNING

- Assess and manage Mr Rocke's pain related to the amputation.
- Ask a social worker to consult with Mr Rocke. Utilising a social worker may assist the person to discuss his current feelings about the amputation.
- Manage risk of contractures and infection.

Expected outcomes

- Verbalise his feelings about the amputation.
- Able to control his own analgesia and be pain free.
- Be allowed to control his pain with a PCA pump.
- Verbalise a decrease in pain.
- Verbalise the importance of turning.
- Turn every 2 hours.
- Allow the staff to monitor his vital signs and administer medications.

IMPLEMENTATION

- Actively listen to the person.
- Offer to arrange a visit with a fellow amputee.
- Assess pain and effectiveness of analgesia.
- Ask the doctor if the person can be placed on a PCA pump.
- Teach the person the importance of turning every 2 hours to prevent contractures.
- Encourage turning and lying prone.
- Teach the importance of antibiotics in preventing and treating infection.

EVALUATION

One week after his surgery, Mr Rocke is actively participating in his care. He has apologised for his behaviour and has explained to Ms Simmons that he was angry about the loss of his leg. He states, 'I thought I knew what to expect, but I didn't.'

CRITICAL THINKING IN THE NURSING PROCESS

1. Once Mr Rocke is ready to assist with his stump care, how would you proceed? Would you give him full responsibility for care and dressings, or would you gradually increase his participation? Why?
2. Which factors in Mr Rocke's home environment and medical history may make self-care more difficult? Do you expect Mr Rocke to follow up on care after his discharge? Why or why not?
3. Mr Rocke states, 'Why should I exercise this leg—it was already cut off!' How would you respond? What is the purpose of exercising the stump?

REFLECTION ON THE NURSING PROCESS

1. Identify the issues regarding the care of Mr Rocke that will be able to assist you with your future nursing care.
2. Looking back at the care you have given Mr Rocke, identify any issues that you feel that you could have seen earlier to improve Mr Rocke's outcome.

- Allow the person to have unlimited visiting hours, if possible. *Unlimited visiting hours allow increased social support.*
- If desired by the person, provide spiritual support by encouraging activities such as visits from a spiritual leader, prayer and meditation. *These activities often provide support during the grieving process.*

Disturbed body image

Although amputation is a reconstructive surgery, the person's body image will be disturbed. Risk of body image disturbance is higher in young people, in whom body image is a particularly important component of self-image.

- Encourage verbalisation of feelings. *This allows the person to communicate concerns and fears and lets the person know the nurse is willing to listen.*
- Allow the person to wear clothing from home. *Familiar clothing provides emotional comfort and helps the person retain a sense of his or her own identity.*
- Encourage the person to look at the stump. *Looking at and touching the stump helps the person face their fear of the unknown and move from denial to acceptance.*
- Encourage the person to care for the stump. *Active participation in care increases self-esteem and independence.*
- Offer to have a fellow amputee visit the person. *A support person who has experienced the same change gives the person the hope that they too can regain independence.*
- Encourage active participation in rehabilitation. *Active participation in rehabilitation increases independence and mobility.*

Impaired physical mobility

If time allows, the person should begin strengthening muscles preoperatively. If the amputation is the result of an emergency, exercises begin within 24 to 48 hours of surgery. The return of independent mobility boosts self-esteem and promotes adaptation to amputation.

- Perform ROM exercises on all joints. *ROM exercises help prevent the development of joint contractures that limit mobility.*
- Maintain postoperative stump shrinkage devices. These may be elastic bandages, shrinker socks, an elastic stockinette or a rigid plaster cast. *In order for the prosthetic limb to fit properly and as the wound heals, therapists will*

work with the person to wrap the stump with elastic wraps or stockings that minimise swelling and shape the stump for the best fit possible into a prosthetic limb.

- Turn and reposition the person every 2 hours. The person with a lower extremity amputation should lie prone every 4 hours, for up to 2 hours. *Repositioning increases blood flow to muscles, forces synovial fluid into joints and helps prevent contractures.*
- Reinforce teaching by the physical therapist in crutch walking or the use of assistive devices. *These devices increase mobility by balancing the person and facilitating ambulation.*
- Encourage active participation in physical therapy. *Physical therapy will fatigue the person in the early stage of healing. Encouragement may increase the person's participation in the physical therapy regimen and thereby increase activity tolerance.*

Community-based care

Person and family teaching includes stump care, prosthesis fitting and care, medications, assistive devices, exercises, rehabilitation, counselling, support services and follow-up appointments. The depth of teaching depends on the cause and site of the amputation and the needs of the person. See the 'Meeting individualised needs' box below.

Holistic nursing care is especially important for the older person with an amputation. The normal ageing process decreases renal and liver function; hence, medications have longer half lives. Altered circulation prolongs wound healing, and slowing of reflexes and alterations in gait may disrupt balance. A walker may be more appropriate than crutches because older people have less strength in the upper extremities. Safety issues, such as decreasing the risk of recurrent falls, must be addressed. The nurse should also assess the person's need for in-home assistance and make appropriate referrals to visiting nurses and home health aides.

MEETING INDIVIDUALISED NEEDS The person with an amputation

Amputation of a limb has significant long-term consequences for the person. The person will grieve the loss of a body part and must adjust to a new self-image. The person's ability to perform normal ADLs and to maintain their usual family and social roles may be significantly affected, at least initially. Depending on the person's occupation, job performance may be affected, necessitating a change of career.

The nurse may be responsible for involving multiple members of the healthcare team in the person's care and rehabilitation, and for coordinating their activities. Following an amputation, the person may need the services of any or all of the following:

- social services to help with rehabilitative and financial arrangements
- physiotherapists to teach ambulation techniques and to provide deep heat or massage
- occupational therapists to assist the person in developing adaptive techniques to deal with the loss of a limb
- prosthetists to develop a prosthesis for the missing limb that will meet the person's needs for ADLs and other activities
- home nursing services for nursing care such as assessments and wound care
- support group services to assist in adapting to the body image change and effects of amputation on ADLs.

Assessing for home care

Preparing the amputee for home care includes a careful assessment of the person's family and support services, and of the home for possible barriers to the person's safety and independence.

Assess the person's acceptance of the amputation and knowledge base about care needs, any activity restrictions or special needs and resources for home care. Discuss home management (i.e. who is responsible for household activities such as cleaning and cooking). Enquire about arrangements that have been made for home care activities and ADLs. Evaluate the person's use of prescription and non-prescription medications, paying particular attention to possible interactions and drugs that may affect the person's balance, mental alertness or appetite. Ask about social habits, such as cigarette smoking, alcohol use or other drug use, that may affect healing or the person's ability to provide self-care.

Assess the person's home environment for possible safety hazards or barriers to ambulation, such as:

- scatter rugs
- stairs between living areas of the house
- presence of grab bars to facilitate toileting and bathing
- access to clean water and other needs for wound care.

Health education for the person and family

The new amputee needs a great deal of teaching to learn to adapt to loss of a limb, whether it is an upper or lower extremity that has been lost. Because the person must be ready to learn before teaching can be effective, use therapeutic communication techniques to encourage the person to verbalise feelings about the amputation and its effects. Use active listening, and teach the person ways to reduce anxiety and deal with feelings of helplessness and loss. Encourage the person to participate in care of the stump to build self-esteem and reinforce teaching. Include the following in teaching for home care:

- Teach the person to wrap the stump appropriately in preparation for fitting the prosthesis.
- Discuss positioning of the stump. Contractures are a particular problem for people with an above-knee amputation and can interfere with the ability to use a prosthesis effectively.
- Teach the person how to perform stump exercises to maintain joint mobility and muscle tone of the affected limb.
- Encourage the person to resume physical activities as soon as possible. This improves the person's health and wellbeing, as well as the person's self-esteem.

MEETING INDIVIDUALISED NEEDS The person with an amputation (continued)

- Discuss household modifications to promote independence, such as grab bars in the bathroom, taps with single-handle controls for water flow and temperature, and handheld shower heads and shower chairs for bathing.

In addition, suggest the following resources:
- Healthdirect—Amputation: www.healthdirect.gov.au/amputation
- Amputees United of Australia: www.monash.edu.au/rehabtech/amputee/AUA.HTM

THE PERSON WITH A REPETITIVE USE INJURY

Repeatedly twisting and turning the wrist, pronating and supinating the forearm, kneeling or raising arms over the head can result in repetitive use injuries. People with repetitive use injuries pose a challenge to the healthcare team. Often these people appear puzzled as they relate a history of manifestations that have worsened over time. They deny abrupt trauma and often worry about the ability to return to work. Repetitive use injuries are common. The number of workers' compensation claims for repetitive use injuries is steadily growing. The increase is believed to be a result of technology advances in the workplace.

Pathophysiology

Common repetitive use injuries include carpal tunnel syndrome, bursitis and epicondylitis.

Carpal tunnel syndrome

The carpal tunnel is a canal through which flexor tendons and the median nerve pass from the wrist to the hand. The syndrome develops from narrowing of the tunnel and irritation of the median nerve. Carpal tunnel syndrome involves compression of the median nerve as a result of inflammation and swelling of the synovial lining of the tendon sheaths. The person complains of numbness and tingling of the thumb, index finger and lateral ventral surface of the middle finger. The person may also complain of pain in this area that interferes with sleep and is alleviated by shaking or massaging the hand and fingers. The affected hand may become weak and the person may be unable to hold utensils or perform activities that require precision.

Carpal tunnel syndrome is one of the three most common work-related injuries. The incidence is believed to be related directly to the number of people using computers. The incidence of carpal tunnel syndrome is higher in women, especially postmenopausal women.

Bursitis

Bursitis is an inflammation of a bursa. A bursa is an enclosed sac found between muscles, tendons and bony prominences. The bursae that commonly become inflamed are in the shoulder, hip, knee and elbow. Constant friction between the bursa and the musculoskeletal tissue around it causes irritation, oedema and inflammation. Manifestations develop as the sac becomes engorged. The area around the sac is tender and extension and flexion of the joint near the bursa produce pain. The inflamed bursa is hot, red and oedematous. The person guards the joint to decrease pain and may point to the area of the bursa when identifying joint tenderness.

Epicondylitis

Epicondylitis is inflammation of the tendon at its point of origin into the bone. Epicondylitis is also referred to as *tennis elbow* or *golfer's elbow*. The exact pathophysiology of epicondylitis is unknown. Current theories attribute inflammation of the tendon to microvascular trauma. Tears, bleeding and oedema are thought to cause avascularisation and calcification of the tendon. Manifestations of epicondylitis include point tenderness, pain radiating down the dorsal surface of the forearm and a history of repetitive use.

INTERPROFESSIONAL CARE

Medical management of repetitive use disorders focuses on relieving pain and increasing mobility. Once the diagnosis is made, treatment can range from conservative measures, such as rest and pharmacological agents, to aggressive measures such as surgery.

Diagnosis

Carpal tunnel syndrome is diagnosed by the person's history and physical examination. The history may reveal an occupation that involves areas such as computer work, jackhammer operation, mechanical work or gymnastics. History of a radial bone fracture or rheumatoid arthritis also increases the risk of carpal tunnel syndrome. Tests specific for carpal tunnel include Phalen's test (see Chapter 37). Bursitis and epicondylitis are diagnosed by history and physical examination.

Medications

The person with a repetitive use injury usually receives NSAIDs. Narcotics may also be administered for acute flare-ups and severe pain. For the person who has epicondylitis or carpal tunnel syndrome, corticosteroids may be injected into the joint.

Treatments

Treatment of repetitive use injuries is performed first by conservative management, followed, if necessary, by surgery.

CONSERVATIVE MANAGEMENT The first steps in the care of all repetitive use injuries are to immobilise and rest the involved joint. The joint may be splinted and ice may be applied (as described in Table 38.1) in the first 24 to 48 hours to decrease pain and inflammation. Ice application may be followed by heat application every 4 hours.

SURGERY Surgery is usually reserved for the person who does not obtain relief with conservative treatment. Surgery for carpal tunnel syndrome includes resection of the carpal ligament to enlarge the tunnel. In epicondylitis and bursitis, calcified deposits may be removed from the area surrounding the tendon or bursa.

Nursing care

The nursing care of a person with a repetitive use injury focuses on relieving pain, teaching about the disease process and treatment, and improving physical mobility.

Nursing diagnoses and interventions

Acute pain

Swelling and nerve inflammation cause pain in the person with a repetitive use injury.

- Ask the person to rate the pain on a scale of 0 to 10 (with 10 being the most severe pain) before and after any intervention. *This facilitates objective assessment of the effectiveness of the chosen pain relief strategy.*
- Encourage the use of immobilisers. *Splinting maintains joint alignment and prevents pain due to movement of inflamed tissues.*
- Teach the person to apply ice and/or heat as prescribed. *Ice causes vasoconstriction and decreases the pooling of blood in the inflamed area. Ice may also numb the tender area. Heat decreases swelling by increasing venous return.*
- Encourage use of NSAIDs as prescribed. *NSAIDs decrease swelling by inhibiting prostaglandins.*
- Explain why treatment should not be abruptly discontinued. *Abrupt discontinuation of treatment may cause reinflammation of the injured area.*

Impaired physical mobility

Joint pain and swelling can impair mobility.

- Suggest interventions to alleviate pain (such as using an immobiliser and taking pain medications). If the joint is pain free, the person will be more likely to take an active role in therapy.
- Refer to a physiotherapist for exercises. *The physiotherapist can assist the person with exercise to prevent joint stiffness.*
- Suggest consultation with an occupational therapist. *Occupational therapy can help the person learn new ways to perform tasks to prevent recurring symptoms.*

Community-based care

Address the following topics for home care:

- Causes of and treatments for repetitive use injury.
- Rehabilitation to allow the person to return to a state of independence.
- Ways to avoid unnecessary exposure to the activities that increase risk of redeveloping the injury. Suggest evaluation of the person's work environment by an environmental risk manager who can prescribe measures to reduce the risk of repetitive use injuries. Wrist supports or an ergonomic keyboard may be useful for the person who uses a computer extensively. Appropriate desk and chair height also are important in maintaining the correct anatomical position while working.
- Information about sources for braces or other assistive devices.

CHAPTER HIGHLIGHTS

- The most commonly reported musculoskeletal injuries are contusions, strains and sprains. Immediate treatment includes RICE (rest, ice, compression, elevation) therapy.
- Dislocations may be congenital, traumatic or pathological. Nursing assessments include monitoring neurovascular status by assessing for increased pain, decreased or absent pulses, pale skin, inability to move a body part or extremity, and changes in sensation.
- Any of the 206 bones of the body may sustain a fracture. Fractures are closed or simple (skin is intact) or open or compound (skin integrity is interrupted); open fractures are at risk of infection. Other fracture descriptors include oblique or spiral, avulsed, comminuted, compressed, impacted or depressed.
- Fractures heal through three phases: inflammatory, reparative and remodelling. Healing is influenced by the age and physical condition of the person and by the type of fracture.
- Fracture complications include compartment syndrome, fat embolism syndrome, deep venous thrombosis, infection, delayed union and non-union, and reflex sympathetic dystrophy.
- Fractures are treated with surgery, traction and/or casts to stabilise the fractured bone, maintain bone immobilisation, prevent complications and restore function.
- Fractures of the hip are most often sustained by older adult women and are usually the result of a fall. They are the most common injury in the older population, resulting in the greatest number of deaths, and cause the most serious health problems of all fractures for people aged 65 years and older.
- Nursing care for the person with a fracture focuses on interventions for acute pain, risk of peripheral vascular dysfunction, risk of infection, impaired physical mobility and risk of disturbed tactile sensory perception.
- An amputation is the partial or total removal of an extremity. This loss has a significant physical and psychosocial effect on the person and the family. The most common cause for amputation of a lower extremity is peripheral vascular disease. Trauma is the most common cause for upper extremity amputation.
- Complications that may follow an amputation include infection, delayed healing, chronic stump pain, phantom pain and contractures. Stump care is necessary to prevent complications and to prepare the stump for a prosthesis.
- Nursing care for the person with an amputation is provided as part of the interprofessional team and is focused on a return to functional health, with interventions to meet needs for acute pain, impaired skin integrity, grieving, disturbed body image and impaired physical mobility.
- Repetitive use injuries, especially common in the workplace, include carpal tunnel syndrome, bursitis and epicondylitis.

CONCEPT CHECK

1 You are teaching a young adult how to provide self-care for a sprained ankle. You explain that the reason for applying ice immediately after the injury is based on the principle that ice:
1 increases the diameter of blood vessels
2 decreases the diameter of blood vessels
3 is helpful in increasing white blood cells
4 lowers the blood pressure and pulse

2 A person with a compound, open fracture has been admitted to the emergency department and is scheduled for immediate surgery. Which of the following nursing diagnoses would be most appropriate in the immediate postoperative period?
1 *Risk of post-traumatic stress disorder (PTSD)*
2 *Impaired transfer ability*
3 *Risk of infection*
4 *Risk of falls*

3 While providing care to an older woman with a cast on her left lower arm (from below the elbow to above the fingers), you perform a neurovascular assessment. Which of the following assessments indicates a possible complication?
1 slightly oedematous fingers
2 warm, pink skin above the cast
3 pale, cold fingers
4 pain rating of 2 on a 1 to 10 scale

4 Which of the following minerals is essential to bone healing?
1 potassium
2 magnesium
3 sodium
4 calcium

5 You are assessing a young man with a newly applied long leg cast. He complains of extreme pain in his leg and his toes are cyanotic and lack sensation. What is your priority intervention?
1 Document the assessments carefully and accurately.
2 Notify the healthcare provider who applied the cast.
3 Elevate the leg on at least three pillows.
4 Apply an ice bag over the painful area.

6 Your assigned person has been diagnosed with DVT of the left lower extremity. What body system would require very careful monitoring?
1 haematological
2 respiratory
3 digestive
4 renal

7 Although nursing diagnoses are always individualised, what is one nursing diagnosis common to all musculoskeletal injuries?
1 *Disturbed body image*
2 *Acute pain*
3 *Chronic pain*
4 *Risk of infection*

8 At what position would you place the remaining extremity following a below-the-knee amputation during the first 24 hours after surgery?
1 elevated above the level of the heart
2 lower than the rest of the body
3 crossed over the intact extremity
4 level with the rest of the body

9 The day following a below-the-knee amputation, the person you are caring for tells you that he feels as though his toes are cramping in the amputated foot. What is this experience called?
1 chronic stump pain
2 contractures
3 attention seeking
4 phantom limb pain

10 A friend is cutting wood with a circular saw. He suddenly screams that he has cut off his finger. What would you do with the amputated finger?
1 Don't worry about it; the important thing is to get him to the hospital.
2 Put it in a storage bag filled with warm water.
3 Tape it to his hand so the emergency personnel will know where it is.
4 Wrap it in a towel, put it in a plastic bag and lay it on ice.

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CHAPTER 39

NURSING CARE OF PEOPLE WITH MUSCULOSKELETAL DISORDERS

ANN-MARIE BROWN

KEY TERMS

LEARNING OUTCOMES

- Explain the pathophysiology, manifestations, complications, interprofessional care and nursing care of metabolic, degenerative, autoimmune, inflammatory, infectious, neoplastic, connective tissue and structural musculoskeletal disorders.
- Compare and contrast the pathophysiology, manifestations, diagnosis and treatments for osteoporosis, osteoarthritis, Paget's disease and rheumatoid arthritis.
- Discuss the purposes, nursing implications and health education for the person and family regarding medications used to treat osteoporosis, Paget's disease, gout, osteomalacia, osteoarthritis, rheumatoid arthritis, systemic lupus erythematosus, osteomyelitis, bone tumours, scleroderma and lower back pain.
- Describe the surgical procedures used to treat people with arthritis.

CLINICAL COMPETENCIES

- Assess functional status of people with musculoskeletal disorders and monitor, document and report abnormal manifestations.
- Use evidence-based research to assess people at risk of osteoporosis and to evaluate the effectiveness of internet use to teach older adults with rheumatoid arthritis.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with musculoskeletal disorders.
- Administer topical, oral and injectable medications used to treat musculoskeletal disorders knowledgeably and safely.
- Provide skilled care to people having a surgical debridement for osteomyelitis and a total joint replacement.
- Integrate interprofessional care into care of people with musculoskeletal disorders.
- Provide teaching appropriate for community-based self-care of musculoskeletal disorders.
- Revise the plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to people with musculoskeletal disorders.

Various metabolic, degenerative, autoimmune, inflammatory, infectious, neoplastic, connective tissue and structural disorders may affect the musculoskeletal system. Many of these diseases have significant physical, psychosocial and financial consequences. When these problems occur, people experience a variety of individualised responses to their altered health status. Nursing care is directed towards meeting physiological needs, providing education and ensuring psychological support for the person and family.

Arthritis refers to inflammation of the joints, while **rheumatic disorders** refer to diseases of the muscles and bones as well as the joints. These diseases affect not only the joints but also the connective tissues of the body. The various types of arthritis are discussed in this chapter in different sections, depending on the primary aetiology of the disorder. Approximately 15% (3.3 million people) of all Australians had arthritis in 2011–2012 (Australian Bureau of Statistics (ABS), 2012). The two most prevalent forms of arthritis are osteoarthritis, which accounts for 51% of cases, and rheumatoid arthritis, which accounts for a further 14% of cases (ABS, 2012). According to the ABS, 37.3% of people who have arthritis don't know which type they have (ABS, 2012).

There are more than 100 different types of arthritis, but the most common are osteoarthritis, rheumatoid arthritis, systemic lupus erythematosus, ankylosing spondylitis and gout (Arthritis Australia, 2015). The aetiology of most rheumatic disorders is not clear; in many cases, the pathophysiological processes involved are often complex and poorly understood. Many are primary disorders; others occur as secondary processes associated with another disease. The wear and tear of ageing, autoimmune processes, metabolic disorders, genetic factors and infection are also implicated as causative factors in some forms of rheumatic disease.

Arthritic disorders are a leading cause of disability; however, their very prevalence may lead the public and healthcare professionals to treat them as normal ageing processes or to discount the validity of the pain and disability experienced by the person with arthritis.

METABOLIC BONE DISORDERS

Metabolic bone disorders originate in the bone remodelling process, which normally involves a sequence of events of bone reabsorption and formation. In the adult, this process is primarily internal remodelling through replacement of trabecular bone. Adults replace about 25% of trabecular bone every 4 months through reabsorption of old bone by osteoclasts and formation of new bone by osteoblasts (Porth & Matfin, 2014). Metabolic bone disorders may result from a variety of factors, including ageing, calcium and phosphate imbalances, genetics and changes in levels of hormones.

THE PERSON WITH OSTEOPOROSIS

Osteoporosis, literally defined as 'porous bones', is a metabolic bone disorder characterised by loss of bone mass, increased bone fragility and an increased risk of fractures. The reduced bone mass is caused by an imbalance of the processes that influence bone growth and maintenance. Although osteoporosis may result from an endocrine disorder or malignancy, it is most often associated with ageing.

In Australia, the number of people suffering from osteoporosis is increasing. Women over the age of 50 are at greatest risk of developing a fracture. In 2011–2012, the estimated prevalence of self-reported diagnosed osteoporosis was 15% of women and 3% of men (AIHW, 2014).

Risk factors

The risk of developing osteoporosis depends on how much bone mass is achieved between ages 25 and 35 and how much is lost later. Certain diseases, lifestyle habits and ethnic backgrounds increase the risk of developing osteoporosis (see the 'Focus on cultural diversity' box below). Different variables affect one's risk of osteoporosis—some can be modified and others cannot. The risk factors are summarised in Box 39.1.

BOX 39.1 Risk factors for osteoporosis

- A family history of osteoporosis
- Personal history of fracture after age 50
- Current low bone mass
- History of fracture in a first-degree relative
- Being female, especially Caucasian or Asian
- Being thin and/or having a small frame
- Menopause-associated low oestrogen levels
- Low testosterone levels in men
- Dietary: low lifetime calcium intake, vitamin D deficiency
- Medication use: anticonvulsants, corticosteroids
- Lifestyle: inactivity, cigarette smoking, excess alcohol
- Presence of certain chronic diseases

Unmodifiable risk factors

Both men and women are susceptible to osteoporosis as they age, because the osteoblasts and osteoclasts undergo alterations that diminish their activity. Women have a significantly higher risk of manifestations and complications of osteoporosis because their peak bone mass is 10–15% less than that of men. In addition, age-related bone loss begins earlier and proceeds more rapidly in women, beginning in their thirties and accelerating before menopause. Oestrogen in women and testosterone in men appear to help prevent bone loss; decreasing levels of these hormones associated with ageing contribute to bone loss. Age-related bone loss in men occurs 15 to 20 years later than in women and at a slower rate.

Indigenous people of both sexes are much more likely than non-Indigenous people to be hospitalised with a minimal hip trauma. Indigenous Australians were slightly more likely to

FOCUS ON CULTURAL DIVERSITY

The person with osteoporosis

- Significant risk is reported for people of all cultural backgrounds, but the highest percentage of cases are in European and Asian women aged 50 or older, with 20% estimated to have osteoporosis and 52% estimated to have low bone mass.
- When differences in age structure are taken into account, the prevalence of osteoporosis in Indigenous Australians is lower than for non-Indigenous Australians. About 2% (1.5% of men and 2.5% of women) of Indigenous Australians have doctor-diagnosed osteoporosis compared with about 3% of non-Indigenous Australians (0.9% of men and 4.7% of women).

Source: Australian Institute of Health and Welfare (2011). *A picture of osteoarthritis in Australia.* Retrieved from www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=10737418747.

report having arthritis than non-Indigenous Australians (18% compared to 15%) and back problems (18% compared to 16%). The proportion of Indigenous Australians reporting osteoporosis was similar to that for non-Indigenous Australians (2% compared to 3%) (ABS, 2011).

Premature osteoporosis is increasing in female athletes, who have a greater incidence of eating disorders and amenorrhoea. Poor nutrition and intense physical training can result in a deficient production of oestrogen. Decreased oestrogen, combined with a lack of calcium and vitamin D, results in a loss of bone density (Porth & Matfin, 2014).

People who have an endocrine disorder such as hyperthyroidism, hyperparathyroidism, Cushing's syndrome or diabetes mellitus are at high risk of osteoporosis. These disorders affect the metabolism, in turn affecting nutritional status and bone mineralisation.

Modifiable risk factors

Modifiable risk factors include behaviours that place a person at risk of developing osteoporosis, as well as physical changes such as menopause whose contribution to osteoporosis can be modified by preventive strategies. Calcium deficiency is an important modifiable risk factor contributing to osteoporosis. Calcium is an essential mineral in the process of bone formation and other significant body functions. When there is an insufficient intake of calcium in the diet, the body compensates by removing calcium from the skeleton, weakening bone tissue. Acidosis, which may result from a high-protein diet, contributes to osteoporosis in two ways. Calcium is withdrawn from the bone as the kidneys attempt to buffer the excess acid. Acidosis also may directly stimulate osteoclast function. A high intake of diet soda with a high phosphate content also can deplete calcium stores.

With menopause and decreasing oestrogen levels, bone loss accelerates in women. Oestrogen promotes the activity of osteoblasts, increasing new bone formation. In addition, oestrogen enhances calcium absorption and stimulates the thyroid gland to secrete calcitonin, a hormone that suppresses osteoclast activity and increases osteoblast activity.

Both cigarette smoking and excess alcohol intake are risk factors for osteoporosis. Smoking decreases the blood supply to bones. Nicotine slows the production of osteoblasts and impairs the absorption of calcium, contributing to decreased bone density. Alcohol has a direct toxic effect on osteoblast activity, suppressing bone formation during periods of alcohol intoxication. In addition, heavy alcohol use may be associated with nutritional deficiencies that contribute to osteoporosis. Interestingly, moderate alcohol consumption in postmenopausal women actually may increase bone mineral content, possibly by increasing levels of oestrogen and calcitonin.

Sedentary lifestyle is another modifiable risk factor that can cause osteoporosis. Weight-bearing exercise, such as walking, influences bone metabolism in several ways. The stress of this type of exercise causes an increase in blood flow to bones, which brings growth-producing nutrients to the cells. Walking causes an increase in osteoblast growth and activity.

Prolonged use of medications that increase calcium excretion, such as aluminium-containing antacids and anticonvulsants, increase the risk of developing osteoporosis. Heparin therapy increases bone resorption and its prolonged use is associated with osteoporosis. Antiretroviral therapy for people with AIDS or HIV infection may cause decreased bone density and osteoporosis (Porth & Matfin, 2014).

Anyone who takes a glucocorticoid medication for more than 3 months is at risk of glucocorticoid-induced osteoporosis. These medications, often prescribed to control many rheumatic diseases, include prednisolone (Prednisone) and dexamethasone (Decadron, Hexadrol). They can directly affect bone cells, slowing the rate of bone formation. They also interfere with how the body uses calcium and affect levels of sex hormones, leading to bone loss. Problems that result, such as an increased possibility of fractures, can be prevented by taking a daily regimen of calcium supplements with added vitamin D and one multivitamin (Mazziotti, Canalis & Giustina, 2010).

Pathophysiology

Although the exact pathophysiology of osteoporosis is unclear, it is known to involve an imbalance of the activity of osteoblasts that form new bone and osteoclasts that resorb bone. Until age 35, when peak bone mass occurs, formation occurs more rapidly than does reabsorption. After peak bone mass is achieved, slightly more is lost than is gained (about 0.3–0.5% per year); this loss is accelerated if the diet is deficient in vitamin D and calcium. In women, bone loss begins to increase to 1–2% per year from the age of 45 years, increases to 2–4% per year at the onset of menopause (with loss of oestrogen), then slows but does not stop at about age 60. In men, bone density tends to remain relatively stable until middle age, decreasing by about 0.5–1.0% per year from the age of 45–55 years (Osteoporosis Australia, 2012).

Osteoporosis affects the diaphysis (shaft of the bone) and the metaphysis (portion of the bone between the diaphysis and the epiphysis). The diameter of the bone increases, thinning the outer supporting cortex. As osteoporosis progresses, trabeculae are lost from cancellous bone (the spongy tissue of bone) and the outer cortex thins to the point that even minimal stress will fracture the bone (Porth & Matfin, 2014).

Manifestations

The most common manifestations of osteoporosis are loss of height, progressive curvature of the spine, lower back pain and fractures of the forearm, spine or hip. Osteoporosis is often called the 'silent disease' because bone loss occurs without symptoms.

The loss of height occurs as vertebral bodies collapse. Acute episodes generally are painful, with radiation of the pain around the flank into the abdomen. Vertebral collapse can occur with little or no stress; minimal movements such as bending, lifting or jumping may precipitate the pain. In some people, vertebral collapse may occur slowly, accompanied by little discomfort. Along with loss of height, characteristic dorsal kyphosis and cervical lordosis develop, accounting for the 'dowager's hump' often associated with ageing. The abdomen tends to protrude and the knees and hips to flex as the body attempts to maintain its centre of gravity (see Figure 39.1).

Complications

The most common fractures are vertebral, hips and wrists. There may be no obvious manifestations of osteoporosis until fractures occur. Some fractures are spontaneous; others may result from everyday activities. While wrist and vertebral fractures have not been shown to increase disability or mortality, persistent pain and associated posture changes may restrict the person's activities or interfere with activities of daily living (ADLs).

INTERPROFESSIONAL CARE

Care of the person with osteoporosis focuses on stopping or slowing the process, alleviating the symptoms and preventing complications. Proper nutrition and exercise are important components of the treatment program.

Diagnosis

The manifestations of osteoporosis can mimic those of other bone disorders, so diagnostic tests are needed to differentiate osteoporosis from other problems.

A bone mineral density (BMD) scan assesses the mass of bone per unit volume—how tightly the bone is packed. Dual-energy x-ray absorptiometry (DEXA) measures bone density in the lumbar spine or hip and is considered to be highly accurate. Ultrasound transmits painless sound waves through the heel of the foot to measure bone density. This 1-minute test is not as sensitive as DEXA, but is accurate enough for screening purposes. These tests are described in Chapter 37.

Laboratory tests include alkaline phosphatase (AST), which may be elevated following a fracture, and serum bone Gla protein (osteocalcin), which can be used as a marker of osteoclastic activity and therefore is an indicator of the rate of

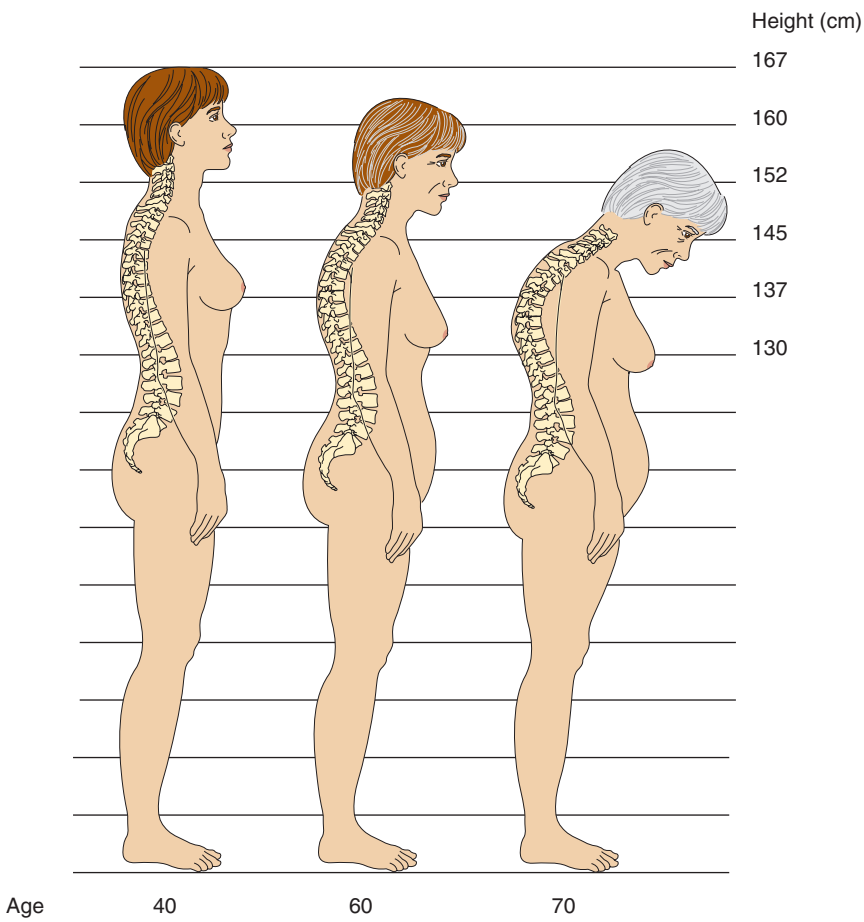


FIGURE 39.1 ■ Spinal changes caused by osteoporosis. As the condition progresses, height can be reduced by as much as 17 cm

TABLE 39.1 Differential features of osteoporosis, osteomalacia and Paget's disease

DIFFERENTIATING FEATURES	OSTEOPOROSIS	OSTEOMALACIA	PAGET'S DISEASE
Pathophysiology	Resorption greater than bone formation	Inadequate mineralisation of bone matrix	Excessive osteoclastic activity and formation of poor-quality bone
Calcium level (serum)	Normal	Low or normal	Normal or elevated (especially in immobilised people)
Phosphate level (serum)	Normal	Low or normal	Normal
Parathyroid hormone level (serum)	Normal	High or normal	Normal
Alkaline phosphatase level (serum)	Normal	Elevated	Increased; not a reliable test for people who have liver disease or are pregnant
Hydroxyproline (urine)	Not applicable	Not applicable	Increased
Radiographic findings	Osteopenia, fractures	Decreased bone density, radiolucent bands known as Looser's zones or pseudofractures	'Punched-out' appearance of bone, increase in bone thickness, linear fractures, mosaic pattern of bone matrix

bone turnover. This test is most useful to evaluate the effects of treatment, rather than as an indicator of the severity of the disease. A comparison of laboratory test results for metabolic bone diseases is outlined in Table 39.1.

Medications

Oestrogen replacement therapy reduces bone loss, increases bone density in the spine and hip, and reduces the risk of fractures in postmenopausal women. It is particularly recommended for women who have undergone surgical menopause before age 50 and is often prescribed for women with other osteoporosis risk factors. Oestrogen therapy alone is associated with an increased risk of endometrial cancer, so it usually is prescribed in combination with progestin (hormone replacement therapy, or HRT). The choice of using HRT to prevent osteoporosis is one that must be made between the woman and her healthcare provider.

Raloxifene (Evista) is a selective oestrogen receptor modulator (SERM) that appears to prevent bone loss by mimicking oestrogen's beneficial effects on bone density in postmenopausal women. It does not have the risks of oestrogen. Hot flushes are a common side effect and this drug should not be taken by a woman with a history of blood clots.

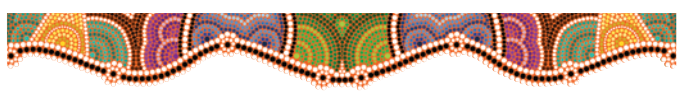
Alendronate (Fosamax) and risedronate (Actonel) are from the class of drugs known as bisphosphonates. Bisphosphonates are potent inhibitors of bone resorption that may be used to prevent and treat osteoporosis. They inhibit bone breakdown, preserve bone mass and increase bone density in the hip and vertebrae. Alendronate is especially useful for men and young adults and to prevent or treat glucocorticoid-induced osteoporosis. The nursing implications of bisphosphonates are found in the 'Medication administration' box on page 1432. Teriparatide (Forteo) is a synthetic parathyroid hormone, administered subcutaneously to stimulate new bone formation and mass. It is used to decrease the risk of bone fracture from osteoporosis in postmenopausal women and in men with primary or secondary hypogonadism.

Ibandronate sodium (Boniva) is the first monthly osteoporosis medication. It is used for both treatment and prevention of postmenopausal osteoporosis and reduces the number of vertebral fractures in women with osteoporosis, as well as increasing bone density in women who do not have the disease. It has been available in Australia since 2006 (Osteoporosis Australia, 2015). Ibandronate sodium needs to be taken on the same day of each month.

Calcitonin is a hormone that increases bone formation and decreases bone resorption. Calcitonin increases spinal bone density and reduces the risk of compression fractures; it may reduce the risk of hip fracture as well. Calcitonin usually is prescribed as a nasal spray, although it also is available in parenteral form. Because calcitonin is a protein, it can precipitate anaphylactic-type allergic responses.

Sodium fluoride stimulates osteoblast activity, increasing bone formation. When used to treat osteoporosis, bone mass of the spine increases and the risk of spinal fractures may be reduced. Fluoride therapy may, however, be associated with an increased risk of hip and other non-vertebral fractures.

Medications being investigated include vitamin D metabolites and other bisphosphonates. See the 'Medication administration' box below for information about calcium, calcitonin and fluoride.



Nursing care

Osteoporosis is both preventable and treatable; therefore, nursing care focuses primarily on planning and implementing interventions to prevent the disease, its manifestations and the resulting injuries. An important aspect of preventing osteoporosis is educating people under age 35. A nursing care plan for a person with osteoporosis is found below.

Health promotion

Health promotion activities to prevent or slow osteoporosis focus on calcium intake, exercise and health-related behaviours.

Nutrition

For people of all ages, stress the importance of maintaining a daily calcium intake that meets recommendations of the National

MEDICATION ADMINISTRATION The person with osteoporosis

CALCIUM

Postmenopausal women, regardless of whether they take replacement oestrogen, are encouraged to take calcium to prevent osteoporosis.

Nursing responsibilities

- Help the person to maintain an adequate dietary intake of calcium. The best dietary source is milk and other dairy products, including yoghurt.
- Postmenopausal women who take oestrogen need 1000 mg of calcium daily. Those who do not take oestrogen need about 1500 mg daily to minimise osteoporosis.
- Identify alternative sources, such as skim milk and low-fat yoghurt, oysters, canned sardines or salmon, beans, cauliflower and dark-green leafy vegetables.

Health education for the person and family

- Take calcium carbonate in divided doses 30 to 60 minutes before meals to allow for absorption.
- Take calcium citrate with meals to minimise gastrointestinal distress.

CALCITONIN

Calcitonin–salmon injection, synthetic Calcimar Miacalin (injection or nasal spray)

In postmenopausal osteoporosis, calcitonin prevents further bone loss and increases bone mass if the person consumes adequate amounts of calcium and vitamin D. Calcitonin may be used in postmenopausal women who cannot or will not take oestrogen.

Nursing responsibilities

- Calcitonin is protein in nature; both the parenteral form and the nasal spray forms may cause an anaphylactic-type allergic response. Observe the person for 20 minutes after administration; have appropriate emergency equipment and drugs available to treat anaphylaxis.
- Alternate nostrils daily when administering calcitonin nasal spray.
- Review medical history for conditions that contraindicate use of calcitonin products: hypersensitivity to salmon calcitonin and lactation (calcitonin is secreted in breast milk and may inhibit lactation).
- Observe for side effects: nausea and vomiting, anorexia, mild transient flushing of the palms of the hands and the soles of the feet, and urinary frequency.
- Teach the person the proper technique for handling and injecting the drug at home.

Health education for the person and family

- Take the medication in the evening to minimise side effects.
- Warm nasal spray to room temperature before using.
- Rhinitis (runny nose) is the most common side effect with calcitonin nasal spray. Other possible side effects include sores, itching or other nasal symptoms. Report nosebleeds to your healthcare provider.
- Nausea and vomiting may occur during initial stages of therapy; they disappear as treatment continues.
- While taking the medication, be sure to consume adequate amounts of calcium and vitamin D.

FLUORIDE

Fluoride is a mineral long recognised as essential for the normal formation of dentine and tooth enamel. Fluoride appears to decrease the solubility of bone mineral and therefore the rate of bone reabsorption. Its use in preventing and treating osteoporosis is relatively new but promising.

Nursing responsibilities

- Monitor serum fluoride levels every 3 months.
- Have bone mineral density studies conducted at 6-month intervals to document progress of bone growth.

Health education for the person and family

- Take sodium fluoride tablets after meals and avoid milk or dairy products; these reduce gastrointestinal absorption of the medication.
- While taking fluoride, be sure to maintain an adequate calcium intake.
- Use fluoride mouth rinse immediately after brushing teeth and just before retiring at night. Do not swallow the rinse and avoid eating or drinking for at least 30 minutes after use.
- Notify the healthcare provider if teeth become stained or mottled after repeated use of fluoride mouth rinse.

Institutes of Health (for Australia and New Zealand) and the National Health and Medical Research Council (NRMRC) and Ministry of Health (MoH) (2015). This is particularly important for adolescent girls and young adult women who may avoid eating many high-calcium foods such as dairy products because of concerns about weight. Optimal calcium intake before ages 30 to 35 probably increases peak bone mass. Emphasise that low-fat (or non-fat) dairy products also contain calcium, although some fat in the product may enhance calcium absorption.

Milk and milk products are the best sources of calcium. The lactose in milk facilitates calcium absorption as well. Other food sources of calcium include sardines, clams, oysters and salmon, as well as dark-green leafy vegetables such as broccoli, bok choy and spinach. For people who avoid dairy products because of lactose intolerance or a vegetarian diet, suggest alternative sources.

Calcium supplements are available in many forms. Most supplements provide calcium carbonate in the range of 200 to 600 mg per tablet. Other forms of calcium, including citrate, gluconate and lactate, generally provide a lower amount of elemental calcium per tablet. A combination of calcium with vitamin D is recommended, particularly for older adults who may have a vitamin D deficiency that impairs their ability to absorb and use calcium.

Exercise

Teach people the importance of physical activity and weight-bearing exercises in preventing and slowing bone loss. Suggest that people participate in regular exercise, such as walking for at least 20 minutes four or more times a week. Inform people that swimming and pool aerobic exercises are not as beneficial for maintaining bone density because of the lack of weight-bearing activity.

NURSING CARE PLAN A person with osteoporosis

Nancy Bauer is a 53-year-old school teacher. She has been married for 36 years and has two children. Mrs Bauer is 165 cm tall. She has smoked one packet of cigarettes a day for 30 years and drinks one to two glasses of wine with dinner each evening. She does not routinely exercise. Mrs Bauer has had symptoms of menopause for 8 years, including hot flushes in the early years and mood swings of late. She has never been on hormone replacement therapy.

Mrs Bauer is currently seeking medical advice for continuous lower back pain. The pain is not relieved with an over-the-counter analgesic and she frequently wakes up during the night because of the pain. She is diagnosed with osteoporosis.

ASSESSMENT

The nurse practitioner notes that Mrs Bauer's vital signs are within normal limits. She has full range of motion of all extremities and is able to stand and bend over, but she reports discomfort when returning to the upright position. Mrs Bauer has a slightly pronounced 'hump' on her upper back and is 2.5 cm shorter than her stated height on admission. Her muscle strength is symmetrical and strong.

DIAGNOSIS

- *Acute pain* of the lower spine related to vertebral compression.
- *Deficient knowledge* related to osteoporosis and treatment to prevent further damage.
- *Imbalanced nutrition: less than body requirements* related to inadequate intake of calcium.
- *Risk of injury* related to effects of change in bone structure secondary to osteoporosis.

PLANNING

Ensure Mrs Bauer is able to manage her pain and educate her regarding her osteoporosis and associated issues.

Expected outcomes

- Verbalise a decrease in back pain.
- Be able to describe ways to treat her osteoporosis and prevent further complications.
- Verbalise an understanding of the current research and treatment regarding osteoporosis.
- Verbalise safety precautions to prevent fractures due to falls.
- Seek consultation for supplements and medications to prevent further bone loss.
- Verbalise how stopping smoking can help prevent further progression of osteoporosis.
- Design a program of physical activity to prevent complications of osteoporosis.

IMPLEMENTATION

- Teach back-strengthening exercises.
- Refer to an osteoporosis support group, if available.
- Assess current knowledge base and correct misconceptions regarding treatment of osteoporosis.
- Provide current educational literature regarding treatment of osteoporosis.
- Review safety and fall precautions, and provide literature regarding how to create a safe home environment.
- Instruct in dietary and calcium supplements that help prevent effects of osteoporosis.
- Provide realistic, yet optimistic, feedback about loss of height and bone integrity and the potential outcomes of treatment.
- Discuss physical exercises that help prevent complications due to osteoporosis.

EVALUATION

On her return visit 6 months later, Mrs Bauer reports that she feels much better. She is no longer irritable and does not experience mood swings because she has been taking her prescribed hormone replacements for 6 months. She is eating products rich in calcium and taking a daily supplement of calcium with vitamin D. Mrs Bauer has reduced her wine intake to one glass in the evening and now drinks decaffeinated coffee and tea. She also states that since she stopped smoking, she has been walking 30 to 45 minutes every day.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the rationale for stopping smoking and limiting caffeine and alcohol intake in the treatment of osteoporosis?
2. Which foods would you encourage for people at high risk of osteoporosis whose serum cholesterol and LDL/HDL ratios indicate a high risk of cardiovascular disease?
3. Which physical activities would you consider beneficial in helping to prevent the effects of osteoporosis in the female person who is wheelchair bound or has limited mobility?
4. Develop a care plan for Mrs Bauer for the nursing diagnosis *Risk of trauma.*

REFLECTION ON THE NURSING PROCESS

1. Reflecting on the care for Mrs Bauer, identify the main issues you have learned from this case study that you will able to apply in future practice.
2. What type of health education is required for Mrs Bauer prior to discharge, and how would this be best delivered to her?

Healthy behaviours

Behaviours that help prevent osteoporosis include not smoking, avoiding excessive alcohol intake and limiting caffeine intake to two or three cups of coffee each day.

Assessment

Collect the following data through the health history and physical examination:

- *Health history:* age, risk factors, history of fractures, smoking history, alcohol intake, medications, usual diet, menstrual history including menopause, usual exercise/activity level, lower back pain (see the following 'Translation to practice' box).
- *Physical examination:* height, spinal curves.

Nursing diagnoses and interventions

Nursing care of people who have osteoporosis focuses on teaching about the disease process, helping maintain physical mobility and nutrition, and solving problems associated with pain and injury.

TRANSLATION TO PRACTICE Evidence-based practice for the person with osteoporosis

Osteoporosis is a major health problem in Australia and New Zealand. Risk factors for osteoporosis include being Caucasian, having a small body frame, not doing weight-bearing exercises and having a family history of osteoporosis. Nursing can do much to prevent the development of osteoporosis by assessing risk factors and teaching about diet, exercise and lifestyle.

IMPLICATIONS FOR NURSING

Assessment is a critical component of the nursing process, enabling nurses to identify people at risk of diseases, to monitor ongoing interventions and to design teaching specific to a person's needs. The risk factors for osteoporosis are genetics, calcium and vitamin D status, body mass index, previous fractures, recurrent falls, medical conditions such as malabsorption disorders, thyroid disease, diabetes, chronic kidney or liver disease, and medications such as antiepileptic drugs, antidepressants and long-term glucocortisoids (Osteoporosis Australia, 2014).

CRITICAL THINKING IN PERSON-CENTRED CARE

1. At what age do women develop maximum bone mass? (Review information in a text or on the internet.) Based on this information, what type of teaching would be most effective?
2. Compare and contrast your teaching for a 24-year-old Indigenous Australian woman who has a calcium-poor diet but is a non-smoker, and a thin, Caucasian, 64-year-old woman who is postmenopausal and smokes.
3. While screening people in a clinic for health risks, including osteoporosis, a man in his seventies says, 'Oh, I can't have bad bones . . . I'm a man!' How would you respond?

Health-seeking behaviours

At multiple points in the person's lifetime, nurses can provide vital information that will help people use self-care strategies to reduce their risk of developing osteoporosis:

- Assess the person's health habits, including diet, exercise, smoking and alcohol use. *The risk of developing osteoporosis in later life is affected by such things as diet, regular participation in weight-bearing exercise and personal habits such as smoking and alcohol consumption.*
- Teach women and men of all ages the importance of maintaining an adequate calcium intake. Provide a list of calcium-rich foods and discuss the use of calcium supplements with people who do not consume adequate dietary calcium. *Calcium needs vary during the course of a lifetime; however, many people never consume adequate amounts of calcium. This affects their peak bone mass and the rate of bone loss with ageing. Calcium in foods is more completely absorbed than that supplied by calcium supplements.*
- Discuss the importance of maintaining a regular schedule of weight-bearing exercise, either through an exercise program or regular physical activity. *Weight-bearing exercise promotes osteoblast activity, helping maintain bone strength and integrity.*
- Refer people to smoking-cessation programs and alcohol treatment programs as appropriate. *Smoking interferes with oestrogen's protective effects on bones, promoting bone loss. Excess alcohol intake affects the nutritional status of the person, increasing the risk of calcium and vitamin D deficiency.*
- Refer people with significant risk factors for osteoporosis to primary care providers or clinics for bone-density evaluation. *Early identification and treatment of osteoporotic changes in bones can reduce the risk and possible long-term consequences of falls and fractures.*

Risk of injury

Falls that would result in little or no injury in the healthy adult may cause fractures in the person with osteoporosis. Even normal movements such as twisting, bending, lifting or rising from bed can precipitate a vertebral fracture.

- Implement safety precautions as necessary for the person who is hospitalised or in a long-term care facility. Maintain the bed in a low position; use side rails if indicated to prevent the person from getting up alone; provide night-time lighting to toilet facilities. *Most falls are preventable, particularly in hospitals and long-term care facilities.*
- Avoid using restraints (if hospitalised or a resident in a long-term care facility) if at all possible. *Restraints may actually increase the person's risk of falling, as well as the risk of injury associated with a fall.*

CONSIDERATION FOR PRACTICE

People may fracture osteoporotic bones when pulling against restraints.

- Teach people who are able to participate in weight-bearing exercises to perform exercises at least three times a week for a sustained period of 30 to 40 minutes. *The mechanical force of weight-bearing exercises promotes bone growth. Bones weaken and demineralise without exercise. Walking is an easy, low-impact form of exercise. Swimming (including walking on the bottom of the pool) does not provide the needed weight-bearing activity.*
- Encourage older adults to use assistive devices to maintain independence in ADLs. *Walking sticks, canes and other assistive devices encourage independence and support activities that promote bone growth.*
- Teach older people about safety and fall precautions. *An assessment of the person's home for safety and fall risks may reduce the risk of fractures and, in turn, the cost of hospitalisation and potential disability and/or death.*

Imbalanced nutrition: less than body requirements

Most Australians do not maintain their recommended daily intake of calcium. People must therefore be made aware of the

relationship between an adequate calcium intake and maintaining strong bones. The recommendations for Australia and New Zealand are:

- 1300 mg/day for girls and boys aged 12 to 18 years
- 1000 mg/day for pregnant and lactating women over 19; 1300 mg/day for pregnant and lactating women under 18
- 1000 mg/day for women aged 19 to 50
- 1000 mg/day for men aged 19 to 70
- 1300 mg/day for women over 50 and men over 70 (NHMRC and MoH, 2015).

Teach people taking calcium supplements the importance of taking the medication at the proper time and the side effects that may occur. Free hydrochloric acid is needed for calcium absorption. Calcium carbonate supplement should be taken 30 to 60 minutes before meals to allow adequate absorption. Calcium citrate supplements should be taken with meals to prevent gastrointestinal distress.

CONSIDERATION FOR PRACTICE
Calcium supplements should be taken in divided doses (two to three times daily) for improved distribution.

Acute pain

Advanced stages of osteoporosis can result in pain and immobilisation. Acute pain usually results from a complicating fracture, especially a compression fracture of the vertebrae.

- Suggest anti-inflammatory pain medications for treatment of both acute and chronic phases of pain. People should be instructed in the amount and frequency as noted on the manufacturer's labels. *Continuous administration of ibuprofen or other non-steroidal anti-inflammatory drugs (NSAIDs) can be useful to provide relief from pain, but people must be cautioned not to exceed dosage recommendations.*

CONSIDERATION FOR PRACTICE
Teach people on long-term anti-inflammatory medications to watch for bright-red bleeding from the stomach (in vomitus) or black bowel movements.

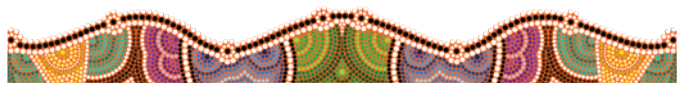
- Suggest the application of heat to relieve pain. *A heating pad may offer temporary pain relief. To avoid the 'rebound effect', the heat should be removed every 20 to 30 minutes.*

Community-based care

The person who has osteoporosis needs education on safety and preventing falls. In addition to home safety, outdoor safety is important, too. People should be taught to use assistive devices for added stability, to wear rubber-soled shoes for traction and to walk on the grass when footpaths are slippery.

Address the following topics when discussing home care:

- resources for medical supplies and assistive devices
- diet, exercise and medications
- pain management
- maintaining good posture to help prevent stress on the spine
- helpful resources:
  - Arthritis Australia: www.arthritisaustralia.com.au
  - Department of Health: www.health.gov.au
  - National Osteoporosis Foundation: www.nof.org/articles/238
  - Osteoporosis and Related Bone Diseases National Resource Centre (NIH): www.bones.nih.gov
  - Osteoporosis Australia: www.osteoporosis.org.au
  - Osteoporosis New Zealand: www.bones.org.nz.



THE PERSON WITH PAGET'S DISEASE

Paget's disease, also called *osteitis deformans*, is a progressive metabolic skeletal disorder that results from excessive metabolic activity in bone, with excessive bone resorption followed by excessive bone formation. This chronic remodelling results in the affected bones being larger and softer, with manifestations of bone pain, arthritis, obvious skeletal deformities and fractures. The disorder affects bones of the axial skeleton, especially the femur, pelvis, vertebrae and skull. The disease may affect one bone or multiple bones. The cause is unknown; however, several theories have been proposed, including hormonal imbalance, vascular disorder, neoplasm, autoimmune disorder and inborn error of connective tissue. It affects up to 4% of Australians over the age of 55 years (Walsh, 2012).

Paget's disease occurs in both men and women, affecting 1.5–8% of the population over the age of 50 in many countries. It is less common in people of Asian, Indian and Scandinavian descent. It is more common in men—2:1 compared to women—and up to 40% of all people with Paget's disease have at least one other relative with the disorder. It has a familial tendency as a result of mutations in several genes. The measles virus has been found in bone lesions and the relevance of that finding is under investigation (Paget Foundation, 2015).

Pathophysiology

Paget's disease progresses slowly. It usually follows a two-stage process: an excessive amount of osteoclastic bone resorption followed by excessive osteoblastic bone formation. The initial phase presents with an abnormal increase in osteoclasts. The bones increase in size and thickness because of the acceleration in bone resorption and regeneration, resulting in a thick layer of coarse bone with a rough and pitted outer surface (Porth & Matfin, 2014). Resorption of cancellous bone occurs rapidly. As new bone tissue tries to replace the loss, fibrous tissue forms in the bone marrow. The bone is at first hyperaemic and soft, and bowing occurs. When this excessive bone cell activity decreases, the result is a gain in bone mass, but the newly formed bone becomes hard and brittle. This brittleness may lead to fractures.

Manifestations

Most people with Paget's disease are asymptomatic for years and the disease often is discovered when typical changes are seen on

an incidental x-ray. Manifestations are often vague and depend on the specific area involved (see the box below). The most common manifestation is localised pain of the long bones, spine, pelvis and cranium. The pain is described as a mild to moderate deep ache that is aggravated by pressure and weight bearing. It is more noticeable at night or when the person is resting. The pain usually is due to metabolic bone activity, secondary degenerative osteoarthritis, fractures or nerve impingement. Because of the increase in blood flow to pagetic bone, flushing and warmth of the overlying skin may be apparent.

MANIFESTATIONS Paget's disease

MUSCULOSKELETAL EFFECTS
- Pain (in the long bones of lower extremities or joints)
- Deformity (enlargement of skull, bowing of lower extremities and deformity of elbows and knees)
- Fractures of lower extremities
- Pathological fractures (especially of the tibia)
- Compression fractures
- Collapse of the vertebrae, resulting in kyphosis and loss of height
- Muscle weakness

NEUROLOGICAL EFFECTS
- Hearing loss
- Spinal cord injuries
- Dementia
- Pain from spinal stenosis
- Bladder and/or bowel dysfunction

CARDIOVASCULAR EFFECTS
- Congestive heart failure

METABOLIC EFFECTS
- Symptoms of hypercalcaemia in immobilised people
- Hypercalciuria and renal calculi
- Increased skin temperature over affected extremities

Complications

Complications of Paget's disease are as follows:
- nerve palsy syndromes from involvement of the upper extremities
- pathological fractures from loss of bone structure
- mental deterioration from compression of the brain when the skull is involved
- compression of the spinal cord from affected cervical vertebrae causing quadriplegia
- cardiovascular disease, resulting from vasodilation of the vessels in the skin and subcutaneous tissues overlying the affected bones
- osteogenic sarcoma, seen in 5–10% of people with severe disease (Porth & Matfin, 2014).

INTERPROFESSIONAL CARE

Care of the person with Paget's disease focuses on relieving pain, suppressing bone cell activity if necessary and preventing or minimising the effects of complications. Many people with Paget's disease are asymptomatic and do not require treatment. For more severely affected people, pharmacological agents are usually effective. Occasionally, surgery may be required.

Diagnosis

Many of the diagnostic tests that are useful for the diagnosis of osteoporosis are equally useful for people with Paget's disease (see Table 39.1). These include x-rays and bone scans to illustrate localised areas of demineralisation in the early stages, seen as 'punched-out' areas that lend a coarse, irregular appearance to the bone. In the later phase, x-rays show enlargement of the bones, tiny cracks in the long bones, and/or bowing of the weight-bearing bones. Computed tomography (CT) scans and magnetic resonance imaging (MRI) help identify possible causes of pain, including degenerative problems, spinal stenosis or nerve root impingement. Diagnostic tests are described in Chapter 37.

Laboratory tests used in diagnosis include a plasma alkaline phosphatase, which will show a steady rise as the disease progresses; the normal level (30 to 115 international units (IU)/L) may be elevated from high normal to more than 3000 international units/L. A urinary collagen pyridinoline test is a sensitive indicator of the rate of bone resorption. Liver function tests, isotope bone scans and vitamin D levels are also useful diagnostic tools (Walsh, 2012).

Medications

People who have mild symptoms often find relief using aspirin or NSAIDs such as ibuprofen and indomethacin. People who are experiencing manifestations and whose diagnostic test results are elevated are usually treated with an agent that retards bone resorption, such as calcitonin or a bisphosphonate.

Bisphosphonates such as alendronate, pamidronate and risedronate are the primary treatments used for severe Paget's disease. These drugs inhibit bone resorption, possibly by attaching to the surface of the calcium/phosphate phase of bone and inhibiting osteoclast activity. They are safe and usually are well tolerated by the person. Alendronate is available as an oral preparation and pamidronate is available for intravenous administration. Oral preparations are poorly absorbed from the GI tract and may cause gastric or oesophageal irritation. Alendronate should be given with a full glass of water on an empty stomach, at least 30 minutes before other medications or food. Pamidronate is given as an intravenous infusion in 5% dextrose or 0.9% sodium chloride. It is given for 3 successive days, generally promoting a rapid response with reduced urinary excretion of hydroxyproline and pyridinium and a fall in alkaline phosphatase. Intravenous pamidronate may cause flu-like symptoms, but these generally are brief. Calcium supplements also are prescribed for people receiving bisphosphonates. After bisphosphonate treatment, people often experience remission of symptoms for a year or more. See the 'Medication administration' box below for nursing implications.

Calcitonin inhibits osteoclastic resorption of bone and is secreted by the thyroid gland. It also works as an analgesic for bone pain. The two derivatives of this medication are salmon (fish) and human. Salmon calcitonin (Calcimar) is generally preferred because it is inexpensive and widely available. Human calcitonin (Cibacalcin) is derived from human thyroid glands, which makes

MEDICATION ADMINISTRATION The person with Paget's disease

BISPHOSPHONATES

Alendronate
Etidronate
Pamidronate
Risedronate
Tiludronate

The bisphosphonates inhibit bone resorption, increasing the mineral density of bones and reducing the incidence of fractures. They are also used in both the prevention and treatment of osteoporosis. When used for Paget's disease, bisphosphonates slow the accelerated bone turnover associated with this disease. Bone pain is relieved and the incidence of pathological fractures is reduced. Cardiac and vascular manifestations of the disease also improve.

Nursing responsibilities

- Administer alendronate with water 30 minutes before food or other medications due to its common side effect of nausea.
- Do not give foods high in calcium, vitamins with mineral supplements or antacids within 2 hours of administering alendronate as this may interfere with the absorption of the drug.
- Instruct the person to avoid lying down for 30 minutes after taking the drug as alendronate causes GI tract irritation and to facilitate delivery of the dose to the stomach (Bryant & Knights, 2014).
- Assess renal function studies before initiating therapy; alendronate is not recommended for use in people with renal insufficiency.
- Dilute the prescribed dose of pamidronate in 1000 mL of 5% dextrose or 0.9% sodium chloride; infuse over at least 4 hours. Do not add to calcium-containing solutions such as Hartmann's solution.
- Monitor the IV site for signs of thrombophlebitis.
- Assess the person for signs of electrolyte imbalance or other adverse responses such as a drug fever.

Health education for the person and family

- Take the medication as directed with clear water only. Consuming other beverages or food within 30 minutes of taking alendronate may interfere with its absorption and effectiveness.
- Do not lie down until after the person has eaten. Alendronate can irritate the oesophagus.
- Report symptoms such as new or worsening heartburn, difficulty swallowing or painful swallowing.
- Fever with or without chills may occur while receiving intravenous pamidronate; this will subside without treatment. Flu-like symptoms also may occur; these will subside within a week or so.
- Report any abnormal symptoms such as tingling around the mouth or numbness and tingling of the fingers or toes, which may indicate an imbalance of electrolytes in the blood.
- Take calcium and vitamin D supplements as instructed by the person's primary care provider.
- Response to these medications is gradual and continues for months after the drug is stopped.

it more expensive and difficult to obtain. The parenteral form of calcitonin is used in treating Paget's disease. (See the 'Medication administration' box on p. 1427 for nursing implications.)

Surgery

Different surgical interventions may be used to treat people with Paget's disease, such as repairing a complete fracture through pagetic bone, realigning a knee through tibial osteotomy to decrease pain or replacing a hip and/or knee for osteoarthritis. Because increased bleeding is a manifestation of Paget's disease, it is important to administer a potent bisphosphonate prior to surgery to decrease hypervascularity and reduce the risk of increased operative blood loss (Paget Foundation, 2015; Walsh, 2012).

Nursing care

Nursing diagnoses and interventions

The nursing interventions for the person with symptomatic Paget's disease focus on pain control, prevention of injury or fractures, and education regarding the disease process and prescribed therapies.

Chronic pain

The most common manifestation of Paget's disease is bone pain. This usually is the manifestation that prompts the person to seek healthcare.

- Assess the location and extent of the pain to determine the bone areas involved. *Bone pain in Paget's disease is poorly localised and is frequently described as 'aching and deep'.*
- Teach the person to take NSAIDs or aspirin on a regular basis as prescribed. *Pain is most noticeable at night or when the person is resting. The pain can become evident when it is aggravated by pressure and weight bearing.*
- Ensure correct placement of prescribed brace or corset. *The person may be required to wear a light brace or corset to relieve back pain and provide support when assuming an upright position. The person may need instruction in the correct application of the device and in the evaluation of pressure areas that may result from wearing the device.*
- Suggest referral for heat therapy and massage. *Heat therapy and massage can alleviate mild discomfort. Care should be taken when applying massage over areas prone to pathological fractures.*

Impaired physical mobility

People with Paget's disease need to maintain or improve mobility so that they can perform necessary self-care activities and prevent complications of immobility.

- Provide an assistive device for use when ambulating. *During the active phase of Paget's disease, the person is prone to fractures. Bone deformities, activity intolerance, fear of falling and pain are all factors that may make the person more prone to falls. An assistive device can provide both physical and psychological support during ambulation, permit the person to ambulate further and provide a device for resting during ambulation.*

CONSIDERATION FOR PRACTICE

Activities as seemingly simple as lifting a heavy box may result in a fracture in the person with Paget's disease.

- Teach good body mechanics. *The person with bone deformities should avoid activities that require lifting and twisting.*
- Reinforce information about exercise protocols and activity regimens. *Exercise and activity protocols should be planned carefully to prevent injury and to minimise fatigue.*

Community-based care

A diagnosis of Paget's disease can be frightening for the person and family. It is important that they understand that this is a treatable disease and that many manifestations of the disease will be relieved with treatment. Inform the person that remissions of the disease often last for a year or more after effective treatment. The Paget Foundation should be suggested as a resource. Discuss the following topics:

- the importance of following the prescribed treatment regimen and keeping scheduled follow-up appointments
- because it may take several weeks to notice a response to treatment, the importance of continuing therapy during this time and after a response is obtained
- if bisphosphonates such as alendronate or pamidronate are ordered, the importance of taking supplemental calcium to prevent low blood calcium levels
- the importance of remaining active
- safety in the home and outdoor environment to prevent falls
- the need to report to the primary care provider any sudden pain or disability, even if no trauma has occurred, because pathological fractures are possible.

THE PERSON WITH GOUT

Gout is a metabolic disease that occurs from an inflammatory response to the production or excretion of uric acid resulting in high levels of uric acid in the blood (hyperuricaemia) and in other body fluids, including synovial fluid. The disorder is characterised by deposits of urates (insoluble precipitates) in the connective tissues of the body. Gout has an acute onset, usually at night, and often involves the first metatarsophalangeal joint (great toe). The initial acute attack is usually followed by a period of months or years without manifestations. As the disease progresses, urates are deposited in various other connective tissues. Deposits in the synovial fluids cause acute inflammation of the joint (*gouty arthritis*). Over time, urate deposits in subcutaneous tissues cause the formation of small white nodules (called **tophi**). Deposits of crystals in the kidneys can form urate kidney stones and result in kidney failure.

Gout may occur as either a primary or a secondary disorder. Primary (inherited) gout is characterised by elevated serum uric acid levels resulting from either an inborn error of purine metabolism or a decrease in renal uric acid excretion due to an unknown cause. Purines are part of the structure of the nuclear compounds DNA and RNA; they also may be synthesised by the body. Impaired uric acid excretion leads to hyperuricaemia in the majority of people with primary gout. In secondary gout, hyperuricaemia occurs as a result of another disorder or treatment with certain medications. Disorders associated with rapid cell turnover, such as some malignancies (leukaemia, in particular), haemolytic anaemia and polycythaemia, can increase purine metabolism. Chronic renal disease, hypertension, starvation and diabetic ketoacidosis can interfere with uric acid excretion, as can certain drugs, including some diuretics (such as frusemide, ethacrynic acid and chlorothiazide), pyrazinamide, cyclosporin, ethambutol and low-dose salicylates. Alcohol ingestion appears to interfere with uric acid excretion and to accelerate its synthesis.

Gout occurs more often in men, usually after the age of 40. In women, attacks of gout are rarely seen until after menopause. The incidence and prevalence of gout are increasing in Australia and New Zealand (Robinson, Taylor & Merriman, 2012). A number of risk factors for gout have been identified (see Box 39.2). Of these, male gender and ageing cannot be modified; the others can be. Consumption of a diet rich in meat and seafood is associated with a higher risk of developing gout, whereas total protein intake and consumption of purine-rich vegetables have not been shown to contribute to its development (Terkeltaub, 2009; Shulten et al., 2009). While alcohol intake has long been known to increase the risk of hyperuricaemia and gout, recent studies have shown that consumption of soft drinks sweetened with sugar or fructose (corn syrup) also increases the risk of gout in men. Obesity and metabolic syndrome (abdominal obesity, hyperlipidemia, hypertension and insulin resistance) are strongly correlated with hyperuricaemia and gout (Rothschild, Francis & Miller, 2015).

Pathophysiology

Uric acid is the breakdown product of purine metabolism. Normally, a balance exists between its production and its

BOX 39.2 Risk factors for gout

- Male gender
- Age
- Diet: higher consumption of meat and seafood
- Alcohol intake, beer in particular
- Consumption of sugar- or fructose-sweetened soft drinks
- Obesity
- Medications: diuretics, aspirin

excretion, with approximately two-thirds of the amount produced each day excreted by the kidneys and the rest in the faeces. The normal serum urate levels for men and women vary, the normal range being 2–7 mg/dL (120–420 μmol/L) for men and 2–6 mg/dL (120–360 μmol/L) for women, although these figures may differ between laboratories or populations. Hyperuricaemia is therefore defined as serum urate levels > 7 mg/dL (> 420 μmol/L) for men and > 6 mg/dL (> 360 μmol/L) for women. Higher levels of uric acid are associated with increased incidence and prevalence rates for gout. Men have higher concentrations of uric acid and therefore a higher prevalence of gout, whereas premenopausal women and children have lower concentrations and therefore a lower prevalence of gout (Perez-Ruiz & Herrero-Beites, 2014).

At levels greater than 7.0 mg/dL, the serum is saturated and monosodium urate crystals may form. It is not known exactly how crystals of monosodium urate crystals are deposited in joints. Several mechanisms may be involved:

- Crystals tend to form in peripheral tissues of the body, where lower temperatures reduce the solubility of the uric acid.
- A decrease in extracellular fluid pH and reduced plasma protein binding of urate crystals are evident.
- Tissue trauma and a rapid change in uric acid levels may also lead to crystal deposition. A rapid increase in uric acid may occur with tissue trauma and release of cellular components.

The monosodium urate crystals may form in the synovial fluid or in the synovial membrane, cartilage or other joint connective tissues. They may also form in the heart, earlobes and kidneys. These crystals stimulate and continue the inflammatory process, during which neutrophils respond by ingesting the crystals. The neutrophils release their phagolysosomes, causing tissue damage, which perpetuates the inflammation.

Manifestations

The manifestations of gout are hyperuricaemia, recurrent attacks of inflammation of a single joint, tophi, kidney disease and kidney stones. Unless treated, the manifestations of gout appear in three stages: asymptomatic hyperuricaemia, acute gouty arthritis and tophaceous gout (see the 'Manifestations' box below).

MANIFESTATIONS Gout

ACUTE GOUTY ARTHRITIS
- Usually monoarticular, affecting metatarsophalangeal joint of great toe, instep, ankle, knee, wrist or elbow
- Acute pain
- Red, hot, swollen and tender joint
- Fever, chills, malaise
- Elevated WBC and erythrocyte sedimentation rate

TOPHACEOUS (CHRONIC) GOUT
- Tophi evident on joints, bursae, tendon sheaths, pressure points, helix of ear
- Joint stiffness, limited ROM and deformity
- Ulceration of tophi with chalky discharge

Asymptomatic hyperuricaemia

The first stage is asymptomatic hyperuricaemia, with serum levels averaging 9 to 10 mg/dL. Most people with hyperuricaemia do not progress to further stages of the disease.

Acute gouty arthritis

The second state is acute gouty arthritis. The acute attack (called a 'flare'), usually affecting a single joint, occurs unexpectedly, often beginning at night. It may be triggered by trauma, alcohol ingestion, dietary excess or a stressor such as surgery. It is often precipitated by an abrupt or sustained increase in uric acid levels. The affected joint becomes red, hot, swollen and exquisitely painful and tender.

Approximately 50% of initial attacks of acute gouty arthritis occur in the metatarsophalangeal joint of the great toe. Other sites for acute attacks include the instep of the foot, ankles, heels, knees, wrists, fingers and elbows. The pain, often intense, peaks within several hours and may be accompanied by fever and an elevated white blood cell (WBC) count and erythrocyte sedimentation rate (ESR). The affected joints are swollen and the skin over the joint is warm and dusky red.

Acute attacks of gouty arthritis last from several hours up to several weeks and typically subside spontaneously. There are no long-lasting sequelae and the person enters an asymptomatic period called the intercritical period. The intercritical period may last up to 10 years; however, approximately 60% of people experience a recurrent attack within 1 year. Successive attacks tend to last longer, occur with increasing frequency and resolve less completely than the initial attack.

Tophaceous (chronic) gout

Tophaceous (or chronic) gout occurs when hyperuricaemia is not treated. The urate pool expands and monosodium urate crystal deposits (tophi) develop in cartilage, synovial membranes, tendons and soft tissues. They are seen most often in the helix of the ear; in tissues surrounding joints and bursae (especially around the elbows and knees); along tendons of the fingers, toes, ankles and wrists; on ulnar surfaces of the forearms; along the shins of the legs; and on other pressure points. The skin over tophi may ulcerate, exuding chalky material containing inflammatory cells and urate crystals. Tophi can also develop in the tissues of the heart and spinal epidura. Although tophi themselves are not painful, they may restrict joint movement and cause pain and deformities of the affected joints. Tophi may also compress nerves and erode and drain through the skin.

Complications

Kidney disease may occur in people with untreated gout, particularly when hypertension is also present. Urate crystals are deposited in renal interstitial tissue. Uric acid crystals also form in the collecting tubules, renal pelvis and ureter, forming stones. The stones can range in size from a grain of sand to a massive structure filling the spaces of the kidney. Uric acid stones can potentially obstruct urine flow and lead to acute kidney failure.

INTERPROFESSIONAL CARE

The classic presentation of acute gouty arthritis is so distinctive that the diagnosis can often be based on the person's history

and physical examination. Treatment is directed towards terminating an acute attack, preventing recurrent attacks and reversing or preventing complications resulting from crystal deposition in tissues and formation of uric acid kidney stones.

Diagnosis

Diagnostic testing is performed to establish an accurate diagnosis and direct long-term therapy. Diagnostic tests are described in Chapter 37.

Serum uric acid is nearly always elevated (usually above 480 mmol/L). The WBC count shows significant elevation, reaching levels as high as 20 000/mm^3 during an acute attack. The ESR is elevated during an acute attack from the acute inflammatory process that accompanies deposits of urate crystals in a joint. In addition, a 24-hour urine specimen is analysed to determine uric acid production and excretion, and analysis of fluid aspirated from the acutely inflamed joint or material aspirated from a tophus shows typical needle-shaped urate crystals, providing the definitive diagnosis of gout.

Medications

Medications are used to terminate an acute attack, prevent further attacks and reduce serum uric acid levels to prevent long-term sequelae of the disease. It is important to treat the acute attack of gouty arthritis before initiating treatment to reduce serum uric acid levels, because an abrupt decrease in serum uric acid may lead to further acute manifestations. Pharmacological therapy is a mainstay of treatment in achieving these goals.

ACUTE ATTACK NSAIDs and colchinine are the treatment of choice for an acute attack of gout. Colchinine has known anti-inflammatory effects but no analgesic properties. That is why it is combined with an NSAID for analgesic effects. Indomethacin (Indocin) is the most frequently used NSAID for gout, although others are equally effective. Other NSAIDs that may be prescribed include ibuprofen (Nurofen), naproxen (Naprosyn, Anaprox), diclofenac (Voltaren) or ketorolac. Although extremely effective, NSAIDs are contraindicated for people with active peptic ulcer disease, impaired kidney function or a history of hypersensitivity reactions to the drugs. As with other anti-inflammatory drugs, people should be aware of possible risks and follow recommended doses carefully.

Colchicine can dramatically affect the course of an acute attack. Joint pain begins to diminish within 12 hours of the initiation of treatment and disappears within 2 days. Colchicine apparently acts by interrupting the cycle of urate crystal deposition and inflammation in an acute attack of gout. It has no anti-inflammatory effect in other forms of arthritis and its use is limited to gout. The use of colchicine is limited by significant side effects. When administered orally, many people develop abdominal cramping, diarrhoea, nausea or vomiting. Intravenous administration is limited by potential toxic effects, including local pain, tissue damage if extravasation occurs during injection, bone marrow suppression and disseminated intravascular coagulation (DIC). It is contraindicated for people who have significant gastrointestinal, kidney, hepatic or cardiac disease.

Corticosteroids may also be prescribed for the person with acute gouty arthritis. If possible, the intra-articular route is preferred for monoarticular arthritis to avoid the multiple systemic effects of steroid therapy. When gout is polyarticular, corticosteroids may be administered either orally or intravenously.

Analgesics may also be prescribed during an acute episode of gouty arthritis. Either codeine or pethidine may be administered to manage the person's pain. Aspirin is avoided because it may interfere with uric acid excretion.

PROPHYLACTIC THERAPY In people at high risk of future attacks of acute gout, prophylactic therapy with daily colchicine may be initiated. Prophylaxis is particularly useful during the first 1 to 2 years of treatment with antihyperuricaemic agents. Although colchicine does not affect the serum uric acid directly, it reduces the frequency of attacks by preventing crystal deposition within the joint. The doses required to achieve this effect are small and few side effects are associated with therapy.

Treatment to reduce serum uric acid levels is typically initiated for people with recurring gout, tophi or renal damage. Asymptomatic hyperuricaemic people require no treatment. Uricosuric agents are used for people who do not eliminate uric acid adequately; allopurinol is prescribed for people who produce excessive amounts of uric acid. Uricosuric drugs block the tubular reabsorption of uric acid, promoting its excretion and reducing serum levels. These drugs reduce the frequency of acute attacks, particularly when administered with colchicine. Probenecid lowers the serum concentration of uric acid by competitively inhibiting the reabsorption of urate at the proximal renal tubule, thus increasing urinary excretion of uric acid (Bryant & Knights, 2014).

Allopurinol (Zyloprim) is a xanthine oxidase inhibitor that lowers plasma uric acid levels and facilitates the mobilisation of tophi. Because of its effectiveness in lowering serum uric acid levels, it may trigger an attack of acute gout. The nursing implications for medications used to treat gout are included in the 'Medication administration' box on next page.

Complementary and alternative therapy

A variety of nutritional and herbal supplements may be used to help prevent gout or decrease the onset of manifestations. These include:

- Vitamin E and selenium may decrease tissue inflammation.
- Amino acids (alanine, aspartic acid, glutamic acid and glycine) increase the ability of the kidneys to excrete uric acid.
- Dark reddish-blue berries (such as cherries and blackberries) are good sources of flavonoids, which help lower uric acid levels, decrease inflammation and prevent or repair joint tissue damage.
- Acupuncture can provide pain relief.

Treatments

Treatments for gout, in addition to medications, include dietary management and rest.

NUTRITION Dietary purines contribute only slightly to uric acid levels in the body and no specific diet may be recommended. If a low-purine diet is recommended, the person should be taught that high-purine foods include all meats and seafood, yeast, beans, peas, lentils, oatmeal, spinach, asparagus, cauliflower and mushrooms. The obese person is advised to lose weight, but fasting is contraindicated for people with gout. Alcohol intake and specific foods that tend to precipitate attacks are avoided.

MEDICATION ADMINISTRATION The person with gout

COLCHICINE

Colchicine is used to terminate an acute attack of gouty arthritis and to prevent recurrent episodes of the disease. Colchicine does not alter serum uric acid levels, but appears to interrupt the cycle of urate crystal deposition and inflammatory response. It may be administered either by mouth or intravenously. Colchicine is also available as a fixed-dose combination with a uricosuric agent, probenecid. Only plain colchicine is used to treat an acute attack of gout; combination therapy is employed to prevent further attacks.

Nursing responsibilities

- Assess for possible contraindications to colchicine therapy, including serious gastrointestinal, renal, hepatic or cardiac disease.
- Administer the following as ordered:
  - *Intravenous doses:* give undiluted or diluted in up to 20 mL sterile 0.9% sodium chloride for injection. Administer over a period of 2 to 5 minutes.
  - *Oral doses:* give on an empty stomach to facilitate absorption.
- Evaluate for adverse effects, including abdominal cramping, nausea, vomiting and diarrhoea, and report promptly, because these side effects may necessitate discontinuation of the drug.

Health education for the person and family

- Drink 3 to 4 L of liquid per day.
- Report adverse responses, including gastrointestinal problems, fatigue, bleeding, easy bruising or recurrent infections, to the healthcare provider.
- Do not drink alcohol.

URICOSURIC DRUGS

Probenecid (Benemid)
Sulfinpyrazone (Anturane)

Probenecid inhibits the tubular reabsorption of urate, promoting the excretion of uric acid and decreasing serum uric acid levels. Sulfinpyrazone potentiates the renal excretion of uric acid, reducing serum uric acid levels. It is used to prevent recurrent attacks of acute gouty arthritis and to treat chronic gout.

Nursing responsibilities

- Assess for prior hypersensitivity responses to this drug.
- Administer after meals or with milk to minimise gastric distress.
- Increase fluid intake to at least 3 L/day to prevent the formation of uric acid kidney calculi.
- Administer sodium bicarbonate or potassium citrate as ordered to maintain an alkaline urine.
- Do not administer aspirin to people receiving probenecid because salicylates interfere with the action of the drug.
- Monitor people receiving the following drugs concurrently with probenecid for increased or toxic effects: penicillin and related antibiotics, indomethacin, paracetamol, naproxen, ketoprofen, lorazepam and rifampin.
- Monitor for possible adverse effects of probenecid, including headache, dizziness, hepatic necrosis, nausea and vomiting, renal colic, bone marrow depression, anaphylaxis, fever, hives and pruritus.
- Administer sulfinpyrazone with meals or antacid to minimise gastric distress.
- Monitor people taking sulfinpyrazone with other sulfa drugs for increased or toxic effects; monitor for hypoglycaemia when receiving insulin or oral hypoglycaemics concurrently; and monitor for bleeding or increased anticoagulant effect when receiving warfarin concurrently.
- Assess for contraindications to therapy with sulfinpyrazone, including active peptic ulcer disease, a history of hypersensitivity to phenylbutazone or other pyrazoles or blood dyscrasias.

Health education for the person and family

- Do not take aspirin or products containing aspirin while taking probenecid. Use paracetamol for relief of mild pain.
- Drink at least 3 L of fluids per day to minimise the risk of kidney stone formation.
- Take sulfinpyrazone with meals to minimise gastric distress and report epigastric pain, nausea or black stools to the healthcare provider promptly.

ALLOPURINOL (ZYLOPRIM)

Allopurinol acts on purine metabolism, reducing the production of uric acid and decreasing serum and urinary concentrations of uric acid. It is used for people with manifestations of primary or secondary gout, including acute attacks, tophi, joint destruction, urinary stones and nephropathy. It is not indicated for use in the treatment of asymptomatic hyperuricaemia.

Nursing responsibilities

- Monitor intake and output and increase fluid intake to approximately 3 L/day.
- Monitor for desired effect of decreased serum uric acid levels and for adverse effects such as nausea, diarrhoea and rash.
- Assess blood urea nitrogen (BUN) and creatinine levels prior to the initiation of and during treatment with allopurinol. Report signs of impaired kidney function such as an elevated BUN and creatinine, decreased urine output, and dilute or frothy urine to the healthcare provider.
- Administer with meals to minimise gastric distress.
- Monitor FBC periodically because allopurinol therapy may cause bone marrow depression.
- In people receiving warfarin concurrently, monitor prothrombin times and be alert to evidence of bleeding, because allopurinol prolongs the half life of warfarin.
- Monitor people receiving chlorpropamide, cyclophosphamide, hydantoin, theophylline, vidarabine or ACE inhibitors concurrently for increased drug effects.
- Discontinue the drug and notify the healthcare provider immediately if the person develops a rash. Rash and hypersensitivity responses occur more frequently in people receiving ampicillin, amoxicillin or thiazide diuretics.

Health education for the person and family

- Stop taking the drug and report any skin rash, painful urination, blood in the urine, eye irritation or swelling of the lips or mouth to the healthcare provider immediately.
- Take the medication after meals to minimise gastric distress.
- Drink 3 to 4 L of fluid daily to maintain a urinary output greater than 2 L/day.
- Acute gouty attacks may occur during the initial stages of allopurinol therapy; continue therapy prescribed for attacks (such as colchicine) to minimise acute episodes.
- Do not take a double dose of medication if you miss a dose.

A liberal fluid intake to maintain a daily urinary output of 2000 mL or more is recommended to increase urate excretion and reduce the risk of urinary stone formation. Urinary alkalinising agents, such as sodium bicarbonate or potassium citrate, may be prescribed as well to minimise the risk of uric acid stones. It is important to monitor people receiving these preparations carefully for signs of fluid and electrolyte or acid–base imbalances.

REST During an acute attack of gouty arthritis, bed rest is prescribed. It is continued for approximately 24 hours after the attack has subsided because early ambulation may bring about recurrence of acute manifestations (McPhee, Papadakis & Tierney, 2015). The affected joint may be elevated, and hot or cold compresses may be applied, for comfort.

Nursing care

People with gout provide self-care at home. Teaching focuses on self-management of pain and altered mobility.

Nursing diagnoses and interventions

Pain is a primary focus for nursing interventions in the person experiencing an acute attack of gout. The person's mobility is also impaired during an acute attack, because of both pain and prescribed activity limitations.

Acute pain

The pain associated with an attack of acute gouty arthritis is intense and accompanied by exquisite tenderness of the affected joint. Measures to alleviate the pain are vital in the initial period until anti-inflammatory medications become effective and the acute inflammatory response is relieved. The following are important in teaching about pain relief:

- Position the affected joint for comfort. Elevate the joint or extremity (usually the foot) on a pillow, maintaining alignment. *Elevation and normal body alignment facilitate blood return from the affected joint, alleviating some of the oedema.*
- Protect the affected joint from pressure, placing a foot cradle on the bed to keep bed covers off the foot. *A foot cradle keeps bed linen from applying pressure on the affected joint.*

CONSIDERATION FOR PRACTICE

The affected joints are so painful that even the weight of a sheet can be unbearable.

- Take anti-inflammatory and antigout medications as prescribed. In the initial period, colchicine may be given hourly. *These medications reduce the acute inflammatory response, gradually relieving discomfort.*
- Take analgesics as prescribed. *Supplemental analgesia may be necessary in the acute period until the inflammatory response is mediated.*
- Maintain bed rest. *It is important to immobilise the affected joint and promote rest to prevent exacerbation of joint inflammation.*

Community-based care

Discuss the following topics with the person:

- The disease and its manifestations. Tell the person that initial attacks cause no permanent damage but that recurrent attacks can lead to permanent damage and joint destruction. Discuss other potential effects of continued hyperuricaemia, including tophaceous deposits in subcutaneous and other connective tissues. Discuss the potential for kidney damage and kidney stones.
- The rationale for and use of prescribed medication. Stress the need to continue the medication until the healthcare provider discontinues it, even though the person is free of manifestations of gout. Tell the person to avoid drugs that increase uric acid blood levels: hydrochlorothiazide (HydroDiuril), cyclosporin (Neoral, Sandimmune), frusemide (Lasix) and high doses of aspirin. People who need to reduce their risk of heart attacks may safely take one baby aspirin each day.
- The importance of a high intake of fluids each day and avoiding the use of alcohol.

THE PERSON WITH OSTEOMALACIA

Osteomalacia, often referred to as *adult rickets*, is a metabolic bone disorder characterised by inadequate or delayed mineralisation of bone matrix in mature compact and spongy bone, resulting in softening of bones. Bone mineralisation requires adequate calcium and phosphate ions in extracellular fluid. When either of these ions is insufficient due to (1) inadequate calcium intake or decreased calcium absorption from the intestines because of insufficient vitamin D, or (2) increased renal losses or decreased intestinal absorption of phosphate, the bone matrix is not mineralised and cannot sustain weight bearing. Marked deformities of weight-bearing bone and pathological fractures occur. The primary causes of osteomalacia are vitamin D deficiency and hypophosphataemia. Osteomalacia can be corrected with treatment.

Osteomalacia has been almost non-existent in Australia and New Zealand because many foods are fortified with vitamin D, but its incidence is increasing among older adults and people who adhere to strict vegetarian diets. It is a significant health problem in cultures whose diets tend to be deficient in calcium and vitamin D. Women in northern China, Japan and northern India have a higher incidence of the disorder (Porth & Matfin, 2014).

The main risk factors for vitamin D deficiency are a diet low in vitamin D, decreased endogenous production of vitamin D because of inadequate sun exposure, impaired intestinal absorption of fats (vitamin D is a fat-soluble vitamin) and disorders that interfere with the metabolism of vitamin D to its active forms. Gastrectomy and small-bowel disorders may reduce the

absorptive surface of the bowel to the extent that nutrients are not completely or adequately absorbed. Both vitamin D and calcium absorption may be affected. Hepatobiliary disorders that interfere with bile production and release and chronic pancreatic insufficiency with inadequate pancreatic enzyme production can affect the absorption of fats and vitamin D from the bowel. Once absorbed, vitamin D is metabolised in the liver and the kidney to its active form; therefore, liver disorders such as cirrhosis and kidney disorders can affect this activation. Certain drugs, such as isoniazid, rifampicin and anticonvulsants, accelerate vitamin D metabolism, resulting in less availability to the tissues. Renal excretion of vitamin D is increased in some kidney disorders such as nephrotic syndrome. (See Box 39.3.)

Hypophosphataemia can be the result of insufficient dietary intake, excessive losses through the urine or stool, or a shift into the cells. Alcohol abuse is the most common cause of hypophosphataemia because of related dietary deficiencies, vomiting, antacid use and increased renal excretion of phosphate. Ingesting large amounts of non-absorbable antacids causes increased phosphate losses in the stool. Several acquired and genetic disorders cause increased losses of phosphate in the urine.

Pathophysiology

The two main causes of osteomalacia are insufficient calcium absorption in the intestine due to a lack of calcium or resistance to the action of vitamin D, and increased losses of phosphorus through the urine (Porth & Matfin, 2014). In its natural form, vitamin D is obtained from certain foods and ultraviolet radiation of the sun. Vitamin D maintains adequate serum levels of calcium and phosphate for normal mineralisation of the bone. Vitamin D deficiency, or resistance to its action, disrupts the normal mineralisation of the bone, causing softening of the bone.

Vitamin D is inactive when it is absorbed from the intestine or synthesised from exposure to ultraviolet light. For vitamin D to become active, a two-step process must occur: (1) vitamin D (and its metabolites) is transported in the blood to the liver, where it is converted to calcidiol, and (2) calcidiol is then transported to the kidney and transformed to an active form, calcitriol.

The active form of vitamin D is needed for optimal absorption of calcium and phosphorus from the intestine. Calcium and phosphorus are transported in the blood to the bones for normal mineralisation. If there is a lack of vitamin D, calcium and phosphorus are not absorbed from the intestine and serum calcium and phosphorus levels therefore fall. A deficiency in these minerals in turn activates the parathyroid glands, with loss of calcium and phosphorus from bone. The continued loss of calcium and phosphate in the bone disrupts bone mineralisation.

Impaired bone mineralisation causes abnormalities in both spongy and compact bone. The osteoid (the soft, non-calcified part of the matrix) continues to be produced but is not mineralised. This abnormal build-up of demineralised bone leads to gross deformities of the long bones, spine, pelvis and skull, because the bone is soft and unable to bear the weight and stress of body movement.

Manifestations

The manifestations of osteomalacia include bone pain and tenderness (see the box below). As the disease progresses, fractures occur. In contrast to osteoporosis, osteomalacia is not associated with a significant occurrence of hip fractures. Instead, pathological fractures occur in the commonly weakened areas (e.g. distal radius and proximal femur).

MANIFESTATIONS Osteomalacia

- Bone pain: may be vague and generalised at first, becoming more intense with activity as the disease progresses; occurs most frequently in the pelvis, long bones of the extremities, spine and ribs.
- Difficulty changing from lying to sitting position and sitting to standing position.
- Muscle weakness: frequently an early sign in severe cases.
- Waddling gait: may be due to pain and muscle weakness.
- Dorsal kyphosis: may occur in severe cases.
- Pathological fractures.

BOX 39.3 Causes of osteomalacia

Vitamin D deficiency
- Inadequate dietary intake
- Lack of sun exposure
- Malabsorption from intestines: gastrectomy, small-bowel disorders, gallbladder disease, chronic pancreatic insufficiency
- Kidney or liver disorders
- Drug effects: isoniazid, rifampicin, anticonvulsants

Phosphate depletion
- Inadequate intake
- Impaired absorption due to chronic antacid use
- Impaired renal tubular reabsorption due to either acquired or genetic disorders

Systemic acidosis
- Renal tubular acidosis
- Ureterosigmoidostomy
- Fanconi's syndrome

Mineralisation inhibitors
- Hypophosphatasia
- Sodium fluoride or disodium etidronate (Didronel)
- Aluminium intoxication

Chronic kidney failure
Calcium malabsorption

INTERPROFESSIONAL CARE

Osteomalacia may be difficult to differentiate from osteoporosis because the manifestations are very similar; however, once the specific cause is determined, appropriate therapy will correct the disorder.

Diagnosis

A history of inadequate dietary intake, kidney failure or some malabsorption states may suggest osteomalacia. Diagnostic tests are described in Chapter 37. Table 39.1 compares the diagnostic findings of osteomalacia with those of osteoporosis and Paget's disease. X-rays demonstrate the effects of generalised bone demineralisation: trabecular bone loss, cyst formation, compression fractures, bowing and bending deformities of the long bones, and osteoid deposits, particularly in the vertebral bodies and pelvis.

Laboratory tests include serum calcium, parathyroid hormone and alkaline phosphatase levels. Calcium may be normal or low, depending on the cause of the disease. Calcium levels may be reduced when calcium absorption is impaired or in severe vitamin D deficiency. Secondary hypoparathyroidism may shift calcium from the bone into extracellular fluid, maintaining a normal serum calcium level. Parathyroid hormone is frequently elevated as a compensatory response to hypocalcaemia in kidney failure or vitamin D deficiency. Alkaline phosphatase is usually elevated.

Medications

Therapeutic management of osteomalacia depends on the cause of the disease. Because the causes are so diverse, it is difficult to generalise treatment. Most people are placed on vitamin D therapy. Calcium and phosphate supplements may be indicated. Radiological evidence of healing often is apparent within weeks of initiating therapy.

Nursing care

Managing the person with osteomalacia includes assessing the person's current dietary intake of vitamin D, calcium and phosphorus, and exposure to ultraviolet light. It also includes managing the person's responses to bone pain and tenderness, fractures and muscle weakness.

Community-based care

Teaching is important not only for the person with osteomalacia, but also for people at risk of developing the disease. When milk and other dairy products began to be fortified with vitamin D, the incidence of childhood rickets decreased dramatically. Now many people are unaware of the importance of vitamin D, calcium and phosphorus to bone health.

Older adults as a group are at high risk of osteomalacia because of dietary deficiencies, age-related intestinal malabsorption and possible physical mobility limitations that restrict their exposure to sunlight. Teach older adults about the importance of maintaining an adequate intake of milk and other dairy products that are not only rich in calcium and phosphorus, but also are fortified with vitamin D. Few other food sources provide enough vitamin D to meet recommended levels. Cod liver oil may be used as a supplement because it contains significant amounts of vitamin D. Supplements are not recommended, however, for people who get adequate vitamin D through dietary sources and sun exposure, because this fat-soluble vitamin may become toxic at high levels. Instruct people who are taking supplements to report to their healthcare provider symptoms such as anorexia, nausea and vomiting, frequent urination, muscle weakness and constipation that may be indicative of hypervitaminosis D.

Teach the person with osteomalacia about safety measures to prevent falls. Discuss the importance of eliminating scatter rugs and clutter from living areas to prevent tripping. Teach the person to place a night-light in hallways and the bathroom to prevent falls associated with night-time toileting. Suggest installing grab bars in the shower and tub and next to the toilet for safety.

Teach people with bone pain and muscle weakness to use assistive devices such as walkers, canes or crutches when ambulating. Provide referrals to physical therapy for teaching people how to safely use these devices. Encourage people to participate in a supervised exercise program such as water aerobics or tai chi to improve muscle strength and balance.

DEGENERATIVE DISORDERS

Degenerative disorders, especially degenerative joint disease, are the most common forms of arthritis in the older adult. Both primary and secondary forms are seen in adults of all ages. Primary or idiopathic osteoarthritis, the most common type, occurs without a clear precipitating factor. Secondary osteoarthritis is associated with an identifiable cause. For instance, it may be related to trauma to a joint, inflammation, skeletal disorders such as congenital hip dysplasia or metabolic disorders. Regardless of cause, degenerative disorders of the joints and muscles can lead to impaired mobility and chronic pain. These problems may in turn cause disability, especially in the performance of ADLs by older adults.

THE PERSON WITH OSTEOARTHRITIS

Osteoarthritis (OA) (also labelled *degenerative joint disease*) is the most commonly occurring of all forms of arthritis and a leading cause of pain and disability in older adults (Porth & Matfin, 2014). This disease is characterised by loss of articular cartilage in articulating joints and hypertrophy of the bones at the articular margins. OA may be idiopathic (without known

FOCUS ON CULTURAL DIVERSITY
The person with osteoarthritis

- Indigenous Australians are more likely than other Australians to report having arthritis, but are much less likely to have hip or knee replacements.
- People in the most disadvantaged areas of Australia are less likely than those in the least disadvantaged areas to have a total hip replacement, but more likely to have a total knee replacement.
- People living in regional and remote areas are more likely to have hip or knee replacements than those living in major cities.

Source: Australian Institute of Health and Welfare (AIHW) (2015).

cause) or secondary (associated with known risk factors). OA affects more than 1.8 million Australians (over 8% of the total population) over the age of 45 years. Men are affected more than women at an earlier age, but the rate of OA in women exceeds men by the middle adult years. The joints most affected are in the hand, wrist, neck, lower back, hip, knee, ankle and feet. Men are more likely than women to have hip OA, whereas postmenopausal women more often have hand OA. In 2012–2013, 20% of Indigenous people reported having a musculoskeletal disease. The prevalence of long-term musculoskeletal diseases for Indigenous Australians was 1.1 times as high as for non-Indigenous Australians (AIHW, 2015).

The cultural and ethnic effects on the development of OA are outlined in the box above.

Localised OA affects only one or two joints. Generalised OA affects three or more joints. Generalised OA may also be classified as nodal (involving the hand) or non-nodal (no hand involvement). Nodal OA may also affect the knees, hips, cervical spine and lumbar spine. Idiopathic OA most commonly affects the terminal interphalangeal joints (*Heberden's nodes*) and less often the proximal interphalangeal joints (*Bouchard's nodes*) (see Figure 39.2), the joints of the thumb, the hip, the knee, the metatarsophalangeal joint of the big toe and the cervical and lumbar spine. Secondary OA may occur in any joint from an articular injury.

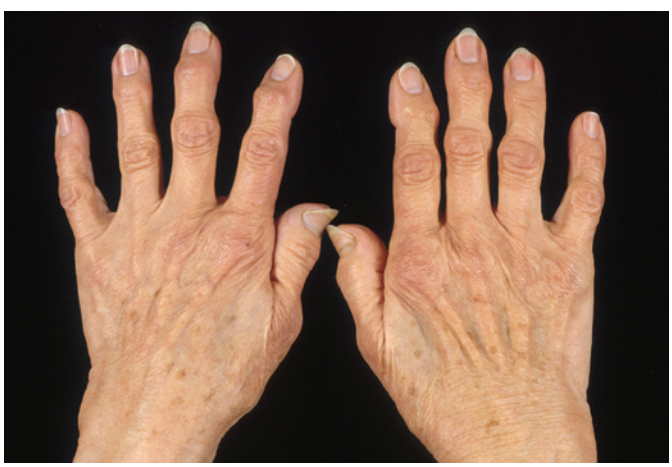


FIGURE 39.2 ■ Typical interphalangeal joint changes associated with osteoarthritis

Source: © L. Samsuri/Custom Medical Stock Photo, Inc.

Risk factors

Idiopathic OA is associated with increasing age. It has been suggested that OA may be inherited as an autosomal recessive trait, with genetic defects causing premature destruction of the joint cartilage. The causes of secondary OA include trauma, mechanical stress and inflammation of joint structures, joint instability, neurological disorders, endocrine disorders and selected medications.

Excessive weight contributes to the development of OA, especially in the hip, ankle and knee. Excess fat may have a direct metabolic effect in the development of the disease. Primary OA of the knee is almost four times more common in obese women and five times more common in obese men (AIHW, 2015). Inactivity is another risk factor. Moderate recreational exercise has been shown to decrease both the chance of developing OA and the progression of manifestations when OA is present. People involved in strenuous, repetitive exercise (such as participating in sports) have an increased risk of developing secondary OA.

Other risk factors that are linked to OA are hormonal factors such as decreased oestrogen in menopausal women, excessive growth hormone and increased parathyroid hormone.

Pathophysiology

The cartilage that lines joints provides a smooth surface, so that the bones of the joint glide over one another without friction, and it distributes the load from one bone to the next, dissipating the mechanical stress that occurs with joint loading. This cartilage normally contains more than 70% water. More than 90% of its dry weight is collagen, which provides strength, and proteoglycans, which provide elasticity and stiffness to compression. Cartilage cells, the chondrocytes, nest in this meshwork of collagen and proteoglycans. Normal articular cartilage exudes some of its water with compression, providing lubrication for joint surfaces. This water is reabsorbed during relaxation of the joint.

In OA, proteoglycans and collagen are lost from the cartilage as a result of enzymatic degradation. The water content of the cartilage increases as the collagen matrix is destroyed. With the loss of proteoglycans and collagen fibres, the cartilage becomes yellow or brownish grey and loses its tensile strength. Surface ulcerations occur and fissures develop in deeper layers of the cartilage. Eventually, large areas of articular cartilage are lost and underlying bone is exposed. The bone thickens in exposed areas, reducing its ability to absorb energy in joint loading. Cysts can also develop in the bone. *Cartilage-coated osteophytes* (bony outgrowths often called 'joint mice') change the anatomy of the joint. As these spurs or projections enlarge, small pieces may break off, leading to mild synovitis (inflammation of the synovial membrane).

Manifestations

The onset of OA is usually gradual and insidious, and the course slowly progressive. Pain and stiffness in one or more joints (usually weight bearing) are the first manifestations of OA. The pain is localised to the affected joints and may be described as a

deep ache. It typically is aggravated by use or motion of the joint and relieved by rest, although it may become persistent as the disease progresses. Pain at night may be accompanied by paraesthesias (numbness, tingling). Pain may also be referred to other parts of the body; for example, OA of the lumbosacral spine may cause severe pain along the path of the sciatic nerve. Following periods of immobility, such as sleeping all night or after a long automobile ride, involved joints may stiffen. Usually only a few minutes of activity are necessary to relieve the stiffness. Range of motion (ROM) of the joint decreases as the disease progresses and grating or crepitus may be noted during movement. Bony overgrowth may cause joint enlargement and flexion contractures may occur because of joint instability. In OA, enlarged joints are characteristically bony hard and cool on palpation. Manifestations specific to affected joints are outlined in the box below.

Complications

OA of the spine may involve the vertebral bodies and intervertebral discs, the diarthrodial joints, or both. *Spondylosis* is degenerative disc disease. As the intervertebral discs degenerate, disc space between the vertebrae is lost. Degenerative disc disease may be complicated by herniated disc, the protrusion of the nucleus pulposus of the disc. Herniation usually occurs in a lateral direction, potentially compressing nerve roots and causing radicular (distributed along the nerve) pain and muscle weakness. See Chapter 42 for further discussion of disc disorders.

Disc degeneration and joint space narrowing alter the mechanics of the spinal column, promoting osteoarthritic changes in the articular processes (the facet joints) of the vertebrae. The cartilage covering the inferior and superior articular processes degenerates, causing localised pain, stiffness, muscle spasm and limited range of motion. Osteophytes may form on articular processes, further contributing to pain and muscle spasm.

The presentation of OA in older people is similar to that in younger adults. However, in this population, the risk of debilitation because of OA is greater and the disease may progress faster. In addition, pain, stiffness and limited ROM increase the risk of falls and fractures in the older adult.

MANIFESTATIONS Osteoarthritis

AFFECTED SITE	MANIFESTATIONS
Interphalangeal joints	■ *Heberden's nodes*—bony enlargements of distal joints; may cause pain, redness, swelling ■ *Bouchard's nodes*—bony enlargement of proximal joints
First carpometacarpal	■ Swelling, tenderness at base of thumb ■ Crepitus with movement ■ 'Squared' appearance of joint
Spine	■ Localised pain and stiffness ■ Muscle spasm ■ Limited range of motion ■ Nerve root compression with radicular pain and motor weakness
Hips	■ Pain referred to inguinal area, buttock, thigh or knee ■ Loss of internal rotation ■ Limited extension, adduction and flexion
Knees	■ Pain and bony enlargement ■ Effusions ■ Crepitus ■ Instability and deformity with advanced disease

INTERPROFESSIONAL CARE

At this time, no treatment is available to arrest the process of joint degeneration. Appropriate management, however, is important to relieve pain and maintain the person's function and mobility. Research is also ongoing on a new class of medications called disease-modifying osteoarthritis drugs (DMOADs) and gene therapy. Vitamin D and chondroitin can slow the rate of degeneration of joints, especially knees (Arthritis Foundation, 2015).

Diagnosis

The diagnosis of OA is generally based on the person's history, physical examination and x-rays of affected joints. Diagnostic tests are described in Chapter 37.

Characteristic changes of OA are visible in x-ray studies of affected joints. Initially, irregular joint space narrowing is seen. Progressive changes include increased density of subchondral (under cartilage) bone, osteophyte formation at the joint periphery and the formation of cysts in the bone. In some cases, MRI or CT scan may be done to determine the extent of joint damage.

Examination of synovial fluid from involved joints can help rule out other types of arthritis (e.g. inflammatory arthritis, gout).

Medications

The pain of OA often can be managed through the use of analgesics such as aspirin. Paracetamol (Panadol) is generally preferred for use in older people because it has fewer toxic side effects. NSAIDs such as ibuprofen (Nurofen), naproxen (Naprosyn), indomethacin (Indocid) and diclofenac (Voltaren) may also be prescribed.

Topical medications include counterirritants, salicylates and capsaicin, sold without prescription as creams, gels, sprays, patches or ointments to relieve pain. Capsaicin, a topical agent, used alone or in conjunction with paracetamol is beneficial. The person should be taught to keep the medications away from their eyes, nose, mouth or any open skin, and not to bandage or apply heat to the treated area. The products should be used no more than three or four times a day and discontinued immediately if severe irritation occurs.

Medications that are effective in decreasing the pain and stiffness of OA are the NSAID COX-2 inhibitors. However, because of the increased risk of adverse cardiovascular (heart attack and strokes) and gastrointestinal (bleeding) effects of most drugs in this category, several were recalled by the Therapeutic Goods Administration (TGA) in 2004 (TGA, 2014). The only COX-2 inhibitor being prescribed as of 2006 was celecoxib (Celebrex).

Potent anti-inflammatory medications, such as systemic corticosteroids, are seldom prescribed for people with OA, although intra-articular corticosteroid injections may be used. With intra-articular injections, a long-acting corticosteroid medication, often mixed with a local anaesthetic such as lignocaine, is injected directly into the joint space of the affected joints. Although this procedure may provide marked pain relief, it can hasten the rate of cartilage breakdown if performed more frequently than every 4 to 6 months.

Treatments

OA is initially treated conservatively, but as pain increases and joint function decreases, surgery often becomes necessary.

CONSERVATIVE TREATMENT The goals of OA treatment are to relieve pain and maintain as much normal joint function as possible. Conservative treatment may include any or all of the following:

- ROM exercises, muscle-strengthening exercises, aerobic exercises
- heat and ice
- a balance between exercise and rest
- use of a cane, crutches or a walker
- weight loss, if indicated
- analgesic and anti-inflammatory medications.

VISCOSUPPLEMENTATION Viscosupplementation is a new treatment for OA of the knee. Hyaluronan, a natural component of synovial fluid, is injected directly into the knee joint. The injection may provide pain relief and improvement in knee function for up to 1 year, but its long-term effects are unknown (Hunter, 2015; NHMRC, 2009).

SURGERY Surgical procedures can provide dramatic results for people with significant chronic pain and loss of joint function. Although elective surgical procedures are frequently avoided in the older adult, even older people can benefit significantly if they do not have a chronic medical condition that contraindicates surgery.

ARTHROSCOPY An *arthroscopy* is a surgical procedure in which an arthroscope (a thin tube that is lit and has a camera in one end) is inserted into a joint. It may be done to diagnose the type of arthritis or to perform debridement by smoothing rough cartilage and flushing out the joint to remove debris. Although arthroscopic debridement and lavage of involved joints have been used, arthroscopy has not proven effective in the treatment of knee OA. It may be useful to remove large pieces of debris or repair a torn cartilage (Mayo Clinic, 2015).

OSTEOTOMY An *osteotomy*, an incision into or transection of the bone, may be performed to realign an affected joint, particularly when significant bony overgrowth or osteophyte formation has occurred. This procedure may also be used to shift the joint load towards areas of less severely damaged cartilage. Although osteotomy does not halt the process of OA, it may have a beneficial effect on joint function and pain, delaying the need for a joint replacement by several years.

JOINT ARTHROPLASTY A *joint arthroplasty* is the reconstruction or replacement of a joint. Arthroplasty is usually indicated when the person has severely restricted joint mobility and pain at rest. Pain is virtually eliminated and the function of the joint is generally improved. Arthroplasty may involve partial joint replacement or reshaping of the bones of a joint. For most people with OA, both surfaces of the affected joint are replaced with prosthetic parts in a procedure known as a *total joint replacement*. Joints that may be replaced include the hip, knee, shoulder, elbow, ankle, wrist and joints of the fingers and toes.

In a total joint replacement, some or all of the synovium, cartilage and bone on both sides of the joint are removed. A metallic prosthesis is inserted to replace one joint surface (generally the load-end or distal portion of a weight-bearing joint). The other joint surface is replaced by a silicone-lined ceramic or plastic prosthesis.

Most prosthetic joints are uncemented; that is, made of porous ceramic and metal components inserted so that they fit tightly into existing bone. The implant is secured by new bone growth into the prosthesis, a process that requires approximately 6 weeks. Although a longer non-weight-bearing period is necessary initially until the prosthesis is fixed in place by the bony growth, the implant appears to have a longer useful lifespan than cemented prostheses. In a cemented joint replacement, methyl methacrylate (a pliable polymer that hardens to hold the prosthesis in place) is used to secure the prosthesis to existing bone. Although the person is able to resume normal activities more rapidly following a cemented joint replacement, methyl methacrylate initiates an inflammatory response and the joint eventually loosens.

- In a *total hip replacement*, the articular surfaces of the acetabulum and femoral head are replaced. The entire head of the femur and part of the femoral neck are removed and replaced with a prosthesis (see Figure 39.3). The acetabulum is remodelled and a prosthesis of high-molecular-weight polyethylene is inserted. The success rate for total hip replacement is reported to be greater than 90%. Most hip replacements last 10 to 15 years, after which a second joint replacement, called

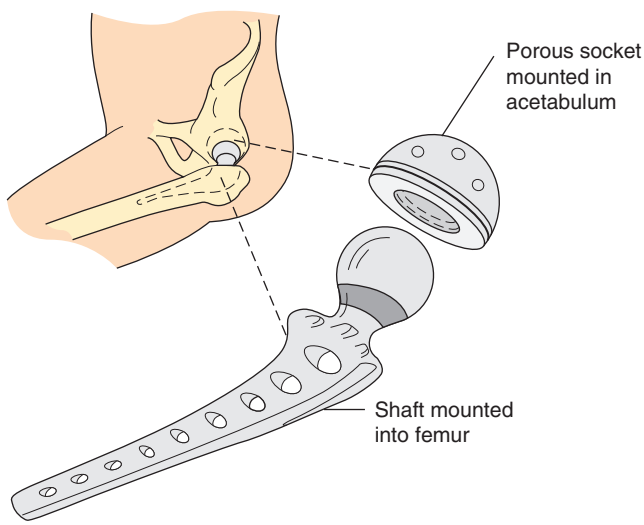


FIGURE 39.3 ■ Total hip prosthesis

a revision, can be performed. Potential problems associated with a total hip replacement include blood clots in leg veins, dislocation within the prosthesis, loosening of joint components from surrounding bone and infection. If recurrent or ineffectively treated, these complications may necessitate removal of the prosthesis, resulting in severe shortening of the extremity and an unstable hip joint.

- *Total knee replacement* is performed if the person has intractable pain and x-ray films show evidence of arthritis of the knee. Several prosthetic devices involving removal of varying amounts of bone are available for knee joint replacement (see Figure 39.4). The femoral side of the joint is replaced with a metallic surface and the tibial side with polyethylene. More than 80% of people obtain significant or total relief of pain with a total knee replacement. They must, however, engage in a vigorous program of rehabilitation to achieve the best results. Joint failure is more common with knee replacement than with a total hip replacement. Loosened joint components, often on the tibial side, are the most common cause of failure. The possible complications following a total knee replacement are the same as for a total hip replacement.
- *Total shoulder replacement* is indicated for unremitting pain and marked limitation of range of motion because of arthritic involvement of both the humeral and glenoid joint surfaces of the shoulder. The joint is immobilised in a sling or abduction splint for 2 to 3 weeks following arthroplasty. Dislocation, loosening of the prosthesis and infection are potential problems associated with total shoulder replacement.
- *Total elbow replacement* involves replacement of the humeral and ulnar surfaces of the elbow joint with a metal and polyethylene prosthesis. Pain and disabling stiffness of the joint are indications for an elbow arthroplasty. Complications, including dislocation, fracture, tricep weakness, loosening and infection, occur frequently.

Infection is the main complication associated with total joint replacement. Not only does infection interfere with healing and prolong recovery, but it may also necessitate removal of the prosthesis and may lead to loss of joint function. Other potential complications include circulatory impairment to the affected limb, thromboembolism, nerve damage and dislocation of the joint.

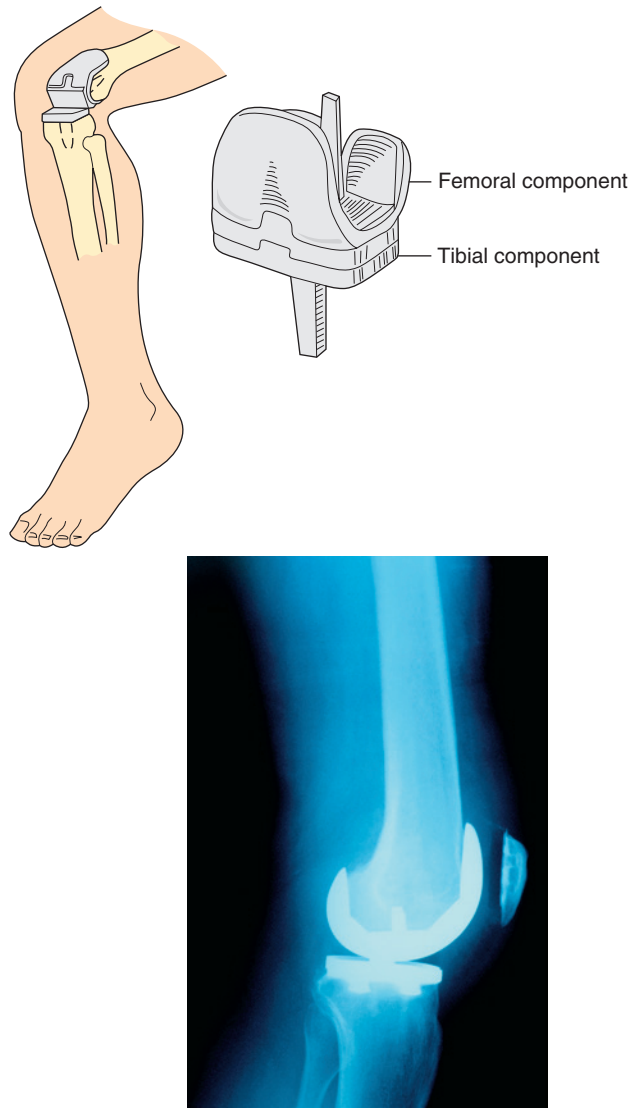


FIGURE 39.4 ■ Total knee replacement

Source: Image © David Frazier/Getty Images.

NURSING CARE OF THE PERSON having total joint replacement

PREOPERATIVE CARE

- Assess the person's knowledge and understanding of the planned operative procedure. Provide further explanations and clarification as needed. *It is important that the person have a clear and realistic understanding of the surgical procedure and expected results. Knowledge decreases anxiety and increases the person's ability to assist with postoperative care procedures.*
- Obtain a health history and physical assessment, including range of motion of the affected joints. *This information not only allows nurses to tailor care to the needs of the individual but also serves as a baseline for comparison of postoperative assessment data.*
- Explain necessary postoperative activity restrictions. Teach how to use the overhead trapeze for changing positions. *The person who learns and practises moving techniques before surgery can use them more effectively in the postoperative period.*
- Provide or reinforce teaching of postoperative exercises specific to the joint on which surgery is to be performed. *Exercises are prescribed postoperatively to: (a) strengthen muscles providing joint stability and support; (b) prevent muscle atrophy and joint contractures; and (c) prevent venous stasis and possible thromboembolism.*
- Teach respiratory hygiene procedures such as the use of incentive spirometry, coughing and deep breathing.

(continued)

NURSING CARE OF THE PERSON having total joint replacement (continued)

Adequate respiratory hygiene is imperative for all people undergoing joint replacement to prevent respiratory complications associated with immobility and the effects of anaesthesia. In addition, many people undergoing total joint replacement are older and may have reduced mucociliary clearance.

- Discuss postoperative pain control measures, including use of patient-controlled analgesia (PCA) or epidural infusion as appropriate. *It is important for the person to understand the purpose and use of postoperative pain control measures to allow early mobility and reduce complications associated with immobility.*
- Teach or provide prescribed preoperative skin preparation such as shower, shampoo and skin scrub with antibacterial solution. *These measures help reduce transient bacteria that may be introduced into the surgical site.*
- Administer intravenous antibiotic as ordered. *Antibiotic therapy is initiated before or during surgery and continued postoperatively to further reduce the risk of infection.*

POSTOPERATIVE CARE

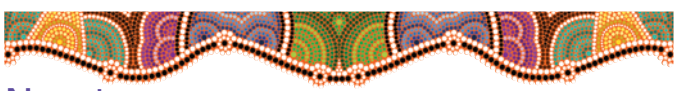
- Monitor vital signs, including temperature and level of consciousness, every 4 hours or more frequently as indicated. Report significant changes to the healthcare provider. *These routine assessments provide information about the person's cardiovascular status and can give early indications of complications such as excessive bleeding, fluid volume deficit and infection.*
- Perform neurovascular checks (colour, temperature, pulses and capillary refill, movement and sensation) on the affected limb hourly for the first 12 to 24 hours, then every 2 to 4 hours. Report abnormal findings to the healthcare provider immediately. *Surgery can disrupt the blood supply to or innervation of the affected extremity. If so, rapid intervention is important to preserve the function of the extremity.*
- Monitor incisional bleeding by emptying and recording suction drainage every 4 hours and assessing the dressing frequently. *Significant blood loss can occur with a total joint replacement, particularly a total hip replacement.*
- Reinforce the dressing as needed. *The dressing is usually changed 24 to 48 hours after surgery but may need reinforcement if excess bleeding occurs.*
- Maintain intravenous infusion and accurate intake and output records during the initial postoperative period. *The person is at risk of fluid volume deficit in the initial postoperative period because of blood and fluid loss during surgery, as well as the effects of the anaesthetic.*
- Maintain bed rest and prescribed position of the affected extremity using a sling, abduction splint, brace, immobiliser or other prescribed device. *Proper positioning of the affected extremity is vital in the initial postoperative period so that the joint prosthesis does not become dislocated or displaced.*
- Help the person shift position at least every 2 hours while on bed rest. *Shifting of position helps prevent pressure ulcers and other complications of immobility.*
- Remind the person to use the incentive spirometer, to cough and to breathe deeply at least every 2 hours. *These measures are important to prevent respiratory complications such as pneumonia.*
- Assess the person's level of comfort frequently. Maintain PCA, epidural infusion or other prescribed analgesia to promote comfort. *Adequate pain management promotes healing and mobility.*
- Help the person get out of bed as soon as allowed. Teach and reinforce the use of techniques to prevent weight bearing on the affected extremity, such as the overhead trapeze, pivot turning and toe touch. *Early mobility prevents complications such as pneumonia and thromboembolism, but appropriate techniques must be used to prevent injury to the operative site.*
- Initiate physical therapy and exercises as prescribed for the specific joint replaced, such as quadriceps setting, leg raising and passive and active ROM exercises. *These exercises help prevent muscle atrophy and thromboembolism and strengthen the muscles of the affected extremity so that it can support the prosthetic joint.*
- Use sequential compression devices or anti-embolism stockings, as prescribed. *These help prevent thromboembolism and pulmonary embolus for the person who must remain immobile following surgery.*
- For the person with a total hip replacement, prevent hip flexion of greater than 90 degrees or adduction of the affected leg. Provide a seat riser for the toilet or commode. *These measures prevent dislocation of the joint.*
- Assess the person with a total hip replacement for signs of prosthesis dislocation, including pain in the affected hip, or shortening and internal rotation of the affected leg.
- For the person with a total knee replacement, use a continuous passive range-of-motion (CPM) device or ROM exercises as prescribed. *Dislocation is not a problem with a knee replacement and more emphasis is placed on ROM exercises in the early postoperative period.*
- Maintain fluid intake and encourage a high-fibre diet. Administer stool softeners or rectal suppositories as needed. *Immobility contributes to the potential problem of constipation; these measures help maintain regular faecal elimination.*
- Encourage consumption of a well-balanced diet with adequate protein. *Adequate nutrition promotes tissue healing.*
- Teach or reinforce post-discharge exercises and activity restrictions. Emphasise the importance of scheduled follow-up physician visits. *People are discharged from the acute care facility before healing is complete. Exercises are prescribed and activities are resumed gradually to protect the integrity of the joint replacement and prevent contractures.*
- For those people needing additional direct care after discharge, arrange placement in a long-term care or rehabilitation facility. *Activity restrictions may preclude discharge to home for some people.*
- Make referrals as needed to community nursing and physiotherapy. *People often require home healthcare for both nursing care needs and continued physiotherapy following discharge from acute or long-term care.*

Nursing care for the person undergoing total joint replacement is outlined in the box below. Refer to Chapter 3 for further discussion of care for the person undergoing surgery.

PHYSICAL THERAPY AND REHABILITATION Recovery from all types of joint replacement requires postoperative physical therapy, focusing on building strength and regaining joint flexibility. Rehabilitation begins in the hospital, most often the day following surgery, and may be continued during home care. Recovery from a hip replacement is 80% complete in 4 weeks and 100% complete in 6 months. Recovery from a knee replacement is 80% complete in 4 weeks and 100% complete after 1 year. During rehabilitation, the person must follow a regimen of exercise, rest and medication (AIHW, 2013; 2015).

COMPLEMENTARY AND ALTERNATIVE THERAPIES The following complementary therapies are examples of those that may be used by people with OA to relieve pain and stiffness. These same therapies are also used by people with rheumatoid arthritis:

- biomagnetic therapy
- rest and joint protection
- weight loss
- acupuncture
- eliminating nightshade foods, such as potatoes, tomatoes, capsicum and eggplant, and tobacco
- taking nutritional supplements, such as glucosamine and chondroitin
- herbal therapy
- massage therapy
- osteopathic manipulation
- vitamin therapy
- yoga.



Nursing care

Osteoarthritis is a chronic process for which there is no cure. The focus of nursing care for the person with OA is providing comfort, helping maintain mobility and ADLs, and assisting with adaptations to maintain life roles. A nursing care plan for a person with OA is found below.

Health promotion

Although OA cannot be prevented, maintaining a normal weight and having a program of regular, moderate exercise will reduce risk factors. Glucosamine and chondroitin are nutritional supplements for OA that are increasingly popular and have been found to be of benefit in reducing manifestations. People should discuss these supplements with their healthcare provider before using them.

Assessment

Collect the following data through the health history and physical examination (see Chapter 37):

- *Health history:* family history of OA, occupation, recreational activities, joint pain and stiffness, ability to carry out ADLs and self-care activities.
- *Physical assessment:* height/weight; gait; joints: symmetry, size, shape, colour, appearance, temperature, pain, crepitus, range of motion, Heberden's nodes, Bouchard's nodes.

Nursing diagnoses and interventions

The priority nursing interventions for people with OA are directed towards managing chronic pain, facilitating physical mobility and improving ability to provide self-care.

Chronic pain

Pain is a primary manifestation of OA. As joint tissues degenerate and changes in joint structure occur, the amount of discomfort generally increases. The pain associated with OA increases with activity and tends to be relieved with rest. Non-pharmacological comfort measures are appropriate, with mild analgesics used to supplement these as needed.

- Monitor the level of pain, including intensity, location, quality and aggravating and relieving factors. *Accurate assessment of pain provides a basis for evaluation of the effect of interventions.*
- Teach people to take prescribed analgesic or anti-inflammatory medication as needed. Analgesics reduce the perception of pain and may decrease muscle spasm as well. *Anti-inflammatory medication may be ordered to decrease local inflammatory response in affected joints.*
- Encourage rest of painful joints. *The pain of OA is often relieved by joint rest.*
- Suggest applying heat to painful joints using the shower, a tub or sitz bath, warm packs, hot wax baths, heated gloves or diathermy, which uses high-frequency electrical currents to generate heat. *Heat application reduces accompanying muscle spasm, relieving pain. Moist heat penetrates deeper than dry heat; diathermy delivers heat directly to lesions in deeper body tissues.*
- Emphasise the importance of proper posture and good body mechanics for walking, sitting, lifting and moving. *Good body mechanics and posture reduce stress on affected joints.*
- Encourage the overweight person to reduce weight. *Excess weight places abnormal stress on joints, particularly the knees.*
- Encourage the use of non-pharmacological pain relief measures such as progressive relaxation, meditation, visualisation and distraction. *These adjunctive pain relief measures can reduce the person's reliance on analgesics and increase comfort.*

Impaired physical mobility

As intra-articular cartilage degenerates and joint structures are altered, the person with OA experiences pain, stiffness and decreased range of motion in affected joints. When the spine, large weight-bearing joints of the hips and knees, or the ankles and feet are affected, physical mobility can be significantly reduced.

- Assess the range of motion of affected joints. *Assessing joint mobility is important as a basis for planning appropriate interventions.*

- Perform a functional mobility assessment, evaluating gait, ability to sit and rise from sitting position, ability to step into and out of the tub or shower, and negotiation of stairs. *The functional assessment provides vital data about the person's ability to maintain ADLs.*
- Teach active and passive ROM exercises as well as isometric, progressive resistance and low-impact aerobic exercises. *Active ROM exercises help maintain muscle tone and mobility of affected joints and prevent contractures.*

Isometric and progressive resistance exercises improve muscle tone and strength; aerobic exercise improves endurance and cardiovascular fitness.

CONSIDERATION FOR PRACTICE

The older woman with OA may be more willing to take part in weight-bearing exercises if she does so as part of a group or organised activity.

NURSING CARE PLAN A person with osteoarthritis

Raymond Nasso is a 72-year-old retired commercial fisherman who has experienced arthritic pain in his hips for the past 10 to 15 years. During the past year, the pain in his right hip has become severe, prompting him to seek medical attention. Significant degenerative changes in both hip joints are noted on x-ray films. The physician recommends a total replacement of the right hip and total replacement of the left hip to follow in 6 to 12 months. Mr Nasso has preoperative teaching and tests the afternoon prior to his surgery, scheduled for 0800 the following morning.

ASSESSMENT

Christie Phlaugh, RN, completes a health history and examination of Mr Nasso on admission. Reviewing his medical record, she notes that Mr Nasso has mild Parkinson's disease and is taking carbidopa/levodopa (Sinemet 25-100) four times a day to control his symptoms. No other chronic medical conditions have been reported. Mr Nasso says he has been essentially healthy his entire life. He has no known allergies to medications, has never smoked and consumes only small amounts of alcohol.

On examination of Mr Nasso, Ms Phlaugh notes that he is alert and oriented. His vital signs are BP 116/64, P 68 regular, R 18, T 36.3°C PO. Peripheral pulses are strong and equal in the upper extremities and slightly weaker but equal in the lower extremities. His feet are cool to the touch but have immediate capillary refill. He has full ROM of his shoulders, elbows and wrists. The ROM of both hips is significantly restricted. Hip flexion beyond 90 degrees prompts pain on both sides. Both flexion and extension of the knees are limited slightly. Mr Nasso walks with a limp, favouring his right hip, and has a shuffling gait.

Preoperative laboratory studies including FBC, coagulation studies, chemistry panel and urinalysis show a serum creatinine of 1.7 mg/dL and BUN of 30 mg/dL, with no other abnormal values noted. His skin swabs, ECG and chest x-ray show no apparent pathologies. Cephazolin 500 mg is to be administered intravenously at 0600 prior to surgery and Mr Nasso is to shower and shampoo with antibacterial soap at bedtime. The physical therapist meets with Mr Nasso to evaluate his mobility and begin teaching him about postoperative weight-bearing restrictions.

DIAGNOSES (POSTOPERATIVE)

- *Acute pain* related to surgical incision.
- *Impaired physical mobility* related to activity and weight-bearing restrictions.
- *Risk of infection* related to disruption in skin integrity.
- *Risk of ineffective right leg tissue perfusion* related to vascular disruption and oedema.

PLANNING

Ensure Mr Nasso is able to manage his pain and prevent postoperative complications.

Expected outcomes

- Maintain an adequate level of comfort postoperatively as demonstrated by verbal expressions of comfort and the ability to move easily within restrictions.
- Remain free of infection.
- Maintain adequate perfusion of affected leg.
- Remain free of injury postoperatively.
- Comply with instructions to cough and breathe deeply.
- Remain free of adverse consequences of immobility such as pneumonia, pressure areas, thromboembolism or contracture.

IMPLEMENTATION

- Assess pain at least hourly during first 24 to 48 hours postoperatively and as needed thereafter.
- Instruct in the use of PCA and monitor its effectiveness.
- Help change position at least every 2 hours; encourage the use of the overhead trapeze to shift position frequently.
- Assess the surgical site frequently; report signs of excess bleeding or inflammation.
- Monitor temperature every 4 hours.
- Maintain sequential compression device and anti-embolic stocking as ordered; remove for 1 hour daily.
- Assist out of bed three times a day after the first 24 hours.
- Maintain abduction of the right hip with pillows.
- Perform passive ROM exercises of unaffected extremities every shift.
- Encourage frequent quadriceps-setting exercises and plantar and dorsiflexion of feet.
- Assess pulses, colour, movement and sensation of right foot hourly for the first 24 hours, then every 2 hours for 24 hours, then every 4 hours.
- Encourage the use of the incentive spirometer hourly for first 24 hours, then at least every 2 hours while awake.

EVALUATION

Mr Nasso returns to the orthopaedic unit from the recovery ward. For the first 36 hours after surgery he is confused and disoriented, but his orientation and thought processes gradually clear. His family has stayed with him and he has not experienced injury or other adverse consequences from his

NURSING CARE PLAN A person with osteoarthritis (continued)

confusion. Otherwise, Mr Nasso has had an uneventful postoperative recovery. Six days after surgery, he is transferred to an extended care rehabilitation facility for further therapy until he is able to ambulate with partial weight bearing on his affected leg. He returns home 5 weeks after surgery, able to use a walker for ambulation. Arrangements are made for an over-bed trapeze, elevated toilet seat and shower chair in his home. A community nurse and physiotherapist visit Mr and Mrs Nasso weekly for a month following his discharge. During this time Mr Nasso gradually resumes full weight bearing. Mr Nasso expresses pleasure with the relief of his hip pain and says he has no fear of having his left hip replaced in the future.

CRITICAL THINKING IN THE NURSING PROCESS

1 Mr Nasso's preoperative laboratory work showed a modest elevation in his serum creatinine and BUN. What do these studies indicate? How might these changes affect nursing responsibilities related to medication administration for Mr Nasso?
2 Mr Nasso became confused postoperatively. Which factors in his history might have alerted the nurses to this possibility? How might anaesthesia and postoperative analgesics have contributed to his confusion?
3 Develop a care plan for Mr Nasso using the nursing diagnosis of *Acute confusion*.

REFLECTION ON THE NURSING PROCESS

1 From caring for Mr Nasso, identify two key issues that you will be able to utilise for future practice.
2 Which health education and teaching techniques are required prior to Mr Nasso's discharge?

Self-care deficit

Just as OA of the lower extremities can reduce the person's mobility, OA of the upper extremities (the wrist, hand and finger joints, in particular) can significantly interfere with performance of ADLs such as cooking and brushing the hair. When the lower extremities are affected, bathing and toileting can be difficult.

- Perform a functional assessment of the upper and lower extremities. For upper extremities, assess the ability to touch the back of the head and to hold and use small items such as eating utensils. *The functional assessment provides important data about the person's ability to provide self-care.*
- Assess the home setting to determine the need for assistive devices such as handrails, grab bars, walk-in shower stall or shower chair, and handheld showerhead. *Many assistive devices are relatively easy and inexpensive to obtain and can significantly improve the person's independence in performing ADLs.*
- Assist in obtaining other assistive devices such as long-handled shoehorns, zipper grabbers, long-handled tongs or grippers for retrieving items from the floor, jar openers and special eating utensils. *These devices can prolong independence in performing ADLs.*

Community-based care

Because of the chronicity of OA, people and their families need appropriate teaching to manage the disease and its consequences effectively. Much of the teaching focus is on preservation of joint function and mobility. Discuss the following topics:

- Safeguard against hazards to safe mobility, such as scatter rugs. Encourage installation of safety devices such as handrails and grab bars.
- Understand the disease process and its chronic degenerative nature.
- Learn exercise techniques, including ROM, isometric, postural, stretching and strengthening, to maintain healthy cartilage, preserve ROM and develop supportive muscles and tendons. A walking program is beneficial for people with OA of the knee.
- Do not overuse or stress affected joints with heavy lifting, excessive stair climbing or bending, or other repetitive actions.
- Sit in a straight chair without slumping; avoid soft chairs or recliners, and sleep on a firm mattress or use a bed board.
- Use pain relief measures including prescribed or over-the-counter analgesic medications and non-pharmacological pain relief measures such as heat, rest, massage, relaxation and meditation.

For the person who has had a total joint replacement, discuss the following:

- use and weight bearing of the affected limb
- appropriate environmental modifications, such as an overhead trapeze for getting out of bed, elevated toilet seats and types of chairs to use and avoid when sitting
- prescribed exercises
- use of assistive devices for ambulation, such as crutches or a walker
- possible complications, including signs of infection or dislocation, and the need to notify the physician promptly if these occur.

Make referrals to home care, physical or occupational therapy, or other community agencies as indicated, and suggest the following resources:

- Arthritis Australia: www.arthritisaustralia.com.au
- Arthritis New Zealand: www.arthritis.org.nz
- Australian Rheumatology Association: http://rheumatology.org.au.

THE PERSON WITH MUSCULAR DYSTROPHY

Muscular dystrophy (MD) is a group of inherited muscle diseases that cause progressive muscle degeneration and wasting. The differences in the types of MD relate to the age at onset, the gender affected by the disorder, the muscles involved

TABLE 39.2 Types of muscular dystrophy

TYPE	SEX AND AGE AT ONSET	CLINICAL MANIFESTATIONS	PROGRESSION
Duchenne's	Males Ages 3 to 5	Weakness of pelvic and shoulder girdles Waddling gait Toe walking Lordosis Cardiac abnormalities Low IQ in 50% of cases	Rapid; person usually confined to wheelchair by age 15; death occurs by age 20
Myotonic	Males and females Any age	Myotonia of hand muscles Muscular weakness of arms and legs Cardiac abnormalities Endocrine abnormalities Mental retardation (common)	Slow; death usually occurs in early fifties
Becker's	Males Ages 5 to 20	Weakness of pelvic and shoulder girdles	Slow; person usually confined to wheelchair at 25 years after onset; normal lifespan
Facioscapulohumeral	Males and females Ages 10 to 20	Weakness of face and shoulder girdles	Slow; normal lifespan
Limb-girdle	Males and females Ages 20 to 40	Weakness of shoulder and pelvic girdles	Extremely variable; usually slow

and the rate at which the disease progresses. These factors are summarised in Table 39.2. In the majority of cases of MD, there is a positive family history.

The most common form of MD, Duchenne's muscular dystrophy, is inherited as a recessive single gene defect on the X chromosome (a sex-linked recessive disorder) and is transmitted from the mother to male children. This disorder affects males exclusively and occurs in 1 of 3500 live male births. It can be recognised early in pregnancy in about 95% of cases by genetic studies or in late pregnancy through amniocentesis. Genetic counselling cannot be reliably used to prevent this disease because there is no way to determine if the woman carries the defective gene. The manifestations appear in early childhood, with the average lifespan being about 15 years after onset (Porth & Matfin, 2014).

Other types of MD have an onset at any age and a slow progression with a normal lifespan.

Pathophysiology

The basic defect in MD is unknown; however, three theories have been proposed. The *vascular* and *neurogenic theories* suggest that the cause is a lack of blood supply to the muscle or a disturbance in the interaction between the nerve and muscle. The *membrane theory* suggests that an alteration in the cell membranes of the muscle causes them to degenerate. Recent genetic studies have shown a deficiency in the amount of dystrophin, a muscle membrane protein, in people with Duchenne's MD. Dystrophin plays an important role in protecting the muscle against mechanical stresses.

Manifestations

All forms of MD exhibit manifestations of muscle weakness. The specific muscles involved depend on the type of MD. As the disease progresses, the person develops difficulty with ambulation and eventually becomes wheelchair bound and finally bed bound. Cardiac abnormalities, endocrine abnormalities and mental retardation may also occur.

INTERPROFESSIONAL CARE

Because there is no cure or specific treatment for MD, care focuses on preserving and promoting mobility. An interdisciplinary approach, involving many members of the healthcare team, is necessary to meet the physical and psychological needs of these people and their families. Diagnosis and classification of the muscular dystrophies are most often based on the manifestations and the pattern of muscle involvement. Biochemical examination, muscle biopsy and electromyography confirm the diagnosis. Diagnostic tests are described in Chapter 37.

Tests include measuring creatine kinase (CK-MM, the isoenzyme found in skeletal muscle) which is elevated in the person with suspected MD; performing a muscle biopsy to identify fibrous connective tissue and fatty deposits that displace functional muscle fibres; and conducting an electromyogram (EMG), which will show a decrease in amplitude in MD.

Nursing care

Nursing care for a person with MD focuses on promoting independence and mobility, and providing psychological support for both the person and family. A holistic approach is essential in planning and implementing care.

Nursing diagnoses and interventions

Self-care deficit

The progressive muscle weakness that is associated with MD impairs the person's ability to perform self-care.

- Provide the person and their family with supportive care during the progress of the disease. The goal of treatment is to prolong each functional stage and delay or prevent deformity. *When transition from ambulation to a wheelchair occurs, depression and grief may occur.*
- Promote independence. Encourage tasks that can be accomplished rather than letting the person struggle with tasks that may prove frustrating. *All forms of MD result in progressive muscle weakness. Management of the disease is directed towards keeping the person as functional as possible while preventing any deformities.*

Community-based care

Teaching the person with MD focuses on maintaining function and independence and preventing deformities. Teach prescribed exercises such as stretching and counterposturing exercises. For the person with braces, discuss skin care and ways to prevent irritation under the brace. Because the person may have weakness involving muscles of respiration, teach the person how to prevent respiratory infections, such as avoiding crowds during flu season and being immunised against pneumococcal pneumonia and influenza. Provide information about support services and organisations such as the Muscular Dystrophy Association.

AUTOIMMUNE AND INFLAMMATORY DISORDERS

Autoimmune and inflammatory disorders of the musculoskeletal system are chronic systemic rheumatic disorders, characterised by diffuse inflammatory lesions and degenerative changes in connective tissues. The disorders have similar clinical features and may affect many of the same structures and organs.

THE PERSON WITH RHEUMATOID ARTHRITIS

Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease that causes inflammation of connective tissue, primarily in the joints. Its course and severity are variable and the range of manifestations is broad. Manifestations of RA may be minimal, with mild inflammation of only a few joints and little structural damage, or relentlessly progressive, with multiple inflamed joints and marked deformity. Most people exhibit a pattern of symmetrical involvement of multiple peripheral joints and periods of remission and exacerbation.

The cause of RA is unknown. A combination of genetic, environmental, hormonal and reproductive factors is thought to play a role in its development. It is speculated that infectious agents, such as bacteria, mycoplasmas and viruses (especially Epstein–Barr virus), may play a role in initiating the autoimmune processes present in RA. Several studies have found that heavy smokers are at increased risk of developing RA. It is known that the incidence of RA has decreased during the past 40 years, supporting the theory that environmental factors may change and either promote or protect against RA (Arthritis Foundation, 2015; Grossman & Porth, 2013).

The course of RA is variable and fluctuating. Remissions are most likely to occur in the first year of the disease. The rate at which joint deformities develop is not constant. Disease progression is fastest during the first 6 years, slowing thereafter. RA contributes to disability and a tendency to shorten life expectancy. About 10% of people with RA go into long-term remission within 1 year; and another 50–60% go into remission within 2 years (Arthritis Foundation, 2015; Grossman & Porth, 2013).

FAST FACTS

- RA is found worldwide, affecting 0.3–2% of the total population and all races.
- RA affects three times as many women as men.
- The onset of RA occurs most frequently between the ages of 30 and 50 years.
- RA is less common than OA, with RA affecting around 400 000 Australians (AIHW, 2009).

The incidence of RA increases with age up to about 70 years. Although the onset and manifestations of RA are much the same in older and younger people, differentiating between RA and OA in the older adult may be difficult at times. It is important to establish an accurate diagnosis, however, because the management of these disorders differs significantly. Clinical features distinguishing RA from OA are listed in Table 39.3.

For older people, RA is managed much as it is for younger people. However, prolonged bed rest or inactivity is not prescribed for acute episodes because it may result in irreversible immobility in the older adult. Also, medications are used with greater caution because of the increased risk of toxicity. In many cases, emphasis is placed less on preventing joint deformity and more on maintaining functional status for the older person with RA.

Pathophysiology

It is believed that long-term exposure to an unidentified antigen causes an aberrant immune response in a genetically

TABLE 39.3 Comparison of the manifestations of rheumatoid arthritis and osteoarthritis

FEATURE	RHEUMATOID ARTHRITIS	OSTEOARTHRITIS
Onset	Usually insidious, may be abrupt	Insidious
Course	Generally progressive, characterised by remissions and exacerbations	Slowly progressive
Pain and stiffness	Predominant on arising, lasting > 1 hour; also occurs after prolonged inactivity	Pain with activity; stiffness following periods of immobility generally relieved within minutes
Affected joints	Appear red, hot, swollen; 'boggy' and tender to palpation; decreased ROM, weakness	Affected joints may appear swollen; cool and bony hard on palpation; decreased ROM
	Multiple joints affected in symmetrical pattern; PIP, MCP, wrists, knees, ankles and toes often involved	One or several joints affected including hips, knees, lumbar and cervical spine, PIP and DIP, wrist and 1st MTP joint
Systemic manifestations	Fatigue, weakness, anorexia, weight loss, fever; rheumatoid nodules; anaemia	Fatigue

susceptible host. As a result, normal antibodies (immunoglobulins) become autoantibodies and attack host tissues. These transformed antibodies, usually present in people with RA, are called *rheumatoid factors (RFs)*. The self-produced antibodies bind with their target antigens in blood and synovial membranes, forming immune complexes. (See Chapter 12 for further information about autoimmune processes.)

The damage to cartilage that occurs in RA is the result of at least three processes:

1. Neutrophils, T cells and other synovial fluid cells are activated and degrade the surface layer of the articular cartilage.
2. Cytokines, especially interleukin-1 (IL-1) and tumour necrosis factor alpha (TNF-α), cause the chondrocytes to attack the cartilage.
3. The synovium digests nearby cartilage, releasing inflammatory molecules containing IL-1 and TNF-α.

Leucocytes are attracted to the synovial membrane from the circulation, where neutrophils and macrophages ingest the immune complexes and release enzymes that degrade synovial tissue and articular cartilage. Activation of B and T lymphocytes results in increased production of rheumatoid factors and enzymes that increase and continue the inflammatory process.

The synovial membrane is damaged by the inflammatory and immune processes. It swells from infiltration of the leucocytes and thickens as cells proliferate and abnormally enlarge. The inflammation spreads and involves synovial blood vessels. Small venules are occluded and vascular flow to the synovial tissue decreases. As blood flow decreases and metabolic needs increase (from the increased number and size of cells), hypoxia and metabolic acidosis occur. Acidosis stimulates synovial cells to release hydrolytic enzymes into surrounding tissues, starting erosion of the articular cartilage and inflammation of the supporting ligaments and tendons.

The inflammation also causes haemorrhage, coagulation and deposits of fibrin on the synovial membrane, in the intracellular matrix and in the synovial fluid. Fibrin develops into *granulation tissue (pannus)* over denuded areas of the synovial membrane. The formation of pannus leads to scar tissue formation that immobilises the joint (see Figure 39.5).

Joint manifestations

The onset of RA is typically insidious, although it may be acute (precipitated by a stressor such as infection, surgery or trauma). Joint manifestations are often preceded by systemic manifestations of inflammation, including fatigue, anorexia, weight loss and non-specific aching and stiffness. People report joint swelling with associated stiffness, warmth, tenderness and pain. The pattern of joint involvement is typically polyarticular (involving multiple joints) and symmetrical. The proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of the fingers, the wrists, the knees, the ankles and the toes are most frequently involved, although RA can affect any joint. Stiffness is most pronounced in the morning, lasting more than 1 hour. It may also occur with

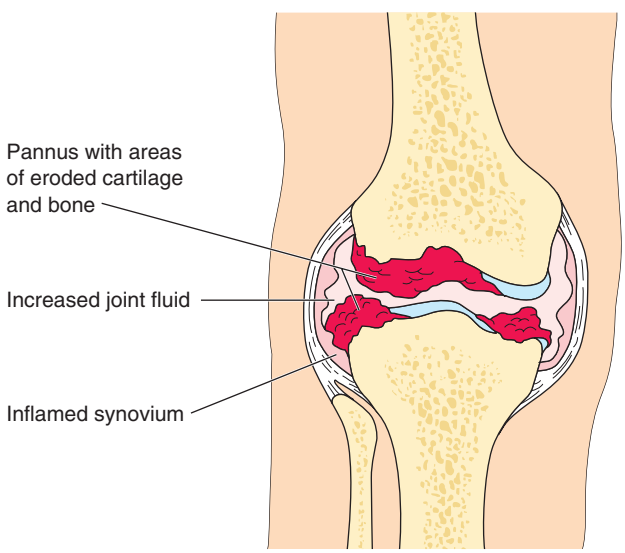


FIGURE 39.5 ■ Joint inflammation and destruction in rheumatoid arthritis. Note synovial inflammation with pannus formation and the erosion of cartilage and underlying bone

prolonged rest during the day and may be more severe following strenuous activity. Swollen inflamed joints feel 'boggy' or sponge-like on palpation because of synovial oedema. Range of motion is limited in affected joints and weakness may be evident.

The persistent inflammation of RA causes deformities of the joint itself and supporting structures such as ligaments, tendons and muscles. As the joint is destroyed, ligaments, tendons and the joint capsule are weakened or destroyed. Joint cartilage and bone are also destroyed. Weakening or destruction of these supporting structures results in lack of opposition to muscle pull, causing deformity.

Characteristic changes in the hands and fingers include ulnar deviation of the fingers and subluxation at the MCP joints. Swan-neck deformity is characterised by hyperextension of the PIP joint with compensatory flexion of the distal interphalangeal (DIP) joints (see Figure 39.6). A flexion deformity of the PIP joints with extension of the DIP joint is called a boutonnière deformity. The ability to effect a pinch is limited by hyperextension of the interphalangeal joint and flexion of the MCP joint of the thumb.

Wrist involvement is nearly universal, leading to limited movement, deformity and carpal tunnel syndrome. Inflammation of the elbows often causes flexion contracture.

The knees are frequently affected in RA, with visible swelling often obliterating normal contours. Instability of the knee joint along with quadriceps atrophy, contractures and valgus (knock-knee) deformities can lead to significant disability. Ambulation may be limited by pain and deformities when the ankles and feet are involved. Typical deformities of the feet and toes include subluxation, hallux valgus (deviation of the great toe towards the other digits of the foot), lateral deviation of the toes and cock-up toes (turned-up toes).

Spinal involvement is usually limited to the cervical vertebrae. Neck pain is common and neurological complications can occur.

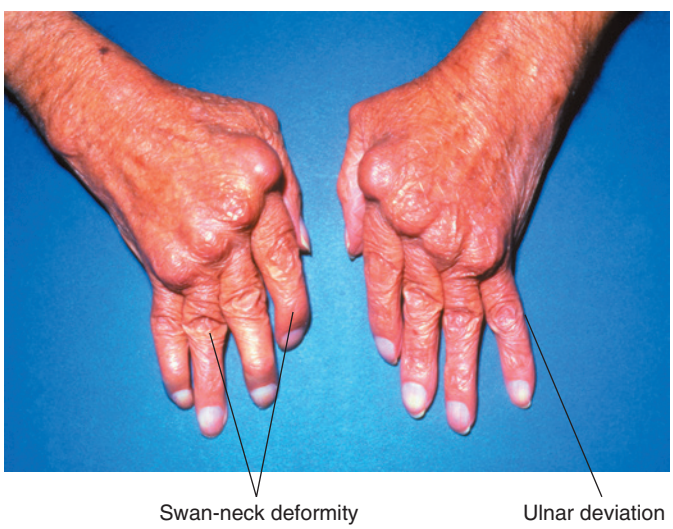


FIGURE 39.6 ■ Typical hand deformities associated with rheumatoid arthritis

Source: © James Stevenson/Photo Researchers, Inc.

Extra-articular manifestations

RA is a systemic disease with a variety of extra-articular manifestations. These are seen particularly in people with high levels of circulating rheumatoid factor. Fatigue, weakness, anorexia, weight loss and low-grade fever are common when the disease is active. Anaemia resistant to iron therapy frequently affects people with RA. Skeletal muscle atrophy is common, usually most apparent in the musculature around affected joints.

Rheumatoid nodules may develop, usually in subcutaneous tissue in areas subject to pressure: on the forearm, olecranon bursa, over the MCP joints and on the toes. Rheumatoid nodules are granulomatous lesions that are firm and either movable or fixed. They may also be found in viscera, including the heart, lungs, intestinal tract and dura.

Other possible extra-articular manifestations of RA include subcutaneous nodules, pleural effusion, vasculitis, pericarditis and splenomegaly (enlargement of the spleen). The multisystem effects of RA are illustrated overleaf.

Increased risk of coronary heart disease

People with rheumatoid arthritis have an increased risk of developing coronary heart disease (CHD). In turn, CHD increases the risk of myocardial infarction and death; in fact, RA is associated with a shortened life expectancy (Arthritis Foundation, 2015; Grossman & Porth, 2013). RA affects the heart by:

- direct effects on the blood vessels, with measures of C-reactive proteins (inflammatory markers) being more predictive of future cardiovascular disease than low-density lipoprotein (LDL) levels
- increased risk of having low high-density lipoprotein, high cholesterol and triglyceride levels, high blood pressure and high levels of homocysteine—all of which increase the risk of CHD
- the damaging side effects that many medications, such as methotrexate and steroids, often have on coronary vessels.

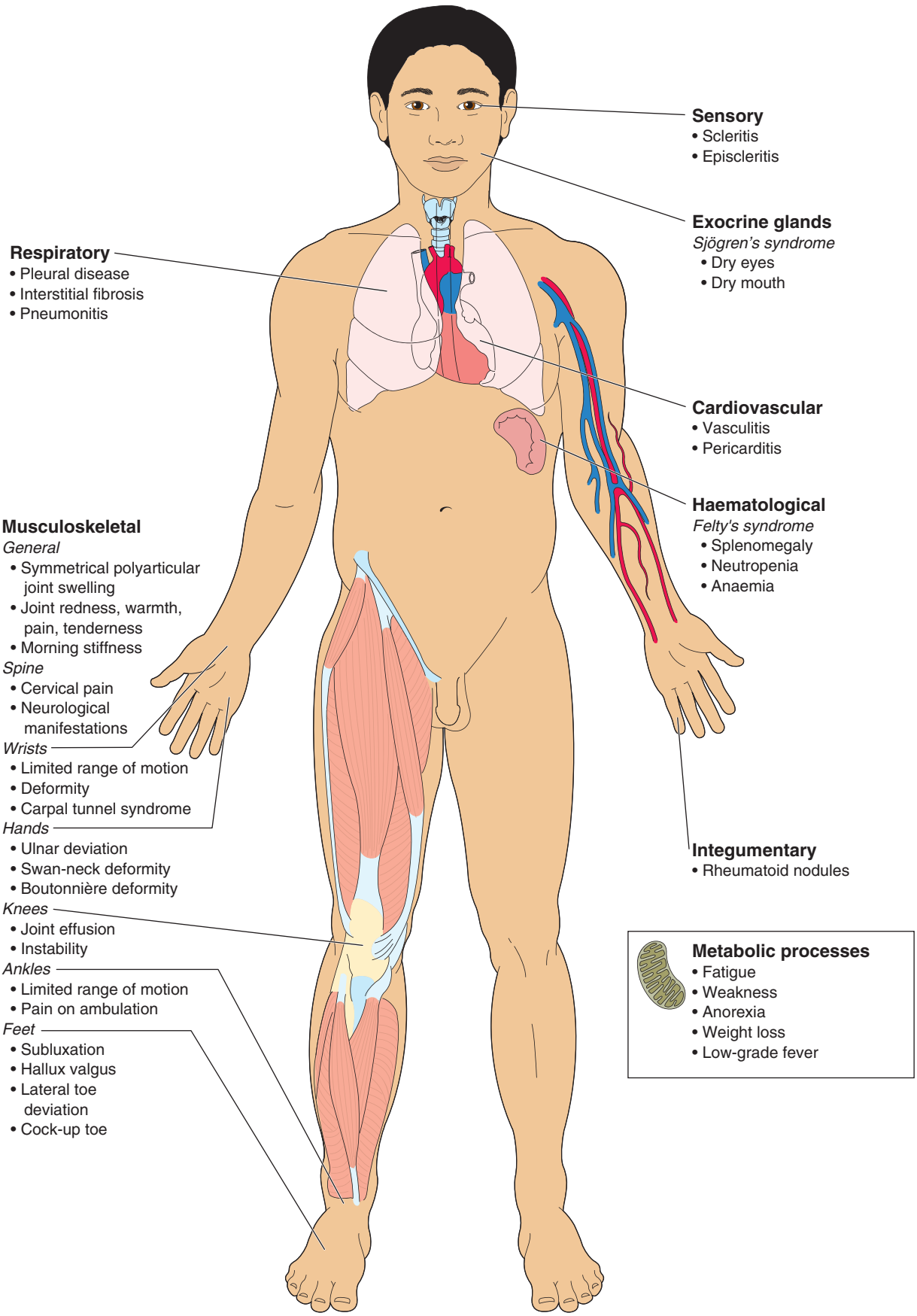
INTERPROFESSIONAL CARE

The diagnosis of RA is based on the person's history, physical assessment and diagnostic tests. Diagnostic criteria developed by the American Rheumatism Association (2015) are used as well (see Box 39.4). At least four of seven criteria must be present to establish the diagnosis. Positive RF occurs in approximately 80% of people and levels rise during active phases of the disease.

Once the diagnosis of RA has been established, the goals of therapy are to relieve pain, reduce inflammation, slow or stop joint damage, and improve wellbeing and ability to function. No cure currently exists for RA; the goal of treatment is to relieve its manifestations. An interprofessional approach is used, with a balance of rest, exercise, physical therapy and suppression of the inflammatory processes.

Because a cure is not available and traditional therapies are not always fully effective, the person with RA is vulnerable to

MULTISYSTEM EFFECTS OF RHEUMATOID ARTHRITIS



Sensory
- Scleritis
- Episcleritis

Exocrine glands
Sjögren's syndrome
- Dry eyes
- Dry mouth

Respiratory
- Pleural disease
- Interstitial fibrosis
- Pneumonitis

Cardiovascular
- Vasculitis
- Pericarditis

Haematological
Felty's syndrome
- Splenomegaly
- Neutropenia
- Anaemia

Musculoskeletal
General
- Symmetrical polyarticular joint swelling
- Joint redness, warmth, pain, tenderness
- Morning stiffness

Spine
- Cervical pain
- Neurological manifestations

Wrists
- Limited range of motion
- Deformity
- Carpal tunnel syndrome

Hands
- Ulnar deviation
- Swan-neck deformity
- Boutonnière deformity

Knees
- Joint effusion
- Instability

Ankles
- Limited range of motion
- Pain on ambulation

Feet
- Subluxation
- Hallux valgus
- Lateral toe deviation
- Cock-up toe

Integumentary
- Rheumatoid nodules

Metabolic processes
- Fatigue
- Weakness
- Anorexia
- Weight loss
- Low-grade fever

BOX 39.4 Diagnostic criteria for rheumatoid arthritis

- Morning stiffness lasting for at least 1 hour and persisting for at least 6 weeks
- Arthritis with swelling or effusion of three or more joints persisting for at least 6 weeks
- Arthritis of wrist, MCP or PIP joints persisting for at least 6 weeks
- Symmetrical arthritis with simultaneous involvement of corresponding joints on both sides of the body
- Rheumatoid nodules
- Positive serum rheumatoid factor
- Characteristic radiological changes of rheumatoid arthritis noted in hands and wrists (American Rheumatism Association, 2015).

quackery. Many non-traditional treatments, including diets, topical preparations, vaccines, hormones, plant extracts and copper bracelets, have been put forth. These treatments are often costly and none has been shown to be effective.

Diagnosis

Diagnostic tests are used to help establish the diagnosis of RA. Testing is also used to rule out other forms of arthritis and connective tissue disorders. Diagnostic tests are described in Chapter 37.

Laboratory tests are used to measure rheumatoid factors and the ESR, which is typically elevated. A full blood count (FBC) is done to identify anaemia. Diagnosing RA in the early stages is often difficult, but a new test is highly effective. In this blood test, people are tested for antibodies to cyclic citrullinated peptide (CCP) with accurate detection of early RA.

Synovial fluid examination will demonstrate changes associated with inflammation, including increased turbidity (cloudiness), decreased viscosity and increased protein and WBC levels. X-rays of affected joints are the most specific test for diagnosis of RA. Early in the disease, few changes may be evident other than soft tissue swelling and joint effusions. As the disease progresses, joint space narrowing and erosions are seen.

Medications

Four general approaches are used in the pharmacological management of people with RA:

1. Aspirin and other NSAIDs and mild analgesics are used to reduce the inflammatory process and manage the manifestation of the disease. Although these drugs may relieve manifestations of RA, they appear to have little effect on disease progression.
2. The second approach uses low-dose oral corticosteroids to reduce pain and inflammation. Recent studies suggest that low-dose oral corticosteroids also may slow the development and progression of bone erosions associated with RA.
3. A diverse group of drugs classified as disease-modifying or slow-acting antirheumatic drugs are employed in the third approach to treating RA. These drugs, which include gold compounds, D-penicillamine, antimalarial agents, infliximab and sulfasalazine, appear to alter the course of the disease, reducing its destruction of joints. Immunosuppressive and cytotoxic drugs are included in this category as well.
4. Intra-articular corticosteroids may be used to provide temporary relief in people for whom other therapies have failed to control inflammation.

ASPIRIN Aspirin is often the first drug prescribed in the treatment of RA unless its use is contraindicated for the person. Aspirin is an inexpensive and effective anti-inflammatory and analgesic agent. The dose of aspirin required to achieve a therapeutic blood level of 15 to 30 mg/dL and its full anti-inflammatory effect is approximately 4 g per day in divided doses (three or four 5 g (325 mg) tablets qid). This effective dose is just under the toxic dose, which produces tinnitus and hearing loss. The person may be instructed to increase the dose of aspirin gradually until either maximal improvement or toxicity occurs. If tinnitus develops, the person reduces the dose by two to three tablets per day until the tinnitus stops.

Gastrointestinal side effects and interference with platelet function are the greatest hazards of aspirin therapy. People are instructed to take aspirin with meals, milk or antacids to minimise gastrointestinal distress and reduce the risk of GI bleeding. Enteric-coated forms of aspirin and non-acetylated salicylate compounds produce less gastric distress than plain or buffered aspirin and reduce the risk of gastric ulceration, but they are more expensive. Salsalate (Disalcid, Mono-Gesic, Salflex) and choline magnesium trisalicylate (Trilisate, Tricosal) are examples of non-acetylated salicylate products. All salicylate products are contraindicated for people with a history of aspirin allergy.

OTHER NON-STEROIDAL ANTI-INFLAMMATORY DRUGS A number of other NSAIDs are available for use in the management of RA if aspirin is not tolerated or effective. All NSAIDs act by inhibiting prostaglandin synthesis. Although the efficacy of all NSAIDs, including aspirin, is equivalent, people's responses are individual. Several trials of different NSAIDs may be necessary to find the most effective drug.

Some NSAIDs are considerably more expensive than aspirin but may cause less gastrointestinal distress and require fewer doses per day. Gastric irritation, ulceration and bleeding remain the most common toxic effects of NSAIDs. They can also affect the lower intestinal tract, leading to perforation or aggravation of inflammatory bowel disorders. All NSAIDs can also be toxic to the kidneys.

NSAIDs commonly prescribed for people with RA are listed in Table 39.4. The TGA (2014) has issued planned regulatory actions for both prescription and over-the-counter (OTC) non-selective NSAIDs. These actions include increased label warnings about the potential serious adverse cardiovascular and gastrointestinal effects of these drugs; the non-prescription drugs include those containing ibuprofen, naproxen and ketoprofen. Nursing implications for the administration of NSAIDs are described in Chapter 11.

TABLE 39.4 Examples of non-steroidal anti-inflammatory drugs used to treat rheumatoid arthritis

DRUG	AVERAGE DOSE	COMMENTS AND PRECAUTIONS
Aspirin	600–900 mg 4 to 6 times daily	Least expensive NSAID; associated with risk of GI ulceration, bleeding and possible haemorrhage; may cause hepatotoxicity
Diclofenac (Voltaren)	50 mg tds or qid; or 75 mg bd	Expensive; risk of hepatotoxicity
Celecoxib	100 mg bd	GIT ulceration and bleeding. Adverse renal effects in some individuals
Flurbiprofen (Ansaid)	50–100 mg tds or qid, not to exceed 300 mg/day	Expensive
Ibuprofen (Nurofen, Advil others)	300 mg qid; 400–800 mg tds or qid	Available in prescription and OTC forms; less gastric distress reported than with aspirin or indomethacin; discontinue if visual disturbances develop
Indomethacin (Indocin)	25–50 mg bd or tds	A potent NSAID used for moderate to severe RA and acute episodes of chronic disease; higher incidence of adverse GI effects and CNS effects such as headache, dizziness and depression
Ketoprofen (Orudis)	50–75 mg tds or qid	Expensive; older adults and people with renal insufficiency require lower doses
Ketorolac	10 mg (max 40 mg)	Increased risk of GI bleeding and other severe effects increase with duration of treatment
Naproxen (Aleve, Anaprox, Naprosyn)	250–500 mg bd	Available in prescription and OTC preparations
Piroxicam (Feldene)	20 mg daily in a single or divided dose	Expensive; GI side effects including stomatitis, anorexia and gastric distress may occur more frequently than with other NSAIDs. Contraindicated in people with renal impairment
Sulindac (Clinoril)	150–200 mg bd	May be safer for use than other NSAIDs in people with chronic renal disease; rare fatal hypersensitivity reaction with fever, liver function abnormalities and severe skin reaction

bd = twice daily; qid = four times daily; tds = three times daily.

CORTICOSTEROIDS Systemic corticosteroids can dramatically relieve the symptoms of RA and appear to slow the progression of joint destruction. The long-term use of corticosteroids is associated with multiple side effects, such as poor wound healing, increased risk of infection, osteoporosis and gastrointestinal bleeding. Severe rebound manifestations can occur when these medications are discontinued. For these reasons, the use of systemic corticosteroids is limited to low dosages daily. The nursing implications for corticosteroid therapy are discussed in Chapter 12.

DISEASE-MODIFYING DRUGS Disease-modifying drugs are a diverse group of medications including drugs that modify immune and inflammatory responses, gold salts, antimalarial agents, sulfasalazine and D-penicillamine (see Table 39.5). They share characteristics that make them useful in the treatment of RA. Although beneficial effects are not apparent for several weeks or months following the initiation of therapy, they can produce not only clinical improvement but also evidence of decreased disease activity. Because their anti-inflammatory effect is minimal, NSAIDs are continued during therapy. As many as two-thirds of people taking disease-modifying drugs show improvement, although these drugs have not been shown to slow bone erosion or facilitate healing. All of these drugs are fairly toxic and close monitoring is necessary during the course of therapy.

Drugs that modify the autoimmune and inflammatory responses in people with RA include leflunomide (Arava) and etanercept (Enbrel). Leflunomide reversibly inhibits an enzyme involved in the autoimmune process and etanercept inhibits the binding of tumour necrosis factor to receptor sites. Infliximab (Remicade) is a biological response modifier and TNF-α receptor antagonist. Given by intravenous infusion, the drug is administered to reduce infiltration of inflammatory cells and TNF-α production. Adalimumab (Humira) is a biological response modifier that is given to people with RA to reduce the inflammatory events of polyarthritis and slow the progression of joint damage. Given by subcutaneous injection, the drug cannot be administered if the person has an acute or chronic infection in any part of the body. Prior to initiating the drug, the person should be tested for tuberculosis.

Gold salts may be administered by mouth but the intramuscular route is preferred because it is more effective. The mode of action of gold is unknown but it may produce clinical remission in some people and decrease new bony erosions. Weekly therapy is continued until significant improvement is noted unless toxic reactions occur. People experiencing benefit from gold therapy may be continued on monthly injections for several years. About one-third of people on gold therapy experience toxic reactions, including dermatitis, stomatitis, bone marrow depression and proteinuria. Mild skin reactions do not always necessitate discontinuation of therapy. FBC and

TABLE 39.5 Disease-modifying drugs used to treat rheumatoid arthritis

CLASS/MEDICATIONS	USUAL DOSE	ADVERSE EFFECTS	COMMENTS/NURSING RESPONSIBILITIES
Gold salts Gold sodium thiomalate (Myochrysine) Aurothioglucose (Solganal) Auranofin (Ridaura Capsules)	Parenteral: 1st dose 10 mg; 2nd dose 25 mg, then 50 mg weekly IM Oral: 6 mg daily	• Pruritus, dermatitis • Stomatitis, metallic taste • Renal toxicity • Blood dyscrasias • Gastrointestinal distress	• Frequent UA and FBC • Monitor the person after injection for flushing, fainting, dizziness, sweating, possible anaphylactic reaction
Antimalarial Hydroxychloroquine (Plaquenil)	200–600 mg daily with meals	• CNS reactions including irritability, nightmares, psychoses • Retinopathy • Alopecia, pruritus • Blood dyscrasias • GI disturbances	• Should not be used during pregnancy • Regular ophthalmological examination required
Other Sulfasalazine (Azulfidine)	2 g/day in divided doses with meals	• Anorexia, nausea, vomiting, gastric distress • Decreased sperm count • Headache • Rash • Blood dyscrasias • Hypersensitivity responses including Stevens–Johnson syndrome • CNS, liver and renal toxicity	• Administer in evenly divided doses • Maintain high fluid intake • May cause yellow-orange skin or urine discolouration • Regular FBC necessary
Penicillamine (Cuprimine, Depen Titratable)	125–250 mg/day initially, slowly increased to a total of 1000–1500 mg/day	• Skin rashes • Fever • Gastrointestinal distress • Oral ulcers, loss of taste • Fever • Bone marrow depression with thrombocytopenia, leucopenia, anaemia • Renal toxicity • May induce immune complex disorders such as Goodpasture's syndrome and myasthenia gravis	• Regular FBC and UA necessary • Administer on an empty stomach • Discontinue during pregnancy • May require 2 to 3 months of therapy before benefit is seen

CNS = central nervous system; FBC = full blood count; UA = urinalysis.

urinalysis are monitored throughout treatment with gold to assess for more severe toxic responses.

Hydroxychloroquine (Plaquenil) is an antimalarial agent sometimes employed in the treatment of RA. Three to 6 months of therapy is required to achieve the desired response and many people do not experience significant benefit. Although hydroxychloroquine has a relatively low toxicity, it can cause pigmentary retinitis and vision loss. People receiving this drug require a thorough vision examination every 6 months.

Sulfasalazine, a drug regularly prescribed for chronic inflammatory bowel disease, may also be prescribed for RA. See Chapter 23 for further discussion of this drug and its nursing implications.

For people not responding to the above preparations, penicillamine may be prescribed. Although this agent may be effective in the management of RA, toxic reactions are common and can be severe, including bone marrow suppression, proteinuria and nephrosis.

IMMUNOSUPPRESSIVE THERAPY Immunosuppressive or cytotoxic drugs are increasingly employed in the management of RA. Indeed, many now consider methotrexate the treatment of choice for people with aggressive RA. Methotrexate may be used along with NSAIDs in the initial treatment plan. A weekly dose can produce a beneficial effect in as few as 2 to 4 weeks. Gastric irritation and stomatitis are the most frequent side effects associated with methotrexate, but side effects may be better controlled if folic acid is taken at the same time. Alcoholism, diabetes, obesity, advanced age and renal disease increase the risk of toxic effects (hepatotoxicity, bone marrow suppression, interstitial pneumonitis).

Other immunosuppressive agents such as cyclosporin, azathioprine and monoclonal antibodies have also been employed in the treatment of people with severe, progressive, crippling disease who have failed to respond to other measures.

Treatments

The primary objectives in treating RA are to reduce pain and inflammation, preserve function and prevent deformity.

REST AND EXERCISE A balanced program of rest and exercise is an important component in the management of people with RA. During an acute exacerbation of the disease, the person may be hospitalised or a short period of complete bed rest may be prescribed. For most people, regular rest periods during the day are beneficial to reduce manifestations of the disease. Additionally, splinting of inflamed joints reduces unwanted motion and provides local joint rest. A variety of orthotic devices are available to reduce joint strain and help maintain function.

Rest must be balanced with a program of physical therapy and exercise to maintain muscle strength and joint mobility. ROM exercises are prescribed to maintain joint function and prevent contractures. Isometric exercises are used to improve muscle strength without increasing joint stress. Isotonic exercises also help improve muscle strength and preserve function. Low-impact aerobic exercises, such as swimming and walking, have been shown to benefit people with RA without adversely affecting joint inflammation or prompting acute episodes.

PHYSICAL AND OCCUPATIONAL THERAPY Physical and occupational therapists can design and monitor individualised activity and rest programs.

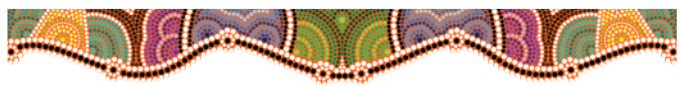
HEAT AND COLD Heat and cold are used for their analgesic and muscle-relaxing effects. Moist heat is generally the most effective and can be provided by a tub bath. Joint pain is relieved in some people through the application of cold.

ASSISTIVE DEVICES AND SPLINTS Assistive devices, such as a cane, walker or raised toilet seat, are most useful for people with significant hip or knee arthritis. Splints provide joint rest and prevent contractures. Night splints for the hands and/or wrists should maintain the extremity in a position of maximum function. The best 'splint' for the hip is lying prone for several hours a day on a firm bed. In general, splints should be applied for the shortest period needed, should be made of lightweight materials and should be easily removed to perform ROM exercises once or twice a day.

NUTRITION For most people with RA, an ordinary, well-balanced diet is recommended. Some people may benefit from substitution of usual dietary fat with omega-3 fatty acids found in certain fish oils.

SURGERY Surgical intervention may be employed for the person with RA at a variety of disease stages. Early in the course of the disease, synovectomy (excision of synovial membrane) can provide temporary relief of inflammation, relieve pain and slow the destructive process, helping to preserve joint function. Arthrodesis (joint fusion) may be used to stabilise joints such as cervical vertebrae, wrists and ankles. Arthroplasty, or total joint replacement, may be necessary in cases of gross deformity and joint destruction. Total joint replacement and nursing care of people undergoing this surgery are discussed in the preceding section on OA.

OTHER THERAPIES Several newer treatments that are not yet in widespread use may be employed in people with progressive RA. Plasmapheresis has been used to remove circulating antibodies, moderating the autoimmune response. Total lymphoid irradiation decreases total lymphocyte levels, although serious adverse effects are associated with this treatment and its continued efficacy has not been established.



Nursing care

People with chronic, progressive, systemic disorders such as RA have multiple nursing care needs involving many functional health patterns. Physical manifestations of the disease often result in acute and chronic pain, fatigue, impaired mobility and difficulty performing routine tasks. The disease also has many psychosocial effects. The person has an incurable chronic disease that may lead to severe crippling. Pain and fatigue can interfere with the person's ability to perform expected roles, such as home maintenance or job responsibilities. Even though the person's hands may appear swollen or deformed, other people may not understand the systemic nature of the disease or appreciate the difference between RA and OA. A nursing care plan for a person with RA is found below.

Health promotion

People with RA have control of their lives by becoming arthritis self-managers. They can help prevent deformities and the effects of arthritis by following prescriptions for exercise, rest, weight management, posture and positioning. The following suggestions are recommended:

- Respect pain as a warning signal. When pain is experienced, change the method of doing things, use equipment or tools if necessary and take intermittent rest periods. Prolonged rest may lead to decreased range of motion.
- A structured exercise program greatly improves the wellbeing of RA sufferers. Stretching, strengthening and aerobic conditioning are all important in the rehabilitation of RA.
- Superficial heat has great effect on hands and feet due to less subcutaneous tissues in these areas. Heat packs and hydrotherapy are excellent for prevention of pain.
- Use adaptive equipment to assist with functional independence (Johns Hopkins Arthritis Center, 2016).

Assessment

Collect the following data through the health history and physical examination (see Chapter 37):

- *Health history:* pain, stiffness, fatigue, joint problems: location, duration, onset, effect on function, fever, sleep patterns, past illnesses or surgery, ability to carry out ADLs and self-care activities.

- *Physical assessment:* height/weight; gait; joints: symmetry, size, shape, colour, appearance, temperature, range of motion, pain; skin: nodules, purpura; respiratory: cough, crackles; cardiovascular: pericardial friction rub, apical bradycardia, S_3.

Nursing diagnoses and interventions

Many nursing diagnoses may be appropriate for the person with RA. This section focuses on those related to its predominant manifestations and their effect on the person's life.

Chronic pain

Pain is a constant feature of RA when the disease is active. Pain accompanies both acute inflammation and lower levels of chronic inflammation. Some people say the pain in joints and surrounding tissue is like a deep, constant toothache. Pain can significantly affect the person's ability to provide self-care and maintain daily activities. It also contributes to the person's fatigue.

- Monitor the level of pain and duration of morning stiffness. *Pain and morning stiffness are indicators of disease activity. Increased pain may necessitate changes in the therapeutic treatment plan.*
- Encourage the person to relate pain to activity level and adjust activities accordingly. Teach the importance of joint and whole-body rest in relieving pain. *Pain is an indicator of excess stress on inflamed joints. Increasing pain indicates a need to decrease activity levels.*
- Teach the use of heat and cold applications to provide pain relief. The person may apply heat by showering or taking tub baths or using warm compresses or other local applications such as paraffin dips. *For people who find that heat increases pain and swelling during periods of acute inflammation, cold packs may be more effective. Both heat and cold have analgesic effects and can help relieve associated muscle spasms.*
- Teach about the use of prescribed anti-inflammatory medications and the relationship of pain and inflammation. *Anti-inflammatory agents reduce chemical mediators of inflammation and swelling, relieving pain.*
- Encourage using other non-pharmacological pain relief measures such as visualisation, distraction, meditation and progressive relaxation techniques. *These techniques can reduce muscle tension and help the person focus away from the pain, decreasing the intensity of the pain experience.*

Fatigue

The pain and chronic inflammatory processes associated with RA lead to fatigue. Other factors contribute as well. Discomfort often disrupts the person's sleep patterns. Anaemia, muscle atrophy and poor nutrition also play a role in the development of fatigue. The person with RA may experience depression or hopelessness, with associated manifestations of fatigue.

- Encourage a balance of periods of activity with periods of rest. *Both joint and whole-body rest are important to reduce the inflammatory response.*
- Stress the importance of planned rest periods during the day. *Rest is vital during acute exacerbations of the disease but also important to maintain the person in remission.*
- Help in prioritising activities, performing the most important ones early in the day. *Assigning priorities helps the person avoid performing relatively unimportant activities at the expense of more meaningful and important ones.*
- Encourage regular physical activity in addition to prescribed ROM exercises. *Aerobic exercise promotes a sense of wellbeing and restful sleep patterns.*
- Refer to counselling or support groups. *Counselling and support groups can help the person develop effective coping strategies and deal with depression and hopelessness.*

NURSING CARE PLAN A person with rheumatoid arthritis

Joanne James is a 42-year-old high school science teacher who began noticing vague joint pain, fatigue, poor appetite and general malaise, which she initially attributed to a case of the flu. However, her symptoms continued and she reports feeling very stiff in the mornings, often taking until 10 or 11 am to begin to feel 'normal'. She then began to notice aching in her hands and wrists, which she attributed to the quilting she loves to do in the evenings. She made an appointment with her family GP when she noticed that her knuckles and finger joints were not just achy but also swollen and hot. Noting that Mrs James has lost 5 kg since her last visit and has mild anaemia and a significantly elevated ESR, the GP referred her to the rheumatology clinic for further evaluation. Following examination, laboratory and radiological testing, the rheumatologist established a diagnosis of rheumatoid arthritis and initiated a multidisciplinary team conference to plan the management of Mrs James's condition.

ASSESSMENT

Cathy Greenstein, RN, completes an assessment of Mrs James. She notes that Mrs James is well groomed and answers questions readily but appears fatigued and ill. Mrs James relates that her job has been extremely stressful because teacher layoffs have resulted in larger class sizes and fewer teaching assistants. Despite her symptoms, she continues to teach full time, but says she feels unable to keep up with all her responsibilities due to her fatigue.

Mrs James states that she is allergic to penicillin. Her past medical history reveals only the usual childhood diseases and three uncomplicated pregnancies, resulting in the births of her children, aged 14, 11 and 9. Physical assessment findings include BP 124/78, P 82 regular, R 18, T 37.8°C PO. Hands: swelling of the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of both hands; second and third PIP and second MCP joints on right hand are red, shiny, hot, spongy and tender to palpation; able to extend fingers to

(continued)

NURSING CARE PLAN A person with rheumatoid arthritis (continued)

180 degrees but cannot make a complete fist with either hand, with flexion limited to less than 90 degrees; grip strength is weak bilaterally; wrist ROM is limited in all directions. Knees are swollen and flexion is slightly limited; positive bulge sign in the right knee. Diagnostic findings are an ESR of 52 mm/h, a haematocrit of 30% and positive for rheumatoid factor. Few changes other than soft tissue swelling are evident on hand and wrist x-rays.

DIAGNOSES

- *Chronic pain* related to joint inflammation.
- *Impaired home maintenance* related to fatigue.
- *Activity intolerance* related to the effects of inflammation.
- *Deficient knowledge of therapeutic regimen*.

PLANNING

Manage Mrs James's pain and educate her in relation to how to manage her condition and continue to undertake her usual activities.

Expected outcomes

- Verbalise effective pain management strategies:
  - Verbalise a plan to reduce responsibilities for home maintenance.
  - Express a willingness to plan rest breaks during the day.
- Demonstrate understanding of the prescribed therapeutic regimen and its importance for both short- and long-term benefit.

IMPLEMENTATION

- Use assistive devices to minimise joint stress with ADLs.
- Teach techniques for relieving pain and morning stiffness, including:
  - Schedule NSAIDs at equal intervals throughout the day.
  - Take morning NSAID dose with milk and crackers approximately 30 minutes before rising.
  - Perform ROM exercises in shower or bathtub.
  - Apply local heat with paraffin dip or compress, use cold packs as needed.
  - Teach techniques to minimise joint stress while performing ADLs.
  - Discuss ways to delegate household tasks to other family members.
  - Explore ways to incorporate 30-minute rest breaks into work schedule.
  - Provide arthritis literature and information.
  - Provide information about the disease process and its manifestations, prescribed medications with desired and adverse effects, and the importance of balancing rest and activity.

EVALUATION

The initial treatment regimen of aspirin, rest, exercise and physical therapy succeeded in partially relieving the acute manifestations of rheumatoid arthritis in Mrs James. However, complete remission has not been achieved. She has had difficulty scheduling rest periods at work and has had to struggle to delegate household tasks. 'I don't look sick to the kids and they seem to think housecleaning is a terrible imposition on their time. It's often easier to just do it myself than to fight about it. Besides, that way it gets done right.' Mrs James has faithfully followed the prescribed medication regimen and exercise routines and she has kept her scheduled appointments and maintained contact with the treatment team.

CRITICAL THINKING IN THE NURSING PROCESS

1. Mrs James is 42 years old. Would your nursing interventions differ if she were 72 years old? If so, how?
2. Rheumatoid arthritis is a chronic illness. What are the physical, emotional and economic implications of a chronic illness that results in chronic pain and deformity?
3. Develop a nursing care plan for Mrs James using the nursing diagnosis of *Ineffective role performance*.

REFLECTION ON THE NURSING PROCESS

1. Identify issues from this case study that would enable you to care for people with rheumatoid arthritis in your future practice.
2. For a person newly diagnosed with rheumatoid arthritis, which education strategies would be appropriate when caring for this person?

Ineffective role performance

Fatigue, pain and the crippling effects of RA can interfere with the person's ability to pursue a career and fill other life roles, such as parent, spouse or homemaker. As the person's role changes, so must the roles of other family members. This can contribute to changes in family processes, increased stress in the family and further difficulty coping with the effects of the disease.

- Discuss the effects of the disease on the person's career and other life roles. Encourage the person to identify changes brought on by the disease. *Discussion helps the person to accept the changes and begin to identify strategies for coping with them.*
- Encourage the person and family to discuss their feelings about role changes and grieve lost roles or abilities. *Verbalisation allows family members to validate and accept feelings about losses and changes, thus helping them to move into new roles.*
- Listen actively to concerns expressed by the person and family members; acknowledge the validity of concerns about the disease, prescribed treatment and the prognosis. *Demonstrating acceptance of these feelings and concerns promotes trust and validates their reality.*

CONSIDERATION FOR PRACTICE

Remember that grief resolution takes time and that people may respond to loss with anger.

- Help the person and family identify strengths they can use to cope with role changes. *Identifying strengths helps the*

person and family to consider role changes that maintain self-esteem and dignity.

- Encourage the person to make decisions and assume personal responsibility for disease management. *People who assume a personal and active role in managing their disease maintain a greater sense of self-control and self-esteem.*

Disturbed body image

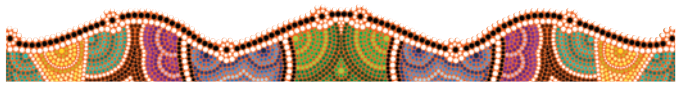
The acute and long-term effects of RA can affect the person's body image, leading to feelings of hopelessness and powerlessness, social withdrawal and difficulty adapting to changes. When inflammation and joint deformity occur despite compliance, the person may have difficulty accepting the need to continue therapeutic measures, particularly those that have side effects or are costly or time consuming. In addition, unproven alternative treatment strategies and quackery may become increasingly attractive to the person.

- Demonstrate a caring, accepting attitude towards the person. *This attitude helps the person accept the physical changes brought on by the disease.*
- Encourage the person to talk about the effects of the disease—both physical effects and effects on life roles. *Verbalisation helps the person identify feelings and gives the nurse an opportunity to validate these feelings.*
- Encourage the person to maintain self-care and usual roles to the extent possible. *Discuss the use of clothing and adaptive devices that promote independence. Independence enhances the person's self-esteem.*
- Provide positive feedback for self-care activities and adaptive strategies. *Positive reinforcement encourages the person to continue adaptive measures and maintain independence.*
- Refer to self-help groups, support groups and other agencies that provide assistive devices and literature. *These groups and agencies can help the person develop adaptive strategies to cope with the effects of RA, enhancing the person's self-concept, body image and independence.*

Community-based care

RA is typically a chronic, progressive disease. As with most diseases of this nature, involvement of the person and family in its management is vital. Education is an important nursing role in caring for people with RA and their families. Address the following topics for home care of the person and for family members:

- disease process and treatments, including rest and exercise
- medications
- management of stiffness and pain
- energy conservation
- use of assistive devices to maintain independence, including self-care aids such as handheld showers, long-handled brushes and shoehorns, and eating utensils with oversized or special handles
- clothing options such as elastic waist pants without zippers, Velcro closures, zippers with large pull-tabs and slip-on shoes
- how to apply splints and take care of skin
- home and equipment modifications, such as a raised toilet seat, grab bars in the bathroom, a bath chair or adapted counter heights for people in a wheelchair
- physical therapy, occupational therapy, community services and home care services
- helpful resources:
  - Arthritis Australia: www.arthritisaustralia.com.au
  - Arthritis New Zealand: www.arthritis.org.nz
  - Australian Rheumatology Association: www.rheumatology.org.au
  - National Institute of Arthritis and Musculoskeletal and Skin Diseases: www.niams.nih.gov
  - New Zealand Rheumatology Association: www.rheumatology.org.nz.



THE PERSON WITH ANKYLOSING SPONDYLITIS

Ankylosing spondylitis (AS) is a chronic inflammatory arthritis that primarily affects the axial skeleton, leading to pain and progressive stiffening and fusion of the spine. The typical age of onset is between 15 and 45. The incidence is greater in men than women and men have more severe disease. AS is difficult to diagnose in the early stages, but may be a major cause of persistent back pain in young adults.

The cause of ankylosing spondylitis is unknown. As with the other spondyloarthropathies, there is a strong genetic component. Approximately 90% of people with AS have the HLA-B27 antigen; about 8% of the general population have this antigen (Porth & Matfin, 2014).

Pathophysiology

Early inflammatory changes are often first noted in the sacroiliac joints. As the cartilage erodes, joint margins ossify and are replaced by scar tissue. The joints of the spine are also affected, with inflammation of the cartilaginous joints and gradual calcification and ossification that leads to ankylosis or joint consolidation and immobility. Other organ systems may be affected as well, including the eyes, lungs, heart and kidneys.

Manifestations

The onset of ankylosing spondylitis is usually gradual and insidious. People may have persistent or intermittent bouts of lower back pain. The pain is worse at night, followed by morning stiffness that is relieved by activity. Pain may radiate to the buttocks, hips or down the legs. As the disease progresses, back motion becomes limited, the lumbar curve is lost and the thoracic curvature is accentuated. In severe cases, the entire spine becomes fused, preventing any motion. People with AS may also experience peripheral arthritis, primarily affecting the hip, shoulders and knee joints. Systemic manifestations include anorexia, weight loss, fever and fatigue. Many people develop uveitis (inflammation of the iris and the middle, vascular layer of the eye).

For most people with AS, the disease is intermittent with mild to moderate acute episodes. These people have a good prognosis with little risk of severe disability.

INTERPROFESSIONAL CARE

Diagnostic testing shows an elevated ESR during periods of active disease and typically a positive HLA-B27 antigen. The diagnosis of ankylosing spondylitis is usually confirmed with x-ray examination of the sacroiliac joints and spine. The sacroiliac joint becomes blurred and gradually obliterated. As the disease progresses, vertebrae become squared and disc spaces narrow.

As with other forms of arthritis, the management of AS is multidimensional. Physiotherapy and daily exercises are important to maintain posture and joint ROM. NSAIDs relieve pain and stiffness and allow the person to perform necessary exercises. Indomethacin (Indocin) is the NSAID most commonly used to treat AS. It may, however, have many adverse effects, including headache, nausea and vomiting, depression and psychosis. Other drugs that may be prescribed include sulfasalazine (Azulfidine) and topical or intra-articular corticosteroids. Severe hip joint arthritis may necessitate total hip arthroplasty.

Nursing care

The primary nursing role in ankylosing spondylitis is to provide supportive care and education. To promote mobility, teach the person to take NSAIDs at regular intervals throughout the day with food, milk or antacid. Encourage the person to maintain a fluid intake of 2500 mL or more per day. Suggest that the person perform exercises in the shower because warm, moist heat prompts mobility. Stress the importance of following the prescribed physical therapy and exercise program to maintain mobility.

Teach the person that proper positioning and posture are important. When sleeping, a bed board may be used to provide firmness and the person should sleep in the supine position using either no pillow or only one small pillow. Other important self-care activities include losing weight if applicable, avoiding smoking and using muscle-strengthening exercises. Suggest occupational counselling if pain and deformity are severe enough to cause work-related problems.

THE PERSON WITH REACTIVE ARTHRITIS

Reactive arthritis (ReA) (*Reiter's syndrome*) is an acute, non-purulent inflammatory arthritis that is believed to be a response to an exposure or infection with certain types of bacteria, including *Chlamydia* (a bacterium contracted during sexual activity) or *Salmonella*, *Shigella*, *Yersinia* or *Campylobacter* (which cause dysentery from contaminated or spoiled food). This type of arthritis most often affects young men who have an inherited HLA-B27 antigen. Reactive arthritis is often found in people with HIV infection, although the reason for the association is not clear. Reactive arthritis is typically self-limited, although it can be recurrent or progressive. About 15–20% of people with ReA develop a chronic arthritis or spondylitis (Spondylitis Association of America, 2009).

Manifestations

Non-bacterial urethritis is often the initial manifestation of Reiter's syndrome. In women, urethritis and cervicitis may be asymptomatic. Conjunctivitis and inflammatory arthritis follow. The arthritis is usually asymmetric, affecting large weight-bearing joints such as the knees and ankles, the sacroiliac joints or the spine. Mouth ulcers, inflammation of the glans penis and skin lesions may occur. The heart and aorta may also be affected.

INTERPROFESSIONAL CARE

The diagnosis of reactive arthritis is based on the person's history and presenting symptoms. Manifestations of ReA typically occur 2 to 4 weeks after the infection and subside in 3 to 12 months. The condition has a tendency to recur. No test is specific for the disorder. Urethral or cervical cultures are obtained to rule out gonococcal infection. When *Chlamydia* is suspected, the person and sexual partner are treated with tetracycline or erythromycin. Reactive arthritis is treated symptomatically, usually with NSAIDs.

Nursing care

People with reactive arthritis usually are seen in primary care settings such as a clinic or GP's office, making the nursing role primarily one of education. Teach the person about the association of the arthritis with the precipitating infection (if identified). Stress the importance of treating the infection effectively if it is still present. Use this opportunity to provide information about sexually transmitted infections and protective measures to prevent their transmission (see Chapter 49). Discuss the usual self-limiting nature of ReA, the appropriate use of prescribed NSAID preparations and symptomatic relief measures such as application of heat and rest.

THE PERSON WITH SYSTEMIC LUPUS ERYTHEMATOSUS

Systemic lupus erythematosus (SLE) is a chronic inflammatory connective tissue disease. It affects almost all body systems, including the musculoskeletal system. The manifestations of SLE are widely variable and are thought to result from cell and tissue damage caused by deposition of antigen–antibody complexes in connective tissues. SLE affects multiple body systems and can range from a mild, episodic disorder to a rapidly fatal disease process.

Although the exact aetiology of SLE is unknown, genetic, environmental and hormonal factors play a role in its development. Twin studies and a familial pattern of the disease point to a genetic component, as does an increased incidence of other connective tissue diseases in relatives of people with SLE. Certain human leucocyte antigen (HLA) genes are seen more frequently in people with SLE. Environmental factors such as viruses, bacterial antigens, chemicals, drugs or ultraviolet light may play a role in activation of the pathological mechanisms of the disease. In addition, it is felt that sex hormones may influence the development of SLE. Women with SLE have reduced levels of several active androgens that are known to inhibit antibody responses. Oestrogens have been shown to enhance antibody responses and have an adverse effect in people with SLE.

The course of SLE is mild in most people, with periods of remission and exacerbation. The number and severity of exacerbations tend to decrease with time. In some people, however, SLE is a virulent disease with significant organ system involvement.

People with active disease have an increased risk of infections, which are often opportunistic and severe. Infections such as pneumonia and septicaemia are the leading cause of death in people with SLE, followed by the effects of renal or central nervous system (CNS) involvement. See the multisystem effects of SLE overleaf.

Pathophysiology

The pathophysiology of SLE involves the production of a large variety of autoantibodies against normal body components such as nucleic acids, erythrocytes, coagulation proteins, lymphocytes and platelets. Autoantibody production results from hyper-reactivity of B cells (humoral response) because of disordered T-cell function (cellular immune response). The most characteristic autoantibodies in SLE are produced in response to nucleic acids, including DNA, histones, ribonucleoproteins and other components of the cell nucleus.

SLE autoantibodies react with their corresponding antigen to form immune complexes, which are then deposited in the connective tissue of blood vessels, lymphatic vessels and other tissues. The deposits trigger an inflammatory response leading to local tissue damage. The kidneys are a frequent site of complex deposition and damage; other tissues affected include the musculoskeletal system, brain, heart, spleen, lung, GI tract, skin and peritoneum. The autoantibodies produced and their target tissue determine the manifestations of SLE.

A number of drugs can cause a syndrome that mimics lupus in people with no other risk factors for the disease. Procainamide (e.g. Procan-SR, Pronestyl) and hydralazine (Apresoline, Hydralyn) are the most common drugs implicated, along with isoniazid (INH).

Renal and CNS manifestations of SLE rarely occur with drug-induced lupus, but arthritic and other systemic symptoms are common. Manifestations of drug-induced lupus usually resolve when the medication is discontinued.

FAST FACTS

- Approximately 1 person in 3500 is affected by SLE in Australia and New Zealand, with women predominating by a ratio of 9:1 over men.
- SLE usually affects women of childbearing age (when the incidence is 30 times greater than in men) but it can occur at any age.
- SLE is more common in Polynesians in New Zealand (3.5 times greater than Europeans), with similar numbers in Indigenous Australians (Porth & Matfin, 2014).
- The incidence of SLE is higher in some families.

Manifestations

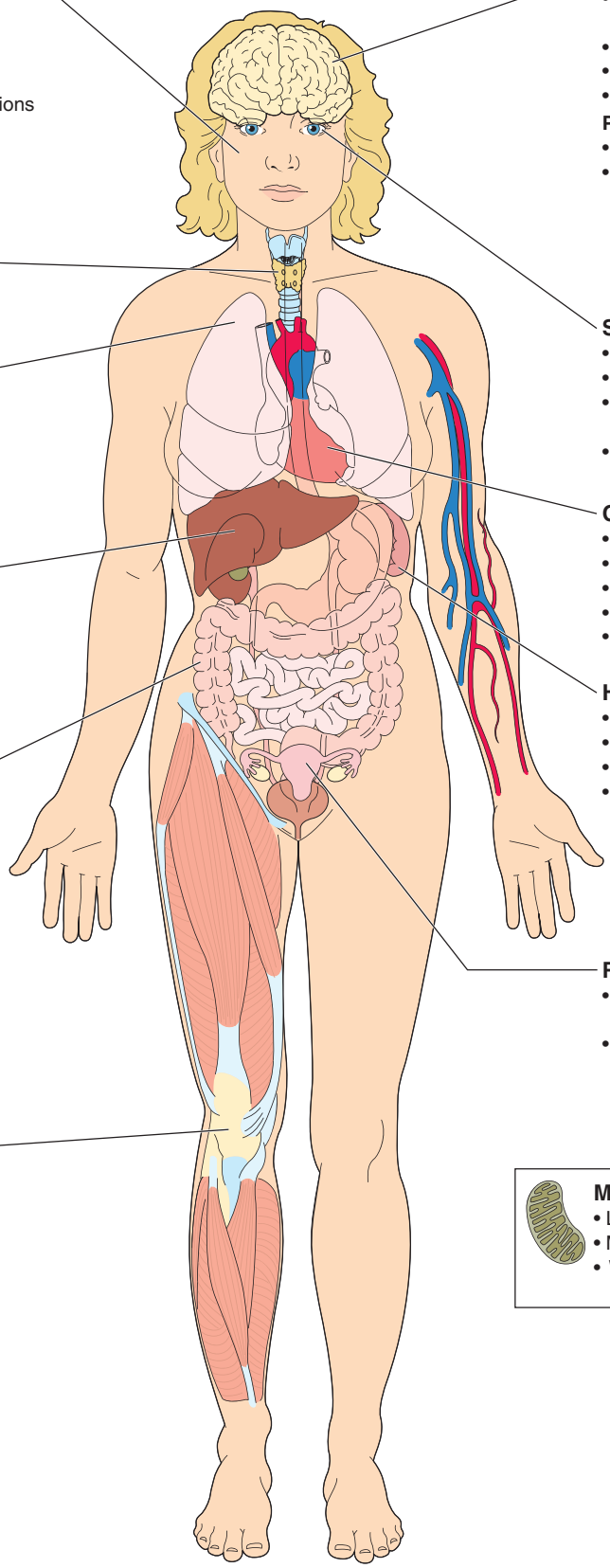
Typical early manifestations of SLE mimic those of rheumatoid arthritis, including systemic manifestations of fever, anorexia, malaise and weight loss, and musculoskeletal manifestations of multiple arthralgias and symmetric polyarthritis. Joint symptoms affect more than 90% of people with SLE. Although synovitis may be present, the arthritis associated with SLE is rarely deforming.

Most people affected by SLE have skin manifestations at some point during their disease. In fact, SLE was originally described as a skin disorder and named for the characteristic red butterfly rash across the cheeks and bridge of the nose (see Figure 39.7). Many people with SLE are photosensitive; a diffuse maculopapular rash on skin exposed to the sun is common. Other cutaneous manifestations include discoid lesions (raised, scaly, circular lesions with an erythematous rim), hives, erythematous fingertip lesions and splinter haemorrhages. Alopecia is common in people with SLE, although the hair usually grows back. Painless mucous membrane ulcerations may occur on the lips or in the mouth or nose. Common manifestations of SLE are listed in the box below.

Approximately 50% of people with SLE experience renal manifestations of the disease, including proteinuria, cellular casts and nephrotic syndrome. Up to 10% develop renal failure as a result of the disease.

Haematological abnormalities such as anaemia, leucopenia and thrombocytopenia are common with SLE. Cardiovascular disorders such as pericarditis, vasculitis and Raynaud's phenomenon often occur. Less frequently, myocarditis, endocarditis and venous or arterial thrombosis may develop.

MULTISYSTEM EFFECTS OF SYSTEMIC LUPUS ERYTHEMATOSUS



Integumentary
- Butterfly rash on face
- Photosensitivity
- Maculopapular rash on exposed body surfaces
- Discoid lesions
- Erythematous fingertip lesions
- Splinter haemorrhages
- Alopecia
- Ulcers (lip, mouth, nose)

Endocrine
- Thyroid abnormalities
- Hyperparathyroidism
- Glucose intolerance

Respiratory
- Pleurisy
- Pleural effusion
- Pneumonitis
- Interstitial fibrosis

Urinary
- Proteinuria
- Cellular casts

Potential complications
- Nephrotic syndrome
- Kidney failure

Gastrointestinal
- Anorexia
- Nausea
- Abdominal pain
- Diarrhoea
- Hepatomegaly

Musculoskeletal
- Arthralgias
- Symmetric polyarthritis
- Joint swelling and effusion
- Morning stiffness

Neurological
- Neuropathies (peripheral and central)
- Seizures
- Depression
- Psychosis

Potential complications
- Stroke
- Organic brain syndrome
  - Intellectual impairment
  - Memory loss
  - Personality changes
  - Disorientation

Sensory
- Conjunctivitis
- Photophobia
- Retinal vasculitis with transient blindness
- Cotton-wool spots on retina

Cardiovascular
- Pericarditis
- Myocarditis
- Endocarditis
- Vasculitis
- Venous or arterial thrombosis

Haematological
- Anaemia
- Leukopaenia
- Thrombocytopaenia
- Splenomegaly

Reproductive
- Pregnancy-induced hypertension, oedema and proteinuria
- Foetal loss

Metabolic processes
- Low-grade fever
- Malaise
- Weight loss

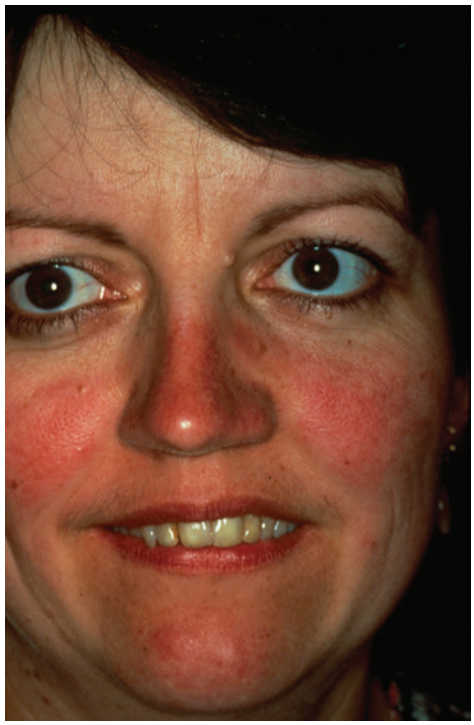


FIGURE 39.7 ■ The butterfly rash of systemic lupus erythematosus

Source: Wellcome Trust/Custom Medical Stock Photo.

Pleurisy, pleural effusions and lupus pneumonitis are common pulmonary manifestations of SLE.

Many people with SLE develop transient nervous system involvement, often within the first year of the disease. Organic brain syndrome manifestations include decline in intellect, memory loss and disorientation. Other possible neurological manifestations include psychosis, seizures, depression and stroke. Ocular manifestations of SLE include conjunctivitis, photophobia and transient blindness due to retinal vasculitis.

Gastrointestinal manifestations of SLE, such as anorexia, nausea, abdominal pain and diarrhoea, may affect up to 45% of people with the disease. The liver may be enlarged and liver function tests may yield abnormal results.

MANIFESTATIONS SLE

- Painful or swollen joints and muscle pain
- Unexplained fever
- Red rash, especially on the face
- Unusual loss of hair
- Pale, cyanotic fingers or toes
- Sensitivity to the sun
- Oedema in legs and around eyes
- Ulcers in the mouth
- Enlarged glands
- Extreme fatigue

INTERPROFESSIONAL CARE

Because of the diversity of organ system involvement and manifestations of SLE, diagnosis can be difficult. No one specific test is available to confirm the presence of this disease in all people suspected of having it. Instead, the diagnosis is based on the person's history and physical assessment, as well as laboratory studies.

As with rheumatoid arthritis, effective management of SLE requires teamwork, with active participation by both the person and members of the healthcare team. Although there is no cure for SLE, the 10-year survival rate is greater than 70% for people with this disease, which was once considered fatal in most cases.

Diagnosis

The multiple autoantibodies produced in SLE cause a number of abnormalities in laboratory tests. Diagnostic tests for the musculoskeletal system are described in Chapter 37.

- *Anti-DNA antibody testing* is a more specific indicator of SLE, because these antibodies are rarely found in any other disorder.
- *ESR* is typically elevated, occasionally to > 100 mm/h.
- *Serum complement levels* are usually decreased as complement is consumed or 'used up' by the development of antigen–antibody complexes.
- *FBC* abnormalities include moderate to severe anaemia, leucopenia and lymphocytopenia, and possible thrombocytopenia.
- *Urinalysis* (*UA*) shows mild proteinuria, haematuria and blood cell casts during exacerbations of the disease when the kidneys are involved. Renal function tests including *serum creatinine* and *blood urea nitrogen (BUN)* may also be ordered to evaluate the extent of kidney disease.
- *Kidney biopsy* may be performed to assess the severity of renal lesions and guide therapy (see Chapter 25).

Medications

The person with mild or remittent SLE may need little or no therapy other than supportive care. Arthralgias, arthritis, fever and fatigue can often be managed with aspirin or other NSAIDs. Aspirin is particularly beneficial for people with SLE because its antiplatelet effects help prevent thrombosis. It may, however, cause liver toxicity and hepatitis.

Skin and arthritic manifestations of SLE may be treated with antimalarial drugs such as hydroxychloroquine (Plaquenil). Hydroxychloroquine has also been shown to be effective in reducing the frequency of acute episodes of SLE in people with mild or inactive disease. Retinal toxicity and possibly irreversible blindness are the primary concerns with this drug. For this reason, the person taking hydroxychloroquine undergoes ophthalmological exam every 6 months.

People with severe and life-threatening manifestations of SLE (such as nephritis, haemolytic anaemia, myocarditis, pericarditis or CNS lupus) require corticosteroid therapy in high doses. Such people may require 40 to 60 mg of prednisone per day initially. The dosage is tapered as rapidly as the person's

MEDICATION ADMINISTRATION Immunosuppressive agents for SLE

CYTOTOXIC AGENTS

Azathioprine (Imuran)
Cyclophosphamide (Cytoxan)
Cyclosporin (Sandimmune)

Certain cytotoxic or antineoplastic drugs are effective as immunosuppressive agents. They act by decreasing the proliferation of cells within the immune system and are widely used to prevent rejection following a tissue or organ transplant. They are usually administered concurrently with corticosteroid therapy, allowing lower doses of both preparations and resulting in fewer side effects.

Nursing responsibilities

- Monitor blood count, with particular attention to the WBC and platelet counts. Notify the healthcare provider if WBCs fall below 4000 or platelets below 75 000.
- Monitor kidney and liver function studies, including BUN, creatinine, creatinine clearance and liver enzyme levels. Report any abnormal levels to the healthcare provider.
- Oral preparations should be administered with food to minimise gastrointestinal effects. Antacids may be ordered.
- Increase fluids to maintain good hydration and urinary output.
- Monitor intake and output.
- Monitor for signs of abnormal bleeding: bleeding gums, bruising, petechiae, joint pain, haematuria and black or tarry stools.
- Use meticulous handwashing and other appropriate measures to protect the person from infection. Assess for signs of infection.
- Pulmonary fibrosis is a potential adverse effect of cyclophosphamide. Therefore, monitor the results of pulmonary function studies and be alert to clinical signs of dyspnoea or cough.

Health education for the person and family

- Avoid large crowds and situations where you might be exposed to infections.
- Report signs of infection such as chills, fever, sore throat, fatigue or malaise to the healthcare provider.
- Use contraceptive measures to prevent pregnancy while you are taking these drugs because they cause birth defects.
- Avoid the use of aspirin or ibuprofen while taking these drugs. Report any signs of bleeding to the healthcare provider.
- You may stop menstruating while you are taking cyclophosphamide. The menses will resume after the drug is discontinued.
- If you are taking cyclophosphamide, be sure to report difficulty breathing or cough to the healthcare provider.

disease allows, although lowering the dosage may precipitate an acute episode. Some people with SLE require long-term corticosteroid therapy to manage symptoms and prevent major organ damage. These people are at increased risk of corticosteroid side effects, such as cushingoid effects, weight gain, hypertension, infection, accelerated osteoporosis and hypokalaemia.

Immunosuppressive agents such as cyclophosphamide or azathioprine may be used, alone or in combination with corticosteroids, to treat people with active SLE or lupus nephritis (see the 'Medication administration' box above). When these agents are used in combination, lower, less toxic doses of each drug can be used. The person receiving immunosuppressive agents is at increased risk of infection, malignancy, bone marrow depression and toxic effects specific to the drug prescribed.

Treatments

Because of the photosensitivity associated with SLE, the person should be cautioned to avoid sun exposure. People should use sunscreens with a sun protection factor (SPF) rating of 15 or higher when outdoors. Topical corticosteroids may be used to treat skin lesions. Some healthcare providers recommend avoiding the use of oral contraceptives, because oestrogen can trigger an acute episode.

People with lupus nephritis who progress to develop end-stage kidney disease are treated with dialysis (haemodialysis or peritoneal dialysis) and kidney transplantation.

Nursing care

Nursing care for the person with mild SLE may be limited to teaching. The person with severe disease, however, has many diverse nursing needs, which vary according to the organ systems involved. Because of the close link between rheumatoid arthritis and SLE, many of the nursing diagnoses and interventions identified for the person with arthritis may be appropriate for the person with lupus. The person with lupus nephritis or end-stage kidney disease has the nursing care needs related to glomerulonephritis and chronic kidney failure. This section focuses on the needs of the person related to the dermatological manifestations of lupus, an increased risk of infection and health maintenance.

Nursing diagnoses and interventions

The priority nursing interventions for the person with SLE are focused on problems with impaired skin integrity, ineffective protection and impaired health maintenance.

Impaired skin integrity

Skin lesions are a common manifestation of SLE. A rash or discoid lesion interrupts the integrity of the skin and the first line of protection against infection, increasing the person's already high risk of infection. These lesions, which usually appear on exposed parts of the skin, can also be disfiguring and cause the person emotional distress.

- Assess knowledge of SLE and its possible effects on the skin. *Assessment allows the nurse to base teaching and information on the person's existing knowledge, improving learning and retention.*
- Discuss the relationship between sun exposure and disease activity, both dermatological and systemic. *It is important for the person to understand that sun exposure may not only cause dermatological manifestations but also trigger an acute episode.*
- Suggest the following strategies to limit sun exposure:
  - Avoid being outdoors during hours of greatest sun intensity (10 am to 3 pm).
  - Use sunscreen with an SPF of 15 or higher when sun exposure cannot be avoided. Apply it 30 minutes before going out into the sun.
  - Reapply sunscreen after swimming, exercising or bathing.
  - Wear loose clothing with long sleeves and wide-brimmed hats when outdoors.

  These strategies can help the person maintain a normal lifestyle while helping to prevent acute episodes.
- Keep skin clean and dry; apply therapeutic creams or ointments to lesions as prescribed. *These measures promote healing and reduce the risk of infection.*

Ineffective protection

Ineffective protection can be a problem for the person with SLE, who is at increased risk of infection and multiple organ system problems because of the disease. In addition, treatment with corticosteroids or immunosuppressive agents further impairs immune responses and the ability to fight infection. The following interventions are for the person who is hospitalised.

- Wash hands before and after providing direct care. *Handwashing removes transient organisms from the skin, reducing the risk of transmission to the person.*

CONSIDERATION FOR PRACTICE

Hands must be washed before and after providing direct care, even if gloves are worn. A decrease in this type of medical asepsis is contributing to the increasing number of hospital-acquired infections that are resistant to antibiotics.

- Use strict aseptic technique in caring for intravenous lines and indwelling urinary catheters or performing any wound care. *Aseptic technique offers protection against external and resident host microorganisms.*
- Assess frequently for infection. Monitor temperature and vital signs every 4 hours. Assess for signs of cellulitis, including tenderness, redness, swelling and warmth. Report signs of infection to the healthcare provider promptly. *Therapy can suppress usual responses, such as elevated temperature and inflammation. The fever of infection may be mistaken for the fever commonly associated with lupus. The person receiving immunosuppressive therapy for the disease has an even higher risk of infection.*
- Monitor laboratory values, including FBC and tests of organ function; report changes to the healthcare provider. *An elevation in the WBC count with a shift to the left (increased numbers of immature leucocytes in the blood) may be an early indication of infection. Changes in liver function studies, renal function studies, myocardial enzymes or other laboratory values may indicate organ system involvement.*
- Initiate reverse or protective isolation procedures as indicated by the person's immune status. *These procedures provide further protection from infection for the severely immunocompromised person.*
- Ensure an adequate nutrient intake, offering supplementary feedings as indicated or maintaining parenteral nutrition if necessary. *Adequate nutrition is important for healing and immune system function.*
- Teach the person the importance of good handwashing after using the bathroom and before eating. *Handwashing reduces the risk of infection with endogenous organisms.*
- Monitor for potential adverse effects of medications, including thrombocytopenia and possible bleeding, fluid retention with oedema and possible hypertension, loss of bone density, osteoporosis and possible pathological fractures, renal or hepatic toxicity, and cardiac effects, particularly in the person with fluid retention and hypervolaemia. *Medications used to treat SLE have many potential adverse effects that can impair normal protective and homeostatic mechanisms.*

Impaired health maintenance

As with other chronic diseases, much of the responsibility for maintaining optimal health rests with the person. Disease manifestations such as fatigue, arthralgias, arthritis and increased risk of infection can interfere with the person's ability to maintain health. Psychosocial issues can also be a significant factor in health maintenance for the person with lupus. These issues may include denial of the significance of the disease, poor coping, lack of financial and other resources, and an inadequate support system.

- Assess the ability to maintain optimal health, identifying physical and psychosocial factors that may affect health maintenance. *Before intervening to improve the person's health maintenance, the nurse must identify and understand factors affecting it.*
- Provide care and teaching in a non-judgmental manner. *To intervene effectively, the nurse must accept the person and family as they are.*
- Encourage the person and family members to discuss the effect of the disease on their lives. *Open discussion helps the person and the nurse identify barriers to health maintenance and begin exploring alternative strategies.*
- Initiate an interprofessional care conference with the person and family. *In this care conference, a number of perspectives can be expressed, improving the planning of strategies for health maintenance activities.*
- Refer the person and family to counselling as needed. *Counselling may help the person and family develop the necessary coping skills to accept and deal with the disease.*

- Refer the person and family to community and social service agencies and local support groups. *These groups and agencies are valuable resources for the person and family.*

Community-based care

Teaching is a critical factor in preparing people with SLE for self-care at home. Address the following topics:

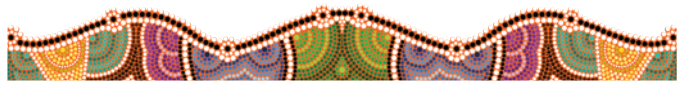
- the disease and its potential effects. Promote an optimistic outlook, stressing that the majority of people do not require long-term corticosteroid therapy and that the disease may improve over time
- the importance of skin care
- the importance of avoiding exposure to infection
- the need to follow the prescribed treatment plan, including rest and exercise, medications and follow-up appointments. Discuss manifestations of an acute episode (often called a flare) and stress the importance of contacting the healthcare provider promptly if any of these manifestations occur.

CONSIDERATION FOR PRACTICE

Warning signs of a flare-up

- Increased fatigue
- Pain, abdominal discomfort
- Rash
- Headache
- Fever
- Dizziness

- The significance of wearing a MedicAlert® tag identifying the condition and therapy such as corticosteroids or immunosuppressives.
- Family planning with the person and spouse. The use of oral contraceptives may be contraindicated for the person; if appropriate, provide information about alternative means of birth control. Pregnancy is not contraindicated for most women with lupus. However, the pregnant person requires close monitoring because acute episodes sometimes accompany pregnancy.
- The need for preventive healthcare for both men and women with SLE. Women should have gynaecological and breast examinations, and men should have prostate examinations, each year. Both men and women should have regular screenings for cholesterol and blood pressure. Annual influenza vaccinations are important, as is pneumococcal vaccinations for older people. If people are taking corticosteroids or antimalarial medications, annual eye examinations should be conducted to screen for and treat any eye problems.
- Helpful resources:
  - Arthritis Australia: www.arthritisaustralia.com.au
  - Arthritis New Zealand: www.arthritis.org.nz
  - Arthritis Victoria (now includes Lupus Foundation Australia): www.arthritisvic.org.au
  - Lupus Association of New South Wales: www.lupusnsw.org.au
  - Lupus Association of Tasmania: www.lupustas.bigpond-hosting.com/home/Welcome.html
  - Lupus Trust of New Zealand: www.lupus.org.nz.



THE PERSON WITH POLYMYOSITIS

Polymyositis is a systemic connective tissue disorder characterised by inflammation of connective tissue and muscle fibres leading to muscle weakness and atrophy. When muscle fibre inflammation is accompanied by skin lesions, the disease is known as dermatomyositis. Polymyositis is an autoimmune disorder of unknown cause that affects more women than men. The onset of the disease typically occurs between the ages of 40 and 60 years, although a childhood-onset form is also seen.

The immune mechanism causing the inflammatory response in polymyositis is not clear, but autoantibodies can be identified in the majority of people with the disease. The activation of complement is thought to contribute to the inflammatory process. Inflammation leads to muscle fibre necrosis and degeneration.

Manifestations

Initial manifestations of polymyositis include muscle pain, tenderness and weakness; rash; arthralgias; fatigue; fever; and weight loss. Skeletal muscle weakness is the predominant manifestation. Its onset may be either insidious or abrupt. Muscle weakness tends to progress over weeks to months. Muscles of the shoulder and pelvic girdles are particularly affected, making it difficult for the person to get out of chairs, climb stairs and reach overhead. Weakness of neck flexor muscles may make it difficult to raise the head from a pillow. Affected muscles may also be tender and painful. A characteristic dusky red rash may be present on the face and upper trunk. Other manifestations include Raynaud's phenomenon, dysphagia, dyspnoea and cough (due to interstitial pneumonitis). The risk of malignancy is increased, particularly in people with dermatomyositis.

INTERPROFESSIONAL CARE

There is no specific test to diagnose polymyositis. Autoantibodies may be identified in blood serum. Serum levels of muscle enzymes are elevated, particularly creatine kinase (CK) and aldolase levels. Biopsy of involved muscle shows patchy muscle fibre necrosis and the presence of inflammatory cells.

A combination of rest and corticosteroid therapy is prescribed for the person with polymyositis. Long-term corticosteroid therapy may be necessary to manage the disease. Immunosuppressive agents such as methotrexate, cyclophosphamide and azathioprine may be used for people who do not respond well to treatment with corticosteroids.

Nursing care

The nursing role in caring for the person with polymyositis is supportive. Measures to promote comfort are important. Muscle weakness may interfere with the person's ability to provide self-care and manage health and home. The person may have difficulty with speech because of pharyngeal muscle weakness. Provide alternate means of communication as needed and use patience in listening. Observe closely while the person eats, because aspiration is a potential problem. Modify the person's diet as needed to maintain nutrition and safety.

Educating the person and their family is an important component of care. Emphasise the need to balance periods of rest and activity. Discuss skin care to prevent dryness and infection. Teach the person about prescribed medications and their short- and long-term side effects. Provide information about safety measures while eating. Encourage family members to become trained in performance of CPR. Discuss signs of respiratory infection and other possible complications of polymyositis, including kidney failure and malignancy.

THE PERSON WITH OSTEOMYELITIS

Osteomyelitis is an infection of the bone. Osteomyelitis may occur as an acute, subacute or chronic process. It occurs as a consequence of bacteraemia (haematogenous osteomyelitis), invasion from a contiguous focus of infection or skin breakdown in the presence of vascular insufficiency (McPhee et al., 2015).

Osteomyelitis can occur at any age, but adults over age 50 are more commonly affected. The older adult is at risk of osteomyelitis for several reasons. Immune function tends to decline with ageing; the older adult also is more likely to have a chronic disease process that affects immune function. Circulatory status in older adults often is compromised by atherosclerotic processes, impairing blood flow to the bone. Older adults have a higher risk of pressure ulcers because of circulatory, skin, sensation and mobility changes associated with ageing. Pressure ulcers that cannot be staged and treated because of eschar formation pose a particular risk. In addition, the older adult may not demonstrate the typical signs of infection and inflammation, thus allowing an infectious process to become well established before it is detected.

Pathophysiology

The cause of osteomyelitis is usually bacterial; however, fungi, parasites and viruses can also cause bone infection. *Staphylococcus aureus* is the most common infecting organism. Other organisms include *Escherichia coli*, *Pseudomonas*, *Klebsiella*, *Salmonella* and *Proteus*.

Direct contamination of bone from an open wound, such as an open fracture or a gunshot or puncture wound, is the most common cause of osteomyelitis; osteomyelitis also may occur as a complication of surgery. The third mode of entry for microorganisms that invade bone tissue is the extension from adjacent soft tissue infection. People with venous stasis or arterial ulcers of the lower extremities or long-term complications of diabetes mellitus are at risk of this type of bacterial invasion.

After entry, bacteria lodge and multiply in the bone, resulting in the inflammatory and immune system response. Phagocytes attempt to contain the infection, releasing enzymes in the process that destroy bone tissue. Pus forms, followed by oedema and vascular congestion. The Haversian canals in the medullary (marrow) cavity of the bone allow the infection to travel to other segments of the bone. If the infection reaches the outer margin of the bone (see Figure 39.8), it raises the periosteum of the bone, spreading along the surface. Lifting of the periosteum from the cortex disrupts the blood vessels that enter the bone. Pressure increases, further compromising the vascular supply and leading to ischaemia and eventual necrosis of the bone. Blood and antibiotics cannot reach the bone tissue once the pressure compromises the vascular and arteriolar systems. In addition, bacteria adhere to damaged bone, coating the underlying bone with a protective film that further impedes host defences.

Haematogenous osteomyelitis

Haematogenous infections are caused by pathogens that are carried in the blood from sites of infection elsewhere in the body. Haematogenous osteomyelitis primarily affects older adults, people with sickle cell anaemia and intravenous drug users. The spine is the usual site of infection in adults. Pathogens enter the well-perfused vertebral bodies of adults via the spinal arteries. From there, the infection spreads into the disc space. The lumbar spine is involved more frequently than the thoracic or cervical spine. Urinary tract infections, soft tissue infection, endocarditis and infected intravenous sites are sources of pathogens.

People with acute haematogenous osteomyelitis experience an acute onset of pain, tenderness and fever. Soft tissue swelling over the affected bone may be noted. The course of vertebral osteomyelitis in intravenous drug users often is subacute, with vague, dull pain in the affected region and a normal or low-grade fever. The pain intensifies over 2 to 3 months and is accompanied by tenderness, muscle spasm and limited range of motion.

Osteomyelitis from a contiguous infection

Infections caused by an extension of infection from adjacent soft tissues fall into this category of osteomyelitis. The infection is a result of or a complication of direct penetrating wounds, joint replacements, decubitus ulcers and neurosurgery. This is the most common cause of osteomyelitis in adults.

The diagnosis of osteomyelitis often is not made until the infection has become chronic because the signs of acute infection may be masked by local tissue inflammation. Failure to heal a surgical wound or fracture or a developing sinus tract may be initial indicators of a bone infection.

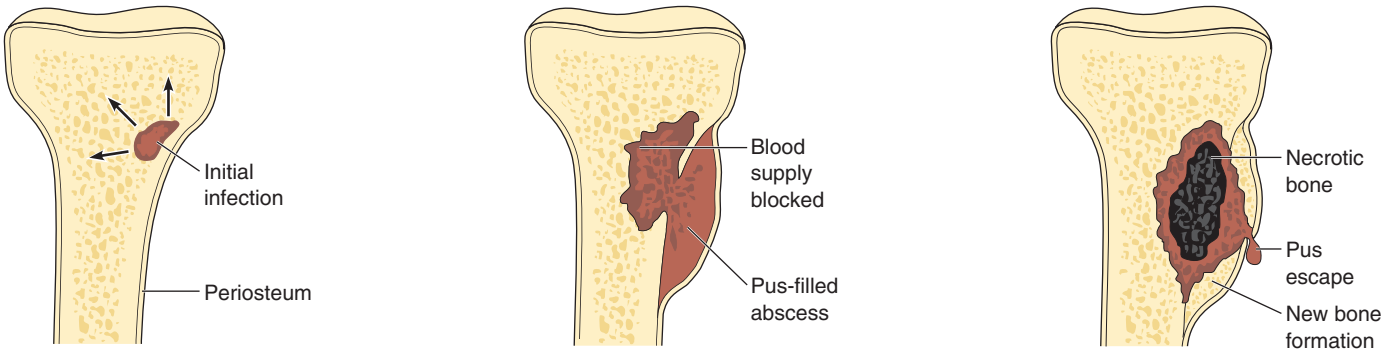


FIGURE 39.8 ■ Osteomyelitis. *A*, Site of initial infection. Bacteria enter and multiply in the bone and the inflammatory response is initiated. *B*, Acute phase, in which infection spreads to other parts of the bone. Pus forms, oedema occurs and the vascular supply is compromised. If the infection reaches the outer margin of the bone, the periosteum is lifted and ischaemia and necrosis eventually occur. *C*, Chronic phase. Necrotic bone separates, a new layer of bone forms around the necrotic bone and a sinus develops to allow the wound to drain

Osteomyelitis associated with vascular insufficiency

People with diabetes and peripheral vascular disease are at risk of developing osteomyelitis involving the feet. Diabetic neuropathy exposes the foot to trauma and pressure ulcers. The person may be unaware of the infection as it spreads into the bone. When tissue perfusion is poor, normal inflammatory responses and wound healing are impaired. The infection often is diagnosed when the person seeks treatment for a non-healing sore, swollen toe or acute cellulitis.

Manifestations

Manifestations of osteomyelitis vary according to the age of the person, the cause and site of involvement, and whether the infection is acute, subacute or chronic (see the box below).

MANIFESTATIONS Osteomyelitis

CARDIOVASCULAR EFFECTS
- Tachycardia

GASTROINTESTINAL EFFECTS
- Nausea and vomiting
- Anorexia

MUSCULOSKELETAL EFFECTS
- Limp in involved extremity
- Localised tenderness, especially in epiphyseal area

INTEGUMENTARY EFFECTS
- Drainage and ulceration at involved site
- Swelling, erythema and warmth at involved site
- Lymph node involvement, especially in the involved extremity

OTHER EFFECTS
- High temperature with chills
- Abrupt onset of pain
- Malaise

INTERPROFESSIONAL CARE

The care of the person with osteomyelitis focuses on relieving pain, eliminating the infection and preventing or minimising complications. Early diagnosis is important to prevent bone necrosis by early administration of the appropriate antibiotic. Most people require both debridement of bone and a long period of antibiotic administration.

Diagnosis

The diagnosis of osteomyelitis is based on bone scans, MRI, blood tests and biopsy. As described in Chapter 37, an MRI, CT scan and bone scan may be conducted to identify abscesses, sinus tracts and bone changes. An ultrasound can detect subperiosteal fluid collections, abscesses and periosteal thickening and elevation associated with osteomyelitis. During an acute infection, ESR and WBC are elevated. Blood and tissue cultures (from affected bone or soft tissue) are obtained to identify the infecting organism and direct antibiotic therapy.

Medications

Antibiotic therapy is mandatory to prevent acute osteomyelitis from progressing to the chronic phase. Parenteral antibiotic therapy begins as soon as cultures (blood and/or wound) are obtained. A penicillinase-resistant semisynthetic penicillin may be given until the culture and sensitivity results are known. These antibiotics are used initially because many cases of osteomyelitis are caused by *Staphylococcus aureus*. When the detailed sensitivity report is obtained from the cultures, more definitive antibiotics are prescribed.

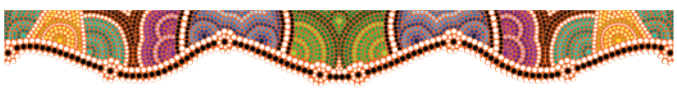
For the person with acute or chronic osteomyelitis, antibiotics are continued for 4 to 6 weeks. Intravenous antibiotic administration or oral therapy is common. Oral therapy with twice-daily ciprofloxacin has been shown to be as effective as parenteral therapy for treating adult people with chronic osteomyelitis caused by susceptible organisms

(McPhee et al., 2015). People can be treated as 'hospital in the home' with long-term IV antibiotics via PICC (peripherally inserted central venous catheter) for up to 6 months if needed.

Surgery

Surgical debridement is the primary treatment for the person with chronic osteomyelitis. The periosteum is excised and the cortex is drilled to release the pressure from accumulated pus. During this procedure, cultures may be obtained and sent to the laboratory for analysis. The wound holes are irrigated and the wound is then closed. The cavity may be kept clean by inserting drainage tubes that are connected to an irrigation and suction system. Postoperatively, the nurse is responsible for instilling and removing diluted antibiotic solutions through the drainage tubes. See the box below for related nursing care.

A musculocutaneous (myocutaneous) flap is another approach used for the treatment of the dead space caused by extensive debridement of the infected site. The procedure involves moving or rotating a muscle and the section of skin fed by the arteries from that muscle into the cavity created by the surgery. A skin graft is performed later.



Nursing care

The person with chronic osteomyelitis faces frequent and lengthy hospitalisations and/or treatment modalities. The prognosis is uncertain and functional deficits and amputation are a constant concern. The ongoing expenses, loss of financial support and role changes within the family are also concerns for the person.

Nursing diagnoses and interventions

Nursing diagnoses associated with acute osteomyelitis focus on preventing the transmission of infection and problems due to immobility. Providing comfort and person teaching are also very important.

Risk of infection

Compromised immune status places the person with osteomyelitis at risk of superinfection. An inadequate kilojoule intake is an additional factor that contributes to the risk.

- Maintain strict handwashing practices. *Meticulous handwashing helps prevent the spread of infection by minimising the entry of organisms into susceptible people.*

CONSIDERATION FOR PRACTICE

Careful handwashing before and after direct care is essential even if gloves are worn.

- Administer antimicrobial therapy at specified time intervals. *Optimal blood levels of antibiotic therapy are mandatory in people with infectious processes.*
- Maintain the person's optimal dietary kilojoule and protein intake. *High kilojoule and protein intake provides the person with sufficient nutritional support for the body's needs during the stressful event of the inflammatory process.*

Hyperthermia

The infection and associated inflammatory process can cause fever in the person with osteomyelitis.

- Monitor temperature every 4 hours and when the person reports chills and/or fever. *Blood cultures are frequently ordered when an acute elevation of temperature occurs. A sudden rise in temperature in people with either acute or chronic osteomyelitis may indicate inadequate antimicrobial management.*
- Maintain a cool environment and provide light clothing and bedding during temperature elevation. *Proper environmental conditions and clothing enhance the evaporative process during acute temperature elevation and promote comfort.*
- Ensure a daily fluid intake of 2000 to 3000 mL. *Dehydration may result from evaporative fluid losses during acute temperature elevations. Furthermore, people taking large doses of antibiotic therapy may experience fluid loss through excessive diarrhoea as a side effect of the therapy. Fluid replacement is necessary during this time to prevent further dehydration.*

Impaired physical mobility

Pain, infection, inflammation and the use of immobilisers can all impair the mobility of the person with osteomyelitis.

- Maintain the affected limb in functional position when immobilised. *The person may hesitate to move the involved extremity because of continuous pain; therefore, the extremity must be maintained in functional position to avoid flexion contracture.*
- Maintain rest and avoid subjecting the affected extremity to weight-bearing activities. *The involved extremity must be immobilised to avoid pathological fractures caused by stress on the weakened bone.*
- Ensure active or passive ROM exercises every 4 hours. *Flexion contracture occurs when the person remains immobile or when there is only minimal joint movement. Consult a physical therapist for plan of exercises to avoid contracture.*

Acute pain

The person with osteomyelitis experiences pain due to swelling.

- Use a splint or immobiliser when the person experiences acute pain from swelling. *Splinting or immobilising the involved extremity provides support and reduces pain caused by movement.*
- Ask the doctor to order scheduled administration of narcotic and non-narcotic analgesics on a 24-hour basis rather than as needed. *The use of 24-hour administration allows blood levels of pain-relieving medications to remain constant.*

NURSING CARE OF THE PERSON undergoing surgical debridement for osteomyelitis

PREOPERATIVE CARE

- Discuss the impending surgery, the person's concerns regarding surgery and its risks, and what steps will be taken if surgery is ineffective. *Open discussion and active listening are important means of gaining the person's trust and encouraging the person to express concerns about the outcome of the surgery. Surgery is frequently performed when 36 to 48 hours of antimicrobial therapy yields no improvement and when prolonged bacteraemia and evidence of an abscess formation are present. The periosteum is excised, allowing access to the purulent material in the infected area. If pus is not apparent, several holes may be drilled into the bone. In some cases, irrigation tubes are inserted and connected to an elaborate system for postoperative antimicrobial therapy.*
- People may need extensive antimicrobial treatment postoperatively if an irrigation system is surgically implanted. Before the procedure, explain to the person that bed rest and an extended period of treatment in the hospital are imperative. *People who understand the events that may occur postoperatively may be more accepting of the required restrictions.*

POSTOPERATIVE CARE

- Provide meticulous care of the dressing and/or irrigation setup. Frequently, the irrigation tubes are connected to a three-way stopcock, which allows irrigation and drainage of the debrided area without separating the tube from the collection device. *Nurses need to be extremely cautious and adhere to strict sterile technique.*
- Assess the person for manifestations of further infection. *Although the person will receive antimicrobial agents, it is important to monitor the person continually for sudden spikes in temperature, pain at the involved site and other indications of superinfection.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- While receiving antimicrobial agents, be sure to drink adequate amounts of fluid and eat a high-kilojoule diet to minimise the risks of damage to the kidneys, yeast infection and adverse gastrointestinal effects.

CONSIDERATION FOR PRACTICE

People are often reluctant to ask for a prn pain medication, allowing the pain to reach a level that is difficult to manage.

- Use non-pharmacological strategies (e.g. heat, distraction, relaxation techniques) for pain management. *Pain of the muscles and joints may be controlled through non-pharmacological interventions. Warm moist packs, warm baths or heating pads to the involved extremity provide comfort due to vasodilation.*
- Avoid excessive manipulation of the involved area; handle the area gently. Carefully assess the person for guarding, limping or unwillingness to move the affected part. Communicate to other healthcare professionals the person's preferences for assistive devices and means of manipulating the involved area. *Gentle handling and minimal manipulation help reduce pain.*

Community-based care

Although people may be hospitalised for acute treatment and surgery, most care is provided at home. Community health services can provide intravenous medications, if prescribed. Discuss the following topics for home care:

- the importance of careful handwashing, especially after toileting and dressing changes
- the importance of taking all antibiotics as prescribed. Include information about helping prevent the yeast infections (of the mouth or vagina) often associated with prolonged antibiotic therapy by eating 1 cup (250 mL) of live-culture yoghurt each day
- the need to take pain medications on a regular basis to prevent pain from becoming severe. Provide information about how to deal with side effects, such as constipation, by increasing fluid and fibre intake
- how to perform wound care and sources for needed equipment and supplies
- rest or limited weight bearing for the affected extremity or body part. Teach how to avoid complications associated with prolonged immobilisation, such as frequently shifting position, keeping skin and linens clean and dry, and doing active ROM exercises for unaffected joints
- the importance of maintaining good nutrition. An adequate supply of kilojoules, protein and other nutrients is necessary for immune function and healing. Suggest frequent small meals and using nutritional supplements such as Ensure to help maintain nutritional intake.

THE PERSON WITH SEPTIC ARTHRITIS

Septic arthritis can develop if a joint space is invaded by a pathogen. The primary risk factors for septic arthritis are persistent bacteraemia (bacteria in the blood) (e.g. due to use of injectable drugs, endocarditis) and previous joint damage (e.g. due to trauma or rheumatoid arthritis). Arthroscopic surgery and total joint replacements that allow potential direct contamination of the joint are additional risk factors (McPhee et al., 2015).

Pathophysiology

The most common bacteria implicated in septic arthritis include gonococci, *S. aureus* and streptococci. Infections by Gram-negative bacteria such as *E. coli* and *Pseudomonas* are seen with

increasing frequency, particularly in people who inject recreational drugs or are immunocompromised (McPhee et al., 2015).

Infection of the joint leads to inflammation with resulting synovitis and joint effusion. Abscesses may form in synovial tissues or bone underlying joint cartilage. If not treated promptly and effectively, septic arthritis can lead to destruction of the affected joint. A single joint, often the knee, is usually affected. Septic arthritis may also affect other joints such as the shoulder, wrist, hip, fingers or elbow.

Manifestations

The onset of septic arthritis is typically abrupt, marked by pain and stiffness of the infected joint. The joint appears red and swollen and is hot and tender to the touch. Effusion (increased fluid within the joint space) is usually present. Systemic manifestations of infection, such as chills and fever, often accompany local manifestations, although these may be muted if the person is taking anti-inflammatory medications.

INTERPROFESSIONAL CARE

Septic arthritis is a medical emergency requiring prompt treatment to preserve joint function. When it is suspected, fluid from the affected joint is aspirated and sent for Gram stain and culture. Cultures also are obtained from all likely sources of the infection, including blood, sputum or wounds. The synovial fluid culture is always positive in non-gonococcal septic arthritis but often is negative for bacteria in early gonococcal arthritis. Infected synovial fluid usually is cloudy, with a high WBC count and a low glucose level. Joint x-ray films are often normal in the initial stages, but soon show demineralisation, bony erosions and joint space narrowing.

The infected joint is treated with rest, immobilisation, elevation and systemic antibiotics. Treatment with a broad-spectrum parenteral antibiotic is initiated before the results of culture are obtained. The medication may be changed or adjusted once the organism has been identified. Antibiotic therapy is continued for at least 2 weeks after inflammatory manifestations have abated. Frequent joint aspirations may be performed to remove excess fluid and pus and to evaluate for the continued presence of bacteria. Surgical drainage may be performed if the hip joint is involved (because of the difficulty of aspirating this joint) or when medical therapy does not rapidly eliminate bacteria from the synovial fluid. Physical therapy is implemented during the recovery period to ensure maintenance of optimal joint function.

Nursing care

Septic arthritis can be frightening to the person who experiences a sudden onset of joint pain and swelling and is faced with the possibility of rapid functional loss of movement. Nursing care is both supportive and educative. People may be hospitalised for initial treatment with intravenous antibiotics. It is important to monitor the person's response to therapy, including systemic manifestations such as fever. Position the affected joint appropriately, using pillows to elevate it as needed. Splints or traction may be used to immobilise the joint. Warm compresses may be ordered for comfort. Active ROM exercises preserve joint mobility and should be initiated as soon as the doctor allows.

The person with septic arthritis needs information about the disorder, its aetiology and its treatment. Teach the person how organisms may gain entry into the joint space. Discuss the role that the use of injected drugs and sexually transmitted infections play in septic arthritis and means to prevent infection as appropriate (e.g. using clean 'works', practising safer sex). Refer the person to a drug treatment program if necessary. Emphasise the importance of complying with all aspects of the treatment plan to prevent joint destruction and disability.

NEOPLASTIC DISORDERS

Bone tumours may be either primary (arising in the bone itself) or metastatic (seeded from a tumour elsewhere in the body). Like other tumours, bone tumours can be either benign or malignant.

THE PERSON WITH BONE TUMOURS

Benign bone tumours tend to grow slowly and do not often destroy surrounding tissues. Primary malignant tumours of the bone are rare, accounting for only about 1% of all adult cancers (Porth & Matfin, 2014). Malignant tumours grow rapidly and metastasise. Virtually every malignant tumour can metastasise to bone. However, the most common metastatic bone tumours originate from primary tumours of the prostate, breast, kidney, thyroid and lung.

Primary bone tumours arise from bone tissue itself—that is, cartilage (chondrogenic), bone (osteogenic), collagen (collagenic) and bone marrow cells (myelogenic). The tissue type, neoplasm classification, sites and incidence of the most common primary bone tumours are summarised in Table 39.6. The focus for discussion in this section is care of the person with a primary bone tumour.

TABLE 39.6 Description of common primary bone tumours

TISSUE TYPE	BENIGN	MALIGNANT	SITE	INCIDENCE
Chondrogenic (cartilage-forming tumours)	Osteochondroma—most common benign tumour		Pelvis, scapula, ribs	Higher in males
	Chondroma		Hands, feet, ribs, spine, sternum or long bones	Ages 30 to 50 Higher in males
		Chondrosarcoma	Femur, pelvis, ribs, head (epiphysis) of long bones	13% of malignant bone tumours Middle age and older Higher in males
Osteogenic (bone-forming tumours)	Osteoid Osteoma		Shaft (diaphysis) of long bones (i.e. femur, tibia)	Ages 20 to 30 Higher in males
		Osteosarcoma—most common malignant tumour	Long bones, knee	38% of malignant bone tumours Predominant in adolescents and people aged 50 to 60
Collagenic (collagen-forming tumours)		Fibrosarcoma	Femur, tibia	4% of malignant bone tumours Wide age distribution, but usually occurs in people aged 40 to 50 Higher in females
Myelogenic (tumours of bone marrow cells)	Giant cell tumour		Shaft (diaphysis) of long bones (i.e. femur, tibia, radius, humerus)	4–5% of bone tumours Wide age distribution Higher in females

Pathophysiology

The aetiology of bone tumours is unknown but there is a connection between increased bone activity and the development of primary bone tumours. Bone tumours frequently occur when primary bone growth is at its peak in adolescence or is overstimulated during disease, such as Paget's disease.

Primary tumours cause bone breakdown, called *osteolysis*, which weakens the bone, resulting in bone fractures. Normal bone adjacent to the tumour responds to tumour pressure by altering its normal pattern of remodelling. The bone's surface becomes altered and the contours enlarge in the area of the tumour growth.

Malignant bone tumours invade and destroy adjacent bone tissue by producing substances that promote bone resorption or by interfering with a bone's blood supply. Benign bone tumours, unlike malignant ones, have a symmetrical, controlled growth pattern. As they grow, they push against neighbouring bone tissue. This weakens the bone's structure until it becomes unable to withstand the stress of ordinary use and frequently causes pathological fracture.

Manifestations

The three main manifestations of bone tumours are pain, a mass and impaired function. Bone pain usually comes on slowly and lasts for as long as a week, is constant or intermittent, and may be worse at night. The mass is described as a swelling or lump on the bones that is firm, slightly tender and may be felt through the skin. The mass may interfere with normal movement and/or cause the bone to break. The manifestations of bone tumours are usually associated with a history of a fall or blow to the extremity that brings the mass to the person's attention. The injury, rather than the growth itself, usually causes the person to seek medical attention. Manifestations of bone tumours are listed in the box below.

INTERPROFESSIONAL CARE

Treatment and care of the person with a bone tumour focuses on prompt diagnosis, removal of the tumour, prevention of complications and education.

Diagnosis

The diagnosis of a bone tumour is critical to the survival of the person and possible preservation of the affected limb. Diagnostic tests are described in Chapter 37.

Radiological studies include x-rays, CT scans and MRI. X-rays show the location of the tumours and the extent of bone involvement. Benign tumours are characterised by sharp margins that are clearly separate from the surrounding normal bone. Metastatic bone destruction has a characteristic 'moth-eaten' pattern in which the growth has a less defined margin that cannot be separated from the normal bone. CT scan and MRI are useful in evaluating the extent of tumour invasion into bone, soft tissues and neurovascular structures. Percutaneous needle biopsy or needle biopsy at the time of surgery is used to determine the exact type of bone tumour.

Laboratory tests include an alkaline phosphatase (elevated with malignant bone tumour) and a calcium level (increased with massive bone destruction).

Treatments

As with other malignant tumours, bone tumours are treated with chemotherapy, radiation therapy and surgery.

MANIFESTATIONS Neoplasms of the musculoskeletal system

	MANIFESTATIONS
BONY SARCOMAS SITE	
Upper or lower extremity or pelvis	■ Worsening deep bony pain ■ Pain at night or during rest that may radiate and become severe ■ Muscular weakness or atrophy
Metaphysis of distal femur, proximal tibia, proximal humerus and pelvis	■ Soft tissue mass extending from bone with erythematous or warm skin over tissue mass ■ Change in ability to perform ADLs ■ Fever
SOFT TISSUE SARCOMAS SITE	
Upper or lower extremity and pelvis	■ Enlarging firm mass with irregular borders, which causes pain in surrounding soft tissue structures
Thigh; shoulder and pelvis	■ Erythema or warmth and venous dilation over skin ■ Muscular weakness and atrophy with limited range of motion change in ability to perform ADLs and change in gait ■ Paraesthesia with neurological involvement and distal swelling ■ Palpable local lymph nodes
Pelvis	■ Altered bowel and bladder habits or pain with intercourse

CHEMOTHERAPY Chemotherapeutic agents are administered to shrink the malignant tumour before surgery, to control recurrence of tumour growth after surgery or to treat metastasis of the tumour. Chemotherapeutic agents used to treat bone tumours are listed in Box 39.5. See Chapter 13 for further discussion of chemotherapy and its nursing implications.

RADIATION THERAPY Radiation therapy may be used in combination with chemotherapy. Radiation therapy is frequently applied to metastatic bone carcinomas as a method of pain control. It is also used to eliminate bony tumours or any remaining tumour after a surgical procedure. Radiation therapy is discussed in Chapter 13.

SURGERY The goal of surgery for the treatment of primary bone tumours is to eliminate the tumour completely. Tumours are removed either by excising the tumour itself or by amputating the affected limb. The type of procedure varies: removal of the tumour only, removal of the tumour along with a small margin of normal tissue surrounding the tumour, removal of the tumour and a wide zone of normal tissue, or removal of the tumour and part or all of the bone in which it lies. Cadaver allografts or metal prostheses often are used to replace missing bone, avoiding amputation. Care of the person undergoing amputation is discussed in Chapter 38.

BOX 39.5 Chemotherapeutic agents used for musculoskeletal neoplasms

Alkylating agents
Ifosfamide
Cyclophosphamide

Antibiotics
Doxorubicin
Bleomycin

Antimetabolites
Methotrexate

Plant alkaloids
Vincristine

Synthetic agents
Cisplatin

Nursing care

Nursing care for the person with bone tumours requires innovative interventions from the time of diagnosis through the rehabilitation phase. In the acute phase, problems associated with pain, lack of knowledge, immobility, coping and anxiety are foremost. If the person develops complications from treatment or if a malignancy metastasises, problems related to home health maintenance management, self-concept and prevention of further complications take priority.

Nursing diagnoses and interventions

The person with a bone tumour requires nursing care to meet many health problems, including prevention of injury, relief of pain, assistance with mobility and teaching about the disease process and treatment.

Risk of injury

In the person with a bone tumour, changes in bone tissue can cause pathological fractures.

- Teach how to avoid falls or injury to the tumour site, such as by using assistive devices when walking and ensuring the home environment is not conducive to falling (e.g. remove throw rugs and use night lights). *Pathological fractures may occur at the tumour site because bone destruction can weaken the area.*

- Provide referral to physical or occupational therapy for fitting of and teaching about assistive devices for ambulating, such as a cane, crutches or a walker. *Assistive devices can reduce the risk of falling when the person has significant weakness of an extremity or when balance has been affected by treatment of the disease.*

Acute pain, chronic pain

In the person with a bone tumour, pain may be related to direct invasion of the tumour or to pathological fractures. People may experience both acute and chronic pain.

- Develop strategies for controlling both acute pain (from surgery, fracture or inflammation) and chronic pain (from progression of the disease). *Analgesics combined with non-pharmacological methods of pain control provide optimum relief of pain. Chronic pain, when mild in nature, is best managed with NSAIDs or aspirin. Moderate pain is best managed with a combination of codeine and NSAIDs. Severe pain is best relieved with long-acting or sustained-relief narcotic analgesics.*
- Provide assistive devices (e.g. canes, walkers, crutches) when the person ambulates. *Assistive devices lessen the pain by supporting weight bearing during ambulation.*

Impaired physical mobility

Pain, muscle wasting or surgical procedures can impair the physical mobility of the person with a bone tumour.

- Begin muscle-strengthening and active and passive ROM exercises immediately after surgery. A continuous passive motion (CPM) machine may be used after surgical procedures to either upper or lower extremities. *Muscle-strengthening exercises must be encouraged as soon as possible to prevent muscle wasting and shorten the rehabilitation period.*
- Encourage exercises that help strengthen the triceps muscles. *The triceps are the major muscles in the arms and must be strengthened to assist in use of crutches or other assistive devices.*
- For the person who has undergone an amputation of a lower extremity, encourage quadriceps and gluteal setting exercises and leg raises. *These exercises will benefit the person when the rehabilitation period begins.*

Decisional conflict

A lack of knowledge about the diagnosis and treatment regimen can impair the person's ability to make informed decisions about the treatment plan.

- Discuss issues related to diagnosis, radiological evaluation, biopsy, surgery, chemotherapy, radiation therapy, potential complications, alternative therapies, risks, benefits, nursing management, discharge plans, home care and long-term treatment and follow up. *The person requires this information in order to make informed decisions about treatment.*

Community-based care

The person with a primary bone tumour needs information about the disease, its potential consequences and treatment options. Present information in a matter-of-fact manner, taking time to listen to and address the person's and family's concerns. Discuss expected effects and potential side effects of surgery, chemotherapy and radiation therapy. Provide information about how to minimise side effects. Teach the postsurgical person about wound care, demonstrating dressing changes and stump care (if amputation has occurred). Provide the person with a list of local resources for obtaining supplies. Discuss activity and weight-bearing restrictions. Refer the person to physical therapy for teaching about ambulation and appropriate muscle-group-strengthening exercises. Ensure that the person who has experienced an amputation is working with or has a referral to a prosthetic specialist. For the person with metastatic disease, discuss hospice services and support groups for people with cancer.

CONNECTIVE TISSUE DISORDERS

Connective tissue is the most abundant and widely distributed body tissue. It not only connects body parts but also provides support; forms bones, cartilage and the walls of blood vessels; and attaches muscles to bones. Connective tissue consists of three elements: (1) long fibres embedded in a (2) non-cellular ground substance and (3) cells specific to the class of connective tissue. Fibres made up primarily of collagen, a protein, are the most abundant in connective tissue.

Connective tissue disorders, also known as collagen diseases, are a group of immune-mediated disorders. Although they appear to have a genetic component, their cause is unknown. Because connective tissue and collagen are widely distributed in many varied tissues, these are systemic diseases with diverse manifestations.

THE PERSON WITH SYSTEMIC SCLEROSIS (SCLERODERMA)

Systemic sclerosis, also known as **scleroderma** ('hardening of the skin'), is a chronic disease characterised by the formation of excess fibrous connective tissue and diffuse fibrosis of the skin and internal organs. The cause of scleroderma is unknown, but genetic, immune and environmental factors are thought to play a role. Although this uncommon disease is distributed worldwide, a higher incidence is noted in coal and gold miners and in people exposed to certain chemicals such as polyvinyl chloride, epoxy resins and aromatic hydrocarbons.

Pathophysiology

Abnormalities in cellular immune function are believed to contribute to the development of scleroderma. Abnormal proliferation of fibrous connective tissue occurs in affected tissues, including the skin, blood vessels, lungs, kidneys and other organs.

Scleroderma may be either localised, affecting the skin only, or generalised (systemic sclerosis), with both skin and visceral organ involvement. Localised involvement may occur as irregularly shaped patches of skin (morphea) or a line of disease on the arm, leg or side of the face (linear scleroderma) (International Scleroderma Network, 2015). Eighty per cent of people with generalised disease have limited involvement, frequently manifested by CREST syndrome, a combination of calcinosis (abnormal calcium salt deposition in the tissues), Raynaud's phenomenon, oesophageal dysfunction, sclerodactyly (localised scleroderma of the fingers) and telangiectasia (dilated, superficial blood vessels). The remainder of people with generalised systemic sclerosis have a diffuse form of the disease and a higher risk of visceral organ involvement. Infections and diseases of the cardiovascular, renal, pulmonary and central nervous systems are the most common causes of death in people with systemic sclerosis.

FAST FACTS

- Scleroderma affects over 5000 people in Australia (Scleroderma Australia, 2015).
- Scleroderma affects women more often than men by a ratio of approximately 3:1.
- Although it can occur at any age from infancy to older adulthood, the onset of scleroderma typically occurs between the ages of 25 and 55 years (Scleroderma Foundation, 2015).

Manifestations

The initial manifestations of systemic sclerosis are usually noted in the skin, which thickens markedly. Diffuse, non-pitting swelling also is noted. As the disease progresses, the skin begins to atrophy, becoming taut, shiny and hyperpigmented (see Figure 39.9). Facial skin tightening leads to loss of skin lines and a pursed-lip appearance. Skin tightness may limit mobility, particularly of the face and hands. Other skin manifestations include telangiectasias (flat, red areas caused by dilation of small blood vessels, usually noted on the face, hands and in the mouth) and calcium deposits, usually noted around joints.

Arthralgias and Raynaud's phenomenon are common early manifestations of systemic sclerosis. Raynaud's phenomenon (intermittent attacks of small-artery vasospasm) is characterised by pallor of the fingers followed by cyanosis and then reactive hyperaemia with redness. Attacks are usually triggered by cold temperatures.

The person with visceral organ involvement may have varied symptoms. Dysphagia is common, because the motility of the oesophagus is affected. Pulmonary involvement can lead to exertional dyspnoea due to impaired gas exchange and right-sided heart failure due to pulmonary hypertension. Involvement of the heart may cause manifestations of pericarditis and arrhythmias. Diarrhoea or constipation, abdominal cramping and malabsorption can occur when the GI tract is affected. Renal effects can lead to proteinuria, haematuria, hypertension and kidney failure.

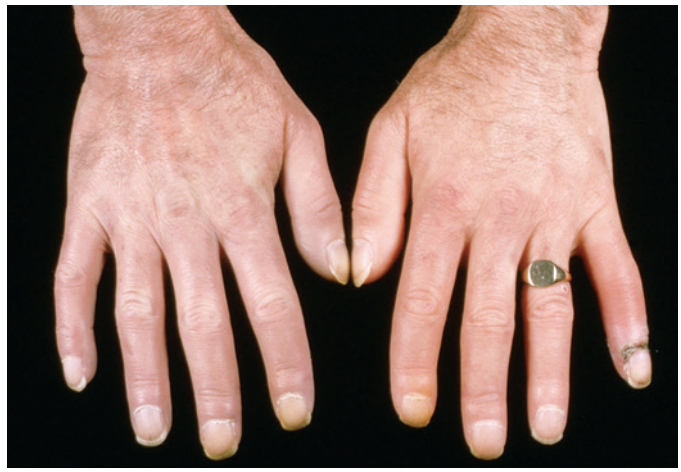


FIGURE 39.9 ■ Characteristic skin changes of scleroderma

Source: Joubert/Photo Researchers, Inc.

The prognosis for localised and limited scleroderma is good; many people have a normal lifespan. The course of diffuse systemic sclerosis is highly variable. This disease is usually progressive; complete remission is rare.

INTERPROFESSIONAL CARE

The manifestations of systemic sclerosis often allow diagnosis with little or no testing. No cure is currently available; treatment is symptomatic and supportive.

Diagnosis

No single diagnostic test is specific for systemic sclerosis, although a titre of 1:40 or higher for antinuclear antibody (ANA) is the most sensitive for diagnosis. Other laboratory studies that are done include an ESR, which is typically elevated from the chronic inflammatory process and an FBC, which will demonstrate anaemia. A skin biopsy may be done to confirm the diagnosis.

Medications

Medications to treat systemic sclerosis are chosen based on the person's symptoms. Immunosuppressive agents and corticosteroids are of limited benefit, but may be used to slow or prevent pulmonary fibrosis and in life-threatening disease. Penicillamine may be used to treat scleroderma and pulmonary fibrosis. Calcium channel blockers such as nifedipine or alpha-adrenergic blockers such as prazosin (Minipress) may be prescribed for people with Raynaud's phenomenon. When manifestations of oesophagitis accompany systemic sclerosis, H_2-receptor blockers

such as cimetidine (Tagamet) or ranitidine (Zantac), antacids or omeprazole (Losec; protein pump inhibitor), which block all gastric secretion, may be ordered. Tetracycline or another broad-spectrum antibiotic may be prescribed to suppress intestinal flora and relieve symptoms of malabsorption. People with kidney disease are usually treated with angiotensin-converting enzyme (ACE) inhibitors such as captopril (Capoten) to control hypertension and preserve kidney function. End-stage kidney disease is managed with dialysis and transplantation.

Physical therapy

Physical therapy is an important part of the management of systemic sclerosis to maintain mobility of affected tissues—the hands and face, in particular. Because the mouth opening, if involved, becomes increasingly smaller as the disease progresses, stretching and strengthening of facial muscles can be vital to maintaining oral food intake.

Nursing care

Nursing care needs of people with systemic sclerosis are individualised to the effects and manifestations of the disease, with interventions summarised in the following section.

Nursing interventions

Skin manifestations are present to some degree in nearly all people with scleroderma. Nursing care related to the skin focuses on maintaining skin integrity and flexibility. Measures to maintain supple skin are important because elasticity cannot be regained once it is lost. Apply moisturisers to prevent dryness and cracking. Protect the skin where it is stretched taut over joints or bony prominences. Perform ROM exercises to help prevent joint contractures due to increasingly tight skin.

Difficulty swallowing and recurrent oesophagitis may interfere with the person's nutritional status. Provide small, frequent meals. Consult with the dietitian and the person to determine which foods are easy to swallow. Keep the person in a sitting or Fowler's position after meals and elevate the head of the bed at night to minimise oesophageal reflux.

The dermatological and systemic effects of the disease may have significant psychological effects on the person, leading to feelings of helplessness and hopelessness and self-esteem disturbance. Establish an atmosphere of trust with the person. Listen actively and acknowledge concerns about the disease and its effects on the person's life and appearance. Encourage the person to share these concerns with family members and significant others. Provide referral to social services or counselling as appropriate.

The person with predominant pulmonary disease has nursing care needs similar to those of other people with restrictive respiratory disorders. If the person with systemic sclerosis has impaired renal function, nursing care is similar to that for people with chronic renal failure (see Chapter 27).

Community-based care

Teach the person with systemic sclerosis about the disease and introduce measures to help manage its effects. Stress the importance of good skin care and physical therapy exercises to maintain mobility, particularly of the hands and face. Discuss the need to avoid chilling (local and whole body) to prevent episodes of Raynaud's phenomenon. Teach the role of proper dress in the winter: loose, warm clothing, gloves and warm stockings. If needed, stress the need to stop smoking because of the vasoconstrictive effect of nicotine and the respiratory effects of the disease. Provide the person with information about manifestations of disease progression and organ involvement. Teach the person to report new or worsening symptoms to the doctor. In addition, suggest the following resources:

- Scleroderma Australia: www.sclerodermaaustralia.com.au
- Scleroderma New Zealand: http://scleroderma.org.nz.

THE PERSON WITH SJÖGREN'S SYNDROME

Sjögren's syndrome is an autoimmune disorder that causes inflammation and dysfunction of exocrine glands throughout the body. Sjögren's syndrome primarily affects women, with a ratio of women to men at 9:1. The highest incidence is between the ages of 40 and 60 years. Although it can occur as a primary disorder, Sjogren's syndrome is often associated with other rheumatic disease, including rheumatoid arthritis, SLE, primary biliary cirrhosis, scleroderma, Hashimoto's thyroiditis and interstitial pulmonary fibrosis (McPhee et al., 2015).

Pathophysiology

In this disease, exocrine glands in many areas of the body are destroyed by infiltration of lymphocytes and deposits of immune complexes. The salivary and lacrimal glands are particularly affected, leading to the characteristic manifestations of *xerophthalmia* (dry eyes) and *xerostomia* (dry mouth). People often experience dry, gritty-feeling eyes and may develop corneal ulcerations. Mucosal dryness affects taste, smell, chewing and swallowing and leads to increased dental caries. Parotid gland enlargement is common. Excess dryness can also affect the nose, throat, larynx, bronchi, vagina and skin. Systemic effects of Sjögren's syndrome include arthritis, dysphagia, pancreatitis, pleuritis, neurological manifestations including migraine and vasculitis. Nephritis may occur, but kidney failure rarely results. People with Sjögren's syndrome have a greatly increased risk of developing malignant lymphoma.

INTERPROFESSIONAL CARE

The diagnosis of Sjögren's syndrome is often based on the person's history and clinical presentation. Schirmer's test, which measures the quantity of tears secreted in a 5-minute period in response to irritation, ocular staining and slit-lamp examination

of the eye may be performed. A definitive diagnosis can be made by biopsy of either the lacrimal or the salivary gland.

Treatment is supportive. Artificial tears are used to decrease eye irritation and dryness. The person can keep the mouth moist by drinking fluids, using a saliva substitute and chewing sugarless gum. Medications that increase mouth dryness, such as atropine and decongestants, should be avoided.

Nursing care

Nurses caring for people with Sjögren's syndrome need to teach measures to protect the person's eyes and oral mucosa. Instil artificial tears as needed. Encourage the person to sip fluids throughout the day. Provide frequent oral hygiene, particularly before and after meals. Ensure that the person has sufficient fluids to drink during meals, because fluids help with chewing and swallowing.

THE PERSON WITH FIBROMYALGIA

Fibromyalgia is a common rheumatic syndrome characterised by musculoskeletal pain, stiffness and tenderness. The cause is unknown, but possible aetiologies include sleep disorders, depression, infections and an altered perception of normal stimuli. Fibromyalgia can be a complication of hypothyroidism, rheumatoid arthritis or (in men) sleep apnoea. It closely resembles chronic fatigue syndrome, except that musculoskeletal pain is predominant in fibromyalgia whereas fatigue is a more significant feature of chronic fatigue syndrome.

Pathophysiology

No inflammatory, structural or physiological muscle changes have been demonstrated in fibromyalgia. A connection between fibromyalgia and the central nervous system is being studied.

Manifestations

A gradual onset of chronic, achy muscle pain is typical, although the onset may be sudden, occasionally following a viral illness. The pain may be localised or involve the entire body. The neck, spine, shoulders and hips are often affected. Pain is produced by palpating localised 'tender points' (see Figure 39.10). Local tightness or muscle spasm may also occur. Systemic manifestations of fibromyalgia include fatigue, sleep disruptions, headaches, morning stiffness, painful menstrual periods and problems with thinking and memory (called the 'fibro fog'). Pain and fatigue are aggravated by exertion.

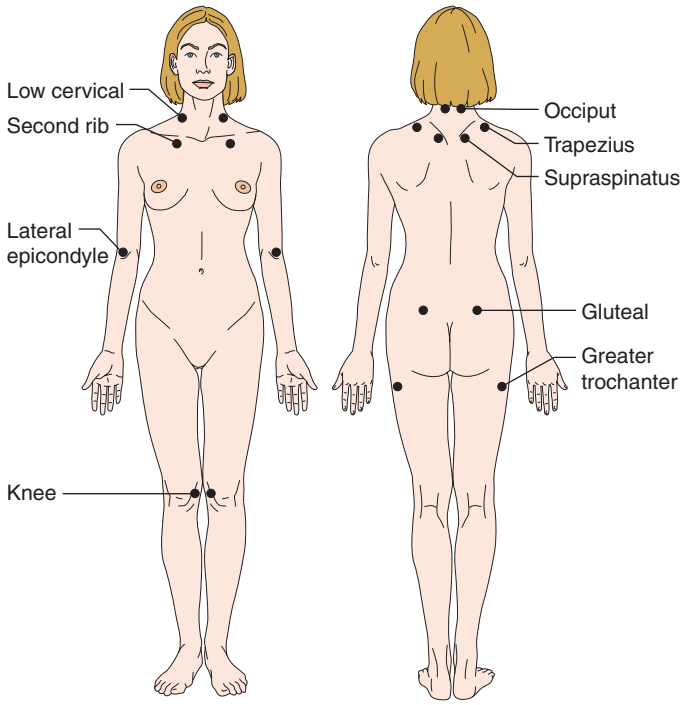


FIGURE 39.10 ■ Location of 'tender points' in fibromyalgia

INTERPROFESSIONAL CARE

The diagnosis of fibromyalgia is based on the history and physical assessment. The criteria—developed by the American College of Rheumatology and used in Australia and New Zealand—that are used for diagnosis are a history of widespread pain that has been present for at least 3 months, and pain at 11 of the 18 tender points on palpation. There are no laboratory or diagnostic tests for the disorder, although tests may be performed to rule out other rheumatic disorders, such as rheumatoid arthritis or SLE. Fibromyalgia also may occur as a complication of hypothyroidism, so thyroid function studies are performed.

Acknowledgement of the person's symptoms and the chronic but treatable nature of this disease is important (Huynh, Yanni & Morgan, 2008). Therapeutic measures include a program of structured aerobic exercise for conditioning, as well as stretching exercises.

Heated pool treatments with or without exercise have been shown to be beneficial. Evidence to support other treatments, such as cognitive behavioural therapy, hypnotherapy, biofeedback and acupuncture, is mixed (Huynh et al., 2008).

There are no medicines currently registered specifically for the treatment of fibromyalgia in Australia. Medical treatments regularly used in the treatment of fibromyalgia in Australia include tricyclic antidepressants (TCAs) which promote better sleep, paracetamol, non-steroidal anti-inflammatories (NSAIDs) and stronger analgesics as required (TGA, 2012). Milnacipran capsules have been listed for the management of fibromyalgia. Milnacipran assists in the treatment of chronic pain and fatigue. It is currently listed as restricted benefit in Australia.

Nursing care

Nursing care for people with fibromyalgia is supportive and educational, and provided in community settings such as clinics and other primary care settings. It is important to validate people's concerns and reassure them that their symptoms are not 'all in the head'. This syndrome is recognisable and manageable; its course is not progressive. Teach people about the disorder and reassure them that it resolves uneventfully in most instances. Provide verbal and written instructions about the use of heat, exercise, stress-reduction techniques and prescribed medications to relieve manifestations. In addition, suggest the following resources:

- Arthritis Australia: www.arthritisaustralia.com.au
- Fibromyalgia SA: sacfs.asn.au/download/fm_overview.pdf.

STRUCTURAL DISORDERS

Structural disorders of the musculoskeletal system most commonly affect the spine. The disorders discussed in this section are spinal deformities and lower back pain.

THE PERSON WITH SPINAL DEFORMITIES

Scoliosis and kyphosis are the two most common deformities of the spinal column. *Scoliosis* is a lateral curvature of the spine. *Kyphosis* is excessive angulation of the normal posterior curve of the thoracic spine. (See Figure 39.11.)

An estimated 1 in 10 people in Australia are affected by scoliosis (Scoliosis Association of Australia, 2015). It usually is diagnosed in adolescence, with girls more affected than boys by a ratio of 8:1. Idiopathic scoliosis is the most common form of the disorder, accounting for approximately 75% of cases. Congenital and neuromuscular disorders such as cerebral palsy, poliomyelitis and muscular dystrophy account for the rest (Porth & Matfin, 2014; Scoliosis Association of Australia, 2015).

Pathophysiology

Detailed discussions of the causes and treatment of scoliosis and kyphosis in younger people can be found in paediatric nursing textbooks. This discussion focuses on the nursing care of adults with these disorders. The manifestations of scoliosis and kyphosis are listed in the box below.

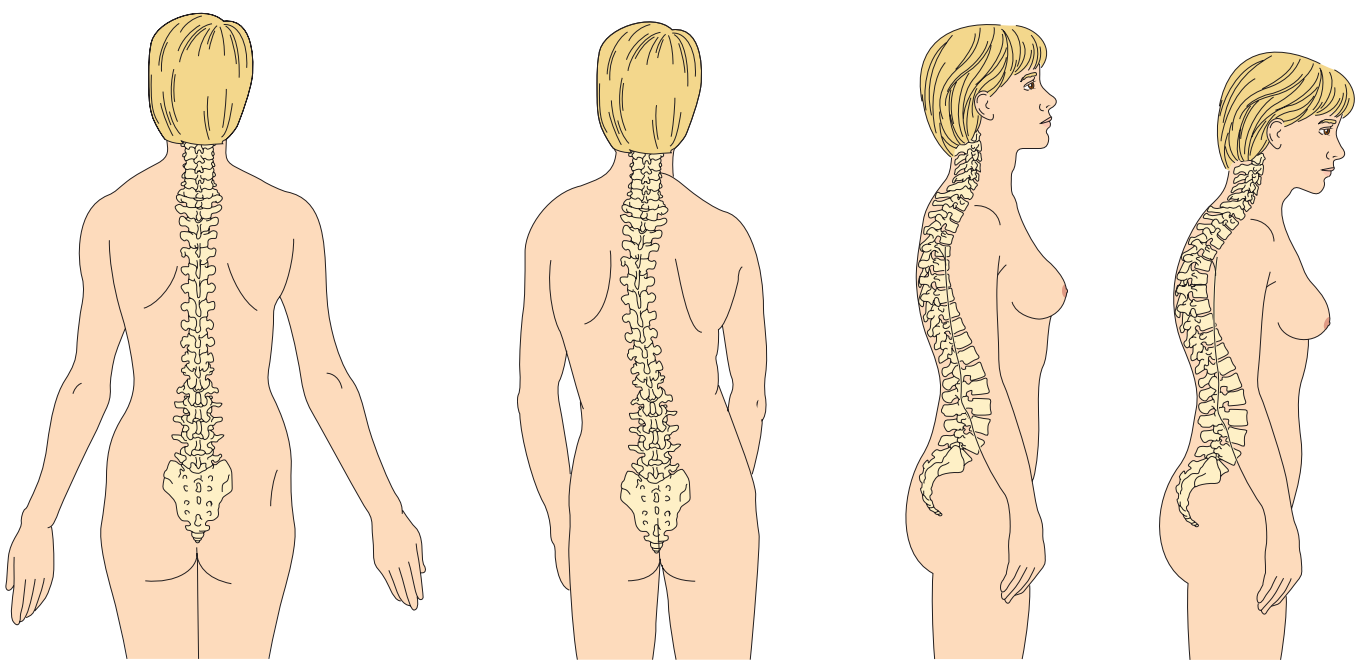


FIGURE 39.11 ■ Common deformities of the spinal column. *A*, Scoliosis is a lateral curvature of the spine. *B*, Kyphosis is an exaggerated posterior curvature of the thoracic spine

MANIFESTATIONS Scoliosis and kyphosis

SCOLIOSIS
- Asymmetry of shoulders, scapulae, waist creases
- Prominence of the thoracic ribs or paravertebral muscles on forward bend
- Lateral curvature and vertebral rotation on posteroanterior x-ray film

SEVERE SCOLIOSIS
- Back pain
- Shortness of breath
- Anorexia, nausea

KYPHOSIS
- Posterior rounding at the thoracic level
- Kyphotic curve of over 45 degrees on x-ray film

Scoliosis

Scoliosis is classified as *postural* when the small curve corrects with bending, and *structural* when the curve does not correct with bending (Porth & Matfin, 2014). Most people requiring treatment have structural scoliosis, a curve caused by a fixed deformity.

The lateral curve that occurs in scoliosis is usually evident in the thoracic, lumbar or thoracolumbar regions of the spine. The vertebral bodies in these spinal regions can be rotated as well as curved to one side or the other.

As scoliosis emerges, the soft tissues (muscles and ligaments) shorten on the concave side of the curvature. Over time, progressive deformities of the vertebral column and ribs develop, causing one-sided compression of the vertebral bodies. The degree of compression and twisting varies according to the location of each vertebra within the curved portion of the spine.

If the lateral curvature is less than 40 degrees when the person's spine reaches maturity, the risk of further progression during adult life is small. However, the spine becomes unstable if the lateral curvature is greater than 50 degrees and curvature likely will worsen throughout the person's lifetime.

Scoliosis is usually first noted by the deformity it causes, such as one shoulder that is higher than the other, a prominent hip or a projecting scapula. Pain is present in severe cases, usually in the lumbar region. Pain also may be caused by pressure on the ribs or the crest of the ilium. Shortness of breath may result from diminished chest expansion and gastrointestinal disturbances may occur because of crowding of the abdominal organs.

Kyphosis

Like scoliosis, kyphosis is classified as postural or structural. Postural kyphosis is caused by a slumping posture. Structural kyphosis may result from congenital malformations or paediatric disorders such as rickets or poliomyelitis. However, kyphosis also may occur during adulthood from vertebral tuberculosis and Paget's disease, or from metabolic disorders such as osteoporosis and osteomalacia. The condition can also result from the surgical removal or radiation of intervertebral discs for the treatment of spinal cord tumours or cysts.

The manifestations of kyphosis include moderate back pain and increased curvature of the thoracic spine as viewed from the side ('hunchback'). Impaired mobility and respiratory problems may occur in cases of severe curvature.

INTERPROFESSIONAL CARE

Diagnosis of scoliosis and kyphosis is important to prevent severe spinal deformity in the adult. The person stands with the arms relaxed and hanging freely at the sides while the examiner evaluates the person from both the back and the front for symmetry of the shoulders, scapulae, waist creases and the length of the arms. The person then bends forwards and the examiner observes for prominence of the thoracic ribs or vertebral muscles. The person is then viewed from the side while the screener looks for increased thoracic rounding or lumbar swayback.

A scoliometer is used to quantify the prominence of any curvatures noted during the examination. The scoliometer is placed at the apex of the curvature. A reading of greater than 10 degrees requires referral to a doctor (Porth & Matfin, 2014).

Diagnosis

Upright posteroanterior and lateral x-rays are used to confirm the diagnosis of curvature of the spine. For the person with scoliosis, the degree of curvature is measured by determining the amount of lateral deviation to the left or right. For the person with kyphosis, anteroposterior and lateral views typically reveal wedging of the vertebrae.

Treatments

Scoliosis and kyphosis may be treated conservatively or with surgery.

CONSERVATIVE TREATMENT Braces, electrical stimulation and traction may be used to prevent progression of scoliosis and kyphosis in younger people whose skeletons have not yet matured. Unfortunately, these approaches are ineffective in adults. Conservative treatment for adults with scoliosis and kyphosis may include weight reduction, active and passive exercises, and the use of braces for support.

SURGERY The use of surgery to correct spinal deformities depends on factors such as the degree of curvature and the person's overall physical, emotional and neurological status. Even with surgery, it is not possible to correct the abnormal curvature completely. The surgical procedure involves attaching metal reinforcing rods to the vertebrae and is usually performed using an anterior approach, although more severe curvature may require both an anterior and a posterior approach. The types of straightening devices used most frequently use bilateral rods with wire hooks or screws that stabilise the spine and correct the deformity.

Nursing care

Nursing diagnoses and interventions

Nursing interventions focus on minimising the risk of injury and neurological impairment.

Risk of injury

People with spinal deformities are at risk of injury from several sources, including structural aspects of bracing both before and after surgical intervention, dislocation of hooks and rods resulting from improper alignment or movement of the back and changes in body position after prolonged immobilisation.

- Assess the environment for safety hazards. *The person needs to learn to use handrails on stairways and take precautions when walking on slippery surfaces or areas with throw rugs.*

CONSIDERATION FOR PRACTICE

Some braces do not allow the person to flex or hyperextend the spinal column.

- Teach the person ways to reduce irritation of skin surfaces beneath the brace: wearing a smooth cotton T-shirt or cotton tube under the brace at all times, changing undergarments at least once daily and washing them with a mild soap. Undergarments should be changed more frequently in warmer weather. *The person wearing a brace is especially prone to skin breakdown and must take precautions to prevent it.*

CONSIDERATION FOR PRACTICE

Teach the person to avoid lotion and body powders; they may irritate the skin.

- Teach the person to loosen the brace during meals and for the first 30 minutes after each meal. *People have difficulty eating if the brace is tight. Loosening the brace during and after each meal will allow adequate nutritional intake and promote comfort.*
- Teach people how to apply the brace and explain ambulatory restrictions. *People requiring a brace need to learn how to apply the brace prior to ambulating. Ambulation is frequently restricted to walking rather than sitting for long periods.*
- Turn people who have undergone spinal surgery by using the log-rolling technique. *People require a position change at least every 2 hours. The use of a turn sheet and sufficient assistance allow the nurse to maintain the person's proper body alignment during the turning procedure.*
- Use a fracture bedpan following surgery. *The fracture bedpan provides minimal misalignment of spine and is more comfortable.*

Risk of peripheral neurovascular dysfunction

Surgical procedures can lead to neurological impairment in the person with a spinal deformity.

- Monitor the movement and sensation of lower extremities every 2 hours for the first 8 hours, then every shift and as needed. *Neurological assessment related to sensation and movement of the lower extremities is necessary because the surgical procedure is in close proximity to spinal nerves. Swelling of the surgical site can impinge on the spinal nerves and cause a loss of sensation and movement.*

Community-based care

People with structural scoliosis or kyphosis need reassurance that the condition was not caused by poor posture. If a brace is prescribed to relieve pain and other symptoms associated with the disorder, provide verbal and written instructions for wearing the brace, such as the number of hours per day it is to be worn and activity restrictions to follow when wearing or not wearing the brace. Teach the person how to protect and care for skin under the brace.

Surgical people need postoperative teaching regarding site care and activities. People who have spinal surgery often are allowed to ambulate fairly soon after surgery, but sitting may be restricted because of the stresses it places on the spine. Instruct the person to notify the healthcare provider if numbness, tingling, pain or weakness of an extremity develops after surgery.

Discuss the importance of not smoking and of avoiding respiratory infections for people with scoliosis or kyphosis that restricts respiratory excursion. Encourage these people to obtain pneumococcal pneumonia and influenza immunisations.

THE PERSON WITH LOWER BACK PAIN

Acute or chronic lower back pain involves the lumbar, lumbosacral or sacroiliac areas of the back. In most cases, lower back pain is due to strains in the muscles and tendons of the back caused by abnormal stress or overuse. Lower back pain caused by degenerative disc disease and herniated vertebral discs is covered in Chapter 42.

Pathophysiology

The pathophysiology of back pain varies with its many causes (see Box 39.6). In general, the five causes and types of back pain are as follows:

1. Local pain is caused by compression or irritation of sensory nerves. Fractures, strains and sprains are common causes of local pain; tumours also may press on pain-sensitive structures.
2. Referred pain may originate from abdominal or pelvic viscera.

BOX 39.6 Factors associated with back pain

Mechanical injury or trauma
- Muscle strain or spasm
- Compression fracture
- Lumbar disc disease

Degenerative disorders
- Spondylosis
- Spinal stenosis
- Osteoarthritis

Systemic disorders
- Osteomyelitis
- Osteoporosis or osteomalacia
- Neoplasms, primary or metastatic

Referred pain
- Gastrointestinal disorders
- Genitourinary disorders
- Gynaecological disorders
- Abdominal aortic aneurysm
- Hip pathology

Other
- Fibromyalgia
- Psychiatric syndromes
- Chronic anxiety
- Depression

3. Pain of spinal origin (i.e. pain associated with pathology of the spine such as disc disease or arthritis) may be referred to other structures such as the buttocks, groin or legs.
4. Radicular back pain is sharp, radiating from the back to the leg along a nerve root. This pain may be aggravated by movements such as coughing, sneezing or sitting.
5. Muscle spasm pain is associated with many spine disorders, although its origin may be unclear. This type of back pain is dull and may be accompanied by abnormal posture and taut spinal muscles.

MANIFESTATIONS Lower back pain

ALTERATIONS IN GAIT AND FLEXION
- Walking in a stiff, flexed state
- Inability to bend at waist
- Limp, which may indicate impairment of the sciatic nerve

NEUROLOGICAL INVOLVEMENT
- When tested for light and deep touch with a pin and cotton ball, may feel sensations in both limbs but experience a stronger sensation in the unaffected side
- Loss of both bowel and bladder control due to involvement of the sacral nerve

PAIN
- Pain in the affected leg when walking on heel or toes
- Continuous, knife-like localised pain in muscles close to the affected disc
- Pain that radiates down posterior of leg
- Sharp, burning pain in the posterior thigh or calf
- Pain in middle of buttock
- Tenderness when muscle close to the affected disc is palpated
- Severe pain with straight leg-raising manoeuvre

Manifestations

People with lower back pain report pain ranging from mild discomfort lasting a few hours to chronic debilitating pain. Acute pain is usually caused when the person participates in an activity that is not usually pursued, such as unusual lifting or bending or playing an active sport. Manifestations are presented in the accompanying box.

INTERPROFESSIONAL CARE

Care of the person with lower back pain focuses on relieving pain, correcting the condition if possible, preventing complications and educating the person.

Diagnosis

The choice of diagnostic tests for the person with low back pain depends on the suspected diagnoses, clinical findings and history. Current guidelines for care recommend that radiography, CT scans and MRI be used only with clinical signs of a potentially serious underlying condition. Diagnostic testing may be considered if pain and other manifestations continue to limit the person after 4 weeks of conservative treatment. Diagnostic tests are described in Chapter 37.

Medications

The medications of choice for lower back pain include NSAIDs and analgesics. NSAIDs block prostaglandin production and reduce inflammation, thus relieving the pain. Muscle relaxants, such as cyclobenzaprine (Flexeril), methocarbamol (Robaxin) or carisoprodol (Soma), may be used, but little evidence supports their efficacy.

Epidural steroid injections may be used to help reduce intense, intractable pain. A steroid solution is injected into the epidural space, which helps decrease the swelling and inflammation of the spinal nerves.

Conservative treatment

The majority of people with acute lower back pain need only a short-term treatment regimen. Limited rest, combined with appropriate exercise and education, is often the primary method of treatment. There is no evidence that activity is harmful or aggravating to the source of pain. In fact, activity promotes bone and muscle strength and may increase endorphin levels. Therefore, active rehabilitation helps to restore function and reduce pain.

Pain may be relieved by an ice bag or hot water bottle (or heating pad) applied to the back. Exercise programs are helpful provided that the person begins gradually and increases activity gradually as the recovery process continues. Physical therapy procedures include diathermy (deep heat therapy), ultrasonography, hydrotherapy and transcutaneous electrical nerve stimulation (TENS) units. These therapies reduce the muscle spasms and pain temporarily. They are frequently used in combination with exercise to provide early mobilisation for the person.

Nursing care

Nursing care of the person with lower back pain focuses on relieving the pain. In addition, most people have very little understanding of the anatomy of the spine, the reasons for the pain, the choices for treatment and the importance of self-management. Therefore, education is another essential aspect of treating lower back pain.

Health promotion

Recommendations for preventing back pain from the National Institute of Neurological Disorders and Stroke (2015) include the following:

- Have a regular exercise program.
- Stretch before working in the garden, jogging and playing sports.
- Quit smoking.
- Lose weight, if needed.
- Maintain a correct posture.
- Use supportive seats when driving.
- Lift by bending at the knees rather than at the waist.
- Reduce emotional stress that causes muscle tension.

In industrial and work settings, nurses should be alert for situations that increase the risk of back pain and injury. Office workers should have chairs with appropriate seat height and length and back support. Modifications of work space or machinery may be necessary for industrial workers to avoid excess stresses on back muscles. Finally, it is important to remember that back pain is a leading cause of lost work time for nurses themselves. Remind co-workers to use good body mechanics and to seek help when lifting or moving people.

Nursing diagnoses and interventions

Nursing interventions for the person with lower back pain are based on problems with acute pain, deficient knowledge and risk of impaired adjustment.

Acute pain

Muscle spasms and inflammation are among the contributing factors of lower back pain.

- Teach the person appropriate comfort measures. *People with lower back pain have discomfort due to muscle spasms and/or inflammation due to nerve compression, surgery or irritation from a brace.*
- Instruct the person to take NSAIDs or analgesics on a routine schedule rather than as needed. *Maintaining a constant blood level of the NSAIDs or analgesics reduces inflammation and provides continuous pain relief.*

Deficient knowledge

The person with lower back pain requires information regarding treatment modalities.

- Encourage the person to stay active. *There is little scientific evidence to show that bed rest is beneficial, but there is ample evidence about the adverse effects of bed rest. Staying in bed for more than 1 to 2 days can actually increase pain and cause joint stiffness and muscle weakness.*
- Teach the person about the 'rebound phenomenon' of prolonged heat or ice therapy. *Ice remaining on the skin longer than 15 minutes or heat longer than 30 minutes causes a reverse effect known as the rebound phenomenon. For example, heat produces maximum vasodilation in 20 to 30 minutes. Continuation of the application beyond 30 to 45 minutes causes tissue congestion and the blood vessels constrict. Likewise, with cold application, maximum vasoconstriction occurs when the skin reaches a temperature of 15°C. Prolonged cold can create a drop in temperature, at which time vasodilation occurs.*
- Provide instructions about appropriate back exercises such as partial sit-ups with the knees bent and knee–chest exercises to stretch hamstrings and spinal muscles. Each exercise should be done 5 times and gradually increased to 10 times. Advise the person to discontinue any exercise that is painful and to seek professional advice before continuing the exercise. *Repetition of prescribed back exercises, such as the pelvic tilt, partial sit-ups and back rolls, will strengthen the muscles that protect the spine and thus prevent back strain.*

Risk of impaired adjustment

The need for lifestyle changes may lead to impaired adjustment in people with back pain.

- Teach the use of appropriate body mechanics in lifting and reaching. The person should be instructed to plan the lift, keep the object being lifted close to the body and avoid twisting when lifting. Encourage the person to obtain help when lifting. *An item is considered excessively heavy if it equals 35% of the lifter's body weight.*
- Instruct the person to modify the workplace or environment to minimise stress to the lower back. *Lumbar supports in chairs, adjustment of chair or table height, and rubber floor mats help prevent back strain or injury.*
- Encourage obese people to lose weight. The trunk of the body must carry excess weight when the person is obese. *Obese people are further away from the objects they lift because of their greater abdominal girth. They may also have more difficulty squatting to lift. The greater the distance between an object and the person's centre of gravity, the higher the risk of straining the lower back.*

Community-based care

Back pain is a common problem in Australia and other industrialised countries. Nurses can have an effect on this significant problem by teaching health practices to prevent back injury to people of all ages. Teach people how to safely lift, bend and turn when engaging in physical activity. Stress the importance of using the large muscle groups of the legs to lift rather than bending and lifting with the smaller muscles of the back. Teach other aspects of good body mechanics, including posture, sleeping on a firm mattress and sitting in chairs that provide good support. Discuss the positive effect of maintaining optimal body weight and good physical fitness.

THE PERSON WITH COMMON FOOT DISORDERS

Hallux valgus, hammer toe and Morton's neuroma are common foot disorders that cause pain or difficulty in walking. All three disorders may be caused by wearing poorly fitting or confining shoes. These disorders are more prevalent among women.

Pathophysiology

Hallux valgus

Hallux valgus, commonly called a *bunion*, is the enlargement and lateral displacement of the first metatarsal (the great toe) (see Figure 39.12). Hallux valgus develops when chronic pressure against the great toe causes the connective tissue in the sole of the foot to lengthen so that the stabilising action of the great toe is gradually lost. The toe bends laterally away from the midline of the body and the metatarsophalangeal joint (MTP) is exposed to friction during walking and becomes enlarged. As the deformity progresses, calluses form over the metatarsal head and bursitis develops in the MTP. In severe cases, the lateral displacement of the great toe may approach 70 to 90 degrees and the second toe may be forced upwards, causing hammer toe. Although bunions may be a congenital disorder, most are caused by wearing pointed, narrow-toed shoes or high heels.

Hallux valgus is obvious on physical examination of the foot. The person may report an inability to fit into shoes. Often, the person may report joint pain or pain around calluses. In advanced or severe cases, the first metatarsal joint may have limited range of motion, particularly in dorsiflexion, and crepitus (crackling or popping) may occur during joint movement.

Hammer toe

Hammer toe (claw toe) is the dorsiflexion of the first phalanx with accompanying plantar flexion of the second and third phalanges. The condition may affect any toe, but the second toe is most commonly affected. People initially experience mild inflammation of the synovial membranes of the involved joints. As the deformity progresses, the dorsiflexed joint rubs against the overlying shoe, causing painful corns to develop.

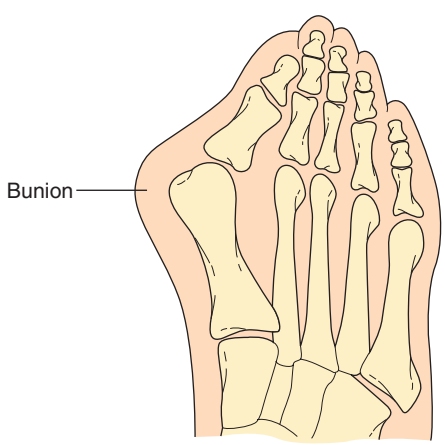


FIGURE 39.12 ■ Hallux valgus (bunion)

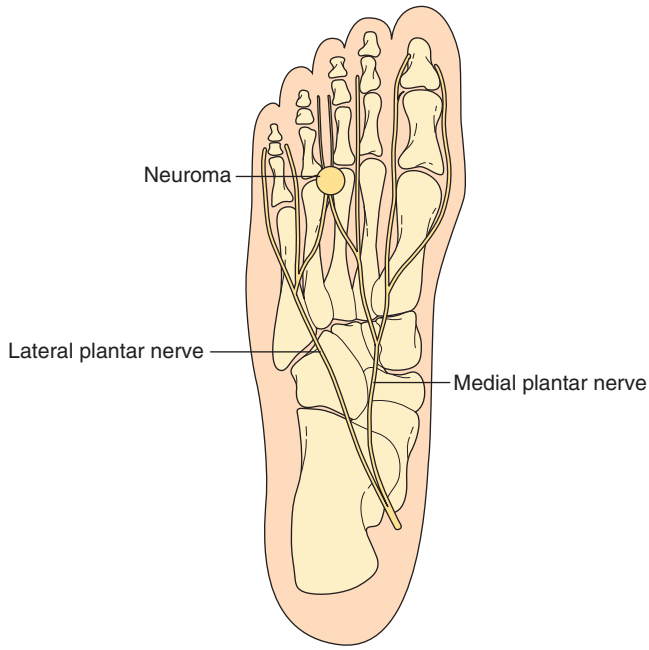


FIGURE 39.13 ■ Morton's neuroma

Morton's neuroma

Morton's neuroma is a tumour-like mass formed within the neurovascular bundle of the intermetatarsal spaces (see Figure 39.13). The neuromas usually occur in only one foot, most frequently in the third web space. Like other common foot disorders, Morton's neuroma usually is caused by wearing tight, confining shoes. The condition develops when repeated compression of the toes causes irritation and scarring of tissues surrounding the plantar digital nerve. The affected nerve becomes inflamed and swells. After repeated episodes of inflammation, the nerve fibres become fibrotic and a neuroma forms.

Manifestations include a burning pain at the web space of the affected foot that radiates into the tips of the involved toes. Weight bearing usually worsens any symptoms; removing the shoe and massaging the foot often relieves the pain. The neuroma may present as a palpable mass between the affected toes. The area over the neuroma usually is tender.

INTERPROFESSIONAL CARE

Care of the person with common foot disorders such as hallux valgus, hammer toe and Morton's neuroma focuses on relieving pain, correcting the structural deformity and preventing re-occurrence. In most cases, all three conditions are diagnosed by inspection. X-ray films of the affected foot are taken if the need for surgery arises.

Conservative treatment for common foot disorders usually involves the use of corrective shoes. Orthotic devices that cushion and stretch the affected joints may be placed within shoes or between the person's toes. For Morton's neuroma, metatarsal pads are used to spread the person's toes and

decompress the affected nerve. Analgesics may be prescribed to relieve pain and inflammation. In severe cases, corticosteroid drugs may be injected into the affected joints or surrounding tissue to relieve acute inflammation.

Surgery is reserved for people with intractable toe deformities or pain. Hallux valgus is treated with bunionectomy; ligaments are lengthened or shortened as needed, and pins are drilled into place so the toe remains in position. Similarly, the correction of hammer toe also involves straightening the affected toe and inserting pins to retain the correction. A cast may be applied over the foot following surgery to correct toe deformities. Surgery for Morton's neuroma causes loss of sensation to a portion of the foot because removing the neuroma involves cutting out a portion of the plantar nerve.

Nursing care

Nursing care for people with these foot deformities focuses on the same areas because the conservative treatment and preoperative and postoperative interventions are similar.

Nursing diagnoses and interventions

Pain relief, prevention of infection and person education are important components of the nursing care of people with foot disorders.

Chronic pain

In the person with a foot deformity, constant pressure of footwear over the involved joint can cause pain.

- Instruct people to wear corrective footwear to assist in the conservative treatment of foot problems. *Pain related to foot problems can result from improper footwear that does not provide proper toe room; in addition, heels higher than 2.5 cm can cause constant flexion and hyperextension problems. In some instances, the person must purchase special shoes or orthotics to ensure correct fit and relief of symptoms.*
- Suggest purchasing appropriate pads to wear over painful bunions, calluses/corns and the ball of the foot. *Protective pads are manufactured for specific foot problems; these include bunion pads, corn pads and metatarsal pads.*
- Instruct people to remove pads and inspect the skin every other day. *People who have difficulty reaching or observing the involved foot should ask another person to do the inspection for them. It is especially important to emphasise the need for inspection to people who have experienced loss of sensation of the feet due to such disorders as diabetes and chronic peripheral vascular disease.*

Risk of infection

Like all surgeries, foot surgery carries a risk of infection. This risk may be increased because of impaired peripheral circulation and exposure of the feet to the environment.

- Teach people proper care and cleaning of exposed pins implanted during the surgical procedure. *Pins inserted into soft tissue of the toes and bones are prone to becoming infected and can potentially result in osteomyelitis.*
- Teach people how to keep pins and casts dry while bathing or ambulating in inclement weather. *People must wear a plastic bag over the cast or pins when bathing or walking in rain or snow. When casts or pins are exposed in water, infection may result.*

Community-based care

For people in all age groups, teach the importance of well-fitting footwear. Discuss the long-term effects of wearing high-heeled shoes with constricting toes, with women in particular. Suggest alternatives for stylish footwear and encourage people to wear supportive and non-restrictive footwear at all times. Discuss the possible effects of bunions on balance and talk about safety measures to prevent falls and injury. Teach people techniques to relieve pressure on affected joints.

CHAPTER HIGHLIGHTS

- Metabolic bone disorders begin in the bone remodelling process and may result from ageing, calcium and phosphate imbalances, genetics and changes in hormone levels. The disorders include osteoporosis, Paget's disease, gout and osteomalacia.
- Osteoporosis is a major health problem in Australia, with fractures being the most common complication. Health promotion activities to prevent development of the disease include a calcium-rich diet, weight-bearing exercise and a healthy lifestyle.
- Gout is characterised by hyperuricaemia and the deposit of tophi in the subcutaneous tissues. Attacks of the disease typically begin with an acutely painful inflammation of the first joint of the great toe.
- Degenerative musculoskeletal disorders include osteoarthritis (OA) and muscular dystrophy (MD). OA is the most commonly occurring of all forms of arthritis and a leading cause of pain and disability in older adults. The disease is characterised by loss of cartilage in articulating joints and hypertrophy of bone at the articular margins. Pain and inflammation are most often conservatively managed with NSAIDs.
- If pain and disability are not controlled in people with arthritis, total joint replacements may be performed.
- Autoimmune and inflammatory disorders of the musculoskeletal system include rheumatoid arthritis (RA), ankylosing spondylitis (AS), reactive arthritis (ReA), systemic lupus erythematosus (SLE) and polymyositis.
- Although the cause of RA is unknown, it is believed to be a combination of genetic, environmental, hormonal and reproductive factors. RA is a systemic disease, affecting one or many joints with the risk of severe contractures and deformity and also causes fatigue, weakness, anorexia,

weight loss and fever. The primary objectives of treatment and care are to reduce pain and inflammation, preserve function and prevent deformity.

- SLE is a chronic inflammatory connective tissue disease, affecting almost all body systems, including the musculoskeletal system. Skin lesions are a common manifestation, exhibited by a characteristic rash on the face. The person with SLE is at increased risk of infection.
- Osteomyelitis and septic arthritis are infectious musculoskeletal disorders. Osteomyelitis may be the result of a blood-borne pathogen, a contiguous infection or a complication of vascular insufficiency. Septic arthritis is a medical emergency, requiring immediate treatment to preserve joint function.
- Bone tumours may be benign or malignant, primary or metastatic. The primary manifestations of a bone tumour are pain, a mass and impaired function. Nursing care is directed towards teaching to prevent injury and interventions to relieve pain.
- Scleroderma is a chronic disease characterised by the formation of excess connective tissue and diffuse fibrosis of the skin and internal organs. It may be either localised or generalised. Other connective musculoskeletal disorders are Sjögren's syndrome and fibromyalgia.
- Structural musculoskeletal disorders affecting the spine are manifested by scoliosis, kyphosis and lower back pain. Those commonly affecting the feet are hallux valgus, hammer toe and Morton's neuroma.

CONCEPT CHECK

1 Although all of the following nursing diagnoses are important when planning care for the person with osteoporosis, which is most significant in terms of long-term disability?
1 *Chronic pain*
2 *Risk of falls*
3 *Activity intolerance*
4 *Acute pain*

2 You are preparing a teaching plan for a woman with osteoarthritis. Which group of medications should you prepare to discuss?
1 opioids
2 antibiotics
3 hormones
4 NSAIDs

3 You are monitoring the laboratory reports for a person with an acute attack of gout. Which of the following measurements would you expect to be increased?
1 haematocrit
2 uric acid
3 alkaline phosphatase
4 creatinine

4 What is a potential complication of both osteoporosis and osteomalacia?
1 infection
2 blood clots
3 fractures
4 contractures

5 You are assessing a woman who has come to an orthopaedic clinic complaining of knee pain. Which of the following assessments you made would indicate an increased risk of osteoarthritis?
1 being overweight by 15 kg
2 having a history of falls
3 eating a diet high in calcium
4 walking 30 minutes each day

6 A postoperative nursing care plan for a person who has had a total knee replacement includes monitoring vital signs and laboratory results. The rationale for these interventions is to:
1 teach the person the importance of these assessments
2 promote rapport between the person and the healthcare providers
3 ensure adequate circulation to the involved extremity
4 prevent the progression of infection

7 When comparing osteoarthritis and rheumatoid arthritis, what assessment finding would be different in the person with rheumatoid arthritis?
1 Health history includes weight loss and fever.
2 Abnormal joint findings are limited to the hands.
3 Stiffness is relieved by activity.
4 Heberden's nodes are located on the finger joints.

8 *Ineffective protection* is an appropriate nursing diagnosis for the person with SLE. What would be your most important intervention for the hospitalised person?
1 Monitor laboratory findings.
2 Provide appropriate skin care.
3 Practise careful handwashing.
4 Administer prescribed medications.

9 Of the different types of arthritis, which one is considered a medical emergency, requiring immediate diagnosis and treatment?
1 osteoarthritis
2 septic arthritis
3 reactive arthritis
4 gouty arthritis

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UNIT 10 BUILDING CLINICAL COMPETENCE

Responses to altered musculoskeletal function

CLINICAL SCENARIO

- You have been assigned to work with the following four people for the 0700 shift on an orthopaedic unit. Significant data obtained during report are as follows:
- Barry Drummond is a 70-year-old Indigenous man with type 2 diabetes mellitus who is 3 days postoperative with bilateral below-the-knee amputations. Vital signs are T 37.2°C, P 88, R 24, BP 150/92. He is complaining of feeling pain in his feet.
- Joyce Stevens is an 84-year-old who is 2 days postoperative for hip replacement surgery. Her vital signs are T 37.5°C, P 100, R 30 and shallow, BP 110/86. She is confused when spoken to. Petechiae have been noted on her arms and legs. She is complaining of difficulty breathing.
- James Gunning, a 21-year-old, was admitted with osteomyelitis of the upper right leg. He has a history of a gunshot wound to the leg. Vital signs are T 39.2°C, P 98, R 22, BP 138/80. He is scheduled for surgical debridement of the wound this morning. He is complaining of pain and requesting pain medication.
- Kim Wong is a 30-year-old who was admitted with manifestations of painful and swollen joints, muscle pain, pale and cyanotic fingers and toes and oedema of the legs and periorbital areas. Her vital signs are T 38.1°C, P 78, R 16, BP 108/72. She is complaining of extreme fatigue. She is to have blood drawn for complete blood count (FBC), anti-DNA antibody testing and serum complement levels.

Critical thinking questions

1 In what order would you visit these people after report?

1.
2.
3.
4.

2 What top two priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Barry Drummond		
Joyce Stevens		
James Gunning		
Kim Wong		

3 After the amputation wound is dressed, what is the person taught to do to toughen the stump?

1. Dangle the stump for 20 minutes every hour while awake.
2. Push the stump into soft and then harder surfaces.
3. Elevate the stump on two pillows, keeping the knee straight.
4. Apply prosthesis over the compression dressing.

4 To prevent hip contractures in the person with an above-the-knee amputation, what does the nurse instruct the person to do?

1. Lie supine for short periods throughout the day.
2. Elevate the stump above the level of the heart.
3. Perform active range-of-motion exercises every 8 hours.
4. Avoid sitting in a chair for prolonged periods of time.

5 The nurse explains to the person with gout that a low-purine diet is recommended. The person understands a low-purine diet when which meal is ordered?

1. ham and asparagus casserole
2. chicken and potatoes
3. chilli and spinach salad
4. prawn and scallop pasta

6 The person's laboratory results are haematocrit of 28%, haemoglobin of 8 g/dL, WBC count of 4000/mm^3, platelet count of 98 000/mL, eosinophil sedimentation rate of 100 mm/h, positive anti-DNA antibodies. What medical diagnosis is supported by these lab values?

1. systemic lupus erythematosus
2. rheumatoid arthritis
3. ankylosing spondylitis
4. polymyositis

7 People who have autoimmune diseases such as systemic lupus erythematosus are at increased risk of developing what disease?

1. chronic renal failure
2. hypertension
3. liver insufficiency
4. coronary heart disease

8 A prescription for ibuprofen (Nurofen) is given on discharge to the person with rheumatoid arthritis. Which toxic effects of the medication does the nurse instruct the person about?

1. diarrhoea, nausea and vomiting
2. blurred vision, tinnitus and headache
3. gastric irritation, ulceration and bleeding
4. dizziness, dry mouth and abdominal cramps

9 When performing a neurovascular assessment, which are included in the initial and focused assessments? (Select all that apply.)

1. pain
2. paroxysm
3. pallor
4. pulses
5. paresis
6. pallaesthesia
7. paraesthesia

10 Which person is at greatest risk of developing osteoporosis?

1. menopausal, Caucasian woman who smokes one packet of cigarettes a day
2. menopausal, Indigenous Australian woman who has diabetes and hypertension
3. premenopausal, underweight Asian woman who is allergic to dairy products
4. premenopausal, obese Indigenous woman who has a sedentary lifestyle

11 An older adult sprained an ankle after tripping on an uneven footpath. Which is the most important intervention?

1. Use a walker when ambulating.
2. Take anti-inflammatory and pain medications to reduce ankle pain.

3. Follow a regimen of rest, ice, compression and elevation.
4. Immobilise the ankle with an air splint.

12 Which actions by the nurse need to be followed when caring for the person with osteomyelitis?

1. Place the person in a private room and use gloves and gown with wound care and good handwashing.
2. Place the person in a semiprivate room with another infected person and use isolation precautions for both people.
3. Place the person near the nurse's station and use standard precautions when caring for the person.
4. Place the person at the end of the hall away from other people to prevent spread of infection and teach the person to use good handwashing.

CASE STUDY

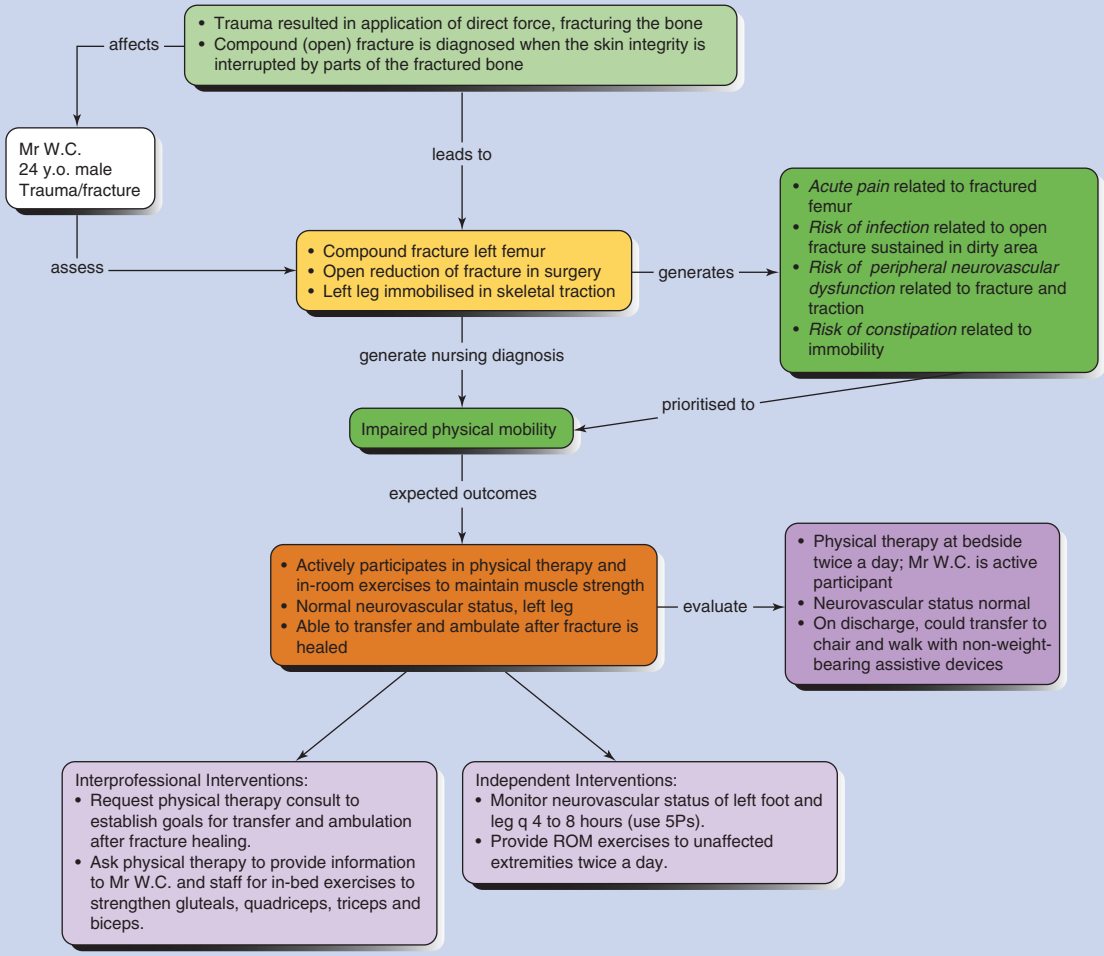
William Comfort is a 24-year-old Caucasian male admitted with a compound fracture of the left femur. He states he was quad biking on a hillside footpath and was thrown from the vehicle. He slid approximately 30 metres down the hill on his left side. His fall was stopped when his foot became tangled in some bushes. On admission, his vital signs were T 37.7°C, P 100 and thready, R 24, BP 116/70. His height is 1.88 m and weight is 89 kg. Assessment revealed an open fracture of the left leg with bleeding and oedema around the open site and severe pain on movement of the leg. Popliteal and pedal pulses are difficult to palpate. His left leg is pale and cool to touch with a capillary refill of 4 seconds. He states his leg feels numb. Multiple lacerations and abrasions are noted on his left trunk and arm. He states he does not have any medical problems and has not seen a doctor in the past 5 years. He is employed as a computer technician. He lives in an apartment with two friends.

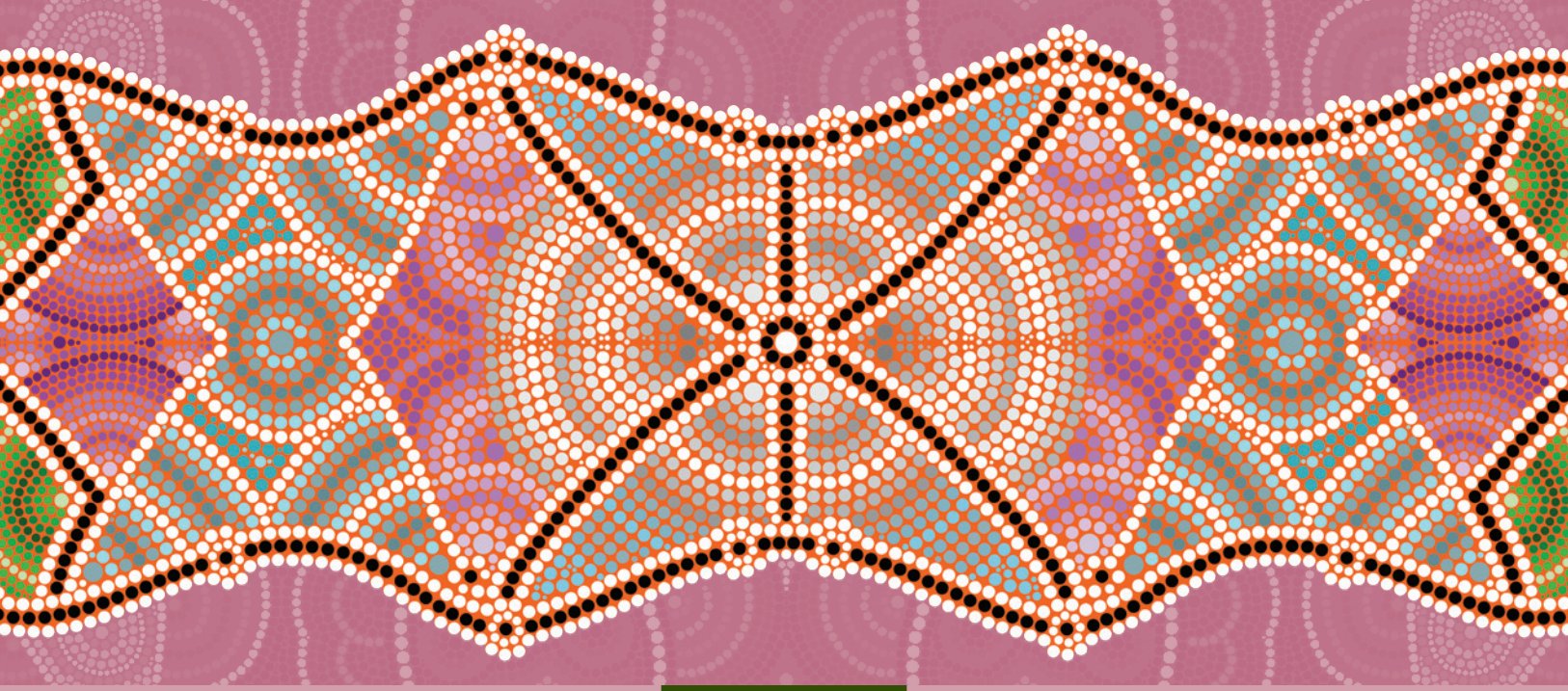
Blood is drawn for a baseline compete blood count (FBC) and urine is obtained for a urinalysis. An intravenous line is started in the right arm with Hartmann's solution infusing at 150 mL/h. He is given a tetanus toxoid immunisation and is medicated with morphine sulfate for pain. X-rays are taken of the left leg, left arm and abdomen. The wounds are cleansed with an antibacterial solution and antibiotic ointment is applied. He went to surgery for an open reduction of the left leg fracture and has been placed in skeletal traction to separate the bony fragments and reduce and immobilise the left leg fracture.

The pathophysiology of a femur fracture is a large amount of force applied to the shaft of the femur, resulting in breaking of the bone. An open fracture is diagnosed when the bone is broken with bone fragments protruding through the skin. Manifestations of a femur fracture are oedema and a deformed and painful thigh. The person is unable to move the hip or knee. Popliteal and pedal pulses are difficult to palpate. Capillary refill time is increased. Pallor and coolness indicate arterial compromise. Sensations to the leg may be burning, numbness, prickly feeling or stinging. Complications of a femur fracture include hypovolaemia, fat embolism, dislocation of the hip or knee, muscle atrophy and ligament damage.

Skeletal traction is the application of a pulling force through placement of pins into the bone. Pins are inserted under sterile conditions into the bone. One or more pulling forces may be applied to maintain alignment of the femur fracture. The disadvantages of skeletal traction are increased anxiety, increased risk of infection and increased discomfort.

When planning nursing care for Mr Comfort, the nursing diagnosis of *Impaired physical mobility* related to fracture of the left femur with skeletal traction is appropriate for implementing nursing interventions.





UNIT 11

RESPONSES TO ALTERED NEUROLOGICAL FUNCTION

CHAPTER 40

A PERSON-CENTRED APPROACH TO ASSESSING THE NERVOUS SYSTEM

DEBRA RAYMOND

KEY TERMS

LEARNING OUTCOMES

- Describe the anatomy, physiology and functions of the nervous system.
- Identify specific topics for consideration during a health history assessment interview of a person with a neurological disorder.
- Explain techniques for assessment of neurological function, including examinations of mental status, cranial nerves, sensory nerves, motor nerves, cerebellar function and reflexes.
- Identify manifestations of impairment of neurological function.
- Describe normal variations in assessment findings for the older adult.

CLINICAL COMPETENCIES

- Conduct and document a health history for a person with or at risk of alterations in the neurological system.
- Conduct and document a physical assessment of neurological structures and functions.
- Perform specific neurological assessments for people with suspected meningeal irritation and for people who are disoriented or comatose.
- Monitor the results of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Cotton balls
- Safety pin
- Tongue depressor
- Tuning fork
- Reflex hammer
- Pencil and paper
- Penlight
- Printed materials
- Substances to test the senses of smell and taste

The nervous system regulates and integrates all body functions, muscle movements, senses, mental abilities and emotions. It collects information from the internal and external environments as sensory input, processes and interprets the input and causes responses that are manifested as motor or sensory output.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE NERVOUS SYSTEM

The nervous system is divided into two regions: the central nervous system (CNS), which consists of the brain and spinal cord; and the peripheral nervous system (PNS), which consists of the cranial nerves, the spinal nerves and the autonomic nervous system.

Nerve cells, action potentials and neurotransmitters

The highly integrated CNS and PNS consist of only two types of cells: neurons, which receive impulses and send them on to other cells; and neuroglia, which protect and nourish the neurons.

Neurons

Each neuron consists of a dendrite, a cell body and an axon (see Figure 40.1). The dendrite is a short process (projection) from the cell body that conducts impulses towards (afferent) the cell body. Cell bodies, most of which are located within the CNS, are clustered in ganglia or nuclei. The cell bodies and dendrites comprise what is often called the grey matter of the CNS. The axon, a long process, conducts impulses away (efferent) from the cell body. Many axons are covered with a myelin sheath, a white lipid substance. It is interrupted at intervals in unmyelinated areas called nodes of Ranvier, which allow movement of ions between the axon and the extracellular fluid. The myelin sheath serves to increase the speed of nerve impulse conduction in axons and is essential for the survival of larger nerve processes. Myelinated nerve fibres comprise the white matter of the brain and spinal cord.

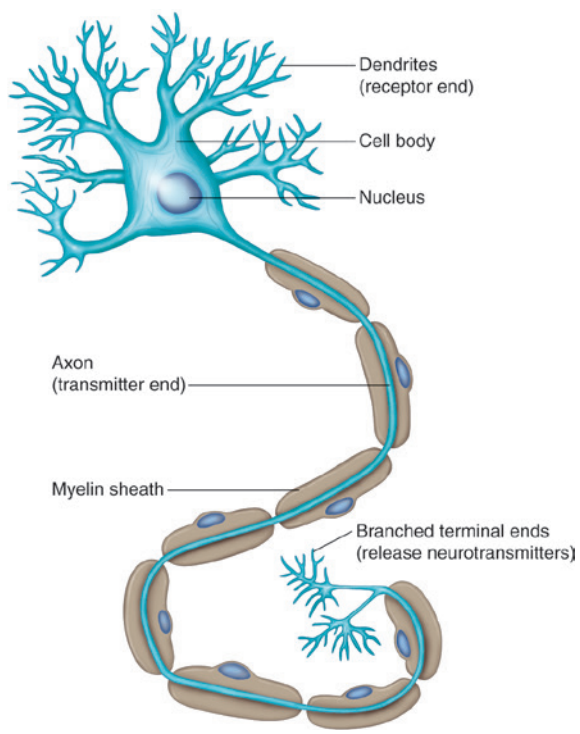


FIGURE 40.1 ■ A typical neuron

Neuroglia

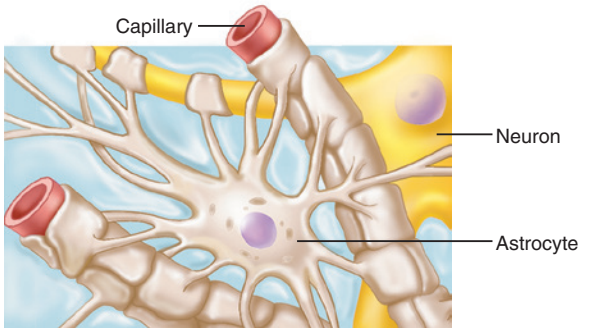
Neuroglia or glia cells are divided into six types of neuroglia—two in the PNS, which are *satellite cells* which surround neuron cell bodies with ganglia, and *Schwann cells* which surround and form myelin sheaths around the larger nerve fibres in the PNS (see Figure 40.2). The four types of neuroglia cells in the CNS are: *astrocytes* which support and bind and nourish the neurons; *oligodendrocytes* that produce myelin that surrounds axons within the CNS forming the myelin sheath; *ependymal cells* that line the central cavities of the brain and the spinal cord and function in the production of cerebral spinal fluid (CSF); and *microglia* which have a phagocytic function and assist in the removal of microorganisms or neuronal debris (Marieb & Hoehn, 2015).

Action potentials

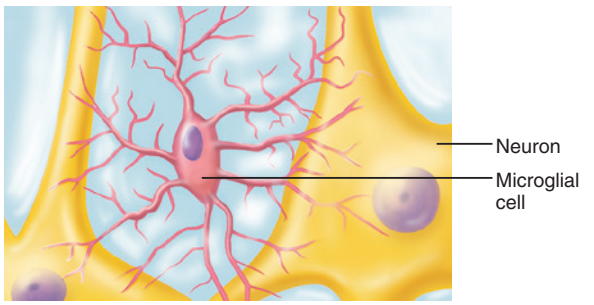
Action potentials are impulses (movements of electrical charge along an axon membrane) that allow neurons to communicate with other neurons and body cells. They are initiated by stimuli and propagated by the rapid movement of charged ions through the cell membrane. When a neuron reaches a certain level of stimulation, an electrical impulse is generated and conducted along the length of its axon. The movement of impulses to and from the CNS is made possible by afferent and efferent neurons. Afferent, or sensory, neurons have receptors in skin, muscles and other organs and relay impulses to the CNS. Efferent, or motor, neurons transmit impulses from the CNS to cause some type of action.

Nerve impulses occur when a stimulus reaches a point great enough to generate a change in electrical charge across the cell membrane of a neuron. A neuron that is not involved in impulse conduction is in a resting, or polarised, state, in which the number of positive ions in the fluid outside the cell membrane is greater than in the fluid within the cell. The chief regulators of membrane potential are sodium and potassium: sodium is the main positive ion in the extracellular fluid and potassium is the main positive ion in the intracellular fluid. In response to an electrical stimulus, the cell membrane becomes permeable to sodium, which moves into the cell. This changes the polarity of the cell membrane and the neuron is said to depolarise. This event stimulates an action potential, or a nerve impulse, to travel down the axon. When the charges and ions return to their original resting state, the neuron is repolarised. The events in an action potential are as follows:

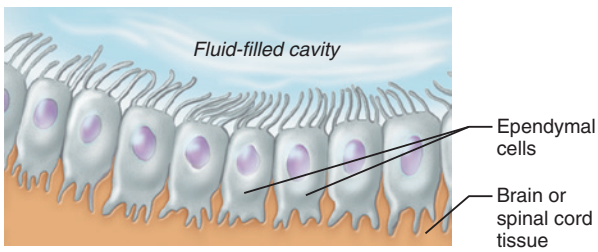
1. Initially, sodium permeability increases. As the membrane is depolarised, sodium channels open and sodium rushes into the cell to a point of depolarisation. (The inside of the cell becomes less negative in comparison to the outside of the cell.)



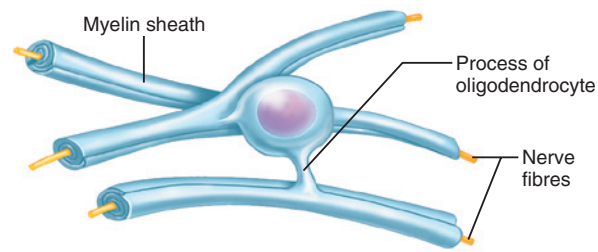
(A) Astrocytes are the most abundant CNS neuroglia



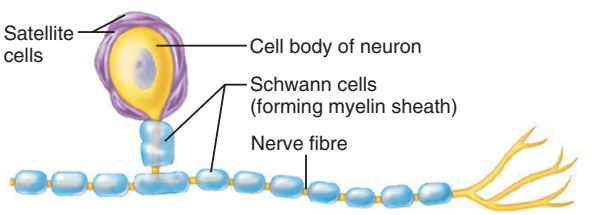
(B) Microglial cells are defensive cells in the CNS



(C) Ependymal cells line cerebrospinal-fluid-filled cavities



(D) Oligodendrocytes have processes that form myelin sheaths around CNS nerve fibres



(E) Satellite cells and Schwann cells (which form myelin) surround neurons in the PNS

FIGURE 40.2 ■ Neuroglia. *A–D*, Supporting cells of the CNS. *E*, Supporting cells of the PNS

Source: E. N. Marieb & K. N. Hoehn (2015). *Human anatomy and physiology* (10th ed.). Upper Saddle River, NJ: Pearson Education. © 2015. Printed and electronically reproduced by permission of Pearson Education, Inc.

2. This is followed by a decrease in sodium permeability, lasting only about 1 millisecond. The sodium gates close and the sodium influx stops.
3. The final event is an increase in potassium permeability. The potassium gates open, potassium rushes out of the cell and the cell interior becomes progressively less positive. The membrane potential moves back to its resting state and is repolarised.

The action potential is generated only at the point of the stimulus; but, once generated, it is propagated along the entire length of the axon regardless of whether the stimulus continues. Conduction of the impulse is rapid in myelinated fibres, with the action potential 'jumping' from one node of Ranvier to the next. The conduction of the impulse is slower in unmyelinated fibres.

Neurotransmitters

Neurotransmitters are the chemical messengers of the nervous system. When the action potential reaches the end of the axon at the presynaptic terminal, a neurotransmitter is released and travels across the synaptic cleft to bind with receptors in the postsynaptic neuron dendrite or cell body. The neurotransmitter may either be inhibitory or excitatory. The excitatory neurotransmitter is almost always acetylcholine (ACh), which is rapidly degraded by the enzyme acetylcholinesterase. Noradrenaline (NA), which may be either excitatory or inhibitory, is another major neurotransmitter.

Nerves that transmit impulses through the release of ACh are called *cholinergic*. Receptors that bind ACh are found in the viscera, skeletal muscle cells and the adrenal medulla (where they stimulate the release of adrenaline). The effect of ACh binding may be either to stimulate or to inhibit a response.

Nerves that transmit impulses through the release of NA are called *adrenergic*. Receptors that bind NA are found in the heart, lungs, kidneys, blood vessels and all target organs stimulated by the sympathetic division except the heart. Adrenergic receptors are further divided into alpha and beta types. Alpha-adrenergic receptors help control such varied functions as arterial vasoconstriction and pupil dilation. Beta-adrenergic fibres may be either $beta_1$- or $beta_2$-receptors. $Beta_1$-receptors are found in the heart, where they regulate the rate and force of contraction. $Beta_2$-receptors are found in receptor cells of the lungs, arteries, liver and uterus; they help regulate bronchial diameter, arterial diameter and glycogenesis. Generally, binding of NA to alpha-receptors stimulates a response, whereas binding to beta-receptors inhibits a response.

Other neurotransmitters include gamma aminobutyric acid (GABA), which inhibits CNS function; dopamine, which may be inhibitory or excitatory and helps control fine movement and emotions; and serotonin, which is usually inhibitory and controls sleep, hunger and behaviour and also affects consciousness.

The central nervous system

The central nervous system consists of the brain and spinal cord, highly evolved clusters of neurons that act to accept, interconnect, interpret and generate a response to nerve impulses originating throughout the body.

The brain

The brain is the control centre of the nervous system and also generates thoughts, emotions and speech. Males and females have equivalent brain sizes, averaging 1450 g in weight for a female and 1600 g for a male. The brain is surrounded by the skull, a bony structure that provides support and protection. The brain has four main regions: the cerebrum, the diencephalon, the brainstem and the cerebellum (see Figure 40.3). The general functions of these regions are summarised in Table 40.1.

The two hemispheres of the cerebrum account for almost 60% of brain weight. The surface of the cerebrum is folded into elevated ridges of tissue called gyri, which are separated by shallow grooves called sulci. Deep grooves, called fissures, further divide the surface of the cerebrum. The longitudinal fissure separates the hemispheres and the transverse fissure separates the cerebrum from the cerebellum. In addition, each cerebral hemisphere is divided into frontal, parietal, temporal and occipital lobes (see Figure 40.4).

The cerebral hemispheres are connected by a thick band of nerve fibres called the corpus callosum, which allows communication between the two hemispheres. Each hemisphere receives sensory and motor impulses from the opposite side of the body. One of the cerebral hemispheres tends to develop more than the other. Most people have a more highly developed left hemisphere, which is responsible for the control of language. The right hemisphere has greater control over nonverbal perceptual functions.

The cerebral cortex is the outer surface of the cerebrum. It consists of neuron cell bodies, unmyelinated fibres, neuroglia and blood vessels. The functions of the different lobes of the cerebrum and the specific areas of the cerebral cortex are shown in Figure 40.4 and listed in Table 40.2.

The diencephalon is embedded in the cerebrum superior to the brainstem. It consists of the thalamus, hypothalamus and epithalamus (see Figure 40.3). The thalamus begins to process sensory impulses before they ascend to the cerebral cortex. It serves as a sorting, processing and relay station for input into the cortical region. The hypothalamus, located inferior to the thalamus, regulates temperature, water metabolism, appetite, emotional expressions, part of the sleep–wake cycle and thirst. The epithalamus forms the dorsal part of the diencephalon and includes the pineal body, which is part of the endocrine system that affects growth and development.

BRAINSTEM The brainstem consists of the midbrain, pons and medulla oblongata (see Figure 40.3). The midbrain is a centre for auditory and visual reflexes. In addition, it functions

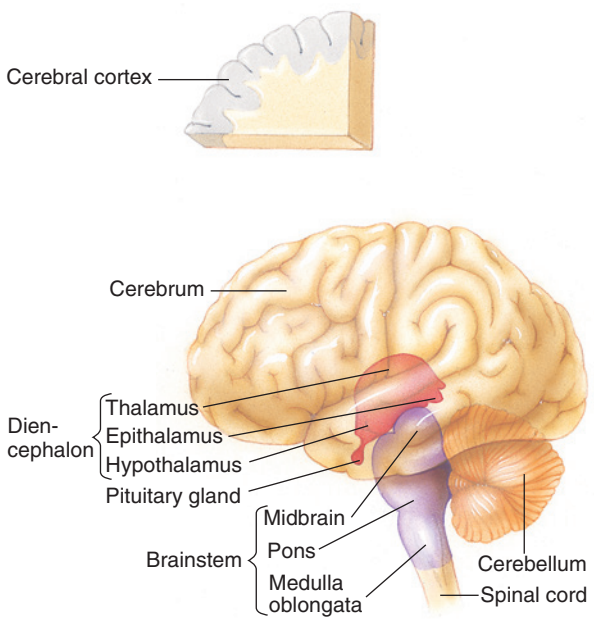


FIGURE 40.3 ■ The four main regions of the brain

TABLE 40.1 General functions of the four regions of the brain

REGION	FUNCTIONS
Cerebrum	Interprets sensory input. Controls skeletal muscle activity. Processes intellect and emotions. Contains skills memory.
Diencephalon	Conducts sensory and motor impulses. Regulates autonomic nervous system. Regulates and produces hormones. Mediates emotional responses.
Brainstem	Serves as conduction pathway. Serves as site of decussation of tracts. Contains respiratory nuclei. Helps regulate skeletal muscles.
Cerebellum	Processes information. Provides information necessary for balance, posture and coordinated muscle movement.

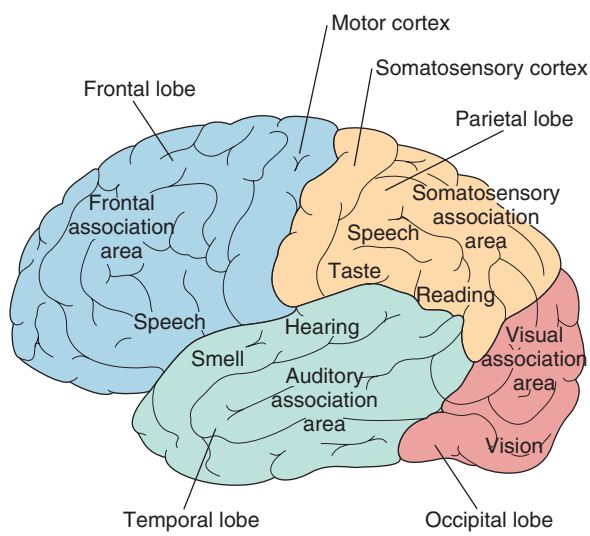


FIGURE 40.4 ■ Lobes of the cerebrum and functional areas of the cerebral cortex

TABLE 40.2 Functions of lobes of the cerebrum and areas of the cerebral cortex

AREA	FUNCTIONS
Parietal lobe (somatic sensory area of cerebral cortex)	Promotes recognition of pain, temperature and light touch. The left side receives input from the right side of the body, and vice versa.
Occipital lobe	Receives and interprets visual stimuli.
Temporal lobe	Receives and interprets olfactory and auditory stimuli.
Frontal lobe	Controls movements of voluntary muscles.
Primary motor area	Facilitates voluntary movement of skeletal muscles.
Speech area	Promotes understanding of spoken and written words.
Motor speech area (Broca's area)	Promotes vocalisation of words.

as a nerve pathway between the cerebral hemispheres and lower brain. The pons is located just below the midbrain. It consists mostly of fibre tracts, but it also contains nuclei that control respiration. The medulla oblongata, located at the base of the brainstem, is continuous with the superior portion of the spinal cord. Nuclei of the medulla oblongata play an important role in controlling cardiac rate, blood pressure, respiration and swallowing.

The cerebellum is connected to the midbrain, pons and medulla. Its functions include coordinating skeletal muscle activity, maintaining balance and controlling fine movements.

VENTRICLES The brain contains four ventricles, which are chambers filled with cerebrospinal fluid (CSF). They are linked by ducts that allow the CSF to circulate. One lateral ventricle is located within each hemisphere. These communicate with the third ventricle through the foramen of Monro. The third ventricle communicates with the fourth ventricle through the cerebral aqueduct that runs through the midbrain. The cerebral aqueduct is continuous with the central canal of the spinal cord.

CEREBROSPINAL FLUID Cerebrospinal fluid, a clear and colourless liquid, is formed by the choroid plexus, which are groups of specialised capillaries located in the brain ventricles. Derived from blood plasma, CSF consists of 99% water and contains protein, sodium, chloride, potassium, bicarbonate and glucose. (See Table 40.3 for normal laboratory values for CSF.) The usual amount of CSF ranges from 80 to 200 mL, averaging about 150 mL, and is replaced several times each day. It is absorbed by arachnoid villi. CSF is normally produced and absorbed in equal amounts. CSF circulates from the lateral ventricles of the cerebral hemispheres into the third ventricle, through the midbrain and into the fourth ventricle. Some CSF flows down the centre of the spinal cord as the rest of it circulates into the subarachnoid space and returns to the blood through the arachnoid villi. CSF forms a cushion for the brain tissue, protects the brain and spinal cord from trauma, helps provide nourishment for the brain and removes waste products of cerebrospinal cellular metabolism.

TABLE 40.3 Normal laboratory values for cerebrospinal fluid

COMPONENT	NORMAL VALUE
Appearance	Clear and colourless
pH	7.35
Specific gravity	1.007
WBCs	0–8 mm^3
Protein	0.15–0.45 g/L
Glucose	2.8–4.4 mmol/L
Chloride	118–132 mEq/L
Pressure	< 200 mmH_2O

MENINGES The brain and spinal cord are covered and protected by three connective tissue membranes called meninges. The meninges form divisions within the skull, enclose venous sinuses and contain CSF. The meninges have three layers (see Figure 40.5). The outermost double layer, the dura mater, is attached to the inner surface of the skull. The middle layer is the arachnoid mater, which encloses the entire CNS. It forms the subarachnoid space that contains CSF. The innermost layer, the pia mater, clings to the brain, spinal cord and segmental nerves and is filled with small blood vessels.

CEREBRAL CIRCULATION AND THE BLOOD–BRAIN BARRIER The brain receives about 750 mL of blood each minute and uses 20% of the body's total oxygen uptake.

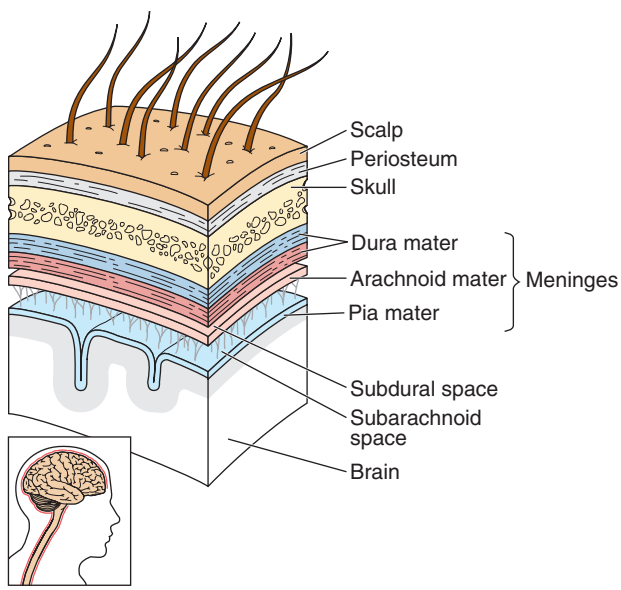


FIGURE 40.5 ■ Anatomy of the meninges

The large amount of oxygen is necessary for metabolism of glucose, which is the brain's sole source of energy. Blood flow to the brain is mostly controlled by autoregulatory or local mechanisms that respond to the brain's metabolic needs. Autoregulation is defined as the ability of the brain to maintain constant cerebral blood flow despite changes in systemic blood pressure. At least three metabolic factors affect cerebral blood flow: carbon dioxide, hydrogen ion and oxygen concentrations. Of these, increased carbon dioxide is the main stimulus for vasodilation with resultant increased cerebral blood flow.

The anterior part of the brain is supplied with blood by the two internal carotid arteries, and the posterior part of the brain is supplied with blood by the vertebral arteries. The internal carotid artery branches into further arteries: the ophthalmic, posterior communicating, anterior choroidal, anterior cerebral and middle cerebral. The brainstem and cerebellum receive their blood supply from the basilar artery. These main arteries are connected by small anterior and posterior communicating arteries, which form a circle of connected blood vessels called the circle of Willis (see Figure 40.6). This circle serves as a protective device, providing alternative routes for brain tissues to receive their blood supply.

The capillaries in the brain have low permeability because the cells that compose their walls join at very tight junctions and are surrounded by a basement membrane and by the processes of supporting cells in the brain (called astrocytes). As a result, the brain is protected from many harmful substances in the blood. This blood–brain barrier allows lipids, glucose, some amino acids, water, carbon dioxide and oxygen to pass through it, thus maintaining a controlled environment. Substances such as urea, creatinine, proteins, some toxins and most antibiotics cannot pass this barrier and enter brain tissue. However, injury to or infection of the brain may cause increased permeability of the blood–brain barrier, altering concentrations of proteins, water and electrolytes.

THE LIMBIC SYSTEM AND THE RETICULAR FORMATION The limbic system and the reticular formation are functional brain systems. These systems, made of networks of neurons, communicate across areas of the brain.

The limbic system consists of structures that form a ring of tissue in the medial side of each hemisphere, surrounding the upper portion of the brainstem and corpus callosum. The limbic system integrates and modulates input to make up the affective part of the brain, providing emotional and behavioural responses to environmental stimuli.

The reticular formation is located through the central core of the medulla oblongata, pons and midbrain. This system has widespread connections throughout the brain and relays sensory input from all body systems to all levels of the brain. The reticular formation includes the reticular activating system (RAS). The RAS is a stimulating system for the cerebral cortex, keeping it alert and responsive to incoming sensory stimuli while filtering out repetitive or unwanted stimuli. The sleep centre inhibits activity of the RAS, and drugs and alcohol may depress it. Other parts of the reticular formation include motor nuclei that help maintain muscle tone and coordinated movements through interconnections with spinal nerves and the vasomotor and cardiovascular regulatory centres, which are part of autonomic regulation of the cardiovascular system.

The spinal cord

The spinal cord extends from the medulla to the level of the first lumbar vertebra (see Figure 40.7). It serves as a centre for conducting messages to and from the brain and as a reflex centre. The spinal cord is about 42 cm long and 1.8 cm thick. The cord is protected by the vertebrae, the meninges and CSF. The grey matter of the cord is on the inside and the white matter is on the outside (the reverse of the arrangement in the brain).

The spinal cord is surrounded and protected by 33 vertebrae: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 fused

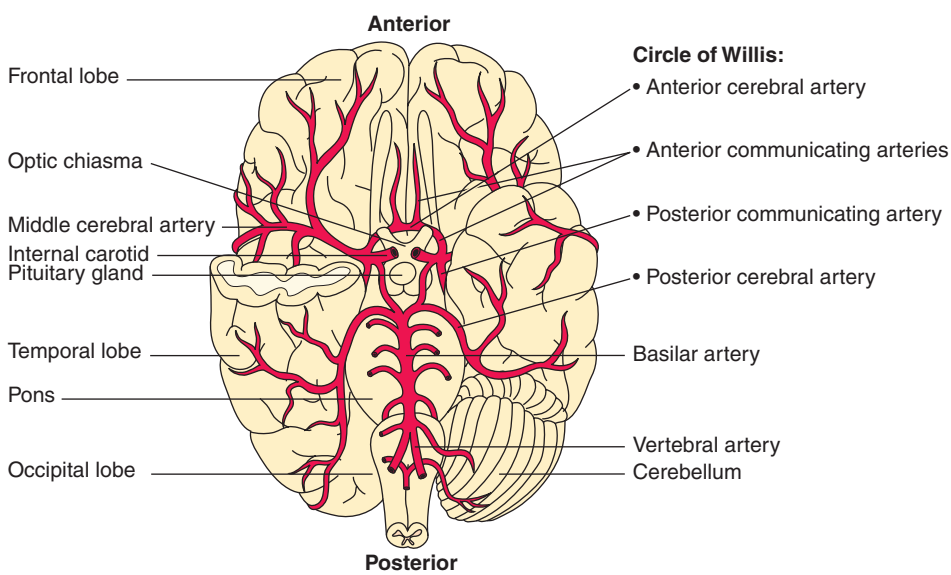


FIGURE 40.6 ■ The main arteries serving the brain and the circle of Willis

vertebrae, which form the coccyx. Each vertebra consists of a body and a vertebral arch formed by projections from the body. This arch encloses a space called the vertebral foramen. The vertebral foramina of all the vertebrae form the vertebral canal through which the spinal cord passes. Intervertebral foramina are spaces between the vertebrae through which spinal nerve roots pass as they exit the vertebral column.

Intervertebral discs are located between each of the movable vertebrae. Each disc is made of a thick capsule surrounding a gelatinous core called the nucleus pulposus. Ligaments that provide mobility and protection surround the vertebral column.

The roots of 31 pairs of spinal nerves, divided into the cervical, thoracic, lumbar, sacral and coccygeal nerves, arise from the cord (see Figure 40.7). Each separates into posterior (sensory) and anterior (motor) roots. Damage to the posterior roots results in loss of sensation, whereas damage to the anterior roots results in flaccid paralysis.

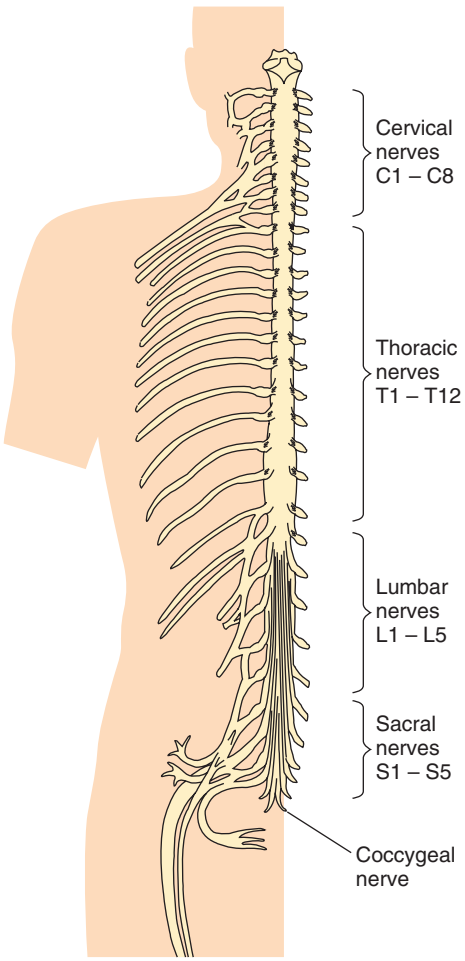


FIGURE 40.7 ■ Distribution of spinal nerves

FUNCTIONS OF THE SPINAL CORD AND SPINAL ROOTS Messages to and from the brain are conducted via ascending (sensory) pathways and descending (motor) pathways (see Figure 40.8). The main ascending tracts are the lateral and anterior spinothalamic tracts, which carry sensations for pain, temperature and crude touch; and the posterior tracts, which carry sensations for fine touch, position and vibration. The lateral and anterior corticospinal (pyramidal) tracts are descending tracts consisting of fibres that originate in the motor cortex of the brain and travel to the brainstem and then down the spinal cord. They mediate voluntary purposeful movements and stimulate certain muscular actions while inhibiting others. They also carry fibres that inhibit muscle tone. The rubrospinal, anterior and lateral reticulospinal and tectospinal (extrapyramidal) tracts include the pathways between the cerebral cortex, basal ganglia, brainstem and spinal cord outside the pyramidal tract. They maintain muscle tone and gross body movements.

UPPER AND LOWER MOTOR NEURONS Upper motor neurons, such as those of the corticospinal and extrapyramidal tract, carry impulses from the cerebral cortex to the anterior grey column of the spinal cord. Damage to upper motor neurons results in increased muscle tone, decreased muscle strength, decreased coordination and hyperactive reflexes. Lower motor neurons

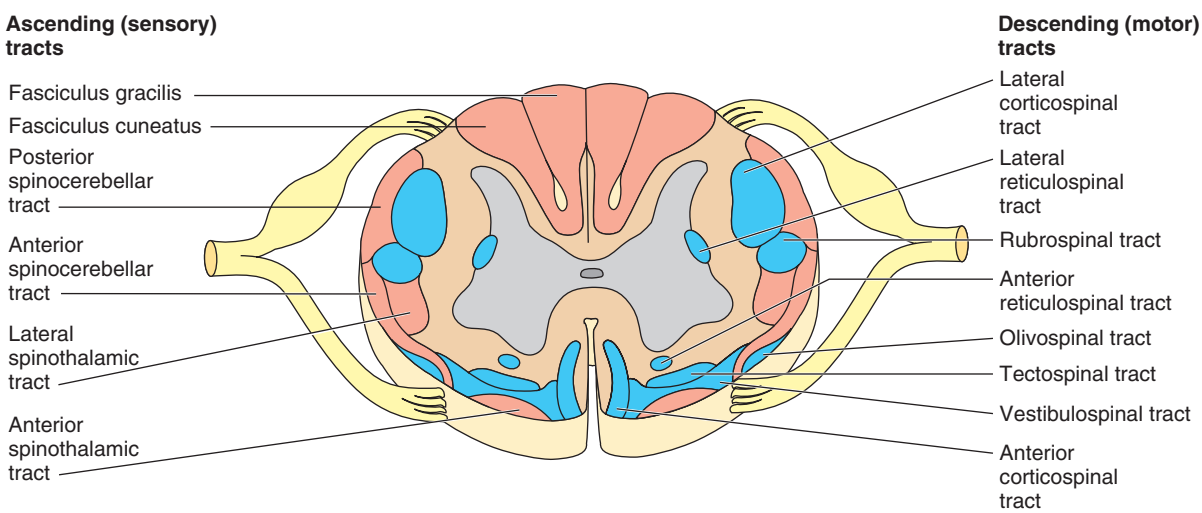


FIGURE 40.8 ■ Ascending and descending tracts of the spinal cord

begin in the anterior grey column of the spinal cord and end in the muscle. Damage to lower motor neurons results in decreased muscle tone, muscle atrophy, fasciculations and loss of reflexes.

The peripheral nervous system

The peripheral nervous system links the CNS with the rest of the body. It is responsible for receiving and transmitting information from and about the external environment. The PNS consists of nerves, ganglia (groups of nerve cells) and sensory receptors located outside—or peripheral to—the brain and spinal cord. The PNS is divided into a sensory (afferent) division and a motor (efferent) division. Most nerves of the PNS contain fibres for both divisions and all are classified regionally as either spinal nerves or cranial nerves.

Spinal nerves

The 31 pairs of spinal nerves (see Figure 40.7) are named by their location:

- cervical nerves: 8 pairs
- thoracic nerves: 12 pairs
- lumbar nerves: 5 pairs
- sacral nerves: 5 pairs
- coccygeal nerves: 1 pair.

Spinal nerves exit the vertebral column through intervertebral foramina to travel to the body regions they serve. The spinal cord does not reach the end of the vertebral column; as a result, the lumbar and sacral nerve roots travel inferiorly through the vertebral canal for some distance before exiting the vertebral column through their associated intervertebral foramina. This collection of descending nerve roots is called the cauda equina.

Each spinal nerve contains both sensory and motor fibres. The sensory fibres are located in the dorsal root and their cell bodies are located within the dorsal root ganglion. The motor fibres are located in the ventral root and their cell bodies are located within the spinal cord. The dorsal and ventral roots merge outside the vertebral canal just past the dorsal root ganglion, forming a spinal nerve. Each spinal nerve further divides into branches called rami.

The ventral rami of the cervical, brachial, lumbar and sacral regions form complex clusters of nerves called plexuses. The main spinal nerve plexuses innervate the skin and the underlying muscles of the arms and legs. For example, the cervical plexus innervates the diaphragm through the phrenic nerve; the brachial plexus innervates the upper extremities through the median, ulnar and radial nerves; and the lumbar plexus innervates the anterior thigh through the femoral nerve.

An area of skin innervated by cutaneous branches of a single spinal nerve is called a dermatome. The dorsal roots of the spinal nerves carry sensations from these specific dermatomes. Dermatomes provide anatomical landmarks that are useful for locating neurological lesions (see Figure 40.9).

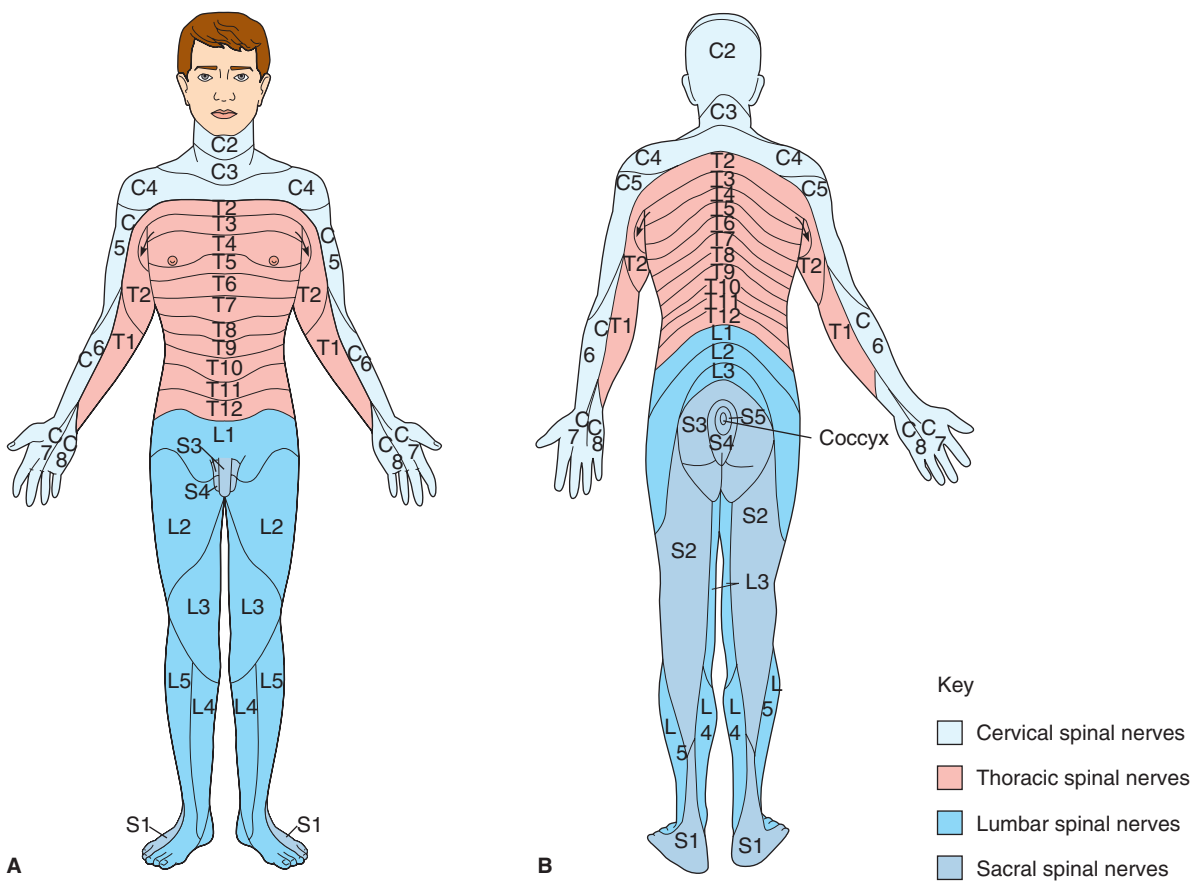


FIGURE 40.9 ■ *A*, Anterior and *B*, posterior dermatomes of the body

Cranial nerves

Twelve pairs of cranial nerves originate in the forebrain and brainstem (see Figure 40.10). The vagus nerve extends into the ventral body cavity, but the 11 other pairs innervate only head and neck regions. Although most are mixed nerves, three pairs (olfactory, optic and acoustic) are solely sensory. The cranial nerves and their related functions are listed in Table 40.4.

Reflexes

A reflex is a rapid, involuntary, predictable motor response to a stimulus. Reflexes are categorised as either somatic or autonomic. Somatic reflexes result in skeletal muscle contraction. Autonomic reflexes activate cardiac muscle, smooth muscle and glands. A reflex occurs over a pathway called a reflex arc.

The essential components of a reflex arc are a receptor, a sensory neuron to carry afferent impulses to the CNS, an integration centre in the spinal cord or brain, a motor neuron to carry efferent impulses and an effector (the tissue that responds by contracting or secreting) (see Figure 40.11).

Somatic reflexes mediated by the spinal cord are called *spinal reflexes*. Many spinal reflexes occur without impulses travelling to and from the brain, with the cord serving as the integration centre, whereas others require brain activity and modulation. *Deep tendon reflexes (DTRs)* occur in response to muscle contraction and cause muscle relaxation and lengthening. DTRs depend on intact sensory and motor nerve roots, functional synapses in the spinal cord, a functional neuromuscular junction and a competent muscle. Thus, an abnormal DTR could indicate a variety of health problems, including a lesion of a spinal nerve. Flexor, or withdrawal, reflexes are caused by actual or perceived painful stimuli and result in withdrawal of the part of the body that is threatened. Superficial responses result from gentle cutaneous stimulation. These responses depend on functional upper motor pathways and on an intact reflex arc.

The autonomic nervous system

The autonomic nervous system (ANS) is a division of the PNS that regulates the internal environment of the body. It is also called the general visceral motor system because it consists of motor neurons that innervate the body's viscera. The skeletal muscle activity and reflexes are regulated by a division of the PNS called the somatic nervous system; the ANS regulates the activity of cardiac muscle, smooth muscle and glands.

The ANS is primarily controlled by the reticular formation in the brainstem. Stimulation of centres in the medulla initiates reflexes that regulate cardiac rate, blood vessel diameter and gastrointestinal function.

The ANS has sympathetic and parasympathetic divisions. Although fibres from both divisions affect the same structures, the actions of the two divisions are opposite in effect and they serve to counterbalance each other. The main neurotransmitters

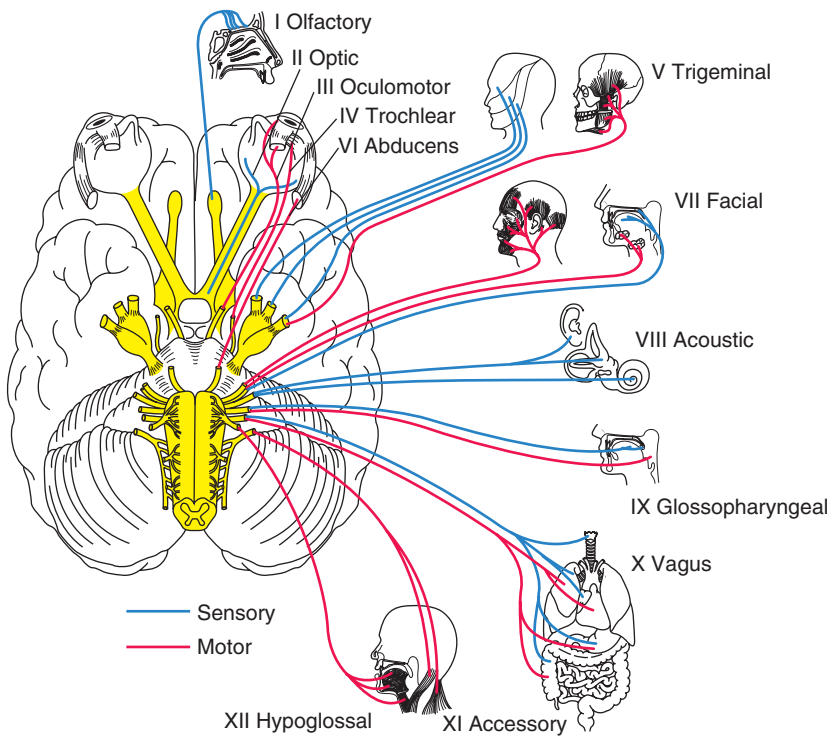


FIGURE 40.10 ■ Cranial nerves

TABLE 40.4 Cranial nerves

NAME	FUNCTION
I Olfactory	Sense of smell
II Optic	Vision
III Oculomotor	Eyeball movement Raising of upper eyelid Constriction of pupil Proprioception
IV Trochlear	Eyeball movement
V Trigeminal	Sensation of the upper scalp, upper eyelid, nose, nasal cavity, cornea and lacrimal gland Sensation of the palate, upper teeth, cheek, top lip, lower eyelid and scalp Sensation of the tongue, lower teeth, chin and temporal scalp Chewing
VI Abducens	Lateral movement of the eyeball
VII Facial	Movement of facial muscles Secretions of lacrimal, nasal, submandibular and sublingual glands Sensation of taste
VIII Acoustic	Sense of equilibrium Sense of hearing
IX Glossopharyngeal	Swallowing Gag reflex Secretions of parotid salivary gland Sense of taste Touch, pressure and pain from pharynx and posterior tongue Pressure from carotid arteries Receptors to regulate blood pressure
X Vagus	Swallowing Regulation of cardiac rate Regulation of respirations Digestion Sensation from thoracic and abdominal organs Proprioception Sense of taste
XI Accessory	Movement of head and neck Proprioception
XII Hypoglossal	Movement of tongue for speech and swallowing

for impulse transmission in the ANS are acetylcholine and noradrenaline. Acetylcholine is the primary neurotransmitter of the parasympathetic division. Noradrenaline is the primary neurotransmitter of the sympathetic division.

Sympathetic division

The sympathetic division of the ANS prepares the body to handle situations that are perceived as harmful or stressful and to participate in strenuous activity. Cell bodies for this division arise in the lateral horns of the spinal cord in the area from

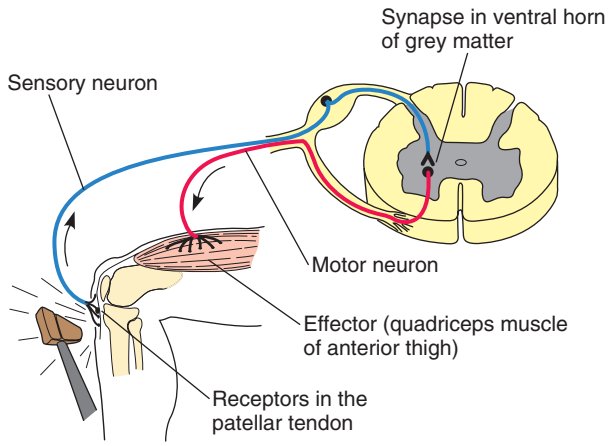


FIGURE 40.11 ■ A typical reflex arc of a spinal nerve. In the two-neuron reflex arc, the stimulus is transferred from the sensory neuron directly to the motor neuron at the point of synapse in the spinal cord

T1 to L2. The fibres separate after leaving the cord and form a chain of ganglia that extends from the neck to the pelvis. Long fibres then extend to the organs that are supplied by the sympathetic division. Stimulation of the sympathetic division can exert the following effects on target organs or tissues:

- dilated pupils
- inhibited secretions
- copious production of sweat (**diaphoresis**)
- increased rate and force of heartbeat
- vasodilation of the coronary arteries
- dilation of the bronchioles
- decreased digestion
- increased release of glucose by the liver
- decreased urine output
- vasoconstriction of arteries
- vasoconstriction of abdominal and skin blood vessels
- increased blood clotting
- increased metabolic rate
- increased mental alertness.

Parasympathetic division

The parasympathetic division of the ANS operates during non-stressful situations. Cell bodies for this division are located in the brainstem (for the cranial nerves) and in the lateral grey matter of S2 to S4. Other than the fibres supplying the cranial nerves III, VII, IX and X, the fibres are carried by the vagus nerve to body tissues, thoracic organs and visceral organs. Stimulation of the parasympathetic division of the ANS produces the following effects:

- constriction of pupils
- stimulation of glandular secretions
- decreased heart rate
- vasoconstriction of coronary arteries
- constriction of the bronchioles
- increased peristalsis and secretion of gastrointestinal fluid.

ASSESSING NEUROLOGICAL FUNCTION

Structures and functions of the neurological system are assessed by findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests. Sample documentation of an assessment of the neurological system is included in the box below.

Health assessment interview

A health assessment to determine problems with neurological structure and/or function may be conducted during a health screening, may focus on a chief complaint (such as headaches) or may be part of a total health assessment. Health problems affecting the neurological system may manifest as problems with musculoskeletal function and an assessment of both systems may be necessary. If the person's level of consciousness is altered, the nurse may need to rely on family members for information. The person's level of consciousness may be assessed by using the Glasgow Coma Scale (see Table 40.5).

If the person has problems with neurological structure or function, analyse its onset, characteristics, course, severity, precipitating and relieving factors, and any associated symptoms, noting the time and circumstances. For example, ask the person the following:

- Describe the location and intensity of the pain you have experienced in your left leg. Is it made worse by coughing, sneezing or walking?
- When did you first notice that you were having numbness in your fingers?
- Describe the difficulty you have when you try to walk.

Questions about present health status include information about numbness, tingling sensations, tremors, problems with coordination or balance, or loss of movement in any part of the body. Ask the person about difficulty with speaking, seeing, hearing, tasting or detecting odours. In addition, elicit information about memory, feeling state (such as anxiety or depression), recent changes in sleep patterns, ability to perform self-care and activities of daily living, sexual activity and weight. If the person is taking prescribed medications, over-the-counter medications or herbal supplements, ask about the type and purpose, as well as the frequency and duration of use.

Ask about any past history of seizures, fainting, dizziness, headaches and any trauma, tumours or surgery of the brain, spinal cord or nerves. Discuss illnesses that may cause neurological manifestations, including cardiac disease, strokes, pernicious anaemia, sinus infections, liver disease and/or kidney failure. Also ask the person about family history of neurological health problems, diabetes mellitus, hypertension, seizures or mental health problems.

SAMPLE DOCUMENTATION

Assessment of the neurological system

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45-year-old male having annual physical examination. No history of injury or infection involving the head or spine. No history of seizures, dizziness, headaches, memory loss or problems with speaking or swallowing. Alert, oriented to person, place and time. Able to follow directions, explain simple proverbs and compare unlike objects. Cranial nerve testing results:

I = Identifies scents correctly.
II = Vision 40/20 in both eyes. Has full visual fields bilaterally.
III, IV, VI = Bilateral full extraocular movements, pupils are equally round, react to light and accommodation. No ptosis present in either eye.
V = Able to identify sharp, dull and light touch to forehead, cheek and chin. Corneal reflex present bilaterally, but decreased (wears contact lenses).
VII = Able to smile, frown, wrinkle forehead, show teeth, puff out cheek, purse lips, raise eyebrows and close eyes against resistance.
VIII = Heard whispered words with both ears.
IX, X = Gag reflex present. Swallows without difficulty.
XI = Equal strength when shrugging both shoulders.
XII = Able to protrude tongue midline without tremors.

All extremities have full range of motion without tremors, tics or weakness. No atrophy of muscles noted. Can perform repetitive alternating movements without difficulty. Gait is steady. Negative Romberg test. All reflexes are 2+ bilaterally. Abdominal reflex present. No Babinski's present.

D. Sassir
(D SASSIR) RN

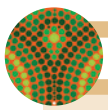
TABLE 40.5 Glasgow Coma Scale

ASSESSMENT	RESPONSE	SCORE*
Eyes open (Record C if eyes are closed by swelling.)	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best motor response (Record best upper arm response.)	Obeys commands	6
	Localises pain	5
	Flexion withdrawal	4
	Abnormal flexion	3
	Abnormal extension	2
	No response	1
Best verbal response (Record T if an endotracheal or tracheostomy tube is in place.)	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Total score:		____

*A higher score indicates a higher level of functioning.

Question the person about occupational hazards, such as exposure to toxic chemicals or materials, use of protective headgear and the amount of time spent performing repetitive motions (e.g. data entry and assembly). Ask questions about self-care to assess the person's diet and use of tobacco, drugs or alcohol, and ask whether they wear a helmet when riding a bike or motorcycle or participating in contact sports, or use a seat belt when driving a vehicle.

Interview questions categorised by functional health patterns are listed in the 'Functional health pattern interview' table below.



FUNCTIONAL HEALTH PATTERN INTERVIEW Neurological system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Have you ever had surgery, injury or illness of the neurological system, such as seizures, stroke, tumour, meningitis? If so, describe the problem and how it was treated. ■ Do you have high blood pressure? If so, how is it treated? ■ Have you ever had problems with the ability to move body parts? Describe. ■ Would you say you think clearly? If not, how and when did the change occur? ■ Are you having any problems with the ability to see, hear, taste or smell? Explain. ■ Have you ever had any diagnostic tests for a neurological problem, such as an MRI or spinal tap? If so, what were the results? ■ Do you take medications for seizures, headaches or other neurological problems? If so, what are they and how often do you take them? ■ Do you now or have you ever smoked, used street drugs or drunk alcohol? If so, what type, how much and for how long? ■ Where were you born and raised as a child?
Nutritional–Metabolic	■ Describe your usual food and fluid intake for a 24-hour period. ■ Have you noticed any problems with chewing or swallowing your food? ■ Do you have trouble with coughing when you eat or drink?
Elimination	■ Has there been any change in your urinary or bowel elimination? If so, describe the change. ■ Do you use laxatives, suppositories or enemas to assist with bowel elimination? If so, what type and how often? ■ Are you able to go to the bathroom without assistance? If not, describe your usual routine.
Activity–Exercise	■ Describe your usual activities in a 24-hour period. ■ Do you have any problems with balance, coordination or walking? Do you use any assistive device when you walk, such as a cane or walker? ■ Have you noticed any weakness in your arms or legs? If so, describe. ■ Are you able to move all of your body parts? If not, explain. ■ Do you trip or fall easily? ■ Have you experienced any shakiness or tremors? Where? ■ If you have seizures, what type do you have? Can you tell when they are going to happen? Does anything specific make you have a seizure? How do you feel after the seizure is over?
Sleep–Rest	■ Does this health problem interfere with your ability to sleep and rest? If so, how? ■ Do you take any medication to help you sleep? If so, what? ■ Describe your energy level. Do rest and sleep restore your energy?
Cognitive–Perceptual	■ Describe any headaches you experience, including frequency, type, location and precipitating/relieving factors. ■ Do you ever feel dizzy or have you fainted? Do you ever feel the room is spinning? Explain. ■ Do you ever experience any numbness, burning or tingling sensations? If so, where and when? ■ Do you have any visual problems, such as double vision, blurring or blind spots? ■ Do you have any problems with hearing? Explain. ■ Has there been any change in your ability to taste or smell? If so, explain. ■ Do you have any difficulty remembering things? If so, describe what you do.
Self-perception–Self-concept	■ How does having this condition make you feel about yourself?
Role–Relationships	■ How has having this condition affected your relationships with others? ■ Has having this condition interfered with your ability to work? Explain. ■ Has anyone in your family had problems with neurological disease? Explain.

(continued)

FUNCTIONAL HEALTH PATTERN INTERVIEW Neurological system (continued)

Sexuality–Reproductive	■ Has this condition interfered with your usual sexual activity?
Coping–Stress–Tolerance	■ Has having this condition created stress for you? If so, does your health problem seem to be more difficult when you are stressed? ■ Have you experienced any kind of stress that makes the condition worse? Explain. ■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments that you would not use to treat this problem?

Physical assessment

Physical assessment begins when the nurse first meets the person and makes an overall evaluation of their mental and physical status. The mental status examination is conducted with both the nurse and the person seated. The rest of the neurological examination may be performed with the person either sitting or standing. A thorough neurological examination is discussed here, but in most instances the nurse will conduct a focused assessment specific to the person's health status.

The neurological system is assessed through inspection, palpation and percussion (with a reflex hammer). When conducting the mental status and cognitive portions of the examination, be aware that fatigue or illness may alter findings. Provide rest periods for the person as needed. When interpreting findings, consider the person's age, educational background and cultural orientation. Normal age-related findings for the older adult are summarised in Table 40.6.

The assessment should take place in a private, comfortable setting. Ask the person to remove outer clothing, shoes and stockings. Provide a gown for the person to wear. It is important to explain that the neurological examination is lengthy and may consist of questions and requests that seem strange to the person. Explain the rationale for each part of the examination.

A brief version of this physical assessment, often referred to as a *neuro check*, may be performed in a shorter time period when a person requires frequent ongoing assessments of neurological status (see Box 40.1).

BOX 40.1 Abbreviated neurological assessment (neuro check)

1. Assess level of consciousness (response to auditory and/or tactile stimulus).
2. Obtain vital signs (BP, P, R).
3. Check pupillary response to light.
4. Assess strength of hand grip and movement of extremities bilaterally.
5. Determine ability to sense touch/pain in extremities.

Diagnostic tests

The results of diagnostic tests of neurological structure and function are used to support the diagnosis of a specific injury or disease, to provide information to identify or modify the appropriate medications or therapy used to treat the disease, and to help nurses monitor the person's responses to treatment and nursing care interventions. Diagnostic tests to assess the structures and functions of the neurological system are described below and summarised in the following bulleted list. More information is included in the discussion of specific injuries or diseases in Chapters 41, 42 and 43.

- Radiological examinations of the skull and spine include standard x-rays, computed tomography (CT) scans, magnetic resonance imaging (MRI), magnetic resonance angiography (MRA) and magnetic resonance spectroscopy (MRS).

TABLE 40.6 Age-related changes in the neurological system

AGE-RELATED CHANGE	SIGNIFICANCE
• ↓ number of brain cells, cerebral blood flow and metabolism • Slower nerve conduction velocity	Delayed response to multiple stimuli and slower reflexes; may need additional time to process and respond to verbal stimuli
• Slower retrieval of information from long-term memory	There is some age-related forgetfulness, which can be improved by using memory aids such as making lists
• Slower response to changes in balance	May contribute to increased risk of falls
• May exhibit less readiness to learn and depend on prior experiences to solve problems • Is more easily distracted and has a decrease in the ability to maintain attention	Learning new skills or knowledge is improved when they are related to previously learned information and when limits are set on times for learning (e.g. no more than 30 minutes at one time).

Findings from these examinations are used to diagnose and evaluate fractures, vertebral displacements, tumours, haemorrhage, aneurysms, cysts, oedema, ischaemia, atrophy, necrosis, seizures, multiple sclerosis, Alzheimer's disease, coma and vascular lesions.

- Both positron emission tomography (PET) and single-photon emission computed tomography (SPECT) are used to assess brain function and blood flow, to differentiate types of dementia, to evaluate the stage of a brain tumour and to identify brain tumours, strokes and seizure disorders.
- Occlusion of the carotid arteries is evaluated by measuring velocity of blood flow with a carotid duplex study; and extracranial blood vessels are evaluated with a transcranial Doppler study.
- Electrical activity of the brain is measured with an electroencephalogram (EEG) to diagnose brain disease and brain death. A magnetoencephalogram (MEG) measures electrical activity of neurons to pinpoint the area of the brain affected by a stroke, brain trauma, brain disorders or seizures.
- Cerebrospinal fluid, removed through a lumbar puncture (LP), is examined in the laboratory to diagnose a variety of brain diseases and infections.
- A myelogram is used to identify lesions of the spinal cord, such as tumours or herniated intervertebral discs.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, assessing for medication use that may affect the outcome of the tests, supporting the person during the examination as necessary, documenting the procedures as appropriate and monitoring the results of the tests.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of the adult. Several neurological diseases that directly affect the nervous system have a genetic component; some are due to mutation of a single gene while others have a more complex method of inheritance (Sobrido et al., 2012). During the health assessment interview, ask about family members with health problems affecting neurological structure or function. In addition, ask about a family history of problems with muscular coordination, Parkinson's disease, narcolepsy, tremor, seizures, Alzheimer's disease or motor neurone disease. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the box below). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

DIAGNOSTIC TESTS The neurological system

NAME OF TEST X-rays of skull and spine

PURPOSE AND DESCRIPTION Standard x-rays of the skull and spine are done to identify fractures, displacement of vertebrae, spinal curves and tissue displacement (as by tumours).

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Computed tomography (CT) scan

PURPOSE AND DESCRIPTION Used to identify intracerebral haemorrhage, tumours, cysts, aneurysms, oedema, ischaemia, atrophy and tissue necrosis. May also be used to evaluate a shift in intracranial contents and differentiate type of stroke. Involves computer-assisted x-rays of several levels of cross-sections of the body part being examined; may be done with or without contrast.

PREPARATION

- Ensure a signed consent form. Check hospital policy on withholding food and fluids. The person is usually on NBM status (except for the medications ordered as part of the test) for 8 hours before the test if it is done in the morning. If the test is done in the afternoon, the person may have a liquid breakfast. Give medications up to 2 hours before test.
- Assess for possible reaction to iodine dye (by asking about allergy to seafood). Document any allergy and inform the physician and radiology department.
- Remove metal hairpins, clips and earrings.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- If applicable, instruct the person not to drink or eat anything before the test except for the ordered medications.
- Inform them that they may be given an intravenous infusion. When the contrast dye is injected, they may feel warm and have a metallic taste in the mouth. The test will last from 30 to 90 minutes and their head will be positioned in a cradle.
- The CT scanner is circular with a round opening. Inform the person that they will be strapped to a special table and the scanner will revolve around the body part to be examined. The scanner will make a clicking noise. The test is painless and someone will always be immediately available during it.

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DIAGNOSTIC TESTS The neurological system (continued)

NAME OF TEST Magnetic resonance imaging (MRI), Functional MRI

PURPOSE AND DESCRIPTION An MRI is done to identify and monitor conditions of the brain and spinal cord, including stroke, tumours, trauma, seizures and multiple sclerosis. It uses magnetic energy to provide images. Gadolinium contrast media may be used to enhance visualisation. A functional MRI is done to evaluate metabolic or blood flow responses of the brain to specific tasks, such as activity and rest.

RELATED NURSING CARE Assess for metal implants (such as a pacemaker or defibrillator), body piercings and shrapnel, which would contraindicate tests.

NAME OF TEST Magnetic resonance angiography (MRA)

PURPOSE AND DESCRIPTION Can provide information about the blood vessels of the brain and identify vascular lesions. The MRA uses the signals from blood vessels to reconstruct only those vessels with blood flow. It can also be done using contrast media.

RELATED NURSING CARE Assess for metal implants (such as a pacemaker) and shrapnel, which would contraindicate tests.

NAME OF TEST Magnetic resonance spectroscopy (MRS)

PURPOSE AND DESCRIPTION Uses a scanner to confirm the presence of Alzheimer's disease, determine the extent of head injury from trauma or stroke, and identify the causes of coma.

RELATED NURSING CARE Assess for metal implants, as for an MRI, which would contraindicate tests.

NAME OF TEST Positron emission tomography (PET), Single-photon emission computed tomography (SPECT)

PURPOSE AND DESCRIPTION When used to study the brain, a PET can assess normal brain function and cerebral blood flow and volume; can differentiate different types of dementia; and can identify stages of brain tumours. A substance containing a radionuclide is given by gas or by injection, and cross-sections of tissue are detected and displayed by computer. A SPECT is similar to a PET, but uses different substances. It can be used to diagnose strokes, brain tumours and seizure disorders.

RELATED NURSING CARE Tell the person not to drink coffee or alcohol or to smoke for 24 hours before the test. Assess glucose levels pre test. Post test, encourage oral fluids to facilitate excretion of the radioactive substance.

NAME OF TEST Cerebral angiogram

PURPOSE AND DESCRIPTION The cerebral angiogram is the definitive diagnostic procedure for aneurysms, arteriovenous malformations, blood vessel patency and stenosis, thrombosis, vasospasm and space-occupying lesions (such as tumours or haematomas). May be performed either as part of a surgical procedure or with local anaesthesia. In radiology, a contrast medium is injected and films are taken at various time intervals.

RELATED NURSING CARE Obtain informed consent, inform about need to remain NBM for 8 hours prior to the procedure and explain that a burning sensation may be felt for a few (4 to 6) seconds behind the eyes or in the jaw, teeth, tongue or lips. Bed rest is maintained for 8 hours after the procedure, vital signs are monitored and fluids are forced to clear the contrast medium.

NAME OF TEST Carotid duplex study, Transcranial Doppler study

PURPOSE AND DESCRIPTION A carotid duplex study evaluates the velocity of blood flow through the carotid arteries and identifies occlusive disease. Uses sound waves produced by the blood flow to produce an image. A transcranial Doppler study follows the same procedure, but is used to evaluate intracranial blood vessels.

RELATED NURSING CARE No special preparation is needed.

DIAGNOSTICTESTS The neurological system (continued)

NAME OF TEST Electroencephalogram (EEG), Magnetoencephalogram (MEG)

PURPOSE AND DESCRIPTION An EEG is used to measure the electrical activity of the brain to diagnose brain disease and brain death. Electrodes are applied to the scalp with skin clips and a graphic picture is obtained (similar to an ECG of the heart). An MEG can identify the area of the brain affected by a stroke, brain disorders or trauma, or seizures. MEG detects magnetic fields generated by activity of neurons.

PREPARATION

- Explain the procedure, emphasising the importance of cooperation. Withhold fluids, foods and medications (as prescribed) that may stimulate or depress brain waves. These include anticonvulsants, tranquillisers, depressants and caffeine-containing foods (e.g. coffee, tea, colas and chocolate). Medications are usually withheld for 24 to 48 hours before the test. Help the person to wash their hair before the test.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Inform the person that the test takes about 1 hour. It is painless and will be performed while sitting in a comfortable chair or lying on a stretcher. The electrodes are applied to the scalp with a thick paste.
- During the test, the person will first be asked to breathe in and out deeply for a few minutes. Then, they will close their eyes while a light is flashed on them and, finally, they will lie quietly with the eyes closed. After the test, the nurse will help the person wash the paste out of their hair.

NAME OF TEST Evoked potentials

PURPOSE AND DESCRIPTION Measures nerve conduction along pathways to evaluate evoked potential of muscle contractions. Used to diagnose and evaluate neuromuscular diseases and identify nerve damage. Transcutaneous or percutaneous electrodes are applied to the skin and provide recordings.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Electromyogram (EMG)

PURPOSE AND DESCRIPTION Measures the electrical activity of skeletal muscles at rest and during contraction; useful in diagnosing neuromuscular diseases. Needle electrodes are inserted into skeletal muscle (as on the legs) and electrical activity can be heard, viewed on an oscilloscope and recorded on graph paper. Normally, there is no electrical activity at rest.

RELATED NURSING CARE Tell the person not to drink fluids containing caffeine or to smoke for 3 hours before the test and not to take medications such as muscle relaxants, anticholinergics and cholinergics.

NAME OF TEST Lumbar puncture (LP)

PURPOSE AND DESCRIPTION The lumbar puncture is used to measure CSF pressure and to obtain a sample of CSF to use in diagnosis of multiple sclerosis or increased intracranial pressure from meningitis, subarachnoid haemorrhage, brain tumour, brain abscess, encephalitis and viral infections. A needle is inserted in L3–L4 or L4–L5 and fluid is aspirated.

PREPARATION

- Ensure a signed consent form is provided. (This consent may be obtained as part of the general consent given on admission to the hospital or agency.)
- Ask the person to empty their bladder before the procedure begins. Help the person to assume a lateral recumbent position near the side of the bed. The person should assume the foetal position (knees flexed towards the head, head bent towards the chest), with their hands clasped around their knees.

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Inform the person that a local anaesthetic will be injected into the skin over the area of the needle insertion. This medication may cause a burning sensation. A long, thin needle will then be inserted into the lower back below the level of the spinal cord. Cerebrospinal fluid will be withdrawn. The cerebrospinal fluid pressure is measured with a calibrated tube called a manometer.
- Inform the person that there may be slight pain down one leg during the procedure and that it is important to remain still during the procedure.
- A small dressing will be used to cover the place where the needle was inserted. After the procedure, the person must remain flat in bed for the number of hours prescribed by the doctor. (This ranges from 4 to 24 hours.) The nurses will take their vital signs and look under the small dressing at regular intervals.

(continued)

DIAGNOSTIC TESTS The neurological system (continued)

- The person should drink fluids so that their body can replace the fluid that was withdrawn. If they have a headache or backache, they can take medications for pain. The person should notify their healthcare provider if they notice increased pain or drainage from the area where the procedure was done.

POST-PROCEDURE NURSING CARE

- Take and record vital signs as indicated by organisational standards. Monitor neurological status at least every 4 hours for 24 hours. Monitor the puncture site for leakage of cerebrospinal fluid or haematoma formation.
- Ensure that the person voids within 8 hours of the procedure. Encourage increased intake of fluids (up to 3000 mL in 24 hours).
- Administer analgesics as prescribed for pain.

RELATED NURSING CARE Related nursing care for the person having a lumbar puncture is described below.

NAME OF TEST Myelogram

PURPOSE AND DESCRIPTION Used to identify lesions of the spinal cord, such as tumours or herniated intervertebral disc. A lumbar puncture is done, a contrast medium is injected into the subarachnoid space and x-rays are taken.

PREPARATION

- Ensure a signed informed consent is provided.
- The meal prior to the procedure is usually omitted. The person should be well hydrated.
- Administer enemas or laxatives as ordered to ensure visualisation of lumbar spine. Administer prescribed pre-test medications, such as a sedative or diazepam (Valium).

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Inform the person that they will remain NBM several hours before the test.
- The examination will last about 1 hour. The position used to perform the examination will depend on the doctor. The person may have to lie on their stomach, sit and lean forward, or sit with the knees to the chest. A strap may be used to prevent falls and the table will be tilted during the examination.
- A lumbar puncture ('spinal tap') will be performed to inject the dye. A local anaesthetic will be used where the needle will be inserted. There may be a feeling of pressure during needle insertion. The needle will be inserted below the level of the spinal cord. Tell the physician if you experience pain.
- Inform the person that they must stay in bed with the head of the bed elevated for at least 6 to 12 hours. (The length of time will depend on physician preference and hospital policy.) The nurse will check their blood pressure, pulse and respirations. The nurse will also check their ability to feel and move at least every 4 hours (or more often) after the examination.

POST-PROCEDURE NURSING CARE

- Take and record vital signs and assess neurological status as prescribed (and at least every 4 hours) for 24 hours post examination. Record and report any changes. Assess the site of the lumbar puncture for leakage of cerebrospinal fluid or bleeding every 4 hours. Notify the physician of leakage or bleeding.
- Encourage increased intake of oral fluids to replace that withdrawn during the examination. (This may also help decrease a post-myelogram headache.) Make sure that the person voids within 8 hours after the examination. If policy permits, allow males to stand at the bedside or people of either gender to use the bathroom. Notify the physician if the person has not voided within 8 hours.
- Administer analgesics as prescribed for post examination pain, headache or muscle spasms. Keep the person's head elevated at least 30 degrees (in bed or in a chair) for 12 hours or as ordered.
- Resume diet if there is no nausea or vomiting. Force oral fluids to 2400 to 3000 mL in 24 hours, beginning immediately after the procedure.
- Administer prescribed medications for nausea. Do not give any phenothiazine derivatives for 48 hours (to reduce the possibility of seizures).

GENETIC CONSIDERATIONS Neurological disorders

- In all types of spinocerebellar ataxia, there is degeneration of the spinal cord and cerebellum, resulting in loss of muscular coordination and spasticity.
- One recently confirmed risk factor for Parkinson's disease is a positive family history of the disease. This neurodegenerative disease affects about 40 000 people in Australia, and is manifested by tremor, muscular stiffness, and difficulty with balance and walking (Brain Foundation, 2013).
- Although multiple sclerosis (MS) is not directly inherited, genetic factors may influence a predisposition to MS within families as well as the severity and course of the disease.
- Narcolepsy, a sleep disorder, does have a familial connection.
- Huntington's disease is an inherited degenerative disorder that leads to dementia. In Australia it currently affects 6 or 7 people in every 100 000.
- Friedreich's ataxia is a rare inherited disease that causes a progressive loss of voluntary muscle coordination and enlargement of the heart.
- Essential tremor, as a primary disorder, affects as many as 14 000 people across Australia and New Zealand (Medtronic, 2010). In more than half of cases, essential tremor is inherited as an autosomal dominant trait, meaning that children of an individual with the disease have a 50% chance of also developing the disorder.
- Epilepsy is one of the most common neurological diseases, characterised by abnormal cell firing in the brain that causes recurring seizures. Recent evidence suggests that there may be a genetic predisposition in up to 70% of cases (American Society of Neurological Surgeons, 2015).
- Charcot–Marie–Tooth syndrome is the most common inherited peripheral neuropathy in the world, characterised by a slowly progressive degeneration of the muscles of the foot, lower leg, hand and forearm.
- Alzheimer's disease (AD) is a leading cause of death in adults, increasing in incidence with age and more common in women. AD tends to run in families, with mutations in four genes believed to be responsible for the disease.
- Motor neurone disease (MND) is a neurological disease that causes progressive degeneration of motor neurons in the brain and spinal cord, resulting in paralysis and death. Chromosome abnormalities have been linked to familial MND.
- Although Tay–Sachs disease is most often considered a disease of children, there is a chronic adult form that causes neuron dysfunction and psychosis.

NEUROLOGICAL ASSESSMENTS

Technique/normal findings	Abnormal findings
Mental status	
Assess appearance, including dress, hygiene, grooming, gait and posture. *The person should be appropriately dressed and clean, with normal gait and posture.*	■ Unilateral neglect (inattention to one side of the body) may occur with some strokes. Poor hygiene and grooming may be seen in people with dementing disorders. ■ Abnormal gait and posture may be seen in transient ischaemic attacks (TIAs), strokes and Parkinson's disease.
Assess behaviour, including actions and affect, content and quality of speech and level of consciousness (LOC). Use the Glasgow Coma Scale (see Table 40.5) to document findings. *A score of 15 on the Glasgow Coma Scale indicates the person is alert and oriented.*	■ Emotional swings or changes in personality may be observed in people who have had a stroke. ■ The face appears mask like (very little expressive movement of facial muscles) in people with Parkinson's disease. ■ Apathy is seen in people with dementing disorders. ■ **Aphasia** (defective or absent language function) may occur in TIAs and strokes. Aphasias are seen with damage to the left cerebral cortex. Aphasias are more often seen with strokes of the left hemisphere than the right hemisphere. ■ **Dysphonia** (change in the tone of the voice) is common in strokes. Dysphonia is seen with paralysis of the vocal cords (cranial nerve X). ■ **Dysarthria** (difficulty speaking) is seen with lesions of upper and lower motor neurons, the cerebellum and the extrapyramidal tract. ■ Damage to the brainstem and/or cerebral cortex may alter LOC. ■ Drowsiness and decreased LOC may be associated with brain trauma, infections, TIAs, stroke and brain tumours. ■ Level of consciousness, ranging from confusion to coma, is usually altered with a stroke.

Technique/normal findings	Abnormal findings
Assess cognitive function. Note orientation to time, place and person. Note attention span and recent and remote memory. Ask the person to: ■ repeat five to seven numbers ■ recall three items after 5 minutes ■ recall their address, breakfast or birthday. Assess thought processes (both content and perceptions) by noting responses to questions. Note ability to understand what is said and to express thoughts. Note ability to make logical and safe judgments. *The person should be oriented to time, place and person; demonstrate attention and ability to remember recent and past events; respond appropriately to questions; and be able to make judgments.*	■ Disorientation to time and place may occur in people with stroke of the right cerebral hemisphere. ■ Memory deficits are often seen in people with a stroke. ■ Perceptual deficits may be seen in strokes. These same deficits may occur following brain trauma and in dementing disorders. ■ Impaired cognition is often noted with strokes, cerebral trauma and brain tumours.
Cranial nerves	
Test CN I (olfactory). Note the person's ability to smell scents (e.g. soap, coffee) with each nostril. This test is usually done only if a problem with the ability to smell is reported. *Sense of smell should be equal in both nostrils.*	■ **Anosmia** (an inability to smell) may be seen with lesions of the frontal lobe and may also occur with impaired blood flow to the middle cerebral artery.
Test CN II (optic). Assess vision in each eye with Snellen chart (see Chapter 44 for guidelines). *Based on previous ability to see and use of visual aids, the person should be able to see with both eyes.*	■ Blindness in one eye may be seen with strokes or with TIAs. Impaired vision or blindness in one side of both eyes (homonymous hemianopia) is associated with stroke. ■ Impaired vision may be seen with strokes and brain tumours. ■ Blindness or double vision may be noted with stroke and TIAs.
Test CN III, IV and VI (oculomotor, trochlear and abducens). Assess extraocular movements by asking the person to follow your finger as you write an H in the air (see Chapter 44). Assess PERRL ('pupils equally round and reactive to light') by covering one eye at a time and shining a bright light directly into the uncovered eye (use a penlight or the ophthalmoscope). See Chapter 44 for more detailed assessment guidelines. *Extraocular movements should be present bilaterally and pupils should be equally round and reactive to light.*	■ Nystagmus (involuntary eye movement) may be seen with strokes. ■ Constricted pupils are associated with impaired blood flow from a stroke.
Assess for ptosis (drooping eyelids). *Eyelids should not droop.*	■ Ptosis (also called Horner syndrome) occurs with strokes, myasthenia gravis and palsy of CN III.

Technique/normal findings	Abnormal findings
Test CN V (trigeminal). Assess ability to feel light, dull and sharp sensations on the face. With the person's eyes closed, check whether sensation is the same on both sides of the face. Stroke the cheek with a wisp of cotton for light touch, with a closed safety pin for dull touch and with a tongue depressor for sharp touch. If the sharp point of a safety pin is used to assess sharp touch, be sure to avoid scratching the surface of the skin and discard the pin after it is used. *Ability to feel light, dull and sharp sensations should be intact.*	■ Changes in facial sensations are noted with impaired blood flow to the carotid artery. ■ Decreased sensations to the face and cornea on the same side of the body, as well as numbness of the lip and mouth, occur with strokes. ■ Loss of facial sensation or contraction of the masseter and temporal muscles is seen with lesions of CN V. ■ Severe facial pain is seen with trigeminal neuralgia (tic douloureux).
Assess the corneal reflex by touching the corneal surface with a wisp of cotton. The reflex may be absent or decreased in people who wear contact lenses. *Normally the person blinks.*	■ The corneal reflex may be impaired with lesions of CN V or VII.
Test CN VII (facial). Assess ability to taste sweet, sour and salt on the anterior two-thirds of the tongue by asking the person to stick out their tongue and applying a sweet, sour or salty substance. Assess ability to frown, show teeth, blow out cheeks, raise eyebrows, smile and close eyes tightly. *Ability to taste sweet, sour and salt is intact. Should be able to frown, show teeth, blow out cheeks, raise eyebrows, smile and close eyes tightly. Muscle movement should be equal bilaterally.*	■ Loss of ability to taste may occur with brain tumours or with nerve impairment. ■ Asymmetry or decreased movement of facial muscles is noted with lesions of the upper and lower motor neurons. ■ Paralysis of the lower motor neurons from injury to CN VII results in the inability to close eyes, a flat nasolabial fold, paralysis of lower face and inability to wrinkle forehead. ■ Paralysis of the upper motor neurons from a stroke results in weakness of eyelids and paralysis of lower face. ■ Pain, paralysis and sagging of facial muscles is seen on the affected side in Bell's palsy.
Test CN VIII (acoustic). Assess ability to hear the ticking of a watch and whispered and spoken words (see Chapter 44). *The person should be able to hear with both ears.*	■ Decreased hearing or deafness may occur with strokes and/or tumours of CN VIII.
Test CN IX and X (glossopharyngeal and vagus). If gag reflex is intact, observe the person swallowing a small drink of water. Observe for a symmetrical rise of the soft palate and uvula as the person says 'ah'. Assess gag reflex by touching back of the person's throat with a tongue depressor. Assess ability to taste sweet, sour or salty substances on the posterior third of the tongue (see previous description). *The person should be able to swallow without difficulty, have symmetrical rise of the soft palate, have intact gag reflex and taste appropriately.*	■ Dysphagia (difficulty swallowing) is common with impaired blood flow to the brain. ■ Unilateral loss of the gag reflex occurs with lesions of CN IX and X.

Technique/normal findings	Abnormal findings
Test CN XI (spinal accessory). Assess the person's ability to turn their head and shrug their shoulders against resistance: ask the person to turn their head to one side against the resistance of your hand; ask them to shrug their shoulders while you exert downward pressure. Observe symmetry, strength and size of muscles. *The person should be able to turn their head and shrug their shoulders against resistance.*	■ Muscle weakness is noted with lower motor neuron disease. Contralateral hemiparesis is seen with strokes.
Test CN XII (hypoglossal). Assess the person's ability to stick out their tongue and move it from side to side against resistance of a tongue depressor. *The person should be able to stick out tongue and move it from side to side against resistance.*	■ Atrophy and **fasciculations** (twitches) of the tongue are seen in lower motor neuron disease. The tongue may deviate towards the involved side of the body.
Sensory function	
Assess ability to perceive various sensations. Touch both sides of various parts of the body (the chest, abdomen, arms and legs) with one or more of the following: ■ cotton wisp ■ sharp object ■ dull object ■ vibrating tuning fork placed on bony prominences. *The person can differentiate between soft and sharp and can feel vibrations appropriately.*	■ Decreased sensation of pain occurs with injury to the spinothalamic tract. ■ Decreased vibratory sensations are seen with injuries to the posterior column tract. ■ Transient numbness of face, arm or hand is seen with TIAs. ■ Sensory loss on one side of the body is seen with lesions of higher pathways to the spinal cord. ■ Bilateral sensory loss is seen in polyneuropathy (a disease in which multiple peripheral nerves are affected, such as Guillain–Barré syndrome or diabetes mellitus). Sensations are impaired with strokes, brain tumours and spinal cord trauma or compression.
Assess sense of position (**kinaesthesia**). Move the person's finger or big toe up or down. Ask the person to describe the movement. *The person can accurately describe position of their finger or toe when moved up or down.*	■ Lesions of the posterior column of the spinal cord may affect sense of position.
Assess ability to discriminate fine touch. Ask the person to identify: ■ object in hand, such as a coin or key (tests stereognosis) ■ number written on hand (tests graphaesthesia) (see Figure 40.12) ■ two points of simultaneous pinpricks on the hand (tests two-point discrimination) (see Figure 40.13) ■ where they are being touched (tests localisation) ■ how many sensations are felt when touched simultaneously on both sides of the body (tests extinction). *The person can identify and discriminate fine touch.*	■ Inability to discriminate fine touch (stereognosis, graphaesthesia, two points, point localisation and extinction) may occur with injury to the posterior columns or sensory cortex.

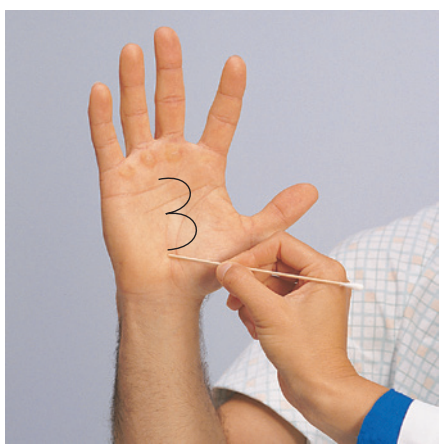


FIGURE 40.12 ■ Testing graphaesthesia

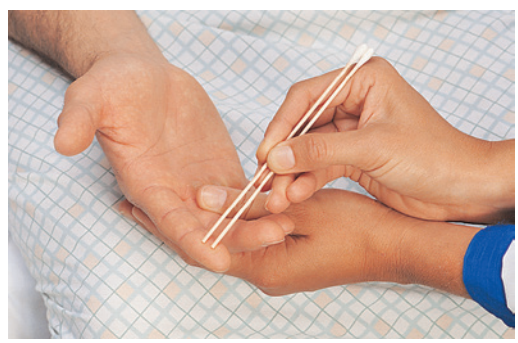


FIGURE 40.13 ■ Testing two-point discrimination

Technique/normal findings	Abnormal findings
Motor function	
Assess bilateral symmetry and size of muscles. Assess for **tremors** (rhythmic movements) and fasciculations (irregular movements). Observe movements as the person is at rest (not making a purposeful movement) and with activity (making a purposeful movement, such as reaching for a glass of water). *Muscles are bilaterally symmetrical and of equal size. Tremors or fasciculations are not present.*	■ Atrophy of muscles is seen with disease of the lower motor neurons. ■ Tremors that occur with activity are seen in multiple sclerosis and diseases of the cerebellar system. ■ Tremors that occur at rest and disappear with movement are common in Parkinson's disease. ■ Fasciculations occur in disease or trauma to the lower motor neurons, as a side effect of medications, in fever, in sodium deficiency and in uraemia.
Assess muscle tone. *Muscle tone is appropriate.*	■ Muscle tone is decreased (**flaccidity**) in disease or trauma of the lower motor neurons and early stroke. ■ Muscle tone is increased (**spasticity**) in disease of the corticospinal motor tract. ■ Muscles are rigid in disease of the extrapyramidal motor tract. ■ Muscles move in small, regular jerky movements (cogwheel rigidity) in Parkinson's disease.
Assess bilateral muscle strength and movement. Ask the person to: ■ squeeze your hands ■ push their feet against the resistance of your hands ■ raise both legs off the bed. See Chapter 37 for a scale to grade muscle strength. *Muscle strength and movement are bilaterally equal and strong.*	■ Weakness of the arms, legs or hands is often seen with TIAs. ■ Hemiplegia (paralysis of one-half of the body vertically) is noted with strokes. ■ Flaccid paralysis is noted with strokes. ■ Paralysis or decreased movement is seen in multiple sclerosis and myasthenia gravis. ■ There is total loss of motor function below the level of injury in complete spinal cord transection and in injuries to the anterior portion of the spinal cord. ■ Spasticity of muscles may occur as a result of incomplete spinal cord injuries.
Cerebellar function	
Assess the gait. Ask the person to walk normally, then in a heel-to-toe fashion, then on toes and finally on heels. *The person has appropriate gait and can walk heel-to-toe, on toes and on heels.* Perform Romberg's test. Ask the person to stand with their feet together and eyes closed. (Stand close to the person to prevent falling.) *There should be minimal swaying for up to 20 seconds.* Assess coordination. Observe ability to pat knees, alternating front and back of hands and increasing speed. Observe ability to touch each finger ofone hand to the thumb. Observe ability to touch the nose, then one of your fingers, then the nose again. Observe ability to run each heel down each shin, while in a supine position (see Figure 40.14). *The person demonstrates coordinated movements.*	■ **Ataxia** is a lack of coordination and a clumsiness of movements, with staggering, wide-based and unbalanced gait. Ataxia is often seen with strokes and cerebellar tumours. Swaying and falling are seen in cerebellar ataxia. Inability to walk on toes, then heels, may indicate disease of the upper motor neurons. ■ Spastic hemiparesis is often associated with strokes or upper motor neuron disease. The person walks with one leg stiffly dragging while the other leg circles out and forwards. One arm is held flexed and close to the side. ■ Steppage gait is noted with disease of the lower motor neurons. The person drags or lifts their foot high, then slaps the foot on to the floor. The person cannot walk on their heels. ■ Sensory ataxia may be associated with polyneuropathy or damage to the posterior columns. The person walks on their heels before bringing down their toes and the feet are held wide apart. Gait worsens with the eyes closed. ■ Parkinsonian gait is often seen in Parkinson's disease. The person stoops over while walking and shuffles their feet. Their arms are held close to the side. ■ A positive Romberg's test may be seen in cerebellar ataxia. ■ Ataxic movements are apparent in cerebellar disease.

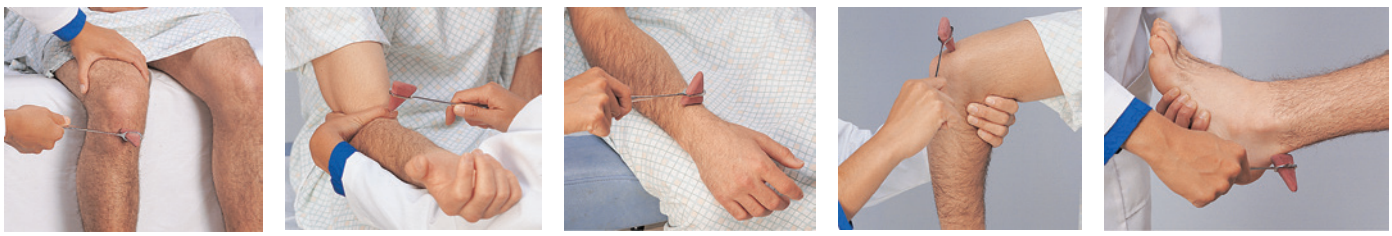
FIGURE 40.14 ■ Heel-to-shin test

REFLEX ASSESSMENTS

A reflex hammer is used to strike the tendon of various reflex sites. To test deep tendon reflexes, ask the person to lock the fingers of both hands together and then pull; this encourages relaxation and promotes reflexes of lower extremities. Superficial reflexes are assessed by lightly stroking the area with the end of a tongue depressor. The following criteria for recording reflexes are often used:

0 = absent or no response
1 = hypoactive; weaker than normal (+)
2 = normal (++)
3 = stronger than normal (+++)
4 = hyperactive, sustained clonus (++++)
A score of 2 is considered normal.

Technique/normal findings	Abnormal findings
Assess the patellar, biceps, brachioradialis, triceps and Achilles deep tendon reflexes (see Figure 40.15).	■ Hyperactive reflexes are present with lesions of upper motor neurons. ■ Decreased reflexes are present with lower motor neuron involvement.



A B C D E

FIGURE 40.15 ■ Deep tendon reflexes *A*, Using reinforcement technique to test the patellar reflex. *B*, Biceps reflex. *C*, Brachioradialis reflex. *D*, Triceps reflex. *E*, Achilles reflex

Assess for clonus by dorsiflexing the person's foot.	■ Clonus, a hyperactive, rhythmic dorsiflexion and plantar flexion, is noted with upper motor neuron disease.
Assess the superficial abdominal and cremasteric reflexes. Abdominal reflex: Lightly stroke the abdomen with a tongue depressor from the side to the midline. Normally the side of the abdomen being stroked will contract towards the umbilicus (see Figure 40.16). Cremasteric reflex: Lightly stroke the inner thigh of the male with a tongue depressor. *Normally, the testicle on the side being stroked will rise.*	■ Superficial reflexes may be absent with disease of the lower and upper motor neurons.

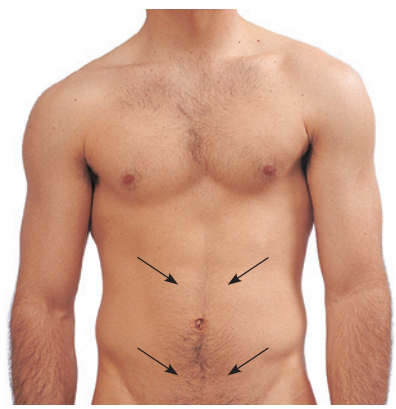


FIGURE 40.16 ■ Location of superficial abdominal reflexes

Assess the Babinski reflex (see Figure 40.17).	■ Dorsiflexion of the big toe and fanning of the other toes is seen with upper motor neuron disease of the pyramidal tract.

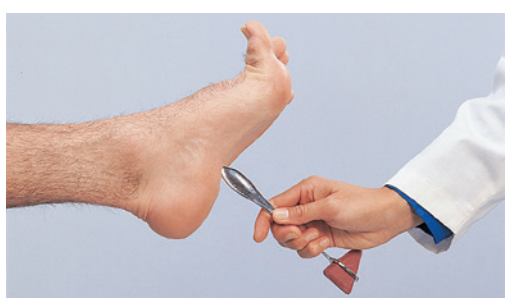


FIGURE 40.17 ■ Assessing the Babinski reflex

Technique/normal findings	Abnormal findings
Special neurological assessments	
Assess for Brudzinski's sign. With the person in the supine position, flex their head to their chest (see Figure 40.18). *There should be no pain, resistance or flexion of the hips or knees.*	■ Pain, resistance and flexion of hips and knees occur with meningeal irritation.
Assess for Kernig's sign. With the person supine, flex their knees and hips, then straighten their knee (see Figure 40.19). *There should be no pain or resistance.*	■ Excessive pain and/or resistance occurs with meningeal irritation.

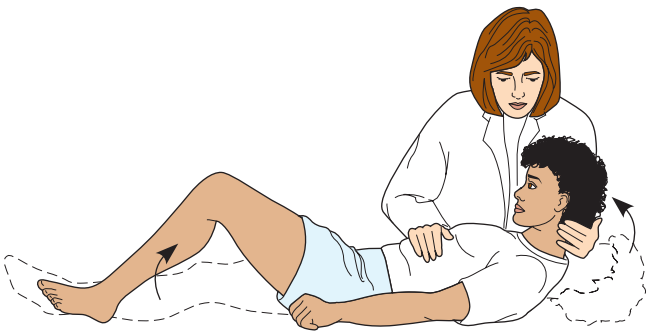


FIGURE 40.18 ■ Assessing for Brudzinski's sign

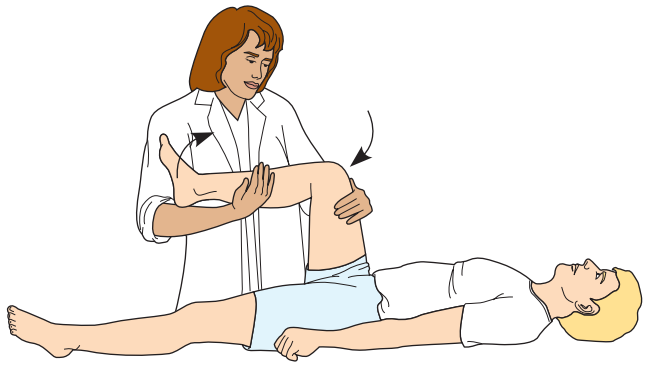


FIGURE 40.19 ■ Assessing for Kernig's sign

Assess for abnormal postures in people who are unconscious. *There should be no abnormal posturing.*

Observe for **decorticate posturing**, in which the upper arms are close to the sides; the elbows, wrists and fingers are flexed; the legs are extended with internal rotation; and the feet are plantar flexed (see Figure 40.20). Decorticate posturing occurs with lesions of the corticospinal tracts.

Observe for **decerebrate posturing**, in which the neck is extended, with the jaw clenched; the arms are pronated, extended and close to the sides; the legs are extended straight out; and the feet are plantar flexed (see Figure 40.21). Decerebrate posturing occurs with lesions of the midbrain, pons or diencephalon.

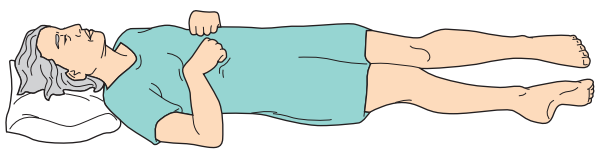


FIGURE 40.20 ■ Decorticate posturing

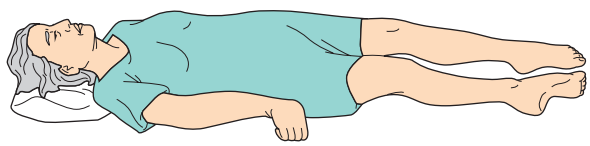


FIGURE 40.21 ■ Decerebrate posturing

CONCEPT CHECK

1 Which component of the brain protects it from harmful substances?
   1 the circulation of cerebrospinal fluid
   2 the large oxygen demand
   3 the structure of neurons
   4 the blood–brain barrier

2 Which pathophysiology results from damage to the lower motor neurons?
   1 loss of cognitive ability
   2 inability to communicate verbally
   3 loss of reflexes
   4 decreasing levels of consciousness

3 Which of the following statements about cerebrospinal fluid (CSF) is true?
   1 If CSF contains glucose, the person has a metabolic disorder.
   2 CSF circulates through the brain via the meninges.
   3 CSF protects the brain and spinal cord from trauma.
   4 A lumbar puncture is done to withdraw CSF from the brain.

4 Following a motorcycle crash, a person has damage to the posterior spinal roots. Which assessment would you expect to find?
1 loss of sensation to dull and sharp
2 flaccid paralysis of the legs
3 changes in peripheral vision in both eyes
4 decreased sense of smell and taste

5 You narrowly miss having a car crash while merging on to the freeway. Your body's responses to this stress are caused by which division of the autonomic nervous system?
1 sympathetic
2 parasympathetic
3 cholinergic
4 adrenergic

6 Which of the physical assessment techniques is not used in the neurological examination?
1 inspection
2 auscultation
3 percussion
4 palpation

7 What would you need to assess function of cranial nerve V (trigeminal)?
1 cotton ball and safety pin
2 stethoscope with bell and diaphragm
3 measuring tape and pencil
4 various scents, such as coffee and vanilla

8 In which of the following cases would assessing the corneal reflex be appropriate?
1 anyone over the age of 50
2 people who wear contact lenses
3 a person with spinal cord trauma
4 an unconscious person

9 You have been asked to assess a person's gag reflex. What equipment would you need to do this?
1 safety pin
2 cotton ball
3 tongue depressor
4 stethoscope

10 Which position best describes decorticate posturing?
1 neck extended, arms extended and pronated, feet plantar flexed
2 arms close to sides, elbows and wrists flexed, legs extended
3 in prone position with arms and knees sharply flexed
4 in supine position, spine extended, legs extended

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CHAPTER 41

NURSING CARE OF PEOPLE WITH INTRACRANIAL DISORDERS

LAURA MELLISH

LEARNING OUTCOMES

- Compare and contrast the pathophysiology, manifestations, interprofessional care and nursing care of people with alterations in level of consciousness and increased intracranial pressure.
- Explain the pathophysiology, manifestations, complications, interprofessional care and nursing care of intracranial disorders, including headaches, epilepsy, traumatic brain injury, central nervous system infections and brain tumours.
- Describe criteria for diagnosing persistent vegetative state and brain death.
- Discuss the purposes, nursing implications and health education for the person and their family in relation to medications used to treat altered cerebral function, headaches, epilepsy, traumatic brain injury, central nervous system infections and brain tumours.
- Discuss surgical options for the treatment of increased intracranial pressure, epilepsy, traumatic brain injury and brain tumours.

CLINICAL COMPETENCIES

- Assess functional status of people with intracranial disorders and monitor, document and report abnormal manifestations.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with intracranial disorders.
- Administer oral and injectable medications used to treat intracranial disorders knowledgeably and safely.
- Provide skilled care to people having intracranial pressure monitoring, tonic–clonic seizures and intracranial surgery.
- Integrate interprofessional care into the care of people with intracranial disorders.
- Provide appropriate teaching and evidence-based practice to facilitate community-based care to promote safety and prevent injury, and to provide information and support necessary for long-term care of people with intracranial disorders.
- Revise the plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to people with intracranial disorders.

KEY TERMS

The person with an intracranial disorder presents a unique nursing challenge. Problems the person experiences in the acute stage of the disorder are often a prelude to long-term problems requiring ongoing management. These long-term problems range from alterations in the body's basic functioning to dysfunctions in the complex processes of the human mind. Systemic problems may accompany or develop secondary to an intracranial disorder. Intracranial disorders may affect both the person's quality of life and that of the person's family. This chapter discusses altered level of consciousness and increased intracranial pressure, followed by discussion of intracranial disorders that may manifest these and other health problems. Information specific to the person with a stroke is provided in Chapter 42.

ALTERED CEREBRAL FUNCTION

The manifestations of altered cerebral function occur as a result of illness or injury. Assessment of the patterns of those manifestations helps determine the extent of the cerebral dysfunction and improvement or deterioration of cerebral function. Except in the case of direct damage to the brainstem and reticular activating system (RAS), brain function deterioration usually follows a predictable progression; that is, a pattern in which higher levels of function are impaired initially, progressing to impairment of more primitive functions. Altered level of consciousness (LOC) and behaviour changes are early manifestations of the deterioration of the function of the cerebral hemispheres. Structures in the midbrain and brainstem are affected sequentially, with characteristic changes in LOC; patterns of respiration, pupillary and oculomotor responses; and motor function. Manifestations of progressive deterioration of cerebral function are outlined in Table 41.1.

THE PERSON WITH ALTERED LEVEL OF CONSCIOUSNESS

Consciousness is a condition in which the person is aware of self and environment and is able to respond appropriately to stimuli. Full consciousness requires both normal rousal and full cognition.

- Rousal, or alertness, depends on the RAS, a diffuse system of neurons in the thalamus and upper brainstem.
- Cognition is a complex process involving all mental activities controlled by the cerebral hemispheres, including thought processes, memory, perception, problem solving and emotion.

These two components of consciousness depend on the normal physiological functions of and connections between the

TABLE 41.1 Progression of deteriorating brain function

LEVEL OF CONSCIOUSNESS	PUPILLARY RESPONSE	OCULOMOTOR RESPONSES	MOTOR RESPONSES	BREATHING
Alert; oriented to time, place and person	Brisk and equal; pupils regular	Eyes move as head turns; caloric testing (ear irrigation) produces nystagmus	Purposeful movement; responds to commands	Regular pattern with normal rate and depth
Responds to verbal stimuli; decreased concentration; agitation, confusion, lethargy; disoriented	Small and reactive	Roving eye movements; doll's eyes positive, with gaze fixed straight ahead; eye deviation away from cold caloric stimulus and towards warm stimulus	Purposeful movement in response to pain stimulus	Yawning, sighing respirations
Requires continuous stimulation to rouse			Decorticate posturing with upper extremity flexion	Cheyne–Stokes respirations with crescendo–decrescendo pattern in rate and depth followed by period of apnoea
Reflexive positioning to pain stimulus	Pupils fixed (non-reactive) in midposition	Caloric testing produces nystagmus	Decerebrate posturing with adduction and rigid extension of upper and lower extremities	Central neurogenic hyperventilation with rapid, regular and deep respirations; apneustic breathing with prolonged inspiration and pauses at full inspiration and following expiration
No response to stimuli	Pupils fixed in midposition	No spontaneous eye movement or nystagmus	Extension of upper extremities with flexion of lower extremities; flaccidity	Cluster or ataxic breathing with irregular pattern and depth of respirations; gasping respirations or apnoea

rousal mechanisms of the reticular formation and the cognitive functions of the cerebral hemispheres. Because rousal and cognition are independent components of consciousness, each can act separately on stimuli. For example, the RAS reacts to the discomfort caused by a full bladder by waking the person in the middle of the night. Once awake, however, the frontal cortex alerts the person that the bladder is full and prompts the person to go to the toilet and empty it.

Conditions that affect either the RAS or the function of the cerebral hemispheres can interfere with the normal level of consciousness. Terms describing LOC are listed and defined in Table 41.2. Nurses should remember that consciousness is a dynamic state: a person may pass from full consciousness to coma within hours or experience a slow diminishment of consciousness that does not become evident for weeks or months. The nurse can help provide effective care for a person with an altered LOC by looking beyond the diagnostic labels of consciousness and accurately assessing the person's behaviour and response to stimuli.

Pathophysiology

Level of consciousness may be altered by processes that affect the rousal functions of the brainstem, the cognitive functions of the cerebral hemispheres, or both. The main causes are: (1) lesions or injuries that affect the cerebral hemispheres directly and widely, or that compress or destroy the neurons of the RAS; and (2) metabolic disorders.

Rousal and cognition

The physiological seat of consciousness, the reticular formation, is a mass of nerve cells and fibres that make up the core of the brainstem, extending from the medulla to the midbrain. The axons of reticular neurons are exceptionally long and branch outwards to cells in the hypothalamus, thalamus, cerebellum and spinal cord. A system of reticular neurons within the RAS passes steady streams of impulses through thalamic relays in order to stimulate the cerebral cortex into wakefulness. The body's sensory tracts interact with RAS neurons; this interrelationship helps control the strength of the RAS's rousing effect on the cerebrum.

Damage to the RAS impairs the person's ability to maintain wakefulness and rousal. Stroke is the most common cause of RAS destruction. Other causes include demyelinating diseases, such as multiple sclerosis, tumours, abscesses and head injury. Function of the RAS may be suppressed by compression of the brainstem, which produces oedema and ischaemia. Pressure and compression of the brainstem may be due to tumours, increased intracranial pressure, haematomas, haemorrhage or aneurysm. Although it is possible to assess LOC or rousal in the person with RAS damage, the impairment in rousal may make it impossible to assess cognitive function.

The function of the brain, especially the cerebral hemispheres, depends on continuous blood flow with unimpeded supplies of oxygen and glucose. Processes that disrupt this flow of blood and nutrients may cause widespread damage to the cerebral hemispheres, impairing rousal and cognition. Bilateral hemispheric lesions (such as global ischaemia) or metabolic disorders (such as hypoglycaemia) are the most common causes of altered LOC related to cerebral dysfunction of the hemispheres. Localised masses, such as a haematoma or cerebral oedema, which displace normal structures and cause direct or indirect pressure on the opposite hemisphere or brainstem, can also affect LOC. The person who has widespread damage to the cerebral hemispheres but an intact RAS has sleep–wake cycles and may rouse in response to stimuli; the person cannot be said to be alert, however, because cognition is impaired.

Both localised neurological processes and systemic disorders can alter LOC. Processes occurring within the brain, which may directly destroy or compress neurological structures, include the following:

- increased intracranial pressure
- stroke
- haematoma
- intracranial haemorrhage
- tumours
- infections
- injury from excitatory amino acids
- demyelinating disorders.

TABLE 41.2 Terms used to describe level of consciousness

TERM	CHARACTERISTICS OF PERSON
Full consciousness	Alert; oriented to time, place and person; comprehends spoken and written words
Confusion	Unable to think rapidly and clearly; easily bewildered, with poor memory and short attention span; misinterprets stimuli; judgment is impaired
Disorientation	Not aware of or not oriented to time, place or person
Drowsy	Lethargic, somnolent; responsive to verbal or tactile stimuli but quickly drifts back to sleep
Stupor	Generally unresponsive; may be briefly roused by vigorous, repeated or painful stimuli; may shrink away from or grab at the source of stimuli
Semicomatose	Does not move spontaneously; unresponsive to stimuli, although vigorous or painful stimuli may result in stirring, moaning or withdrawal from the stimuli, without actual rousal
Coma	Unrousable; will not stir or moan in response to any stimulus; may exhibit non-purposeful response (slight movement) of area stimulated but makes no attempt to withdraw
Deep coma	Completely unrousable and unresponsive to any kind of stimulus, including pain; absence of brainstem reflexes, corneal, papillary and pharyngeal reflexes, and tendon and plantar reflexes

Any systemic condition that affects the delivery of blood, oxygen and glucose to the brain or alters cell membranes may also alter LOC. If cerebral blood flow is impaired or the person becomes hypoxic or hypoglycaemic, cerebral metabolism is impaired and level of consciousness declines rapidly. Severe hypoxia quickly leads to ischaemia. Ischaemia may be focal (e.g. following a stroke) or global (as from cardiac arrest or hypovolaemic shock). Widespread global ischaemia causes almost immediate unconsciousness (Grossman & Porth, 2013). People at particular risk include those with poorly controlled diabetes and those with cardiac or respiratory failure.

Other metabolic alterations that can affect LOC include fluid and electrolyte imbalances, such as hyponatraemia or hyperosmolality and acid–base alterations, such as hypercapnoea (an elevated arterial carbon dioxide level). Accumulated waste products and toxins from liver or kidney failure can affect neuronal and neurotransmitter function, altering LOC. Drugs that depress the central nervous system (e.g. alcohol, analgesics, anaesthetics) suppress metabolic and membrane activities in the RAS and cerebral hemispheres, thereby affecting LOC. Glutamate, the main excitatory neurotransmitter in the brain, may accumulate during prolonged ischaemia, resulting in acute glutamate toxicity and cell death.

Seizure activity, with abnormal electrical discharges from a local area of the brain or from the entire brain, commonly affects LOC. It appears that the spontaneous, disordered discharge of activity that occurs during a seizure exhausts energy metabolites or produces locally toxic molecules, altering LOC for a time after the seizure. Consciousness returns when the metabolic balance of the neurons is restored.

As the impairment of brain function progresses, more stimuli are required to elicit a response from the person. Initially, the person may rouse to verbal stimuli and respond appropriately to questions, remaining oriented to time, place and person. With deterioration of neurological function, the person becomes more difficult to rouse and may become agitated and confused when awakened. Orientation to time is lost initially, followed by orientation to place and then to person. Continuous stimulation or vigorous shaking is required to maintain wakefulness as LOC decreases. Eventually, the person does not respond, even with deep painful stimuli.

Patterns of respirations

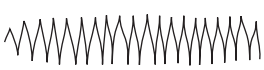
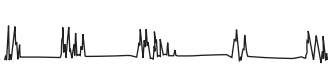
Progressive impairment of neural function also causes predictable changes in respiratory patterns as respiratory centres are affected. In normal respirations, a rhythmic pattern is maintained by neural centres in the pons and medulla that respond to changes in arterial levels of oxygen (PaO_2) and carbon dioxide ($PaCO_2$). When there is damage to the RAS or cerebral hemispheres, neural control of these centres is lost and lower brainstem centres regulate breathing patterns by responding only to changes in $PaCO_2$, resulting in irregular respiratory patterns. The initial manifestations of deteriorating brain function are yawning and sighing. As outlined in Table 41.1, and illustrated in Table 41.3, progressive deterioration in brain function is accompanied by decreasing LOC and changes in breathing patterns. The types of respirations, by area of cerebral damage, are as follows:

- diencephalon: *Cheyne–Stokes respirations* (alternating regular periods of deep, rapid breathing followed by periods of apnoea)
- midbrain: *neurogenic hyperventilation* (may exceed 40 per minute), the result of uninhibited stimulation of the respiratory centres
- pons: *apneustic respirations*, characterised by sighing on mid-inspiration or prolonged inhalation and exhalation; results from excessive stimulation of the respiratory centres
- medulla: *ataxic/apnoeic respirations* (totally uncoordinated and irregular), probably as a result of the loss of responsiveness to CO_2.

Pupillary and oculomotor responses

The brainstem areas that control rousal are adjacent to areas that control the pupils. A predictable progression of pupillary and oculomotor responses occurs as the level of consciousness deteriorates towards coma (see Table 41.1). If the lesion or process affecting neurological function is localised, effects may initially be seen in the ipsilateral pupil (the pupil on the same side as the lesion). With generalised or systemic processes, pupils are affected equally. If the pupils are small and equally reactive, metabolic processes affecting LOC may be present. With compression of cranial nerve III at the midbrain, the

TABLE 41.3 Breathing patterns characteristic of altered level of consciousness

PATTERN		DESCRIPTION
Cheyne–Stokes respirations		A regular crescendo–decrescendo pattern with increasing, then decreasing, rate and depth of respirations followed by a period of apnoea
Central neurogenic hyperventilation		A sustained pattern of rapid, regular, deep respirations (hyperapnoea)
Apneustic breathing		Prolonged inspiration with a pause at full inspiration followed by expiration and a possible pause following expiration
Cluster breathing		Clusters of several breaths with irregular periods of apnoea between clusters
Ataxic respirations		Respirations that are completely irregular in pattern and depth with irregular periods of apnoea

pupils may become oval or eccentric (off centre). As the level of functional impairment progresses, the pupils become fixed (unresponsive to light) and, eventually, dilated.

In deteriorating LOC and coma, spontaneous eye movement is lost and reflexive ocular movements are altered. Normally, both eyes move simultaneously in the same direction; injury to the cranial nerve nuclei in the midbrain and pons can impair normal movement. Doll's eye movements are reflexive movements of the eyes in the opposite direction of head rotation; they are an indicator of brainstem function (see Figure 41.1). As a result of the oculocephalic reflex, the eyes move upwards with passive flexion of the neck and downwards with passive neck extension. As brainstem function deteriorates, this reflex is lost. The eyes fail to turn together and, eventually, remain fixed in the midposition as the head is turned.

Motor responses

The level of brain dysfunction and the side of the brain affected may be assessed by motor responses. These responses are the most accurate identifier of changes in mental status. In altered LOC, motor responses to stimuli range from an appropriate response to a command (e.g. 'squeeze my hand' or 'push my hands away with your feet') to flaccidity (see Table 41.1). Initially, the person may be able to move purposefully away from a noxious stimulus; for example, to brush the examiner's hand away from the face. As function declines, movements become more generalised (withdrawal, grimacing) and less purposeful. Reflexive motor responses may occur, including decorticate posturing with flexion of the upper extremities accompanied by extension of the lower extremities. With further decline, decerebrate posturing is seen, with adduction and rigid extension of the upper and lower extremities. Without intervention, the person eventually becomes flaccid, with little or no motor response to stimuli.

Coma states and brain death

Possible outcomes of altered LOC and coma include full recovery with no long-term residual effects, recovery with residual damage (such as learning deficits, emotional difficulties or impaired judgment) or more severe consequences, such as persistent vegetative state (cerebral death) or brain death. Resources for families are listed in Box 41.1.

PERSISTENT VEGETATIVE STATE **Persistent vegetative state** (also called irreversible coma) is a permanent condition of complete unawareness of self and the environment and loss of all cognitive functions. Usually the result of severe brain trauma or global ischaemia, this condition results from death of the cerebral hemispheres with continued function of the brainstem and cerebellum. While the homeostatic regulatory functions of the brain continue, the ability to respond meaningfully to the environment is lost. The diagnosis of persistent vegetative state requires that the condition has continued for at least 1 month (Grossman & Porth, 2013).

The person has sleep–wake cycles and retains the ability to chew, swallow and cough, but cannot interact with the environment. When awake, the eyes may wander back and forth across the room, but they cannot track an object or person. In a minimally conscious state, the person is aware of the environment and can follow simple commands, manipulate objects, gesture or verbalise to indicate 'yes/no' responses and make meaningful movements (such as blinking or smiling) in response to a stimulus. With appropriate supportive care, the person may remain in this state for years.

LOCKED-IN SYNDROME **Locked-in syndrome** is distinctly different from persistent vegetative state in that the person is alert and fully aware of the environment and has intact cognitive abilities, but is unable to communicate through speech or movement because of blocked efferent pathways from the brain. Motor paralysis affects all voluntary muscles, although the upper cranial nerves (I to IV) may remain intact, allowing the person to communicate through eye movements and blinking. In essence, the person is 'locked' inside a paralysed body while remaining fully conscious of self and environment. Infarction or haemorrhage of the pons that disrupts outgoing nerve tracts but spares the RAS is the usual cause of locked-in syndrome. This condition may also result when the corticospinal tracts between

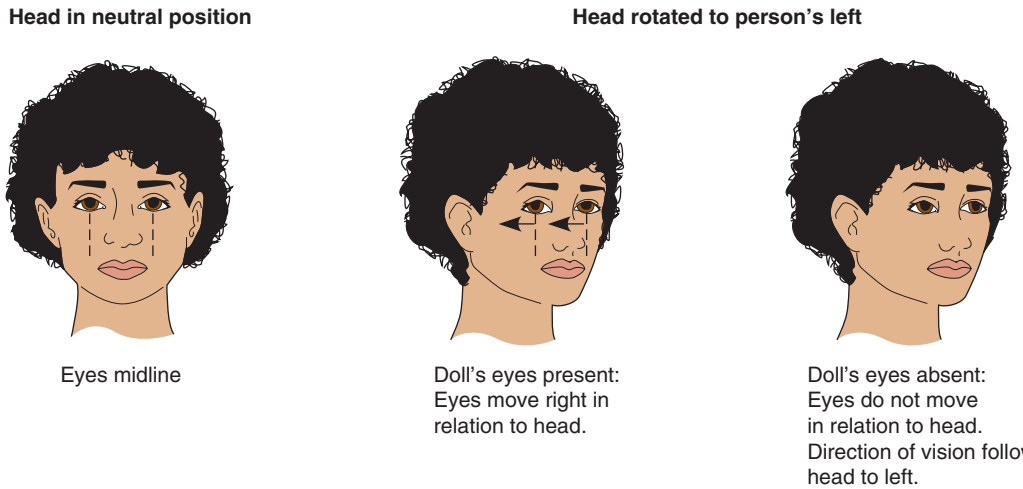


FIGURE 41.1 ■ Doll's eye movements characteristic of altered LOC

BOX 41.1 Organisations providing information for families of people in a coma

Brain Injury Australia
Suite 5, Hodson Building,
Royal Rehabilitation Centre
Sydney
241 Morrison Road
Putney NSW 2112
Ph: 1800 BRAIN1
(1800 272 461)
www.braininjuryaustralia.org.au

Carers Australia
Unit 1, 16 Napier Close
Deakin ACT 2600
Ph: (02) 6122 9900
www.carersaustralia.com.au

Brain Foundation
Suite 21, Regent House
37–43 Alexander Street
Crows Nest NSW 2065
Ph: 1300 886 660 or
(02) 9437 5967
www.brainfoundation.org.au

the midbrain and pons are interrupted. Disorders of the lower motor neurons or muscles, such as acute polyneuritis, myasthenia gravis or motor neurone disease may also paralyse motor responses, leading to locked-in syndrome.

BRAIN DEATH **Brain death** is the cessation and irreversibility of all brain functions, including the brainstem. Although the exact criteria for establishing brain death may vary somewhat from state to state, it is generally agreed that brain death has occurred when there is no evidence of cerebral or brainstem function for an extended period (usually 6 to 24 hours) in a person who has a normal body temperature and is not affected by a depressant drug or alcohol poisoning. Generally recognised criteria are:

- unresponsive coma with absent motor and reflex movements
- no spontaneous respiration (apnoea)
- pupils fixed (unresponsive to light) and dilated
- absent ocular responses to head turning and caloric stimulation (caloric stimulation is performed by irrigating the ear with ice-cold water to test the oculovestibular reflex, a reflex controlled by the brainstem. Normally, the cold causes the eyes to move first towards the irrigated side, followed by a return to midline)
- flat electroencephalogram (EEG) and no cerebral blood circulation present on angiography (if performed)
- persistence of these manifestations for 30 minutes to 1 hour and for 6 hours after onset of coma and apnoea.

Apnoea in the comatose person is determined by the apnoea test. The ventilator is removed while maintaining oxygenation by tracheal cannula and allowing the PCO_2 to increase to 60 mmHg or higher. This level of carbon dioxide is high enough to stimulate respiration if the brainstem is functional. The EEG may be used to establish the absence of brain activity when brain death is suspected. A flat (isoelectric) EEG over a period of 6 to 12 hours in a person who is not hypothermic or under the influence of drugs that depress the central nervous system (CNS) is generally accepted as an indicator of brain death.

Prognosis

The prognosis for people with altered levels of consciousness and coma varies according to the underlying cause and pathological process. Age and general medical condition also play a role in determining outcome. Young adults may fully recover following deep coma from head injury, drug overdose or other cause. Recovery of consciousness within 2 weeks is associated with a favourable outcome. In general, the prognosis is poor for people who lack pupillary reaction or reflex eye movements 6 hours after the onset of coma.

INTERPROFESSIONAL CARE

Management of the person with an altered LOC or coma must begin immediately. The focus of management is to identify the underlying cause, preserve function and prevent deterioration if possible. Airway and breathing must be maintained during the initial acute stage until the diagnosis and prognosis can be established. Intravenous fluids are used to support circulation and to correct fluid, electrolyte and acid–base imbalances. Treatment protocols to reduce increased intracranial pressure or control seizure activity (discussed later in this chapter) may be initiated. Changes in LOC associated with craniocerebral trauma, such as haematomas, often require immediate surgical intervention.

Diagnosis

Although the person's history and physical examination findings often indicate the cause of alterations in LOC, several diagnostic tests may be useful in establishing the diagnosis. The tests used to evaluate for possible metabolic, toxic or drug-induced disorders include both radiological and laboratory tests.

CT and MRI scanning are done to detect neurological damage due to haemorrhage, tumour, cyst, oedema, myocardial infarction or brain atrophy. These tests may also identify displacement of brain structures by large or expanding lesions. Radioisotope brain scan is performed to identify abnormal lesions in the brain and evaluate cerebral blood flow. Cerebral angiography allows radiographic visualisation of the cerebral vascular system. This exam can identify lesions such as aneurysms, occluded vessels or tumours, and may also be used to determine cessation of cerebral blood flow and brain death. Transcranial Doppler studies use an ultrasound velocity detector that records sound waves reflected from RBCs in blood vessels to assess cerebral blood flow. Lumbar puncture with cerebrospinal fluid (CSF) analysis is performed when infection and possible meningitis are suspected as a cause of altered LOC. EEG is used to evaluate the electrical activity of the brain. (See Chapter 40 for further information and nursing implications of neurological tests.)

Laboratory tests are used to identify and monitor altered LOC. These may include any or all of the following:

- *Blood glucose* is measured immediately when coma is of unknown origin and hypoglycaemia is suspected or possible. When the blood glucose falls to less than 2.2 to 2.7 mmol/L, cerebral function declines rapidly. The person with type 1 diabetes is at particular risk of hypoglycaemia-induced coma.
- *Serum electrolytes*—sodium, potassium, bicarbonate, chloride and calcium, in particular—are measured to assess for metabolic disturbances and to guide intravenous therapy. Hyponatraemia, in which serum sodium levels are below

115 mmol/L (normal level: 135 to 145 mmol/L), is associated with coma and convulsions, especially if it develops rapidly.

- *Serum osmolality* is evaluated. Both hyperosmolar and hypo-osmolar states may be associated with coma. Hyperosmolality (above 320 mOsm/kg H_2O) causes cellular dehydration of brain tissue as fluid is drawn into the vascular system by osmosis. Hypo-osmolality (less than 250 mOsm/kg H_2O), by contrast, leads to cerebral oedema and swelling, impairing consciousness.
- *Arterial blood gases* (ABGs) are drawn to evaluate arterial oxygen and carbon dioxide levels, as well as acid–base balance. Hypoxaemia is a frequent cause of altered LOC; increased levels of carbon dioxide are also toxic to the brain and can induce coma, particularly when the onset of hypercapnoea is acute.
- *Liver function tests*, including bilirubin, aspartate transaminase (AST), alanine transaminase (ALT), lactate dehydrogenase (LDH), serum albumin and serum ammonia levels, are determined to evaluate hepatic function. High ammonia levels seen in hepatic failure interfere with cerebral metabolism and neurotransmitters, affecting LOC.
- Toxicology screening of blood and urine is done to determine if altered LOC is the result of acute drug or alcohol toxicity. Serum alcohol levels are measured and the blood is assessed for the presence of substances such as barbiturates, carbon monoxide or lead.

Medications

Medications are used to support homeostasis and normal function for the person with altered LOC, as well as to treat specific underlying disorders. An intravenous catheter is inserted and fluid balance is maintained using isotonic or slightly hypertonic solutions, such as normal saline or Hartmann's solution. The person's response to fluid administration is monitored carefully for evidence of increased cerebral oedema.

If hypoglycaemia is present, 50% glucose is administered intravenously to restore cerebral metabolism rapidly. Conversely, insulin is administered to the person with hyperglycaemia to reduce the blood glucose level and thus the serum osmolality. With narcotic overdose, naloxone is administered. Naloxone is a narcotic antagonist that competes for narcotic receptor sites, effectively blocking the depressant effect of the narcotic. Thiamine may be administered with glucose, particularly if the person is malnourished or known to abuse alcohol, to prevent exacerbation of Wernicke's encephalopathy, a haemorrhagic encephalopathy due to thiamine deficiency that is associated with chronic alcoholism (Papadakis, McPhee & Rabow, 2015).

Any underlying fluid and electrolyte imbalance is corrected by administering medications or appropriate electrolytes. For the person who is hyponatraemic and has a low serum osmolality, frusemide (Lasix) or an osmotic diuretic such as mannitol may be administered to promote water excretion. Appropriate antibiotics are administered intravenously to the person with suspected or confirmed meningitis.

Surgery

Although surgery is not indicated for most people with altered LOC, it may be necessary if the cause of coma is an intracerebral tumour, haemorrhage or haematoma. Surgical intervention is discussed later in this chapter, in the section on brain tumours. When there is a risk of increased intracranial pressure, the person is monitored continuously. These measures are discussed in the section on increased intracranial pressure that follows.

Other treatments

Support of the airway and respirations is vital in the person with an altered LOC. The person who is drowsy but rousable may need little more than an oral pharyngeal airway. With more severe alterations in consciousness, the person may need endotracheal intubation to maintain airway patency, particularly if the cough and gag reflexes are absent. Mechanical ventilation is indicated when hypoventilation or apnoea is present. Unless a not-for-resuscitation (NFR) order is in effect, mechanical ventilation should be initiated even if it has not been established that the disorder is reversible; without ventilatory support, cerebral anoxia develops rapidly and brain death may ensue. ABGs are monitored frequently to determine the adequacy of ventilation. Cautious hyperventilation may be used to reduce $PaCO_2$ and promote cerebral vasoconstriction to reduce cerebral oedema.

Nutrition

In people with long-term alterations in consciousness, such as vegetative state or locked-in syndrome, measures to maintain nutritional status are initiated. Enteral feedings with a gastrostomy tube are preferred if the person is unable to take enough food by mouth without aspirating. In some cases, total parenteral nutrition (TPN) may be used.

Nursing care

Nursing care of the person with an altered LOC is planned and implemented for a variety of responses of both the person and their family.

Support of the family

Family members of a person with an altered level of consciousness are often very anxious. It is difficult for the family to deal with the person's uncertain prognosis. They may experience various conflicting emotions, such as guilt and anger. Reinforce information provided by the doctor and encourage the family to talk to the person as though they are able to understand. Explain that this communication may initially seem awkward, but in time it will feel appropriate. Evaluate the family's readiness to receive explanations regarding the person's treatment and care. The presence of many tubes (e.g. intravenous line, catheter, ventilator) may be overwhelming to the family. They may not perceive the seriousness of the situation if a thorough explanation is not given. Include family members in the person's care as much as they wish to be involved.

Allow significant others to stay with the person when possible. Reinforce the need for family members to care for themselves by encouraging adequate meals and rest. Offer to contact support services such as friends, neighbours and social services that the hospital may provide. Ask family members to leave a telephone number where they can be reached and assure them that they will be called if any significant changes occur. Encourage family members to call if they have questions or concerns.

Nursing diagnoses and interventions

Nursing diagnoses and interventions discussed in this section are directed towards the unconscious person and focus on problems with airway maintenance, skin integrity, contractures and nutrition.

Ineffective airway clearance

Ineffective airway clearance related to loss of the cough reflex and the inability to expectorate is a major problem for the unconscious person. The cough reflex may be absent or impaired when conditions that produce coma depress the function of the medullary centres.

- Assess ability to clear secretions. Monitor breath sounds, rate and depth of respirations, dyspnoea, pulse oximeter and the presence of cyanosis. *The person's ability to clear secretions serves as the initial assessment base for developing further interventions.*
- In unconscious people or those without an intact cough reflex, maintain an open airway by periodic suctioning, limiting the time of suctioning to 10 to 15 seconds or less. Periodic suctioning may be necessary to clear the airway of mucus, blood or other drainage. *Suctioning for more than 15 seconds in the person with increased intracranial pressure may cause hypercapnia, which in turn vasodilates cerebral vessels, increases cerebral blood volume and increases intracranial pressure.*

CONSIDERATION FOR PRACTICE

If the person has a base-of-skull fracture or CSF draining from the ears or nose, never suction nasally.

- Turn from side to side every 2 hours and maintain a side-lying position with the head of the bed elevated approximately 30 degrees. Do not position the unconscious person on the back. *Turning the person from side to side facilitates respirations, prevents the tongue from obstructing the airway and helps prevent pooling of secretions in one area of the lungs (thus decreasing the risk of pneumonia).*
- If the person has a tracheostomy, provide tracheostomy care every 4 hours and suction when secretions are present (see Chapter 34) *to maintain an open airway.*
- Monitor the results of arterial blood gas analysis and pulse oximetry. Maintain records of trends. *ABGs and pulse oximetry directly measure the oxygen content of blood and are good indicators of the lungs' ability to oxygenate the blood.*

Risk of aspiration

The unconscious person with a depressed or absent gag and swallowing reflex is at high risk of aspiration. Drainage, mucus or blood may obstruct the airway and interfere with oxygenation. Pooling of aspiration secretions in the lungs also increases the risk of pneumonia.

- Assess swallowing and gag reflexes every shift as appropriate to the person's level of consciousness. *Deepening levels of unconsciousness may cause a loss in swallow and gag reflexes.*
- Monitor for and report manifestations of aspiration: crackles and wheezes, dullness to percussion over an area of the lungs, dyspnoea, tachypnoea and cyanosis. *Early recognition facilitates prompt intervention.*
- Provide interventions to prevent aspiration:
  - Maintain NBM status.
  - Place in the side-lying position.
  - Provide oral hygiene and suctioning as needed.

  The side-lying position allows secretions to drain from the mouth rather than into the pharynx. Oral hygiene and suctioning remove secretions that might otherwise be aspirated.

CONSIDERATION FOR PRACTICE

Never give oral food and fluids to people with a decreased LOC because of the risk of aspiration.

Risk of impaired skin integrity

The unconscious person is at risk of impaired skin integrity as a result of immobility and the inability to provide self-care. On average, healthy people change positions during sleep every 11 minutes; the unconscious person often cannot maintain the movement needed to prevent pressure on the skin, especially over bony prominences. As a result, the skin and subcutaneous tissues may become ischaemic and prone to develop pressure injury. Perspiration and incontinence of urine and stool may exacerbate the problem. Nursing interventions are directed at maintaining the integrity not only of the skin but also of the lips and mucous membranes.

- Assess skin every shift, especially over bony prominences, the back of the scalp and around genitals and buttocks. *The large surface area of the skin bears weight and is in constant contact with the surface of the bed. The skin, subcutaneous tissue and muscles, especially those tissues over bony prominences, undergo constant pressure. This impairs normal capillary blood flow, which interferes with the exchange of nutrients and waste products. Tissue ischaemia and necrosis may result and lead to the development of pressure injury.*
- Provide proper positioning. Reposition bed-bound people at least every 2 hours if this is consistent with the overall treatment goals. Keep the head of the bed elevated no higher than 30 degrees unless prescribed differently. Provide special pads and mattresses that distribute weight more evenly (e.g. alternating pressure air mattresses, egg-crate foam cushions, turning frames). Consider requesting/using a special therapeutic bed that automatically turns the person at regular intervals. Lift the person instead of dragging the person across the sheet. *When the head of the bed is elevated above 30 degrees,*

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 8: Preventing and Managing Pressure Injuries.

'The intention of this standard is to prevent patients from developing pressure injuries and effectively managing pressure injuries when they do occur.' (Australian Commission on Safety and Quality in Health Care (ACSQHC), 2012, p. 54).

Implementing this standard is achieved by the establishment of systems that ensure thorough screening for patients at risk and the utilisation of clinically appropriate prevention and management strategies.

Caring for individuals with decreased LOC will result in the need for vigilant pressure area care due to the person's inability to move voluntarily, which increases their risk of developing pressure injuries. Pressure area care of the person with decreased LOC involves regular repositioning as permitted by their clinical condition; the use of pressure-relieving devices such as alternating pressure air mattresses; the correct use of lifting aids such as slide sheets; keeping the person's skin clean and dry; regular inspection of pressure areas to detect deterioration; and the management of pressure injuries according to best practice guidelines.

Source: © Australian Commission on Safety and Quality in Health Care.

the person's torso tends to slide down towards the foot of the bed. Friction and perspiration cause the skin and superficial fascia to remain fixed against the bed linen while the deep fascia and skeleton slide downwards. When a person is pulled rather than lifted, the skin remains fixed to the sheet while the fascia and muscles are pulled upwards. These shearing forces promote tissue breakdown.

- Provide interventions to prevent breakdown of the skin and mucous membranes:
  - Keep bed linen clean, dry and wrinkle free.
  - Provide daily bath with mild soap.
  - Cleanse the skin after urine and faecal soiling with a mild cleansing agent.
  - Provide oral care and lubricate the lips every 2 to 4 hours.
  - Maintain accurate intake and output records.
  - Keep the cornea moist by instilling methyl cellulose solution (0.5–1%) and apply protective eye shields or close the eyelids with adhesive strips if the corneal reflex is absent.

  Keeping linen clean, dry and wrinkle free decreases the risk of injury from the shearing force of bed rest and protects against environmental factors that cause drying. Adequate hydration of the stratum corneum appears to protect the skin against mechanical insult. Preventing dehydration maintains circulation and decreases the concentration of urine, thereby minimising skin irritation in people who are incontinent. Proper eye care prevents corneal abrasion and irritation.

Impaired physical mobility

People who are unconscious are unable to maintain normal musculoskeletal movement and are at high risk of contractures related to decreased movement. Flexor and adductor muscles are stronger than the extensors and abductors, resulting in the rapid development of flexor and adductor contractures without preventive measures. Passive ROM exercises must be performed routinely to maintain muscle tone and function, to prevent additional disability and to help restore impaired motor function.

- Maintain extremities in functional positions by providing proper support devices. Remove support devices every 4 hours for skin care and passive ROM exercises. Provide pillows for the axillary region; rolled washcloths may be placed in elevated hands; use splints to prevent plantar flexion (foot drop). *Pillows in the axillary region help prevent adduction of the shoulder. Rolled washcloths help decrease oedema and flexion contracture of the fingers. Splints are useful in preventing plantar flexion.*
- Collaborate with a physiotherapist to develop and implement passive range-of-motion (ROM) exercises (unless contraindicated, as for the person with increased intracranial pressure) at least four times a day, keeping the following principles in mind:
  - Place one hand above the joint being exercised. The other hand gently moves the joint through its normal range of motion.
  - Move the body part to the point of resistance and stop.

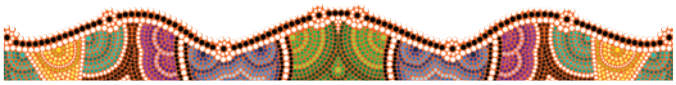
  Placing one hand above the joint provides support against gravity and prevents unwanted movement. ROM exercises help prevent contractures by stretching muscles and tendons and maintaining joint mobility.

Risk of imbalanced nutrition: less than body requirements

The unconscious person is at risk of an alteration in nutrition related to a reduced or complete inability to eat. This is especially true for the person who is unconscious as the result of an infection or trauma, both of which increase metabolic requirements.

- Monitor nutritional status through daily weights (on bed scales) and laboratory data. For accuracy, weigh the person at the same time each day, using the same scales. Ensure that the person wears the same clothing. *Changes in laboratory data with decreased nutrition include a decrease in the levels of serum prealbumin and serum transferrin.*
- Assess the need for alternative methods of nutritional support (tube feeding or total parenteral nutrition) through

collaboration with a dietitian. People unable to take oral food require parenteral nutrition or liquid feedings through a nasogastric, gastrostomy or jejunostomy tube. *Needs for protein, kilojoules, zinc and vitamin C increase during wound healing.*



THE PERSON WITH INCREASED INTRACRANIAL PRESSURE

Increased intracranial pressure (IICP) (also called *intracranial hypertension*) is sustained elevated pressure (10 mmHg or higher) within the cranial cavity (Dorman Wagner, Johnson & Hardin-Pierce, 2013). Transient increases in ICP occur with normal activities such as coughing, sneezing, straining or bending forward. These transient increases are not harmful; however, sustained IICP can result in significant tissue ischaemia and damage to delicate neural tissue. Cerebral oedema is the most frequent cause of sustained increases in ICP. Other causes include head trauma, tumours, abscesses, stroke, inflammation and haemorrhage.

Pathophysiology

In the adult, the rigid cranial cavity created by the skull is normally filled to capacity with three essentially non-compressible elements: the brain (80%), cerebrospinal fluid (8%) and blood (12%). A state of dynamic equilibrium exists; if the volume of any of the three components increases, the volume of the others must decrease to maintain normal pressures within the cranial cavity. This is known as the *Monro–Kellie hypothesis*. The normal intracranial pressure is 5 to 10 mmHg (measured intracranially with a pressure transducer while the person is lying with the head elevated 30 degrees) or 60 to 180 cm H_2O (measured with a water manometer while the person is lying in a lateral recumbent position).

Cerebral blood flow and perfusion are important concepts for understanding the development and effects of IICP. Whereas blood and CSF contribute roughly an equal percentage to normal intracranial volume, vascular factors account for twice the amount of increase in ICP that CSF does. The brain requires a constant supply of oxygen and glucose to meet its metabolic demands; 15–20% of the resting cardiac output goes to the brain to meet its metabolic needs. Interruption of the cerebral blood flow leads to ischaemia and disruption of the cerebral metabolism.

Pressure and chemical autoregulation are compensatory mechanisms in which cerebral arterioles change diameter to maintain cerebral blood flow when ICP increases. In pressure autoregulation, stretch receptors within small blood vessels of the brain cause smooth muscle of the arterioles to contract. Increased arterial pressure stimulates these receptors, leading to vasoconstriction; when arterial pressure is low, stimulation of these receptors decreases, causing relaxation and vasodilation. Chemical, or metabolic, autoregulation works in much the same way as pressure autoregulation. In this case, the stimulus is a build up of metabolic by-products of cell metabolism, including lactic acid, pyruvic acid, carbonic acid and carbon dioxide. Carbon dioxide and increased hydrogen ion concentration are potent cerebral vasodilators that may act locally or systemically to increase cerebral blood flow. Conversely, a fall in $PaCO_2$ causes cerebral vasoconstriction. Arterial oxygen tension (PaO_2) also affects cerebral blood flow, although it is a less powerful mechanism than that exerted by carbon dioxide and hydrogen ions.

IICP may result from an increase in intracranial contents from a space-occupying lesion, hydrocephalus, cerebral oedema (swelling), excess CSF or intracranial haemorrhage. Displacement of some CSF to the spinal subarachnoid space and increased CSF absorption are early compensatory mechanisms. The low-pressure venous system is also compressed and cerebral arteries constrict to reduce blood flow. Brain tissue's ability to accommodate change is relatively restricted. The relationship between the volume of the intracranial components and intracranial pressure is known as *compliance*. When the capacity to compensate for pressure in the cranium is exceeded, increased intracranial pressure (hypertension) develops. Intracranial hypertension is a sustained state of IICP and is potentially life threatening.

Autoregulatory mechanisms have a limited ability to maintain cerebral blood flow. When autoregulation fails, cerebrovascular tone is reduced and cerebral blood flow becomes dependent on changes in blood pressure. Autoregulation may be lost either locally or globally because of several factors, including increasing intracranial pressure, local or diffuse cerebral tissue ischaemia or inflammation, prolonged hypotension and hypercapnoea or hypoxia.

Manifestations

With loss of autoregulation, intracranial pressure continues to rise and cerebral perfusion falls. Cerebral tissue becomes ischaemic and manifestations of cellular hypoxia appear. The manifestations of IICP are listed in the following box.

Level of consciousness

Because the neurons of the cerebral cortex are most sensitive to oxygen deficit, changes in cortical function are the earliest manifestations of increasing ICP (Grossman & Porth, 2013). Behaviour and personality changes occur; the person may become irritable and agitated. Memory and judgment are impaired and speech pattern changes may be noted. The person's LOC decreases. As cerebral hypertension and hypoxia progress, the LOC continues to decrease in a predictable pattern to coma and unresponsiveness.

Motor responses

Pressure on the pyramidal tract often causes weakness (hemiparesis) on the contralateral side early in IICP. As ICP continues to increase, hemiplegia and abnormal motor responses, such as decorticate or decerebrate posturing, develop. (See Chapter 40 for an illustration of these postures.)

Vision and pupils

Altered vision is an early manifestation of IICP; it is caused by pressure on the visual pathways and cranial nerves. Blurred vision, decreased visual acuity and diplopia are common.

MANIFESTATIONS Increased intracranial pressure

- Decreased level of consciousness: early—confusion; restlessness, lethargy; disorientation, first to time, then to place and person; late—comatose with no response to painful stimuli.
- Pupillary dysfunction: sluggish response to light progressing to fixed pupils; with a localised process, pupillary dysfunction is first noted on the ipsilateral side.
- Oculomotor dysfunction: inability to move eye(s) upwards; ptosis (drooping) of the eyelid.
- Visual abnormalities: decreased visual acuity, blurred vision, diplopia.
- Papilloedema: may be a late sign.
- Motor impairment: early—hemiparesis or hemiplegia of the contralateral side; late—abnormal responses such as decorticate or decerebrate positioning; flaccidity.
- Headache: uncommon but may occur with processes that slowly increase ICP; worse on rising in the morning and with position changes.
- Projectile vomiting without nausea.
- Cushing's response: increased systolic blood pressure, widening pulse pressure, bradycardia.
- Respirations: altered respiratory pattern related to level of brain dysfunction.
- Temperature: may be significantly elevated as compensatory mechanisms fail.

Pupillary and oculomotor responses are affected as well. Because the cause of IICP is often localised at first, pupillary changes, including gradual dilation and sluggish response to light, may initially be limited to the ipsilateral side.

Vital signs

Ischaemia of the vasomotor centre in the brainstem triggers the CNS ischaemic response, a late sign of IICP. Neuronal ischaemia in the vasomotor centre causes a marked increase in the mean arterial pressure (MAP), with a significant increase in systolic blood pressure and increased pulse pressure. The increased MAP causes reflexive slowing of the cardiac rate. This trio of manifestations (increased MAP, increased pulse pressure and bradycardia) is known as *Cushing's response* (or triad) and represents the brainstem's final effort to maintain cerebral perfusion (Grossman & Porth, 2013). The respiratory pattern also changes, often in the predictable progression outlined earlier in Table 41.1. Although the temperature is usually normal in early stages, as ICP continues to increase, hypothalamic function is impaired and the temperature may rise dramatically.

Other manifestations

Additional manifestations of IICP include headache, particularly on rising, that worsens with position changes. Headache is more common with slowly developing IICP and occurs because of pressure on pain-sensitive structures, such as the middle meningeal arteries, the venous sinuses and the dura at the base of the skull. Papilloedema (oedema and swelling of the optic disc) may be noted on funduscopic examination. Vomiting, often projectile and occurring without warning, may develop.

Cerebral oedema

Cerebral oedema is an increase in the volume of brain tissue due to abnormal accumulation of fluid. Cerebral oedema is often associated with increased IICP; it may occur as a local process in the area of a tumour or injury, or it may affect the entire brain. Two types of cerebral oedema have been identified and are described as follows (Grossman & Porth, 2013):

1. *Vasogenic oedema*, an increase in the capillary permeability of cerebral vessels, occurs with impairment of the blood–brain barrier, allowing diffusion of water and protein into the interstitial spaces of the brain. A variety of pathologies, such as ischaemia, haemorrhage, brain tumours and injuries and infections (such as meningitis), may cause the increase in capillary permeability. The site of the brain injury, the level of increase in capillary permeability and the person's systemic blood pressure influence the rate and extent of the oedema's spread. Vasogenic oedema is manifested by focal (localised) neurological deficits, altered levels of consciousness and severe intracranial hypertension.
2. *Cytotoxic oedema*, actual swelling of the brain cells from an increase in intracellular fluid, involves changes in the functional or structural integrity of cell membranes due to pathologies such as water intoxication (such as from the syndrome of inappropriate secretion of antidiuretic hormone (SIADH)) or severe ischaemia, intracranial hypoxia, acidosis and brain trauma. With abnormally low cerebral perfusion, oxygen and nutrients are depleted, intracranial cells switch to anaerobic metabolism and the sodium–potassium pump in the cell walls is impaired. Sodium diffuses into the cells, pulling fluid with it. The cells swell and intracranial pressure rises. Accumulated metabolic waste products, such as lactic acid, contribute to a rapid deterioration of cell function. Cytotoxic oedema is a slowly progressive process that results in altered consciousness. The oedema may be so severe that it causes cerebral infarction with brain tissue necrosis.

Cerebral oedema tends to be proportional to the extent of the pathology precipitating it. Brain function is not disrupted by cerebral oedema unless the oedema causes an increase in ICP. When it does, a vicious cycle can ensue: cerebral oedema increases ICP, which in turn decreases cerebral blood flow. Brain tissue becomes hypoxic and ischaemic, increasing toxic metabolic by-products, hydrogen ion concentration and carbon dioxide levels in the tissue. Autoregulatory mechanisms cause vasodilation and increase cerebral blood flow, further increasing cerebral oedema and intracranial pressure. Without effective intervention, the person's condition can deteriorate rapidly; ICP increases to the point where brain structures herniate.

Hydrocephalus

Hydrocephalus refers to a progressive dilation of the ventricular system, which becomes dilated as the production of CSF exceeds its absorption (Hickey, 2013). Hydrocephalus may increase ICP when it develops acutely. It is generally classified as either non-communicating or communicating hydrocephalus. Non-communicating hydrocephalus occurs when CSF

drainage from the ventricular system is obstructed. It may develop when a mass or tumour, inflammation or haemorrhage, or congenital malformation obstructs the ventricular system. Communicating hydrocephalus is a condition in which CSF is not effectively reabsorbed through the arachnoid villi. It may occur secondarily to subarachnoid haemorrhage or scarring from infection. In *normal pressure hydrocephalus*, seen most often in adults aged 60 or older, ventricular enlargement causes cerebral tissue compression but the CSF pressure on lumbar puncture is normal. This condition may follow cerebral trauma or surgery, or the cause may not be known.

Manifestations of hydrocephalus depend on the rate of its development. They may be mild and insidious in onset, presenting as progressive cognitive dysfunctions, gait disruptions and urinary incontinence. If the process causing hydrocephalus is an acute one, the manifestations are those of IICP.

Brain herniation

If IICP is not treated, cerebral tissue is displaced towards a more compliant area. This can result in brain herniation, the displacement of brain tissue from its normal compartment under dural folds of the falx cerebri or through the tentorial notch or incisura of the tentorium cerebelli (Grossman & Porth, 2013). Herniation of the cerebellum through the tentorium exerts pressure on the brainstem, with subsequent herniation through the foramen magnum. This is a lethal complication of IICP because it puts pressure on the vital centres of the medulla.

Brain herniation syndromes are generally categorised as supratentorial or infratentorial, depending on their location above or below the tentorium cerebelli (see Figure 41.2).

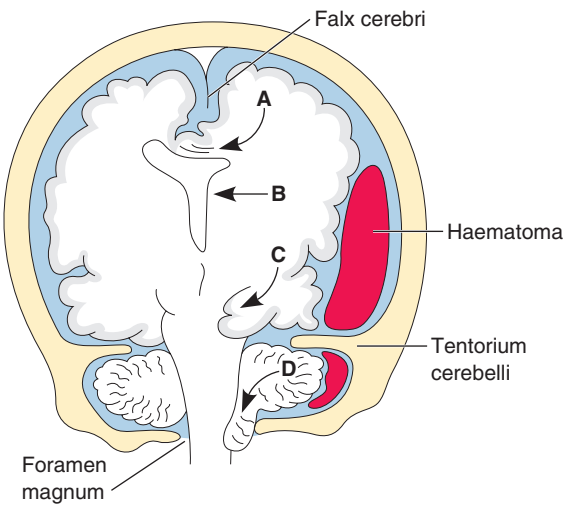


FIGURE 41.2 ■ Forms of brain herniation due to intracranial hypertension. *A*, Cingulate herniation occurs when the cingulate gyrus is compressed under the falx cerebri. *B*, Central herniation occurs when a centrally located lesion compresses central and midbrain structures. *C*, Lateral herniation occurs when a lesion at the side of the brain compresses the uncus or hippocampal gyrus. *D*, Infratentorial herniation occurs when the cerebellar tonsils are forced downwards, compressing the medulla and top of the spinal cord

Supratentorial herniation syndromes include cingulate herniation, central or transtentorial herniation, uncal or lateral transtentorial herniation, and infratentorial herniation.

- *Cingulate herniation* (see Figure 41.2A) occurs when the cingulate gyrus is displaced under the falx cerebri. Local blood supply and cerebral tissue are compressed, resulting in ischaemia and further increases in intracranial pressure.
- *Central or transtentorial herniation* is the downward displacement of brain structures, including the cerebral hemispheres, basal ganglia, diencephalon and midbrain through the tentorial incisura (see Figure 41.2B). The person's neurological signs may deteriorate rapidly, with decreased LOC progressing to coma, Cheyne–Stokes respirations progressing to central neurogenic hyperventilation and pupils progressing from small and reactive to midsized and fixed. The person may demonstrate abnormal motor responses with unilateral decorticate posturing.
- *Uncal or lateral transtentorial herniation* occurs when a lateral mass displaces cerebral tissue centrally, forcing the medial aspect of the temporal lobe under the edge of the tentorial incisura (see Figure 41.2C). The oculomotor nerve (cranial nerve III) often becomes trapped between the uncus and the tentorium, causing ipsilateral pupillary dilation. Other manifestations include alterations in LOC, motor deficits (which may occur on the same side as the herniation because of compression of the cerebral peduncle on the opposite side), decreased sensation, respiratory changes, abnormal positioning and eventual respiratory arrest.
- *Infratentorial herniation* results from increased pressure within the infratentorial compartment. Herniation may occur either upwards, with structures displaced through the tentorial incisura, or downwards, with displacement through the foramen magnum (see Figure 41.2D). Downward displacement compresses the medulla, including its centres for controlling vital functions. Manifestations associated with medullary compression include coma, altered respiratory patterns, fixed pupils and decorticate or decerebrate posturing. Respiratory or cardiac arrest may occur.

INTERPROFESSIONAL CARE

Management of the person with IICP is directed towards identifying and treating the underlying cause of the disorder and controlling ICP to prevent herniation syndrome. IICP is a medical emergency and there is little time to complete lengthy diagnostic tests. The diagnosis must be made on the basis of observation and neurological assessment; even subtle changes may be clinically significant.

Diagnosis

Diagnostic tests focus on identifying the presence of IICP and its underlying cause. A CT scan or MRI is generally the initial test used to identify the possible causes of IICP (such as space-occupying lesions or hydrocephalus) and to evaluate therapeutic options. In general, a lumbar puncture is not performed when IICP is suspected because the sudden release of the pressure in the skull may cause cerebral herniation.

In addition to the diagnostic tests listed in the previous section for altered LOC, the following specific tests are usually ordered and their results closely monitored:

- Serum osmolality is an indicator of hydration status in the person with IICP. The test measures the number of dissolved particles (electrolytes, urea, glucose) in the serum. The normal range for the adult is 280 to 300 mOsm/kg H_2O. In addition to the restriction of fluids in the person with IICP, serum osmolality is maintained at a slightly elevated level (325 mOsm/kg H_2O) to draw excess intracellular fluid into the vascular system.
- ABGs are monitored frequently to assess pH and levels of oxygen and carbon dioxide. Hydrogen ions and carbon dioxide are both potent vasodilators; hypoxaemia also causes vasodilation, although to a lesser degree.

Medications

Medications play an important role in the management of IICP. Diuretics, particularly osmotic diuretics, are commonly used to reduce ICP and are the mainstays of pharmacological treatment. Nursing implications for these medications are described in the 'Medication administration' box below.

MEDICATION ADMINISTRATION — Increased intracranial pressure

Note: Because the person with IICP often has an altered level of consciousness, education of the person and their family is not discussed here.

OSMOTIC DIURETICS

Mannitol (osmitrol)
Urea
Glucose

Osmotic diuretics (hyperosmotic agents) draw fluid out of brain cells by increasing the osmolality of the blood. The effects of these drugs vary with the type of injury. Mannitol therapy is often initiated if the person's ICP exceeds 15 to 20 mmHg for at least 10 minutes. Both intravenous bolus and continuous infusion techniques are used. Repeated use of mannitol can lead to continual elevations in serum osmolality, with attendant risk of seizures and serious fluid and electrolyte imbalance. Urea is seldom administered intravenously because a severe local reaction may result if leakage occurs at the injection site. Mannitol and urea are used cautiously if renal disease is present.

Nursing responsibilities

- Monitor vital signs, urinary output, central venous pressure (CVP) and pulmonary artery pressures (PAP) before and every hour throughout administration.
- Assess person for manifestations of dehydration.
- Assess person for muscle weakness, numbness, tingling, paraesthesia, confusion and excessive thirst.
- Assess person for pulmonary oedema while administering the medication.
- Monitor neurological status and intracranial pressure readings.
- Monitor renal function and serum electrolytes throughout therapy.
- Do not administer the medication if crystals are present in the solution. Administer with an in-line filter. Observe infusion site frequently for infiltration.
- Do not administer mannitol solution with blood.
- Do not discontinue medication abruptly. Rebound migraine headaches may occur.

LOOP DIURETICS

Frusemide (Lasix)
Ethacrynic acid (Edecrin)

Loop diuretics such as frusemide and ethacrynic acid inhibit sodium and chloride reabsorption at the ascending loop of Henle. They cause a reduction in the rate of CSF production, thus reducing the ICP.

Nursing responsibilities

- Monitor vital signs and electrolytes closely.
- Assess fluid status throughout therapy.
- Monitor blood pressure and pulse before and during administration.
- Monitor renal laboratory studies closely.
- Use an infusion pump to ensure accurate dosage.

INTRAVENOUS FLUIDS

Keeping the person moderately dehydrated to maintain serum osmolality can be effective in reducing cerebral oedema. When giving intravenous fluids, closely monitor the osmolality of the solutions; if people with increased ICP are given hypo-osmolar solutions, increased cerebral oedema can occur. Preferred solutions include 0.45–0.9% sodium chloride solutions.

Nursing responsibilities

- Monitor fluid status closely.
- Monitor neurological status closely.
- Avoid administering hypo-osmolar solutions, such as 5% dextrose in water.
- Half-strength normal saline (0.45% sodium chloride) is considered a suitable fluid for a person who has IICP.
- Take care not to restrict fluids excessively in people receiving dehydrating agents (such as osmotic or loop diuretics).

OTHER PHARMACOLOGICAL INTERVENTIONS FOR IICP

- Antipyretics, such as paracetamol, are used to reduce hyperthermia, thereby decreasing the high cerebral metabolism that contributes to IICP.
- Anti-ulcer drugs, such as histamine H_2 antagonists (e.g. ranitidine (Zantac) or sucralfate (Carafate), are used in people with IICP to decrease the development of stress ulcers.
- Antihypertensive agents, such as beta-adrenergic blocking agents, may be used if the mean arterial pressure (MAP) is high.
- Vasopressors may be used if the MAP is low.
- Anticonvulsants may be given to prevent or treat seizures.

Osmotic diuretics work by increasing the osmolarity of the blood, thereby drawing water out of oedematous brain tissue and into the vascular system for elimination via the kidneys. The effects of these drugs vary with the type of injury. Regardless of the agent used, the optimal dose is the lowest that reduces ICP. Mannitol is the most commonly employed osmotic diuretic. Glucose, urea and glycerol are other osmotic diuretics that may be used. Urine output by indwelling catheter is monitored. Electrolyte levels are carefully assessed and potassium is replaced as indicated.

Loop diuretics, such as frusemide (Lasix) (the drug of choice) and ethacrynic acid (Edecrin), may be prescribed for some people with IICP. These diuretics act on the renal tubule and are extremely effective in promoting diuresis. Additionally, loop diuretics may be used to manage the rebound effect that may occur with mannitol administration.

Sedation and paralysis are used as chemical restraints to control restlessness and agitation because these movements increase blood pressure, ICP and cerebral metabolism. Paralysis with neuromuscular blockage is most often accomplished with pancuronium. Close monitoring during treatment for residual muscle weakness and signs of respiratory distress are essential. A peripheral nerve stimulator may be used for this purpose.

Antipyretics, such as paracetamol, are used alone or in combination with a hypothermia blanket to treat hyperthermia. Hyperthermia increases the cerebral metabolic rate and exacerbates an existing increase in ICP. Anticonvulsants are often required to manage seizure activity associated with brain injury and IICP. Gastrointestinal prophylaxis with intravenous histamine H_2 antagonists or proton pump inhibitors are often used, because people with IICP are at increased risk of developing stress gastritis and ulcers (Papadakis et al., 2015).

Intravenous fluids are usually necessary to maintain the person's fluid and electrolyte balance as well as vascular volume. If the person's blood pressure is unstable, vasoactive medications may be administered to maintain the MAP in a range that supports cerebral perfusion while minimising increases in ICP. When enteral feeding is not possible, total parenteral nutrition (TPN) may be administered.

Surgery

People with IICP may undergo various intracranial surgical techniques to treat the underlying cause (see the discussion in the later section on brain tumours). In addition, infarcted or necrotic tissue may be resected to reduce brain mass. A drainage catheter or shunt may be inserted laterally via a burr hole into a ventricle to drain excess CSF and reduce hydrocephalus. The removal of even a small amount of CSF may dramatically reduce IICP and restore cerebral perfusion pressure.

ICP monitoring

Critical to preserving brain function and preventing secondary brain damage from IICP are careful assessments and monitoring with ICP monitors, measuring cerebral blood flow and cerebral perfusion pressure, and measuring oxygen levels of brain tissue. Intracranial pressure monitors facilitate continual assessment of ICP and the effects of medical therapy and nursing interventions on ICP. In addition, cerebral perfusion pressure (the difference between MAP and ICP) can be readily calculated, allowing more precise manipulation of therapeutic measures to maintain cerebral perfusion and thereby prevent ischaemia. The criteria for ICP monitoring depends on the person, but in general, people who are comatose and have a Glasgow Coma Score (described in Chapter 40) of 8 or less should be monitored.

Basic monitoring systems include an intraventricular catheter, subarachnoid bolt or screw, and epidural probe (see Figure 41.3). Intraventricular fluid-filled catheters are placed in the anterior horn of the lateral ventricle (most often in the right side). Ventricular catheters can both drain CSF and measure ICP. The ICP value is measured deep in the brain and is considered the most reflective of the whole brain pressure. Subarachnoid devices are placed in the subarachnoid space. A fibre-optic transducer-tipped catheter can be placed in the epidural, subdural or parenchymal space, with ICP values considered very accurate. Once the intracranial sensor is implanted, it is connected to a transducer that converts the impulses to a signal that the recording device can translate into an oscilloscope tracing, digital value or graphic recording. Factors that increase the risk of infection during ICP monitoring are listed in Table 41.4.

Transcranial blood flow is monitored with transcranial Doppler studies (TCD) to measure the velocity of blood flow

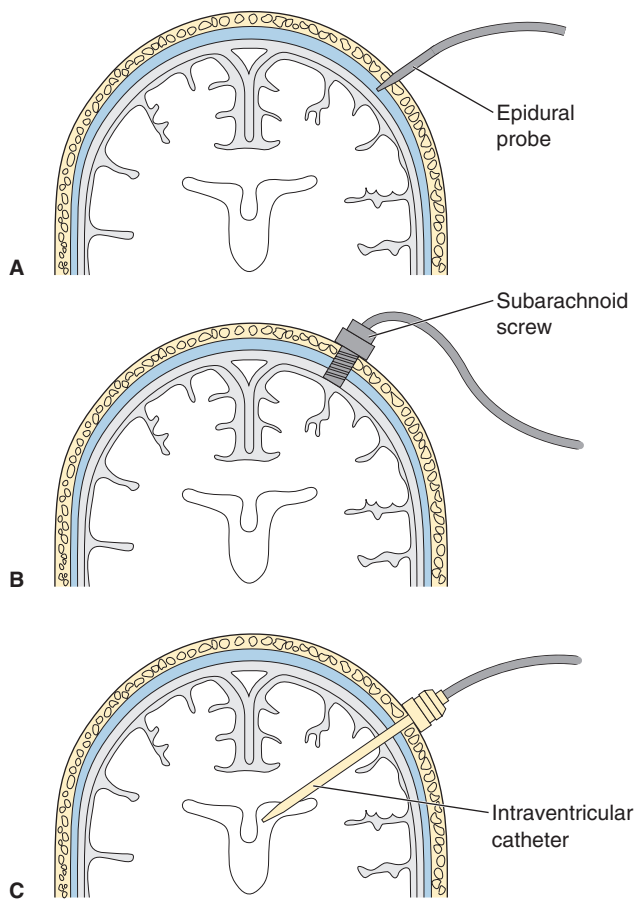


FIGURE 41.3 ■ Types of intracranial pressure monitoring. *A*, Epidural probe. *B*, Subarachnoid screw. *C*, Intraventricular catheter

TABLE 41.4 Risk factors for infection with intracranial pressure monitoring

FACTOR	RATIONALE
Intraventricular catheter	Is more invasive than other monitoring devices
Open head trauma or neurosurgery	Disrupts protective skin and skeletal barriers
Intracranial haemorrhage	Necessitates frequent flushing of catheter to maintain patency
Older adult	Tends to have impaired immune defences
Monitoring for more than 3 to 5 days; or open system or frequent irrigation	Offers increased opportunity for pathogens to enter and grow

in the cerebral vessels. Cerebral perfusion pressure (CPP) is the pressure required for the heart to provide the brain with blood, calculated by subtracting ICP from MAP. (Normal CPP is 70 to 95 mmHg.) Brain oxygenation monitoring may be conducted by using a jugular bulb oxygen saturation (SjO_2) monitor connected to a small fibre-optic catheter inserted into the jugular vein. (Normal SjO_2 is 50–75%.) Another device used to monitor brain tissue oxygenation is the LICOX system, which includes information about ICP, oxygen status and temperature status within the brain tissue itself (Keddie & Rohman, 2012). In addition, cerebral microdialysis catheters can provide information about the nature of the cerebral interstitial fluid.

Mechanical ventilation

People with IICP often require intubation and are placed on a ventilator for respiratory management. Mechanical ventilation may be used to maintain partial pressure of oxygen and carbon dioxide, thus preventing hypoxaemia and hypercapnoea, both of which can increase intracranial pressure. It is important to maintain adequate oxygenation with a partial pressure of arterial oxygen at about 100 mmHg and a partial pressure of arterial carbon dioxide of about 35 mmHg. The person with IICP and signs of impending herniation may be judiciously hyperventilated to cause cerebral vasoconstriction; however, this also increases cerebral ischaemia.

Nursing care

The nursing care of people with IICP involves identifying those at risk and managing factors known to increase intracranial pressure. A major focus is protecting the person from sudden increases in ICP or a decrease in cerebral blood flow.

Nursing diagnoses and interventions

Nursing interventions include performing neurological assessments, maintaining the patency of the airway, ensuring adequate ventilation, positioning and moving, instituting seizure precautions and monitoring fluids and electrolytes. Additionally, both the person and their family need emotional support during this period. The person with IICP has varied responses to actual or potential changes in physiological processes.

Ineffective tissue perfusion: cerebral

A number of disorders may lead to IICP, including cerebral oedema, hydrocephalus, space-occupying lesions and haemorrhage, herniation syndromes and changes in carbon dioxide concentrations. Increasing intracranial pressure alters cerebral perfusion and oxygenation of brain cells. The person with IICP requires intensive care and often needs ventilator assistance.

- Assess for and report manifestations of IICP every 15 minutes to 1 hour and as necessary. Assessment areas include LOC, behaviour, motor/sensory functions, pupillary size and reaction to light, and vital signs, including temperature. Look for trends, because vital signs alone do not correlate well with early deterioration. *Assessment of neurological status establishes the person's clinical condition and provides a baseline for measuring changes. Sudden changes in neurological signs often indicate deterioration. An elevated temperature with increased oxygen consumption further increases intracranial pressure. Pupillary responses mirror the status of the midbrain and pons. Pressure on the brainstem may compromise the function of cranial nerves IX and X and protective mechanisms, such as the gag and cough reflexes.*

CONSIDERATION FOR PRACTICE

Often, the earliest manifestations of a change in intracranial pressure are alterations in the level of consciousness and respirations.

- For the person on a ventilator: maintain patency of the airway; pre-oxygenate with 100% oxygen before suctioning; limit suctioning to 10 seconds; suction gently. *Pre-oxygenation helps maintain oxygen levels during suctioning. Suctioning stimulates the cough reflex and Valsalva manoeuvre. Correct suctioning minimises the risk of hypoxaemia.*
- Monitor ABGs. *ABGs provide a reliable indicator of oxygen and carbon dioxide levels. If oxygen concentration is low, oxygen may be given or increased.*
- Elevate head of the bed to 30 degrees or keep flat, as prescribed; maintain the alignment of the head and neck to avoid hyperextension or exaggerated neck flexion; avoid prone position. *Keeping the head of the bed elevated facilitates venous drainage from the cerebrum. Obstruction of jugular veins can impede venous drainage from the brain.*
- Monitor bladder distension and bowel constipation. Administer stool softeners and use the Credé technique (applying pressure to the suprapubic region with the fingers of one or both hands) to empty the bladder. If the Credé technique is not effective, evaluate the pros and cons of urinary catheterisation if the bladder remains distended.

Constipation and bladder distension increase intrathoracic or intra-abdominal pressure and place the person at risk of impaired venous drainage from the brain.

- If alert, assist the person in moving up in bed. Do not ask to push with heels or arms or push against a footboard. Avoid a footboard and restraints. *Moving up in bed requires pushing. Helping the person move prevents initiation of the Valsalva manoeuvre, which increases intracranial pressure.*
- Plan nursing care so that activities are not clustered together; avoid turning the person, getting the person on the bedpan or suctioning within the same time period. Schedule nursing care to provide rest periods between procedures. *Multiple procedures, including certain nursing care activities, can increase ICP. Constant stimulation tends to increase ICP. Individualised nursing care ensures optimal spacing of activities and rest.*
- Provide a quiet environment, limiting noxious stimuli. Avoid jarring the bed. Try to limit situations that cause emotional upset; maintain a calm, reassuring manner; caution family members to refrain from unpleasant conversations or conversations that may be emotionally stimulating to the person. *Noxious stimuli and emotional upsets cause an elevation in ICP.*
- Maintain fluid limitations, if prescribed. *Restricting fluids helps decrease cerebral oedema by reducing total body water.*

Risk of infection

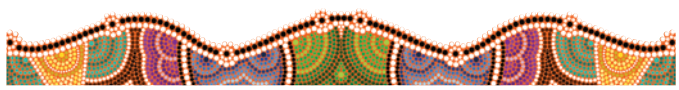
Although any person with an open head wound is at risk of infection, the interventions discussed here are for the person with an intracranial monitoring device. Most clinical units have written protocols for managing these systems. The following nursing actions serve only as a general guide.

- Keep dressings over the catheter dry and change dressings on a prescribed basis (usually every 24 to 48 hours). *Wet dressings are conducive to bacterial growth.*
- Monitor the insertion site for leaking CSF, drainage or infection. Monitor for manifestations of infection, including changes in vital signs, chills, increased WBC counts and positive cultures of drainage. *Close monitoring helps detect the earliest signs of infection and helps prevent major complications. Fever is usually considered the key assessment. However, fever in a person with a neurological disorder may be due to damage to the hypothalamus. Headache, generalised muscle aches, shivering and chills may also be seen in the person with infection.*
- Use strict aseptic technique when in contact with the device. Check drainage system for loose connections. *The use of aseptic technique and monitoring drainage systems for loose connections help prevent hospital-acquired infections. Most such infections are transmitted by healthcare workers who fail to wash their hands properly, to change gloves between people or to follow aseptic technique protocols. Invasive procedures provide an excellent opportunity for microbes to enter the body.*

Health education for the person and family

Teach the person at risk of or having IICP (and able to follow instructions) to avoid coughing, blowing the nose, straining to have a bowel movement, pushing against the bed rails or performing isometric (muscle-contracting) exercises. Advise the person to maintain head and neck alignment when turning in bed and to take rest periods.

Encourage the family to talk to the person, but maintain a quiet environment with a minimum of stimuli. Inform family members that upsetting the person may increase intracranial pressure and that they should avoid discussions that may distress the person. For people unable to make decisions about treatment and to sign informed consent, the family must carry out these functions.



LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 3:

Preventing and Controlling Health Care Associated Infections.

'The intention of this standard is to prevent patients from acquiring preventable healthcare associated infections and effectively manage infections when they occur by using evidence-based strategies.' (ACSQHC, 2012, p. 26)

Implementing this standard is achieved by ensuring adequate strategies that address both prevention and control of healthcare-associated infections are developed and applied to clinical practice. These strategies include the development of appropriate infection-control policies and procedures; adhering to infection-control policies and procedures; surveillance of healthcare-associated infection rates; monitoring of staff compliance with infection-control procedures; training and education for all staff; and education for individuals and their families and caregivers.

Caring for individuals with head injuries requires strict adherence to infection-control policies and procedures, particularly for those individuals with open skull fractures or ICP monitors in place. People with head injuries are also at risk of developing pneumonia and urinary tract infections.

Source: © Australian Commission on Safety and Quality in Health Care.

THE PERSON WITH A HEADACHE

Headache, one of the most frequent manifestations of a health problem people experience, is pain within the cranial vault. Headaches may occur as a result of benign or pathological conditions, intracranial or extracranial conditions, diseases of other body systems, stress, musculoskeletal tension or a combination of these factors.

Most headaches are mild, transient and relieved by a mild analgesic. However, some headaches are chronic, intense and recurrent. Manifestations of headache vary according to the cause, type and precipitating symptoms.

Pathophysiology

The bones and brain tissue itself lack pain-sensitive nerve fibres, but selected structures within the cranial vault are sensitive to pain. Headache is experienced when there is traction, pressure, displacement, inflammation or dilation of nociceptors (nerve endings that are receptors of noxious stimuli) in areas sensitive to pain (Hickey, 2013). Pain-sensitive structures include supporting structures, such as the skin, muscles and periosteum; the nasal cavities and sinuses; portions of the meninges, cranial nerves II, III, IV, V, VI, IX and X; and cerebral vessels, including extracranial arteries and the venous sinuses. Most facial and scalp structures are sensitive to pain. The most common types of headaches are migraine, cluster and tension headaches (see Table 41.5).

Migraine headache

Migraine headache is a recurring vascular headache lasting from 4 to 72 hours, often initiated by a triggering event and usually accompanied by a neurological dysfunction. It affects about 3 million people worldwide, with three times as many women as men having migraines (Headache Australia, 2015). It is more common between the ages of 12 and 40 years with prevalence declining thereafter. Migraine headaches may occur daily or as infrequently as once a year.

There are two types of migraine headaches: common migraine (without an aura) and classic migraine (with an aura; most often experienced as a visual disturbance prior to the pain). Common migraines occur in 80% of the people who are affected by this disorder. Headaches classified as migraines may differ in intensity, duration and frequency. The exact causes of migraine are not fully understood, but they are believed to be the result of abnormalities in cerebrovascular blood flow, a reduction in brain and electrical activity, or increased release of sensory substances such as serotonin, noradrenaline substance P, nitric oxide or glutamate.

A variety of factors are believed to trigger the onset of a migraine headache. Rapid changes in blood glucose levels, stress, emotional excitement, fatigue, hormonal changes due to menstruation, stimuli such as bright lights, and food high in tyramine or other vasoactive substances (e.g. aged cheese, nuts, chocolate and alcoholic beverages) have been associated with migraine attacks. Hypertension and fever may make the disorder worse.

COMMON MIGRAINE This type is the most common and is associated with hereditary factors. The aura stage is absent; people are aware only that a headache is imminent. The headache develops gradually, lasting hours to days, and may occur in women during periods of premenstrual tension and fluid retention. Chills, nausea and vomiting, fatigue and nasal congestion are often present.

TABLE 41.5 Comparison of migraine, cluster and tension headaches

TYPE	RISK FACTORS	FREQUENCY AND DURATION	DESCRIPTION	PRODROMAL AND ASSOCIATED MANIFESTATIONS
Migraine	Female Family history of migraine headache	Episodic: • Tends to occur with stress and crisis • Often correlates with menstrual cycle • Can last hours to days	Slow onset; pain becomes more severe, involving one side of head more than other	Prodromal manifestations: visual defects, confusion, paraesthesias Associated manifestations: nausea, vomiting, chills, fatigue, irritability, sweating
Cluster	Male Use of alcohol or nitrates May begin in early childhood	Episodes are clustered together in rapid succession for a few days or weeks with remissions that last for months Can last a few minutes to a few hours	May begin in infraorbital region and spread to head and neck; throbbing, deep pain, often unilateral	Prodromal manifestations: uncommon Associated manifestations: flushing, tearing of eyes, nasal congestion, sweating and swelling of temporal vessels
Tension	Related to tension and anxiety No family history Often begins in adolescence	Episodic: • Varies with amount of stress • Duration also varies; can be constant	Tight, pressing, vice-like; may involve neck and shoulders	Prodromal manifestations: uncommon Associated manifestations: sustained contraction of neck muscles

CLASSIC MIGRAINE The classic migraine headache has several stages, as follows:

- The aura stage is characterised by sensory manifestations, usually visual disturbances such as bright spots or flashing lights zigzagging across the visual fields. This stage lasts from 5 to 60 minutes. Less common sensory symptoms include numbness or tingling of the face or hand, weakness of an arm or leg, mild aphasia, confusion, drowsiness and lack of coordination. Additionally, some people experience a premonition the day prior to an attack. They may feel nervous or have other mood changes. The aura period corresponds with the initial physiological change of vasoconstriction.
- The headache stage is characterised by vasodilation, a decline in serotonin levels and the onset of throbbing headache. It appears that the pain is related to increased vessel permeability and polypeptide exudation by perivascular nerve endings rather than the vasodilation itself. Cerebral arteries are dilated and distended, with walls that are oedematous and rigid. Beginning unilaterally, the headache eventually may involve both sides as it increases in intensity during the next several hours. Nausea and vomiting often occur. The person may be acutely ill and is often extremely irritable. The sensory organs often become hypersensitive and the person withdraws from sound and light. The scalp is tender. The headache may last from several hours to a day or two.
- During the post-headache phase, the headache area is sensitive to touch and a deep aching is present. The person is exhausted. Vessel size and serotonin levels return to normal.

Cluster headache

A *cluster headache* is an extremely severe, unilateral, burning pain located behind or around the eyes. The cluster headache is predominantly experienced by men between the ages of 20 and 40 years. The headaches occur in groups or 'clusters' of one to eight each day for several weeks or months, followed by remission lasting months to years (Hickey, 2013). The physiological mechanism underlying cluster headaches is not well understood, but involves a vascular disorder, a disturbance of serotonergic mechanisms, a sympathetic defect or deregulation of the hypothalamus.

Although the headache may occur at any time, it typically begins 2 to 3 hours after falling asleep, awakens the person and then lasts from 15 to 180 minutes. Prodromal signs are absent. Intense unilateral pain around or behind one eye is accompanied by rhinorrhoea, lacrimation, flushing, sweating, facial oedema and possible miosis or ptosis on the affected side. The same side of the head is involved in each cluster of attacks.

The headaches often occur in spring and autumn and then disappear for an extended period. Attacks may be triggered by drinking alcohol, eating specific foods, medications such as glyceryl trinitrate (nitroglycerine), or there may be no known precipitating event.

Tension headache

Tension headache is characterised by bilateral pain, with a sensation of a band of tightness or pressure around the head. Sharply localised painful spots (trigger points) may be present. The onset is gradual and the intensity, frequency and duration of the attack vary greatly. This type of headache is caused by sustained contraction of the muscles of the head and neck. It is often precipitated by stressful situations and anxiety. Secondary causes include prolonged computer use and disorders of the eyes, ears, sinuses or cervical vertebrae. Abnormal posture associated with occupations that require bending over a desk (e.g. office workers, students) often precipitates tension-type headache. Additionally, slouching while reading or watching television can lead to muscle contraction. Most headaches are tension-type headaches.

INTERPROFESSIONAL CARE

Identifying the underlying cause(s) of the headache is the initial focus of interprofessional care. If the underlying cause is treatable, the headache will often decrease or disappear. An accurate diagnosis of the type of headache is key to the treatment.

Therapeutic management for migraine headache includes a combination of education, medications and measures to control contributing factors. Dietary changes such as eliminating caffeine, cured meats, monosodium glutamate (MSG) and foods containing tyramine (red wine, aged cheese and others) may be necessary. Stress management or biofeedback is also part of the overall strategy. Treatment protocols for cluster headache include eliminating aggravating factors (e.g. consumption of alcohol) and using medications and oxygen inhalation. The management of tension headaches is directed towards reducing the person's level of stress and relieving pain with ice and aspirin or non-steroidal anti-inflammatory drugs (NSAIDs).

Diagnosis

Diagnosis and treatment are based on history, identifying triggering or precipitating events, and the type of headache. A thorough history and physical examination are integral parts of the assessment. Neurodiagnostic testing may be done to rule out a structural disease process. Testing may include a brain scan, MRI, x-ray studies of the skull and cervical spine, EEG or lumbar puncture for CSF if inflammation is suspected. Serum metabolic screens and hypersensitivity testing also may be performed if systemic problems are suspected.

Medications

Pharmacological management depends on the type of headache. The goals of treatment are to reduce the frequency and severity of headaches and to limit or relieve a headache that is beginning or in progress.

The management of migraine headache includes administering medications to prevent pain (prophylactic therapy) as well as drugs to stop (or abort) a headache in progress. The person with frequent migraine headaches is a candidate for prophylactic therapy. Drugs used to reduce the frequency and severity of migraine include:

- Methysergide maleate (Deseril) is a serotonin antagonist that competitively blocks serotonin receptors in the CNS and is also a potent vasoconstrictor.
- Propranolol hydrochloride (Inderal) is a beta-blocker that prevents dilation of vessels in the pia mater and inhibits serotonin uptake.
- Topiramate (Topamax) and sodium valproate (Epilim) are CNS agents and anticonvulsants.

When the manifestations of migraine are recognised early, several medications may be used to abort or limit the severity and duration of the headache. Ergotamine tartrate (Cafergot) is a complex drug that reduces extracranial blood flow, decreases the amplitude of cranial artery pulsation and decreases basal artery hyperperfusion. Administered at the onset of an attack, ergotamine controls up to 70% of acute attacks. Sumatriptan (Imigran) is available in oral, nasal spray or subcutaneous injection forms. It binds with serotonin receptors and is rapidly effective. Zolmitriptan (Zomig), a selective serotonin receptor agonist, is administered orally and is effective in the treatment of acute headache. Once a migraine is in progress, a narcotic analgesic such as codeine or pethidine may be required. Anti-emetics may be prescribed to control nausea and vomiting.

Many of the same medications used for migraine also prevent or treat cluster headache. Because the onset of cluster headaches is abrupt, abortive therapy is not possible. Medications such as ergotamine tartrate may be given in suppository form at bedtime to prevent headache during the episodic attacks. Inhaling 100% oxygen at 7 L/min for 15 minutes at the onset of an attack may relieve a person's headache (Papadakis et al., 2015).

Non-narcotic analgesics such as aspirin or paracetamol may relieve tension headaches. Additionally, tranquillisers such as diazepam may reduce muscle tension.

Nursing implications for drugs commonly prescribed for headaches are described in the 'Medication administration' box below.

Alternative and complementary therapies

The following alternative and complementary therapies are used to relieve the pain of headaches:

- vitamin D, elemental calcium, riboflavin (vitamin B) and magnesium
- acupuncture
- relaxation, guided imagery, massage
- melatonin, 5-HTP, CoQ10
- magnetic field therapy
- herbal therapy
- osteopathic manipulation.

Nursing care

In addition to the nursing care discussed in this section, a nursing care plan for a person with a migraine headache follows.

Health promotion

Teach people with tension headaches relaxation techniques, such as massage and biofeedback. Counselling for chronic anxiety may also be helpful. Triggers for migraine or cluster headache should be identified and, if possible, eliminated. For example, avoiding physical and emotional stress, having regular and consistent sleep patterns, eating meals regularly, and avoiding specific foods or alcohol can be incorporated into daily life and are helpful. Specific suggestions are outlined in Box 41.2.

BOX 41.2 Suggestions to decrease incidence of migraine headaches

- Wake up at the same time each morning.
- Exercise at least three times a week.
- No smoking or caffeine after 3 pm.
- No artificial sweeteners.
- No MSG.
- Reduce or eliminate red wine, cheese, alcohol, chocolate and caffeine.
- Try a gluten-free diet.

Assessment

Collect the following data through the health history and physical examination.

- *Health history:* history of intracerebral trauma, tumour or infection; detailed history and description of headache characteristics; family history; triggering factors; usual diet; effects of recurring headaches on lifestyle, activities of daily living (ADLs) and role performance.
- *Physical assessment:* skin (diaphoresis, pallor, flushing), eyes (sensitivity to light, tearing), muscle strength and movement.

Nursing diagnoses and interventions

The primary response of the person requiring nursing interventions is acute pain. Develop nursing interventions to help the person identify strategies for controlling the pain and discomfort of the headache.

Acute pain

Headaches originate from both intracranial and extracranial sources and range in severity from benign, transient discomfort to severe, incapacitating pain. Interventions focus on teaching the person self-care measures to control or relieve the pain and reducing any associated problems, such as nausea and vomiting or anxiety.

- Advise to maintain a diary of headaches, including duration, onset, location, relation to menstruation or food intake and related manifestations such as factors that relieve or intensify the pain. A thorough assessment of the headache is essential for both the person and the healthcare provider to identify the circumstances and patterns of headache occurrence.
- Ask the person to rate the pain or discomfort on a scale of 0 to 10 (with 10 being the worst pain). Using a scale to rate the pain provides an objective measure of the person's subjective experience of the pain or discomfort. The scale can also be used to evaluate the effectiveness of pain relief measures.
- Advise to minimise light, noise and activity, and to rest in a quiet, non-stimulating environment when experiencing a headache. Manipulating the environment helps reduce noxious stimuli that may increase pain.
- Advise to use non-invasive and non-pharmacological pain relief measures such as deep breathing or relaxation to facilitate self-management of pain (see Chapter 8). Alternative strategies to control pain can help to reduce tension and increase the person's sense of control over the pain.

MEDICATION ADMINISTRATION Headaches

BETA-BLOCKERS

Propanolol hydrochloride (Inderal)
Pindolol (Barbloc)
Atenolol (Tenormin)
Timolol (Blocadren)

Beta-blockers are effective in the prophylactic treatment of headache. They act by combining with beta-adrenergic receptors to block the response to sympathetic nerve impulses, circulating catecholamines or adrenergic drugs.

Nursing responsibilities

- Before beginning therapy, determine pulse and blood pressure in both arms with the person lying, sitting and standing.
- Assess baseline and monitor serum glucose level, FBC, electrolytes and liver and renal function studies.
- Note any history of diabetes or impaired renal function.
- Note the rate and quality of respirations; drugs in this category may cause dyspnoea and bronchospasm.
- Administer the drug with meals to prevent gastrointestinal disturbances.
- Be alert that beta-blockers cause bradycardia and the heart rate may not rise in response to stress, such as exercise or fever. Notify the primary healthcare provider if pulse falls below 50 or if blood pressure changes significantly.
- Teach the person or family member how to take a pulse and blood pressure reading.

Health education for the person and family

- Take the medication with meals to provide a coating for the gastrointestinal tract and prevent gastrointestinal disturbances.
- Return for blood work as prescribed.
- Take the last dose of the day at bedtime.
- Rise from a sitting or lying position to a standing position slowly to avoid dizziness and falls.
- Take pulse and blood pressure each day and maintain a record of readings.
- Avoid excessive intake of alcohol, coffee, tea or cola. Consult with the healthcare provider before taking any over-the-counter medications.
- Report any cough, nasal stuffiness or feelings of depression to the healthcare provider.

TRICYCLIC ANTIDEPRESSANTS

Imipramine hydrochloride (Tofranil)
Amitriptyline hydrochloride (Endep)

The tricyclic antidepressants have been successful in the prophylaxis of cluster and migraine headaches. Although the exact mechanism is not known, they do prevent the re-uptake of noradrenaline or serotonin or both. They are chemically related to the phenothiazines and as such they exhibit many of the same pharmacological effects (e.g. anticholinergic, antiserotonin, sedative, antihistaminic and hypotensive effects).

Nursing responsibilities

- Assess baseline FBC and liver function studies, heart sounds and neurological status before initiating prescribed therapy.

Health education for the person and family

- Make position changes slowly.
- Chew sugarless gum to relieve dry mouth.
- Do not abruptly quit taking the medication.

ERGOT ALKALOID DERIVATIVES

Methysergide maleate (Deseril)

Methysergide is an ergot alkaloid derivative structurally related to LSD. It acts by stimulating smooth muscle, leading to vasoconstriction. It is thought that methysergide prevents headaches by blocking the effects of serotonin, a powerful vasodilator believed to play a role in vascular headaches. It also inhibits the release of histamine from mast cells and prevents the release of serotonin from platelets.

Nursing responsibilities

- Note any history of kidney or hepatic disease.
- Assess baseline eosinophil and neutrophil counts before beginning therapy.
- Administer the drug with meals or milk to minimise gastrointestinal irritation due to increased hydrochloric acid production.
- Assess for kidney, CNS and cardiovascular complications.
- Drug dosage should be gradually reduced over 2 to 3 weeks to prevent rebound headaches. A drug-free interval of 3 to 4 weeks is required with each 6-month course of therapy to prevent complications.
- Monitor for signs of ergotism, such as coldness or numbness of the fingers and toes, nausea, vomiting, headache, muscle pain and weakness. Vasoconstriction may further impair peripheral circulation and increase blood pressure.

Health education for the person and family

- Take the medication with meals or milk to minimise gastrointestinal upset.
- Report to the healthcare provider nervousness, weakness, rashes, hair loss or swelling of the extremities.
- Weigh daily and report any unusual weight gain to the healthcare provider.
- Return to the healthcare provider for a check-up at least every 6 months or as instructed. Do not take the drug on a regular basis for longer than 6 months, but do not abruptly stop taking it.
- Return for follow-up blood work as ordered.

SEROTONIN SELECTIVE AGONISTS

Sumatriptan succinate (Imigran)
Zolmitriptan (Zomig)

These agents bind to vascular receptors to vasoconstrict cranial blood vessels and relieve migraine headache.

Nursing responsibilities

- Assess for history of peripheral vascular disease, kidney or hepatic problems, and pregnancy.
- Evaluate relief of migraine headache and assess for side effects of photophobia, sound sensitivity, and nausea and vomiting.

Health education for the person and family

- Do not use more than two injections in a 24-hour period and allow at least 1 hour between injections.

MEDICATION ADMINISTRATION Headaches (continued)

- Use the autoinjector to administer the medication and follow instructions for the proper method of giving the injection and disposing of the syringe.
- Report wheezing, heart palpitations, skin rash, swelling of the eyelids or face, or chest pain to the healthcare provider immediately.

CALCIUM CHANNEL BLOCKERS

Verapamil (Isoptin)
Nifedipine (Adalat)

The calcium channel blockers may have value in controlling cerebral vasospasms by two mechanisms: inhibiting the influx of calcium into the cerebral artery; and interfering with the destruction of erythrocytes and aggregation of platelets.

Nursing responsibilities

- These drugs cause peripheral vasodilation. Therefore, monitor blood pressure and pulse during the initial administration of the drug. Any excessive hypotensive response and tachycardia may precipitate angina. Request written parameters for safe drug administration.
- Monitor intake and output and daily weights. Assess for manifestations of congestive heart failure: weight gain, peripheral oedema, dyspnoea, rales and jugular vein distension.
- Teach the person and family members how to take pulse and blood pressure readings.

Health education for the person and family

- Take the medication with meals to reduce gastrointestinal irritation.
- Take pulse and blood pressure before taking medications each day at the same time and follow instructions regarding when to withhold medication and when to contact the healthcare provider. Keep a record of pulse and blood pressure readings.
- Report any side effects, such as dizziness, vertigo, unusual flushing, facial warmth or headaches, to the healthcare provider.
- Report immediately any swelling of the hands or feet, pronounced dizziness or chest pain accompanied by sweating, shortness of breath or severe headaches.

NON-STEROIDAL ANTI-INFLAMMATORY DRUG (NSAID): SALICYLATE

Acetylsalicylic acid, or aspirin, is a non-narcotic analgesic, antipyretic, anti-inflammatory agent used to relieve headache pain.

Nursing responsibilities

- Determine the type and pattern of pain. If aspirin was used in the past for pain control, note its effectiveness.
- Note any history of peptic ulcers or other conditions that may suggest potential problems with the use of salicylates.
- Assess people receiving anticoagulant therapy for bruises, bleeding of the mucous membranes or blood in the urine or stool.

Health education for the person and family

- Take aspirin after meals or before meals with an antacid and a full glass of water to minimise gastric irritation.
- Report ringing in the ears, unusual bleeding of gums, bruising or black tarry stools to the primary healthcare provider.
- Monitor blood glucose levels carefully (if you have diabetes) and report hypoglycaemia if it occurs.

ERGOTAMINE

Caffeine-ergotamine tartrate combination (Cafergot)
Ergotamine tartrate (Gynergen)

Ergot alkaloids vasoconstrict the cerebral blood vessels, decreasing the amplitude of the pulsations of the cranial arteries. The major use of ergot alkaloids is for the treatment of migraine headaches. Cafergot has the same actions as Gynergen; in addition, the caffeine it contains provides a vasoconstrictive action, enhancing the effects of ergotamine.

Nursing responsibilities

- Because the drug accumulates in the body and is eliminated slowly, ergotamine poisoning may occur. Sepsis, renal and vascular disease, heavy smoking, malnutrition, pregnancy, contraceptive hormones and fever can increase the risk of ergotamine poisoning.
- These drugs are contraindicated in people with diabetes mellitus, sepsis, hepatic or kidney disease, peripheral and coronary artery disease, hypertension and pregnancy.

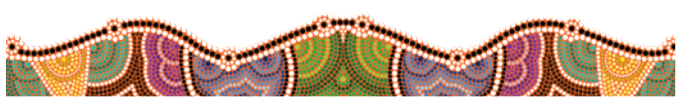
Health education for the person and family

- Take the drug immediately at onset of headache.
- Report the following to your healthcare provider: pain in the leg muscles, weakness and coldness or numbness of fingers or toes.
- A dose of Cafergot taken late in the day may prevent sleep because of the effects of caffeine.

- Suggest application of cold compresses or dry heat to the head and neck. The application of cold causes vasoconstriction, which helps reduce pain in vascular headaches. Application of heat can reduce muscle tension and improve circulation.
- Advise to follow good nutrition guidelines, get regular exercise and sleep, and minimise stress. Headaches are more likely to occur when ill, tired or under stress.

Community-based care

In addition to implementing comfort measures, personal education has a high priority. Develop a teaching plan to help the person learn how to limit attacks (e.g. by avoiding precipitating factors) and reduce the effects of the headache. Provide specific information about prescribed medications. Referrals for methods of stress reduction may be necessary for people with long-term or migraine headaches.



NURSING CARE PLAN A person with a migraine headache

Becky Friedman is a 30-year-old primary school teacher. Her friends and the other teachers regard Ms Friedman as an enthusiastic person who sets high standards for herself and strives for perfection. During the third term (the start of spring), Ms Friedman begins to miss work and sometimes appears very nervous. One day, another teacher notices Ms Friedman running down the hall and into the bathroom; the teacher finds Ms Friedman vomiting. As she washes up, Ms Friedman tells the other teacher that she has been having headaches since she began menstruating, but that they have never been as intense and frequent as during this past year. Ms Friedman agrees to see the nurse practitioner, Jane Schickadanz, at the school clinic for evaluation.

ASSESSMENT

During her health history, Ms Friedman relates that each month before her menstrual cycle she becomes nervous and sees flashing lights. She also has difficulty expressing herself and thinking clearly. The next day she develops a 'sick headache'. She states that the headache can last 1 to 2 days and that afterwards she cannot brush her hair because her scalp hurts. Ms Friedman attributes these symptoms to PMS and adds that she thinks she is allergic to cheese and nuts because she gets sick after eating them. After assessment and in consultation with the doctor, Ms Schickadanz diagnoses Ms Friedman's problem as a migraine with aura headache. Sumatriptan succinate (Imitrex) is prescribed.

DIAGNOSES

- *Acute pain* related to vasodilation of cerebral vessels and a decreased serotonin level manifested by the presence of headaches.
- *Knowledge deficit* related to pain management, evidenced by inadequate use of analgesia.
- *Altered role performance* related to pain, evidenced by an inability to perform occupational tasks.

PLANNING

- Provide education about migraine and methods of treatment.

Expected outcomes

- Experience reduced frequency and duration of pain.
- Identify available resources for helping with self-management of pain.

IMPLEMENTATION

- Ask Ms Friedman to keep a diary of her headaches for the next month, noting times of their occurrence, location and duration of pain, and factors that trigger the onset, such as her menstrual period or certain foods.
- Advise Ms Friedman to take medication at the first awareness of an impending attack.
- Suggest an appointment with a counsellor to learn methods of relaxation and stress relief.
- Request a dietary referral for elimination of foods that might precipitate headaches.

EVALUATION

Four weeks after beginning medication therapy with Imitrex and relaxation techniques, Ms Friedman has noted a decrease in the intensity of the headaches. She reports that the medication has stopped the headaches, which, she has noted, tend to occur more frequently immediately before her menstrual period. She is walking for 30 minutes each day and has made changes in her usual diet. Ms Friedman states, 'I feel good about going to work with my kids at school and knowing I can control my pain.'

CRITICAL THINKING IN THE NURSING PROCESS

1. List the questions you would include in a health history that would identify stressors consistent with migraine headaches.
2. Develop a teaching plan for Ms Friedman that includes methods of reducing fluid retention before her menstrual period, as well as a suggested diet based on the food guide pyramid.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future nursing practice.
2. Which education strategies could you use to develop a plan of care for Ms Friedman for the nursing diagnosis of *Disturbed sleep pattern* related to pain, evidenced by fatigue?

THE PERSON WITH EPILEPSY

Epilepsy (also called *seizure disorder*) is a chronic disorder of abnormal, recurring, excessive and self-terminating electrical discharge from neurons. Epilepsy is characterised by recurring seizures accompanied by some type of change in behaviour. A **seizure** (sometimes called a convulsion) is a single event of abnormal electrical discharge in the brain resulting in an abrupt and temporary altered state of cerebral function (Hickey, 2013). This abnormal neuronal activity, which may involve all or part of the brain, disturbs skeletal motor function, sensation, autonomic function of the viscera, behaviour and/or consciousness. Epilepsy is categorised as a paroxysmal disorder because its manifestations are discontinuous; that is, minutes, days, weeks or even years may elapse between seizures.

Incidence and prevalence

Epilepsy is one of the most common neurological conditions, affecting an estimated 275 000 people in Australia (Epilepsy Centre, 2015). There is a strong genetic component. Although people of any age may be affected, the prevalence and incidence of epilepsy increases dramatically in older adults (see the following 'Meeting individualised needs' box).

The incidence of epilepsy is increasing. Researchers have suggested that the increase may be due to technological advances in obstetric and paediatric care that allow extremely high-risk neonates to survive, and to other technological advances that have improved survival rates after craniocerebral trauma.

Isolated seizure episodes may occur in otherwise healthy people for a variety of reasons, including an acute febrile state,

FAST FACTS

- Epilepsy affects people of all ages and cultural backgrounds.
- Approximately 275 000 Australians have epilepsy (Epilepsy Centre, 2015).
- Epilepsy is not a mental disorder.

infection, metabolic or endocrine disorder (such as hypoglycaemia) or exposure to toxins. Epilepsy may be idiopathic (i.e. it may have no identifiable cause) or it may be secondary to birth injury, infection, vascular abnormalities, trauma or tumours.

Pathophysiology

Normally, when the mind is actively working, electrical activity in the brain is unsynchronised; when the mind is at rest, electrical activity is mildly synchronised. It is believed that most seizures arise from a few unstable, hypersensitive and hyper-reactive neurons in the brain. During a seizure, these neurons produce a rhythmic and repetitive hypersynchronous discharge. Although the exact initiating factor for seizure activity has not been identified, several theories have been proposed (Grossman & Porth, 2013):

- alterations in the permeability of, or ion distribution across, cell membranes
- alterations in the excitability of neurons resulting from neuroglia (neuroglia are CNS connective or supporting cells; they include astrocytes, oligodendroglia and microglia)
- scarring or decreased inhibition of activity in the cerebral cortex or thalamic region
- imbalances of excitatory and inhibitory neurotransmitters such as acetylcholine (ACh) or gamma aminobutyric acid (GABA).

All people have a seizure threshold; when this threshold is exceeded, a seizure may result. In some people, the seizure threshold may be abnormally low, increasing their risk of seizure activity; in other people, pathological processes may alter the seizure threshold (Grossman & Porth, 2013). The neurons that initiate seizure activity are called the *epileptogenic focus*. Abnormal neuronal activity may remain localised, causing a partial or focal seizure, or it may spread to involve the entire brain, causing generalised seizure activity.

Seizures may also be provoked or unprovoked. Unprovoked (primary or idiopathic) seizures have no identifiable cause, with multiple episodes diagnosed as a seizure disorder. Provoked (secondary) seizure aetiologies include febrile seizures in children, toxaemia of pregnancy, rapid withdrawal from alcohol or barbiturates, systemic metabolic conditions (such as hypoglycaemia, hypoxia, uraemia and electrolyte imbalances) and pathologies of the brain (such as meningitis, cerebral bleeding or cerebral oedema).

Metabolic needs of the brain increase dramatically during seizure activity. The demand for adenosine triphosphate (ATP), the energy source of the brain, increases by approximately 250%. Consequently, the demand for glucose and oxygen (which are needed to produce ATP) increases and oxygen consumption increases by about 60%. To supply this increased oxygen need and remove carbon dioxide and other metabolic by-products, cerebral blood flow increases to about 2.5 times that of the normal rate. As long as oxygenation, blood glucose levels and cardiac function remain normal, cerebral blood flow can respond to this increased metabolic demand of the brain. If cerebral blood flow cannot meet these needs, however, cellular exhaustion and cellular destruction may result.

Manifestations

Although seizures may be categorised in several different ways, the classification developed by the International League Against Epilepsy is the most useful clinically (cited in Papadakis et al., 2015). In this classification, seizures are divided into those that affect only part of the brain (partial seizures) and those that are generalised (affect all of the brain). An individual may have more than one type in what are called mixed seizures.

Partial seizures

Partial (or focal) seizures involve the activation of only a restricted part of one cerebral hemisphere. A partial seizure

MEETING INDIVIDUALISED NEEDS Epilepsy in older adults

For years, epilepsy was believed to be a disease that only affected children. However, the incidence of epilepsy is higher in older adults than in children. In people aged 65 years and over there has been a significant increase in the development of new seizure activity (Epilepsy Action Australia, 2015). These data have important implications for nursing assessments and care.

- The most common cause of epilepsy in older adults is arteriosclerosis of the cerebrovascular system (with up to 80% of the older population having arteriosclerosis).
- The manifestations of epilepsy in older adults are different from those in younger adults and children. Although 60% of younger people have generalised tonic–clonic seizures, only 30% of older adults have generalised tonic–clonic seizures. The most common type of seizure in older adults is a complex partial seizure.
- Older adults tend to have longer post-seizure manifestations than younger adults.
- Epilepsy that begins in older adults is often easier to control with anti-epileptic drugs (AEDs) than that in younger people. However, some AEDs decrease the effect of statins used to treat arteriosclerosis (the most common cause of epilepsy in older adults).

accompanied by no alteration in consciousness is called a simple partial seizure; one in which consciousness is impaired is called a complex partial seizure.

SIMPLE PARTIAL SEIZURES The manifestations of *simple partial seizures* depend on the involved area of the brain. Manifestations may include alterations in motor function, sensory signs or autonomic or psychic symptoms. Typically, the motor portion of the cortex is affected, causing recurrent muscle contractions of the face or a contralateral part of the body, such as a finger or hand. This motor activity may stay confined to one area or spread sequentially to adjacent parts, a phenomenon known as a *Jacksonian march* or *Jacksonian seizure*. Manifestations of a simple partial seizure involving the sensory portion of the brain may include abnormal sensations or hallucinations. Disruptions in the function of the autonomic nervous system, with resulting tachycardia, flushing, hypotension and hypertension, or psychic manifestations, such as a sense of déjà vu (a feeling that 'this has happened before') or inappropriate fear or anger, may also be experienced during a simple partial seizure.

COMPLEX PARTIAL SEIZURES During a *complex partial seizure*, consciousness is impaired and the person may engage in repetitive, non-purposeful activity, such as lip smacking, aimless walking or picking at clothing. These behaviours are known as automatisms. During the seizure, the person loses conscious contact with the environment; amnesia is common after the seizure and several hours may elapse before the person regains full consciousness. Complex partial seizures usually originate in the temporal lobe and may be preceded by an aura, such as an unusual smell, a sense of déjà vu or a sudden intense emotion.

Generalised seizures

Generalised seizures involve both hemispheres of the brain as well as deeper brain structures, such as the thalamus, basal ganglia and upper brainstem. Consciousness is always impaired with generalised seizures. Absence and tonic–clonic seizures are the common forms of generalised seizure activity; they occur more frequently (especially in children) than partial seizures.

ABSENCE SEIZURES *Absence (petit mal) seizures* are characterised by a sudden brief cessation of all motor activity accompanied by a blank stare and unresponsiveness. Absence seizures are more common in children than in adults. The seizure typically lasts only 5 to 10 seconds, although some may last for 30 seconds or more. Movements such as eyelid fluttering, or automatisms such as lip smacking, may occur during an absence seizure. Seizure activity may vary from occasional episodes to several hundred per day.

TONIC–CLONIC SEIZURES *Tonic–clonic seizures* (grand mal) are the most common type of seizure activity in adults. This type of seizure activity follows a typical pattern. A warning *aura* may precede generalised seizure activity. The aura may be a vague sense of uneasiness or an abnormal gustatory, visual, auditory or visceral sensation (such as a metallic taste in the mouth, a smell of burning rubber or seeing a bright light). Often, however, the seizure occurs without warning.

The seizure begins with a sudden loss of consciousness and sharp tonic muscle contractions (the tonic phase of the seizure). With the muscle contraction, air is forced out of the lungs and the person may cry out. Postural control is lost and the person falls to the floor in the opisthotonic posture (see Figure 41.4A). Muscles are rigid, with the arms and legs extended and the jaw clenched. Urinary incontinence is common; bowel incontinence may also occur. Breathing ceases and cyanosis develops during the tonic phase of a seizure. The pupils are fixed and dilated. The tonic phase lasts an average of 15 seconds, although it may persist for up to a minute.

The clonic phase, which follows the tonic phase, is characterised by alternating contraction and relaxation of the muscles in all the extremities along with hyperventilation (see Figure 41.4B). The eyes roll back and the person froths at the mouth. The clonic phase varies in duration and subsides gradually. The entire tonic–clonic portion of the seizure generally lasts no more than 60 to 90 seconds.

Following the clonic phase of seizure activity, the person remains unconscious and unresponsive to stimuli. This period is known as the postictal period or phase. The person is relaxed and breathes quietly. The person regains consciousness gradually and may be confused and disoriented on waking. Headache, muscle aches and fatigue often follow the seizure and the person may sleep for several hours. Amnesia of the seizure is usual; the person also may not recall events just prior to the seizure activity.

Because of the lack of warning with tonic–clonic seizures, the person may experience injury. Head injury, fractures, burns or motor vehicle crashes may occur secondarily to seizure activity.

Status epilepticus

Status epilepticus can develop during seizure activity. In this case, the seizure activity becomes continuous, with only very

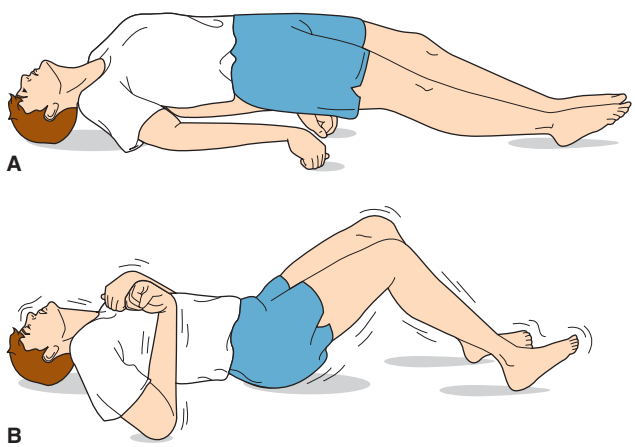


FIGURE 41.4 ■ Tonic–clonic seizures in grand mal seizures. *A*, Tonic phase. *B*, Clonic phase

short periods of calm between intense and persistent seizures. The repetitive seizures may be of any type, although they are usually generalised tonic–clonic (Grossman & Porth, 2013). Repeated seizures have a cumulative effect, producing muscular contractions that can interfere with respirations. The person is in great danger of developing hypoxia, acidosis, hypoglycaemia, hyperthermia and exhaustion if the convulsive activity is not halted. Status epilepticus is considered a life-threatening medical emergency that requires immediate treatment.

INTERPROFESSIONAL CARE

Initial treatment focuses on controlling the seizure; the long-term goal is to determine the cause and prevent future seizures. Interprofessional care includes diagnostic testing, medications and, in some cases, surgery.

Diagnosis

Diagnostic testing is performed to confirm the seizure diagnosis and to determine any treatable causes and precipitating factors. (See Chapter 40 for a description of neurological tests and related nursing care.) Radiological examinations include an MRI or CT scan to determine abnormalities in the brain and a skull x-ray to identify any bony abnormalities. An electroencephalogram (EEG) helps localise any brain lesions and confirm the diagnosis. A lumbar puncture may be performed to assess spinal fluid for CNS infections (increased WBCs) or tumours (increased protein levels). Blood studies are used to assess blood count, electrolytes, blood urea and blood glucose.

Medications

Anti-epileptic drugs (AEDs) (also called anticonvulsant drugs) can reduce or control most seizure activity. More than 20 drugs are used in the treatment of epilepsy. These medications do not cure the disorder; they only manage its manifestations. AEDs generally act in one of two ways: by raising the seizure threshold or by limiting the spread of abnormal activity within the brain.

The goals of medications for epilepsy are to protect the person from harm and to reduce or prevent seizure activity without impairing cognitive function or producing undesirable side effects. Ideally, the lowest possible dose of a single medication that will control the person's seizures is prescribed; often, however, several medications must be tried before the most effective is identified and a combination of drugs may be needed to manage the person's seizures. Therapy is individualised, based on the type of seizure activity and the person's response to the medication. Some drugs recommended for newly diagnosed adults with either partial or mixed seizures, and commonly used in Australia, are phenytoin (Dilantin), sodium valproate (Epilim), gabapentin (Neurontin), lamotrigine (Lamictal), oxcarbazepine (Trileptal) and topiramate (Topamax). Examples of and nursing implications for AEDs are described in the 'Medication administration' box below; drug interactions are listed in Box 41.3.

BOX 41.3 Drug interactions with AEDs

- *Sodium valproate (Epilim) and phenobarbital*. Blood levels of phenobarbital may rise significantly when sodium valproate is added to the person's medication regimen.
- *Phenobarbital and digoxin*. This combination may increase the metabolism of digoxin, resulting in decreased digoxin levels.
- *Phenobarbital and warfarin (Coumadin).* Phenobarbital may decrease the absorption of warfarin from the gastrointestinal tract and decrease the drug's anticoagulant response.
- *Disulfiram (Antabuse) and phenobarbital.* This combination may inhibit the metabolism of the anticonvulsant drug and increase the incidence of side effects associated with the anticonvulsant drug.
- *Carbamazepine and oral contraceptives.* Carbamazepine decreases the effectiveness of oral contraceptives.
- *Other drugs.* Other drugs reported to interact with anticonvulsant drugs include aspirin, certain antibiotics, isoniazid, acetazolamide (Diamox), antacids, folic acid and narcotics.

If the person has been seizure free for at least 3 years, withdrawal of medications may be considered, with the dose of one drug at a time reduced over weeks or months. There is no way to predict which people can remain seizure free without medication, but if seizures reoccur, the same medications usually provide good control.

Status epilepticus requires immediate intervention to preserve life. Establishing and maintaining the airway is a priority. A solution of 50% dextrose is administered intravenously to prevent hypoglycaemia. Diazepam (Valium) or lorazepam (Ativan) is given intravenously and the dose repeated in 10 minutes if necessary to stop seizure activity. Phenytoin (Dilantin) is administered intravenously for longer-term control of seizures. Phenobarbital may also be administered to people in status epilepticus.

Surgery

Resective surgery, with removal of the epileptogenic focus, is an option that is still in its early stages. Candidates for this type of surgery include those who are unresponsive to medical management, who have a unilateral focus and who have impaired quality of life from seizures. Resections of the temporal lobe are most commonly performed and are most effective for partial complex seizures. An estimated 5% of people with epilepsy may be candidates for surgery. The goal of surgery is to reduce the person's uncontrollable seizures.

To be selected as a candidate for surgery, the person must be highly motivated and psychologically prepared. A psychological screening is required because the preoperative preparation is extensive and time consuming and the

surgery is long and requires that the person remain awake during surgery so that they can cooperate and respond to commands. The EEG is monitored during surgery to identify the epileptogenic focus and evaluate the effect of surgical intervention.

General postoperative care for the person with intracranial surgery follows the nursing management guidelines outlined later in the chapter. Specific preoperative and postoperative care for a person with a seizure disorder is described in the box below.

Vagal nerve stimulation therapy

Vagal nerve stimulation (VNS) therapy is approved as a treatment for people with partial-onset seizures who do not respond to AEDs. The therapy does not stop the seizures, but rather reduces their number and improves the person's quality of life. It is almost always necessary to continue taking AEDs. The therapy is designed to prevent seizures by sending regular small pulses of electrical energy to the brain via the vagus nerve. A flat, round battery (about the size of a dollar coin) is implanted in the chest wall, and electrodes are threaded under the skin and wound around the vagus nerve in the neck. The battery is programmed to deliver a few seconds of electrical energy every few seconds. If the person feels that a seizure is about to happen, a discharge can be activated by passing a small magnet over the battery. In some people this stops the seizures. Side effects are hoarseness and throat discomfort.

Nursing care

See below for a nursing care plan for a person with seizures.

Health promotion

Health promotion activities for the person with seizures focus on teaching to reduce the incidence of seizure activity and to promote safety. Stress the following:

- Know the importance of follow-up care, of keeping medical appointments and of continuing to take AEDs as prescribed even when no seizures are experienced.

MEDICATION ADMINISTRATION Seizures

ANTI-EPILEPTIC DRUGS (AEDS)

Examples of AEDs are:
Phenytoin (Dilantin)
Phenobarbital
Primidone (Mysoline)
Carbamazepine (Tegretol)
Sodium valproate (Epilim)
Ethosuximide (Zarontin)
Clonazepam (Rivotril)
Gabapentin (Neurontin)
Lamotrigine (Lamictal)
Tiagabine hydrochloride (Gabitril)
Levetiracetam (Keppra)

AEDs are used to control chronic seizures and involuntary muscle spasms or movements characteristic of certain neurological diseases. These drugs act in the motor cortex of the brain to reduce the spread of electrical discharges from the rapidly firing epileptic foci in this area. These agents control seizures without impairing the normal functions of the CNS. Drugs effective against one type of seizure may not be effective against another; anticonvulsant therapy must be individualised.

Nursing responsibilities

- Monitor blood pressure, pulse and respirations.
- Note evidence of CNS side effects, such as blurred vision, dimmed vision, slurred speech, nystagmus or confusion. Gingival hyperplasia may be noted in people taking phenytoin.
- Recognise that if people are to be on prolonged therapy, they may need a diet rich in vitamin D.
- Monitor the serum calcium level as ordered; phenytoin can contribute to demineralisation of bone.
- When administering anticonvulsants intravenously, monitor closely for respiratory depression and cardiovascular collapse.
- Administer gabapentin 2 hours after antacids.
- Administer tiagabine hydrochloride with food.

Health education for the person and family

- Take the exact dosage prescribed. Do not increase, decrease or discontinue the dosage without obtaining the healthcare provider's approval; doing so may lead to convulsions.
- Avoid hazardous tasks until the drug has been regulated. AEDs may at first decrease mental alertness and cause drowsiness, headache, dizziness and incoordination of muscles. These effects are usually dose related and may disappear with a change of dosage or continued therapy.
- If you are taking phenytoin (Dilantin), maintain good oral hygiene: use a soft toothbrush, massage the gums and floss daily.
- It is very important to obtain liver function studies regularly as ordered by the healthcare provider. This will help detect early signs of hepatitis and other liver problems. Report for all scheduled laboratory studies, including complete blood count, kidney and liver function studies, and drug levels.
- Carry identification indicating the type of seizures for which you are being treated.
- Do not take gabapentin 1 hour before or less than 2 hours after an antacid.
- If you are taking lamotrigine and develop a rash, tell your healthcare provider.
- Take tiagabine hydrochloride (Gabitril) with food.

- Review any state and local laws that apply to people with seizure disorders. Driving a motor vehicle is usually prohibited for 6 months to 2 years after a seizure episode. Usually, a driver's licence can be reinstated or obtained after a seizure-free period and a letter from the nurse practitioner or doctor.
- Know drug interactions with other prescribed drugs, over-the-counter (OTC) drugs, street drugs and alcohol.
- Teach family members first aid for a seizure:
  - Cushion the head.
  - Loosen anything tight around the neck.
  - Turn on the side.
  - Nothing in the mouth.
  - Don't hold down.
- Teach family members to call for medical assistance:
  - if the seizure lasts for more than 5 minutes
  - if there is slow recovery, a second seizure or difficulty breathing after the seizure
  - if there are signs of injury (such as bleeding from the mouth).

Assessment

Collect the following data through the health history and physical examination:

- *Health history:* past seizures; age when the first seizure occurred, most recent seizure; factors precipitating a seizure, any warning signs (aura); prophylactic anticonvulsant therapy; and specific concerns the person may have about the seizures.
- *Physical assessment:* important data used in determining an accurate diagnosis describes manifestations obtained from nursing assessments before, during and after a seizure. (Table 41.6 lists nursing assessments with rationale.)

Nursing diagnoses and interventions

Nursing care of people with a seizure disorder focuses on providing care during and immediately after the seizure and on personal/family teaching. The person with seizures has a wide variety of responses to actual or potential changes in health status; interventions discussed in this section focus on facilitating physical and psychological comfort and safety.

Risk of ineffective airway clearance

During a seizure, the tongue may fall back and obstruct the airway, the gag reflex may be depressed, and secretions may pool at the back of the throat. These may put the person at risk of an obstructed airway. Most seizures occur in the home or community; therefore, teach these interventions to the person's family:

- Provide interventions to maintain a patent airway:
  - Loosen clothing around the neck.
  - Turn on the side.
  - Do not force anything into the mouth.
  - If prescribed and available, administer oxygen by mask.
  - Although it was at one time believed that it was necessary to place a padded tongue blade in the person's

TABLE 41.6 Nursing assessments before, during and after a seizure

ASSESSMENT	RATIONALE
What was the person's level of consciousness? If consciousness was lost, at what point?	Indicates area of brain involved and type of seizure
What was the person doing just before the attack?	May suggest precipitating factors
In which part of the body did the seizure start?	May indicate the site of seizure activity in the brain tissue; for example, if jerking movements were first observed in right hand, the seizure focus may be in left motor cortex
Was there an epileptic cry?	Usually indicates the tonic stage of a generalised tonic–clonic seizure
Were any automatisms such as eyelid fluttering, chewing, lip smacking or swallowing observed?	Often seen in complex, partial and absence seizures
How long did movements last? Did the location or character change (tonic to clonic)? Did movements involve both sides of the body or just one?	Indicates areas in which focal activity originated
Did the head and/or eyes turn to one side and, if so, which side?	Helps localise the focus of the seizure. During the seizure, the head and eyes typically will turn away from the side of the epileptogenic focus
Were there changes in pupillary reactions?	Indicates involvement of the autonomic nervous system
If the person fell, was the head hit?	Skull x-ray studies may be needed to rule out subdural haematoma or fracture
Was there foaming or frothing from the mouth?	Usually indicates a tonic–clonic seizure

NURSING CARE OF THE PERSON with seizures who is having surgery

PREOPERATIVE CARE

- For most people, AEDs are withheld the morning or evening of the day before surgery. *AEDs may interfere with intraoperative EEG monitoring.*
- For people with frequent and/or severe seizures, however, a partial dose of medication may be administered. *This prevents seizures or status epilepticus during surgery.*
- A low dose of analgesics is administered before surgery. *The person must remain awake throughout the lengthy procedure to respond to commands during EEG recording.*

POSTOPERATIVE CARE

- AEDs are administered parenterally until the person can tolerate oral fluids; medications are then continued orally. *It is common for the person to have seizures in the early postoperative period.*
- Steroids are administered for the first 3 days after surgery and are tapered and then discontinued during the following week. *Steroids are given to decrease cerebral oedema.*

NURSING CARE PLAN A person with a seizure disorder

Janet Carlson is a 19-year-old university student who lives with her parents and one younger sister. Although Janet had seizures while she was at high school, they have been controlled with medication. However, she had a tonic–clonic seizure yesterday and immediately made an appointment with her family GP. She is currently taking phenytoin (Dilantin) 300 mg/day as a maintenance medication to prevent seizures.

ASSESSMENT

Evita Farias, RN, completes a health history for Ms Carlson. During the history, she tells Ms Carlson that she has been under stress because of difficulties in completing her course requirements this semester. She has not been sleeping as many hours per night and sometimes she forgets to take her medication. Ms Carlson's serum phenytoin level is 8 mg/mL. Therapeutic level is 10 to 20 mg/mL.

DIAGNOSES

- *Risk of injury* related to recurrence of generalised tonic–clonic seizure activity and low serum phenytoin levels, evidenced by increased seizure activity.
- *Deficient knowledge* related to activities that may trigger seizure occurrence, the effect of stress on seizures and medication information manifested by increased seizure activity.

PLANNING

- Improved management of epilepsy through compliance with management regimens and increased knowledge of epilepsy.

Expected outcomes

- Awareness of precipitating and triggering factors related to the onset of seizures.
- Awareness of the relationship between emotional and physical stress and seizures.
- Awareness of the importance of taking AEDs.

IMPLEMENTATION

- Teach Ms Carlson and her family the following:
  - current information about seizures
  - care during and after a seizure
  - medication protocols
  - factors and activities that can trigger seizures
  - the importance of follow-up care.
- Refer Ms Carlson and her family to a local epilepsy support group.
- Recommend that she purchase and wear a MedicAlert® bracelet.

EVALUATION

Ms Carlson is instructed to continue taking Dilantin 300 mg/day. She states the importance of nutrition, rest and measures to reduce stress. She also discusses the importance of maintaining the proper blood levels of her medication, stating that too little or too much of the medication could cause problems. Ms Carlson recognises that the seizures had recurred during a busy time in high school during which she had forgotten to take her medication. She is now wearing a MedicAlert® bracelet. Ms Carlson is provided with the telephone number of Epilepsy Action Australia.

CRITICAL THINKING IN THE NURSING PROCESS

1. If you were Ms Carlson's nurse, would your teaching differ if she were living alone? If so, how?
2. Ms Carlson tells you that although she knows she should not drive a car, she often drives her friend to work. How would you approach this problem?

REFLECTION ON THE NURSING PROCESS

1. Which safety strategies could family members implement in the event of a person having a seizure?
2. Outline what you have learned from this case study that you will apply to your future nursing practice.

mouth during a seizure, this is no longer recommended; an improperly placed tongue blade can obstruct the airway. *Turning the person on the side allows secretions to drain from the mouth.*

- Teach family members or significant others how to care for the person during a seizure to prevent airway obstruction. *Family members are often the only people present to provide this emergency intervention.*

Anxiety

The person with a seizure disorder is understandably anxious about the future, with questions about ability to go to school or university, work, have a family and drive a car. Feelings of embarrassment about having a seizure in public and rejection by others are common and also increase the person's anxiety.

- Provide support by explaining that concerns are normal. It is important to be sensitive to the effect of seizures on the person's self-concept and body image; alterations in these areas not only increase anxiety but also cause withdrawal from socialisation with others. *Demonstrating acceptance of the person's concerns allows further discussion.*
- Help identify safe leisure activities. *Worrying about being hurt if a seizure occurs may cause withdrawal from social activities that are pleasurable.*
- Provide information about sources and support groups. *Sharing information with other people with similar health problems allows for a more realistic viewpoint; accurate information can clear up misconceptions that cause anxiety.*
- Provide accurate information about hiring practices and legal limitations on driving or operating heavy or dangerous machinery. *Accurate information decreases anxiety about the unknown. The federal* Disability Discrimination Act *in Australia prohibits discrimination; however, there are legal limitations on driving until the person is proved free of seizures.*

Community-based care

Teaching follows a systematic assessment of the needs of both the person and their family. Include family members so that they can learn seizure management, including the care and observations necessary before and during a seizure. Stress the importance of safety and keeping the airway patent.

Help both the person and their family adjust to a diagnosis of epilepsy. Address the following topics:

- the importance of wearing a MedicAlert® bracelet or carrying a medical alert card at all times
- avoiding alcoholic beverages and limiting coffee intake
- taking showers versus baths because of safety issues during a generalised seizure
- factors that may trigger a seizure, such as abrupt withdrawal from medication, constipation, fatigue, excessive stress, fever, menstruation, sights and sounds such as television, flashing video and computer screens
  - helpful resources, including:
  - Epilepsy Action Australia: www.epilepsy.org.au
  - Epilepsy Australia: www.epilepsyaustralia.net
  - Epilepsy Foundation: www.epinet.org.au.

TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) (also called *craniocerebral trauma*) refers to any injury of the scalp, skull (cranium or facial bones) or brain. TBI is the second-largest cause of death and disability in Australia and worldwide, with occurrence three times greater in the Indigenous Australian population, particularly females. TBI may be defined as a traumatic insult to the brain capable of causing physical, intellectual, emotional, social and vocational changes. A TBI may be classified as a penetrating (open) head injury (e.g. resulting from a knife, bullet or baseball bat) or a closed head injury (a blunt injury to the brain that does not result in an open skull fracture). TBI may cause problems with cognition, movement, sensation and emotions. Even mild brain injuries, if repeated over an extended period of time, can result in cumulative neurological and cognitive deficits.

FAST FACTS

- Each year, 1000 Australians sustain a TBI (Australian Trauma Quality Improvement Program, 2012).
- In 2004–2005, 22 710 Australians were hospitalised with TBI as the primary or associated diagnosis. Of these, 980 died (Australian Institute of Health and Welfare (AIHW), 2007).
- Approximately 438 300 Australians live with a disability as the result of a TBI (AIHW, 2007).

In Australia, the leading causes of TBI are falls (40%), followed by transportation accidents (31%) and assaults (14%). Elevated blood alcohol levels, not wearing motorcycle helmets and not wearing seat belts contribute significantly to the risk of crashes and subsequent injury. Other causes of head injury include sports injuries and occupational and recreational injuries (Brain Injury Australia, 2015). Adults aged 15 to 44 are at the greatest risk, with a male-to-female ratio in the Anglo-Saxon population of 3:1 (Hickey, 2013). Other risk factors include being over the age of 75 and living in a high-crime area.

Specific damage following craniocerebral injuries is related to the mechanism of the injury (how it occurs), the nature of the injury (type) and the location of the injury (where it occurs).

Head injuries may be classified as blunt or penetrating and can occur through several mechanisms:

- Acceleration injury is sustained when the head is struck by a moving object, such as a swinging bat.
- Deceleration injury occurs when the head hits a stationary object, such as a concrete wall.
- Acceleration–deceleration injury (also called a *coup–contrecoup* phenomenon) occurs when the head hits an object and the brain 'rebounds' within the skull (see Figure 41.5). The brain is injured at the point of impact (the coup) and on the opposite side of the impact (the contrecoup).

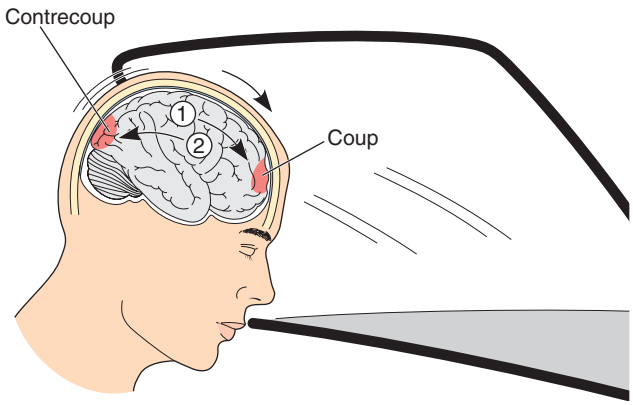


FIGURE 41.5 ■ Coup–contrecoup head injury. Following the initial injury (coup), the brain rebounds within the skull and sustains additional injury (contrecoup) in the opposite part of the brain

TABLE 41.7 Types of skull fractures

TYPE	DESCRIPTION
Linear	Simple, clean break in skull
(simple)	Occurs with low-velocity injuries
Comminuted	Bone is crushed into small, fragmented pieces
	Usually seen with high-impact injuries
Depressed	Inward depression of bone fragments
	Usually due to a powerful blow to the skull
	The dura may or may not be intact
	Bone fragments may penetrate into the brain tissue
Basilar	Occurs at the base of the skull
	May be linear, comminuted or depressed

Two or more areas of the brain can be injured as a result of this phenomenon.

- Deformation injuries are those in which the force deforms and disrupts the integrity of the impacted body part (e.g. skull fracture).

Types of craniocerebral trauma include injuries to the skull (including fractures), injuries to the brain (including concussion and contusion) and intracranial haemorrhage (including haematomas). Brain injury can result either from the direct effects of the trauma on brain tissue or from secondary responses to trauma, such as cerebral oedema, haematoma (blood clot), swelling or increased intracranial pressure.

THE PERSON WITH A SKULL FRACTURE

A *skull fracture* is a break in the continuity of the skull. It may occur with or without damage to the brain; however, intracranial trauma often results from skull fractures. The considerable force of impact significantly increases the risk of underlying haematoma formation. Disruption of the skull can also cause cranial nerve injury, allow bacteria to enter the cranial vault, and/or allow CSF to leak out.

Pathophysiology

Skull fractures are classified as open or closed. In an open fracture, the dura is torn; and in a closed fracture, the dura is not torn. Skull fractures are further classified into one of four categories: linear, comminuted, depressed or basilar (see Table 41.7).

Linear fractures

Linear fractures are the most common, accounting for 80% of all skull fractures. They typically extend from the point of impact towards the base of the skull. Although the risk of infection or CSF leakage is minimal with this type of fracture because the dura usually remains intact, subdural or epidural haematomas (a collection of blood) frequently underlie the fracture. A haematoma (discussed later in this chapter) places pressure on underlying brain tissue, increasing both intracranial pressure and the risk of brain damage.

Comminuted and depressed fractures

Comminuted and depressed skull fractures increase the risk of direct damage to brain tissue from bruising (contusion) and bone fragments. However, the risk of secondary brain injury may be reduced in these fractures, because in breaking the bone, the traumatic impact energy is distributed and dissipated. If the skin overlying the fracture is lacerated or the dura is torn, the risk of infection is increased.

Basilar fractures

Basilar skull fractures involve the base of the skull and usually are extensions of adjacent fractures, although they may occur independently. Although most basilar skull fractures are uncomplicated, they may involve the sinuses of the frontal bone or the petrous portion of the temporal bone (middle ear). If the dura is disrupted, CSF may leak through the tear. CSF leakage may include *rhinorrhoea* (CSF leakage through the nose) or *otorrhoea* (CSF leakage from the ear). Blood may be visible behind the tympanic membrane (haemotympanum) or ecchymosis may be noted over the mastoid process (Battle's sign). Bilateral periorbital ecchymosis ('raccoon eyes') is another possible manifestation. If CSF leakage is present, the risk of infection is high. Other complications of basilar skull fractures include injury to the internal carotid artery and compression of cranial nerve I, II, III, IV, V, VII or VIII.

INTERPROFESSIONAL CARE

Treatment of the person with a skull fracture depends on the type and location of the fracture. Skull fracture may be only one of several head injuries.

A simple linear fracture generally requires bed rest and observation for underlying injury to brain tissue or haematoma formation. No specific treatment is required. Depressed skull fractures require surgical intervention, usually within 24 hours of the injury, to debride the wound completely and remove bone fragments, which may become embedded in brain tissue or cerebral blood vessels. If depressed deeply, the bone may be elevated. If cerebral oedema is not present, a cranioplasty with insertion of acrylic bone may be performed. Basal skull fractures do not require surgery unless CSF leakage persists. Regular neurological assessments and observation for manifestations of meningitis are required for the hospitalised person. Antibiotics may be administered prophylactically.

Nursing care

The person with a craniocerebral trauma may have a variety of responses and healthcare needs, depending on the location and extent of the trauma. Many of those problems, with related nursing interventions, are discussed in other sections of this chapter, including seizures, increased intracranial pressure and bleeding within the brain.

Nursing diagnoses and interventions

Risk of infection

The person with a skull fracture is at increased risk of infection related to access to the cranial contents through a tear in the dura. In an open, depressed fracture, the wound may be contaminated by dirt, hair or other debris.

- Monitor for otorrhoea or rhinorrhoea. *Open fractures of the skull increase the possibility of leakage of CSF from the ears or nose.*
- Test drainage of clear fluid from ear and nose for glucose by using a glucose reagent strip, such as Dextrostix. *Clear drainage that tests positive for glucose indicates leakage of CSF; however, be aware that false positives may occur.*
- *Observe blood-tinged fluid for 'halo' sign. CSF dries in concentric rings on gauze or tissues. This sign is suggestive of CSF leakage.*
- Keep the nasopharynx and the external ear clean. Place a piece of sterile gauze in the ear or tape a sterile cotton pad loosely under the nose; change dressings when they become wet. *Wet dressings facilitate movement of organisms.*
- Instruct person not to blow nose, cough or inhibit sneeze; sneeze through open mouth. *Blowing the nose and coughing increase ICP. Withholding a sneeze forces bacteria backwards.*
- Use aseptic technique at all times when changing head dressings or ICP monitor dressings and insertion sites. *Using aseptic technique reduces the possibility of introducing infection.*

Knowledge deficit: skull fracture

The person and their family need to be informed about the degree of injury that has occurred with the skull fracture. The person with a linear fracture, who may not be hospitalised, will need teaching that focuses on the need to monitor progress closely. To prevent complications, advise the person and their family to go to the emergency room if the person experiences any of the following:

- growing drowsiness or confusion
- difficulty waking (instruct a family member to wake the person every 2 hours during the first night home)
- vomiting (especially if projectile)
- blurred vision
- slurred speech
- prolonged headache
- blood or clear fluid leaking from the ears or nose
- weakness in an arm or leg
- stiff neck
- seizure.

THE PERSON WITH A FOCAL OR DIFFUSE TRAUMATIC BRAIN INJURY

Even when the skull and other structures overlying the brain remain intact, a blow to the head can cause significant brain injury. Closed head injuries may result in either focal or diffuse damage to the brain. They range in severity from mild to severe.

Pathophysiology

Brain injury results from both primary and secondary mechanisms. Primary injury results from the impact. A blow to the head, even with no break in the skull, can cause serious and diffuse brain injury. Injury to axons disrupts oligodendroglia and direct mechanical disruption is caused by debris and leakage. The immediate vascular response to the injury results in increased capillary permeability to solutes.

Secondary injury is the progression of the initial injury resulting from events that affect perfusion and oxygenation of brain cells. These events include intracranial oedema, haematoma, infection, hypoxia or ischaemia. Cerebral ischaemia is the most common cause of secondary brain injury (Grossman & Porth, 2013). Ischaemia leads to cerebral hypoxia, with consequences of increased glial permeability to sodium (cytotoxic oedema), an influx of calcium with changes in electrophysiology and release of free fatty acids and lactic acidosis.

TABLE 41.8 Systemic effects of acute brain injury

CAUSE	EFFECT
• Stimulation of the sympathetic nervous system, which stimulates the adrenal cortex and medulla to increase glucocorticoid and mineralocorticoid levels	• Increased metabolism of carbohydrates, fats and proteins • Retention of sodium and water
• Stimulation of the sympathetic nervous system, increasing the serum catecholamine levels	• Hypertension • EEG changes • Arrhythmias (bradycardia, sinus tachycardia)
• Altered release of ADH from the posterior pituitary	• Retention of water or diuresis and diabetes insipidus
• Neurogenic pulmonary dysfunction	• Abnormal respiratory patterns • Reduced residual capacity with retention of CO_2, vasodilation and increased ICP • Pulmonary oedema
• Stress response to trauma	• Hyperglycaemia
• Increased platelet, plasma fibrinogen and thromboplastin levels	• Decreased clotting and prothrombin times • Vascular occlusion • Disseminated intravascular coagulation • Anaemia
• Immunosuppression	• Infection
• Decreased gastric motility and increased gastric acidity	• Gastritis • Gastric ulcers

Acute brain injury affects all body systems as well as the central nervous system. Systemic effects of acute brain injury are listed in Table 41.8.

Focal brain injuries

Focal brain injuries are specific, grossly observable brain lesions confined to one area of the brain. They include contusions, lacerations and intracranial haemorrhage. The force of an impact produces contusions from direct contact with the inside of the skull that in turn may cause epidural haemorrhage and subdural and intracerebral haematomas. The mechanisms of injury are coup and/or contrecoup damage to the brain at the point of the impact and the rebound effect. The damaged brain area is surrounded by oedema, contributing to IICP. Infarction and necrosis, multiple haemorrhages and oedema are found within the contused areas. The maximum effects of the injury peak in 18 to 36 hours.

Intracranial haemorrhage can result directly from the trauma (e.g. beneath a fracture) or from shearing forces on cerebral arteries and veins that occur with acceleration–deceleration. Depending on the site and rate of bleeding, manifestations may appear immediately or may not become evident for hours or even weeks. Intracranial haemorrhages and the haematomas they cause place pressure on surrounding structures, causing manifestations of an expanding focal lesion. They also cause IICP, leading to altered levels of consciousness and potential herniation syndromes. Intracranial haematomas are classified by their location as epidural, subdural or intracerebral. Table 41.9 compares the frequency, locations/common sites, precipitating factors and manifestations of intracranial haematomas; Figure 41.6 illustrates their locations.

CONTUSION A *contusion* is a bruise of the surface of the brain, typically accompanied by small, diffuse venous haemorrhages. Both white and grey matter may have a bruised, discoloured appearance. A decrease in pH, with accumulation of lactic acid and decreased oxygen consumption, may hinder cell function. Contusions (and other focal brain injuries) occur when the brain strikes the inner skull, often with a coup (point of impact) lesion and a contrecoup lesion on the opposite side of the brain. Contusions occur most frequently near bony prominences of the skull. Cerebral oedema can follow contusion, resulting in IICP. Contusions—small, diffuse venous

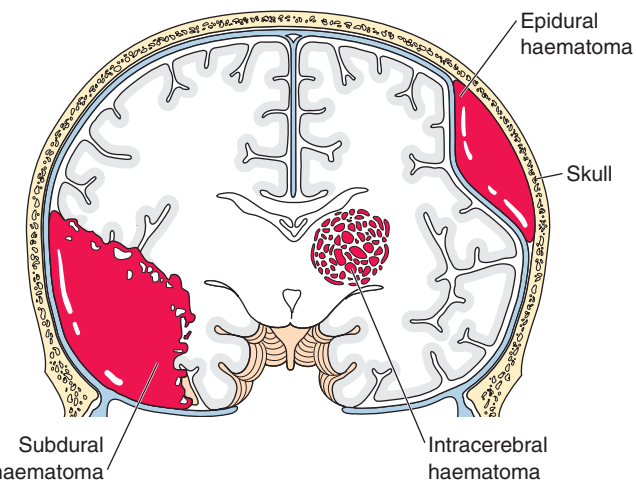


FIGURE 41.6 ■ Three types of haematoma: epidural haematoma, subdural haematoma and intracerebral haematoma

TABLE 41.9 Comparison of intracranial haematomas

TYPE/FREQUENCY	LOCATION/COMMON SITE	PRECIPITATING FACTORS	MANIFESTATIONS
Epidural haematoma 2–6% of all types of head injuries	Located in the space between the skull and the dura mater Common site: the temporal bone (over the middle meningeal artery)	Skull fractures Contusion	Momentary loss of consciousness followed by a lucid period lasting from a few hours to 1 to 2 days Rapid deterioration in LOC (drowsiness to confusion to coma) Seizures Headache Hemiparesis (may be ipsilateral or contralateral) Fixed dilated ipsilateral pupil Rise in blood pressure, with decreases in pulse and respirations, indicates a rapidly increasing haematoma
Subdural haematoma Approximately 29% of all types of head injuries	Located in the space below the dural surface (between the dura and arachnoid and pia mater layers of meninges) Common site: may occur any place in cranium	Closed head injury Acceleration–deceleration injury Cerebral atrophy (seen in older adults) Chronic alcoholism Use of anticoagulants Contusion	Acute: • headache • drowsiness • agitation • slowed thinking • confusion Subacute: • same as those of acute subdural haematoma but develop more slowly Chronic: • manifestations may not appear until weeks to months after injury • confusion, slowed thinking, drowsiness
Intracerebral haematoma 14–15% of all types of head injuries	Located directly in the brain tissue Common sites: frontal or temporal region	Gunshot wounds Depressed bone fractures Stab injury Long history of systemic hypertension Contusions	Headache Deteriorating consciousness to deep coma Hemiplegia on contralateral side Dilated pupil on the side of the clot

haemorrhages—and brain swelling are at their peak 12 to 24 hours after injury.

Manifestations of the contusion depend on the size and location of the brain injury. An initial loss of consciousness occurs; LOC may remain altered and behaviour changes such as combativeness may persist for an extended period. Full consciousness may be regained extremely slowly and residual deficits may persist; in some people, full LOC never really returns. Focal effects of the contusion may cause loss of reflexes, hemiparesis (muscular weakness of one-half of the body) or abnormal posturing. Manifestations of IICP may occur if cerebral oedema develops. Regaining full LOC may take an extended period of time and residual deficits may persist.

EPIDURAL HAEMATOMA An **epidural haematoma** (also called an *extradural haematoma*) develops in the potential space between the dura and the skull, which normally adhere to one another. As the blood collects, the expanding haematoma strips the dura away from the skull. Epidural haematomas affect young to middle-aged adults more frequently than older adults, because the dura becomes more tightly attached to the skull with ageing.

Epidural haematomas usually result from a skull fracture that tears an artery, often the middle meningeal artery. Because epidural haematomas are arterial in origin, they tend to develop rapidly. The person may lose consciousness with the initial injury and then have a brief lucid period before the LOC rapidly declines from drowsiness to coma as the haematoma expands, stripping the dura away from the skull and placing pressure on brain tissue. Other manifestations include headache, vomiting, a fixed, dilated pupil on the same side (ipsilateral) as the haematoma, contralateral (opposite side) hemiparesis or

hemiplegia and possible seizures. Because epidural haematomas usually develop rapidly, timely intervention is vital to prevent significant increases in ICP and herniation.

SUBDURAL HAEMATOMA **Subdural haematomas**, in which a localised mass of blood collects between the dura mater and the arachnoid mater, are more common than epidural haematomas. Acute subdural haematomas are usually located at the top of the head and develop within 48 hours of the initial head injury. Chronic subdural haematomas develop over weeks or months. The chronic type is seen most often in the older adult and people who have some brain atrophy with subsequent enlarged epidural space. These haematomas are often venous in origin, although they may involve bleeding from small arteries as well. Subdural haematomas may form without direct trauma or contusion; acceleration–deceleration forces may tear the bridging veins that connect veins on the surface of the cerebral cortex to the dural sinuses. As blood collects, it places direct pressure on underlying brain tissue.

Acute subdural haematomas develop rapidly following head injury. Although a lucid period may occur, the person commonly develops drowsiness, confusion and enlargement of the ipsilateral pupil within minutes or hours of the injury. If responsive, the person may complain of a unilateral headache. Hemiparesis and respiratory pattern changes may occur.

Chronic subdural haematomas are often associated with relatively minor trauma such as a fall. Weeks to months may elapse before manifestations of the haematoma occur; the initial trauma may have been forgotten. Chronic subdural haematomas may also occur spontaneously in the older adult or in people with bleeding disorders. Manifestations of the haematoma develop slowly and may be mistaken for the onset of dementia in the older adult. Slowed thinking, confusion, drowsiness or lethargy are common early manifestations. Other manifestations include headache, dilation and sluggishness of the ipsilateral pupil, and possible seizures.

INTRACEREBRAL HAEMATOMA **Intracerebral haematomas** may be single or multiple and are associated with contusions. They may occur in any location but usually are found in the frontal or temporal lobes. They may result from closed head trauma, particularly contusion or shearing of small blood vessels deep within the hemispheres. Intracerebral haematomas can also accompany other types of head trauma such as lacerations. Older adults are particularly vulnerable to intracerebral haemorrhage because cerebral blood vessels are more fragile and easily torn.

The manifestations of intracerebral haematoma vary according to the location of the haematoma. Headache may develop, along with decreasing LOC, hemiplegia and dilation of the ipsilateral pupil. The expanding clot increases intracranial pressure and herniation may occur.

Diffuse brain injuries

A diffuse brain injury (DBI) affects the entire brain and is caused by a shaking motion, with twisting movement (rotational acceleration) as the primary mechanism of injury. DBIs include concussions and diffuse axonal injuries. Shearing stresses on brain tissue cause axonal damage from shearing, tearing or stretching of nerve fibres. The most serious axonal injuries are located furthest from the brainstem, with the frontal and temporal axonal tracts being most vulnerable to injury. Physical deficits resulting from DBIs include spastic paralysis, peripheral nerve injury, swallowing disorders, visual and hearing impairments, and taste and smell disorders. Damage decreases the speed of information processing and responding and disrupts attention, resulting in serious cognitive and affective impairments. Cognitive deficits that may result include disorientation and confusion, short attention span, problems with memory and learning, perceptual problems and poor judgment. Possible behavioural deficits include agitation, impulsivity, depression and social withdrawal.

Initially, the damage involves tearing of axons, blood vessels and brain tissue (visible only by electron microscope). The number of damaged axons progressively increases, with pathology involving the nuclei and axons. The damaged axons, which resemble sausage links, regress into round balls called retraction balls (visible with light microscopy). After several weeks, the retraction balls are replaced by clusters of microglia. In the final phase, astrocytosis (equivalent to scarring) occurs at the site of axonal damage, accompanied by demyelination of long axon tracts.

The categories of DBI include mild concussion, classic cerebral concussion and diffuse axonal injury. Manifestations of a concussion are listed in the box below.

MILD CONCUSSION The word **concussion** means violent shaking. A concussion involves temporary axonal disturbances. It is defined as a momentary interruption of brain function. A concussion is associated with an immediate, brief loss of consciousness on impact. Altered consciousness may last only seconds or may persist for several hours. Amnesia for events immediately preceding (antegrade amnesia) and following (retrograde amnesia) the injury is common. Other manifestations of concussion include headache, drowsiness, confusion, dizziness and visual disturbances such as diplopia or blurred vision.

MANIFESTATIONS Concussion

- Immediate loss of consciousness (lasting usually no longer than 5 minutes)
- Amnesia regarding events surrounding injury
- Headache
- Drowsiness, confusion, dizziness
- Visual disturbances
- Possible brief seizure activity with transient apnoea, bradycardia, pallor and hypotension

POST-CONCUSSION SYNDROME

- Persistent headache
- Dizziness
- Irritability and insomnia
- Impaired memory and concentration, learning problems

CLASSIC CEREBRAL CONCUSSION A classic cerebral concussion involves diffuse cerebral disconnection from the brainstem RAS. An immediate loss of consciousness occurs, lasting less than 6 hours. Both retrograde and anterograde amnesia occur. Cerebral contusions may be present. In a severe concussion, a brief seizure and respiratory arrest may occur; transient pallor, bradycardia and hypotension may accompany loss of consciousness.

Following concussion, people may develop post-concussion syndrome with persistent headache, dizziness, irritability, insomnia, impaired memory and concentration, and learning problems. Post-concussion syndrome may last for several weeks or, rarely, up to a year.

DIFFUSE AXONAL INJURY Diffuse axonal injury (DAI) is a brain injury in which a high-speed acceleration–deceleration injury, typically associated with motor vehicle crashes, causes widespread disruption of axons in the white matter. Focal lesions may be found in the corpus callosum, midbrain and brainstem. An immediate loss of consciousness occurs. The prognosis is poor; most people with severe DAI either die or remain in a persistent vegetative state.

DAIs may range from mild to severe. In mild DAI, coma lasts 6 to 24 hours, and cognitive, psychological and sensorimotor deficits may persist. In moderate DAI, injury and impairment is spread throughout the cerebral cortex and diencephalon. There is axonal tearing, coma lasting more than 24 hours and often incomplete recovery. In severe DAI, axonal injury occurs in both cerebral hemispheres, the diencephalon and the brainstem. Immediate autonomic dysfunction occurs and IICP is manifested. Profound cognitive and sensorimotor deficits occur, involving movement, verbal and written communication, ability to learn and reason, and ability to modulate behaviour.

INTERPROFESSIONAL CARE

The person with a brain injury may receive medical and/or surgical treatment. Specific guidelines for the medical management of head injury have been developed for concussion and acute TBI by each state and territory Department of Health. These guidelines include the following:

- *Concussion*. Following a concussion, the person may be observed for 4 hours in the emergency department (ED) and then discharged home with instructions for further observation to detect manifestations of secondary injury. If the loss of consciousness lasted for more than 2 minutes, the person may be admitted to the hospital for observation.
- *Acute TBI*. Recognition and management of acute TBI with transport to an ED is essential to personal outcomes. Morbidity and mortality increase with hypotension (systolic pressure less than 90 mmHg) and hypoxia (PaO_2 less than 60 mmHg), so fluids are given to support a mean systolic arterial blood pressure at more than 90 mmHg (Hickey, 2013). Assessment of the person's airway, breathing and circulation (ABCs), with management of dysfunction, is necessary to decrease the secondary effects of the brain injury. The fluid of choice for intravenous fluids is hypertonic saline, because it reduces intracranial hypertension. An intracranial pressure monitor probe may be inserted to assess ICP and monitor therapy to reduce cerebral oedema and maintain cerebral perfusion. Osmotic diuretics such as mannitol also may be administered to reduce cerebral oedema. Adequate oxygenation is vital to maintain cerebral metabolism; carbon dioxide is a potent vasodilator, and increased levels may contribute to cerebral oedema and IICP.

On admission to the ICU from the ED, the person may be placed on a special bed and connected to various monitoring devices. Invasive lines are inserted, including a central venous pressure (CVP) catheter, arterial line, pulmonary catheter, ventriculostomy, ICP monitor and, perhaps, a retrograde jugular catheter. In most instances, an endotracheal tube is inserted and connected to a mechanical ventilator, cardiac monitoring is initiated, bilateral sequential calf compressors are applied, pulse oximetry is started and a rectal temperature probe is inserted. All values are monitored for changes to ensure early detection of cerebral hypoxia and impending ischaemia to prevent secondary brain injury.

Diagnosis

Diagnostic testing may be done to monitor haemodynamic status and detect conditions that may contribute to cerebral oedema. Radiological examinations include skull x-rays (to identify skull fractures and assess penetrating objects) and CT scan or MRI to detect contusions and lacerations associated with diffuse axonal injury. ABGs are analysed, with particular attention to oxygen and carbon dioxide levels.

Managing ICP

ICP is managed (as described in a previous section) to re-establish equilibrium of the intracranial contents and prevent secondary brain damage. Treatments include airway management, hyperventilation (used if signs of herniation appear), fluid resuscitation, positioning, temperature regulation and medications. Medications other than those previously discussed include a category of drugs called neuroprotectants. These drugs are used to treat or alter some of the pathological pathways that occur in ischaemia and must be administered within a short time of the injury to be effective. Classifications of the drugs include lipid peroxidase inhibitors, free radical scavengers, receptor antagonists, calcium channel blockers and gangliosides.

Surgery

Small subdural haematomas can frequently be reabsorbed and may be treated conservatively, with close observation and supportive care. However, the treatment of choice for epidural haematomas and large acute subdural haematomas is surgical evacuation of the clot. This can often be

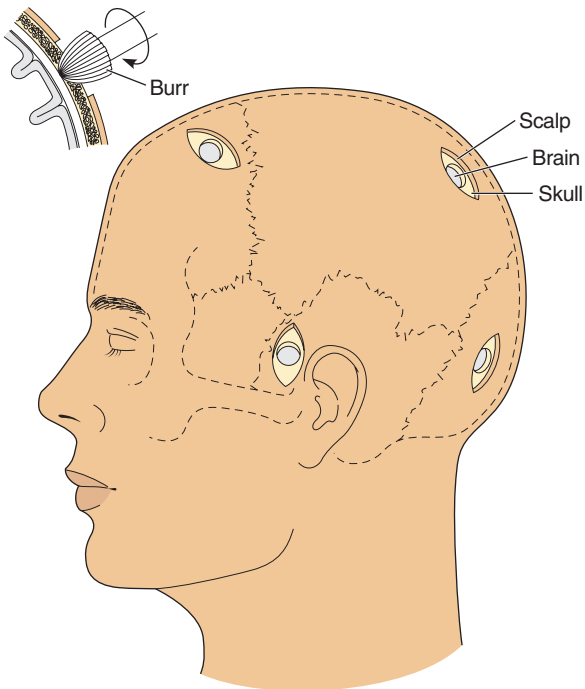


FIGURE 41.7 ■ Possible locations of burr holes

performed through burr holes made into the skull (see Figure 41.7). In an epidural haematoma, the bleeding vessel can also be ligated during this procedure, preventing further bleeding. Rebleeding may occur following evacuation of an acute subdural haematoma in older adults and in people with chronic alcoholism. A craniotomy is necessary to evacuate chronic subdural haematomas because the haematoma tends to solidify, making it difficult or impossible to remove through burr holes. Surgery is less successful in treating intracerebral haematomas because of widespread tissue damage. Supportive care to manage intracranial pressure and prevent complications is provided.

Nursing care

In addition to the nursing care discussed in this section, a nursing care plan for a person with a subdural haematoma follows.

Health promotion

The best way to treat any injury is to prevent it from happening. Public education must continue to stress the importance of safe driving, the dangers of driving under the influence of alcohol or drugs, and the necessity of wearing seat belts and cycle helmets. Legislation changes mandating the use of seat belts, child safety restraints and installation of airbags are all examples of health promotion. Other behaviours that can reduce the morbidity and mortality associated with TBI are following gun safety rules, promoting farm safety and teaching older adults about safety (such as preventing falls) in the home.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40):

- *Health history:* a history of the injury is helpful in understanding the nature of the craniocerebral trauma; knowledge about loss of consciousness assists the nurse in planning care.
- *Physical examination:* neurological assessment, including pupils, LOC, Glasgow Coma Scale, brainstem reflexes (cornea, cough, gag, extraocular movements), vital signs; skull and face (deformity, lacerations, bruising, bleeding); movement of extremities.

Nursing diagnoses and interventions

Nursing care of the person in the acute care phase initially focuses on maintaining an effective airway and breathing pattern. Nursing care is also directed towards continuous assessment and monitoring of neurological function as well as other body systems. This close monitoring provides early recognition and treatment of problems and complications and initiation of aggressive forms of therapy that may be needed.

Many nursing diagnoses associated with traumatic brain injury correspond with those outlined previously in the sections on the person with altered LOC and IICP. Specific nursing diagnoses discussed in this section focus on problems with intracranial adaptive capacity, airway clearance and breathing patterns.

Decreased intracranial adaptive capacity

The person with a traumatic brain injury has or is at high risk of IICP. As the mechanisms that normally compensate for changes in intracranial pressure are compromised, intracranial pressure increases in disproportional response to a variety of stimuli. (See the discussion earlier in the chapter for other nursing diagnoses and interventions for the person with IICP.)

- Monitor for manifestations of IICP, including eye opening response, motor response and verbal response. *These responses evaluate the ability to integrate commands with conscious and involuntary movement.*
- Monitor for changes in vital signs: bradycardia or tachycardia, varying breathing patterns, hypertension, and/or widening pulse pressure. *Vital signs vary depending on the site of impairment. Cushing's triad (bradycardia, increased systolic blood pressure and increased pulse pressure) indicates brainstem ischaemia leading to cerebral herniation.*

NURSING CARE PLAN A person with a subdural haematoma

Wong Lee is a 50-year-old boat mechanic who is married and has three sons. Although Mr Lee has been through rehabilitation twice for alcoholism, he has not been able to quit drinking. His doctor has explained the physical consequences and the possible interaction between alcohol and the anticoagulant Mr Lee is taking for chronic atrial fibrillation. While attending a family reunion, during which he eats a large meal and drinks several beers, Mr Lee joins a game of cricket. Mrs Lee is concerned that Mr Lee has consumed too much alcohol to play cricket in the heat, but Mr Lee is adamant and states that he wants to bowl. During Mr Lee's first over the batter hits a ball that strikes Mr Lee in the head. Mr Lee stumbles and drops to the ground, holding his head. He does not lose consciousness and gets up on his own. His sons and wife try to persuade him to go to the hospital, but Mr Lee insists he feels fine.

Two weeks later, after an evening of consuming several mixed drinks, Mr Lee develops a headache. He attributes the headache to a hangover, but instead of improving the next day, the headache becomes steadily worse. He becomes confused and disoriented. His wife, concerned that his drinking is increasing again, calls the doctor, who admits Mr Lee to the detoxification centre at the local hospital. A CT scan is performed. The diagnosis of a subdural haematoma is made and Mr Lee is transferred to the neurosurgical ward.

ASSESSMENT

When Saundra Knight, the nurse on the neurosurgical ward, enters the room, she notices that Mr Lee is sitting in bed, laughing softly and unable to hold himself upright while sitting in the bed, and is giddy. As she begins to talk to Mr Lee, he states, 'Don't ask me anything—I can't think. My headache is getting worse.' Over the next few hours, the giddiness subsides and Mr Lee becomes drowsy. Ms Knight reports a Glasgow Coma Scale score of 11. An ICP monitor is inserted and reveals increased intracranial pressure. Mr Lee is scheduled to have burr holes and haematoma evacuation that afternoon.

DIAGNOSES

- *Risk of ineffective breathing pattern related to pressure on* the respiratory centre by an intracranial haematoma, evidenced by either an increase or decrease in respiratory rate.
- *Ineffective cerebral tissue perfusion* related to increased intracranial pressure secondary to cerebral oedema, evidenced by a decreased LOC.

PLANNING

Maintain ICP within normal limits through close observation and assessment.

Expected outcomes

- Maintain a respiratory rate and rhythm within normal limits.
- Maintain adequate cerebral perfusion, as evidenced by stable vital signs, stable neurological status and no decrease in level of consciousness.

IMPLEMENTATION

- Perform neurological assessment every 2 hours or as needed.
- Monitor vital signs every 2 hours or as needed.
- Explain to the family the procedure for intracranial surgery.

EVALUATION

The first day postoperatively, Mr Lee begins breathing on his own without ventilatory support. His respiratory rate and rhythm are within normal limits, with no signs of abnormal breath sounds. The ICP monitor readings are appropriate and Mr Lee shows significant improvement in level of consciousness, with a Glasgow Coma Scale score of 15. Mr Lee continues to improve and is discharged home 5 days after surgery.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the similarities and differences between Mr Lee's disorder and the manifestations of other types of intracranial haematomas.
2. Mr Lee keeps trying to pull out his ICP line. You know he should not be restrained because pulling against restraints increases restlessness and increases intracranial pressure. What would you do?

REFLECTION ON THE NURSING PROCESS

1. Develop a care plan for the nursing diagnosis of *Acute confusion* related to increased intracranial pressure, evidenced by an altered LOC.
2. Outline what you have learned from this case study that you will apply to your future nursing practice.

- Monitor for vomiting, headache, lethargy, restlessness, purposeless movements and changes in mentation. *These manifestations may be early indicators of intracranial pressure changes.*
- Monitor temperature and initiate hypothermia treatment as prescribed. *Impaired hypothalamic function can interfere with temperature regulation. Hyperthermia may increase ICP.*
- Monitor fluid status: regularly compare intake and output, review serum osmolality and use an infusion pump to administer IV fluids (if prescribed). *Osmotic diuretics, if used to treat cerebral oedema, may cause hypotension and decreased cardiac output.*

CONSIDERATION FOR PRACTICE

Overhydration from rapid infusion of IV fluids may cause or further increase IICP.

Ineffective airway clearance

The primary objective in the care of any trauma person is maintaining a patent airway to prevent hypoxia. However, in the initial acute care phase, the risk of cervical vertebral fractures and spinal cord injury may complicate the process of establishing a patent airway. In addition, other

multisystem injuries may complicate the interpretation of vital signs. In general, all unconscious people with a head injury should be intubated with an endotracheal tube to prevent aspiration. Peoples with head trauma may also require a tracheostomy to provide an airway and may be placed on a ventilator.

- Monitor neurological manifestations on a regular schedule. *Changes in neurological manifestations may indicate IICP, with the risk of further depression of the respiratory system and respiratory arrest.*
- Maintain head and neck in neutral alignment, immobilised until injury is determined. *Head rotation and neck flexion are associated with IICP, decreased jugular venous outflow and localised changes in cerebral blood flow. Immobilisation prevents spinal cord injury in suspected or actual fractures of the cervical spine; spinal cord injury at this level would further impair respiratory function.*
- Clear the nose and mouth of mucus and blood. *This helps maintain patency of the upper airway.*
- Suction the airway as needed, limiting suctioning time to no more than 10 seconds at one time. Do not suction the nasal passages until a dural tear has been ruled out. *Suctioning is usually necessary to maintain a patent airway.*

Ineffective breathing pattern

The person with a traumatic brain injury and haematoma is at high risk *of Ineffective breathing pattern* related to IICP. If ICP increases dramatically, tentorial herniation may occur, leading to sudden respiratory arrest.

- Monitor the respiratory pattern for rate, depth and rhythm every 2 hours or as needed if the person is not on a ventilator. Assess breath sounds, presence of cyanosis, restlessness and use of accessory respiratory muscles. Monitor pulse oximetry and blood gas levels. *Head injuries may cause alterations in respirations. An increased respiratory rate may indicate hypoxia. A decrease in respiratory rate may be the result of depression of the medullary respiratory centre.*
- Monitor ICP readings. *Continuous measurement of ICP is used to diagnose and monitor increased intracranial pressure. As ICP increases, herniation may occur, leading to respiratory arrest and death.*

CONSIDERATION FOR PRACTICE

In general, an initial increase in intracranial pressure causes respirations to slow; as the pressure continues to increase, respirations become rapid.

- If the person is not intubated, prepare for oxygen administration and/or tracheal intubation if respiratory distress occurs. *Supplying oxygen prevents hypoxia until a haematoma can be evacuated, relieving pressure on the respiratory centre.*
- Prepare for cranial surgery if deteriorating respiratory pattern and neurological changes are noted. *Surgical intervention usually consists of placing several burr holes in the skull or performing a craniotomy to remove the haematoma. (Intracranial surgery is discussed later in the chapter.) However, the cerebral oedema and increased intracranial pressure may cause death even if surgery is performed.*

Community-based care

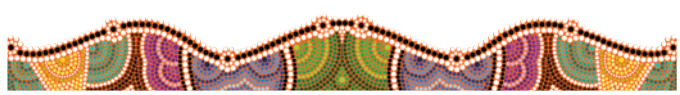
Concussion

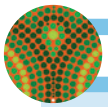
Advise the person and their family that a post-concussion syndrome sometimes occurs. If the person experiences persistent headaches and dizziness, is uncharacteristically emotional, seems overly tired or has difficulty paying attention or remembering, the healthcare provider should be notified. Explain that these manifestations may persist for some time. Rehabilitation may help the person compensate for memory impairment and attention deficits.

Acute brain injury

People who survive an acute brain injury will require long-term physical care and rehabilitation. Although recovery is highly individualised, many people who regain consciousness require lifelong care; others remain in a coma or vegetative state. The family often expects the person to recover fully after the coma subsides and they need information about the real possibility of residual deficits in self-care, emotional responses, cognition, communication and movement (see the 'Translation to practice' box below). Topics that should be addressed for home care include:

- The need to encourage self-care and independence as much as possible.
- Information to enhance recovery (Synapse, 2013):
  - Get lots of rest. Don't rush back to work or university.
  - Avoid anything that could cause another blow or jolt to the head.
  - Consult with doctor about when it will be safe to drive a car, ride a bike or use heavy equipment. (Reaction time is often slower after a TBI.)
  - Take only prescribed drugs and don't drink alcohol.
  - Write things down if you are having problems remembering.
  - If the injury was severe, therapy may be needed to learn lost skills, such as speaking, walking or reading.
  - Safety issues.
  - Equipment needs, such as a mobility aids and hospital bed.
  - Vocational counselling and services.
  - Referral to community resources and support groups.
  - Helpful resources:
    - Brain Foundation: www.brainfoundation.org.au
    - Brain Injury Australia: www.braininjuryaustralia.org.au
    - Brain Research Institute: Australia: www.brain.org.au
    - Disability Services Australia: www.dsa.org.au.





TRANSLATION TO PRACTICE Evidence-based practice: caregivers of young adults with TBI

Traumatic brain injury (TBI) is a leading cause of death and disability across all age groups, but the age group most affected is young adults, especially young men. This is the result of increased risk-taking behaviour in this age group, including drinking alcohol and using drugs while operating a motor vehicle. This population of TBI survivors is large and caring for them is expensive. Most research of TBI survivors has focused on problems of the survivor, but few have examined the burden placed on the family. This is especially true for the experience of the survivors' mothers, who are in almost all cases the primary caregiver. A study was conducted by Kao and Stuifbergen (2004) to describe the meaning of the experience of the relationship between young adult TBI survivors and their mothers. Both the survivors and the mothers were interviewed and the data were analysed to describe the experience. The survivors described having a sense of being abnormal; the mothers struggled with a balance of protecting their children and letting them become independent. The mothers also struggled to maintain relationships inside and outside of the family. All these experiences resulted in ongoing stress for the mothers.

IMPLICATIONS FOR NURSING

The findings of this study have implications for community-based teaching and interventions for TBI survivors and their caregivers. Each year a significant number of TBI survivors leave the acute care setting and return to their homes, where caregivers are often unprepared for the lifelong multiple disabilities and problems they will face.

Developing an appropriate tool to measure family caregiver burden for those with disabilities from TBI is recommended, so as to be able to design and implement individualised interventions and teaching. The teaching and interventions should be developed as part of a systematic and integrated discharge plan, a community-based program and a follow-up approach to assist TBI survivors and their families to effectively decrease the stressors that are a part of caregiver load.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. You are making a home visit to a 22-year-old woman who has recently been discharged from acute care following a serious car crash resulting in mild diffuse axonal injury. She says to you, 'This is my third year of nursing at university. Will I be able to be a nurse?' How would you respond?
2. On discharge from the hospital, a nursing diagnosis for a young adult TBI survivor who would be cared for at home by his single mother was *Risk of parental role conflict related to caring for a child with TBI manifested by lack of independence*. Do you think this is an appropriate diagnosis? Why or why not?
3. If you were to outline topics for a program to reduce the incidence of TBI in a rural community, how would it differ from one for residents of a large urban city?

Source: Data from Love and load: The lived experience of the mother–child relationship among young adult traumatic brain-injured survivors by H. Kao & A. Stuifbergen (2004*). Journal of Neuroscience Nursing*, *36*(2), 73–81.

CENTRAL NERVOUS SYSTEM INFECTIONS

THE PERSON WITH A CENTRAL NERVOUS SYSTEM INFECTION

The central nervous system, including the meninges, neural tissues and blood vessels, may be directly affected by bacteria, viruses, fungi, protozoans and rickettsiae. The CNS may also be affected by toxins from bacterial infections. In general, organisms enter the brain in two ways: through the bloodstream by crossing the blood–brain barrier, or by direct invasion through a skull fracture or bullet hole. Very rarely, a CNS infection occurs as a result of contamination of a surgical field or lumbar puncture. The main CNS infections include meningitis, encephalitis and brain abscesses.

The incidence of pathogenic infections of the CNS increases with the onset of AIDS. People who are HIV positive may have CNS infections caused by toxoplasmosis, cryptococcus, tuberculosis, herpes simplex, cytomegalovirus (CMV) or a polyoma virus (resulting in progressive multifocal leucoencephalopathy).

Pathophysiology

When pathogens enter the CNS and the meninges, an inflammatory process results. The pathology of CNS infections includes the invading pathogens, the subsequent inflammation and the increase in intracranial pressure that may result from the inflammatory processes. Both the pathogenic damage and the IICP may result in brain damage and life-threatening complications.

Meningitis

Meningitis is an inflammation of the pia mater, the arachnoid and the subarachnoid space. Inflammation spreads rapidly throughout the CNS because of the circulation of CSF around the brain and spinal cord. Meningitis may be acute or chronic, and it may be bacterial, viral, fungal or parasitic in origin. Meningococcal meningitis may occur in epidemics among people who are in close contact with one another, such as members of the armed forces and students living in

residential colleges. Pneumococcal meningitis, in contrast, primarily affects the very young and very old.

The organism responsible for meningitis must overcome non-specific and specific host defence mechanisms to invade and replicate in the CSF. These defences include the skin barrier, the blood–brain barrier, the non-specific inflammatory response and the immune response. Host response to the particular pathogen is responsible for the manifestations of clinical meningitis. The organisms that initiate the host response in meningitis demonstrate an affinity for the nervous system. They colonise and invade the nasopharyngeal mucosa, survive intravascularly and penetrate the CNS if the blood–brain barrier is damaged, as can happen during surgery.

Infection of the CSF and meninges causes an inflammatory response in the pia, arachnoid and CSF. Because the meninges and subarachnoid space are continuous around the brain, spinal cord and optic nerves, the infection and inflammatory response are always cerebrospinal, involving both the brain and the spinal cord. Inflamed blood vessels in the area leak fluids as cell permeability increases. Purulent exudate infiltrates cranial nerve sheaths and blocks the choroid plexus and subarachnoid villi. IICP occurs as brain tissue responds to the pathogen. With an increase in ICP, cerebral perfusion decreases and cerebral autoregulation is lost.

BACTERIAL MENINGITIS The causative organisms of bacterial meningitis include *Neisseria meningitis*, meningococcus, *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Escherichia coli*. Risk factors include head trauma with a basal skull fracture, otitis media, mastoiditis, sinusitis, neurosurgery, systemic sepsis or immunocompromise (Grossman & Porth, 2013). Even when appropriate antibiotics are used, the mortality rate for adults remains at approximately 25%.

Once the pathogen enters the central nervous system, it or its toxic products (free radicals) initiate an inflammatory response in the meninges, CSF and ventricles. Meningeal vessels become engorged and their permeability increases. Phagocytic white blood cells migrate into the subarachnoid space, forming a purulent exudate that thickens and clouds the CSF and interferes with its flow. Rapid exudate formation causes further inflammation and oedema of meningeal cells. Blood vessel engorgement, exudate formation, impaired CSF flow and cellular oedema cause the intracranial pressure to increase.

Manifestations The person with bacterial meningitis typically presents with fever and chills, headache, back and abdominal pain, and nausea and vomiting. (The older adult may not have a high fever, but may instead exhibit confusion.) Meningeal irritation causes nuchal rigidity (stiff neck) and positive Brudzinski's sign (flexion of the neck that causes the hip and knee to flex) and positive Kernig's sign (inability to extend the knee while the hip is flexed at a 90-degree angle). Photophobia is present; the person may also experience diplopia. With meningococcal meningitis, a rapidly spreading petechial rash involving the skin and mucous membranes may be noted. The person may also have IICP, manifested by decreased LOC, seizures, changes in vital signs and respiratory pattern, and papilloedema. The manifestations of bacterial meningitis are listed in the box below.

MANIFESTATIONS Bacterial meningitis

- Restlessness, agitation and irritability
- Severe headache
- Signs of meningeal irritation:
  - Nuchal rigidity
  - Positive Brudzinski's sign
  - Positive Kernig's sign
- Chills and high fever
- Confusion, altered LOC
- Photophobia (aversion to light), diplopia
- Seizures
- Signs of increased ICP (widened pulse pressure and bradycardia, respiratory irregularity, decreased LOC, headache and vomiting)
- Petechial rash (in meningococcal meningitis)

Complications Complications of bacterial meningitis include arthritis, cranial nerve damage and hydrocephalus. Cranial nerve VIII, the auditory nerve, is frequently affected, with resulting nerve deafness. Thrombophlebitis may develop in cerebral vessels, with infarction of surrounding tissues (Grossman & Porth, 2013).

VIRAL MENINGITIS Acute viral meningitis, also called *aseptic meningitis*, is a less severe disease than bacterial meningitis. It can be caused by numerous viruses, such as herpes simplex, herpes zoster, Epstein–Barr virus or cytomegalovirus (CMV). Viral meningitis most often appears after a case of mumps. Although viral infection also triggers the inflammatory response, the course of the disease is benign and of short duration. Recovery is uneventful.

Manifestations The manifestations of viral meningitis are similar to those of bacterial meningitis, although usually milder. The person may have a mild flu-like illness prior to the onset of meningitis. Headache is intense and is accompanied by malaise, nausea, vomiting and lethargy. Photophobia may be present. The person generally remains oriented, although possibly drowsy. Temperature is mildly elevated. Neck stiffness, positive Brudzinski's sign and positive Kernig's sign are usually present.

Encephalitis

Encephalitis is an acute inflammation of the parenchyma of the brain or spinal cord. It is almost always caused by a virus, but it may also be caused by bacteria, fungi and other organisms. Other less common causes include ingested lead; post-vaccination encephalitis (from vaccines for measles, mumps and rabies) and HIV (Grossman & Porth, 2013). See Table 41.10 for a list of the most common causes of encephalitis.

VIRAL ENCEPHALITIS Viruses depend on living tissue for reproduction and become highly destructive when they invade brain tissue. The inflammatory response extends over the cerebral cortex, the white matter and the meninges, with

TABLE 41.10 Causes of encephalitis

CAUSE	COMMENTS
Arboviruses	Transmitted by bites from ticks and mosquitoes. Bites from ticks occur more frequently in spring. Bites from mosquitoes occur in middle to late summer. Most common type is the Ross River virus. May destroy major parts of the lobe or hemisphere. 20% of people who develop Murray Valley encephalitis die and 40% develop permanent neurological damage (e.g. seizures, blindness, deafness, speech disorders or mental retardation). Young Indigenous children in Western Australia have significantly poor outcomes. The incubation is 5 to 15 days. Mortality rates associated with arboviruses are higher than those associated with enteroviruses.
Enteroviruses, such as echovirus, coxsackievirus, poliovirus, paramyxovirus (the virus that causes mumps), and varicella zoster (the virus that causes chickenpox)	Infection occurs more frequently in summer (except infection by the mumps virus, which occurs more frequently in early winter). Some degree of protection can be afforded by immunisation against measles, mumps and poliomyelitis. Mortality rates are lower than those associated with herpes simplex type 1 virus.
Herpes simplex type 1 virus	Most common non-epidemic encephalitis in Australia. Can occur any time of year and throughout the world. Has an affinity for the frontal and temporal lobes. Prognosis is grave but not hopeless: mortality rate can be as high as 40% and the person may die within 2 weeks.
Amoebic meningoencephalitis due to infection by *Naegleria* and *Acanthamoeba* protozoa	Both protozoa are found in warm freshwater. Enter the nasal mucosa of people swimming in dams or lakes. May also be found in soil and decaying vegetation. Incidence of infection is increasing.
Exogenous poisoning	May occur after ingestion of lead or arsenic or inhalation of carbon monoxide.

degeneration of the neurons. The pathology of encephalitis includes local necrotising haemorrhage, which ultimately becomes generalised, with prominent oedema. There is progressive degeneration of nerve cell bodies. The inflammatory response in encephalitis does not cause exudate formation as it does in meningitis. Certain viruses show a propensity for specific areas of the brain (e.g. herpes simplex virus involves frontal and temporal lobes). The virus gains access to the CNS via the bloodstream or along peripheral or cranial nerves, or it may already be present in the meninges in the person with meningitis.

The manifestations of viral encephalitis vary, depending on the organism and area of the brain affected. Usual manifestations are similar to those of meningitis, including fever, headache, seizures, stiff neck and altered LOC. The person may be disoriented, agitated and restless, or lethargic and drowsy. As the disease progresses, the LOC deteriorates and the person may become comatose.

ARBOVIRUS ENCEPHALITIS The arboviruses are arthropod (mosquito or tick)-borne agents that infect humans. They include many different types, including dengue, Ross River virus, Barmah Forest virus, Japanese encephalitis, Murray Valley encephalitis and Kunjun. In Australia, adults are most often infected with Ross River virus, with older adults affected more often. The arthropods may live in small mammals and birds or may be carried by horses and deer.

The arthropod-borne agents cause widespread degeneration of nerve cells, and oedema and necrosis—with or without haemorrhage—occur. IICP may develop. Manifestations include fever, malaise, sore throat, nausea and vomiting, stiff neck, tremors, paralysis of extremities, exaggerated deep tendon reflexes, seizures and altered LOC.

Brain abscess

A brain abscess is an infection with a collection of purulent material within the brain tissue. Approximately 80% are found in the cerebrum and 20% are cerebellar.

The causes of a brain abscess include open trauma and neurosurgery; infections of the mastoid, middle ear, nasal cavity or nasal sinuses; metastatic spread from distant foci (such as heart, lungs, skin, abscessed teeth and dirty needles); and arising from other associated areas of infection. The immunocompromised are at increased risk of abscesses. The most common pathogens causing the abscess are streptococci, staphylococci and bacteroids. Yeast and fungi may also cause brain abscess.

A brain abscess results from the presence of microorganisms in the brain tissue. If the abscess is encapsulated, it has the ability to enlarge and, therefore, behave as a space-occupying lesion within the cranium. This predisposes the person not only to the systemic effects of the inflammatory process but also to the serious consequences of increased intracranial pressure. Occasionally, the abscess does not become encapsulated;

instead, it spreads through the brain tissue to the subarachnoid space and ventricular system.

Initially, the person exhibits the general symptoms associated with an acute infectious process, such as chills, fever, malaise and anorexia. Because brain abscess generally forms after infection, the person may consider these signs to be an exacerbation of that illness. The person may experience seizures, altered LOC and manifestations of IICP. As the abscess enlarges, specific symptoms are related to location; for example, the person with a frontal lobe abscess may have contralateral hemiparesis, expressive aphasia, focal seizures and frontal headache.

INTERPROFESSIONAL CARE

Bacterial meningitis is a medical emergency that, if not treated immediately, can be fatal within days. Successful management depends on rapid diagnosis and aggressive treatment with antibiotics and corticosteroids to eradicate the infecting organism and support vital functions. The person may be placed in strict or respiratory isolation until the organism has been identified, depending on hospital policy. Universal precautions apply to CSF as well as blood.

Treatment for viral meningitis focuses on managing symptoms and is supportive. Antipyretics and analgesics may provide relief. Antibiotic therapy is not indicated and isolation precautions are not required.

Treatment of the person with a brain abscess focuses on prompt initiation of antibiotic therapy. Other manifestations are treated symptomatically, as with the person diagnosed with meningitis or encephalitis. If pharmacological management is not effective, the abscess may be drained or, if it is encapsulated, removed.

Diagnosis

The diagnosis of meningitis is based on manifestations and diagnostic test results. Gram stain and culture of the CSF are used to determine if a bacterial infection is present and to determine the specific infectious agent. Counterimmunoelectrophoresis (CIE) is a laboratory test that may be ordered to determine the presence of viruses or protozoa. Polymerase chain reaction techniques may be used to detect viral DNA or RNA in spinal fluid. CT scan will show an area of increased contrast surrounding a low-density core with brain abscess.

Lumbar puncture with examination of the CSF is the definitive diagnostic measure for bacterial meningitis. Data that indicate bacterial meningitis include turbid, cloudy fluid; a markedly increased white blood cell (WBC) count and protein content; and a decreased glucose content. The opening pressure on the lumbar puncture is elevated. In contrast, the person with encephalitis may have a normal CSF analysis and pressure or may have some lymphocytes. The person with a brain abscess will have a markedly elevated pressure with elevated protein content and elevated WBC count. Glucose content is normal. (Because a lumbar puncture in the presence of a space-occupying lesion can result in brain herniation and death, a CT scan is performed first if neurological findings support such a lesion.)

Medications

Immediate intravenous administration of a broad-spectrum antibiotic that crosses the blood–brain barrier into the subarachnoid space is instituted in cases of bacterial meningitis. Once culture reports identify the causative organism, drug therapy is continued from 7 to 21 days, using the most effective drug or drugs specific to that bacterium. The cephalosporin antibiotics are preferred. A major concern in the treatment of CNS infections is penicillin-resistant streptococci. Recommendations for treatment are for a broad-spectrum cephalosporin, such as rifampicin (Rifadin), cefotaxime (Cefotaxime Sandoz) or vancomycin (Vancomycin Sandoz). However, as the bacteria are killed, the toxins they release increase production of inflammatory cytokines, which are potentially lethal. Steroids such as dexamethasone (Decadron) are often given with the antibiotics to suppress inflammation. The CDC recommends that the person remain on isolation for 24 hours after the start of antibiotic therapy.

Treatment for encephalitis consists of administering specific medications and preventing complications. Fungal meningitis is treated with antifungal agents, such as amphotericin-B (Amphotec), flucytosine (Ancontil) and fluconazole (Diflucan). Viral encephalitis is treated with intravenous aciclovir (aciclovir intravenous infusion).

Antibiotic therapy is the primary treatment for brain abscess. A combination of broad-spectrum antibiotics is used if the infecting organism is unknown.

Anticonvulsant medications such as phenytoin (Dilantin) are often prescribed to prevent or control seizure activity. Antipyretic and analgesic medications may provide symptomatic relief; however, analgesics that have a depressant effect on the CNS (such as opiates) are avoided to prevent masking of early manifestations of deteriorating LOC. The person initially may require anti-emetics to control nausea and vomiting. Fluid and electrolyte status is maintained through intravenous fluid replacement until the person is able to resume oral intake.

Surgery

Surgical drainage of an encapsulated abscess may be necessary. The decision to perform surgery is based on the person's general condition, the stage of abscess development and the site of the abscess.

Nursing care

Central nervous system infections are serious illnesses, with potentially life-threatening effects and complications. Nursing assessments and interventions are critical in identifying changes in the person's neurological status and preventing complications from IICP. In addition to the nursing care described in this section, a nursing care plan for a person with bacterial meningitis follows.

Health promotion

As with many other intracranial injuries and disorders, educational activities to promote health by preventing CNS infections are important nursing interventions. The following information should be provided:

- Vaccinations for meningococcal meningitis are recommended or required for members of the armed forces and tertiary students (groups at increased risk of invasive meningococcal meningitis).
- Administration of prophylactic rifampicin (Rifadin) is recommended for people exposed to meningococcal meningitis.
- Mosquito control with repellents, insecticides and protective clothing.
- Destruction of the insect larvae and elimination of breeding places, such as pools of stagnant water.
- Vaccination against Japanese B encephalitis (recommended for summer travellers to rural East Asia).
- Prompt diagnosis and treatment of infections of the head, neck and respiratory system.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40). Further focused assessments are described with nursing interventions below.

NURSING CARE PLAN A person with bacterial meningitis

Monty Cook is a 22-year-old musician who plays in a local rock band. He is unmarried and lives with his parents. He is known by everyone in the community as a quiet, low-key, easygoing person and an excellent guitar player. During a performance 2 days ago at the local pub, he had difficulty playing his guitar, complaining of bright stage lights blazing in his eyes. When he tried to keep his head down to prevent the lights from hurting his eyes, he noticed his neck was very stiff. After the performance, one of the newest members of the band remarked that it certainly was not their best performance. Monty responded angrily that maybe the new members of the group needed more practice. Then he stomped out and went home to bed.

He wakes at 4 am with a severe headache, sweating and chills; his temperature is 38.9°C and he cannot bend his neck without severe pain. His mother recognises that he is agitated and irritable, which is uncharacteristic. Frightened, she rushes him to the hospital emergency room. A lumbar puncture performed in the emergency room reveals turbid, cloudy fluid; a markedly increased WBC count; and protein with a decreased glucose content. Bacterial meningitis is the medical diagnosis. Mr Cook is admitted to the hospital for treatment and care.

ASSESSMENT

When the nurse, Aisha Aldi, enters Mr Cook's isolation room, she sees him thrashing about in the bed, talking incoherently and becoming more agitated. On assessment, Ms Aldi notes dry mucous membranes, cracked lips and small petechiae over the upper torso and abdomen. Mr Cook's temperature is 40°C. Kernig's sign is positive. Intravenous broad-spectrum antibiotics are prescribed and initiated. After the first 2 hours of care, Ms Aldi notes a decrease in Mr Cook's level of consciousness.

DIAGNOSES

- *Hyperthermia* related to infection and abnormal temperature regulation by hypothalamus, evidenced by an increased body temperature.
- *Disturbed thought processes* related to intracranial infection, evidenced by confusion, agitation and uncharacteristic behaviours.
- *Risk of injury* related to progression of illness manifested by increased confusion and agitation.

PLANNING

Monitor the person's condition, provide appropriate nursing care and maintain the person's safety.

Expected outcomes

- Have a decrease in body temperature.
- Become less restless and agitated.
- Remain free of injury.

IMPLEMENTATION

- Monitor vital signs every 2 hours.
- Provide sponge baths if temperature continues to rise.
- Provide a quiet, non-stimulating environment with the shades drawn.
- Provide oral care every 4 hours.
- Measure and compare intake and output every 2 hours.
- Perform neurological assessments every 2 to 4 hours.
- Monitor for and report seizure activity and decreasing level of consciousness.
- Keep bed in low position with side rails elevated.
- Administer prescribed intravenous antibiotics.

EVALUATION

After 4 days of antibiotic therapy, Mr Cook's temperature has returned to near normal. Ms Aldi notes that he has begun opening his eyes and visually tracking her as she moves about the room. Mr Cook responds to a request to squeeze Ms Aldi's fingers and after several hours asks her what had happened. On day 5, Mr Cook states that he feels better and his headache is gone. He asks for sips of juice and begins urinating regularly. Seven days after admission, Mr Cook is discharged and is able to go home with his mother. He has some weakness in his legs, but otherwise has no evidence of neurological deficits.

CRITICAL THINKING IN THE NURSING PROCESS

1. Which strategies should the nurse use to decrease the environmental stimuli for Mr Cook and what is the rationale for doing these?
2. If you were caring for Mr Cook in the initial phase of the illness and he became combative, what would you do?

REFLECTION ON THE NURSING PROCESS

1. Develop a plan of care for the nursing diagnosis of *Acute pain* related to meningitis manifested by headaches. Consider the effect of narcotics on respiratory function in designing the plan.
2. Outline what you have learned from this case study that you will apply to your future nursing practice.

- *Health history:* risk factors (concurrent infections, other illnesses, travel), when manifestations began, severity of manifestations, current nausea and headache, seizures.
- *Physical examination:* Glasgow Coma Scale, level of consciousness, vital signs, motor function, pupillary check, cranial nerves, neck ROM, Brudzinski's sign, Kernig's sign, skin (rash, petechiae, purpura), muscle movement and strength, speech.

Nursing diagnoses and interventions

In planning and implementing nursing care for the person with a CNS infection, the prognosis may depend on the supportive care given. The person is often very ill and the combination of fever, dehydration and cerebral oedema may predispose the person to seizures. Airway obstruction, respiratory arrest or cardiac arrhythmias may occur. Nursing diagnoses and interventions previously discussed for the person with an altered LOC, IICP and seizures are also appropriate for the person with a CNS infection. Nursing interventions in this section focus on *Ineffective protection* and *Risk of fluid volume deficit*.

Ineffective protection

People with CNS infections are less able to protect themselves against insults from both internal and external sources. The effects of the inflammation and resulting pathophysiological processes may include pain, fever, altered LOC, seizures, IICP and cranial nerve dysfunction. In addition, pathophysiological effects on the brain from toxins or thrombosis of a cerebral vessel may lead to permanent neurological deficits, such as loss of motor function or dementia.

- Monitor neurological status on a regular basis. *Many complications are evidenced by changes in neurological manifestations.*
- Monitor vital signs, including temperature, on a regular basis. *The person often has a high temperature throughout the illness, ranging from 38°C to 40.5°C.*
- Monitor levels of consciousness. Assess levels of orientation, memory, attention span and response to stimuli. *Early in the infection, the person often has problems with memory and orientation. There may be problems with following commands, restlessness, irritability and combativeness. As the illness progresses, the LOC decreases to lethargy and finally into deep coma.*

CONSIDERATION FOR PRACTICE

Hyperthermia may result from increased intracranial pressure, while an increased temperature can also increase ICP.

- Monitor for manifestations of seizure activity and institute seizure precautions:
  - Monitor twitching of hands or face and tonic–clonic movements.
  - Have an oral airway and suction equipment readily available.
  - Pad side rails, maintain bed in low position and keep side rails up.

  Irritation of the cerebral cortex secondary to meningeal inflammation may cause seizures. Careful monitoring and seizure precautions are necessary to prevent injury.
- Monitor for manifestations of cranial nerve damage; monitor extraocular movements, facial movement, dizziness, ability to hear, double vision, drooping upper eyelids (ptosis) and pupillary changes. *Cranial nerve dysfunction may result from inflammation or vascular changes in the brain.*
- Monitor for manifestations of increased intracranial pressure: decreased pulse, increased blood pressure, widening pulse pressure, respiratory changes and vomiting. *IICP from infectious or inflammatory exudate, cerebral oedema and hydrocephalus.*
- Administer prescribed medications and maintain prescribed fluid restrictions. *Diuretics are often prescribed to prevent increases in ICP, anticonvulsants are prescribed to prevent or control seizures, and antibiotics are prescribed to eradicate the bacteria. Fluids may be restricted to help prevent IICP.*

Risk of fluid volume deficit

The person is at risk of fluid volume deficit related to increased metabolic rate, diaphoresis and fluid restrictions.

- Monitor for presence or worsening of fluid volume deficit.
- Measure and compare intake and output every 2 to 4 hours.
- Monitor daily body weights.
- Monitor skin turgor.
- Monitor condition of mucous membranes.
- Monitor urine amount, colour and odour.
- Monitor BUN:creatinine ratio.

The elastic property of the skin depends partially on interstitial fluid volume. If there is a fluid volume deficit, skin flattens more slowly after a pinch is released. Mucous membranes are dry. In fluid volume deficit, urine output is decreased, urine is dark in colour and concentrated with a strong odour, and urine specific gravity is greater than 1.020 and BUN will rise out of proportion to serum creatinine.

CONSIDERATION FOR PRACTICE

A weight loss of 0.5 kg represents a fluid loss of approximately 500 mL.

- When administering fluids, either orally or parenterally, consider concurrent illnesses. *People with IICP or kidney failure require complex management.* See Chapter 9 for a further discussion of fluid volume deficit.

Community-based care

The importance of preventive measures, such as recognising predisposing conditions, is a major focus for education. People who have had close contact with the person with meningitis should be monitored for fever, headache or neck stiffness. Some doctors believe that those closest to the person are

candidates for antimicrobial prophylaxis. Also address the following topics:

- the need to report any manifestations of ear infection, sore throat or upper respiratory infection
- the names and purposes of all medications that may be prescribed
- the importance of taking all medication until completely gone, because some people may think it is acceptable to stop the medication as soon as they feel better.

TUMOURS OF THE BRAIN

THE PERSON WITH A BRAIN TUMOUR

Brain tumours are growths within the cranium, including tumours in brain tissue, meninges, the pituitary gland or blood vessels. Brain tumours may be benign or malignant, primary or metastatic, and intracerebral or extracerebral. Regardless of type or location, brain tumours are potentially lethal as they grow within a closed cranial vault and displace or impinge on CNS structures.

Incidence and prevalence

An estimated 1400 new cases of malignant brain tumours are diagnosed annually in Australia and approximately 1200 people die each year (Brain Foundation, 2015). Although brain tumours can occur in any age group, the highest incidence in adults is in those aged 50 to 70. In the adult population, the most common tumour is glioblastoma multiforme, followed by meningioma and cytoma. Glioblastomas represent more than 50% of all primary intracranial lesions.

The cause of many brain tumours is unknown. Although a number of chemical and viral agents can cause brain tumours in laboratory animals, there is no evidence that these agents cause tumours in humans. Other factors associated with brain tumours include heredity, cranial irradiation and exposure to some chemicals (Grossman & Porth, 2013).

Pathophysiology

Brain tumours may be classified as benign or malignant, based on the tissue type and characteristics of the cells. The use of the term *benign* may be misleading. A tumour that is benign by histological examination but is surgically inaccessible may continue to grow and expand, increasing intracranial pressure and causing neurological deficits, herniation and finally death. In discussions of brain tumours, the term *malignant* is used to describe the lack of cell differentiation, the invasive nature of the tumour and its ability to metastasise.

Brain tumours also may be classified as primary or metastatic, depending on their origin (see Table 41.11). Primary tumours of CNS tissue arise from the cells and structures that are found within the brain; for example, neurons and neuroglia. The primary intracranial tumours that originate in the skull cavity but not from brain tissue itself arise from the supporting structures, including the meninges, pituitary gland and pineal gland. Primary brain tumours rarely metastasise outside the CNS. Metastatic brain tumours originate from structures outside the brain, such as the breasts, lungs and prostate gland.

Focal disturbances take place when there is compression of brain tissue and infiltration or direct invasion of brain parenchyma with destruction of neural tissue. As the tumour grows, oedema develops in adjacent tissues. The mechanism is not completely understood, but it is thought that an osmotic gradient causes the tumour to absorb fluid. Some tumours may cause haemorrhage. Venous obstruction and oedema due to breakdown of the blood–brain barrier increase intracranial volume and intracranial pressure. Obstruction of the circulation of CSF from the lateral ventricles to the subarachnoid space causes hydrocephalus.

An estimated 25% of people with cancer develop brain metastasis. Metastatic brain tumours present in the same way as primary brain tumours, with IICP and focal and/or diffuse cerebral dysfunction. The most common source of intracranial metastasis is cancer of the lung. Other common primary sites are the breast, kidney and gastrointestinal tract. The metastasis reaches the brain through the circulation. In most cases, the tumours are multiple and are scattered through the cerebellum and cerebrum.

Manifestations

Multiple manifestations can develop as a result of the growth of the tumour, while others are related to the location of the lesion. Some of the more common manifestations include changes in cognition or consciousness, a headache that is usually worse in the morning, seizures and vomiting. Compression of brain tissue and the invasion of the brain tumour into the cerebral tissue may lead to changes typically seen with cerebral oedema and IICP. Cerebral blood supply may diminish as the tumour compresses blood vessels. Shifts in brain tissue can occur, leading to brain herniation syndromes and, if untreated, death. See the ‘Manifestations’ box below.

INTERPROFESSIONAL CARE

Treatment for a brain tumour may involve chemotherapy, radiation therapy, surgery or any combination of these. Several variables are considered when selecting the appropriate treatment modality: the size and location of the tumour, the type of tumour, related symptoms (such as neurological deficits) and the person’s overall condition.

TABLE 41.11 Classification of brain tumours

TUMOUR TYPE	TUMOUR NAME	CHARACTERISTICS
Primary tumours:		
Intracerebral tumours: Account for 40–50% of all brain tumours Originate from neuroglia and invade brain tissue Most common type of brain tumour	Glioma • Astrocytoma • Glioblastoma multiforme • Ependymoma • Oligodendroglioma • Astroblastoma	 Most common glioma Graded I to IV according to degree of cell differentiation Most malignant form Fast-growing tumour that develops from lining of ventricles Graded I to IV according to degree of cell differentiation Slow growing Rare, slow growing May be encapsulated Benign
Extracerebral tumours: Tumours arising from the supporting structures of the nervous system Account for 10–15% of all brain tumours	Medulloblastoma Meningioma Acoustic neuroma	Fast growing and malignant Occurs primarily in children; can occur in adults Found in cerebellum Slow growing Develops in meninges (especially dura) Firm and encapsulated Slow growing Benign Originates from Schwann cells of the cranial nerve XIII May also affect cranial nerves V, VII, IX and X Also called neurofibromatosis Genetic origin due to autosomal dominant Mendelian trait Firm, encapsulated lesions attached to nerve
Congenital (developmental) tumours: Account for 4–8% of all brain tumours	Haemangioblastoma Craniopharyngioma	Vascular tumour Slow growing Originates from Rathke's pouch Solid or cystic tumour Compresses pituitary gland Presses on the third ventricle and may cause blockage of cerebrospinal fluid (CSF)
Pituitary adenomas: Account for 8–12% of all brain tumours	Chromophobic Eosinophilic Basophilic	Account for 90% of pituitary tumours Non-secreting tumour Slow growing Secreting tumours that produce growth hormone Secreting tumours that produce adrenocorticotropic hormone Fast growing
Secondary tumours		
Metastatic brain tumours: Account for 10% of all brain tumours		Slow-growing tumours that arise from other parts of the body Usually well differentiated from the brain Spread from tumours of the lung, breast, lower gastrointestinal tract, pancreas, kidney, skin

MANIFESTATIONS Brain tumours

FRONTAL LOBE TUMOURS
- Inappropriate behaviour
- Personality changes
- Inability to concentrate
- Impaired judgment
- Recent memory loss
- Headache
- Expressive aphasia
- Motor dysfunctions

PARIETAL LOBE TUMOURS
- Sensory deficits: paraesthesia, loss of two-point discrimination, visual field deficits

TEMPORAL LOBE TUMOURS
- Psychomotor seizures

OCCIPITAL LOBE TUMOURS
- Visual disturbances

CEREBELLUM TUMOURS
- Disturbances in coordination and equilibrium

PITUITARY TUMOURS
- Endocrine dysfunction
- Visual deficits
- Headache

Diagnosis

The following diagnostic tests may be ordered.

- A CT scan or an MRI with gadolinium enhancement can locate the tumour and define its size, shape, extent to which normal anatomy is distorted and the degree of any associated cerebral oedema.
- Arteriography may show stretching or displacement of cerebral vessels by the tumour, as well as the presence of tumour vascularity.
- EEG provides information about cerebral function, may demonstrate focal or diffuse changes, and is useful if seizures are present.
- Endocrine studies are conducted if a pituitary tumour is suspected.

Medications

The choice of drug for treatment is based on the type of tumour, its location and the person's response to therapy. The use of chemotherapy to treat brain tumours is still emerging. An intraventricular method of medication administration uses an Ommaya reservoir that has been surgically implanted into a lateral ventricle of the brain (see Figure 41.8). Other medications that may be prescribed include corticosteroids and anticonvulsants.

Surgery

Surgery is used to remove tumours, to reduce the size of the tumour or for symptom relief (palliation). The type of procedure, the surgical approach and the timing of surgery (emergency versus planned procedure) influence the overall nursing management of the person having intracranial surgery.

Some of the more common intracranial neurosurgical procedures follow:

- *Burr hole:* a hole made in the skull with a special drill. The hole may facilitate the evacuation of an extracerebral clot or a series of holes may be made in preparation for craniotomy (see Figure 41.7).
- *Craniotomy:* a surgical opening into the cranial cavity (see Figure 41.9). For a craniotomy, a series of burr holes are made. The bone between the holes is then cut with a special saw called a craniotome. The tumour is excised and the bone flap is returned to the opening. A craniotomy may also be performed to repair defects associated with traumatic head injuries or to repair a cerebral aneurysm.
- A supratentorial craniotomy refers to surgery above the tentorium. It provides access to the frontal, temporal, parietal and occipital lobes. The incision for this procedure is usually within the hairline over the area involved.
- An infratentorial craniotomy refers to surgery below the tentorium. Access is provided to lesions in the cerebellum and the brainstem. The incision is made at the nape of the neck, around the occipital lobe.
- *Craniectomy:* an excision of a portion of the skull and complete removal of the bone flap. This procedure may be done to provide decompression after cerebral oedema. Pressure on the brain structures is lessened by providing space for expansion.
- *Cranioplasty:* plastic repair to the skull in which synthetic material is inserted to replace the cranial bone that was removed. This procedure may be performed after a large craniectomy. The plastic repair restores the contour and integrity of the cranium.

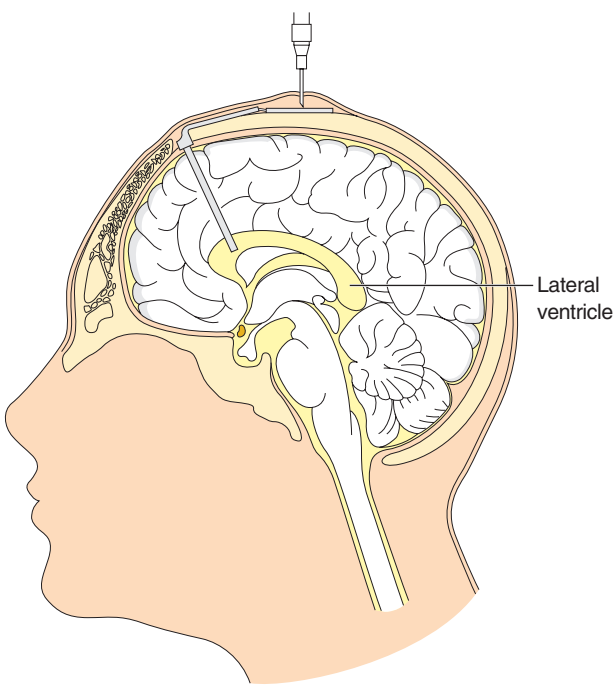


FIGURE 41.8 ■ Ommaya reservoir for medication administration

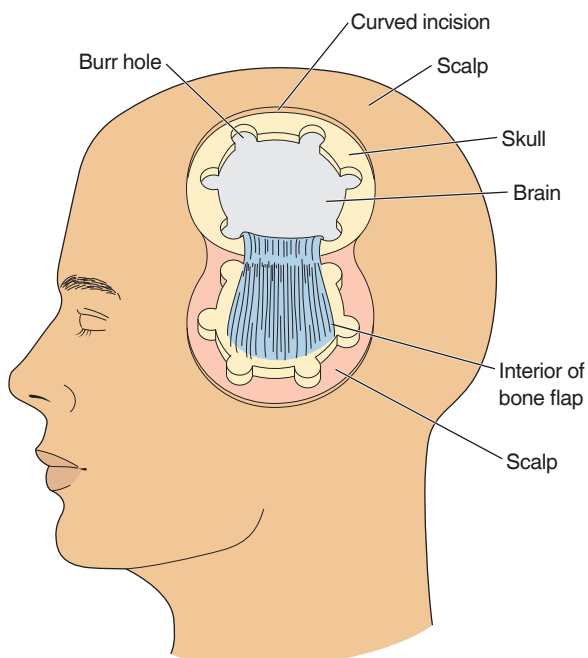


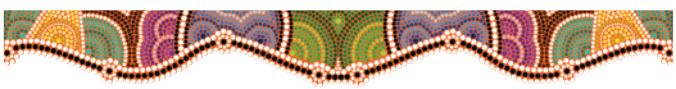
FIGURE 41.9 ■ In a craniotomy, a portion of the skull and overlying scalp is removed to allow access to the brain

Radiation therapy

Radiation therapy may be administered alone or as adjunctive therapy with surgery. Radiation is often the treatment of choice for surgically inaccessible tumours; it may also be used to decrease the size of a tumour prior to surgery. Tumours that were not completely excised by surgery may also be treated with radiation.

Specialty procedures

Technological advances—including the development of special instruments, the use of stereotaxic techniques for localising a specific target and the use of the laser beam—have greatly advanced neurosurgical practice. Microsurgery involves an operating microscope with microinstruments and supportive illumination equipment. Using stereotaxic techniques to precisely locate a specific target point allows for location of discrete areas of the brain that control specific functions and exact locations of deep brain lesions. The use of a laser beam for excision of a tumour results in less damage to surrounding tissue and less postoperative swelling. The gamma knife, which is not actually a knife but a gamma unit, consists of a heavily shielded helmet containing 201 sources of cobalt-60, which is capable of destroying deep and otherwise inaccessible lesions in a single treatment session. A new area of chemotherapy is that of biodegradable anhydrous wafers, which are impregnated with the chemotherapy drug and implanted into the tumour at the time of surgery. The wafers are made so that they slowly release the drug over a period of many months (Grossman & Porth, 2013).



Nursing care

The nursing care of the person with a brain tumour includes support during the diagnostic period and specific management as directed by the selected treatment. The foundation for care is data from the health history and physical assessment, which includes identifying neurological deficits. This information directs planning and implementing care. Many of the alterations in health commonly experienced by the person with a brain tumour have been discussed throughout this chapter, including altered level of consciousness, increased intracranial pressure and seizures. The person will require intensive care in the immediate postoperative period. In addition to the nursing care described in this section, a nursing care plan for a person with a brain tumour follows.

Nursing diagnoses and interventions

This section of the chapter focuses on nursing interventions for the person who has intracranial surgery. The nursing diagnoses discussed are *Anxiety*, *Risk of infection*, *Ineffective protection*, *Acute pain* and *Low self-esteem*.

Anxiety

The diagnosis of a brain tumour brings anxiety and feelings of uncertainty about the future. Both the person and family members are likely to be apprehensive and require education and emotional support.

- Explain routine medical procedures, including blood work and radiological studies. *Baseline laboratory and radiological studies are needed to ensure that the person has no other pre-existing medical condition. Explaining the procedures and assisting the person through this process help decrease anxiety.*
- Reinforce, clarify and repeat information. *Both the person and their family may have limited understanding of the scheduled diagnostic tests, procedures and treatment modalities. The person may be confused or have altered thought processes as a result of the tumour. Information may need to be repeated or re-explained.*
- Encourage the person and their family to verbalise feelings, questions and fears; provide realistic information appropriate to their level of understanding. *Verbalisation helps reduce anxiety and fear.*
- Review the person and their family's strengths and effective coping skills. *Personal strengths, support systems and coping skills can aid in the development of appropriate strategies to reduce anxiety.*
- Arrange for a member of the clergy to visit if desired. *Faith in a higher being is often a strong source of strength and support.*
- Provide preoperative teaching, including the following information:
  - type of anaesthesia and surgery
  - time surgery will begin and expected length of procedure and recovery room stay
  - where the person will be taken after surgery (ICU) (if possible, show the person and their family the ICU and introduce them to the nurse who will be in charge of care after the surgery)
  - where the family can wait during and following surgery
  - appearance of the person after surgery, which may include swollen, bruised eyelids and other facial features; a large dressing covering the head; a partially or fully shaved head; and a tracheostomy or endotracheal tube
  - behaviour of the person after surgery, which will differ depending on the site of surgery, although cognitive and behavioural changes are common.

  Information about what to expect reduces anxiety.
- Allow time for the person and their family to be together. *People need quiet time together with their families to support each other and prepare emotionally for surgery.*

Risk of infection

The person who has had intracranial surgery is at risk of infection from multiple invasive lines, the scalp wound and the risk of introduction of bacteria into the operative area. The nurse provides interventions to monitor for and prevent infection.

- Monitor for leakage of CSF:
  - presence of glucose in clear drainage from ears, nose or wound
  - complaints of 'something dripping down the back of the throat'
  - constant swallowing.

  These manifestations indicate an opening in the dura, which provides an avenue for an ascending infection.

- Provide interventions to prevent contamination of area leaking CSF:
  - If leaking from the nose: keep head of bed elevated 20 degrees unless contraindicated; do not suction nasally; do not clean nose; tell person not to put finger in nose; do not insert packing.
  - If leaking from the ear: position person on side of leakage unless contraindicated; do not clean ear; tell person not to put finger in ear; do not insert packing.
  - Place a sterile dressing over the area of drainage and change as soon as it becomes damp.

  Leakage of CSF indicates a break in the dura and increases the risk of an ascending infection. Surgery may be necessary to repair the break; however, the leak usually heals spontaneously in about 1 week.
- Monitor for and report manifestations of infection:
  - Take and record temperature on a regular basis.
  - Assess IV insertion sites for redness, swelling, drainage and pain.
  - Assess scalp wound for redness, swelling, bulging, drainage and pain.
  - Assess for manifestations of meningitis: fever and chills, increasing headache, neck stiffness, positive Kernig's or Brudzinski's sign, photophobia.
  - Monitor laboratory reports for increased WBC count.

  Intact skin is the first line of defence against infection. Any break in the skin increases the risk of infection. Intracranial surgery increases the risk of meningitis, with infectious agents ascending into the brain.
- Implement interventions to prevent infection:
  - Use strict aseptic technique when changing dressings and when caring for wound drains and ICP monitor lines.
  - Keep the person's hands away from drains and dressings; use mitten restraints if necessary.
  - Administer prescribed antibiotics.

  Sterile technique decreases the risk of introducing infection into a wound. Antibiotics are usually prescribed prophylactically to prevent infection.

Ineffective protection

The person who has intracranial surgery does not have normal human defences against changes in intracranial pressure and is also at risk from cerebral oedema and a shift of intracerebral contents. In addition, the surgery may cause cerebral bleeding or haematoma formation.

- Monitor for manifestations of increased intracranial pressure:
  - restlessness, agitation and decreasing LOC
  - headache
  - vomiting
  - seizures
  - decreasing sensory and motor function
  - changes in pupil size and reaction
  - changes in vital signs: altered respiratory rate or depth, increasing pulse pressure, decreasing pulse rate, increasing blood pressure
  - abnormal posturing.

  Increasing intracranial pressure is manifested by alterations in the functions and centres controlled by the brain.
- Implement interventions to decrease the risk of IICP:
  - Elevate the head of the bed 15 to 30 degrees as prescribed (unless contraindicated).
  - Avoid neck flexion or rotation; keep head in midline position unless a large bone flap or mass was removed; then position the person on unoperated side to decrease venous congestion in the operative area.
  - Do not take rectal temperatures.
  - Avoid clustering activities that increase intracranial pressure: suctioning, turning and bathing.
  - Administer medications to prevent vomiting.
  - Do not suction for more than 10 seconds at one time.
  - Teach the person (if possible) to avoid coughing, sneezing and straining to have a bowel movement.
  - Maintain fluid restrictions as prescribed.
  - For internal shunts: avoid pressure on the shunt, reservoir or tubing. Pump the shunt only if prescribed.
  - For external shunts: avoid kinks in tubing and maintain the drainage collecting device and the person's head at the prescribed levels.

  Keeping the head of the bed slightly elevated facilitates venous drainage from the brain. Neck flexion or rotation disrupts circulation to and from the brain. Rectal stimulation, suctioning, turning, bathing, coughing, sneezing and straining to have a bowel movement all initiate the Valsalva manoeuvre, which constricts the jugular veins and impairs venous return from the brain. Fluid restriction may be prescribed to dehydrate the person slightly and lessen ICP.
- Maintain (as much as possible) a quiet, calm, softly lighted environment. *Avoid excessive sensory stimulation. These interventions promote rest and decrease stimulation, thereby reducing ICP.*
- Implement interventions to prevent seizures or, if they occur, to prevent injury to the person:
  - Pad side rails of the bed.
  - Place bed in lowest position and keep side rails up.
  - Carry out interventions to prevent and treat IICP.
  - Have an oral airway (Guedel's) and suction equipment immediately available.
  - Administer prescribed anticonvulsants.
  - If a seizure occurs: maintain a patent airway; do not restrain the person; do not force anything into the person's mouth; provide physical and emotional support.

  These interventions promote safety and help prevent injury. Anticonvulsants are often prescribed prophylactically to prevent seizures after intracranial surgery.
- Carefully monitor hydration status. Compare trends in intake and output, laboratory results of serum osmolality and urine specific gravity and osmolality. *Changes in*

NURSING CARE PLAN A person with a brain tumour

Claire Lange is a 44-year-old morning television presenter. During a midweek program, she confuses several major news items so badly that her co-anchor tries to correct her. Ms Lange responds angrily that she does not need any help and then rises and storms off the set. As she leaves the camera area, she limps noticeably and appears to drag her left leg. The show's producer asks her what is wrong; she screams that nothing is wrong—she simply has another headache. He follows her to her dressing room and enquires about her headaches. She tells him that they come and go but have been getting worse lately. He then asks her if she has injured her left leg; she responds that the leg was weak because she was tired. As the producer leaves the dressing room, Ms Lange begins to shake and collapses on the floor. The producer recognises that she is having a seizure and calls for an ambulance.

Ms Lange is admitted to the neurology ward of the local hospital for evaluation. A CT scan, MRI and EEG are completed and identify an intracranial mass. A biopsy of the mass is positive for malignant cells. A glioma in the frontal lobe is identified and surgery is scheduled for that week.

ASSESSMENT

When Clara Rosetti, RN, enters Ms Lange's room, she sees Ms Lange looking at her shoulder-length hair in the mirror. Ms Lange tells Ms Rosetti that she has never in her life worn her hair any shorter and 'Now you're going to cut it all off!' She paces the room and makes the statement, 'I guess the hair isn't really important if I survive this situation.' She also says that she has a headache.

DIAGNOSES

- *Acute pain* related to tumour and increase in intracranial pressure manifested by the onset of headaches.
- *Disturbed body image* related to surgery manifested by anxiety surrounding potential hair loss and appearance postoperatively.
- *Anxiety* related to unknown future following surgery, evidenced by emotional distress.

PLANNING

- Reduce anxiety and fear and manage pain effectively.

Expected outcomes

- Verbalise the causes of pain.
- Verbalise an understanding of the changes in body appearance that are associated with the scheduled intracranial surgery (e.g. shaving of the head prior to surgery, cranial incision and facial swelling postoperatively).
- Identify measures that will help minimise the effect of the hair loss.
- Verbalise a reduction in anxiety.

IMPLEMENTATION

- Assess level of discomfort using a rating scale of 0 to 10.
- Provide a quiet, non-stimulating environment.
- Position the person for comfort, keeping the head of the bed elevated to promote venous drainage.
- Assess level of consciousness for potential increases in ICP.
- Encourage to verbalise feelings about the surgery.
- Suggest measures that may help minimise the hair loss, such as the use of turbans, scarves, hats and wigs.
- Suggest relaxation techniques to decrease anxiety.

EVALUATION

By the time of surgery, Ms Lange has recognised the relationship between the brain tumour and the headache. She states that lying in a flat position and coughing increase the headache. The head of the bed is kept at a 30- to 45-degree angle. Daily activities are spaced to provide periods of rest. Ms Lange demonstrates no significant changes in level of consciousness. She has talked about the effect of the hair loss and her television responsibilities. Ms Lange has learned that the hair preparation would be done in surgery and that the hair would be saved for her. She states she has already consulted her hairdresser and that 'scarves and turbans are on the way'.

CRITICAL THINKING IN THE NURSING PROCESS

1. Outline interventions to decrease intracranial pressure both before and after surgery.
2. When making your initial assessments on the morning of surgery, you find that Ms Lange has a decreased pulse and increased blood pressure. She tells you her headache is worse and suddenly vomits. What do you do now?
3. Ms Lange asks you to be sure that she has absolutely no visitors after surgery because she knows how ugly she will look. How would you respond?

REFLECTION ON THE NURSING PROCESS

1. Which communication and education strategies could you use when caring for people undergoing neurosurgery?
2. Outline what you have learned from this case study that you will apply to your future nursing practice.

fluid balance and osmolality may result from excess intravenous fluids, osmotic diuretics, surgically induced diabetes insipidus or syndrome of inappropriate antidiuretic hormone secretion, fever, diarrhoea, tube feedings or hyperglycaemia.

Acute pain

The person who has intracranial surgery has pain, manifested as a headache, as a result of either compression or displacement of brain tissue or from increased intracranial pressure. A headache may also be a manifestation of meningitis.

- Assess the location, duration and intensity of the pain, using a scale from 0 (no pain) to 10 (worst pain) in the person who can verbally communicate. *The person is the best source of information about pain.*
- Implement interventions to reduce the pain:
  - Raise the head of the bed slightly.
  - Reduce noise and bright lights in the room.

- If allowed, loosen head dressing.
- Administer narcotic analgesics with caution.

Non-pharmacological measures may be used to reduce IICP and headache.

CONSIDERATION FOR PRACTICE

Narcotic analgesics mask changes in eye signs and depress respirations.

Situational low self-esteem

The person who has intracranial surgery has many alterations that affect self-esteem and body image. Physical changes include a loss of hair on the scalp, swelling and bruising in the eyelids and face, and perhaps an indentation in the skull. The person is no longer independent in self-care, but must depend on others to meet basic needs. There are often long-term neurological deficits, affecting areas such as speech, vision and motor abilities, which require changes in roles and relationships.

- Assess for verbal and non-verbal manifestations of negative self-esteem:
  - denial of changes
  - preoccupation with changes
  - refusal to look in the mirror
  - withdrawal from family and friends
  - expressions of grief and loss (see Chapter 4).

  Low self-esteem can be initiated by stressful situations and changes in body image.
- Provide interventions to improve self-concept:
  - Limit negative self-assessment.
  - Help focus on positive areas of life.
  - Help identify sources of support and strength.
  - Help identify and use helpful coping methods.
  - Encourage significant others to visit.
  - Encourage independence in self-care.

  Self-esteem is derived from one's own perceptions of competence and from the responses of others. When one's self-concept and self-ideal are congruent, self-esteem is enhanced.

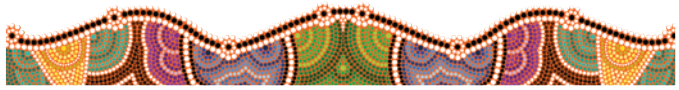
Community-based care

The effect of the possible outcomes following the surgery produces fear in both the person and their family, interfering with their ability to retain information. The person may have cognitive or neurological deficits that interfere with learning. Family members also must be assessed for their ability to cope with the stress of the surgery. Information may have to be repeated several times.

People and their families who have experienced intracranial surgery require emotional support. The process of recovery is often extended and may involve adaptation to changes in body image and management of any motor or sensory deficits. The family should be involved in the care of the person. If family members are willing, they may begin to assist with ADLs while the person is in the hospital, such as assisting with personal hygiene and meals. People should also be encouraged to take an active role in their own care. Discharge planning includes a discussion of the following topics: medication information; wound care; the use of wigs, turbans, hats or colourful scarves; and the importance of follow-up visits. In addition, emphasise the importance of reporting manifestations such as stiff neck, increasing headache, elevated temperature, new motor or sensory deficits, vision changes or seizures.

Provide information about the overall treatment plan, management of deficits and/or disabilities, and future needs. Specific teaching topics are as follows:

- safety measures for motor deficits, sensory deficits, lack of coordination, seizures and cognitive deficits
- comfort measures for nausea, vomiting and pain
- measures for communication if aphasia is present
- measures to improve vision if visual deficits are present
- how to buy wigs and hairpieces
- referrals to support groups and community resources
- helpful resources:
  - Brain Foundation: brainfoundation.org.au
  - Brain Tumour Alliance Australia: www.btaa.org.au
  - Cancer Council Australia: www.cancer.org.au
  - Sydney Neuro-Oncology Group (SNOG): www.snog.org.au.



CHAPTER HIGHLIGHTS

- Altered level of consciousness (LOC) is a common response to intracranial disorders and is an early manifestation of deterioration of the function of the cerebral hemispheres. The alteration in cerebral function occurs in a sequential pattern, with characteristic changes in LOC, respiratory patterns, pupillary and oculomotor responses, and motor function. Coma states include persistent vegetative state and locked-in syndrome.
- Increased intracranial pressure (IICP) is a sustained elevated pressure (≥ 10 mmHg) within the cranial cavity. IICP may result from cerebral oedema, hydrocephalus, head trauma, tumours, abscesses, inflammation, haemorrhage or stroke.
- The manifestations of IICP include a decreasing LOC, abnormal motor weakness and responses, altered vision, altered vital signs, headache, papilloedema and projectile vomiting. If untreated, IICP causes a displacement (herniation) of cerebral tissue, herniation of the cerebellum through the tentorium, followed by herniation of the brainstem through the foramen magnum. This is a lethal complication of IICP because it puts pressure on the vital centres in the medulla. IICP is primarily managed with osmotic diuretics and monitored with continuous intracranial pressure monitors.
- Headaches, a common type of intracranial pain, are categorised as tension, migraine and cluster. A classic migraine is characterised by an aura; a common migraine does not have an aura.
- Epilepsy is a chronic disorder of abnormal, recurring, excessive and self-terminating electrical discharges from neurones. A seizure is a single event of abnormal electrical discharge.

Seizures are categorised into those that affect only a part of the brain (partial seizures) and those that affect all of the brain (generalised). The most common type of seizure in adults is a tonic–clonic generalised seizure. Seizures are treated medically with anti-epileptic drugs (AEDs), surgery, and/or vagal nerve stimulation therapy.

- Traumatic brain injury (TBI) refers to any injury of the scalp, skull or brain and is a leading cause of death and disability. TBIs include skull fractures and focal or diffuse brain injury. An acute brain injury affects all body systems and carries the risk of secondary injury to the brain from hypoxia and ischaemia.
- An epidural haematoma develops in the potential space between the dura and the skull. A subdural haematoma collects between the dura mater and the arachnoid mater. Diffuse brain injuries include contusions, concussions and diffuse axonal injury. People with an acute TBI must have immediate transport and treatment in an ED, followed by care in a critical care unit. They will require long-term physical care and rehabilitation.
- Central nervous system infections may be caused by bacteria, bacterial toxins, viruses, fungi, protozoans and rickettsiae. Organisms may enter the brain through the bloodstream or by direct invasion. The main CNS infections are meningitis, encephalitis and brain abscess. CNS infections are treated with broad-spectrum antibiotics and antifungal agents.
- Brain tumours are growths within the cranium, including on or in brain tissue, the meninges, the pituitary gland or blood vessels. Brain tumours may be benign or malignant, primary or metastatic, and intracerebral or extracerebral. Regardless of the type or location, brain tumours are potentially lethal because they displace or impinge on CNS structures within a closed bony system.

CONCEPT CHECK

1 Which of the following pathophysiological events results in irregular respiratory patterns as LOC decreases?
1. pressure on the meninges
2. reflexive motor responses
3. loss of the oculocephalic reflex
4. brainstem responses to changes in $PaCO_2$

2 The unconscious person has depressed or absent gag and swallowing reflexes. Which nursing diagnosis would be appropriate?
1. *Decreased intracranial adaptive capacity*
2. *Risk of aspiration*
3. *Imbalanced nutrition: less than body requirements*
4. *Ineffective breathing pattern*

3 What is the rationale for the use of osmotic diuretics to treat IICP?
1. Hyperthermia increases the cerebral metabolic rate and exacerbates IICP.
2. Increased blood osmolality draws oedematous fluid into the vascular system.
3. People with IICP are at increased risk of gastrointestinal haemorrhage.
4. Brain injury and IICP often cause seizures.

4 You are monitoring the neurological status of a person in a coma. Which of the following commands would be most accurate in identifying changes in mental status?
1. 'Tell me your name.'
2. 'Look at this light when I shine it in your eyes.'
3. 'Squeeze my hand.'
4. 'Are you having trouble breathing?'

5 On admission to the ED, a person who has altered LOC has a variety of laboratory tests to facilitate the diagnosis of the aetiology of the condition. Which tests would likely be performed? (Select all that apply.)
1. blood glucose
2. serum electrolytes
3. blood and urine toxicology
4. urine for WBCs
5. spinal fluid osmolarity

6 Of the following diagnostic tests, which one is the most accurate indicator of hydration status in the person with altered LOC?
1. FBC
2. urinalysis
3. serum osmolality
4. blood culture

7 Which manifestation is consistently assessed in people with generalised seizures?
1. loss of consciousness
2. repetitive non-purposeful activity
3. tonic movements
4. clonic movements

8 When assessing a person with a head injury, you test fluid dripping from one ear for glucose. What are you assessing for?
1. infection
2. blood
3. CSF
4. serum

9 You are administering an anti-epileptic drug to a person newly diagnosed with seizures. The person says, 'Will this cure my convulsions?' What would you say?
1. 'No, but it will relieve your headache.'
2. 'No, but it will help decrease the aura you experience.'
3. 'No, not for the first year.'
4. 'No, but it may reduce or control them.'

10 Which of the following statements is true of brain tumours?
1. All brain tumours are potentially lethal.
2. Only malignant tumours are lethal.
3. Metastatic brain tumours are benign tumours.
4. Benign brain tumours rarely require treatment.

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CHAPTER 42

NURSING CARE OF PEOPLE WITH CEREBROVASCULAR AND SPINAL CORD DISORDERS

DEBRA RAYMOND

KEY TERMS

LEARNING OUTCOMES

- Identify prevalence, incidence and risk factors responsible for disorders of cerebral blood flow and spinal cord structure and function.
- Explain the pathophysiology, manifestations, complications, interprofessional care and nursing care of people with stroke, ruptured intracranial aneurysm, arteriovenous malformation, spinal cord injury, herniated intervertebral disc and spinal cord tumour.
- Compare and contrast the acute treatment and care of the person with a stroke or ruptured intracranial aneurysm and a spinal cord injury.
- Discuss the pathophysiological effects of injuries and tumours of the spinal cord by level of injury.
- Discuss the purposes, nursing implications and health education of the person and family for medications used to treat stroke, ruptured intracranial aneurysm and spinal cord injury.
- Describe the methods used to stabilise and immobilise spinal cord injuries.
- Describe the surgical procedures used to treat cerebrovascular and spinal cord disorders.

CLINICAL COMPETENCIES

- Assess functional status of people with cerebrovascular and spinal cord disorders and monitor, document and report abnormal manifestations.
- Use evidence-based research to promote early recognition and treatment of the warning signs of a stroke.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with cerebrovascular and spinal cord disorders.
- Administer oral and injectable medications used to treat cerebrovascular and spinal cord disorders knowledgeably and safely.
- Provide skilled care to people having a carotid endarterectomy, halo fixation and a posterior laminectomy.
- Integrate interprofessional care into the care of people with cerebrovascular and spinal cord disorders.
- Provide appropriate teaching to facilitate self-catheterisation, self-care of a ruptured intervertebral disc and community-based self-care of disabilities resulting from cerebrovascular and spinal cord disorders.
- Revise the plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to people with cerebrovascular and spinal cord disorders.

The health problems discussed in this chapter result from alterations in cerebral blood flow and disorders of the spinal cord. People with disorders of cerebral blood flow and the spinal cord experience a wide variety of neurological deficits that affect cognitive and perceptual functional health patterns. They also require treatment and care for both acute and long-term health problems.

Nursing care for people with these disorders is tailored to meet the needs of the person and is individualised according to the person's responses to alterations in intracranial and spinal cord structure and function. This chapter's discussion of nursing care includes consideration of both acute and long-term healthcare needs. The disabilities and long-term effects resulting from cerebrovascular disorders and spinal cord injuries almost always cause loss and grief, not only in the person but also in the family of the person. Chapter 4 provides information about person responses to loss and nursing interventions to help reduce grieving.

CEREBROVASCULAR DISORDERS

THE PERSON WITH A STROKE

A **stroke** (*cerebral vascular accident* (*CVA*)) is a condition in which neurological deficits result from a sudden decrease in blood flow to a localised area of the brain. Strokes may be *ischaemic* (when blood supply to a part of the brain is suddenly interrupted by a thrombus (blood clot), embolus (foreign matter travelling through the circulation) or stenosis (narrowing)) or *haemorrhagic* (when a blood vessel breaks open, spilling blood into spaces surrounding neurons). The neurological deficits caused by ischaemia and the resultant necrosis of cells in the brain vary according to the area of the brain involved, the size of the affected area and the length of time blood flow is decreased or stopped. A major loss of blood supply to the brain can cause severe disability or death. When the duration of decreased blood flow is short and the anatomical area involved is small, the person may not be aware that damage has been done.

Incidence and prevalence

On average, someone in Australia has a stroke every 10–13 minutes. Stroke is the second leading cause of death in Australia and a major cause of disability: approximately 60 000 people suffer a stroke each year. Of these, many people who survive are left with some type of functional impairment. Although strokes occur in every age group, the highest incidence occurs in people over 75 years of age. Strokes occur more frequently in men than women and Australian men are also more likely to suffer a stroke at a younger age than women (National Stroke Foundation, 2012). Indigenous Australians are almost twice as likely to experience cerebrovascular disease and twice as likely to die as a result than non-Indigenous Australians (Gray, Brown & Thomson, 2012).

Risk factors

Certain diseases and lifestyle habits increase the risk of a stroke, including the following (Australian Institute of Health and Welfare (AIHW), 2006; National Stroke Foundation, 2012):

- *Hypertension*. Hypertension is the greatest risk factor for a stroke. Increased systolic and diastolic blood pressure is associated with damage to all blood vessels, including the cerebral vessels. People with hypertension have a four to six times greater risk of stroke than those without hypertension. Thirty per cent of Australian adults over 25 years of age have hypertension.
- *Heart disease*. Atrial fibrillation increases the risk of stroke five-fold (American Heart Association (AHA), 2009). Other cardiovascular problems that increase the risk of a stroke are mitral valve stenosis, patent foramen ovale and cardiac surgery.
- *Diabetes mellitus*. Diabetes leads to vascular changes in both the systemic and cerebral circulation and increases the risk of hypertension. (The prevalence of hypertension is 40% higher in people with diabetes.) The person with diabetes is three times more likely to have a stroke than those without diabetes. Some 7.6% of Australian adults over the age of 25 years have diabetes.
- *Blood cholesterol levels*. Increased blood cholesterol levels contribute to the risk of atherosclerosis, including arteries in the cerebral circulation. Fifty-one per cent of the Australian population over 25 years have an elevated cholesterol level.
- *Smoking*. Cigarette smoking doubles a person's risk of ischaemic stroke and increases the risk of cerebral haemorrhage by up to 3.5%. Smoking is directly responsible for more strokes in young adults. Seventeen per cent of Australians over the age of 14 years smoke daily.
- *Substance abuse*. The injection of unpurified substances increases the risk of a stroke and abuse of certain drugs can decrease cerebral blood flow and increase the risk of intracranial haemorrhage. Substances associated with strokes include marijuana, anabolic steroids, heroin, amphetamines and cocaine.

FAST FACTS

Estimated cost of stroke in Australia

- Total healthcare cost of stroke was $606 million in 2008–2009.
- The largest portion of the cost is spent on hospital care.
- The total financial cost in 2012 for stroke was estimated to be $5 billion a year.

Sources: AIHW (2008); National Stroke Foundation (2016).

Other risk factors include a family history of stroke, obesity, a sedentary lifestyle, recent viral and bacterial infections, and previous transient ischaemic attacks. Risk factors specific to women are oral contraceptive use, pregnancy, childbirth, menopause, migraine headaches with aura, autoimmune disorders (such as diabetes and lupus) and clotting disorders.

In addition, having a stroke is a major risk factor for having another stroke (called recurrent stroke); about 25% of people who have a stroke and recover have another stroke within 5 years. The risk is highest immediately after a stroke, then decreases with time (American Heart Association (AHA), 2009; National Institute of Neurological Disorders and Stroke (NINDS), 2012a).

Pathophysiology

The brain, which makes up only 2% of total body weight, receives approximately 20% of the cardiac output each minute (about 750 mL) and accounts for 20% of the body's oxygen consumption. Cerebral blood flow, especially in the deep cerebral vessels, is largely self-regulated by the brain to meet metabolic needs. This self-regulation (also called autoregulation) allows the brain to maintain a constant blood flow despite changes in systemic blood pressure. However, autoregulation is not effective when systemic blood pressure falls below 50 mmHg or rises above 160 mmHg. In the latter case, the increased systemic pressure (as in hypertension) causes an increase in cerebral blood flow with resultant over-distension of cerebral vessels. Cerebral blood flow also increases in response to increased carbon dioxide concentrations, increased hydrogen ion concentrations and decreased oxygen concentrations.

When blood flow to and oxygenation of cerebral neurons are decreased or interrupted, pathophysiological changes at the cellular level take place in 4 to 5 minutes. Cellular metabolism ceases as glucose, glycogen and adenosine triphosphate (ATP) are depleted and the sodium–potassium pump fails. Cells swell as sodium draws water into the cell. Cerebral blood vessel walls also swell, further decreasing blood flow. Even if circulation is restored, vasospasm and increased blood viscosity can continue to impede blood flow. Severe or prolonged ischaemia leads to cellular death. A central core of dead or dying cells is surrounded by a band of minimally perfused cells, called the penumbra. Although cells in the penumbra have impaired metabolic activities, their structural integrity is maintained. The survival of these cells depends on a timely return of adequate circulation, the volume of toxic products released by adjacent dying cells, the degree of cerebral oedema and alterations in local blood flow. The potential survival of cells in the penumbra has led to the use of fibrinolytic agents in the early treatment of ischaemic stroke (Porth, 2010).

The neurological deficits that occur as a result of a stroke can often be used to identify its location. Because the motor pathways cross at the junction of the medulla and spinal cord (decussation), strokes lead to loss or impairment of sensorimotor functions on the side of the body opposite the side of the brain that is damaged. This effect, known as a **contralateral deficit**, causes a stroke in the right hemisphere of the brain to be manifested by deficits in the left side of the body (and vice versa).

A stroke is characterised by a gradual or rapid onset of neurological deficits due to compromised cerebral blood flow. Strokes may result from a variety of problems, including cerebral thrombosis, cerebral embolism and cerebral haemorrhage.

Ischaemic stroke

Ischaemic strokes result from blockage and/or stenosis of a cerebral artery decreasing or stopping blood flow and ultimately causing a brain infarction. This type of stroke accounts for about 80% of all strokes (NINDS, 2012a). The blockage may result from a blood clot (either as a thrombus or an embolus) or from stenosis of a vessel resulting from a build up of plaque. Plaque may cause stenosis in large blood vessels (called large vessel disease) or small blood vessels (called small vessel disease). Large vessel disease usually is the result of thrombi. Small vessel strokes, called lacunar infarcts, are small to very small infarcts in the deep, non-cortical areas of the brain or the brainstem. Ischaemic strokes are classified as transient, thrombotic or embolic.

TRANSIENT ISCHAEMIC ATTACK A **transient ischaemic attack (TIA)**, sometimes called a mini-stroke, is a brief period of localised cerebral ischaemia that causes neurological deficits lasting for less than 24 hours (usually less than 1 to 2 hours) (Porth, 2010). The deficits may be present for only minutes or may last for hours. TIAs are often warning signals of an ischaemic thrombotic stroke. One or many TIAs may precede a stroke, with the time between the TIA and a stroke ranging from hours to months. One in five people who have a TIA will have an acute stroke within 3 months (National Stroke Foundation, 2012). The aetiology of TIA includes inflammatory artery disorders, sickle cell anaemia, atherosclerotic changes in cerebral blood vessels, thrombosis and emboli. Neurological manifestations of a TIA vary according to the location and size of the cerebral vessel involved. Manifestations have a sudden onset and often disappear within minutes or hours. Commonly occurring deficits include contralateral numbness or weakness of the leg, hand, forearm and corner of the mouth (due to middle cerebral artery involvement); aphasia (due to ischaemia of the left hemisphere); and visual disturbances such as blurring (due to involvement of the posterior cerebral artery) (Porth, 2010). The person may also experience a visual disturbance called *amaurosis fugax* (a fleeting blindness of one eye, described as a shade coming down over vision in the affected eye).

THROMBOTIC STROKE A thrombotic stroke is caused by occlusion of a large cerebral vessel by a thrombus (blood clot). Thrombotic CVAs most often occur in older people who are resting or sleeping. The blood pressure is lower during sleep, so there is less pressure to push the blood through an already narrowed arterial lumen and ischaemia may result.

Thrombi tend to form in large arteries that bifurcate and have narrowed lumens as a result of deposits of atherosclerotic plaque. The plaque involves the intima of the arteries, causing the internal elastic lamina to become thin and frayed with exposure of underlying connective tissue. This structural change causes platelets to adhere to the rough surface and release the enzyme adenosine

diphosphate. This enzyme initiates the clotting sequence and the thrombus forms. A thrombus may remain in place and continue to enlarge, completely occluding the lumen of the vessel, or a part of it may break off and become an embolus.

The most common locations of thrombi are the internal carotid artery, the vertebral arteries and the junction of the vertebral and basilar arteries. Thrombotic strokes affecting the smaller cerebral vessels are called lacunar strokes, because the infarcted areas slough off, leaving a small cavity or 'lake' in the brain tissue. A thrombotic stroke usually affects only one region of the brain that is supplied by a single cerebral artery.

A thrombotic stroke occurs rapidly but progresses slowly. It often begins with a TIA and continues to worsen over 1 to 2 days; the condition is called a *stroke in evolution*. When maximum neurological deficit has been reached, usually in 3 days, the condition is called a *completed stroke*. At that time, the damaged area of brain tissue is oedematous and necrotic.

EMBOLIC STROKE An embolic stroke occurs when a blood clot or clump of matter travelling through the cerebral blood vessels becomes lodged in a vessel too narrow to permit further movement. The area of the brain supplied by the blocked vessel becomes ischaemic. The most frequent sites of cerebral emboli are at bifurcations of vessels, particularly those of the carotid and middle cerebral arteries. This type of stroke is typically seen in people who are younger than those experiencing thrombotic strokes and occurs when the person is awake and active.

Many embolic strokes originate from a thrombus in the left chambers of the heart, formed during atrial fibrillation. These are referred to as cardiogenic *embolic strokes*. Emboli result when parts of the thrombus break off and are carried through the arterial system to the brain. Cerebral emboli may also be caused by carotid artery atherosclerotic plaque, bacterial endocarditis, recent myocardial infarction, rheumatic heart disease and ventricular aneurysm.

An embolic stroke has a sudden onset and causes immediate deficits. If the embolus breaks down and is absorbed by the body, manifestations will disappear in a few hours to a few days. If the embolus is not absorbed, manifestations will persist. Even if the embolus is absorbed, the vessel wall where the embolus lodges may be weakened, increasing the potential for cerebral haemorrhage.

Haemorrhagic stroke

A **haemorrhagic stroke**, or intracranial haemorrhage, occurs when a cerebral blood vessel ruptures. It occurs most often in people with sustained increase in systolic–diastolic blood pressure. Intracranial haemorrhage usually occurs suddenly, often when the affected person is engaged in some activity. Although hypertension is the most common cause, a variety of factors may contribute to a haemorrhagic stroke, including rupture of a brittle plaque-encrusted artery wall, ruptured intracranial aneurysms, trauma, erosion of blood vessels by tumours, arteriovenous malformations, anticoagulant therapy and blood disorders. Of all forms of stroke, this form is most often fatal and occurs in about 20% of all strokes (NINDS, 2012a). There are two types of haemorrhagic strokes: intracerebral haemorrhage and subarachnoid haemorrhage. Haemorrhagic strokes that result from ruptured cerebral aneurysm or an arteriovenous malformation are discussed in the following sections of the chapter.

As a result of the blood vessel rupture, blood enters the brain tissue, the cerebral ventricles or the subarachnoid space, compressing adjacent tissues and causing blood vessel spasm and cerebral oedema. Blood in the ventricles or subarachnoid space irritates the meninges and brain tissue, causing an inflammatory reaction and impairing absorption and circulation of cerebrospinal fluid (CSF).

The onset of manifestations from a haemorrhagic stroke is rapid. Manifestations depend on the location of the haemorrhage, but may include vomiting, headache, seizures, hemiplegia and loss of consciousness. Pressure on the brain tissue from increased intracranial pressure (discussed in Chapter 41) may cause coma and death.

Manifestations

Manifestations of a stroke vary according to the cerebral artery involved and the area of the brain affected. Manifestations are always sudden in onset, focal and usually one sided. The most common manifestation is weakness involving the face and arm, and sometimes the leg. Other common manifestations are numbness on one side, loss of vision, speech difficulties, a sudden severe headache and difficulties with balance. The various deficits associated with involvement of a specific cerebral artery are collectively referred to as stroke syndromes, although the deficits often overlap, as shown in the box below.

Early warning signs of strokes are shown in Box 42.1.

MANIFESTATIONS Stroke by involved cerebral vessel

INTERNAL CAROTID ARTERY
- Contralateral paralysis of the arm, leg and face
- Contralateral sensory deficits of the arm, leg and face
- If the dominant hemisphere is involved: aphasia
- If the non-dominant hemisphere is involved: apraxia, agnosia, unilateral neglect
- Homonymous hemianopia

MIDDLE CEREBRAL ARTERY
- Drowsiness, stupor, coma
- Contralateral hemiplegia of the arm and face
- Contralateral sensory deficits of the arm and face
- Global aphasia (if dominant hemisphere involved)
- Homonymous hemianopia

ANTERIOR CEREBRAL ARTERY
- Contralateral weakness or paralysis of the foot and leg
- Contralateral sensory loss of the toes, foot and leg
- Loss of ability to make decisions or act voluntarily
- Urinary incontinence

VERTEBRAL ARTERY
- Pain in face, nose or eye
- Numbness and weakness of the face on involved side
- Problems with gait
- Dysphagia

BOX 42.1 Early warning signs of strokes

Making sure the public has a good understanding of early warning signs using the FAST acronym is vital to ensuring rapid treatment of a person suffering a stroke.

- **F**ACE—check their face. Has their mouth drooped?
- **A**RMS—can they lift both arms?
- **S**PEECH—is their speech slurred? Do they understand you?
- **T**IME—time is critical. If you see any of these signs, call 000 or mobile 112 immediately!

Source: National Stroke Foundation (2012).

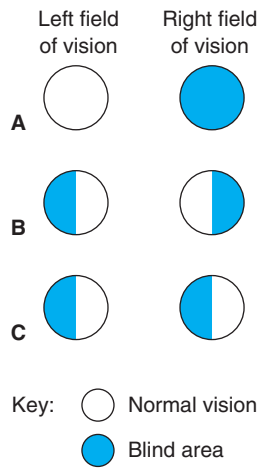


FIGURE 42.1 ■ Abnormal visual fields. *A*, Normal left field of vision with loss of vision in right field. *B*, Loss of vision in temporal half of both fields (bitemporal hemianopia). *C*, Loss of vision in nasal field of right eye and temporal field of left eye (homonymous hemianopia)

Complications

Typical complications include sensoriperceptual deficits, cognitive and behavioural changes, communication disorders, motor deficits and elimination disorders. These may be transient or permanent, depending on the degree of ischaemia and necrosis as well as time of treatment. As a result of the neurological deficits, the person with a stroke has complications that involve many different body systems (see the box below). The disabilities resulting from a stroke often cause serious alterations in functional health status.

Sensoriperceptual deficits

A stroke may involve pathological changes in neurological pathways that alter the ability to integrate, interpret and attend to sensory data. The person may experience deficits in vision, hearing, equilibrium, taste and sense of smell. The ability to perceive vibration, pain, warmth, cold and pressure may be impaired, as may proprioception (the body's sense of its position). The loss of these sensory abilities increases the risk of injury. Deficits may include:

- **hemianopia**—the loss of half of the visual field of one or both eyes; when the same half is missing in each eye, the condition is called *homonymous hemianopia* (see Figure 42.1)
- **agnosia**—the inability to recognise one or more subjects that were previously familiar; agnosia may be visual, tactile or auditory
- **apraxia**—the inability to carry out some motor pattern (e.g. drawing a figure, getting dressed) even when strength and coordination are adequate.

Another form of sensory–perceptual deficit is the **neglect syndrome** (or *unilateral neglect*), in which the person has a disorder of attention. In this syndrome, the person cannot integrate and use perceptions from the affected side of the body or from the environment on the affected side and ignores that part. In severe cases, the person may even deny the paralysis. This deficit is more common following a stroke of the right hemisphere where damage to the parietal lobe (a centre for mediation of directed attention) results in perceptual deficits.

Pain and discomfort may accompany a stroke, with the person experiencing acute pain, numbness or strange sensations. Although not common, damage to the thalamus may cause *central stroke pain* or *central pain syndrome*. The pain in this syndrome includes hot and cold, burning, tingling and sharp stabbing pain, most often in the extremities. It is worsened by movement and temperature changes. The painful sensations are not relieved by pain medications, nor are there any specific treatments.

Cognitive and behavioural changes

A change in consciousness, ranging from mild confusion to coma, is a common manifestation of a stroke. It may result from tissue damage following ischaemia or haemorrhage involving either the carotid or the vertebral arteries. Altered consciousness may also be the result of cerebral oedema or increased intracranial pressure.

Behavioural changes include emotional lability (in which the person may laugh or cry inappropriately), loss of self-control (manifested by behaviour such as swearing or refusing to wear clothing) and decreased tolerance for stress (resulting in anger or depression). Intellectual changes may include memory loss, decreased attention span, poor judgment and an inability to think abstractly.

Communication disorders

Communication is a complex process, involving motor functions, speech, language, memory, reasoning and emotions. Communication disorders are usually the result of a stroke affecting the dominant hemisphere. The left hemisphere is dominant in about 95% of right-handed people and 70% of left-handed people (Porth, 2010).

Many different impairments may occur and most are partial. Disorders of communication affect both speech (the mechanical act of articulating language through the spoken word) and language (the vocal or written formulation of ideas to communicate thoughts and feelings). Language involves oral and

MANIFESTATIONS AND COMPLICATIONS Stroke by body system

INTEGUMENT
- Decubitus (pressure) ulcers

NEUROLOGICAL
- Hyperthermia
- Neglect syndrome
- Seizures
- Agnosias
- Communication deficits
  - a Expressive aphasia
  - b Receptive aphasia
  - c Global aphasia
  - d Agraphia
- Visual deficits
  - a Homonymous hemianopia
  - b Diplopia
  - c Decreased acuity
- Cognitive changes
  - a Memory loss
  - b Short attention span
  - c Distractibility
  - d Poor judgment
  - e Poor problem-solving ability
  - f Disorientation
- Behavioural changes
  - a Emotional lability
  - b Loss of social inhibitions
  - c Fear
  - d Hostility
  - e Anger
  - f Depression
- Increased intracranial pressure
- Alterations in consciousness
- Sensory loss (touch, pain, heat, cold, pressure)

RESPIRATORY
- Respiratory centre damage
- Airway obstruction
- Decreased ability to cough

GASTROINTESTINAL
- Dysphagia
- Constipation
- Stool impaction

GENITOURINARY
- Incontinence
- Frequency
- Urgency
- Urinary retention
- Renal calculi

MUSCULOSKELETAL
- Hemiplegia
- Contractures
- Bony ankylosis
- Disuse atrophy
- Dysarthria

written expression and auditory and reading comprehension. These disorders include:

- aphasia—the inability to use or understand language; aphasia may be expressive, receptive or mixed (global)
- expressive aphasia—a motor speech problem in which one can understand what is being said but can respond verbally only in short phrases; also called *Broca's aphasia*
- receptive aphasia—a sensory speech problem in which one cannot understand the spoken (and often written) word. Speech may be fluent but with inappropriate content; also called *Wernicke's aphasia*
- mixed or global aphasia—language dysfunction in both understanding and expression
- dysarthria—any disturbance in muscular control of speech.

Motor deficits

Body movement results from a complex interaction between the brain, spinal cord and peripheral nerves. The motor areas of the cerebral cortex, the basal ganglia and the cerebellum initiate voluntary movement by sending messages to the spinal cord, which then transmits the messages to the peripheral nerves. A stroke may interrupt the central nervous system (CNS) component of this relay system and produce effects in the contralateral side ranging from mild weakness to severe limitation of any kind of movement.

Depending on the area of the brain involved, strokes may cause weakness, paralysis and/or spasticity. The deficits include:

- **hemiplegia**—paralysis of the left or right half of the body (see Figure 42.2)
- **hemiparesis**—weakness of the left or right half of the body
- flaccidity—absence of muscle tone (hypotonia)
- spasticity—increased muscle tone (hypertonia), usually with some degree of weakness. The flexor muscles are usually more strongly affected in the upper extremities and the extensor muscles are more strongly affected in the lower extremities.

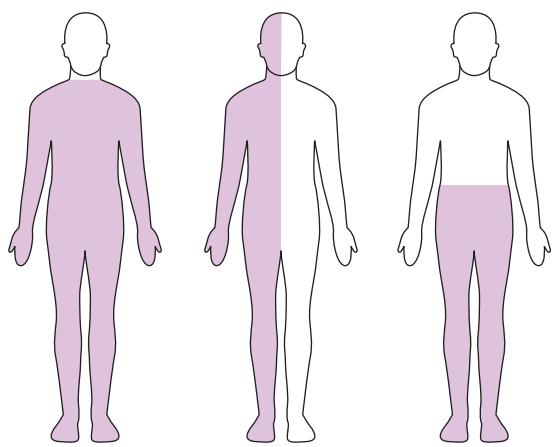


FIGURE 42.2 ■ Types of paralysis. *A*, Quadriplegia is complete or partial paralysis of the upper extremities and complete paralysis of the lower part of the body. *B*, Hemiplegia is paralysis of one half of the body when it is divided along the median sagittal plane. *C*, Paraplegia is paralysis of the lower part of the body

When the corticospinal tract is involved, the affected arm and leg almost always are initially flaccid and then become spastic within 6 to 8 weeks. Spasticity often causes characteristic body positioning: adduction of the shoulder, pronation of the forearm, flexion of the fingers and extension of the hip and knee. There is often foot drop, outward rotation of the leg and dependent oedema in the involved extremities.

The motor deficits may result in altered mobility, further impairing body function. The complications of immobility

involve multiple body systems and include orthostatic hypotension, increased thrombus formation, decreased cardiac output, impaired respiratory function, osteoporosis, formation of renal calculi, contractures and decubitus ulcer formation.

Elimination disorders

Disorders of bladder and bowel elimination are common. A stroke may cause partial loss of the sensations that trigger bladder elimination, resulting in urinary frequency, urgency or incontinence. Control of urination may be altered as a result of cognitive deficits. Changes in bowel elimination are common, resulting from changes in level of consciousness (LOC), immobility and dehydration (Hickey, 2013).

INTERPROFESSIONAL CARE

The type of treatment a person with a stroke receives depends on the stage of the disease. In general, there are three treatment stages: stroke prevention, acute care immediately after a stroke and rehabilitation after a stroke. The person with an acute stroke may receive medical and/or surgical treatment. The focus in the acute care phase is on diagnosing the type and cause of the stroke, supporting cerebral circulation and controlling or preventing further deficits. The goals of stroke care are to minimise brain injury and maximise recovery. In Australia, it is recommended that a person suffering from a stroke is cared for in a specialist stroke unit where possible; these units have been shown to decrease morbidity and mortality rates (AIHW, 2006).

Diagnosis

Diagnosis begins with a complete history and careful physical assessment, including a thorough neurological examination. The time of the onset of stroke manifestations is a critical part of assessment. The National Institute of Health (NIH) Stroke Scale is a clinical evaluation tool widely used to assess neurological outcome and degree of recovery. Part of the Scale is detailed in Table 42.1. The tool measures LOC, vision, facial paralysis, motor abilities, ataxia, sensation, language and attention.

Imaging tests are used to identify an increased risk of a stroke or to identify pathophysiological changes after a stroke has occurred.

Computed tomography (CT) is the first imaging technique used to demonstrate the presence of haemorrhage, tumours, aneurysm, ischaemia, oedema and tissue necrosis. A CT scan can also demonstrate a shift in intracranial contents and is useful in distinguishing the type of stroke (e.g. a haemorrhagic stroke results in an increase in density). Cerebral infarctions usually are visible with a CT scan 6 to 8 hours post stroke; haemorrhage is visible immediately. Other imaging tests that may be used for diagnosis include cerebral arteriogram,

TABLE 42.1 NIH Stroke Scale: assessment of level of consciousness

INSTRUCTIONS	SCALE DEFINITION	SCORE
1a. **Level of consciousness (LOC)**: The investigator must choose a response, even if a full evaluation is prevented by such obstacles as an endotracheal tube, language barrier, orotracheal trauma/bandages. A 3 is scored only if the person makes no movement (other than reflexive posturing) in response to noxious stimulation.	0 = Alert, keenly responsive. 1 = Not alert, but arousable by minor stimulation to obey, answer or respond. 2 = Not alert, requires repeated stimulation to attend or is obtunded and requires strong or painful stimulation to make movements (not stereotyped). 3 = Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid and flexic.	_____
1b. **LOC questions**: The person is asked the month and their age. The answer must be correct. There is no partial credit for being close. Aphasic and stuporous people who do not comprehend the questions will score 2. People unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier or any other problem not secondary to aphasia are given a 1. It is important that only the initial answer be graded and that the examiner not 'help' the person with verbal or non-verbal cues.	0 = Answers both questions correctly. 1 = Answers one question correctly. 2 = Answers neither question correctly.	_____
1c. **LOC commands**: The person is asked to open and close the eyes and then to grip and release the non-paretic hand. Substitute another one-step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the person does not respond to command, the task should be demonstrated to them (pantomime) and the results scored (i.e. follows none, one or two commands). A person with trauma, amputation or other physical impediments should be given suitable one-step commands. Only the first attempt is scored.	0 = Performs both tasks correctly. 1 = Performs one task correctly. 2 = Performs neither task correctly.	_____

Note: This is a sample of only one part of the NIH Stroke Scale. The entire scale may be viewed as a PDF file at www.ninds.nih.gov/doctors/NIH_Stroke_Scale.pdf.

transcranial Doppler ultrasound, MRI, MRA, PET and SPECT (see the 'Diagnostic tests' box in Chapter 40).

A lumbar puncture may be performed to obtain CSF for examination if there is no danger of increased intracranial pressure (ICP). (Removal of CSF when intracranial pressure is increased can result in herniation of the brainstem.) A thrombotic stroke may elevate CSF pressure; after a haemorrhagic stroke, frank blood may be seen in the CSF.

In addition to imaging tests, a blood test has recently been approved to screen for recurrent stroke risk. The PLAC test scans the blood for high levels of lipoprotein-associated phospholipase A2 (Lp-Pla2), found to be more common in a person who has had a stroke.

Medications

Medications are administered to prevent a stroke in a person with a TIA or a previous stroke and to treat the person during the acute phase of a stroke.

PREVENTION Antiplatelet agents are often used to treat a person with a TIA or who has had a previous stroke. Platelets are concentrated in arteries with a high blood flow where they adhere to endothelial tissue damaged by atherosclerosis and occlude the vessel. The drugs used to prevent clot formation and blood vessel occlusion include aspirin, clopidogrel (Plavix), dipyridamole (Persantine) and ticlopidine hydrochloride (Ticlopidine Hexal).

Daily low-dose aspirin reduces TIA occurrence and stroke risk by interfering with platelet aggregation. Ticlopidine hydrochloride is a platelet-aggregation inhibitor that has shown reduction in thrombotic stroke risk.

ACUTE STROKE Medications are used to treat the person during the acute phase of an ischaemic stroke to prevent further thrombosis formation, increase cerebral blood flow and protect cerebral neurons. The type of medication used varies according to the type of stroke.

Anticoagulant drug therapy (discussed in Chapter 32) is often ordered for an ischaemic stroke. The most commonly used anticoagulants are warfarin (Coumadin), heparin and enoxaparin (Clexane). Anticoagulants are never administered to a person with a haemorrhagic stroke. Anticoagulants do not dissolve an existing clot but prevent further extension of the clot and formation of new clots. Sodium heparin may be given subcutaneously or by continuous IV infusion, or warfarin sodium (Coumadin) may be given orally.

Fibrinolytic therapy, using a tissue plasminogen activator such as recombinant tissue plasminogen activator alteplase (rt-PA, tPA), sometimes given concurrently with an anticoagulant, is used to treat thrombotic stroke. The drug converts plasminogen to plasmin, resulting in fibrinolysis of the clot. To be effective, it must be given intravenously as soon as possible after the onset of manifestations, after confirming (with a CT scan) that the person has had an ischaemic stroke. Within Australia current guidelines state the drug must be given within 4.5 hours of onset of symptoms (National Stroke Foundation, 2010). Antithrombotic drugs, which inhibit the platelet phase of clot formation, have been used as a preventive measure for the person at risk of embolic and thrombotic CVA. Both aspirin and dipyridamole have been used for this purpose. These drugs are sometimes also used in combination with other drugs during acute treatment. Antiplatelet agents are contraindicated in the person with a haemorrhagic stroke.

Management of hypertension is controversial but if the person is eligible for fibrinolytic therapy, blood pressure control is essential to decrease the risk of bleeding. If the blood pressure is sustained at levels 185 mmHg systolic or 110 diastolic, the person cannot be treated with IV tPA (AHA, 2005).

Corticosteroids, such as prednisone or dexamethasone, have been used to treat cerebral oedema, but the results are not always positive. If the person has an increased ICP, hyperosmolar solutions (such as mannitol) or diuretics (such as frusemide) may be administered. Anticonvulsants, such as phenytoin (Dilantin), and barbiturates may be prescribed if increased ICP causes seizures. Increased ICP is discussed in Chapter 41.

Treatments

The treatments used in the management of a stroke include surgery and rehabilitation.

SURGERY Surgery may be performed to prevent the occurrence of a stroke, to restore blood flow when a stroke has already occurred or to repair vascular damage or malformations. A carotid endarterectomy at the carotid artery bifurcation may be performed to remove atherosclerotic plaque in the person who has suffered a TIA (see Figure 42.3). Nursing care for the person in the initial postoperative period following a carotid endarterectomy is described in the following box.

When an occluded or stenotic vessel is not directly accessible, an extracranial–intracranial bypass may be performed. Bypass of the internal carotid, middle cerebral or vertebral arteries may be required. The indications for the bypass are manifestations of ischaemia caused by TIAs or a mild completed stroke. The procedure re-establishes blood flow to the affected area of the brain.

A carotid angioplasty with stenting is a newer option for treating cerebral stenosis. During the procedure an angioplasty balloon catheter is inserted through an artery in the person's arm or leg. Under fluoroscopy, the catheter is advanced to the area of carotid artery stenosis and a small filter is inserted to catch any

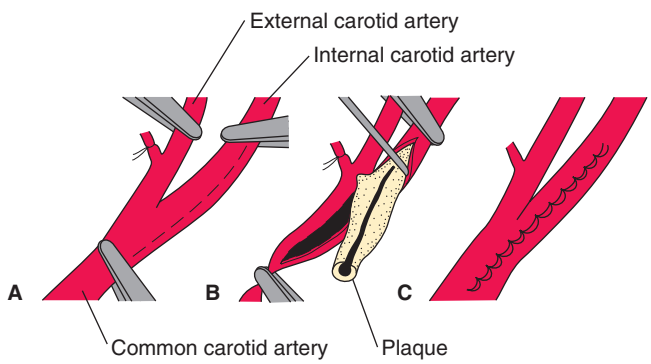


FIGURE 42.3 ■ Carotid endarterectomy. *A*, The occluded area is clamped off and an incision is made in the artery. *B*, Plaque is removed from the inner layer of the artery. *C*, To restore blood flow through the artery, the artery is sutured or a graft is completed

clots or pieces of debris that might break loose. The balloon is then inflated to widen the artery, followed by insertion of a permanent stent in the area of the angioplasty (Palmieri, 2006).

REHABILITATION Various types of therapy are necessary for post-stroke rehabilitation. Listed below are the types and goals of therapies used:

- Physiotherapy may help prevent contractures and improve muscle strength and coordination. Physiotherapists teach exercises to enable the person to relearn how to walk, sit, lie down and change from one type of movement to another.
- Occupational therapy provides assistive devices and a plan for regaining lost motor skills that greatly improve quality of life after a stroke. These skills include eating, drinking, bathing, cooking, reading, writing and toileting.
- Speech therapy is provided to help the person relearn language and communication skills, as well as improve swallowing.

Nursing care

Even though many people who have a stroke have full recovery, a substantial number are left with disabilities that affect their physical, emotional, interpersonal and family status. The required nursing care is often complex and multidimensional, requiring consideration of continuity of care for the person in acute care settings, long-term care settings, rehabilitation centres and the home.

Nurses caring for people who have had a stroke require knowledge and skill to meet their needs during both the acute and the rehabilitative phases of care. The person may have multiple losses: loss of mobility, ability to provide self-care, communications, concept of self and interpersonal or intimate relationships with others. Holistic, individualised nursing care is essential in all settings and focuses on promoting the achievement of maximum potential and quality of life.

The person's family is often faced with many changes. The young to middle-aged adult with a family member who has had a stroke may be faced with economic difficulties and social isolation. The middle-aged adult family member may become the caretaker for an older parent—in essence, switching roles with the parent. An older adult may not be able to care for a spouse and may have to accept residential care placement. In addition, the older adult who has no family may have to struggle alone to regain the ability to function independently. Although not all of these problems are amenable to nursing solutions, the nurse is most often the healthcare provider who assesses and identifies the needs of each individual and provides information and referrals to the person and families to help meet those needs.

Because a stroke has the potential to cause many different health problems, a wide variety of nursing diagnoses may be appropriate. It is important to remember that each person will be affected differently, depending on the degree of ischaemia and the area of the brain involved. Nursing diagnoses discussed in this section focus on problems with cerebral tissue perfusion (specific to nursing care during the acute phase), physical mobility, self-care, communication, sensory–perceptual deficits, bowel and urine elimination, and swallowing (specific to prevention of complications and rehabilitation). A nursing care plan for a person with a stroke is found overleaf.

NURSING CARE OF THE PERSON having a carotid endarterectomy

POSTOPERATIVE CARE

- Position on the unoperated side and either maintain a flat position or elevate the head of the bed 30 degrees as prescribed. Maintain head and neck alignment and avoid rotating, flexing or hyperextending the head. *Pressure on the wound is undesirable. Elevating the head decreases oedema in the operative site. Maintaining head and neck alignment prevents additional tension or pressure on the operative side.*
- Support the head when changing position. Teach to support the head with the hands when able to move about. *Supporting the head helps prevent stress on the operative site (which may cause bleeding and haematoma formation); it also helps reduce stress on the suture line.*
- Perform focused assessments to monitor for complications:
  a. *Haemorrhage*. Assess the dressing and the area under the neck and shoulders for drainage. Assess for increased pulse and decreased blood pressure. *The most common cause of respiratory problems is pressure on the trachea from a haematoma formation.*
  b. *Respiratory distress*. Assess respiratory rate, rhythm, depth and effort. Observe for restlessness. Keep a tracheostomy tray at the bedside. *Respiratory distress may result from oedema and haematoma formation, which may compress the trachea.*
  c. *Cranial nerve impairment*. Observe and record any facial drooping, tongue deviation, hoarseness, dysphagia or loss of facial sensation. *Cranial nerves may be stretched during surgery, leading to temporary deficits in cranial nerve function.*
  d. *Hypertension or hypotension*. Take and record blood pressure at least hourly. Report any changes immediately and implement orders for medications to treat hypertension or hypotension. *About one-half of all people having a carotid endarterectomy develop unstable blood pressure related to surgical denervation of the carotid sinus. Uncontrolled hypertension may precipitate a CVA. The most common problem is hypotension, possibly related to stimulation of the carotid body baroreceptors, which are exposed during surgery. Hypotension may result in myocardial ischaemia.*

Health promotion

Health promotion activities focus on stroke prevention, especially for the person with known risk factors. It is important to discuss, as appropriate, the importance of stopping smoking and drug use with people of all ages. Maintaining a normal weight through diet and exercise can help reduce obesity, which increases the risk of hypertension and type 2 diabetes mellitus. (Both, in turn, increase the risk of a stroke.) Cholesterol levels should be screened regularly to monitor for hyperlipidemia. Regular healthcare to monitor for and treat cardiovascular disorders and to detect and treat infections such as infective endocarditis is important. It is also important to increase public awareness of the signs of a TIA or stroke, and of the need to call 000 or mobile 112, or to seek immediate medical care if the following warning signs or symptoms suddenly occur:

- weakness or numbness of the face, arm or leg, especially on one side of the body
- confusion, difficulty speaking or understanding speech
- trouble walking, dizziness, loss of coordination
- difficulty with vision in one or both eyes
- severe headache without a cause.

Information about public awareness of stroke manifestations and the need for immediate treatment are discussed in the 'Translation to practice' box.

Assessment

The following data is collected through the health history and physical examination (see Chapter 40). Further focused assessments are described with nursing interventions. Gender-specific questions are required to assess risk factors for stroke; risk factors are different for men and women (see box below).

- *Health history:* risk factors, previous stroke, drug use (prescribed, over-the-counter, street drugs), smoking history, when manifestations began, severity of manifestations, presence of incontinence, LOC, family support system.
- *Physical assessment:* LOC, motor strength, coordination, communication, cranial nerves, sensory function.

MEETING INDIVIDUALISED NEEDS

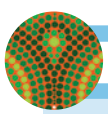
Risk factors for stroke in women

Some risk factors for stroke apply only to women—most specifically pregnancy, childbirth and menopause. These risks are the result of fluctuations in hormones that occur at different stages of life. However, other risks are also more common for women and information should be collected during a health history. For accurate assessment, ask the following questions, depending on the woman's age:

- How many pregnancies have you had?
- Have you had a miscarriage? If so, how many?
- How many births have you had? When was your last delivery?
- When was your last menstrual cycle?
- Do you take any type of hormone replacement therapy?
- Do you take birth control pills?
- Do you have migraine headaches? If so, do you have an aura?
- Have you ever been diagnosed with diabetes or lupus?
- Have you ever been diagnosed with a clotting disorder? Have you ever had a clot in your leg?

Nursing diagnoses and interventions

The acute phase of a stroke is most often the time from admission to the hospital until the person is stabilised, usually 24 to 72 hours after admission (Hickey, 2013). Depending on the severity of the stroke, the person may be admitted to the intensive care unit. Regardless of the hospital setting, the nurse provides interventions to maintain body functions and prevent complications.



TRANSLATIONTO PRACTICE Evidence-based practice: improved rapid treatment of a stroke

Stroke is the second leading cause of death in Australia and is also a leading cause of severe, long-term disability (National Stroke Foundation, 2012). The risk of disability and death can be reduced in people who experience a sudden ischaemic stroke by the administration of tissue plasminogen activator (tPA). To be effective, tPA must be administered within 4.5 hours of the warning signs of a stoke, but the public's awareness of stroke manifestations and of the need for immediate treatment remains poor. In Australia, 39% of people presenting to hospital with a stroke arrive within 3 hours; however, only 3% of all ischaemic stroke victims receive thrombolytic therapy (National Stroke Foundation, 2010). Further emphasis on achieving higher rates of thrombolytic treatment is required to improve morbidity and mortality rates.

IMPLICATIONS FOR NURSING

Nurses provide information to the public in a wide variety of health promotion activities, including stroke awareness and the need for immediate treatment to ensure the best outcomes of care. It is important that the programs be geared towards the specific population most at risk and target people from all socioeconomic and cultural backgrounds. Although not measured in this study, factors that affect behaviour, such as perceived risk, benefits and barriers of care, readiness to change and self-efficacy, are areas that may be effective in designing educational programs to increase stroke awareness.

CRITICAL THINKING IN PERSON CARE

1. If you were planning a stroke awareness education program, where would you implement it in order to reach the largest population? How would you advertise stroke warning signs to reach the most people?
2. Think of a slogan for the public that increases awareness of the 4.5-hour time limit for treatment with tPA.

Ineffective tissue perfusion: cerebral

The initial assessment and care of the person admitted for intensive care focuses on identifying changes that may indicate altered cerebral perfusion. The person's airway, breathing, circulation and neurological status are monitored, and interventions are provided to maintain cerebral perfusion.

- Monitor respiratory status and airway patency. Auscultate pulmonary sounds and monitor respiratory rate and results of arterial blood gas studies.
- Suction as necessary, using care to suction no longer than 10 seconds at any one time and using sterile technique.
- Place in a side-lying position.
- Administer oxygen as prescribed.
  The person is often unconscious and breathing may be impaired. Suctioning removes secretions that not only obstruct airflow but also pose a risk of aspiration and pneumonia. Suctioning for longer than 10 seconds at a time may increase intracranial pressure (Hickey, 2013). Respiratory complications develop rapidly, as manifested by crackles and wheezes, rapid respirations and respiratory acidosis. The administration of oxygen decreases the risk of hypoxia and hypercapnia, which can increase cerebral ischaemia and intracranial pressure.

CONSIDERATION FOR PRACTICE

Positioning the person on the side allows secretions to drain out of the mouth, helping to prevent aspiration.

- Monitor mental status and LOC (Glasgow Coma Scale): restlessness, drowsiness, lethargy, inability to follow commands, unresponsiveness.
- Monitor strength and reflexes and assess for pain, headache, decreased muscle strength, sluggish pupillary reflexes, absent gag or swallowing reflexes, hemiplegia, Babinski's sign and decerebrate or decorticate posturing.
  Frequent monitoring of neurological status is necessary to detect changes. Alterations in mental status, LOC, movement, strength and reflexes indicate increased intracranial pressure, the main cause of death in the acute phase of a stroke.
- Continuously monitor cardiac status, observing for arrhythmias. *A stroke may cause cardiac arrhythmias, including bradycardia, PVCs (premature ventricular contractions), tachycardia and AV block. Characteristic ECG changes include a shortened PR interval, peaked T waves and a depressed ST segment.*
- Monitor body temperature. *Hyperthermia may develop if the hypothalamus is affected.*
- Maintain accurate intake and output records; measure urinary output via a Foley catheter. *A stroke may damage the pituitary gland, resulting in diabetes insipidus and the possibility of dehydration from greatly increased urinary output.*

CONSIDERATION FOR PRACTICE

Diabetes insipidus is indicated by a large output of dilute urine; dehydration is indicated by scanty amounts of dark, concentrated urine.

- Monitor for seizures. Pad the side rails and administer prescribed anticonvulsants. *Seizures may be the result of cerebral tissue damage or increased intracranial pressure. Padded side rails prevent injury if a seizure occurs. Anticonvulsants prevent or treat seizures.*

Impaired physical mobility

The goals of care for the person with impaired mobility are to maintain and improve functional abilities (by maintaining normal function and alignment, preventing oedema of extremities and reducing spasticity) and to prevent complications.

- Encourage active range-of-motion (ROM) exercises for unaffected extremities and perform passive ROM exercises for affected extremities every 4 hours during day and evening shifts, and once during the night shift. Support the joint during passive ROM exercises. *Active ROM exercises maintain or improve muscle strength and endurance and help to maintain cardiopulmonary function. Passive ROM exercises do not strengthen muscles but do help maintain joint flexibility.*

CONSIDERATION FOR PRACTICE

Both active and passive exercises increase venous return, decreasing the risk of thrombophlebitis.

- Turn every 2 hours around the clock, following a posted schedule for side-to-side and supine-to-prone position changes. (Verify prone positioning with the doctor.) Maintain body alignment and support extremities in proper position with pillows. *Turning on a regular basis, accompanied by proper positioning, maintains joint function, alleviates pressure on bony prominences that can lead to skin breakdown, decreases dependent oedema in hands and feet, and lessens the risk of complications resulting from immobility (see Figure 42.4).*

CONSIDERATION FOR PRACTICE

When lying on the affected side, the person may be restless because they do not have normal sensation and feel as if they may fall.

- Monitor the lower extremities each shift for symptoms of thrombophlebitis. Assess for increased warmth and redness in calves; measure the circumference of the calves and thighs. *People on bed rest (especially those with loss of muscle strength and tone) are particularly prone to the development of deep venous thrombosis. Promptly report manifestations of thrombophlebitis.*
- Collaborate with the physiotherapist as the person gains mobility, using consistent techniques to move the person from the bed to the wheelchair and to help the person ambulate. *The use of consistent techniques facilitates rehabilitation.*

Self-care deficit

The person who has had a stroke may have a self-care deficit as a result of impaired mobility or mental confusion. It is important for the person to perform as many of their own

NURSING CARE PLAN A person with a stroke

Phillip Boren is a 63-year-old male who had a stroke due to right cerebral thrombosis 1 week ago. He is a history teacher at the local high school. His hobbies are wood carving and gardening. For the past 2 years, Mr Boren has been taking medication for hypertension, but his wife Emily reports that he often forgets to take it and that his blood pressure was high at his last physical examination. Mrs Boren tells the staff that she has never had to worry about her husband's health before and that she wants to learn everything she can to care for him at home. However, she says that her husband was always the one to make the decisions and pay the bills. Mrs Boren adds that all the children, grandchildren and neighbours want to see Mr Boren back at home as soon as possible.

ASSESSMENT

Carol Merck, RN, the nurse assigned to Mr Boren, completes a health history and physical assessment, with Mrs Boren providing information for the history. Mrs Boren reports that her husband did have several spells of dizziness and blurred vision the week before his stroke, but they lasted only a few minutes and he believed them to be due to 'old age and working out in the sun'. On the morning of admission, Mr Boren woke up and could not move his left arm or leg; he also could not speak sensibly. Mrs Boren called 000 and an ambulance took her husband to the hospital.

Physical assessment findings include the following: Mr Boren is drowsy but responds to verbal stimuli. Although he does not respond verbally, he can nod his head to indicate 'yes' when asked questions. Flaccid paralysis is present in his left arm and left leg, with no response noted to touch in those extremities. (He is left handed.) Visual fields are decreased in a pattern consistent with homonymous hemianopia. A CT scan, negative on admission, is repeated on the day after admission and confirms the medical diagnosis of a right-brain stroke due to a thrombus of the middle cerebral artery.

Mr Boren's medical treatment includes heparin sodium administered by continuous intravenous drip, with clotting studies to be performed every 4 hours and the dose adjusted accordingly.

DIAGNOSES

- *Risk of feeding self-care deficit* related to loss of the ability to use the left hand and arm and manifested by a failure to feed self.
- *Risk of impaired physical mobility* related to neurological deficits causing left hemiplegia and manifested by difficulty mobilising.
- *Risk of impaired skin integrity* related to inability to change position and manifested by the development of pressure area sores.
- *Risk of disturbed visual sensory perception* related to changes in visual fields and manifested by difficulty in performing activities of daily living (ADLs).
- *Risk of impaired verbal communication* related to cerebral injury and manifested by slurred or incoherent speech.

PLANNING

- Arrange mealtimes so that Mr Boren is sitting up by the window in a clean and private environment.
- Encourage Mrs Boren to visit at mealtimes, to assist with meals and periodically to bring a favourite food from home.
- Establish and maintain a regular schedule for turning when Mr Boren is in bed.
- Ensure marker and paper are available for alternative communication.

Expected outcomes

- Learn to use his right hand to feed himself.
- Participate in exercises necessary to maintain muscle strength and tone.
- Maintain skin integrity.
- Indicate understanding that visual fields may improve in a few weeks.
- Practise and implement speech therapy activities while at the same time using alternative methods of communication.

IMPLEMENTATION

- Provide adaptive devices (silverware with thick handles and non-slip plates).
- Provide passive ROM exercises for his left arm and leg; schedule active ROM exercises for his right extremities, as well as quadriceps and gluteal sets every 4 hours during waking hours.
- Keep his skin clean and dry at all times.
- Place objects (e.g. call bell, tissues) on unaffected side and approach him from that side.
- Support attempts to communicate verbally; when he is not understood, he may prefer to use a large marker and tablet.

EVALUATION

Mr Boren is discharged to his home after being in the hospital for 10 days. During the first 2 months after discharge, Martha Grimes, RN, the community health nurse, visits Mr and Mrs Boren at home. At the end of 2 months, Mr Boren is using his right hand to feed himself. He has regained partial use of his left arm and leg, and is using a walker to move around the house and yard; he is even able to work in his flower garden. His skin has remained intact and his vision is back to normal. He is slowly relearning speech; this has been the most difficult change for him to accept.

CRITICAL THINKING IN THE NURSING PROCESS

1. Hypertension is sometimes referred to as 'the silent killer'. Provide justifications for this statement.
2. What would be your reply if, after you had completed passive ROM on Mr Boren's left arm, he wrote: 'I just ignore that part of my body—it doesn't work anyway.'?
3. Which communication and education strategies would you implement to assist Mr Boren and his family?

REFLECTION ON THE NURSING PROCESS

1. Reflecting on your experience with people who have suffered a stroke, which allied team members should become involved in Mr Boren's care? Why?
2. What information could you provide to Mr Boren's wife to assist her when Mr Boren is discharged home?

FIGURE 42.4 ■ Positioning the person with hemiplegia is important in preventing deformity of the affected extremities. *A*, With the person in a supine position, place a pillow in the axilla (to prevent adduction) and under the hand and arm, with the hand higher than the elbow (to prevent flexion and oedema). *B*, When the person is lying supine, use a pillow from the iliac crest to the middle of the thigh to prevent external rotation of the hip. *C*, When the person is in the prone position, place a pillow under the pelvis to promote hip hyperextension

ADLs as possible to promote functional ability, increase independence, decrease feelings of powerlessness and improve self-esteem.

Before establishing a plan to increase self-care, determine which hand was dominant before the stroke. If the person's dominant side is affected, self-care will be more difficult.

- Encourage use of the unaffected arm to bathe, brush teeth, comb hair, dress and eat. *Use of the unaffected arm promotes functional ability and independence.*
- Teach the person to put on clothing by first dressing the affected extremities and then dressing the unaffected extremities. *This technique facilitates self-dressing with minimal assistance.*
- Collaborate with the occupational therapist in scheduling times for training for upper extremity functioning necessary for ADLs. Encourage the use of assistive devices (if required) for eating, physical hygiene and dressing. *Following a regular schedule in daily routines promotes learning. The use of assistive devices promotes independence and decreases feelings of powerlessness. Optimal grooming facilitates positive self-concept.*

Impaired verbal communication

The person who loses communication abilities requires intensive speech therapy and emotional support. It is important to determine the specific nature of the impairment when planning interventions and helping family members understand specific problems. Although the speech therapist is usually most involved with speech rehabilitation, nurses must plan interventions to meet communication needs during all phases of care.

Use the following guidelines:

- Approach and treat the person as an adult.
- Do not assume that the person who does not respond verbally cannot hear. Do not use a raised voice when addressing the person.
- Allow adequate time for the person to respond.
- Face the person and speak slowly.
- When you do not understand the person's speech, be honest and say so.
- Use short, simple statements and questions. *Accepting the person and providing dignity and respect enhances the nurse–person relationship. Allowing adequate response time and using short verbal statements or questions while facing the person motivates the person to communicate and decreases frustration.*
- Accept frustration and anger as a normal reaction to the loss of function. *Anger represents the person's frustration at the inability to control the loss of function.*
- Try alternative methods of communication, including writing tablets, flash cards and computerised talking boards. *The person who is unable to communicate verbally may use other methods effectively.*

Risk of impaired urinary elimination and constipation

Both urinary and bowel elimination may be altered because of neurological deficits, impaired mobility, cognitive impairment, communication deficits or pre-existing problems (especially if the person is an older adult). Other causes include changes in food and fluid intake and side effects of medications. Urinary incontinence or retention, and constipation and faecal impaction are the usual manifestations.

- Assess for urinary frequency, urgency, incontinence, nocturia and voiding in small amounts. In addition, assess the person's ability to respond to the need to void, to use the call light and to use toileting equipment.

CONSIDERATION FOR PRACTICE

Voiding small amounts of urine frequently may be a manifestation of a bladder dysfunction. Assess for a distended bladder.

- Encourage bladder training by having the person void on schedule, such as every 2 hours, rather than in response to the urge to void.
- Educate the person about Kegel exercises. To perform Kegel exercises, the person contracts the perineal muscles as though stopping urination, holds the contraction for 5 seconds and then releases.
- Use positive reinforcement (verbal praise) for successful management of urinary elimination.
  Voiding every 2 hours or on schedule promotes bladder tone and urine storage. Kegel exercises increase pubococcygeal muscle tone and bladder control, decreasing incontinence. Positive reinforcement can be a useful part of the teaching program.
- Discuss pre-stroke bowel habits, as well as the pattern of bowel elimination since the stroke.
- If the person is able to swallow without difficulty, encourage fluids (up to 2000 mL per day) and a high-fibre diet.
- Increase physical activity as tolerated.
- Assist in using the toilet facilities at the same time each day (based on usual patterns of bowel elimination), ensuring privacy and having the person sit in an upright position if at all possible.
- Administer prescribed stool softeners if the person is following a bowel elimination routine or is not drinking sufficient fluids.
  Increased fluids, fibre and activity stimulate intestinal motility. Establishing a regular daily time for bowel movements in the upright position and in privacy promotes normal bowel elimination. Stool softeners help prevent the formation of hard stool that is more difficult to expel.

Impaired swallowing

A stroke may impair the ability to swallow. Weakness or lack of coordination of the tongue, attention deficits and deficits involving the swallowing reflex all play a role. Dysphagia (difficulty swallowing) may result in choking, drooling, aspiration or regurgitation. Nursing care focuses on maintaining safety by preventing aspiration and on ensuring adequate nutrition.

- Monitor results of swallowing studies prior to providing oral food and fluids. The speech therapist should be consulted to access the person for the most appropriate diet.
- Ensure safety when eating:
  - Position in upright sitting position with neck slightly flexed.
  - Order puréed or soft food. Liquids should be of the same consistency as honey.
  - Feed or teach the person to eat by putting food behind the front teeth on the unaffected side of mouth and tilting the head slightly backwards. Teach to swallow one bite at a time.
  - Assess for coughing with eating or drinking. *Coughing may be indicative of dysphagia.*

CONSIDERATION FOR PRACTICE

After eating, check the mouth for 'pocketing' of food, especially in the affected cheek.

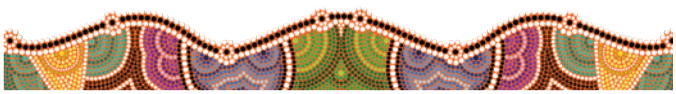
- Have suction equipment available at the bedside in case of choking or aspiration.
  Sitting upright with the head and neck first slightly flexed and then tilted back helps the person swallow. The person can usually swallow puréed or soft foods more easily than liquid or solid foods. Using the unaffected side of the mouth helps prevent food from collecting in the mouth and makes swallowing safer; in addition, food is less likely to fall out of the mouth.
- Monitor lung sounds. *Coarse lung sounds heard in the right upper and/or lower lobes may indicate aspiration as the right bronchus is the first division of the bronchi and where the majority of aspirations occur.*
- Minimise distractions and, if necessary, give step-by-step instructions for eating. *Distractions increase the risk of aspiration. Complex activities are easier to perform when broken down into small steps.*

Community-based care

Throughout the rehabilitation process, it is important to encourage self-care as much as possible but also to involve family members in the plan of care. Stress that ADLs may take twice as long as they did before the stroke. Emphasise that physical function may continue to improve for up to 3 months and speech may continue to improve for even longer. Address the following topics in preparing the person and family for community-based care:

- physical care, medications, physiotherapy, occupational therapy, speech therapy
- realistic expectations
- time off for the caregiver; respite care services
- distributors for equipment and supplies
- home environment conducive to using equipment (e.g. a wheelchair or walker)
- home and equipment modifications (e.g. a raised toilet seat, grab bars in the bathroom, a bath chair, a vise lid opener, a long-handled shoehorn)
- home health services
- community resources, such as Meals on Wheels, senior centres, elder care, large-print telephone buttons, stroke clubs, VitalCall (emergency alerting systems through a local hospital

or agency). Financial assistance may be available within the community for housekeeping and personal care assistance
- Helpful organisations:
  - National Stroke Foundation (with state branches): https://strokefoundation.com.au
  - StrokeLine: 1800 787 653
  - Stroke Association of ACT: 6269 2636
  - Stroke Association of Queensland: 3277 3838
  - Stroke Association of Victoria: http://www.strokeassociation.com.au
  - Stroke Recovery Association of NSW: http://www.strokensw.org.au
  - Stroke SA Inc.: http://stroke.org.au



THE PERSON WITH AN INTRACRANIAL ANEURYSM

An **intracranial aneurysm** is a saccular outpouching of a cerebral artery that occurs at the site of a weakness in the vessel wall. The weakness may be the result of atherosclerosis, a congenital defect, trauma to the head, ageing or hypertension. A ruptured cerebral aneurysm is the most common cause of a haemorrhagic stroke.

Incidence and prevalence

Studies suggest the average prevalence of intracranial aneurysms is around 2% of the population; most go through life without any manifestations of bleeding. However, it is estimated that 50–80% of these will never rupture (Brain Aneurysm Foundation, 2011). Intracranial aneurysms are most common in adults aged 30 to 60 (Hickey, 2013).

The exact aetiology is unknown, but theories of cause include: (1) a developmental defect in the vessel wall; and (2) degeneration or fragility of the vessel wall due to conditions such as hypertension, atherosclerosis, connective tissue disease or abnormal blood flow. Hypertension and cigarette smoking may be contributing factors.

Pathophysiology

Intracranial aneurysms tend to occur at the bifurcations and branches of the carotid arteries, and the vertebrobasilar arteries at the circle of Willis, with most aneurysms (85%) located anteriorly. They range in size from smaller than 15 mm to larger than 50 mm. Intracranial aneurysms tend to enlarge with time, making the vessel wall thin and increasing the probability of rupture.

There are several different types of intracranial aneurysms. A *berry aneurysm* is probably the result of a congenital abnormality of the tunica media of the artery. The aneurysm usually ruptures without warning. A *saccular aneurysm* is any aneurysm with a saccular outpouching which distends only a small portion of the vessel wall. This type of aneurysm is often caused by trauma. In a *fusiform aneurysm*, the entire circumference of a blood vessel swells to form an elongated tube. Most aneurysms of this type occur as a result of the changes of arteriosclerosis. Fusiform aneurysms act as space-occupying lesions. In a *dissecting aneurysm*, the tunica intima pulls away from the tunica media of the artery and blood is forced between the two layers. It may result from atherosclerosis, inflammation or trauma. *Mycotic aneurysms* are caused by emboli from infections such as bacterial endocarditis.

Intracranial aneurysms typically rupture from the dome rather than the base, forcing blood into the subarachnoid space at the base of the brain. The aneurysm may also rupture and force blood into brain tissue, the ventricles or the subdural space. This discussion focuses on intracranial haemorrhages due to rupture of a cerebral aneurysm. See Chapter 41 for further discussion of types of intracranial bleeding and haematomas.

Manifestations

An intracranial aneurysm is usually asymptomatic until it ruptures, although very large aneurysms may cause headache and/or neurological deficits due to pressure on adjacent intracranial structures. Small leakages of blood may occur periodically, causing headache, nausea, vomiting and pain in the neck and back. The person may also have prodromal manifestations before the rupture occurs, such as headache, eye pain, visual deficits and a dilated pupil.

The manifestations of a ruptured intracranial aneurysm (and subsequent subarachnoid haemorrhage) include a sudden, explosive headache; loss of consciousness; nausea and vomiting; a stiff neck and photophobia (due to meningeal irritation); cranial nerve deficits; stroke syndrome manifestations; and pituitary malfunctions (that result primarily from changes in ADH secretion).

The severity of the rupture is often inferred from the manifestations of the subarachnoid haemorrhage. The Hunt–Hess classification of subarachnoid manifestations is frequently used to classify non-traumatic subarachnoid haemorrhages. The grades of severity are:
- grade 1—asymptomatic or minimal headache and slight neck rigidity
- grade 2—moderate to severe headache, neck rigidity, cranial nerve deficits
- grade 3—drowsy, lethargic, mild neurological deficits
- grade 4—stuporous, moderate to severe hemiparesis, early decerebrate rigidity
- grade 5—deep coma, decerebrate rigidity, moribund appearance.

Fibrin and platelets seal off the bleeding point, but the escaped blood forms a clot that irritates the brain tissue. The resulting inflammatory response causes cerebral oedema and both the oedema and the haemorrhage increase intracranial pressure (Hickey, 2013). Bleeding into the subarachnoid space causes meningeal irritation. Hypothalamic dysfunction and seizures are also potential complications.

Complications

The main complications of a ruptured intracranial aneurysm are rebleeding, vasospasm and hydrocephalus.

Rebleeding

The greatest risk of rebleeding is within the first day after the initial rupture and again in 7 to 10 days (when the initial clot breaks down). Rebleeding is manifested by a sudden severe

headache, nausea and vomiting, decreasing levels of consciousness and new neurological deficits (Hickey, 2013). The mortality from rebleeding is as high as from the initial rupture.

Vasospasm

Cerebral vasospasm is a common but dangerous complication that occurs between 3 and 10 days after a subarachnoid haemorrhage. It is associated with a large number of deaths and disability. A cerebral vasospasm narrows the lumen of one or more cerebral vessels, causing ischaemia and infarction of tissue supplied by the affected vessels. The actual cause is unknown, but it occurs in blood vessels surrounded by thick blood clots, suggesting that some substance in the clot initiates the spasm. The manifestations vary according to the degree of spasm and the area of brain affected. Regional alterations may cause focal deficits (such as hemiplegia), whereas global alterations cause loss of consciousness.

Hydrocephalus

Hydrocephalus, an abnormal accumulation of CSF within the cranial vault and dilation of the ventricles, is a potential complication of a ruptured intracranial aneurysm. Hydrocephalus is thought to be the result of obstruction of reabsorption of CSF through the arachnoid villi. The obstruction is caused by an increased protein content of the CSF because of lysis of blood in the subarachnoid space (Porth, 2010). The accumulation of CSF increases intracranial pressure. Initial manifestations of hydrocephalus are typically non-specific but commonly include decreasing levels of consciousness.

INTERPROFESSIONAL CARE

The care of the person with a ruptured intracranial aneurysm includes determining the location of the aneurysm, treating the manifestations of the haemorrhage and preventing rebleeding and vasospasm. Interventions using radiology, angiography and a variety of procedures may prevent aneurysm rupture or stop the bleeding. Surgery is usually the treatment of choice to repair the bleeding artery.

Diagnosis

The diagnostic tests conducted to identify the site and extent of a ruptured intracranial aneurysm, as well as rebleeding, are a CT scan and bilateral carotid and vertebral cerebral angiograms. A cerebral angiogram is the gold standard for evaluating cerebral aneurysm; it can demonstrate the source of the aneurysm about 80–85% of the time (Hickey, 2013). A lumbar puncture will reveal blood-tinged spinal fluid. These tests are described in Chapter 40.

Medications

Calcium channel blockers, such as nimodipine (Nimotop), are used to improve neurological deficits due to vasospasm following subarachnoid haemorrhage from ruptured intracranial aneurysms. Administered for 21 consecutive days, they have been found to enhance collateral blood flow and reduce the incidence of ischaemic deficits from arterial spasm without side effects (Hickey, 2013).

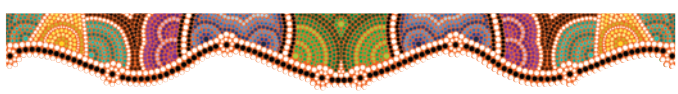
Other medications that may be prescribed include anticonvulsants, such as phenytoin (Dilantin), to prevent seizures if the person has increased intracranial pressure; analgesics for headache; and stool softeners to prevent constipation and straining with a bowel movement (which increases intracranial pressure and blood pressure, and may cause rebleeding).

Procedures used to treat aneurysm

Treatments for an intracranial aneurysm are performed either to prevent rupture or to isolate the vessel to prevent further bleeding. The person with good neurological status may have surgery soon after the rupture. In the person with significant neurological deficits, surgery may be delayed until they are more stable and less at risk of vasospasm; however, the trend is towards surgery as soon as possible.

Several different types of procedures are used to repair a ruptured intracranial aneurysm or to prevent the rupture of an existing large aneurysm. These include:

- The skull is opened (craniotomy) and the aneurysm is located. The neck of the aneurysm may be clipped with a metal clip (preventing the entry of blood into the aneurysm) or the involved artery may be clipped both proximally and distally to the aneurysm to isolate the affected area.
- Endovascular Guglielmi detachable coils (GDCs) are a method used to treat non-ruptured aneurysms. One or more small platinum coils are inserted through a microcatheter and threaded through the carotid or femoral artery to the site of the aneurysm, where they are released and fill the body of the aneurysm. The coils initiate the immune response and the body produces a blood clot inside the aneurysm, strengthening the artery walls and reducing the risk of rupture. After the aneurysm is stabilised, it can be clipped with less risk of haemorrhage and death (NINDS, 2012a). However, endovascular coil procedures are also effective without surgery, especially for smaller aneurysms.
- Stents, which are coil or mesh tubes introduced into the body through a catheter, are used to cover the neck of an aneurysm, while coils are deposited within the body of the aneurysm.
- Balloon remodelling is used for large, multiple or surgically inaccessible aneurysms. A balloon is placed across the neck of the aneurysm and coils are inserted into the body of the aneurysm. The balloon prevents the coils from moving out to the aneurysm.
- Parent vessel occlusion is performed to occlude the parent vessel that supplies blood to the aneurysm. Prior to permanent occlusion, the risk of neurological impairment is assessed by monitoring motor, sensory and cognitive functions in an awake person while temporary occlusion is conducted.



Nursing care

Nursing care is planned and implemented for the person with a ruptured intracranial aneurysm to prevent rebleeding, as well as to meet needs resulting from neurological deficits.

Nursing diagnoses and interventions

Appropriate nursing diagnoses and interventions are described earlier in the chapter in the discussion of nursing care for the person with a stroke. The priority interventions in the acute care stage of a ruptured intracranial aneurysm focus on ineffective cerebral tissue perfusion.

Ineffective tissue perfusion: cerebral

These interventions are for the care of the person immediately after the intracranial aneurysm ruptures. The expected outcome of care is preventing rebleeding and improving cerebral tissue perfusion.

- Institute aneurysm precautions to prevent rebleeding, as follows:
  - Keep the person in a private, quiet, darkened room. Disconnect or remove the telephone. Avoid using bright overhead lights. *A quiet environment helps prevent an increase in blood pressure, which could precipitate rebleeding. The person may experience photophobia (abnormal sensitivity to light) if haemorrhage has damaged the oculomotor nerve.*
  - Elevate the head of the bed 30 to 45 degrees; follow prescribed activity orders (usually complete bed rest, but in some cases bathroom privileges may be approved). *Elevating the head of the bed promotes venous return from the brain and thus decreases intracranial pressure. Decreasing activity reduces the likelihood of increases in blood pressure.*
  - Limit visitors to two family members at any one time and limit the duration of visits. Monitor the person's response to visitors and decrease interactions if the person becomes agitated or upset. *Psychological stress may increase blood pressure and the risk of rebleeding; however, social isolation may increase anxiety and stress. Each person (and family) must be individually evaluated.*
  - Allow reading, watching television or listening to the radio to promote relaxation. *Although these passive activities were previously contraindicated for the person on aneurysm precautions, current therapy is based on the belief that these activities promote relaxation and help control blood pressure.*
- Prevent constipation and straining to have a bowel movement. Administer stool softeners as prescribed. Collaborate with the person and doctor about use of a bedside commode or the bathroom. Do not administer enemas. *The person is at risk of constipation as a result of decreased mobility and the administration of narcotics (such as codeine) for headache. When straining to have a bowel movement, the person uses the Valsalva manoeuvre, which increases intracranial pressure and may precipitate rebleeding.*
- If the person is alert (and depending on doctor preference), allow them to feed self and provide own personal care. *In many instances, self-care causes less anxiety and stress than care provided by the nurse. The extent of care provided varies according to the person's condition and the doctor's preferences. The person who may have to go to theatre will be kept nil by mouth.*

CONSIDERATION FOR PRACTICE

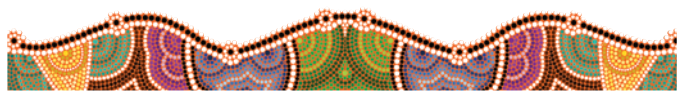
Maintaining a daily stool chart is an important assessment in preventing constipation.

- Monitor vital signs and neurological status as indicated by the person's condition. (Frequency of assessments may range from every 15 minutes to every 4 hours.) *Vital signs and neurological assessments provide ongoing data for evaluation of changes indicative of increasing intracranial pressure and decreasing neurological function. Report any change immediately to the doctor.*

CONSIDERATION FOR PRACTICE

Restlessness and changes in respirations are often early manifestations of increased intracranial pressure.

- Maintain seizure precautions: have suction equipment and an oropharyngeal tube at the bedside, maintain the bed in the low position and keep the side rails padded and raised. *Applying suction and inserting an oropharyngeal airway may be necessary to maintain an open airway in case of seizure. A lowered bed and padded, raised side rails prevent injury if a seizure occurs.*
- Avoid positioning and activities that increase intracranial pressure such as coughing, sneezing, vomiting, sharply flexing the neck, blowing the nose, enemas or moving self up in bed. *These measures help to prevent increasing intracranial pressure and rebleeding.*



THE PERSON WITH AN ARTERIOVENOUS MALFORMATION

An arteriovenous (AV) malformation is a congenital intracranial lesion, formed by a tangled collection of dilated arteries and veins that allows blood to flow directly from the arterial into the venous system, bypassing the normal capillary network. Most AV malformations (90%) are located in the cerebral hemispheres; the remainder are found in the cerebellum and brainstem.

Rupture of vessels in the malformations accounts for 2% of all strokes. People with this condition develop manifestations before 40 years of age; it affects men and women equally (Porth & Matfin, 2014). The manifestations are the result of spontaneous bleeding from the lesion into the subarachnoid space or brain tissue.

Pathophysiology

AV malformations displace, rather than encompass, normal brain tissue (Hickey, 2013). The pathophysiological effects of an AV malformation are the result of the shunting of blood from the arterial to the venous system and of altered perfusion of cerebral tissue near the malformation. The shunting of arterial blood directly into the venous system within the malformation transfers the higher arterial pressure directly into the lower-pressure venous

system. This increased pressure is likely to cause spontaneous bleeding or progressive expansion and rupture of a blood vessel.

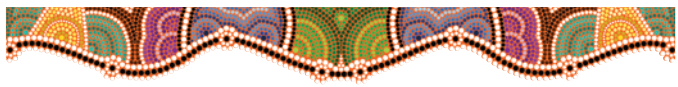
Altered cerebral perfusion results when blood flow through a large, high-flow malformation is diverted from the normal cerebral circulation, causing tissue ischaemia of the area surrounding the malformation. This is sometimes called a vascular 'steal' phenomenon.

AV malformations range in size from very small to very large. Large malformations are usually initially manifested by seizure activity. In contrast, the manifestations of a small malformation are more often due to a haemorrhage that causes neurological deficits. In both instances, the person may have recurrent headaches that do not respond to treatment.

INTERPROFESSIONAL CARE

AV malformations are diagnosed with the same diagnostic tests used to diagnose an intracranial aneurysm.

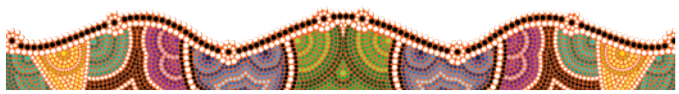
If the malformation is accessible, the ideal treatment is excision of the malformation and removal of any haematoma. Large malformations may be treated by embolisation. In this procedure, substances such as Gelfoam or metallic pellets are introduced into the involved area of the cerebral circulation, where they form emboli and gradually obstruct blood flow in the malformation. Inaccessible malformations are also treated with radiation therapy or laser therapy to coagulate blood in the malformation and thicken its vascular elements, eventually obstructing it. When the malformation is excised or obstructed, blood flow is no longer shunted and cerebral perfusion improves.



Nursing care

Nursing care depends on the condition of the malformation. If haemorrhage has not occurred, teach the person to avoid activities that raise blood pressure or could cause injury. The person is usually given medications to control blood pressure and prevent seizures.

If the malformation ruptures and causes an intracranial haemorrhage, nursing care is the same as for any person who has had a haemorrhagic stroke (discussed earlier in this chapter).



SPINAL CORD DISORDERS

THE PERSON WITH A SPINAL CORD INJURY

Nursing care of the person with a spinal cord injury takes place from the acute management phase through ongoing rehabilitation in a variety of settings. Although priorities of care may change depending on the person and the setting, care focuses on maximising functional health status to preserve quality of life. The nurse provides care and also collaborates with other healthcare professionals in meeting this goal.

Incidence and prevalence

A **spinal cord injury (SCI)** is usually due to trauma. The main causes of SCI are contusion, laceration, transection, haemorrhage and damage to blood vessels that supply the spinal cord. If vertebrae are fractured and ligaments are torn, bony fragments can damage the cord and make the spinal column unstable. Injury to blood vessels supplying the cord can cause permanent damage. The injury is identified by vertebral level. For example, a C6 spinal cord injury is at the sixth cervical vertebra.

Risk factors

The three main risk factors for SCIs are age, gender and alcohol or drug abuse. Young men are more prone than women to risk-taking behaviours. Older adults are more likely to have a cord injury from even minor trauma as a result of age-related vertebral degeneration. Motor vehicle crashes while under the influence of alcohol or drugs are a major source of trauma to people of all ages.

FAST FACTS

- Approximately 300–400 Australians have an SCI each year.
- An estimated 10 000 people in Australia are living with SCIs.
- The cost associated with care of people with SCIs is estimated at $500 million per year.
- The majority of SCIs (79%) are due to traumatic injuries such as motor vehicle crashes, acts of violence, falls and sports injuries.
- The highest prevalence of SCI occurs in the 15–24-year-old age group.

Source: AIHW (2010).

Pathophysiology

The spinal cord provides a two-way pathway for the conduction of impulses and information to and from the brain and the body, serves as a major reflex centre and (through its attached spinal nerves) is involved in the sensory and motor innervation of the entire body below the head. It consists of an outer region of white matter and an inner region of grey matter. The grey matter comprises the central canal of the cord, the posterior horns, the anterior horns and the lateral horns. It is divided into a sensory half (dorsally) and a motor half (ventrally) and innervates somatic and visceral regions of the body. The white matter consists of tracts or pathways that convey information. The ascending (sensory)

pathways carry information about proprioception, fine touch, discrimination, pain, temperature, deep pressure and touch. The descending (motor) pathways carry information about movement. The pyramidal tracts control skilled voluntary movements (such as writing). The extrapyramidal tracts (all tracts other than the pyramidal tracts) bring about all other body movements. See Chapter 40 for further information.

When the spinal cord is injured, the primary injury causes microscopic haemorrhages in the grey matter of the cord and oedema of the white matter of the cord. These initial pathological changes are followed by the secondary injury, with mechanisms that increase the area of injury. The haemorrhages extend, eventually involving the entire grey matter. Microcirculation to the cord is impaired by oedema and haemorrhage. The injured tissue releases noradrenaline, serotonin, dopamine and histamine; these vasoactive substances cause vasospasm and further decrease microcirculation. As a result, vascular perfusion and oxygen tension of the affected area are decreased, which leads to ischaemia.

When ischaemia is prolonged, necrosis of both grey and white matter begins within a few hours and within 24 hours the function of nerves passing through the injured area is lost. Although circulation returns to the white matter of the cord in about 24 hours, decreased circulation in the grey matter continues. Because oedema extends the level of injury for two cord segments above and below the affected level, the extent of injury cannot be determined for up to 1 week.

Tissue repair occurs over a period of 3 to 4 weeks. Phagocytes enter the area 36 to 48 hours after the initial injury. Neurons degenerate and are removed by microphages in the first 10 days after the injury. RBC disintegrate and the haemorrhages are reabsorbed. Eventually the area of injury is replaced by acellular collagenous tissue and the meninges thicken.

Forces resulting in SCI

SCIs are the result of the application of excessive force to the spinal column. The most common causes of abnormal spinal column movements are acceleration and deceleration (forces that are applied to the body; for example, in motor vehicle crashes and falls). Acceleration occurs when external force is applied in a rear-end collision; the upper torso and head are forced backwards and then forwards. Deceleration occurs in a head-on collision; the external force is applied from the front. The head and body move forwards until they meet a stationary object and then are forced backwards. The following forces and movements (see Figure 42.5) may cause a variety of SCIs, with the extent of injury depending on the amount and direction of motion and the rate of application of force:

- *Hyperflexion*, or forcible forward bending, may compress vertebral bodies and disrupt ligaments and intervertebral discs.
- *Hyperextension*, or forcible backward bending, often disrupts ligaments and causes vertebral fractures. A whiplash injury is a less severe form of hyperextension, with injury to soft tissues but no vertebral or spinal cord damage.
- *Axial loading*, a form of compression, is the application of vertical force to the spinal column (e.g. by falling and landing on the feet or buttocks, or by diving into shallow water).

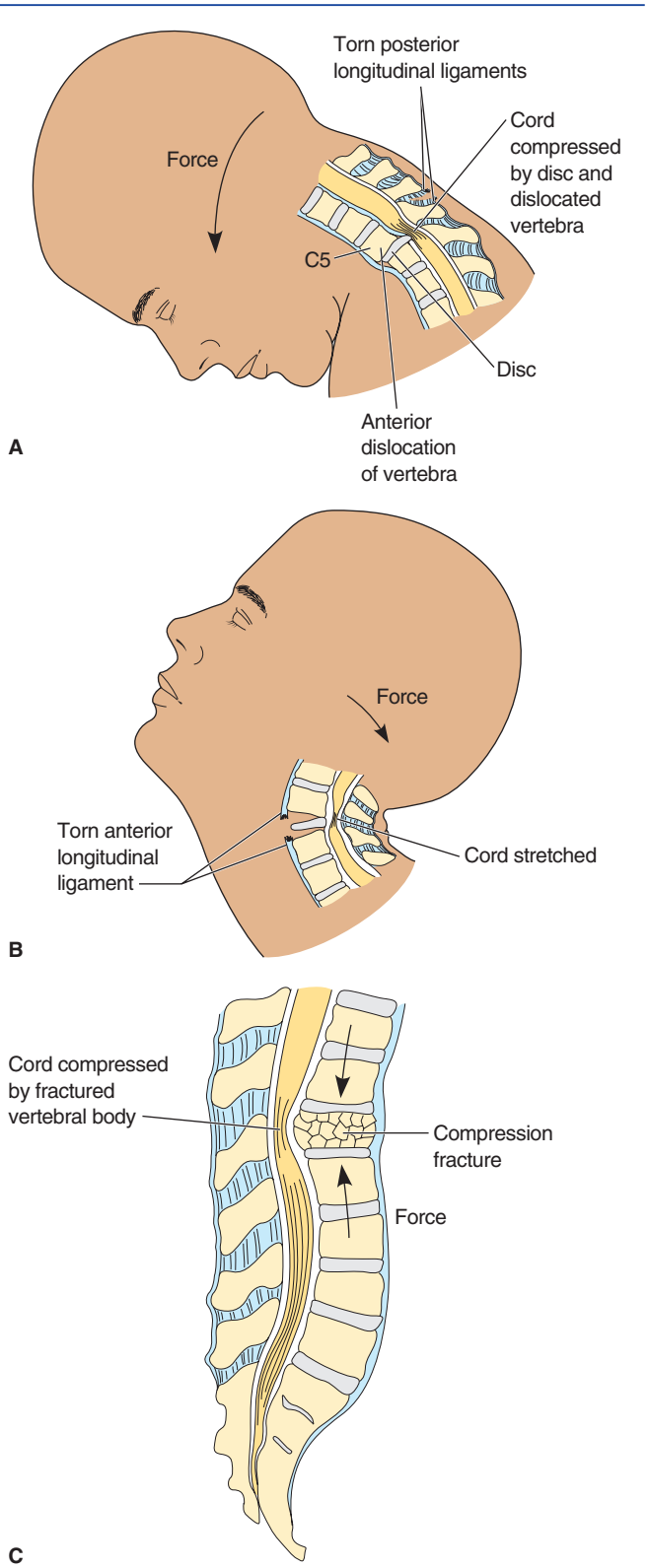


FIGURE 42.5 ■ Spinal cord injury mechanisms. *A*, Hyperflexion. *B*, Hyperextension. *C*, Axial loading, a form of compression

- *Excessive rotation*, in which the head is excessively turned, may tear ligaments, fracture articular surfaces and cause compression fractures.

The alteration of the spinal cord and soft tissues caused by these abnormal movements is called deformation.

The spinal cord may be penetrated by bullets and other foreign objects (e.g. sharp objects used as weapons, shrapnel from explosions). Penetrating injuries may cause vertebral fractures, tear ligaments and muscles, or cut through a part or all of the spinal cord. Complete severing of the cord is rare.

Sites of pathology

Injuries occur most often in the lumbar and cervical regions. The most frequent sites of injury of the cord are at the first, second and fourth to sixth cervical vertebrae (C1, C2, C4 to C6); and the eleventh thoracic to second lumbar vertebrae (T11 to L2). Because the cervical spine has a wider range of movement than the rest of the spine, the cervical portion is more likely to be affected by externally applied forces. In addition, the cord fills most of the vertebral canal in the cervical and lumbar regions and thus is more easily injured. Damage to the vertebrae and ligaments causes the spinal column to become unstable, increasing the possibility of compression or stretching of the spinal cord with any further movement.

Classification of SCIs

SCIs are classified according to systems—for instance: (1) as complete or incomplete cord injury, (2) by cause of injury, and (3) by level of injury. In clinical practice, these classifications often overlap. In a *complete SCI* (about 45% of all injuries), the motor and sensory neural pathways are completely interrupted (transected), resulting in total loss of motor and sensory function below the level of the injury. However, 'complete' does not necessarily mean the spinal cord has been severed. In an *incomplete SCI* (about 55% of all injuries), the motor and sensory pathways are only partially interrupted, with variable loss of function below the level of injury. Incomplete SCIs are further classified into syndromes, as outlined in Table 42.2. Both complete and incomplete injuries can occur in paraplegia and quadriplegia. The alterations in function that occur as the result of SCIs vary greatly depending on the amount of tissue damage and the level of injury.

Manifestations

The spinal cord, the vertebrae, the intervertebral discs, the spinal nerves, the ligaments and the surrounding soft tissue structures are in such close anatomical proximity that any condition or injury affecting one structure may well affect any one or all of the other structures. The conditions with the most critical effects are disorders affecting the spinal cord. Disorders and injuries of the spinal cord have the potential to affect movement, perception, sensation, sexual function and elimination. Manifestations and complications of SCI by body system are listed in the box below.

Spinal shock is the temporary loss of reflex function (called *areflexia*) below the level of injury. This response begins immediately after complete transection of the spinal cord, when connections between the brain and the spinal cord are interrupted and the cord does not function at all. The response also occurs (although in varying degrees) after partial transection, as well as after spinal cord contusions, compression and ischaemia.

Normal activity of the spinal cord is dependent on constant impulses from the higher centres of the brain. When damage from an injury stops these impulses, spinal shock follows. There is loss of motor function, tendon reflexes and autonomic function. Spinal shock may begin within 1 hour of the injury. The condition may last from a few minutes to several months

TABLE 42.2 Incomplete spinal cord injury syndromes

TYPE	CAUSE	LOCATION	DEFICITS
Central syndrome	Cord transection Hyperextension	Cervical	Spastic paralysis of the upper extremities Variable paralysis of the lower extremities Variable effects on the bowel, the bladder and sexual function
Anterior syndrome	Damage to the anterior spinal artery Infarction of the anterior spinal artery Hyperflexion	Anterior two-thirds of the cord	Paralysis below the level of injury Loss of temperature and pain sensation below the level of injury
Posterior syndrome	Vertebral dislocation Herniated disc Compression	Nerve roots	Weakness in isolated muscle groups Tingling, pain Decreased or absent reflexes in the involved area Bowel or bladder dysfunction
Brown–Séquard syndrome	Penetrating trauma	Hemisection of the anterior and posterior cord	Paralysis below the level of injury on the ipsilateral (same) side of the body Contralateral loss of temperature and pain sensation below the level of injury Ipsilateral loss of proprioception below the level of injury
Homer's syndrome	Incomplete cord transection	Cervical sympathetic nerves	Ipsilateral ptosis of the eyelid, constricted pupil and facial anhidrosis (inability to perspire)

MANIFESTATIONS AND COMPLICATIONS Spinal cord injury by body system

INTEGUMENT
- Decubitus (pressure) ulcers

NEUROLOGICAL
- Pain
- Areflexia
- Hypotonia
- Autonomic dysreflexia

CARDIOVASCULAR
- Spinal shock
- Paroxysmal hypertension
- Orthostatic hypotension
- Cardiac arrhythmias
- Decreased venous return
- Hypercalcaemia

RESPIRATORY
- Limited chest expansion
- Decreased cough reflex
- Decreased vital capacity

GASTROINTESTINAL
- Stress ulcers
- Paralytic ileus
- Stool impaction
- Stool incontinence

GENITOURINARY
- Urinary retention
- Urinary incontinence
- Neurogenic bladder
- Impotence
- Testicular atrophy
- Inability to ejaculate
- Decreased vaginal lubrication

MUSCULOSKELETAL
- Joint contractures
- Bone demineralisation
- Osteoporosis
- Muscle spasms
- Muscle atrophy
- Pathological fractures
- Paraplegia
- Quadriplegia

(although it usually lasts from 1 to 6 weeks) and then reflex activity returns. Spinal shock ends slowly, with the gradual reappearance of reflexes, hyperreflexia (increased reflex responses), muscle spasticity and reflex bladder emptying.

The manifestations of acute spinal shock (which vary in degree) include the following:

- flaccid paralysis of skeletal muscles below the level of injury
- loss of all spinal reflexes below the level of injury
- loss of sensations of pain, touch, temperature and pressure below the level of injury
- absence of visceral and somatic sensations below the level of injury
- bowel and bladder dysfunction
- loss of the ability to perspire below the level of injury.

A person with a cervical or upper thoracic SCI may also have neurogenic shock, resulting in cardiovascular changes. These changes are due to the inability of higher centres in the brainstem to modulate reflexes. As a result, vascular beds below the level of injury dilate and the cardiac accelerator reflex is suppressed. The person experiences hypotension and bradycardia. Other manifestations may include respiratory insufficiency due to loss of innervation of the diaphragm in C1 to C4 injuries, hypothermia, paralytic ileus, urinary retention and oliguria.

Both bradycardia and hypotension may persist even after the spinal shock resolves. In addition to losing sympathetic control of the heart rate, the person with a high-level SCI experiences decreased peripheral resistance and loss of muscle activity. These changes result in sluggish blood flow and decreased venous return, increasing the risk of thrombophlebitis.

Complications

The complications of an SCI involve many different body systems and result often in permanent disability and loss of functional health status. The complications include, but are not limited to, upper and lower motor neuron deficits, paraplegia and quadriplegia, and autonomic dysreflexia. Other complications, depending on the level and severity of the injury, are ineffective respirations, altered skin integrity, increased risk of thrombosis and alterations in bowel elimination, urinary elimination and sexual pattern.

Upper and lower motor neuron deficits

Injuries to the spinal cord are often classified as either *upper motor neuron lesions* or *lower motor neuron lesions*. Motor neurons are functional units that carry motor impulses. The upper motor neurons (located in the cerebral cortex, thalamus, brainstem and corticospinal and corticobulbar tracts) are responsible for voluntary movement. When these motor pathways are interrupted, the person experiences spastic paralysis and hyperreflexia and may be unable to carry out skilled movement.

Lower motor neurons (located in the anterior horn of the spinal cord, the motor nuclei of the brainstem and the axons that reach the motor end plate of skeletal muscles) are responsible for innervation and contraction of skeletal muscles. Interruption of lower motor neurons results in muscle flaccidity and extensive muscle atrophy, with loss of both voluntary and involuntary movement. If only some of the motor neurons supplying a muscle are affected, the person experiences partial paralysis (paresis); if all motor neurons to a muscle are affected, the person experiences complete paralysis. Hyporeflexia is also present.

Paraplegia and quadriplegia

Two common neurological deficits resulting from an SCI are paraplegia and quadriplegia (see Figure 42.2). **Paraplegia** is paralysis of the lower portion of the body, sometimes involving the lower trunk. Paraplegia occurs when the thoracic, lumbar and sacral portions of the spinal cord are injured, causing loss or impairment of sensory and/or motor function. **Quadriplegia**, also called *tetraplegia*, occurs when cervical segments of the cord are injured, impairing function of the arms, trunk, legs and pelvic organs.

Autonomic dysreflexia

Autonomic dysreflexia (also called *autonomic hyperreflexia*) is an exaggerated sympathetic response that occurs in the person with SCIs at or above the T6 level. This response, which is seen only after recovery from spinal shock, occurs as a result of a lack of control of the autonomic nervous system by higher

centres. When stimuli are unable to ascend the cord, mass reflex stimulation of the sympathetic nerves below the level of the injured cord area occurs, triggering massive vasoconstriction. In response, the vagus nerve causes bradycardia and vasodilation above the level of injury. If untreated, autonomic dysreflexia can cause seizures, a stroke or a myocardial infarction, and is potentially fatal (Hickey, 2013).

Autonomic dysreflexia is triggered by stimuli that would normally cause abdominal discomfort (a full bladder is the most common cause), by stimulation of pain receptors and by visceral contractions (Porth, 2010). Causes include faecal impaction, bladder infections or stones, intrauterine contractions, ejaculation, peritonitis and stimulation from pressure injuries or ingrown toenails. The most common precipitating event is a blocked urinary catheter.

The manifestations of this condition include: pounding headache; bradycardia; hypertension (with readings as high as 300/160); flushed, warm skin with profuse sweating above the lesion and pale, cold and dry skin below it; and anxiety (Porth, 2010). Dysreflexia is a neurological emergency and requires immediate treatment.

BOX 42.2 Assessment findings in acute SCI

Cervical injury
- Paralysis or weakness of extremities
- Respiratory distress manifested by changes in ABG studies, cyanosis, flaring of the nostrils, use of accessory muscles of respiration and restlessness
- Pulse rate below 60 and systolic BP below 80
- Decreased peristalsis

Thoracic and lumbar injury
- Paralysis or weakness of extremities

Spinal shock
- Loss of skin sensation
- Flaccid paralysis, areflexia
- Absent bowel sounds
- Bladder distension
- Decreasing blood pressure
- Absence of the cremasteric reflex in males (retraction of the left or right testicle in response to stimulation of the skin of, respectively, the inner left or right thigh)

INTERPROFESSIONAL CARE

The person with an acute SCI requires emergency assessment and care, and medications; sometimes the person also requires immobilisation and surgery. The person is first assessed and stabilised at the scene of the accident, initially treated in the emergency room and then admitted to the hospital's critical care unit.

Emergency care

The danger of death from SCI is greatest when there is damage to or transection of the upper cervical region. When the injury is at the C1 to C4 level, respiratory paralysis is common and the person who survives requires ventilator assistance to breathe. Injuries below C4 may increase the risk of respiratory failure if oedema ascends the cord. It is of critical importance not to complicate the initial injury by allowing the fractured vertebrae to damage the cord further during transport to the hospital. Although at one time injuries to the high cervical cord were almost always fatal, advances in trauma care have greatly improved the survival rate.

All people who have sustained trauma to the head or spine, or who are unconscious, should be treated as though they have a spinal cord injury. Pre-hospital management includes rapid assessment of the ABCs (airway, breathing, circulation), immobilising and stabilising the head and neck, removing the person from the site of injury, stabilising other life-threatening injuries and rapidly transporting the person to the appropriate facility. Guidelines for emergency care are as follows:

- Avoid flexing, extending or rotating the neck.
- Immobilise the neck, placing rolled towels or blankets on each side of the person's neck, or apply a cervical collar before moving the person onto a backboard.
- Secure the head by placing a belt or tape across the forehead and securing it to the stretcher.
- Maintain the person in the supine position.
- Transfer directly from the stretcher with backboard still in place to the type of bed that will be used in the hospital.

Assessment findings at the scene of the accident or in the emergency room vary according to the level of injury. The assessment findings common to the level of injury and spinal shock are outlined in Box 42.2.

The person in the emergency department with a suspected or identified SCI is also treated for respiratory problems, paralytic ileus, atonic bladder and cardiovascular alterations. Respiratory distress in the person with a cervical-level injury is treated by placing the person on a ventilator. Oxygen is administered to the person with a thoracic-level injury. Paralytic ileus (obstruction of the intestines due to lack of peristalsis) is common in the person with a spinal cord injury and is treated by the insertion of a nasogastric tube with connection to suction. To prevent over-distension of an atonic bladder, an indwelling catheter is inserted and connected to dependent drainage. Cardiovascular status is assessed on a continuous basis by inserting invasive monitoring devices, such as a Swan–Ganz catheter, or continuous cardiac output monitoring and attaching the person to a cardiac monitor, or by arterial monitoring to identify hypotension and to draw arterial blood gases (ABGs).

High-dose corticosteroid protocol using methylprednisolone must be implemented within 8 hours of the injury to improve neurological recovery. Clinical research indicates that the use of this adrenocorticosteroid is effective in preventing secondary spinal cord damage from oedema and ischaemia. Treatment with GM1 ganglioside for 3 to 4 weeks is an experimental approach that has been effective for some people (Papadakis & McPhee, 2015).

Diagnosis

Diagnostic tests are ordered to identify the level and extent of injury and to detect any complications. The tests include x-ray of the spine, CT or MRI of the spine and somatosensory evoked potential studies to locate the level of spinal cord injury by

stimulating peripheral nerves and measuring response times. ABGs are measured to establish a baseline or to identify problems due to respiratory insufficiency.

Medications

The pharmacological treatment of the person with an SCI is symptomatic. It is directed primarily towards decreasing oedema from the injury, treating hypotension and bradycardia, and treating spasticity.

- Corticosteroids, discussed earlier in this section, may be used to decrease or control oedema of the cord.
- Vasopressors are used in the immediate acute care phase to treat bradycardia or hypotension due to spinal and neurogenic shock. Examples of drugs are dopamine to treat hypotension in neurogenic shock and dobutamine to support cardiac function. Atropine should be available at the bedside to treat bradycardia.
- Antispasmodics are used to treat spasticity in the person with SCI. Both baclofen and diazepam may be used. A discussion of nursing implications of treatment with antispasmodics is found in the 'Medication administration' box below.
- Analgesics such as non-steroidal anti-inflammatory drugs (NSAIDs) and narcotics are administered to reduce pain.
- Proton pump inhibitors, such as omeprazole, esomeprazole, rabeprazole or pantoprazole, are often administered to prevent stress-related gastric ulcers, a common complication in SCI.
- Unless contraindicated, anticoagulants (heparin or warfarin) may be given to prevent thrombophlebitis.
- Stool softeners may be administered as part of a bowel retraining program.

Treatments

The treatments used in the management of an SCI include surgery, stabilisation and immobilisation.

SURGERY Early surgical treatment may be necessary if there is evidence of compression of the spinal cord by bone fragments or a haematoma. Surgery may also be done to stabilise and support the spine. However, many people are treated with stabilisation devices and do not require surgery. Surgeries that may be performed include a decompression laminectomy, a spinal fusion and insertion of metal rods. Surgeries of the spine are discussed later in the chapter.

STABILISATION AND IMMOBILISATION As a result of one or more dislocations or fractures of the cervical vertebrae, the person with an SCI may be immobilised in some type of traction or external fixation device to stabilise the vertebral column and prevent any further damage (see Figure 42.6).

FIGURE 42.6 ■ External fixation device

Source: © Rodolfo Arpia/Alamy.

MEDICATION ADMINISTRATION Antispasmodics in spinal cord injury

ANTISPASMODICS

Baclofen
Diazepam
Orphenadrine citrate

These drugs depress the CNS and inhibit the transmission of impulses from the spinal cord to skeletal muscle. They are used to control muscle spasm and pain associated with acute or chronic musculoskeletal conditions. They are not always effective in controlling spasticity resulting from cerebral or spinal cord conditions.

Nursing responsibilities

- Assess the person's spasticity and involuntary movements to obtain baseline data for comparison with results of therapy.
- Do not expect therapy to have effects for 1 week.
- Administer oral medications with food to decrease gastrointestinal symptoms.

Health education for the person and family

- These drugs may cause drowsiness, diplopia and impotence.
- Take your medications with meals to decrease gastric irritation.
- Physical improvement may take several weeks.
- Report slurred speech, drooling or inability to carry out usual functions to the doctor.
- Do not stop taking the medication without consulting your healthcare provider.

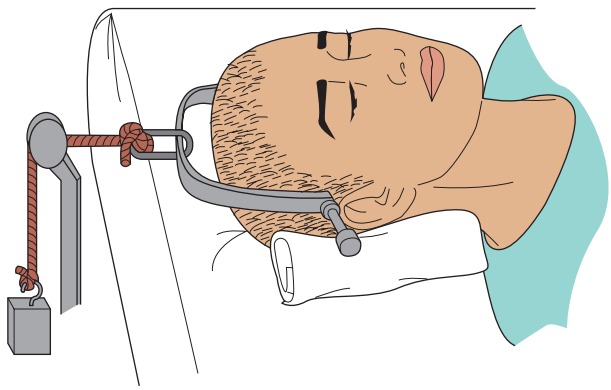


FIGURE 42.7 ■ Cervical traction may be applied by several methods, including Gardner–Wells tongs

Traction may also be used to stabilise the spinal column for the person who is not yet in a condition to have surgery or who has severe bleeding and oedema of the injured cord. The doctor applies the traction or fixation device; the nurse is responsible for assessments and interventions following the application.

Although used less frequently today, various devices provide cervical traction. For example, Gardner–Wells tongs may be used (see Figure 42.7). In this type of traction, the doctor applies pins to the skull, approximately 1 cm above each ear and weights are attached to the device.

The halo external fixation device is often used to provide stabilisation if there is no significant involvement of the ligaments (see Figure 42.8). It is most often used to provide stability for fractures of the cervical and high thoracic vertebrae without cord damage. This device allows greater mobility, self-care and participation in rehabilitation programs. The device is secured with four pins inserted into the skull: two in the frontal bone and two in the occipital bone. The halo ring is then attached to a rigid plastic vest lined with sheepskin. Nursing care of the person using a halo fixation device are described in the box below.

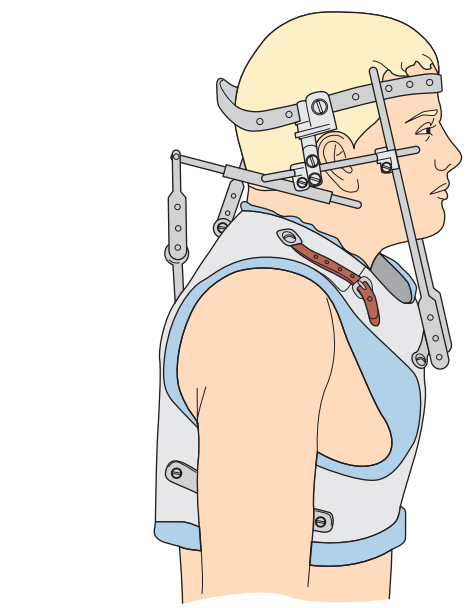


FIGURE 42.8 ■ The halo external fixation device

Nursing care

During both the acute phase and the rehabilitative phase, the person with an SCI has complex needs that involve all members of the healthcare team. Because these injuries are more common in younger people, consideration of lifelong effects on both the person and the family is essential. The nurse coordinates person care and develops and implements a care plan that is individualised to each person and family. The focus of the plan is to prevent the secondary complications of immobility and altered body functions, to promote self-care and to educate the person and family. A nursing care plan for a person with an SCI is found below.

Health promotion

Health promotion for SCIs primarily involves preventing injuries. Nurses can provide valuable information in the community and in the workplace to prevent SCIs. Programs that focus on wearing seat belts and using approved infant seats and child booster chairs in motor vehicles can do much to help decrease the number of SCIs each year. Education programs that promote workplace safety should include information on preventing falls and using heavy equipment safely.

Assessment

The following data are collected through the health history and physical examination (see Chapter 40). Further focused assessments are described with nursing interventions in the next section.

- *Health history:* time, location and type of event causing injury; location, duration, quality and intensity of pain; dyspnoea; sensation; paraesthesia.
- *Physical examination*: vital signs, motor strength, movement, spinal reflexes, bowel sounds, bladder distension.

Nursing diagnoses and interventions

Because an SCI has many possible effects, many nursing diagnoses may be appropriate. Nursing diagnoses discussed in this section focus on problems with physical mobility, respirations, dysreflexia, bowel and bladder elimination, sexual dysfunction and self-esteem.

Impaired physical mobility

After the initial period of spinal shock and areflexia, the person regains spinal reflex activity and muscle tone that is not

NURSING CARE OF THE PERSON in halo fixation

- Maintain integrity of the halo external fixation device.
  a. Inspect pins and traction bars for tightness; report loosened pins to the doctor.
  b. Tape the appropriate wrench to the head of the bed for emergency intervention.
  c. Never use the halo ring to lift or reposition the person.

  Loosening of the apparatus poses the risk of further damage to the cord. It is the responsibility of the nurse to maintain the integrity of the apparatus and the safety of the person.
- Assess muscle function and skin sensation every 2 hours in the acute phase and every 4 hours thereafter.
  a. Assess motor function on a scale of 0 to 5, with 0 being no evidence of muscle contraction and 5 being normal muscle strength with full range of motion.
  b. Assess sensation by comparing touch and pain, moving from impaired to normal areas and testing both the right and left sides of the body.

  Monitoring muscle function and skin sensation allows early identification of potential neurological deficits.
- Monitor pin sites each shift and follow hospital policy for pin care. Some general guidelines are:
  a. Assess pin sites for redness, oedema and drainage.
  b. Depending on policy, clean each pin site with a sterile applicator dipped in hydrogen peroxide, apply a topical antibiotic and cover with sterile 5 cm split gauze squares.

  Organisms can enter the body through the pin-insertion site; assessments and care are provided to detect signs of and prevent infection.
- Maintain skin integrity.
  a. Turn the immobile person every 2 hours.
  b. Inspect the skin around edges of the vest every 4 hours.
  c. Change the sheepskin liner when it is soiled and at least once each week.

  These interventions prevent skin injury and irritation.

under the control of higher centres. The person with injuries above the level of T12 experiences involuntary spastic movements of skeletal muscles. These movements reach a peak about 2 years after the injury and then gradually subside (Porth, 2010). Spasms impair the ability to carry out ADLs and work. In addition, the paraplegia or quadriplegia increases the potential for impaired skin integrity, thrombophlebitis and contractures.

The goals of care for the person with impaired mobility related to an SCI are to reduce the effects of spasticity and to prevent complications involving the skin, the cardiovascular system and joint function.

- Perform passive ROM exercises for all extremities at least twice a day. Identify stimuli that cause spastic movements and either avoid the stimuli (such as certain exercises) or teach the person to expect the movements. *ROM exercises help prevent contractures and stretch spastic muscles, promoting rehabilitation.*
- Maintain skin integrity by turning every 2 hours, assessing pressure points at least once each shift and using a pressure-relieving mattress if necessary. The person may be placed on a regular or special bed, such as a kinetic bed. *Immobility compresses soft tissues and promotes the development of decubitus ulcers. The lack of sensory warning mechanisms and of voluntary motor control of skin dermatomes further increases the risk of altered skin integrity. Special beds allow movement or turning while keeping the spinal column in alignment.*
- Assess the lower extremities each shift for manifestations of thrombophlebitis. Observe for redness and for increased heat every shift; measure thigh and calf circumference daily. If anti-embolic stockings (TEDs) are ordered, remove for 30 to 60 minutes each shift. Assess for skin impairment and provide skin care while TEDs are removed. *The person with neurological deficits is at high risk of deep venous thrombosis (DVT) as a result of immobility, vasomotor dysfunction and decreased venous return with venous stasis. Anti-embolic stockings help to prevent the pooling of blood in the lower extremities and increase venous return, lessening the risk of venous stasis and thrombus formation. Sequential compression devices or calf compressors reduce venous stasis by intermittently squeezing blood from the deep veins in the legs, thereby lessening the risk of DVT (Rolls, 2008).*

CONSIDERATION FOR PRACTICE

Removing TED stockings each shift not only promotes healthy skin but also lets the nurse assess skin integrity.

Impaired gas exchange

Injuries at the level of T1 to T7 leave the phrenic nerve intact, but the innervation of intercostal muscles is affected, compromising respiratory function. In addition, because the abdominal muscles are paralysed, the person cannot expel secretions by coughing. The person with cord injuries at C3 or above has paralysis of the respiratory muscles and cannot breathe without a ventilator.

- Monitor vital capacity and respiratory effectiveness, assessing for tachycardia, restlessness, PaO_2 less than 60 mmHg, $PaCO_2$ greater than 50 mmHg and vital capacity less than 1 L. *People with cervical cord injuries frequently require ventilatory support because of reduced vital capacity and inability to expel secretions by coughing.*

CONSIDERATION FOR PRACTICE

Changes in ABGs and vital capacity signal respiratory insufficiency.

- Monitor for signs of ascending oedema of the spinal cord, including difficulty in swallowing or coughing, respiratory stridor, use of accessory muscles of respiration, bradycardia and increased motor and sensory loss. *Haemorrhage and oedema can further impair respiratory function.*
- Help the person to cough, as follows: place the hand between the umbilicus and xiphoid process and push in and up as the person exhales and coughs. *The person who is unable to cough effectively and has decreased ventilatory capacity may develop atelectasis, pneumonia and respiratory failure.*

NURSING CARE PLAN A person with an SCI

Jim Colins, a 19-year-old university student, is admitted to the hospital by ambulance following a car crash. His family (father, mother and sister) live 200 km away and cannot visit often, although they are very concerned. On admission to the hospital, a CT scan of the spine shows a fracture and partial laceration of the cord at the C7 level. Mr Colins is in halo traction. One night, he tells the nurse, 'I wish I had just died when I got hurt. I don't think I can stand to live like this.'

ASSESSMENT

When Mr Colins is admitted to the critical care unit, he has flaccid paralysis involving all extremities. He has no sensation below the clavicle or in portions of his arms and legs. His bladder is distended and bowel sounds are absent. Other assessment findings include BP 90/56, P 50, T 36.1°C, arterial blood gases PH 7.4, PaO_2 96, $PaCO_2$ 37, SaO_2 96%. Oxygen per nasal prongs is given at 2 L/min and halo traction is applied. A Foley catheter is inserted into his bladder and a nasogastric tube is inserted and attached to low-pressure continuous suction.

After 7 days, Mr Colins is moved from the critical care unit to the neurosurgical unit for continuing care and planning for transfer to a rehabilitation hospital in his home town. His vital signs have stabilised and are normal for his age; respirations and oxygenation are normal. Other neurological assessments remain the same.

DIAGNOSES

- *Risk of impaired physical mobility* related to paralysis of lower and upper extremities and manifested by inability to mobilise independently.
- *Risk of bowel incontinence* related to lack of voluntary sphincter control and manifested by faecal incontinence.
- *Dysfunctional grieving* related to denial of loss and manifested by depression, anger or denial.

PLANNING

- Plan to begin mobilisation and ROM exercises in the morning when Mr Colins is not tired.
- His usual time for a bowel movement is after breakfast; schedule retraining program for that time.
- Discuss the grief process with Mr Colins and his family.

Expected outcomes

- Be actively involved in exercise programs.
- Have a soft, formed stool every second or third day.
- Verbally express his grief to parents and staff.

IMPLEMENTATION

- Conduct passive exercises on all extremities four times a day.
- Provide progressive mobilisation by initially raising the head of the bed 90 degrees (repeat two to three times during the first day of movement); if blood pressure remains normal, dangle the legs for 5 minutes before transferring him to a chair.
- Encourage a diet high in fibre and fluids. Likes wholemeal bread, orange juice and cola; does not like water.
- Promote grief work by providing time to express feelings. Explain to the family that his denial and anger are part of the grieving process.
- Determine food likes and dislikes, and order preferred foods from the menu. Encourage his friends to bring in his favourite foods periodically.
- Take and record weight every third day, using the bed scales.

EVALUATION

By the time Mr Colins is transferred to the rehabilitation hospital he is looking forward to learning how to use special equipment and getting his own motorised wheelchair. He is able to sit up in a chair without dizziness or hypotension. The use of ordered stool softeners combined with a high-fibre diet and fluid intake of 2000 to 3000 mL per day has maintained bowel elimination. Mr Colins and his parents have spent 3 hours talking about their feelings related to the accident and the future. Although the discussion is emotionally difficult, all three say they now feel much better. Mr Colins still has episodes of angry outbursts and tears, but he is more optimistic about what can be done and believes he can finish university. He selects foods from the menu each day and eats most of his meals, but he especially enjoys the times his friends bring in pizza or Thai takeaway.

CRITICAL THINKING IN THE NURSING PROCESS

1. Considering Mr Colins's age and developmental level, do you think his emotional responses to his injury were appropriate?
2. Issues of sexuality are obviously important to the person with a spinal cord injury. How would you approach Mr Colins about this topic?
3. Which issues in this case study will influence your future practice?

REFLECTION ON THE NURSING PROCESS

1. Design an education strategy to help educate Mr Colins about pressure areas and care needed to prevent them.
2. Outline a nursing discharge program for Mr Colins, incorporating useful information for Mr Colins's family about available resources.

Ineffective breathing patterns

Respiratory function is impaired in the person with an SCI in the cervical and thoracic levels if the diaphragm (innervated at C3 to C5), the intercostal muscles (innervated at T1 to T7) and the abdominal muscles are affected. In the person with injury at higher levels, assisted ventilation and a tracheostomy are necessary; when the injury is at lower levels, the person's ability to take a deep breath and cough is diminished. The goal of nursing interventions is to maintain normal respiratory rate (12 to 20 breaths per minute) and to prevent pulmonary complications such as atelectasis and pneumonia.

- Assess respiratory rate, rhythm and depth every 4 hours (or more frequently if needed). Auscultate breath sounds as a part of respiratory assessment. *Injury to the cord in the cervical or thoracic regions can decrease respiratory function and increase the risk of respiratory problems.*

CONSIDERATION FOR PRACTICE

Auscultate the lungs for crackles and wheezes.

- Monitor results of oxygen saturation with pulse oximetry and ABG studies. *ABG studies provide information about gas exchange; decreasing pH, oxygen and oxygen saturation levels, and increasing carbon dioxide levels, signal respiratory acidosis.*
- Administer supplemental oxygen as prescribed. *Oxygen saturation must be maintained at 100% with supplemental oxygen to prevent hypoxaemia and secondary SCI in all acute SCI.*
- Help the person turn, cough and deep breathe at least every 2 hours. Use assisted coughing as necessary. *Paralysis of intercostal or abdominal muscles decreases the ability to expel secretions by coughing; retained secretions increase the risk of pneumonia. The inability to breathe deeply may result in atelectasis.*
- Increase fluids given by mouth to 3000 mL per day (if oral intake is approved), according to the person's preference for type of liquids and predicated on the person's ability to swallow. *Increased fluid intake thins secretions, which can more easily be expelled and expectorated.*

Dysreflexia

Autonomic dysreflexia is an emergency that requires immediate assessment and intervention to prevent complications of extremely high blood pressure (loss of consciousness, seizure and even death).

- Elevate the head of the person's bed and remove TEDs or sequential compression boots. *These measures increase pooling of blood in the lower extremities and decrease venous return, thus decreasing blood pressure.*
- Assess blood pressure every 2 to 3 minutes while at the same time assessing for stimuli that initiated the response (such as a full bladder, impacted stool or skin pressure). *The most serious danger in dysreflexia is elevated blood pressure, which could precipitate a stroke, myocardial infarction, arrhythmias or seizures. If the person has a Foley catheter, ensure that there are no kinks in the tubing. If the person does not have a Foley catheter, drain the bladder with a straight catheter. If manifestations persist, assess for a faecal impaction. If an impaction is present, insert lignocaine into the anus, wait 10 minutes, and manually remove the impaction.*

CONSIDERATION FOR PRACTICE

Blood pressure readings may be as high as 300/160.

- If blood pressure remains dangerously elevated, the doctor may prescribe intravenous administration of an antihypertensive agent such as diazoxide. Other medications that may be used include nifedipine and hydralazine.

CONSIDERATION FOR PRACTICE

It is important to closely monitor for hypotension following administration of antihypertensive medications, especially if the stimulus for the dysreflexia has been removed.

Impaired urinary elimination and constipation

Depending on the level of the injury, the person with an SCI may have alterations in bowel and bladder function. The person with injuries to the cord at or above the S2 to S4 levels will have a neurogenic bladder, with deficits in control of micturition. Voluntary and involuntary bowel control is affected in the person with a lower motor neuron injury. Both bowel and bladder retraining are possible; if not, some form of assisted elimination is necessary. Although an indwelling catheter may be used in the acute phase of care, the goal is to re-establish a catheter-free state.

- Monitor for manifestations of a full bladder. *Over-distension stretches the bladder and can lead to backflow of urine into the ureters and kidney; stasis of urine in an incompletely emptied bladder increases the risk of infection.*

CONSIDERATION FOR PRACTICE

A distended bladder can be palpated over the lower abdomen above the pubic symphysis.

- Teach the person to use trigger voiding techniques prior to straight catheterisation. These techniques include stroking the inner thigh, pulling the pubic hair, tapping on the abdomen over the bladder and (in females) pouring warm water over the vulva. *These trigger voiding techniques stimulate parasympathetic nerve fibres to cause reflex activity and may facilitate voiding.*
- Teach self-catheterisation to the person who will be able to carry out the procedure alone or with minimal assistance (see Procedure 42.1). *Straight catheterisation at regular intervals is part of bladder training because periodic*

PROCEDURE 42.1 Self-catheterisation

Self-catheterisation on an intermittent basis (usually as part of self-care at home) is a clean, rather than sterile, procedure. The hands should be washed before and after the procedure and the urinary meatus should be cleaned by washing with soap and water.

Attempt to void. If urine is not of sufficient quantity (at least 100 mL), or if you cannot void at all, perform self-catheterisation. *A large amount of residual urine means that more frequent catheterisations (every 4 to 6 hours) are necessary.*

FEMALE SELF-CATHETERISATION

- While sitting on the wheelchair or the commode, locate the urethra. Visualise the urethra by looking in a mirror or palpate the urethra with a fingertip. *Visualisation or palpation of the meatus is necessary for proper catheter insertion.*
- Lubricate the meatus with a water-soluble lubricant. *Lubrication facilitates the insertion of the catheter and reduces trauma to tissues.*
- Take a deep breath and insert the catheter tip 5 to 7 cm or until urine flows. *The catheter enters the bladder more easily when the sphincter is relaxed. The deep breath relaxes the sphincter. The female urethra is 4 to 6.5 cm long.*
- Hold the catheter securely and allow urine to drain until the flow stops. *Withdrawing and reinserting the catheter increase the risk of infection.*

MALE SELF-CATHETERISATION

- Sit either on the commode or in the wheelchair. Hold the penis with slight upward tension and extend it to its full length. *Extending the penis straightens the urethra.*
- Lubricate the catheter from the tip to about 15 cm downwards. *Lubrication is especially important for male catheterisation because of the length of the urethra.*
- Take a deep breath and insert the catheter 15 to 17 cm or until urine flows. *The catheter enters the bladder more easily when the sphincter is relaxed. The deep breath relaxes the sphincter. The male urethra is about 15 cm long.*
- Hold the catheter securely and allow urine to drain until flow has stopped. *Withdrawing and reinserting the catheter increase the risk of infection.*

FOLLOWING SELF-CATHETERISATION

For both female and male techniques, withdraw the catheter and wash it with soap and water. Store the catheter in a clean container. *The catheter can be reused until it is too soft or too hard to be directed into and through the urinary meatus.*

distension and relaxation of the muscles of the bladder promote reflex bladder activity. In addition, self-care fosters independence.

- Monitor residual urine throughout the bladder retraining program. *A residual urine amount of less than 80 mL after a triggered voiding is considered satisfactory.*
- Institute a bowel retraining program as follows:
  - Assess usual patterns of bowel elimination to establish best times for an individualised program.
  - Maintain a high-fluid, high-fibre diet.
  - Use stool softeners as prescribed; rectal suppositories and enemas may be used 30 minutes after meals to stimulate stronger peristalsis and facilitate evacuation.
  - Maintain an upright position if at all possible and ensure privacy.
  - If the person is unable to evacuate, digital stimulation or manual removal on a regular basis may be the most effective long-term management.

  A bowel retraining program to regulate the bowel through reflex activity may be instituted in the person with upper motor neuron injuries. The person with a lower motor neuron injury loses the defecation reflex and bowel retraining is more difficult (if not impossible).

Sexual dysfunction

Sexual intercourse is often still possible for the person with an SCI. In men, the general rule is that the higher the level of injury the greater the potential to have reflexogenic erections, although ejaculation or orgasm may not occur and fertility is usually lower as a result of a lack of temperature control of the testes. However, ejaculation may be stimulated and the sperm used to inseminate the person's partner so that fatherhood is a possibility. Men who have sacral-level injuries do not have reflexogenic erections but may have psychogenic erections. They are also more likely to remain fertile.

Women with an SCI generally do not have sensation during sexual intercourse, but pregnancy is possible. However, pregnant women with an SCI are at increased risk of autonomic dysreflexia during labour and delivery. Birth-control options should be discussed prior to discharge from the acute care setting.

A person with an SCI may be deeply concerned about alterations in sexual function. These concerns may lead to lowered self-esteem, altered self-image or changes in feelings about being an attractive and desirable person. Assess concerns and provide a climate that is receptive to discussion about sexuality. Examples of objectives for sexual counselling for the person with an SCI are that the person will understand how the injury has altered sexual functioning, be aware of alternative ways of achieving sexual pleasure and have a positive self-concept and body image.

- Include data about sexuality when obtaining the nursing history and database. *Sexuality is a private matter for most people and the person may not discuss it unless the nurse introduces the topic.*

- Provide accurate information about the effect of the SCI on sexual function. *Accurate information gives the person a realistic picture of how the injury will affect sexuality.*
- Initiate a discussion with the person and partner of alternative means of gaining sexual satisfaction; these include the use of vibrators and oral–genital and manual stimulation. *Alternatives to intercourse can meet sexual needs and help maintain the relationship with a significant other.*
- Refer for sexual counselling, if appropriate, or to local support groups where questions can be answered by others with similar experiences. *Knowing that others have had similar experiences can decrease social isolation and provide a means of learning alternative methods of sexual functioning.*

Low self-esteem

An SCI is often the result of sudden trauma. Within moments, a formerly independent, fully functioning individual is suddenly unable to move and faces enormous adjustments in social, economic and personal roles and relationships. Body image, self-esteem and role performance are all affected by the damage. As a result, the person often demonstrates behaviours that may be difficult for the nurse to handle: depression, denial and anger are seen in the period immediately after the injury. In addition to these responses, the young adult person may act out by making sexually overt statements.

- Encourage talking about all aspects of physical function and care. *Talking provides a safe outlet for fears and frustrations and also increases self-awareness. Acceptance of self facilitates rehabilitation.*
- Encourage self-care and independent decision making. *Participating in self-care can promote positive coping; making decisions decreases feelings of powerlessness.*
- Help identify strategies to increase independence in desired roles; include both short- and long-term goals. Discuss assistive devices (such as hand-operated motor vehicles). *Identifying strategies to increase independence in the future fosters a positive self-concept and motivates the person to achieve rehabilitation goals.*
- Include family members and important others in discussions. *The realisation that others do care and will continue to provide support is important in fostering positive self-regard.*
- Refer the person and family to support groups or for psychological counselling. *Adjustment to change is more likely when the person and family seek peer and professional assistance.*

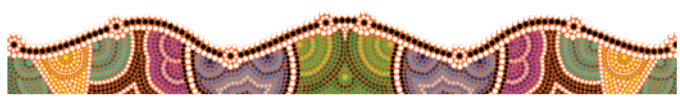
Community-based care

Rehabilitation of the person with an SCI is an ongoing process that moves from intensive care to intermediate care, to rehabilitation, and then to community-based and home care. Nursing interventions are necessary at all points in the process to prevent the complications of altered physical mobility and body functions, and to teach the person and family measures that promote independence in self-care.

Discharge planning should be addressed even in the initial plan of care while the person is in the critical care setting. Advance planning ensures continuity of care when the person leaves the hospital setting.

The following should be included in teaching the person and family about care at home:

- self-care activities (ADLs, exercises, bowel and bladder programs, skin care)
- mobility (use of assistive devices: wheelchair, crutches, special motor vehicles)
- preparation of the home environment
- if the person is in a wheelchair, will steps, stairs, doors or carpeted floors present physical barriers?
- if a special bed is necessary, have arrangements been made and is it in the home?
- psychological support
- independent activities
- coping skills for the person and caregiver
- referral to a community health agency and physiotherapist for the person who is returning home
- helpful resources:
  - Spinal Cord Injuries Australia: www.scia.org.au
  - Christopher Reeve Paralysis Foundation: www.christopherreeve.org
  - Australian Quadriplegic Association: www.aqavic.org.au.



THE PERSON WITH A HERNIATED INTERVERTEBRAL DISC

A herniated intervertebral disc—also called a ruptured disc, herniated nucleus pulposus or a slipped disc—is a rupture of the cartilage surrounding the intervertebral disc with protrusion of the nucleus pulposus (see Figure 42.9). Perhaps few neuro-orthopaedic disorders are as challenging as those involving the intervertebral discs. The person with herniation (rupture) of a disc has not only excruciating pain but also limited mobility. These problems may in turn cause alterations in role function, coping and the ability to perform ADLs.

Incidence and prevalence

A herniated intervertebral disc may occur at any adult age. However, it is more common as people enter middle age and age-related changes occur. The nucleus pulposus loses fluid content and the discs are less able to absorb shocks. The discs become smaller and slip out of place more easily. Ageing causes degeneration in the annulus fibrosus and the posterior longitudinal ligaments, and the vertebrae and discs are less able to respond to movement and are more easily injured.

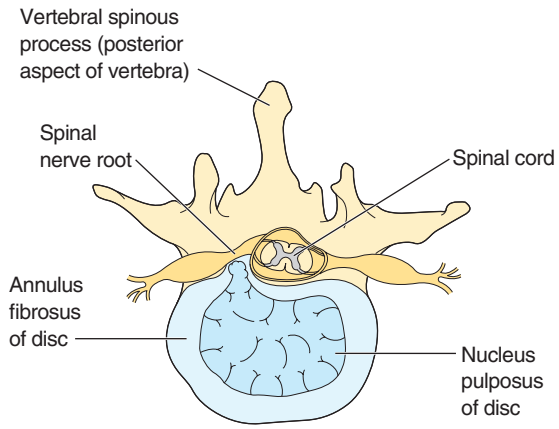


FIGURE 42.9 ■ A herniated intervertebral disc. The herniated nucleus pulposus is applying pressure against the nerve root

FAST FACTS

- Herniated intervertebral discs are more common in men than women.
- They occur most often in people between the ages of 30 and 50.
- The majority of herniated discs occur in the lumbar region (L4 or L5 to S1); when discs herniate in the cervical region, they most commonly do so at C6 to C7.
- Multiple herniations are not common, occurring in only about 10% of all people.

Source: Hickey (2013).

Pathophysiology

The intervertebral discs, located between the vertebral bodies, are made of an inner nucleus pulposus and an outer collar (the annulus fibrosus). The discs allow the spine to absorb compression by acting as shock absorbers. A herniated intervertebral disc occurs when the nucleus pulposus protrudes through a weakened or torn annulus fibrosus of an intervertebral disc (see Figure 42.9). This protrusion may occur anywhere along the vertebral column, but herniation of thoracic discs is uncommon. The protrusion may occur spontaneously or as a result of trauma, with trauma (such as lifting heavy objects or falling) causing about half of all cases. Rupture of the disc allows herniation of the nucleus pulposus in a posterolateral direction, with compression of the associated nerve root. The resulting pressure on adjacent spinal nerves causes characteristic manifestations, which vary with the location and the amount of protruding disc material (see the box below). Occasionally, the herniation is central rather than posterolateral, with pressure on the spinal cord.

The herniation may be abrupt or gradual. Lifting incorrectly or suddenly twisting the spine can cause rupture with immediate intense pain and muscle spasms. Gradual herniation is the result of degenerative changes, osteoarthritis or ankylosis spondylitis. People with a gradual herniation have a slow onset of pain and neurological deficits.

Lumbar disc manifestations

The classic manifestation of a ruptured lumbar disc is recurrent episodes of pain in the lower back. The pain typically radiates across the buttock and down the posterior leg, although it may be experienced only in the leg. **Sciatica** is a term used to describe lumbar back pain that radiates down the posterior leg to the ankle and is increased by sneezing or coughing (the result of pressure on nerve roots L4, L5, S1, S2 or S3, which give rise to the sciatic nerve). Sciatica may be elicited by straight leg raising: the person feels pain when lifting one leg while dorsiflexing the foot of that leg. Sciatica pain varies in intensity, ranging from mildly uncomfortable to excruciating. It is aggravated by a variety of positions and activities, including sitting, straining, coughing, sneezing, climbing stairs, walking and riding in a car.

Other manifestations include postural deformity, motor deficits, sensory deficits and changes in reflexes. In about 60% of people with ruptured lumbar discs, the normal lumbar lordosis is absent. When standing, the person typically has a slight forward tilt to the trunk, scoliosis of the lumbar spine, slight flexion of the hip and knee on the affected side and

MANIFESTATIONS A ruptured intervertebral disc

L4 TO L5 LEVEL (AFFECTS FIFTH LUMBAR NERVE ROOT)

- Pain in hip, lower back, posterolateral thigh, anterior leg, dorsal surface of foot, great toe
- Muscle spasms in affected areas
- Paraesthesia over lateral leg and web of great toe
- Foot drop (rare)
- Decreased or absent ankle reflex
- Cauda equina syndrome (with complete nerve root compression): bowel and bladder incontinence, paralysis of lower extremities

L5 TO S1 LEVEL (AFFECTS FIRST SACRAL NERVE ROOT)

- Pain in midgluteal region, posterior thigh, calf to heel, plantar surface of the foot to the fourth and fifth toes
- Paraesthesias in posterior calf and lateral heel, foot and toes
- Difficulty walking on toes

C5 TO C6 LEVEL (AFFECTS SIXTH CERVICAL NERVE ROOT)

- Pain in neck, shoulder, anterior upper arm, radial area of forearm, thumb
- Paraesthesia of forearm, thumb, forefinger and lateral arm
- Decreased biceps and supinator reflex
- Triceps reflex normal to hyperactive

paravertebral muscle spasms (Hickey, 2013). Motor deficits include weakness and, in some people, problems with sexual function and urinary elimination. Sensory deficits include paraesthesias and numbness. Knee and ankle reflexes are decreased or absent.

Cervical disc manifestations

Cervical discs that herniate laterally cause pain in the shoulder, neck and arm. Other manifestations of lateral cervical herniation include paraesthesias, muscle spasms and stiff neck, and decreased or absent arm reflexes. Central cervical herniations result in mild, intermittent pain; however, the person may also experience lower extremity weakness, unsteady gait, muscle spasms, urinary elimination problems, altered sexual function and hyperactive lower extremity reflexes.

INTERPROFESSIONAL CARE

Considerations for the person with a ruptured intervertebral disc include identifying the location of herniation and determining whether conservative treatment or surgery is indicated. Nursing care is directed towards preparing the person for diagnostic tests and providing teaching and care for the person who has either medical or surgical interventions.

Diagnosis

Diagnostic tests are ordered to differentiate the cause of back pain; for example, back and leg pain is also caused by spinal tumours, degenerative processes or abdominal diseases. Assessing pain is an important part of diagnosis. The tests include x-rays and CT scans of the lumbosacral or cervical area to identify skeletal deformities and narrowing of the disc spaces (see Chapter 40). Electromyography (EMG), which measures electrical activity of skeletal muscles at rest and during voluntary contraction, may be conducted to identify specific muscles affected by the pressure of the herniation on the nerve roots.

A myelogram with contrast medium is done to illustrate areas of herniation, although it does not provide the detail found with CT or MRI. However, myelography is diagnostic in 80–90% of all cases and is used both to rule out tumours and locate the herniation. Nursing implications for the care of a person having a myelogram are described in Chapter 40.

Medications

The person with a ruptured intervertebral disc is treated with medications to relieve pain and reduce swelling and muscle spasms. Pain is usually managed with NSAIDs (see Chapter 8). Muscle spasms are treated with muscle relaxants.

Treatments

A ruptured intervertebral disc may be treated conservatively or with surgery.

CONSERVATIVE TREATMENT A ruptured intervertebral disc is usually managed conservatively unless the person is experiencing severe neurological deficits. The goals of treatment are pain relief and healing of the involved disc by fibrosis. Conservative treatment is usually prescribed for 2 to 6 weeks. If the person continues to have pain after that time, surgery may be considered. The treatment regimen depends on the severity of the manifestations. Decreasing activity level with bed rest is no longer recommended; and in many cases, the person is advised to continue with normal activities while taking prescribed medications for pain, inflammation and muscle spasms.

Medications used to treat back pain include non-narcotic analgesics, anti-inflammatory drugs such as the NSAIDs, muscle relaxants and sedative–tranquillisers.

SURGERY Surgery is indicated for people who do not respond to conservative management or have serious neurological deficits. Several surgical interventions are used to treat a ruptured intervertebral disc. The type of surgery chosen depends on the location of the disc and the stability of the spinal column.

- A **laminectomy**, the type of surgery most often performed, is the removal of a part of the vertebral lamina. The surgery is done to relieve pressure on the nerves. It is often combined with removal of the protruding nucleus pulposus (*nuclectomy*). Nursing care for the person having a laminectomy is discussed in the box below. A *discectomy* is the removal of the nucleus pulposus of an intervertebral disc. Discectomy may be performed alone or with a laminectomy.
- Spinal fusion is the insertion of a wedge-shaped piece of bone or bone chips between the vertebrae to stabilise them. The bone is usually taken from a person donor site, such as the iliac crest. A spinal fusion may also be performed through a spinal implant with a device called a BAK (a hollow titanium cylinder with holes), which is packed with grafted bone from a donor site and placed in the space where a disc is removed. Although not appropriate for all people requiring a spinal fusion, this does facilitate a short hospital stay and convalescence.
- Foraminotomy is an enlargement of the opening between the disc and the facet joint to remove bony overgrowth compressing the nerve. The location and size of the incision vary according to the surgeon's preference and the location and size of the ruptured disc. The posterior approach is taken for lumbar surgery. Either the posterior or the anterior approach may be taken for cervical discs.
- Intradiscal electrothermal therapy (IDET) uses thermal energy to treat pain from a bulging spinal disc. A special needle is inserted into the disc and heated to a high temperature. The heat thickens and seals the disc wall and decreases bulging of the disc.
- A *microdiscectomy*, in which microsurgical techniques are used, is performed through a very small incision. This type of surgery decreases the possibility of trauma to surrounding structures during surgery and allows early postoperative mobility and a short hospital stay.

NURSING CARE OF THE PERSON having a posterior laminectomy

PREOPERATIVE TEACHING

- Demonstrate and ask the person to practise log-rolling; explain that it will be done by the nurses for the first day or two and then the person can do it alone. *To ensure healing, the spinal column must remain in alignment when turning and moving.*
- Explain the importance of taking pain medications regularly and of asking for them before the pain is severe. Include information about the possibility of the pain being much the same after surgery. *Pain is easier to control if medications are taken before the pain is severe. Pain may be the same following surgery for a herniated intervertebral disc because oedema due to surgery irritates and compresses the nerve roots.*
- Demonstrate the use of a fracture bedpan and ask the person to practise its use. The person usually must remain flat in bed for a period of time following surgery. *A fracture bedpan is more comfortable for the person who must lie flat.*
- Explain that the person may need to eat while lying flat. *This position prevents flexion of the spine.*
- Demonstrate and ask the person to demonstrate deep breathing, the use of the incentive spirometer and leg exercises. *These measures prevent respiratory and circulatory complications.*

POSTOPERATIVE CARE

- Maintain the person in a position that minimises stress on the surgical wound. For people with cervical laminectomy:
  a. Elevate the head of the bed slightly.
  b. Position a small pillow under the neck.
  c. Maintain the position of the cervical collar.
- For people with lumbar laminectomy:
  a. Keep the bed flat or elevate the head of the bed slightly.
  b. Place a small pillow under the head.
  c. Place a small pillow under the knees or use a pillow to support the upper leg when the person lies on one side.

  These positions minimise stress on the surgical wound and suture line. A cervical collar provides stability and prevents flexing or twisting the neck.
- Turn the person every 2 hours, using the log-rolling technique. Teach the person not to use the side rails to change position. Maintain proper body alignment in all positions. *The person's body is turned as a single unit (usually with a turning sheet) to avoid movement of the operative area. Pulling on the side rails puts stress on the operative area and may also cause misalignment of the vertebral column.*
- Monitor the person for signs of nerve root compression.
  a. Cervical laminectomy: assess hand grips and arm strength, ability to move the fingers and ability to detect touch.
  b. Lumbar laminectomy: assess leg strength, ability to wriggle the toes and ability to detect touch.

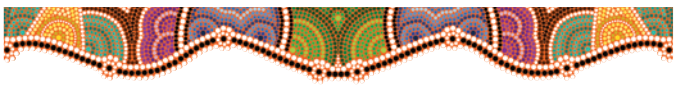
  Compare bilateral findings. Report muscle weakness or sensory impairment to the doctor immediately. *Loss of motor and sensory function may indicate nerve root compression.*
- Assess for haematoma formation as manifested by severe incisional pain that is not relieved by analgesics and decreased motor function. Report these findings immediately. *A haematoma may form at the surgical site. If untreated, it may cause irreversible neurological deficits including paraplegia and bowel/bladder dysfunctions (Hickey, 2013).*
- Assess for leakage of cerebrospinal fluid. Assess the dressing for increased moisture. Check the sheets for wetness when the person is lying supine; check for clear liquid running down the back when the person is sitting or standing. Gently palpate the sides of the wound to detect a bulge. Use a Dextrostrix strip to assess any leakage for the presence of glucose, a positive indicator of cerebrospinal fluid. *Although uncommon, leakage of cerebrospinal fluid greatly increases the risk of infection of the wound and of the meninges.*
- Assess for nerve root injury. Assess the person's ability to dorsiflex the foot (lumbar laminectomy) and the person's grip strength (cervical laminectomy). Assess the person who has had a cervical laminectomy for hoarseness. Report hoarseness and further assess the person's ability to swallow. *Nerve root compression may cause permanent damage, resulting in footdrop (in lumbar laminectomy) and hand weakness (in cervical laminectomy). Damage to the laryngeal nerve may cause permanent hoarseness. Impaired ability to swallow puts the person at risk of aspiration.*
- Assess for urinary retention. The person should void within 8 hours after surgery. If the doctor allows, let males stand to void. Compare intake and output for each 8-hour period. *All people who have received a general anaesthetic are at risk of urinary retention. The person who has had a lumbar laminectomy may have even more difficulty voiding as a result of stimulation of sympathetic nerves during surgery.*
- Assess for pain using a scale from 0 (no pain) to 10 (severe pain). Administer prescribed analgesics on a regular basis or teach the person to use patient-controlled analgesia (PCA), if prescribed. Discuss the person's concerns about pain that is unrelieved by surgery. *Compression of the nerve root over time results in oedema and inflammation. Because of surgery-induced oedema, the person is likely to experience either the same pain or perhaps more severe pain in the period immediately after surgery. This pain usually persists for several weeks after surgery. In addition, many people who have had a lumbar laminectomy have muscle spasms in the lower back, abdomen and thighs for the first few days after surgery.*
- Assess for infection by taking and recording vital signs at least every 4 hours; report increased body temperature. Assess the wound and dressing for signs of infection: increased redness, drainage, pain and pus. Use sterile technique to change dressings. *The surgical person is always at risk of infection; the person with a laminectomy is also at risk of arachnoiditis. This inflammation of the arachnoid layer of the spinal meninges*

(continued)

NURSING CARE OF THE PERSON having a posterior laminectomy (continued)

results from wound infection or contamination during surgery and may cause the formation of painful adhesions.

- Encourage deep breathing and the use of the incentive spirometer every 2 hours; coughing may be discouraged. *Anaesthesia and immobility depress respiratory function. Coughing may be discouraged because it can disrupt healing tissues, especially in the person having a cervical laminectomy.*
- Increase mobility as prescribed. (The time frame for ambulation is prescribed by the doctor; the routine here is representative.) People often sit on the side of the bed and dangle their legs the evening after surgery or the first day thereafter. Many people ambulate the first or second postoperative day. To help the person out of bed, first elevate the head of the bed. Then bring the person's legs over the side of the bed at the same time that the upper body moves into the upright position. *The person should not ambulate without assistance until they are no longer dizzy or weak. Early ambulation increases respiratory and circulatory function and decreases the risk of thrombophlebitis of the lower extremities. The vertebral column should remain in alignment while the person sits and stands. Safety must be considered throughout care.*



Nursing care

Nursing care for the person with a ruptured intervertebral disc may be provided through information in community and work settings, during conservative treatment and during pre- and postoperative treatment. The pain of the ruptured disc is often discouraging and debilitating and may well affect the person's ability to work.

Health promotion

Proper body mechanics may help prevent the occurrence of a ruptured intervertebral disc. Educating the person about the proper method of lifting and moving heavy objects should begin when children enter school. This information should also be given to all workers (including nurses) who have lifting as part of their responsibilities. The guidelines for proper body mechanics are as follows:

- Begin activities by spreading the feet apart to broaden the base of support.
- Use large muscles of the arms to lift and the legs to push when lifting.
- Work as closely as possible to the object that is to be lifted or moved.
- Slide, roll, push or pull an object rather than lift it.
- When lifting, bend the knees and lift up over your centre of gravity.
- When lifting, use a back support belt.

Assessment

The following data are collected through the health history and physical examination (see Chapter 40).

- *Health history:* type of employment, risk factors, pain (location, duration, intensity).
- *Physical assessment:* muscle strength and coordination, sensation, reflexes.

Nursing diagnosis and interventions

Nursing care for the person with a herniated intervertebral disc focuses largely on pain management, both during conservative management and after surgery.

Acute pain

People with a ruptured intervertebral disc experience acute back and leg pain. Acute pain may be related to preoperative muscle spasms or nerve root compression. After surgery, the person may have pain at the site of the incision and in the surgical area.

- Assess the degree of pain on a 0 to 10 scale (10 being greatest pain) and identify contributing and relieving factors. *Pain is a subjective experience. The nurse needs to assess it thoroughly before initiating interventions.*
- Use a firm mattress or place a board under the mattress. *A firm bed supports the spinal column and muscles.*
- Educate the person about avoiding turning or twisting the spinal column and to assume positions that decrease stress on the vertebral column (e.g. when in the supine position, flex the hips slightly). A small pillow may be placed under the knees (for the person with a herniated lumbar disc) or under the neck (for the person with a herniated cervical disc). *Correct body positions can decrease intradisc pressure.*
- Provide analgesic medications around the clock. *Intense pain can increase muscle spasms; maintaining serum levels of analgesics often prevents severe pain.*

CONSIDERATION FOR PRACTICE

It is important to maintain a constant level of pain relief. Healthcare providers have the responsibility of relieving pain with adequate medications.

Chronic pain

The person with a ruptured intervertebral disc often has pain for an extended period of time. Despite conservative treatment or previous surgery, pain may be ongoing or intermittent. If previous surgery has not relieved the pain, the person may be depressed or angry. Caring for a person with chronic pain is frustrating and the person is often regarded as difficult.

- Treat the person's reports of pain with respect. *The person is the one experiencing the pain and is thus the expert about it.*
- Do not refer to the person as being addicted to pain medication. *All types of pain medications may be used legitimately to manage pain.*

- Monitor the person carefully for any changes in condition. *Significant changes in the person's condition may go unrecognised when pain is present for a prolonged period of time.*

CONSIDERATION FOR PRACTICE

Although the person may develop tolerance to a narcotic analgesic, tolerance does not imply addiction.

- Maintain written plans of care for pain management that are individualised and ensure continuity of care. *When the person makes several visits (e.g. to an emergency department or a pain clinic), written records help caregivers determine what is effective in managing pain and what is not.*
- Teach the person alternative methods of pain management. *Consider the person's coping style when recommending methods. People who have a passive coping style are often better able to manage pain by depending on others, taking medications and resting. People with an active coping style are probably better able to manage pain by learning self-management methods, taking part in activities and staying busy.*
- Develop effective methods of improving rest and sleep. *Problems with rest and sleep make pain management more difficult. Sleeping poorly at night contributes to decreased motivation, confused thinking, depression and muscle aches.*
- Refer the person to a physiotherapist for an exercise program, if appropriate. *The person needs to know exactly what exercises to do, how many repetitions are recommended, for how long and how often. The person should not exercise to the point of causing increased pain.*
- Assess the need for referrals (and make them, if necessary) for the person who is depressed or anxious. *Anxiety and depression often are a part of long-term chronic pain, making pain management more difficult. Suggest that referrals for help with the frustration (rather than 'depression') may make a significant difference in the person's ability to manage pain.*

Constipation

The person with a ruptured intervertebral disc often has problems with constipation because of reduced mobility. Nursing interventions to alleviate and prevent constipation are important because straining to have a bowel movement can increase intradisc pressure, thus increasing pain.

- Assess the person's usual bowel routine, including diet, fluid intake and the use of laxatives or enemas. *Effective interventions are based on individualised needs.*

CONSIDERATION FOR PRACTICE

People who use laxatives or enemas for long periods of time may be dependent on those methods of having a bowel movement.

- Encourage a fluid intake of 2500 to 3000 mL per day unless contraindicated by the presence of renal or cardiac disease. *Adequate fluid intake facilitates the passage of faeces.*
- Increase fibre and bulk in the diet. If the person is unable to tolerate increased fibre, consult with the doctor about the use of stool softeners or bulk-forming agents. *Bulk and fibre promote regularity by retaining water in the large intestine.*

Community-based care

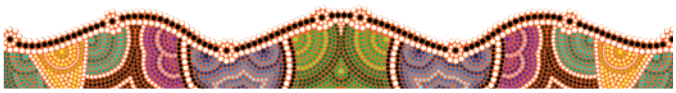
It is the nurse's responsibility to teach the person and family about chronic pain control, including specific interventions to alleviate pain. The nurse's role may be that of advocate and creative problem solver (see the 'Meeting individualised needs' box below). The following topics should be addressed:

- Often the goal is to control pain so that the person can perform normal ADLs, rather than to reach a pain-free state.

MEETING INDIVIDUALISED NEEDS Educating the person with a ruptured intervertebral disc

- Sleep on a firm mattress; use a bed board if necessary.
- When lying in the supine position, flex the knees to approximately a 45-degree angle with a small pillow and use a small pillow under the head.
- Avoid any activities that flex the spine, such as bending or lifting, and do not twist the back.
- Follow your diet to maintain body weight or to lose weight if needed.
- Follow the prescribed exercise program—for example:
  - a Lie flat on your back on the floor. Tighten your abdominal and buttock muscles and tilt your pelvis forward so that your lower back is flat on the floor. (This is called a *pelvic tilt.*) Hold the position for 3 seconds and repeat for the prescribed number of times.
  - b Lying on the back on a firm surface, press the feet to the floor, tighten the abdominal muscles and lift the upper half of the body off the floor. Hold the position for 3 seconds and repeat as prescribed.
  - c Lying on your back on a firm surface, bring your knees up to the chest. Put your hands around your knees and raise the buttocks off the floor. Repeat as prescribed.
  - d Sit upright on the floor or a firm surface. Keep one leg straight and bend the other knee. Reach for the toes of the straightened leg. Switch legs. Repeat as prescribed.
  - e Stand upright. Squat down, flexing the hips and knees. Straighten your back. Stand upright by straightening the knees. Repeat as prescribed.
- Wear flat-heeled shoes that provide good support.
- Use proper lifting techniques. For instance, squat and use your thigh muscles to lift an object from the floor and spread your feet to get a wide base of support when you lift while you are standing.

- Non-pharmacological methods of pain management include relaxation techniques, guided imagery, distraction, hypnosis and music. Joining a support group may be an effective intervention in coping with and managing pain.
- The person may be referred to a physiotherapist for education about body mechanics and back-strengthening exercises. Nurses should have the person demonstrate the exercises to reinforce teaching.



THE PERSON WITH A SPINAL CORD TUMOUR

Spinal cord tumours may be benign or malignant, primary or metastatic. They may arise at any level of the spinal column. Of all spinal cord tumours, 50% are thoracic, 30% are cervical and 20% are lumbosacral. They constitute about 0.5–1% of all tumours (Hickey, 2013). Tumours of the spinal cord are seen equally in men and women, and they most often occur between the ages of 20 and 60. They are rarely seen in the older adult.

Classification

Spinal cord tumours are classified by anatomical location as either intramedullary or extramedullary tumours. Intramedullary tumours, which make up about 10% of spinal tumours, arise from within the neural tissues of the spinal cord; those that occur include astrocytomas, ependymomas, glioblastomas and medulloblastomas (Papadakis & McPhee, 2015). Extramedullary tumours arise from tissues outside the spinal cord, with commonly occurring tumours including neurofibromas, meningiomas, sarcomas, chordomas and vascular tumours.

Extramedullary tumours are further categorised as intradural (arising from the nerve roots or meninges within the subarachnoid space) or extradural (arising from epidural tissue or the vertebrae outside the dura).

Tumours of the spinal cord are also classified as either primary or secondary (metastatic). Primary tumours, arising from the epidural vessels, spinal meninges or glial cells, have an unknown cause. Secondary tumours are metastatic in origin, most commonly the result of malignancies of the lung, breast, prostate, gastrointestinal tract or uterus.

Pathophysiology

Depending on their anatomical location, spinal cord tumours result in pathological changes as a result of compression, invasion or ischaemia secondary to arterial or venous obstruction. Extramedullary tumours (whether benign or malignant) alter normal function through compression of the spinal cord, with destruction of white matter and eventual filling of the space around the spinal cord. Cord compression interferes with normal blood flow and membrane potentials, altering afferent and efferent motor, sensory and reflex impulses. Compression of the spinal cord also causes oedema, which can ascend the cord and cause further neurological deficits. Intramedullary tumours both compress and invade. As the tumour grows within the cord, the cord also enlarges and distorts the white matter.

Manifestations

The manifestations of a spinal cord tumour depend on the anatomical location, level of occurrence, type of tumour and spinal nerves involved. General manifestations of a spinal cord tumour include pain, motor and sensory deficits, changes in bowel and/or bladder elimination, and changes in sexual function. Specific manifestations by anatomical level are outlined in the box below.

Pain is often the first manifestation of a spinal cord tumour. It is caused by compression of the spinal cord, tension on the spinal nerves or tumour attachment to the proximal dura (the covering of the spinal cord). The pain may be either localised or radicular. Localised pain is felt when pressure is applied over the spinous process of the involved area; this type of pain often accompanies metastatic tumours involving the vertebrae. Radicular pain is felt along the course of a nerve as a result of compression, irritation or tension of a nerve root. The pain is often made worse by any

MANIFESTATIONS Spinal cord tumours

CERVICAL CORD TUMOURS
- Ipsilateral arm motor involvement, followed by ipsilateral and contralateral leg involvement, followed by contralateral arm involvement
- Paresis of the arms and legs
- Stiffness of the neck
- Paraplegia
- Pain in the shoulders and arms
- Hyperactive reflexes

THORACIC CORD TUMOURS
- Paresis and spasticity of one leg, followed by paresis and spasticity of the other leg
- Pain in the back and chest
- Positive Babinski reflex
- Bowel and bladder dysfunction
- Sexual dysfunction

LUMBOSACRAL CORD TUMOURS
- Paresis and spasticity of one leg, followed by paresis and spasticity of the other leg
- Pain in the lower back, radiating to the legs and perineal area
- Loss of sensation in the legs
- Bowel and bladder dysfunction
- Sexual dysfunction
- Decreased or absent ankle and knee reflexes

activity that causes intraspinal pressure, such as sneezing or coughing.

Motor manifestations resulting from a spinal cord tumour include paresis and paralysis below the level of the tumour, spasticity and hyperactive reflexes. The Babinski reflex may be positive. These deficits are the result of involvement of the corticospinal tracts.

Many different sensory manifestations may occur, depending on the location and level of the tumour. Lateral tumour growth and compression affect the lateral spinothalamic tracts, causing pain, numbness, tingling and coldness. If the tumour involves the posterior columns, the senses of vibration and proprioception of body parts are affected.

Bladder and bowel elimination and sexual function are often affected. Bowel elimination deficits include constipation that may progress to paralytic ileus. Initial bladder elimination deficits include frequency, urgency and difficulty voiding. The deficits may progress to urinary retention and a neurogenic bladder. In addition, the male person may be impotent.

Syringomyelia is a complication of some spinal cord tumours. In this condition, a fluid-filled cystic cavity forms in the central intramedullary grey matter. This syndrome causes pain, motor weakness and spasticity.

INTERPROFESSIONAL CARE

The medical management of the person with a spinal cord tumour focuses first on diagnosis. Treatment depends on the type of tumour, its location and the person's condition.

Diagnosis

The person with a spinal cord tumour undergoes many of the same diagnostic tests as the person with a ruptured intervertebral disc. The tests used to identify the tumour include x-rays, CT scans, MRI and myelogram (see Chapter 40). A lumbar puncture of the person with a spinal cord tumour will demonstrate CSF that is commonly xanthochromic (having a yellow colour), has increased protein, has few to no cells and clots immediately. (This cluster of findings is called Froin's syndrome.)

Medications

The person with a spinal cord tumour is given medications to relieve pain and control oedema. If the pain is severe and the result of a metastatic tumour, narcotic analgesic may be administered via an epidural catheter. Pain management for the person with a spinal cord tumour is provided by narcotic analgesics (see Chapter 8). Steroids, such as dexamethasone, are administered to control oedema of the cord.

Surgery

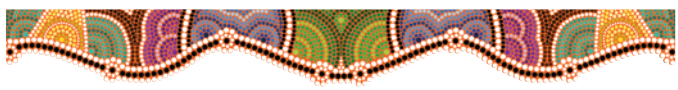
Intramedullary and intradural tumours are surgically excised when possible. Advances in microsurgical techniques and laser surgery have increased the possibility of tumour excision. Metastatic tumours may be partially excised to reduce cord compression; rapidly growing metastatic lesions may require surgical decompression to preserve motor, bowel or bladder function.

The surgical excision is made through a laminectomy. The person with a tumour involving more than two vertebrae often has a spinal fusion and may also have rods inserted to stabilise the spinal column.

Radiation therapy

Radiation therapy is used to treat metastatic spinal cord tumours for several different reasons. It may be used on an emergency basis to treat the person with rapidly progressing neurological deficits. It may be used to reduce pain. Radiation may also be used following surgical excision of as much tumour mass as possible.

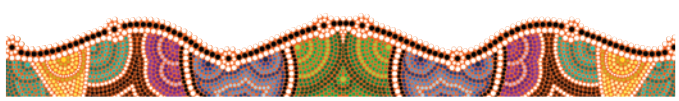
Radiation of the spinal cord may cause the development of radiation-induced myelopathy. This complication of radiation exposure occurs over time, with manifestations of *Brown–Séquard syndrome* (weakness or paralysis on one side of the body and loss of sensation on the opposite side) developing 12 to 15 months after therapy. The manifestations may progress to paraplegia, sensory loss and loss of bowel and bladder control (Hickey, 2013).



Nursing care

Nursing care for the person with a spinal cord tumour is individualised in accordance with the type of tumour and the type of treatment. The person with a benign tumour that is removed by surgery has different healthcare needs to the person with a metastatic tumour, even though they may have similar neurological deficits. The person with a spinal cord tumour (regardless of type) requires nursing care to monitor for neurological changes, to provide pain management and to manage motor and sensory deficits in order to preserve quality of life.

The assessments and nursing interventions for the person with a spinal cord tumour are similar to those described for the person with an SCI or who is undergoing surgery for a ruptured intervertebral disc. Following surgical treatment, the person may be transferred to a rehabilitation centre or may go home for the recovery period. Referrals for home care, occupational therapy and physiotherapy often help the person regain functional abilities. Teach family members how to move the person in the bed and from the bed to a chair. Also teach them how to provide physiotherapy, care for any appliances (such as an indwelling catheter) and prevent or treat constipation.



CHAPTER HIGHLIGHTS

- A stroke is a condition in which neurological deficits result from a sudden decrease in blood flow to a localised area of the brain. Strokes may be ischaemic or haemorrhagic. Ischaemic strokes result from a blockage of a cerebral artery by formation of a blood clot or by a clot or foreign substance (such as fat or bacteria) lodging in a blood vessel; they include transient ischaemic attacks, thrombotic strokes and embolic strokes. Haemorrhagic strokes occur when a cerebral blood vessel ruptures.
- Depending on the size and location of cerebral tissue damage, strokes may cause cognitive and behaviour changes, sensory–perceptual deficits, language disorders and motor deficits. Treatment of an ischaemic stroke with fibrinolytic therapy within 3 hours of the onset of manifestations may reverse damage to cerebral neurons.
- Nursing care is directed towards both prevention of a stroke through community-based education programs and interventions to promote recovery and decrease complications.
- Intracerebral haemorrhage may follow rupture of an intracranial aneurysm or arteriovenous malformation. Intracranial aneurysms occur at the site of a weakness in a cerebral blood vessel, while AV malformations are a tangled collection of dilated arteries and veins.
- Spinal cord injuries are almost always the result of trauma, with the main risk factors being age (young adults), gender (male) and alcohol or drug abuse. The causes of injury to the spinal cord include contusion, laceration, transection, haemorrhage and damage to spinal cord blood vessels.
- In a complete SCI, the motor and sensory pathways in the spinal cord are completely interrupted (transected), resulting in total loss of motor and sensory function below the level of the injury. In an incomplete SCI, the motor and sensory pathways are only partially interrupted, resulting in variable loss of function below the level of injury. Injuries of the spinal cord have the potential to affect movement, perception, sensation, sexual function and elimination.
- Spinal shock is the temporary loss of all reflexes (areflexia) below the level of injury. Manifestations of spinal shock include bradycardia, hypotension and flaccid paralysis.
- Autonomic dysreflexia is an exaggerated sympathetic response in the person with an SCI at or above the T6 level. Triggered by noxious stimuli (such as a blocked urinary catheter or a faecal impaction), this condition results in extreme hypertension and, if untreated, may cause seizures, stroke or a myocardial infarction.
- Rehabilitation of the person with an SCI is an ongoing process from intensive care to home care. Nursing interventions are necessary in all settings to promote independence in self-care.
- A herniated intervertebral disc is a rupture of the cartilage surrounding the intervertebral disc with protrusion of the nucleus pulposus. The main manifestation of lumbar discs is lower back and sciatic pain on the affected side. Cervical discs cause pain in the shoulder, neck and arm. A variety of medications, treatments and surgical procedures are available for the person.
- Spinal cord tumours may be benign or malignant, primary or metastatic. Depending on their size and location, they cause pathological changes in spinal cord function through compression, invasion or ischaemia.

CONCEPT CHECK

1 Which of the following manifestations would alert you to the possibility that a person has had a TIA?
1. sudden severe pain over the left eye
2. numbness and tingling in the corner of the mouth
3. complete paralysis of the right arm and leg
4. loss of sensation and reflexes in both legs

2 Although all of the following are risk factors for a stroke, which one is the greatest risk?
1. hypertension
2. heart disease
3. diabetes
4. high cholesterol levels

3 You have been assigned to care for a person who has had an acute ischaemic stroke of a left cerebral vessel. You read the chart and realise the person has contralateral deficits. What does this mean?
1. Both sides of the body are involved.
2. The person will have neurological deficits on the left side of the body.
3. The person will have neurological deficits on the right side of the body.
4. Deficits will be present below the level of the stroke.

4 What is the rationale for administration of a tissue plasminogen activator within the first 3 hours of a thrombotic stroke?
1. to reduce the risk of vasospasm
2. to decrease the risk of infection
3. to increase platelet aggregation
4. to cause fibrinolysis of the clot

5 Oxygen is often administered to the person who has had a stroke. Preventing hypoxia and hypercapnia through this treatment will lessen the risk of which complication?
1. fluid accumulation in the lungs
2. pulmonary emboli
3. increased intracranial pressure
4. rebleeding

6 What is the primary pathophysiological process of spinal shock?
1. temporary loss of reflex function below the level of injury
2. loss of control of cardiovascular mechanisms
3. exaggerated sympathetic response
4. damage to the lower motor neurons

7 A person has manifestations of autonomic dysreflexia. Which of these assessments would indicate a possible cause for this condition?
1. extreme hypertension
2. kinked catheter tubing
3. respiratory wheezes and stridor
4. skin breakdown over the coccyx

8 A person is admitted to the emergency department following a motor vehicle accident. An SCI at the cervical level is identified. What will be done to facilitate respirations?
1. No treatments are necessary.
2. Oxygen per nasal prongs will be administered.
3. The person will be placed on a ventilator.
4. The head of the bed will be elevated.

9 Many different medications may be given to the person with an acute SCI. Which of the following will possibly be administered? (Select all that apply.)

1 corticosteroids
2 vasopressors
3 antibiotics
4 analgesics
5 antihistamines

10 You are conducting a class at a factory to teach methods to prevent a ruptured intervertebral disc. What should be included? (Choose all that apply.)

1 Spread the feet apart to broaden the base of support.
2 Bend from the waist to lift articles from the floor.
3 Use large leg muscles to push when lifting.
4 Always lift articles rather than rolling or pushing them.
5 Work as closely as possible to the object to be moved.

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CHAPTER 43

NURSING CARE OF PEOPLE WITH NEUROLOGICAL DISORDERS

LAURA MELLISH

KEY TERMS

LEARNING OUTCOMES

- Identify prevalence, incidence and risk factors for degenerative neurological, peripheral nervous system, cranial nerve and infection- and neurotoxin-caused neurological disorders.
- Explain the pathophysiology, manifestations, complications, interprofessional care and nursing care of people with neurological disorders.
- Compare and contrast the manifestations of the progressive stages of Alzheimer's disease.
- Discuss the purposes, nursing implications and health education for the person and their family of medications used to treat Alzheimer's disease, multiple sclerosis, Parkinson's disease and myasthenia gravis.
- Describe the procedures (thymectomy, percutaneous rhizotomy, plasmapheresis) used to treat selected neurological disorders.

CLINICAL COMPETENCIES

- Assess functional status of people with neurological disorders and monitor, document and report abnormal manifestations.
- Use evidence-based research to design nursing interventions specific to the needs of ageing people with multiple sclerosis.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualised nursing interventions for people with neurological disorders.
- Administer oral and injectable medications used to treat neurological disorders knowledgeably and safely.
- Provide skilled care to people having a thymectomy, percutaneous rhizotomy or plasmapheresis.
- Integrate interprofessional care into the care of people with neurological disorders.
- Provide appropriate teaching to facilitate safety and communication, prevent neurological infections and toxins (rabies, tetanus and botulism), and facilitate community-based acute and chronic self-care for healthcare needs resulting from neurological disorders.
- Revise the plan of care as needed to provide effective interventions to promote, maintain or restore functional health status for people with neurological disorders.

This chapter discusses a variety of neurological disorders. Included are degenerative disorders, peripheral nervous system disorders, cranial nerve disorders and disorders caused by neurotoxins and viruses. For many of the disorders, nursing care is based on similar nursing diagnoses. To avoid repeating those diagnoses and interventions for each disorder, they have been divided between the nursing care discussions as appropriate.

DEGENERATIVE NEUROLOGICAL DISORDERS

Degenerative neurological disorders affect the central nervous system and the peripheral nerves. By progressively disrupting cognitive processes or motor functions, disorders such as Alzheimer's disease, Parkinson's disease and multiple sclerosis strike at the core of an individual's sense of personal autonomy and wellbeing and can be psychologically and emotionally devastating to family members and caregivers.

Ongoing medical research into degenerative neurological disorders offers an increasing measure of hope to people and their families. The discovery of genetic or biochemical markers associated with some of these disorders is leading to the development of effective screening and diagnostic methods. In addition, new drugs may make it possible to halt the progression of the disorders in some people, transforming the disorders into manageable conditions. This chapter begins with a discussion of dementia, which is not a specific disease but rather a collection of manifestations caused by a variety of disorders that affect the brain.

DEMENTIA

Dementia affects multiple cortical functions, calculation, learning capacity, language and judgment. Impairments of cognitive function are usually accompanied by deterioration in emotional control, social behaviour and motivation. People with dementia lose their ability to solve problems and may also have personality changes such as agitation and hallucinations. All forms of dementia result from death of neurons and/or the loss of communication between the cells. Although the exact cause is not always known, many forms of dementia are characterised by abnormal structures in the brain called inclusions, and there is clearly a genetic component in the development of some kinds of dementia.

More than 342 800 people in Australia have dementia. Of these, three in 10 people are aged 85 and older. However, despite increased incidence in older people, dementia is not a normal part of ageing (Alzheimer's Australia, 2015a). The incidence of dementia in the Indigenous population is nearly five times greater than in the total Australian population. Dementia affects one in eight (12.4% of the population) Indigenous people aged 45 and older (Australian Indigenous HealthInfoNet, 2011).

Many different diseases and conditions may cause dementia, including Alzheimer's disease, vascular dementia, Huntington's disease, Creutzfeldt–Jakob disease, medications, metabolic disorders, poisoning and anoxia. Table 43.1 provides an overview of the most common causes of dementia. Doctors do not diagnose dementia unless two or more brain functions (such as memory, language skills, perception, reasoning or judgment) are significantly impaired without loss of consciousness.

Even though the actual cause of all dementias may not be known, factors that increase the risk of developing one or more kinds of dementia have been identified. These risk factors include advancing age, a family history of a disease that causes dementia, smoking and alcohol use, atherosclerosis, high cholesterol and plasma homocysteine levels, diabetes, mild cognitive impairment and Down syndrome.

TABLE 43.1 Common causes of dementia

NAME	CAUSE AND PRIMARY PATHOPHYSIOLOGY
Alzheimer's disease (the most common cause of dementia in people aged 65 and older)	Unknown cause; characterised by two abnormalities in the brain: amyloid plaques and neurofibrillary tangles.
Vascular dementia (the second most common cause of dementia)	Caused by brain damage from cerebrovascular and cardiovascular problems (usually strokes). May also be caused by cerebral blood vessel damage from genetic disorders, endocarditis, myeloid angiopathy, vasculitis and profound hypotension.
Lewy body dementia	Cause usually unknown, although familial cases have been reported. Cells die, and remaining cells in the substantia nigra contain abnormal structures called Lewy bodies.
Frontotemporal dementia	Nerve cells, especially in the frontal and temporal lobes, degenerate. In many people, abnormal tau protein accumulates in neurofibrillary tangles.

Although sometimes confused with dementia, people often experience other conditions that may mimic dementia. These include:

- age-related cognitive decline, resulting from slower information processing and mild memory impairment. With ageing, the brain often decreases in volume and some neurons are lost. These changes are normal and are not considered a part of dementia
- mild cognitive impairment, which may progress to dementia but is not severe enough to be initially diagnosed as such
- depression or other emotional problems, causing people to be passive, slow, confused or forgetful
- delirium, characterised by confusion, rapidly altering mental states, disorientation and possible personality changes. Delirium is usually caused by a treatable physical or mental health illness and, when treated, results in a full recovery.

THE PERSON WITH ALZHEIMER'S DISEASE

Alzheimer's disease (AD) (also called *dementia of Alzheimer type* or *senile disease complex*) is a form of dementia characterised by progressive, irreversible deterioration of general intellectual functioning. People with AD live about 8 to 10 years following diagnosis, although some live as long as 20 years. The cause of death is often aspiration pneumonia because of the loss of the ability to swallow late in the disease.

Memory loss is usually the first sign of Alzheimer's disease. Memory deficits are initially subtle and family members and friends may not suspect a problem until the disease progresses and manifestations become more noticeable. Family members and people with AD may also deny the manifestations and hide deficits until the person exhibits unsafe or extremely unusual behaviour. Progression of the disease varies, but the course is one of deteriorating cognition and judgment with eventual physical decline and total inability to perform activities of daily living (ADLs). With the loss of the ability to perform even the most basic ADLs, the burden of meeting the person's needs shifts to the caregiver.

Incidence and prevalence

Alzheimer's disease is the most common degenerative neurological disorder and the most common cause of cognitive impairment in older adults (Grossman & Porth, 2013). It accounts for up to 70% of cases of dementia in Australia, affecting adults in middle to late life (Alzheimer's Australia, 2015b).

Two types of AD exist*: familial AD* follows an inheritance pattern; *sporadic AD* has no obvious inheritance pattern. AD is further described as early onset (occurring in people younger than 65) and late onset (occurring in people aged 65 and older). Early-onset AD affects people aged 30 to 60, is relatively rare and often progresses more rapidly than late-onset AD.

Risk factors and warning signs

As one ages, the risk of developing AD increases. With numbers of older people increasing, the incidence of AD is predicted also to increase. The risk factors for AD are older age, family history and female gender. Warning signs are:

- memory loss that affects job skills
- difficulty performing familiar tasks
- problems with language
- disorientation to time and place
- poor or decreased judgment
- problems with abstract thinking
- misplacing things
- changes in mood or behaviour
- changes in personality
- loss of initiative.

Recognising early manifestations is important, because the cause of dementia (such as from depression or hypothyroidism) may be reversible. Further information can be obtained from Alzheimer's Australia. Dementia from AD is not reversible. Treatment, however, can maximise quality of life and allow the affected person to plan for the future.

FAST FACTS

- Scientists estimate that in 2050, one in every 85 people worldwide will be affected by AD (Western Australian Neuroscience Research Institute, 2014).
- AD usually occurs after the age of 65, with the risk increasing with age.
- Early-onset forms of AD, which are usually genetic, may appear as early as age 30.
- In Australia, one in 15 people over the age of 65, and almost one in four of all people over the of age 85, have AD (Neuroscience Research Australia, 2015).
- 2706 people died from AD in 2010, accounting for 1.9% of all registered deaths for that year. In 2010, female deaths (1865) due to AD were higher than male deaths (841); the median age at death was 87.3 years.

Source: Australian Bureau of Statistics (ABS) (2012). *Causes of death, Australia, 2010: Diseases of the nervous system (G00-G99).* (Cat. no. 3303.0). Retrieved from www.abs.gov.au/ausstats/abs@.nsf/Products/E9AE6DDF5D8153E9CA2579C6000F6FF6?opendocument.

Pathophysiology

Characteristic findings in the brains of AD people are loss of nerve cells and the presence of *neurofibrillary tangles* and *amyloid plaques* (see Figure 43.1). Neurofibrillary tangles result when *tau*, a kind of protein in the neurons, becomes distorted and twisted. Tau normally holds together the microtubules which guide nutrients and molecules to the end of the axon. In AD, tau changes and twists into pairs of filaments, which then join to form tangles. Because tau no longer maintains the transport system, communication is lost between neurons. Death of neurons may follow, contributing to the development of dementia.

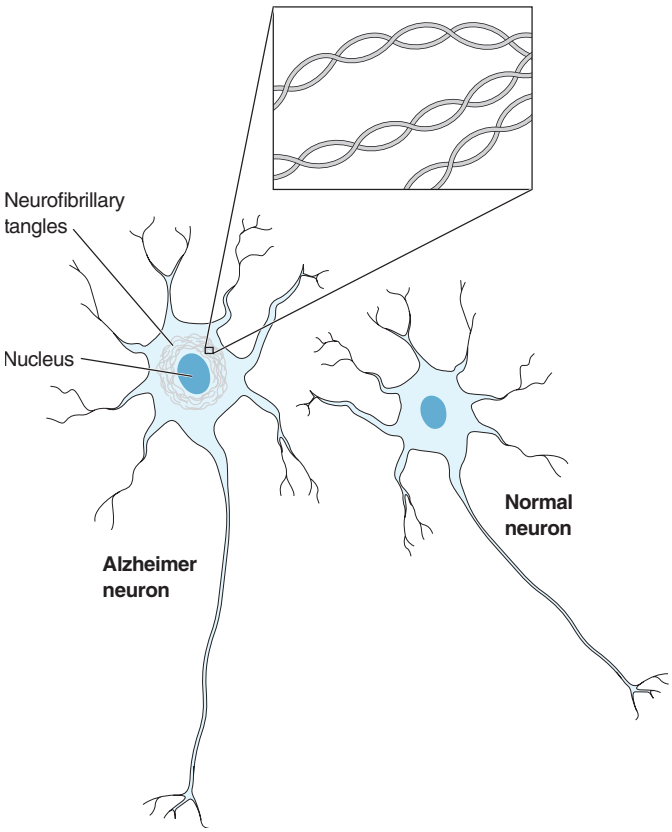


FIGURE 43.1 ■ Neuron with neurofibrillary tangles seen in Alzheimer's disease

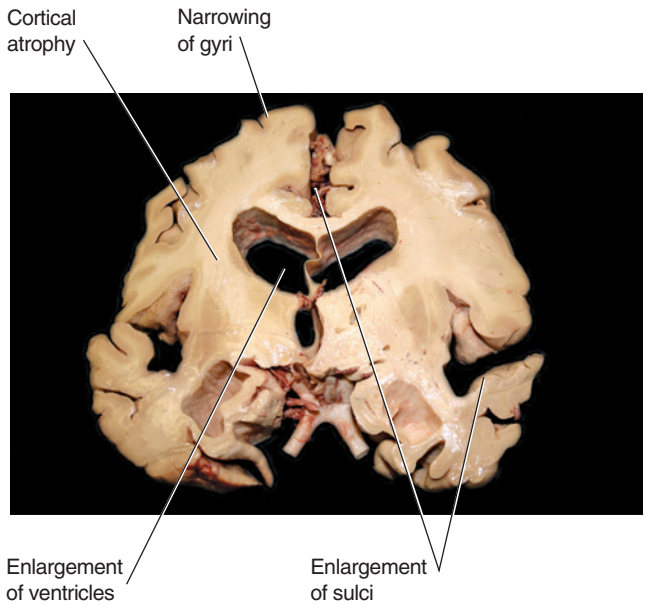


FIGURE 43.2 ■ Changes in neuroanatomy associated with Alzheimer's disease. Note areas of cortical atrophy, narrowing of the gyri, enlargement of sulci and ventricular dilation

Source: © Living Art Enterprises/Science Source.

Groups of nerve cells (especially the terminal axons) degenerate and clump around an amyloid core as plaques, and are found in the spaces between the neurons of the brain. These plaques, which develop first in areas used for memory and cognition, disrupt transmission of nerve impulses. The plaques consist primarily of insoluble deposits of beta-amyloid, a protein fragment from a larger protein called amyloid precursor protein, mixed with other neurons and non-nerve cells. It is not yet known if plaque formation causes AD or if plaques are a by-product of the AD process.

Blood flow to the affected areas of the brain is decreased. The brain atrophies and corresponding enlargement of ventricles and sulci is evident (see Figure 43.2). As AD progresses, more areas of the brain are affected, with manifestations correlating to those affected areas of the brain. For example, neuronal and neurotransmitter losses in the parietal lobe result in problems with perception and interpretation of environmental stimuli; deficits in the frontal lobe cause changes in personality and emotional lability.

AD is characterised by atrophy of the cortical area of the brain and loss of neurons, especially in the parietal and temporal lobes. With significant atrophy and loss of brain tissue, the ventricles enlarge (a form of hydrocephalus) (Grossman & Porth, 2013). Several structural and chemical changes in the brain occur with AD, especially in the hippocampus and the frontal and temporal lobes of the cerebral cortex. As AD destroys neurons in the hippocampus and related structures, short-term memory fails, and the ability to perform easy and familiar tasks declines. The effect of AD on neurons in the cerebral cortex is loss of language skills and judgment. Emotional outbursts and behaviour changes (such as wandering and agitation) begin to occur and become more frequent as the disease progresses. Eventually, other areas of the brain are affected; all affected areas begin to atrophy and the person becomes totally helpless and unresponsive.

The exact cause of AD is unknown. Theories include a decrease in choline acetyltransferase activity in the cortex and hippocampus. This enzyme is necessary for the synthesis of acetylcholine, a neurotransmitter associated with memory. The decrease in choline acetyltransferase is about equal to the severity of AD. Other theories include a mutation for encoding amyloid precursor protein and alteration in apolipoprotein E. Other possible causes are gene defects on chromosomes 14, 19 or 21, which may lead to clumping and precipitation of insoluble amyloid as plaques. The role of protein kinase C, the link between AD and aluminium, a viral cause, an autoimmune cause and mitochondrial defects that alter cell metabolism and protein processing are being studied.

Manifestations

Alzheimer's disease is classified into three stages based on the person's manifestations and abilities, as outlined in the box below. It is important to note that the progression of AD varies for each individual and may not precisely follow the model.

Stage 1 AD

In stage 1, a person typically appears physically healthy and alert, and cognitive deficits can go undetected unless thorough and periodic evaluations are performed. People may seem restless, forgetful or uncoordinated; they may lack spontaneity and be disoriented as to time and date. Usually, family members are the first to notice lapses in memory, subtle changes in personality or problems in doing simple calculations. People with AD and their families may consciously or unconsciously compensate for cognitive deficits by adjusting schedules and routines.

Stage 2 AD

In stage 2, memory deficits are more apparent and the person is less able to behave spontaneously. People may wander and get lost, even in their own homes. Although progression of manifestations continues and orientation to place and time deteriorates, people with AD may still have periods of mental lucidity and engage in time-oriented conversations. Generally, however, people become more confused and lose their sense of time, leading to changes in sleeping patterns, agitation and stress. They may demonstrate repetitive behaviour and eat ravenously. People with AD are less capable of making even simple decisions and adapting to environmental changes, and are often unable to carry out ADLs. **Sundowning** is another behavioural change, characterised by increased agitation, time disorientation and wandering behaviours during afternoon and evening hours; it is accelerated on overcast days.

Language deficits are common in stage 2. They include *paraphasia* (using the wrong word), *echolalia* (repetition of words or phrases) and *scanning speech*, in which the person appears to search for words. Eventually, *total aphasia* (absence of speech) may occur. Frustration and depression are common in people with AD as the full extent and implications of the deficits become obvious.

The person with AD slowly loses the ability to perform simple tasks required for hygiene or eating because sequencing of tasks is lost. For example, the person may open a can of soup but not remember to pour it into a pan to heat it. Instead, the person might place the can directly on the burner and leave the heat on high even after a smoke alarm sounds. The person with AD may falsely interpret the smoke alarm as a telephone ringing or an ambulance siren. Thus, safety is a high priority for the person in stage 2.

Sensorimotor deficits in stage 2 include *apraxia*, the inability to perform purposeful movements and use objects correctly; *astereognosis*, the inability to identify objects by touch; and *agraphia*, the inability to write properly. Problems related to malnutrition and decreased fluid intake, such as anaemia and constipation, may be evident. Sleep pattern disturbances are also common and are related to the loss of time orientation, sundowning phenomenon and depression.

Stage 3 AD

Stage 3 brings increasing dependence, with inability to communicate, loss of urinary and faecal continence, and progressive loss of cognitive abilities. Common complications include pneumonia, dehydration, malnutrition, falls, depression, delusions, seizures and paranoid reactions. People with AD are indifferent to food and lose weight. They are unable to recognise family or friends, or even themselves. The average life expectancy is 1 to 2 years from the onset of stage 3, although the individual may live as long as 10 years. Most people with AD are institutionalised during this final stage of the disease. Death frequently occurs from pneumonia secondary to aspiration.

INTERPROFESSIONAL CARE

There is no cure for AD and the main objective of care is to provide an environment that matches the person's functional abilities. Nurses, doctors, physical therapists and social workers collaborate with the person's family to provide the least restrictive environment in which the person can safely function.

MANIFESTATIONS Alzheimer's disease

STAGE 1: APPROXIMATELY 2TO 4YEARS

- Short-term memory loss: forgets location and names of objects and has difficulty learning new information; long-term memory is unaffected.
- Decreased attention span.
- Subtle personality changes: lacks spontaneity; denial, irritability and depression are possible.
- Mild cognitive deficits: attempts to adjust to and cover up memory loss.
- Visuospatial deficits: some problems with depth perception.

STAGE 2: APPROXIMATELY 2TO 12YEARS

- Impaired cognition: obvious memory deficits and confusion; loss of abstract thinking; astereognosis and agraphia; inability to do maths calculations; loss of ability to tell time and time disorientation, manifested as 'sundowning'; wandering behaviour.
- Personality changes: becomes easily agitated and irritable; may have delusions or hallucinations.
- Visuospatial deficits: is unable to dress self; has poor spatial orientation.
- Impaired motor skills: paces and is restless at times; motor apraxia is evident when using familiar objects.
- Impaired judgment: diminished social skills; inability to drive a car; inability to make decisions (e.g. choose clothing).

STAGE 3: APPROXIMATELY 2TO 4YEARS OR LONGER

- Cognitive abilities grossly decreased or absent: is usually disoriented to time, place and person.
- Communication skills usually absent: is frequently mute.
- Motor skills grossly impaired or absent: limb rigidity and posture flexion; bowel and bladder incontinence.

Diagnosis

Alzheimer's disease is diagnosed by ruling out causes for the person's manifestations. The only definitive method of diagnosis is postmortem examination of brain tissue. An extensive work-up is especially important because the dementia may be due to a reversible or treatable condition. For example, an older person's misuse of medications can lead to overdosing and resulting confusion. Other categories of conditions that may be considered and ruled out include depression, infection, hypothyroidism, dehydration, heart disease, stroke and chronic obstructive respiratory disease. Mental status is assessed with tests such as the Folstein Mini-Mental Status Examination (Folstein, 1975). This examination assesses areas of function such as the person's orientation to time, ability to repeat a series of words, ability to name objects and ability to follow written instructions.

National policies and practice guidelines for the early recognition and assessment of AD have been established by Alzheimer's Australia. A diagnosis of Alzheimer's disease requires the documented presence of dementia, onset between ages 40 and 90 years (most often after age 65), no loss of consciousness and absence of systemic or brain disorders that could cause mental changes.

Medications

There is no cure for AD, but some medications are effective in slowing the progression of the disease. Tacrine hydrochloride (Cognex) was the first medication specifically approved for the treatment of AD. Donepezil hydrochloride (Aricept) is used to treat mild to moderate AD dementia with some success. Rivastigmine tartrate (Exelon) is also used to treat mild to moderate AD manifestations. It improves the ability to carry out ADLs, decreases agitation and delusions, and improves cognitive function. Galantamine hydrobromide (Reminyl) is believed to increase the concentration of acetylcholine in the central nervous system (CNS) and is used as a treatment of mild to moderate AD. Memantine (Ebixa) improves cognitive function in moderate to severe AD and mild to moderate vascular dementia. Memantine acts by blocking receptors for glutamate, resulting in decreased calcium accumulation into neurons (increased calcium accumulation damages neurons). See the box below for information about selected medications used to treat AD.

Depression often accompanies AD and is treated with the appropriate medication. Antihistamines and tricyclic antidepressants that have high anticholinergic activity are usually avoided because they can increase AD manifestations. Occasionally, people with AD require tranquillisers such as thioridazine (Mellaril) or haloperidol (Serenace) to manage severe agitation. Other therapies under study to prevent or delay the onset of AD include antioxidants such as vitamin E, anti-inflammatory agents and antihypertensive drugs to lower hypertension.

Alternative and complementary therapy

The following types of alternative and complementary therapies may be used in treating the manifestations of AD:

- massage, which decreases agitation
- herbs:
  - ginkgo biloba and vitamin E, which are thought to improve cognition
  - huperzine A, a traditional Chinese medicine, which acts as an acetylcholinesterase inhibitor

MEDICATION ADMINISTRATION The person with Alzheimer's disease

CHOLINERGICS (PARASYMPATHOMIMETICS); CHOLINESTERASE INHIBITORS

Tacrine hydrochloride (Cognex)
Donepezil hydrochloride (Aricept)
Rivastigmine tartrate (Exelon)
Tacrine hydrochloride (Cognex)

In the early stages of AD, the pathological changes in neurons result in a deficiency of acetylcholine (a key neurotransmitter involved in cognitive functioning). Cholinesterase inhibitors slow the breakdown of acetylcholine release by the remaining intact neurons. In addition, rivastigmine tartrate inhibits the G1 form of acetylcholinesterase (found in higher levels in the brain of people with AD), so less acetylcholine is degraded. The drugs are used to improve memory in mild to moderate AD dementia.

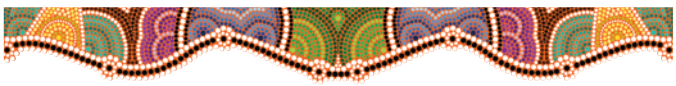
Nursing responsibilities

- Administer tacrine hydrochloride 1 hour before meals, if possible.
- Administer donepezil hydrochloride at bedtime.
- Administer rivastigmine tartrate (both capsules and liquid) with food. Liquid form may be administered undiluted or mixed with water, juice or soft drink. Stir to completely dissolve.
- Monitor for jaundice, increased bilirubin levels and other signs of liver involvement, such as rising serum aminotransferase (AST, ALT) levels. Therapy is usually decreased when the enzyme level exceeds four times normal limits and discontinued when the level reaches five times normal.
- Observe for gastrointestinal bleeding and gastric ulcer pain.
- Monitor for cholinergic-related problems: bladder outlet obstruction, seizures and slowed heart rate.
- Assist with ambulation because dizziness is a common side effect.
- Monitor glycaemic control in people with diabetes.
- Assess for improvement in AD symptoms, especially in reasoning, memory and ADLs.

Health education for the person and family

- Notify the healthcare provider promptly if jaundice, seizures, slowed heart rate, GI bleeding or difficulty in urinating occurs.
- Follow directions for times and instructions about administration of specific medication.
- Follow your healthcare provider's recommendation for periodic EEGs, blood tests and urine tests.
- These medications do not cure AD and will at some point become ineffective as the disease progresses.

- coenzyme Q10, an antioxidant that occurs naturally in the body
- supplements, such as zinc, selenium and evening primrose oil
- therapies involving art, music, sound and dance.



Nursing care

People with AD often require intensive, supportive nursing interventions directed at the physical and psychosocial responses to illness. Equally important, the nurse can facilitate the long-term support of these people by providing teaching and referrals to follow-up care in the community. A nursing care plan for the person with AD is found below.

Health promotion

Health promotion for the person with AD focuses on maintaining functional abilities and safety. If the person will be cared for at home, address safety considerations (see the box below) as well as the caregivers' abilities to meet the person's basic needs, such as maintaining hygiene and other ADLs. Adapt nursing interventions and teaching to the person's stage of Alzheimer's disease. Nurses also promote health in the caregiver; information about caregiver support systems and respite care should be provided.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40). Further focused assessments are described with nursing interventions below.

- *Health history:* family member/caregiver support, living arrangements, ability to carry out ADLs, drug use, work history (e.g. exposure to metals), previous history of multiple strokes, brain injury or brain infection, family history of dementia, sleep pattern, changes in cognition and memory, ability to communicate, changes in behaviour.
- *Physical assessment:* height/weight, orientation, abstract reasoning, mental status.

Nursing diagnoses and interventions

During the early stage of AD, nursing care focuses on helping the person make minor adaptations to his or her environment. As the person becomes progressively unable to manage self-care tasks, more adaptations are required. Equally important, the caregiver needs much support—both physical and psychosocial—as the person becomes increasingly dependent.

Impaired memory

Impaired memory is an appropriate nursing diagnosis in stage 1 AD. At this stage, techniques to help with the memory loss should be included in teaching for both the person and the caregiver.

- Suggest complementary therapies, such as meditation, massage or exercise. *These activities can help reduce stress; stress can aggravate memory loss.*
- Suggest using a calendar, keeping lists of reminders or asking someone else to remind of appointments and events. *Written or verbal reminders are helpful if memory is impaired.*
- Recommend using a medication box labelled with days and times. A *medication box is a good way to remember to take medications.*

NURSING CARE PLAN A person with AD

Arthur and Ruth Joste, both aged 73, have been married for 47 years; he is a retired school teacher and she has been a homemaker. They have four children; two live in the same town and two live out of state. Arthur has noticed that he is having problems remembering friends' names and phone numbers; his wife has been asking him if he is driving in the correct direction when they go shopping.

Mrs Joste has severe osteoarthritis and is unable to lift heavy objects or perform all but light housekeeping tasks. For about 18 months, Mrs Joste has been aware of her husband's progressive cognitive decline, including forgetting current news from last night's TV news; miscalculating bank balances; neglecting his hygiene needs; and confusing their children's and grandchildren's names. The Jostes are referred to a neurologist for evaluation.

ASSESSMENT

Martha Spital, RN, assesses Mr Joste at the neurologist's office. She notes that he is unable to recall his home address without prompting, to name the correct date (although he does know the day of the week), to subtract serial 7s more than twice and to recall two of three objects. He is alert to his surroundings. Mrs Joste states that the problems seem to be getting worse with time and that she has had to 'cover up' mistakes for her husband. Mr Joste seems easily agitated and his wife reports that his sleep habits are 'jumbled'; he has long periods of wakefulness in the night-time hours.

Following a thorough evaluation and diagnostic testing to rule out other possible disorders, the neurologist tells the couple that Mr Joste has probable dementia of the Alzheimer's type. Both have feared this diagnosis; they want to know how they can be sure that Mr Joste has this disease and what they can do to prevent further decline. Both are obviously much saddened and they verbalise their feelings of being overwhelmed. The Jostes intend to remain in their home 'for as long as we can'.

NURSING CARE PLAN A person with AD (continued)

DIAGNOSES

- *Chronic confusion* related to deterioration of brain function and dementia manifested by forgetfulness.
- *Self-care deficits* related to forgetfulness and declining physical abilities, evidenced by poor hygiene.
- *Risk of injury* related to decreased orientation manifested by a decreased awareness of surroundings.
- *Disturbed sleep pattern* related to time disorientation, evidenced by periods of wakefulness.
- *Caregiver role strain* (wife) related to need to care for self and husband manifested by husband's increased need for assistance with ADLs.

PLANNING

Provide education about Alzheimer's disease, evaluate the environment, assess available support and determine needs in order to establish a management plan.

Expected outcomes

- The Joste family becomes aware of the characteristics of Alzheimer's disease and its progression.
- Remain free of injury.
- Navigate home environment with modifications as needed.
- Participate in grooming and hygiene activities with prompting and supervision.
- Obtain a minimum of 7 uninterrupted hours of sleep a night.
- Mrs Joste will participate in a minimum of two out-of-home activities a week.

IMPLEMENTATION

The community nurse, Eric Montane, RN, makes a home visit to evaluate the environment, assess available support and determine needs. He meets two of the Jostes' children, Dawn and Jay, who live in the same community and are willing to participate as much as possible in providing care and modifying the home.

Mr Montane discusses the importance of establishing and maintaining a consistent daily routine. He emphasises the importance of matching activities to Mr Joste's mental abilities to avoid frustration and increased agitation. Mr Montane recommends labelling drawers with their contents, such as Mr Joste's sock drawer. Labelling rooms may be necessary eventually.

Because his inability to comprehend and process information distresses and agitates Mr Joste, Mr Montane teaches the family to modify their communications to fit Mr Joste's cognitive ability, such as using simple, direct statements and directions.

Mr Montane recommends that family members keep background noise to a minimum because this may be a source of confusion.

After assessing the home, Mr Montane makes the following recommendations about safety:

- Remove throw rugs from hallways and tack down any remaining carpets.
- Secure the kitchen, bathroom and workshop cabinets, as well as the controls on the oven and stove.
- Modify the doors so that negotiating locks requires a two-step system of unlocking, such as with a deadbolt and a key.
- Provide extra lighting in dark areas, especially a night light in the bathroom.

Mr Montane explains that Mrs Joste will need assistance with housekeeping as Mr Joste continues to decline. Mr Montane provides referrals to community services, including Meals on Wheels, which can supply a daily meal. He also suggests that the Jostes obtain home care services to provide daily hygiene care. Most of the remaining home maintenance needs can be met with the children's help.

Mr and Mrs Joste and the two children attend the weekly local support group meetings for Alzheimer's disease and related disorders for approximately 3 months; thereafter, Mrs Joste attends with her daughter.

EVALUATION

Six months after the initial home visit and family planning session, Mr Joste:

- Has not had a fall, burn or other injury.
- Has periods of confusion when outside his home, but 90% of the time is oriented to place when at home.
- Has attended several support group meetings until 3 months ago. Currently, his wife attends weekly and a daughter occasionally accompanies her. She has continued to participate in their church and maintains contact with a few friends. She is finding it harder to leave her husband unattended for even a few minutes.
- Is able to clean and dress himself with prompting; he is not able to choose his own clothing. If hygiene articles are 'set up' (e.g. if the toothpaste is placed on the toothbrush), he remembers to perform the hygiene activity. The children have been replacing buttons and zippers with Velcro closures on his clothing.
- Sleeps an average of 6 hours a night with a 30-minute nap in the afternoon; this pattern is consistent with his previous sleep pattern.
- Has been more easily agitated for the past month. He wanders from room to room, apparently looking for something. These behaviours are worse in the evening and on cloudy days. Mrs Joste acknowledges her progressive inability to care for her husband.

CRITICAL THINKING IN THE NURSING PROCESS

1. Develop a tool to teach safety needs for the person and the family with Alzheimer's disease.
2. List five interventions to decrease agitation in cognitively impaired older adults; give three additional examples of activities suited to an older adult with AD who has osteoarthritis.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from this case study that you will apply to your future nursing practice.
2. Which education strategies could you use to ensure a person with AD receives adequate nutrition?

MEETING INDIVIDUALISED NEEDS **Safety interventions for the person with AD**

Decreasing the risk of falls

- Assess usual environment for hazards, such as throw rugs, electrical cords and slick floors.
- Observe areas of special concern, such as the bathroom, kitchen and stairs, and modify as needed; for example, provide skid-proof surfaces and mark stairs to show depth.
- Evaluate muscle strength and gait; consult a physiotherapist to plan exercises to increase strength and balance.
- Check shoes for fit and support.
- Enquire about alcohol use and medications that affect balance or cause mobility problems; for example, antihypertensive agents can cause dizziness with position changes.
- Use night lights and increase daytime lighting in dark areas such as hallways.
- Keep traffic areas free from clutter.

Decreasing the injuries related to cognitive impairments

- Secure items that may be mistakenly ingested, such as cleaning preparations and house plants.
- Modify potentially unsafe areas, such as unenclosed porches.
- Provide double-lock systems to outside doors and doors to rooms that are off limits.
- Protect from fire hazards; for example, make matches and cigarettes inaccessible.
- Fence the yard with a locked gate to prevent wandering.
- Modify the controls on the oven and stove.
- Adjust the water heater to a safe temperature.

General safety considerations

- Plan a calling system for emergencies; have children call at about the same time every day as a check.
- Ensure that the cognitively impaired family member has no access in the home to objects such as knives and guns.

- If safety is a concern (such as turning on the stove and forgetting it), suggest using alternatives such as a microwave. Program emergency numbers into the telephone. Ask the person to consider a personal emergency alarm system. *These measures can increase safety.*
- Suggest using cues, such as an alarm on a watch or a mobile phone, to trigger actions at designated times. *Cues are often helpful when memory loss is a problem.*

CONSIDERATION FOR PRACTICE

It may be necessary for the caregiver to arrange for the person's medications to be supplied by the pharmacy in a prearranged pack such as a Webster pack.

Chronic confusion

People with AD often have memory deficits that make functioning in a non-structured environment difficult. Many of the nursing interventions for this diagnosis need to be modified over time as the person continues to lose cognitive function.

- Label rooms, drawers and other items as needed. *Visual cues promote the highest possible degree of independence for the person.*
- Remove potential hazards (such as sharp knives or potentially harmful liquids or chemicals) from the environment. *Ensuring safety is a critical factor in providing care.*
- Keep environmental stimuli to a minimum: decrease noise levels; speak in a calm, low voice; and take an unhurried approach. *Minimising sensory input and maintaining a calm manner may decrease anxiety.*
- Begin each interaction by identifying yourself and calling the person by name. See Box 43.1 for other communication techniques. *These techniques provide information for the person with memory loss.*
- Limit questions to those that require a simple 'yes' or 'no' response. Questions need to be appropriate to the person's ability as decision-making and verbal skills decline.
- Orient to the environment, person and time as able, and place large, easy-to-read calendars and clocks in the

BOX 43.1 Communicating with the person with AD

- Face the person and talk directly to them; call the person by name.
- When first approaching the person, identify yourself.
- Use simple sentences and words with few syllables.
- Speak in a calm, low voice.
- Ask one question at a time. Use questions that require only a 'yes' or 'no' response.
- Keep non-verbal communication relaxed and parallel to the verbal communication.
- Avoid giving the impression of being in a hurry; try to have a relaxed approach.
- Observe for anxiety—wringing hands, pacing, darting eye movements—and alter your approach to decrease anxiety.
- Avoid arguing with people; do not insist on orienting person to reality; the person's point of reference may not be based in reality.
- Give plenty of time for the person with AD to process what you are trying to say; do not expect people to perform skills beyond their abilities.
- Repeat explanations in simple terms.

person's line of vision. Make references to the season or day of the week when conversing with the person. Orient the person according to their own level of ability. *Orienting to precise time may not be possible in the later stages of AD.*
- Provide boundaries by placing red or yellow tape on the floor. *Boundaries help the person stay within safe areas.*

CONSIDERATION FOR PRACTICE
Red and yellow are more easily seen by older adults.

- Provide continuity in nursing staff. *This not only promotes consistency of care for the person but also allows the nurse to determine more accurately changes in the person's condition.*
- Repeat explanations simply and as needed to decrease anxiety. *Loss of short-term memory leads to loss of a point of reference; eventually, people with AD think they are experiencing everything for the first time.*

Anxiety

Managing the AD person's behaviours associated with anxiety, restlessness and confusion is a major challenge confronting nurses and caregivers. Frequently, people are relatively calm in the morning hours, only to experience increasing periods of agitation in the afternoon and evening hours. The person with AD may even wake from the night's sleep with confusion, fearfulness or panic attacks.
- Monitor for early behaviours of fatigue and agitation. *Early assessment of problems results in prompt intervention to promote rest or to remove the person from the situation causing anxiety.*
- Remove from situations that are causing increased anxiety, such as noisy activities involving large groups. *High-stimulus situations may increase anxious feelings and agitation.*
- Keep daily routine as consistent as possible. *Providing a structured day enhances feelings of familiarity and decreases stress.*
- Schedule rest periods or quiet times throughout the day. *Fatigue contributes to anxiety and lowers the ability to tolerate stress.*
- Provide quiet activities, such as listening to music, in the afternoon or early evening. *Quiet activities may help decrease sundowning.*
- If confusion and agitation persist or escalate, assess for physical causes such as decreased oxygenation, infections, fatigue, constipation and electrolyte imbalance. *Physical factors can increase agitation in people with AD.*
- Use therapeutic touch or gentle hand massage. *These activities induce relaxation and have a calming effect.*

Hopelessness

As the person and their family recognise the effect of AD on their lives, they may feel a sense of hopelessness. They may not have the coping skills to deal effectively with the diagnosis and anticipated problems. The increasingly degenerative, irreversible nature of the disorder tends to diminish hope; only the ability to adapt to the many problems can restore it.
- Assess the person's and family's response to the diagnosis and understanding of AD; encourage expression of feelings. *Understanding the person/family's perspective enables the nurse to dispel myths about AD.*
- Provide realistic information about the disorder; provide information at the person/family's level of understanding. *The person and their family may need to have separate sessions. Factual information provides a foundation for decision making.*
- Avoid criticising or judging expressed feelings. *An environment accepting of the expression of real feelings promotes both further expression of feelings and willingness to discuss other issues.*
- Support positive family bonds and enhance communication between family members; promote mutual positive regard. *Strong family relationships can provide direction for living and convey willingness to share the burden.*
- Encourage the person to make as many decisions as possible. *Self-determination enhances a feeling of control over a situation and may give a sense of hope.*
- Encourage the person and their family to seek spiritual guidance that previously inspired hope. *The person's religious affiliation is a legitimate support system. Belief in a higher being can inspire hope beyond present circumstances.*

Caregiver role strain

Most caregivers of people with AD are spouses or other family members. Because AD is a chronic and eventually debilitating disorder, caregivers may feel overwhelmed by their responsibilities. The caregiving spouse faces not only the responsibility for the person's multiple physical demands but also economic and psychosocial stressors. An area that must be discussed is the ability and safety of the person in driving an automobile. Although it may be necessary, the loss of independence represented by the loss of the ability to drive may further trigger anxiety and anger. Fear of the future, loss of income, loss of companionship and a mate—combined with fatigue—make the caregiver vulnerable. Caregivers may become physically and mentally exhausted and socially isolated because of the overwhelming responsibilities of providing total care to the incapacitated family member.
- Teach the caregivers self-care techniques, such as taking rest periods and avoiding fatigue. *Fatigue adds to stress and potentially leads to poor decision making.*
- Have the caregivers list and regularly take part in physical activities they enjoy, such as walking or swimming. *Regular physical exercise decreases stress.*
- Refer the caregivers to local AD support groups. Suggest books pertinent to the subject. *Explicit suggestions in locating support systems and providing specific information promote coping.*

- Refer the caregivers to Meals on Wheels, home care, respite care and other community services. *Community agencies can relieve some of the daily care burdens, thus providing time for other activities. Programs that support caregivers have been shown to delay nursing home placement.*
- Ensure the family knows that hospice care is available during the end stages of AD. *Hospice services can support the family during this difficult time*.

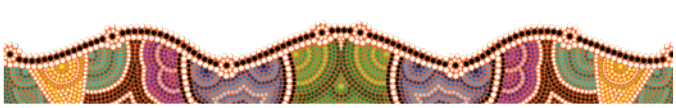
Community-based care

Teaching for people and families initially centres on explaining the disorder and exploring available support systems. Anticipate the need to re-explain the disorder and its consequences because people and families may be in shock or denial during the initial period of the disease.

In addition to explaining the anticipated changes with AD, suggest practical solutions to identified problems. It is important to evaluate both the person and caregivers; interventions must be appropriate for the family's situation and resources. Maintaining the least restrictive environment that promotes safety for the person is a major goal of teaching. Using memory cues, such as labelling drawers to indicate the specific types of clothing and labelling rooms, can help orient the person and foster independence. Consistency in the environment and daily routine is an essential part of care. Emphasising realistic expectations means adjusting care and communication techniques to the person's level of ability.

Address the following topics for home care of the person and for the caregiver:

- Support groups and peer counselling are helpful in handling caregiver stress.
- A person with AD who is confused or agitated is not comfortable and is usually frightened.
- Plan care that matches the person's level of coping, using a consistent routine.
- Provide regular rest periods to decrease the person's stress and fatigue. (These do not increase night-time wandering.)
- Plan care for the caregiver. Periodic adult day care or respite care during the initial stages, with plans for increasing assistance to meet the person's daily needs as the disease progresses, may be sufficient. Referrals to the appropriate agency for long-term care, including skilled nursing facilities, may be indicated. Family members may need help adjusting to the idea of extended care but may be relieved to relinquish the physical care needs.
- Suggest the following resources:
  - Alzheimer's Australia: www.fightdementia.org.au
  - National Dementia Helpline: 1800 100 500
  - Carers Australia: www.carersaustralia.com.au
  - My Aged Care: www.myagedcare.gov.au or 1800 200 422



THE PERSON WITH MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is a chronic demyelinating neurological disease of the CNS (brain, optic nerves and spinal cord), associated with an abnormal immune response to an environmental factor. The manifestations of MS vary according to the area of the nervous system affected. The initial onset may be followed by a total remission, making diagnosis difficult. In about 60% of people, MS is characterised by periods of exacerbation, when manifestations are highly pronounced, followed by periods of remission, when manifestations are not obvious. The end result, however, is progression of the disease with increasing loss of function.

Incidence and prevalence

The onset of MS is usually between 20 and 40 years of age, with three times as many women as men being diagnosed. MS is the most prevalent CNS demyelinating disorder and is a leading cause of neurological disability in young adults. MS is more common in Caucasians, especially those of northern European ancestry; however, MS does occur in people of African, Asian and Hispanic descent. A definite genetic factor has not been established but studies suggest that genetic factors may make some individuals more susceptible than others (MS Australia, 2015).

Pathophysiology

MS is believed to occur as a result of an autoimmune response to a prior viral infection in a genetically susceptible person. The infection, which is thought to occur early in life, activates T cells. T cells usually move in and out of the CNS across the blood–brain barrier but, for an unknown reason, they remain in the CNS in people with MS. The T cells facilitate infiltration by other leucocytes and an inflammatory process follows. Inflammation destroys myelin and oligodendrocytes (myelin-producing cells), leading to axon dysfunction.

Myelin sheaths are fatty, segmented wrappings that normally protect and insulate nerve fibres and increase the speed of transmission of nerve impulses. In multiple sclerosis, these myelin sheaths of the white matter of the spinal cord, brain and optic nerve are destroyed in patches, called *plaques*, along the axon (see the following 'Pathophysiology illustrated' feature). The demyelination of nerve fibres slows and distorts the conduction of nerve impulses and sometimes results in the total absence of impulse transmission. The neurons usually affected by MS are located in the spinal cord, brainstem, cerebral and cerebellar areas, and the optic nerve.

Both plaques and diffuse lesions form as demyelinating lesions. Plaques typically are scattered through the white matter of the CNS, although they may extend into adjacent grey matter. Early manifestations are the result of inflammatory oedema in and around the plaque and partial demyelination. These manifestations typically disappear within weeks after the initial episode. With progression of the disease, the demyelination and plaque formation result in scarring of glia (*gliosis*) and degeneration of axons. Continued loss of function leads to permanent disability, usually over about 20 years.

FAST FACTS

- Approximately 23 000 people in Australia have MS; the incidence is 2.5 million worldwide.
- Females are affected three times as often as males, and the incidence is highest in young adults under 55 years old.
- The disease occurs more commonly in temperate climates. This association is established by approximately age 15, and moving to or from a temperate climate after that age does not change it. In Australia, there is a significantly higher incidence of MS in Tasmania than in mainland Australia. No significant increase of disease incidence is noted in Indigenous Australians.

Sources: MS Australia (2015); Grossman & Porth (2013).

There are four classifications of MS: relapsing–remitting, primary progressive, secondary progressive and progressive–relapsing (see Box 43.2). Most people with MS present with the relapsing–remitting type.

Various stressors have been suggested as triggers for MS. These stressors include febrile states, pregnancy, extreme physical exertion and fatigue. These precipitating factors can also cause a relapse of the manifestations during the course of the disease.

Manifestations

The manifestations of MS vary according to the areas destroyed by demyelination and the affected body system (see the 'Multisystem effects of MS' feature below). Fatigue is one of the most disabling manifestations and affects almost all people with MS. The manifestations, categorised by the established syndromes of MS, are listed in the box below.

Brief attacks of manifestations are described as short lived or paroxysmal. Short-lived attacks of neurological deficits indicate the appearance or worsening of manifestations. Conditions that cause short-lived attacks include: (1) minor increases in body temperature or serum calcium concentrations (both increase the leakage of current through demyelinated neurons); and (2) functional demands that exceed conduction capacity. Paroxysmal attacks are sensory or motor manifestations that occur abruptly and last for only seconds or minutes; the manifestations are paraesthesias, dysarthria and ataxia, and tonic head turning. Paroxysmal attacks, which may occur many times a day, result from the direct transmission of nerve impulses between adjacent demyelinated axons.

BOX 43.2 Classifications of multiple sclerosis

Relapsing–remitting: the most common clinical course of MS, characterised by exacerbations (acute attacks) with either full recovery or partial recovery with disability.

Primary progressive: steady worsening of disease from the onset with occasional minor recovery.

Secondary progressive: begins as with relapsing–remitting, but the disease steadily becomes worse between exacerbations.

Progressive–relapsing: this rare form continues to progress from the onset but also has exacerbations.

MANIFESTATIONS Multiple sclerosis

MIXED OR GENERALISED TYPE (50% OF CASES)

- Visual deficits, with visual blurring, fogginess or haziness; impaired colour perception, decreased central visual acuity, area of diminished vision in the visual fields, acquired colour-vision deficit (especially to red and green) and an altered pupillary reaction to light.
- Brainstem lesions (cranial nerves III to XII) with nystagmus, dysarthria, deafness, vertigo, vomiting, tinnitus, facial weakness, decreased sensation, diplopia and eye pain; and cognitive dysfunctions involving concentration, short-term memory, word finding and planning.
- Mood alterations are manifested as depression more often than euphoria.

SPINAL TYPE (25% OF CASES)

- Weakness and/or numbness in one or both extremities (most often the legs).
- Upper motor neuron involvement is manifested by stiffness, slowness, weakness (spastic paresis).
- Bladder dysfunctions include urgency, hesitancy and incontinence.
- Bowel dysfunction is most often seen as constipation.
- Neurogenic impotence is noted.

CEREBELLAR TYPE (5% OF CASES)

- Manifestations of nystagmus, ataxia and hyptonia.

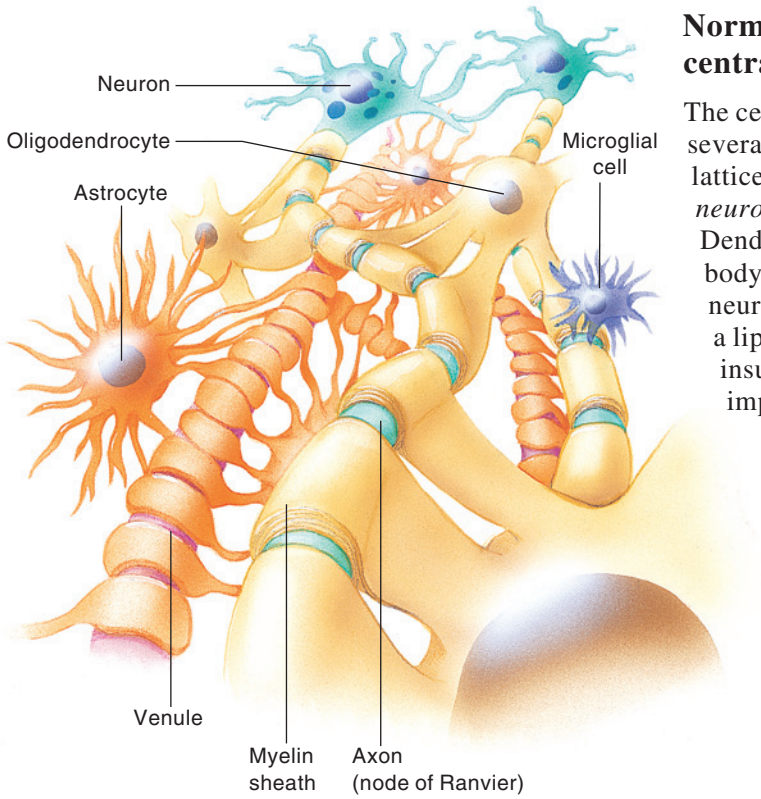
AMAUROTIC FORM (5% OF CASES)

- Blindness

INTERPROFESSIONAL CARE

Management of the person with MS varies according to the severity of the manifestations. The focus is on retaining the optimal level of functioning possible, given the degree of disability. Rehabilitation—physical, occupational/vocational and psychosocial—is a cornerstone of an interprofessional approach to treatment. During exacerbations, the focus of interventions shifts to controlling manifestations and quickly returning to remission.

Multiple sclerosis



Normal anatomy of the central nervous system

The central nervous system (CNS) is composed of several cell types arranged in a dense, interconnected lattice. The basic functional cell of the CNS is the *neuron*, which transmits electrochemical impulses. Dendrites, thin projections extending from the neuron body, receive impulses that are passed down the neuronal axon for transmission to other cells. Myelin, a lipid-protein substance, surrounds the axons, insulating them and speeding nerve impulse transmission.

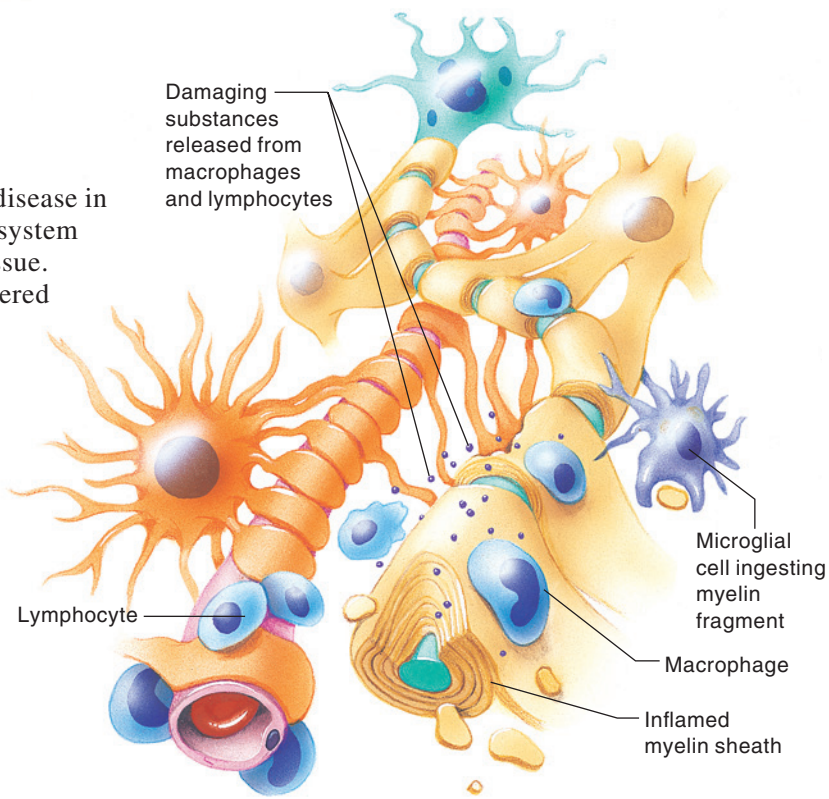
Neurons are surrounded by a network of cells:

- *Astrocytes* support neurons and connnect them to surrounding capillaries and venules.
- *Microglia* are motile phagocytic cells.
- *Oligodendrocytes* wrap concentric layers of myelin around nearby axons.

Acute attack

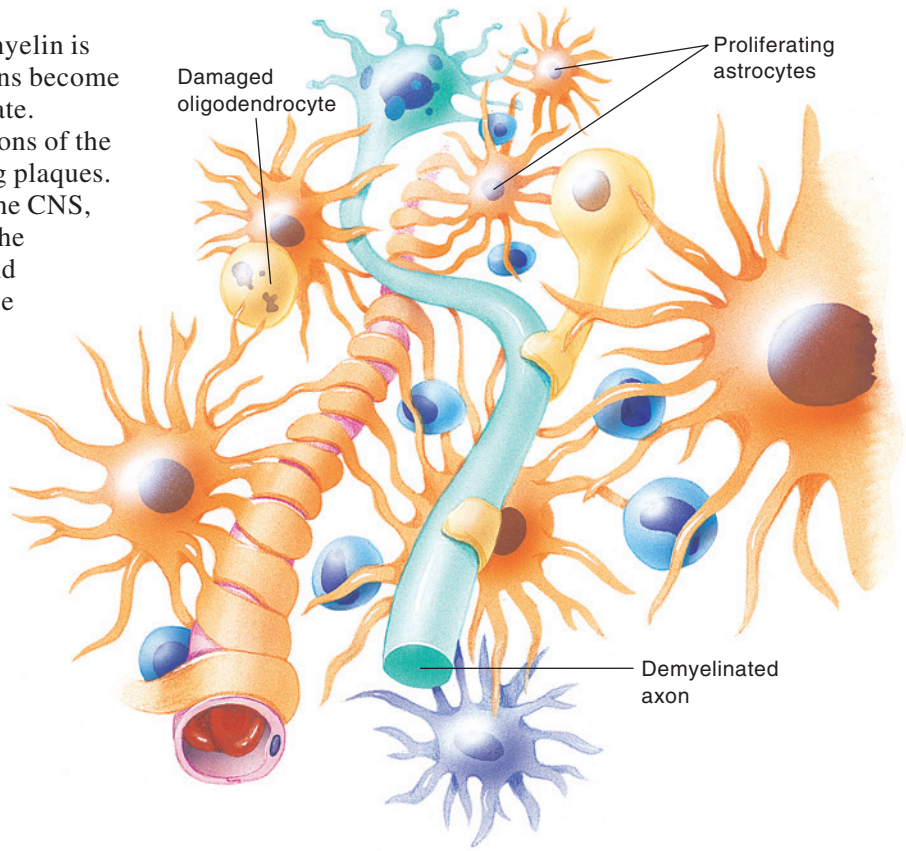
Multiple sclerosis (MS) is a demyelinating disease in which axonal myelin in the central nervous system is eroded, destroyed and replaced by scar tissue.

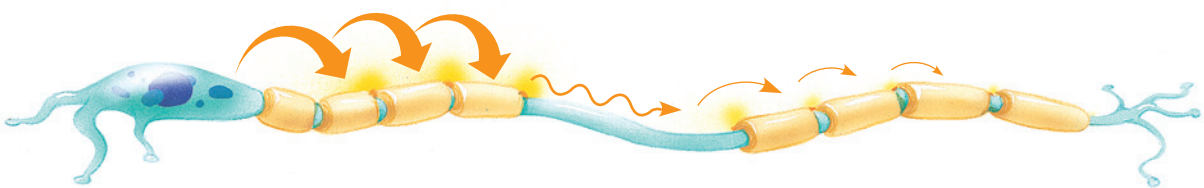
An autoimmune process apparently triggered by genetic and environmental factors is believed to cause inflammation of venules in the CNS. This disrupts the blood–brain barrier, allowing lymphocytes to enter CNS tissue. These lymphocytes proliferate and produce IgG, an antibody that attacks and damages myelin and causes the release of inflammatory chemicals and oedema. As the inflammation subsides, the myelin regenerates and manifestations of the disease subside.



Chronic lesion

After repeated inflammatory attacks, myelin is irreparably damaged. Segments of axons become totally demyelinated and may degenerate. Astrocytes proliferate in damaged regions of the CNS (a process called *gliosis*), forming plaques. The plaques are scattered throughout the CNS, appearing as grey or pinkish lesions. The relapsing-remitting character of MS and the scattered areas of damage within the CNS account for the variable nature of MS manifestations.

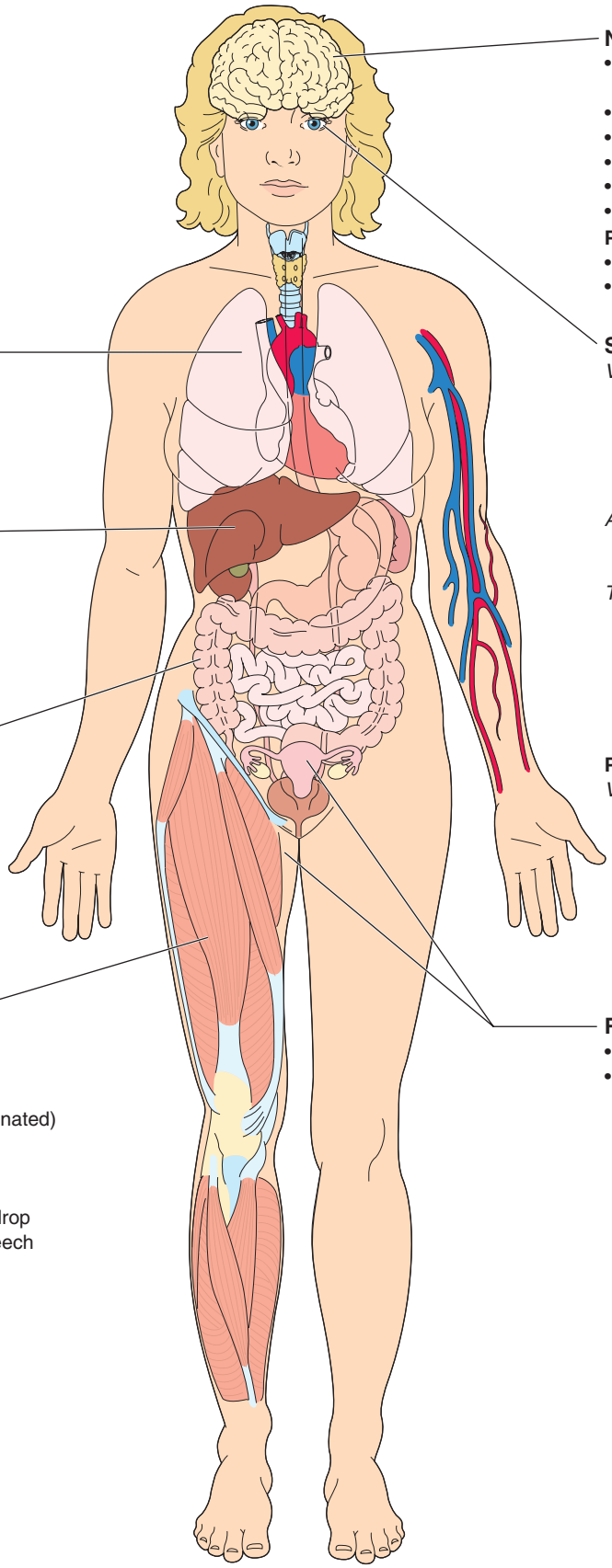




Abnormal nerve impulse transmission

In an undamaged neuron, nerve impulses travel down the axon by 'leaping' from one node of Ranvier to the next, thus greatly increasing the speed of impulse transmission. When nerve impulses travel down an axon damaged by MS, they are significantly slowed and weakened as they pass across the surface of demyelinated areas. Impulses may be blocked entirely when axons degenerate. The weakening or interruption of the transmission of nerve impulses and plaque formation within the CNS cause the manifestations of MS, including extremity weakness, paraesthesias, visual disturbances, bladder dysfunction and vertigo.

MULTISYSTEM EFFECTS OF MULTIPLE SCLEROSIS



Neurological
- Emotional lability (euphoria or depression)
- Forgetfulness
- Apathy
- Scanning speech
- Impaired judgment
- Irritability

Potential complications
- Convulsive seizures
- Dementia

Sensory

Visual
- Blurred vision
- Diplopia
- Nystagmus
- Visual field defects (blind spots)
- Eye pain

Auditory
- Vertigo
- Nausea

Tactile (especially hands or legs)
- Numbness
- Paraesthesias (tingling, burning sensation)
- Diminished sense of temperature
- Pain with spasms
- Loss of proprioception

Potential complication

Visual
- Blindness

Respiratory
- Diminished cough reflex

Potential complication
- Respiratory infections

Urinary
- Hesitancy
- Frequency
- Retention
- Reflex bladder emptying

Potential complications
- Recurring UTIs
- Incontinence

Gastrointestinal

Oral/oesophageal
- Difficulty chewing
- Dysphagia

Upper/lower GI
- Decreased or absent sphincter control
- Bowel incontinence
- Constipation

Musculoskeletal
- Fatigue
- Limb weakness
- Ataxic movements (shaky, irregular, uncoordinated)
- Intention tremors
- Spasticity
- Muscular atrophy
- Dragging of foot and foot drop
- Dysarthria with slurred speech

Reproductive
- Impotence (male)
- Loss of genital sensation

Diagnosis

Diagnosis of MS is challenging because the disease does not present uniformly. A diagnosis requires that the person has one of the following: (1) two or more exacerbations separated by 1 month or more and lasting more than 24 hours, followed by recovery; (2) a history of repeated exacerbations and remissions with or without complete recovery, followed by progressively more severe manifestations lasting for 6 months or more; or (3) slowly increasing manifestations for at least 6 months.

Diagnostic tests vary with the presenting complaints. Magnetic resonance imaging (MRI) with findings of lesions is the most definitive test available; however, it is only one of several laboratory and diagnostic tests that may be performed when establishing the diagnosis. Other tests (described in Chapter 40) include:

- Cerebrospinal fluid (CSF) analysis reveals an increased number of T lymphocytes that are reactive with antigens, indicating the presence of an immune response in the person (but is not specific to MS). Eighty per cent of people with MS have elevated levels of immunoglobulin G (IgG) in the CSF.
- Computed tomography (CT) scan of the brain shows atrophy and white matter lesions. In about 25% of people with MS, enlarged ventricles are visible on CT.
- Positron emission tomography (PET) scan measures brain activity. In people with MS the scan reveals areas with changes in glucose metabolism.
- Evoked response testing of visual, auditory or somatosensory impulses may show delayed conduction.

Medications

Medications slow the progression of MS and decrease the number of attacks. (See the 'Medication administration' box below.) Medications are used for a variety of reasons, including to treat manifestations, modify the course of the disease or interrupt the progression of the disease.

The medications used during an exacerbation are aimed at decreasing inflammation to inhibit manifestations and induce remission. Frequently, a combination of adrenal corticosteroid hormone (ACTH) and glucocorticoids is used to decrease inflammation and suppress the immune system. Immunosuppressive agents, including azathioprine (Imuran) and cyclophosphamide (Endoxan), are also used. Interferon and glatiramer acetate are used to reduce exacerbations in people with relapsing–remitting MS. Interferon alpha, beta and gamma (Roferon-A, Intron A, Wellferon, Imukin, Avonex or Rebif, Betaferon, Actimmune) enhance immune function, while glatiramer acetate (Copaxone) stimulates parts of the myelin basic protein to reduce the relapse rate of MS. Both drugs are given by injection and are usually well tolerated.

Other medications treat the manifestations of MS. Anticholinergics are administered for bladder spasticity; cholinergics are given if the person has a problem with urinary retention related to flaccid bladder. Depression is treated with antidepressant drugs.

Surgery

Surgery may be indicated for people who experience severe spasticity and deformity. However, physical therapy can prevent most severe problems. Foot drop from severe plantar flexion can be relieved with an Achilles tenotomy, a surgical procedure in which the Achilles tendon is transected.

Nutrition and fluids

Several diets involving manipulation of fats are currently under investigation. People with MS may be overweight because of their inability to ambulate; depression may contribute to the problem because people who are depressed tend to eat more. Ideally, the person should maintain a weight as close as possible to that recommended for the person's height and body type.

As MS progresses, the person's ability to prepare food and eat is compromised. Changes in muscle tone, tremor, weakness and ataxia all contribute to nutritional problems. Dysphagia also is a common problem. The diet must be adapted to accommodate changes in the person's ability to chew and swallow.

Rehabilitation

Physical and rehabilitative therapies are tailored to the person's level of functioning. The long-term goal is to enable the person to retain as much independence as possible. One major intervention is to maintain and increase existing muscle strength.

Spasticity is managed with stretching exercises, gait training and braces, splints or other assistive devices. To maintain balance, the person is encouraged to widen the base of support by standing with the feet slightly further apart. Walkers and canes may be weighted to provide support and balance for the ataxic person.

An interprofessional approach to rehabilitation will provide supportive services: speech therapy for problems with phonation, occupational therapy to maintain strength in the upper extremities and carry out ADLs, and occupational counselling. Referrals to a urologist are indicated for problems with urinary incontinence, urinary tract infections, retention and impotence. Consultation with a physiotherapist may be needed if the person develops chronic respiratory infections due to the inability to cough, move secretions or breathe deeply, especially with increased debilitation.

Nursing care

Because the disease most often affects young adults in the prime of life, the psychosocial and economic effect can be devastating. People with MS have to make adjustments to body image changes while simultaneously adapting to the altered relationships and decreased earnings usually encountered with the disease. A once-healthy spouse becomes wheelchair bound; a person once independent may eventually become dependent for even the most basic ADLs. The unpredictable course of MS is a challenge for long-term planning. A nursing care plan for the person with MS is given below.

- Suggest performing tasks in the morning hours. *Biorhythm studies indicate that people usually have greater energy reserves in the morning hours and diminished reserves in the afternoon.*

MEDICATION ADMINISTRATION The person with multiple sclerosis

IMMUNOMODULATORS

Interferon beta-1a (Avonex)
Interferon beta-1b (Betaferon)
Glatiramer acetate (Copaxone, Copolymer-1)

Interferon beta-1a, interferon beta-1b and glatiramer acetate are administered to people with relapsing–remitting MS to prolong the time of onset to disability. Their use is based on the assumption that MS is an immunologically mediated disease. Interferon beta-1b produces a decrease in the MS lesions in some people. Some people, however, develop a decrease in the absolute neutrophil count and increases in the levels of liver enzymes. Anxiety, confusion and depression with suicidal tendencies also have been reported. Other adverse reactions include pain, inflammation, hypersensitivity at the injection site and generalised flu-like manifestations. Some women experience menstrual disorders. Pregnant women should not take these medications.

Nursing responsibilities

- Assess baseline parameters to evaluate drug side effects: psychological profile, liver function tests and FBC with differential.
- Monitor FBC and liver function tests every 3 months or as prescribed.
- Assess injection site and report ulceration promptly. (Pain and redness are common reactions.)
- Evaluate the person's baseline neurological, sensory and motor function. Monitor changes in condition and function.
- Report if the person is pregnant or breastfeeding.

Health education for the person and family

- These drugs may cause depression and thoughts of suicide; report these feelings immediately to the healthcare provider.
- Administer medication within 3 hours of reconstitution. Rotate injection sites and avoid any areas that are red or show other skin reactions.
- Seek follow-up care to monitor neurological changes, FBC and liver function.
- Avoid prolonged exposure to sunlight.

ADRENAL CORTICOSTEROIDS

Adrenocorticotropic hormone (ACTH) (Acthar)
Prednisone (Deltasone, Meticorten, Predsone, Prednisolone)
Methylprednisolone (Depo-Medrol, Solu-Medrol)

Adrenal corticosteroids are used both to sustain a remission and to treat exacerbations of MS. ACTH is usually given to induce a remission; it is administered intravenously for 1 week and may be followed by oral prednisone therapy. Another protocol involves administering ACTH intravenously for 3 days followed by intramuscular injections every 12 hours for 1 week (Hickey, 2013). The drugs are given to suppress the immune system, which is implicated in the aetiology of MS. If the drug is used long term the usual steroid precautions are indicated, such as monitoring for glucose intolerance, osteoporosis and cataract formation. The drugs are used with caution in pregnant and lactating women.

MUSCLE RELAXANTS

Baclofen (Lioresal)
Dantrolene (Dantrium)
Diazepam (Valium)

Muscle relaxants are given to people with MS to relieve muscle spasms. Baclofen and diazepam act by suppressing CNS reflexes that regulate muscle activity; neither drug affects muscle strength. Baclofen therapy should be discontinued over 1 to 2 weeks; sudden withdrawal may cause seizures and paranoid ideation. In contrast to diazepam and baclofen, dantrolene acts directly on skeletal muscles and may affect muscle strength. Dantrolene may cause hepatotoxicity and should not be administered when hepatitis or cirrhosis is present.

Nursing responsibilities

- Evaluate baseline muscle strength and spasticity, range of movement and dexterity.
- Maintain safety or fall precautions; dizziness and drowsiness are common side effects.
- For the person taking dantrolene, monitor liver function tests (enzymes and bilirubin) for signs of hepatotoxicity.

Health education for the person and family

- These drugs may cause sedative effects. Take appropriate safety measures (e.g. avoid driving).
- Avoid CNS depressants (antihistamines, alcohol); they can increase the sedative effects of the medication.
- Continue follow-up care; if you are taking dantrolene, for example, liver function will need to be monitored.
- If you are taking baclofen, do not suddenly stop the medication.
- Increase fibre and fluids in the diet to prevent constipation.
- Change positions slowly to minimise dizziness and other effects of orthostatic hypotension.

IMMUNOSUPPRESSANTS

Azathioprine (Imuran)
Cyclophosphamide (Endoxan)

Immunosuppressants are given to people with MS because of the autoimmune component of the disease. Both medications can cause bone marrow suppression and increase the risk of cancer. Azathioprine may produce hepatitis. Toxic effects of cyclophosphamide include haemorrhagic cystitis, sterility and stomatitis.

Nursing responsibilities

- Monitor baseline parameters: FBC with platelet count and differential, urinalysis, liver function tests, hepatitis profile.
- Assess for anaemia: fatigue, lethargy, pallor.
- Watch for bleeding.
- Protect against and observe for subtle signs of infection.

Health education for the person and family

- Report infection, bleeding and anaemia immediately.
- Drink at least 2 L of fluid a day and observe urine for blood.
- Report jaundice immediately.
- Check oral cavity daily for changes or ulcers.
- Avoid becoming pregnant while taking these drugs.
- Obtain follow-up care, including frequent blood tests.

- Advise to avoid temperature extremes, such as hot showers or exposure to cold. *Maintaining a relatively constant body temperature may avoid exacerbation of the disorder. Heat can delay impulse transmission across demyelinated nerves, which contributes to fatigue.*
- Refer to the appropriate professionals to manage fatigue: stress management groups, support groups, occupational or physical therapist, as indicated. *Support groups and therapy can facilitate self-management and improve coping.*

Health promotion

Following an overview of the disorder, the person needs to understand how to prevent fatigue and exacerbations. Advise the person to avoid stress, extremes of cold and heat, high humidity, physical overexertion and infections. Because pregnancy can exacerbate manifestations, counselling about this risk is indicated. Also, address preventive measures to avoid risk of respiratory and urinary tract infections.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40):

- *Health history:* history of childhood viral illnesses, geographical residence when a child, exposure to physical or emotional stressors (pregnancy/delivery, extremes of heat), medications, symptom onset, severity of manifestations.
- *Physical assessment:* affect, mood, speech, eye movements, gait, tremors, vision and hearing, reflexes, muscle strength and movement, sensation.

Nursing diagnoses and interventions

Interventions for the person with MS vary with the acuity of exacerbations and the presenting problems. Many nursing diagnoses relate to the inability to perform ADLs—for example, *Self-care deficit* and *Impaired home maintenance*. Others reflect problems with musculoskeletal changes or altered nerve conduction—for example, *Impaired physical mobility*, *Ineffective breathing pattern*, *Constipation* and *Functional urinary incontinence*. The nursing diagnoses discussed in this section are *Fatigue* and *Self-care deficit*.

Fatigue

Fatigue is an overwhelming, sustained sense of exhaustion and decreased capacity for physical and mental work at the usual level. Fatigue affects every aspect of the life of a person with ms: the ability to remain independent and perform self-care, sexual function, mobility, airway clearance and, ultimately, self-concept and coping. A great deal of teaching is needed to help the person and their family understand fatigue and how to adapt. People and their families need assistance managing fatigue in a society in which energy is highly valued.

- Assess degree of fatigue and identify contributing factors. *Fatigue is a subjective experience that needs to be evaluated thoroughly before planning can begin.*
- Arrange daily activities to include rest periods. *Rest is essential to manage feelings of fatigue; periods of relaxation may help replenish energy reserves.*

CONSIDERATION FOR PRACTICE

It is important to remember that the fatigue from chronic illnesses such as MS is very different from being 'tired', and that rest and sleep may not result in improvement.

- Ask the person to consider which activities are really necessary and to set priorities. *Prioritising activities promotes independence and self-control.*

Self-care deficit

People with MS may need assistance with bathing, toileting, dressing, grooming and feeding. The help needed can range from minimal guidance to total dependence. The person's ability to perform self-care activities is the gauge by which family members and caregivers need to adjust assistance. Self-care encompasses both the decisions about care and the provision of care; most people are capable of making decisions even after physical limitations prevent physical self-care. The need to maintain self-determination cannot be overemphasised and must be incorporated into each intervention. As the person with MS ages, there may be even more need for teaching to provide self-care.

- Assess the extent of the person's self-care deficit; refer to other health team members for assessment as appropriate. For example, refer to a speech pathologist to assess swallowing and gag reflex, if indicated. *An accurate assessment is crucial to individualising interventions.*
- Suggest adaptive devices, such as arm or wrist braces, as needed. *Meeting hygiene needs and feeding self are essential for positive self-concept, self-esteem and socialisation.*
- Teach to use assistive devices, such as plate guards; to modify consistency of foods; and to eat when energy level is better. If unable to buy and prepare meals, provide referral to Meals on Wheels. *Proper nutrition is basic to health; adapting utensils and foods can facilitate meeting nutritional needs.*
- Teach interventions related to altered bowel and bladder function: fluid intake of at least 2 L daily, bowel routine as indicated to prevent constipation, self-catheterisation skills as necessary. *Maintaining optimal bowel and bladder function decreases the risk of urinary tract infection and bowel impaction.*

Community-based care

The inconsistent and erratic nature of MS can make teaching for self-care difficult. Initial teaching focuses on a realistic explanation of MS. Referral to a support group early in the course of the disease is indicated. Social support can make a positive difference to a person's ability to cope with MS. Address the following topics in preparing the person for home care:

- various treatment options and their side effects
- information about medications, particularly steroid use, and about possible interactions with prescription or over-the-counter (OTC) medications

NURSING CARE PLAN A person with MS

George McMurphy, a 45 year old from Gosford, NSW, was diagnosed with MS approximately 5 years ago. He states that he probably had mild symptoms as long ago as 10 years. He works as a manager for a large grocery store chain near his home. He lives at home with his wife and two children, aged 12 and 15. Recently, Mr McMurphy has had increasing problems with urinary incontinence, lack of energy, weakness, extreme fatigue and altered mobility from spasticity in his leg muscles. He also has a fever, chest congestion and a cough productive of green sputum. He is admitted to the hospital for evaluation and treatment of pneumonia and exacerbation of his MS.

ASSESSMENT

Denise Miller, RN, primary care nurse, is assigned to care for Mr McMurphy. His main complaint is the inability to 'bring up all this sputum; I feel rotten from being so congested. I hate not being able to get to work and for my wife to have to tend to my personal needs'. Vital signs are as follows: BP 134/84, P 94, R 30, T 38.8°C. Mr McMurphy is admitted for an acute exacerbation of the disorder, probably triggered by pneumonia. He will be treated with ACTH and intravenous antibiotics during this admission.

DIAGNOSES

- *Ineffective airway clearance* related to lung infection and thick mucus, evidenced by tachypnoea and a productive cough.
- *Activity intolerance* related to fatigue and spasticity, evidenced by the inability to attend work and attend to own ADLs.
- *Self-care deficit: toileting, feeding and grooming* related to muscle weakness, evidenced by the inability to attend to own ADLs without assistance.

PLANNING

Management of symptoms of pneumonia and implementation of strategies to cope with an exacerbation of MS.

Expected outcomes

- Be able to clear airway.
- Have breath sounds clear to auscultation and pulse oximetry readings above 95%.
- Be able to ambulate using assistive devices, if needed.
- Perform self-care activities without becoming overly fatigued and tired.
- Verbalise methods to adapt daily routine to his level of tolerance.

IMPLEMENTATION

- Initiate pulmonary hygiene measures (e.g. incentive spirometry, turning, deep breathing and coughing, breathing exercises and postural drainage) at least every 2 hours. Assess lung sounds, oxygen saturation and ability to clear airway.
- Teach the importance of maintaining an oral fluid intake of at least 2 L per day to prevent tenacious sputum and urinary tract infections. Teach signs and symptoms of urinary and respiratory infections.
- Encourage participation in decision making about care.
- Assist with ADLs only as needed, based on level of fatigue and muscle weakness.
- Plan self-care activities so that they are performed during periods of peak level of energy; intersperse rest periods throughout the day.
- Refer to an MS support group.
- Refer to physical and occupational therapists for counselling regarding control of spasticity and possible splinting of spastic muscles.
- Consult a urologist for assessment of bladder incontinence; teach intermittent catheterisation. Alternatively, the use of an external condom catheter may be indicated.

EVALUATION

Mr McMurphy is discharged 3 days following admission. He states that he feels stronger; on discharge, he has no problem clearing his airway. Although he continues to pace his activities to avoid fatigue, his muscle strength and 'tiredness' have improved. He is able to complete ADLs unassisted.

Pulmonary function has returned to normal pre-hospitalisation levels: ABGs and pulse oximetry are within normal limits. Both Mr McMurphy and his wife have listed several ways to modify their daily routine to allow more rest and decreased stress. Follow-up visits to Mr McMurphy's primary care provider have been arranged and the McMurphys have been provided with information about the local MS support group.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe approaches the nurse could take to ensure that Mr McMurphy does not exceed his activity tolerance.
2. Develop a teaching plan for Mr McMurphy to help prevent future respiratory infections.

REFLECTION ON THE NURSING PROCESS

1. Which education and communication strategies could you suggest to reduce the risk of injury related to fatigue, muscle weakness and spasticity?
2. Outline what you have learned from this case study that you will apply to your future nursing practice.

- ongoing care from nurses, counsellors and physical, occupational and speech therapists, as well as the doctor and community health nurse
- helpful resources:
  - MS Australia: www.msaustralia.org.au
  - National Institute of Neurological Disorders and Stroke (NINDS): www.ninds.nih.gov
  - Brain Foundation: www.brainfoundation.org.au/disorders/multiple-sclerosis

THE PERSON WITH PARKINSON'S DISEASE

Parkinson's disease (PD) is a progressive, degenerative neurological disease characterised by tremor (shaking), muscle rigidity and bradykinesia (slowness of movement) (Grossman & Porth, 2013). People with PD are faced with multiple problems involving independence in ADLs, emotional wellbeing, financial security and relationships with caregivers.

Incidence and prevalence

PD is one of the most common neurological disorders affecting older adults. It is estimated that there are as many as 70 000 (one in every 340) Australians who currently have PD; of these, 53% are male and 47% are female. Eighty two per cent of people develop PD after the age of 65; however, 18% of people living with PD are of working age (15–64 years). The recent discovery of inherited forms of PD suggests a genetic role in the development of this disease (Deloitte Access Economics, 2015).

Parkinson's-like manifestations, called *secondary Parkinsonism*, may result from other disorders such as trauma, encephalitis, tumours, toxins and drugs. Drug-induced Parkinsonism, which is usually reversible, may occur in people taking neuroleptics, anti-emetics, antihypertensives and illegal designer drugs containing MPTP, a toxic chemical (Grossman & Porth, 2013). Carbon monoxide or cyanide poisoning can also cause secondary Parkinsonism. This discussion focuses on primary Parkinson's disease, the cause of which is unknown.

Pathophysiology

Coordinated, voluntary body movement is achieved through the actions of neurotransmitters in the basal ganglia of the brain. Some neurotransmitters facilitate the transmission of excitatory nerve impulses, while other neurotransmitters inhibit their transmission. Together, this system allows control of movement. A disturbed balance between excitatory and inhibitory neurotransmitters causes disorders of voluntary motor function, such as PD.

In PD, neurons in the cerebral cortex atrophy and are lost, the dopaminergic nigrostriatal (pigmented) pathway degenerates and the number of specific dopamine receptors in the basal ganglia decreases. These pathological processes cause a decrease in the production of dopamine (a neurotransmitter that helps regulate nerve impulses involved in motor function) from the substantia nigra. The usual balance of dopamine (an inhibitory neurotransmitter) and acetylcholine (an excitatory neurotransmitter) in the brain is disrupted and dopamine no longer inhibits acetylcholine. The failure to inhibit acetylcholine is the underlying basis for the manifestations of the disorder. PD has five stages, outlined in Box 43.3.

Manifestations

Parkinson's disease begins with subtle manifestations. People complain of feeling tired and seem to move more slowly; a slight tremor may accompany the fatigue. Over time, the manifestations progressively increase in severity. The manifestations and complications of PD are presented in the box below.

BOX 43.3 Stages of Parkinson's disease

1. Unilateral involvement only, usually with minimal or no functional impairment.
2. Bilateral or midline involvement, without impairment of balance.
3. First sign of impaired righting reflexes, evidenced as unsteadiness as the person turns or demonstrated when the person is pushed from standing equilibrium with the feet together and eyes closed. Functionally, the person is somewhat restricted in activities but may have some employment potential, depending on the type of employment. People are physically capable of leading independent lives and their disability is mild to moderate.
4. Fully developed, severely disabling disease; the person is still able to walk and stand unassisted but is markedly incapacitated.
5. Person is confined to bed or wheelchair unless aided.

MANIFESTATIONS AND COMPLICATIONS

Parkinson's disease

RELATED TO MOTOR DYSFUNCTION

- Non-intentional tremor
- Bradykinesia or akinesia
  a. Slowed movements; inability to initiate voluntary movements
  b. Slowed speech, low amplitude
  c. Poor articulation
  d. Decreased eye movements (i.e. blinking)
  e. Mask-like, expressionless face
- Rigidity
- Posture and gait disturbances
  a. Trunk tilted forwards
  b. Shuffling gait, propulsive at times
  c. Retropulsion
- Complications: falls, fractures, impaired communication, social isolation

RELATED TO AUTONOMIC SYSTEM DYSFUNCTION

- Skin problems
  a. Seborrhoea
  b. Excess sweating on face and neck, absence of sweating of trunk and extremities
  c. Mottled skin
- Heat intolerance
- Postural hypotension
- Constipation
- Complications: skin breakdown, dizziness, falls, constipation

RELATED TO COGNITIVE AND PSYCHOLOGICAL DYSFUNCTION

- Dementia
  a. Memory loss
  b. Lack of insight and problem-solving ability
  c. Declining intellectual abilities
- Anxiety
- Depression
- Complications: loss of ability to function, social isolation

Tremor

Tremor at rest is usually the first manifestation experienced, with one of the upper extremities more often affected. Resting tremors of the hand show a 'pill-rolling' motion of the thumb and fingers. (This name reflects the way in which medicinal pills were formed in the early days of medicine.) The tremor may be controlled with purposeful, voluntary movement and is worsened by stress and anxiety. People have progressive impairment in performing skills that require dexterity and fine muscle control, such as writing and eating.

Rigidity and bradykinesia

Manifestations related to motor and postural effects include rigidity, bradykinesia and uncoordinated movements. Rigidity (resulting from involuntary contraction of all skeletal muscles) makes both active and passive movement difficult. It is manifested as increased resistance to passive range of motion. Although the extremity moves, it does so in a jerky motion, called *cogwheel rigidity*. The first manifestation of rigidity may be muscle cramps in the toes or hands, but most often the person describes stiffness, heaviness or aching in muscles.

Bradykinesia, experienced as difficulty in starting, continuing or coordinating movements, is the most common and crippling manifestation. All striated muscles are affected, including those that involve chewing, swallowing and speaking. Slowed or delayed movements affect the eyes, mouth and voice, causing a mask-like face and softened or muffled voice. Disorders of swallowing result in problems with eating and with drooling. People have a staring gaze with minimal change in expression (see Figure 43.3). People describe being 'frozen' in place as voluntary movement is lost and they sit or lie in one position without movement for long periods of time. Movement is interspersed with freezing, brought about by turning, increasing the effort to move or making visual or touch contacts.

FIGURE 43.3 ■ In Parkinson's disease, the person's face lacks expression or animation

Source: Scott Houston/Corbis.

Abnormal posture

The loss of normal postural reflexes results in postural abnormalities, including disorders of postural fixation, equilibrium and righting. Involuntary flexion of the head and shoulders means the person with PD cannot maintain an upright position of the trunk when sitting or standing. This problem of postural fixation results in the characteristic stooped, forwards-leaning position. Disorders of equilibrium follow loss of postural fixation, with an inability to make adjustments when leaning or falling, increasing the risk of injury from falls. (The person usually falls backwards.) The person takes short, accelerated steps to try to maintain an upright position when walking.

Autonomic and neuroendocrine effects

Many manifestations result from the loss of functions controlled by the autonomic nervous system. Elimination problems include constipation and urinary hesitation or frequency. People may experience problems related to orthostatic hypotension, including dizziness with position change. Eczematous skin changes and seborrhoea are related to the increase in sweat gland activity secondary to increased sebotrophic hormone production.

Mood and cognition

Both depression and dementia are pathologies associated with PD. Depression occurs in half of all people, and one-third have dementia. Dementia, resulting from loss of cholinergic cells, loss of neurons, senile plaques, neurofibrillary tangles and amyloid changes in small blood vessels, occurs in 20% of people with PD and develops later in the disease (Grossman & Porth, 2013). The person has manifestations similar to the person with Alzheimer's disease, including confusion, disorientation, memory loss, distractibility and changes in abstraction and judgment. *Bradyphrenia* may also occur, resulting in slow thinking and a decreased ability to form thoughts, to plan or to make decisions.

Sleep disturbances

People with PD commonly have sleep disturbances, although they may experience decreased manifestations during sleep in the early stages. The ability to fall and stay asleep is affected by acetylcholine. Muscle rigidity may compromise sleep because of the inability to change position. This lack of muscle movement causes the person to awaken and consciously shift position.

Interrelated effects

Some of the manifestations that people with PD experience have multiple contributing factors. For example, constipation is common because of decreased peristalsis. However, decreased peristalsis is not the only cause: immobility, tremors (resulting in being unable to drink from a glass easily) and dietary changes from dysphagia all contribute to the problem of constipation.

Complications

The following complications are associated with PD:

- oculogyric crisis, in which the eyes become fixed with a lateral and upwards gaze
- paranoia and hallucinations, which may accompany dementia
- impaired communication due to changes in speech, handwriting and expressiveness
- falls from balance, posture and motor changes
- infections, such as pneumonia, related to immobility
- malnutrition related to dysphagia and inability to prepare meals
- altered sleep patterns due to loss of dopamine, levodopa side effects (nightmares, dreams) or side effects of anticholinergics (hyperreflexia, muscle twitching) and depression
- skin breakdown and pressure ulcers associated with urinary incontinence, malnutrition and sweat reflex changes
- depression and social isolation.

INTERPROFESSIONAL CARE

Prognosis is poor, owing to the progressive degeneration that ultimately affects multiple physiological systems and their function. Psychosocial effects are equally devastating and the family needs more support as the person's debilitation increases. Total disability is usually seen 10 to 20 years after diagnosis. The leading cause of death is pneumonia.

Diagnosis is based primarily on a thorough history and physical examination and is made based on having two of the following manifestations: tremor at rest, bradykinesia, rigidity and postural instability. Interventions vary with the clinical stage of the disorder and include medication, surgery and rehabilitation to retain the optimal level of functioning possible. An interprofessional approach is essential for people with PD.

Diagnosis

Diagnostic studies may support a potential diagnosis of PD; no test clearly differentiates PD from other neurological disorders (Hickey, 2013). However, a PET scan will show decreased uptake of 6-[18F]-fluorodopa.

Medications

The goal of drug therapy is to control manifestations to the extent possible. Generally, medications vary with the stage of the disease; however, response is individualised and guides the selection of medications. Types of drugs used include monoamine oxidase (MAO) inhibitors, dopaminergics, dopamine agonists and anticholinergics. Information about these drugs is presented in the 'Medication administration' box below.

Initially people are treated with selegiline (Carbex, Eldepryl), amantadine (Symmetrel) or anticholinergics. As the disease progresses, levodopa (Dopar, Larodopa) in combination with carbidopa (Lodosyn) is used in a medication named carbidopa–levodopa (Sinemet). Because levodopa eventually loses its effectiveness, dopamine agonists are added to increase its effectiveness. Eventually, pharmacotherapeutic agents lose their efficacy and the disease continues to progress despite treatment. Response to the drugs fluctuates; this phenomenon is called the 'on–off' response.

Bromocriptine (Parlodel) and pergolide (Permax), agents that inhibit the breakdown of dopamine, are used to delay progression of the disease. Catecholamine-O-methyl transferase (COMT) inhibitors (tolcapone (Tasmar) and entacapone (Comtan)) are used in conjunction with carbidopa–levodopa therapy to reduce the metabolism of levodopa, leading to more sustained dopaminergic stimulation of the brain. Selegiline (Eldepryl) increases dopaminergic activity and is used as an adjunctive therapy for people who have fluctuations in response, or become unresponsive, to levodopa.

Other medications may be used to treat problems related to PD. Antidepressants may be prescribed. Propranolol (Inderal) may be used to treat tremors; it should be used cautiously when people have orthostatic hypotension. Botulism toxin injections may be given to treat eyelid spasms and abnormal posturing (dystonia) involving the extremities.

Deep brain stimulation

Activa® tremor control therapy uses an implanted pacemaker-like device to deliver mild electrical stimulation to block the brain impulses that cause tremor, rigidity, stiffness, slowed movement and problems with walking. In this procedure, an insulated wire is surgically placed in the thalamus and connected to an implanted pulse generator (similar to an advanced cardiac pacemaker) near the clavicle. It is used only for people who cannot adequately control manifestations with medications (NINDS, 2015c).

Surgery

Pallidotomy is a surgical technique for Parkinson's disease and its results have been helpful for many people. In this procedure, the neurosurgeon locates the affected areas of the globus pallidus and destroys the involved tissue. As a result, people who could not previously ambulate are able to walk and tremors cease. The long-term effects are still being evaluated.

Stereotaxic thalamotomy (an x-ray is taken during neurosurgery to guide the insertion of a needle into a specific area of the brain) has been used only for people who do not respond to medications—generally, younger people with extreme unilateral tremor. The surgeon destroys a small amount of tissue by creating a lesion in the ventrolateral nucleus of the thalamus. This surgery decreases tremors and rigidity in the contralateral extremity.

Foetal tissue transplantation is a controversial surgical procedure limited to a few medical centres. In this procedure, brain cells from aborted foetuses are implanted into the brain in the hope that the new cells will grow and produce enough dopamine to restore some lost mobility.

Rehabilitation

Depending on their individual needs, people frequently benefit from rehabilitation therapy with a physiotherapist, social worker, psychologist and/or speech therapist.

Physiotherapists (PTs) can implement an individual exercise program to improve coordination, balance, gait and transfers.

MEDICATION ADMINISTRATION The person with Parkinson's disease

DOPAMINERGICS

Levodopa (Larodopa, Dopar)
Carbidopa–levodopa (Sinemet)
Amantadine (Symmetrel)

These drugs have their main effect on the akinesia of DP, improving mobility while decreasing muscle rigidity and tremor. Levodopa is a metabolic precursor of dopamine, but unlike dopamine it can cross the blood–brain barrier. Levodopa is converted to dopamine in the brain by decarboxylase, a catalytic enzyme, and stimulates dopamine receptors to balance the dopamine/acetylcholine concentrations. Carbidopa prevents decarboxylase from converting levodopa to dopamine in the peripheral tissues; therefore, carbidopa is frequently given in combination with levodopa. Amantadine is used to treat dyskinesia and also elevates mood.

Levodopa is avoided in people with narrow-angle glaucoma, severe angina pectoris, transient ischaemic attacks or melanoma. The 'on–off' phenomenon occurs after the person takes levodopa for several years; this phenomenon is characterised by unexpected dyskinesias and lack of symptom control.

Common side effects are nausea and vomiting; darkening of urine and sweat; dyskinesias, especially in the first few months of therapy; arrhythmias; orthostatic hypotension; and psychological reactions, such as hallucinations and vivid dreams. Older adults are particularly susceptible to psychological disturbances.

Nursing responsibilities

- Establish the person's baseline functional abilities in performing ADLs and administering the medication; assess motor control and coordination.
- To avoid adverse reactions, assess the person's overall health status before initiating therapy.
- Monitor medications known to cause adverse drug interactions: anticholinergics, pyridoxine and antipsychotic agents alter the effectiveness of levodopa; MAO-B inhibitors can cause severe hypertension because of their vasoconstrictive effects.
- Withhold levodopa for 8 hours prior to administering carbidopa–levodopa to avoid potentiating the effects of the circulating levodopa.

Health education for the person and family

- Levodopa may not take effect for several weeks to months.
- Do not alter dosages of medications; taking more of a medication may not result in better symptom control and can cause severe side effects.
- Your protein intake should be divided into equal amounts for the day's meals. Avoid foods high in pyridoxine, such as pork, beef, ham, avocado, beans and oatmeal.
- Levodopa may cause a darker colour of urine; however, this is harmless.
- To prevent side effects:
  - Prevent nausea by taking medication with food.
  - Change position slowly to avoid a drop in blood pressure and risk of falling.
  - Prevent constipation by increasing fluid intake and exercising regularly.
- Notify the healthcare practitioner if you begin to have difficulty making voluntary movements, or cardiac or psychological symptoms develop.
- Watch for the 'on–off' phenomenon, in which periods of symptom control alternate with periods when the drug fails to control symptoms.

MONOAMINE OXIDASE INHIBITORS

Selegiline (Eldepryl, Carbex)

Selegiline works by selectively inhibiting the enzyme that inactivates dopamine in the brain. It may be administered alone or as an adjunct therapy with levodopa: selegiline inhibits the enzyme system that would otherwise break down and destroy dopamine. This synergistic effect lasts approximately 1 to 2 years. The combination of selegiline and levodopa increases the adverse reactions of dopamine; nurses must be alert for orthostatic hypotension, changes in movement, hallucinations and confusion. These responses can be modified by lowering the dose of levodopa. Because it is highly selective for the MAO-A enzyme, selegiline does not have antidepressant effects like the MAO-B inhibitors. The risk of severe hypertension is low.

Nursing responsibilities

- Establish baseline functional abilities: motor control and movements, position changes, mental status.
- Monitor problems with insomnia.
- Assess for orthostatic hypotension; look for unsteadiness with position change and complaints of dizziness.
- Assess for hypertension, which can occur with higher than usual doses.

Health education for the person and family

- It is very important to take the medication as directed, especially the dose and time of administration.
- Notify the healthcare practitioner if insomnia occurs.
- Report signs of dizziness when changing positions or standing, changes in ability to move or psychological changes.
- Change positions slowly, especially when moving from a sitting to a standing position.
- Keep follow-up appointments for evaluation of the medication's effectiveness.

DOPAMINE AGONISTS

Bromocriptine (Parlodel)
Pergolide (Permax)
Pramipexole (Sifrol)
Ropinirole (Repreve)

Dopamine agonists act by directly activating dopamine receptors in the brain. They are frequently used in combination with levodopa therapy: when dopamine agonists are given with levodopa, they increase

MEDICATION ADMINISTRATION The person with Parkinson's disease (continued)

the therapeutic effects of levodopa and reduce fluctuations in motor symptoms. Adverse reactions are similar to those of levodopa: nausea, orthostatic hypotension and psychological disturbances are common. Nursing responsibilities, and personal and family teaching information, are similar to those for the dopaminergics.

COMT INHIBITORS

Tolcapone (Tasmar)
Entacapone (Comtan)

COMT inhibitors inhibit catechol-O-methyltransferase (COMT), which is responsible for metabolising dopamine. The concurrent administration of a COMT inhibitor with levodopa increases the amount of levodopa available to the brain to control Parkinson's disease.

Nursing responsibilities

- Monitor liver function test results and manifestations of liver impairment (dark urine, jaundice).
- Administer with food.
- If given concurrently with warfarin, monitor APTT and INR.

Health education for the person and family

- Avoid using alcohol and sedatives.
- Rise slowly from a sitting or lying position to avoid falling.
- Nausea is common at the beginning of therapy.
- Do not abruptly stop taking the medication.
- Report increased loss of muscle control, yellow skin or eyes, dark urine, hallucinations, severe diarrhoea.

ANTICHOLINERGICS

Benzhexol (Artane)
Benztropine (Cogentin)
Biperiden (Akineton)
Cycrimine (Pagitane)
Procyclidine (Kemadrin)
Chlorphenoxamine (Phenoxene)

Anticholinergics are effective in PD because they block the excitatory action of the neurotransmitter acetylcholine. They are frequently used during the early stages of the disease or when the person can no longer take levodopa. They may be given in combination with carbidopa–levodopa therapy. These medications ease drooling, tremors and rigidity; however, side effects are common and may include blurred vision, dry mouth, constipation, delayed gastric emptying, urinary retention, photophobia and tachycardia. Older adults are especially susceptible to heat stroke and psychological side effects, including confusion, depression, delusions and hallucinations. Anticholinergics should be tapered slowly when discontinued to avoid enhancing Parkinsonian symptoms.

Nursing responsibilities

- Perform baseline assessment for presence of glaucoma, cardiac dysfunction and prostatic hypertrophy.
- Note other medications, including OTC medications that have anticholinergic effects, such as antihistamines and tricyclic antidepressants.
- Monitor for side effects, especially changes in vision, elimination, gastric emptying and mentation.

Health education for the person and family

- Inform your healthcare practitioner if you begin taking any new medications or notice any new symptoms.
- Avoid overexposure to heat and take precautions to avoid heat stroke: drink fluids, keep cool and avoid strenuous activity on hot days.
- Drink adequate amounts of fluid to minimise constipation.
- Practise home safety to prevent falls associated with blurred vision.
- Avoid taking OTC antihistamines or sleeping aids; these have anticholinergic activity.
- Have the eyes examined annually to check for glaucoma; wear dark glasses if photophobia develops.
- Do not suddenly stop taking anticholinergics.

Preventing contractures is an important goal of exercise therapy. It is crucial that family and healthcare personnel permit the person adequate time to perform not only exercise regimens but also ADLs. Activities should not be rushed.

An occupational therapist (OT) helps the person adapt to changing abilities pertinent to work, self-care and recreational activities. Some rehabilitation centres assign OT personnel the responsibility of addressing the person's upper extremity functions while assigning PT personnel to manage lower extremity problems. For example, skills related to cooking and grooming would be supervised by the OT, whereas mobility and posture skills would be supervised by the PT.

Speech therapists frequently address not only the person's speech but also chewing and swallowing. These therapists evaluate people and plan treatment regimens. The challenge with people who have PD is that they have not only vocalisation problems but also dexterity deficits; speech therapists therefore must evaluate the potential benefits of assistive devices, such as a magic slate, voice synthesiser or computer, for each person.

Nursing care

The chronic and eventually debilitating nature of PD poses many challenges to people, families and healthcare professionals. Dependence due to declining physical and mental abilities is of major concern. In the early stages, most people are able to

remain at home, with the family assisting with or providing many of the person's ADL needs. As the disease progresses and the burden of care increases, the person and their family may prefer placement in a long-term care facility. A nursing care plan for the person with PD is given below.

Health promotion

Teaching preventive measures is extremely important when caring for people who have PD. Preventing malnutrition, falls and other environmental accidents, constipation, skin breakdown from incontinence or immobility, and joint contracture requires teaching and reinforcement.

In addition to incorporating information about safety needs, teach ways to prevent orthostatic hypotension when the person changes positions; some people may also benefit from wearing individually fitted compression hose. In addition, address safety considerations about proper administration of medications.

Assessment

Collect the following data through the health history and physical examination (see Chapter 40). Further focused assessments are described with nursing interventions. When assessing the older person, be aware of normal changes with ageing, outlined in Chapter 40.

NURSING CARE PLAN A person with PD

Walter Avneil, aged 78, was diagnosed with PD at age 64. His wife died 5 years ago and he has no other family living. Mr Avneil worked for more than 40 years as a mechanic in a large factory. He now lives in a residential aged care facility. During his last clinic visit for a review of his medications, the following assessment was made.

ASSESSMENT

Caucasian male with history of PD for the past 14 years. Skin oily and damp. Tremors in both hands and the lips. Gait is slow and shuffling, with a forwards-leaning posture. Speech slow and slurred. Face expressionless. Has lost 5 kg since last visit 3 months ago. Has been on levodopa with carbidopa since diagnosis. States his main problems are 'eating problems, bowel problems, walking problems'.

DIAGNOSIS

- *Constipation* related to lack of exercise, decreased food intake and effects of medications manifested by difficulty opening bowels.
- *Impaired verbal communication* related to lip tremors, slow/slurred speech and facial muscle involvement of PD, evidenced by difficulty communicating needs to others.
- *Poor nutrition* related to difficulty swallowing and chewing, evidenced by weight loss.
- *Impaired physical mobility* related to rigidity and bradykinesia, evidenced by slow, shuffling gait.

PLANNING

- Provide interventions to manage complications of PD.

Expected outcomes

- Have a soft stool at least every other day.
- Practise exercises provided by speech therapist twice a day.
- Increase number of kilojoules, fluids and fibre in diet provided at long-term facility.
- Improve joint mobility and ability to ambulate.

IMPLEMENTATION

- Discuss problems with bowel elimination with staff at long-term facility; suggest increasing fluids to 3 L per day and also increasing fibre in the diet, with oatmeal for breakfast and more fruit and vegetables at meals.
- Encourage exercises provided by speech therapist to improve speech and swallowing. If these are not effective, make a referral for another evaluation.
- Discuss diet plan with dietitian at the long-term care facility, including consistency of foods and number of kilojoules. Suggest dietitian be part of the swallowing evaluation by the speech therapist.
- Refer for physiotherapy and occupational therapy for a program to improve gait and joint mobility and to decrease risk of falling.

EVALUATION

In a return visit 3 months later, Mr Avneil reports that 'my bowels are working better'. He has gained 3.2 kg and the staff report that this is related to multiple factors, including practising his swallowing exercises, getting more exercise which stimulated his appetite and changing his diet to six small meals a day of soft or puréed foods. The staff are offering him liquids at meals and snack times and he usually drinks all they give him. His speech is not much improved. His posture and gait are somewhat better and he is doing the exercises provided by the physiotherapist and occupational therapist. Mr Avneil's functional abilities have improved so much that the staff are considering training sessions specific to care of residents with PD.

CRITICAL THINKING IN THE NURSING PROCESS

1. Although Mr Avneil did not mention it, the staff report that he is frustrated by not being able to dress himself. Which suggestions could you make to facilitate his independence?
2. Mr Avneil spends most of his time alone, although he enjoys the company of the other residents. List assessments and interventions you might provide to increase his diversional activity.
3. The loss of his wife and the debilitating effects of his disease increase Mr Avneil's risk of the nursing diagnosis of *Chronic sorrow*. What might you suggest the long-term staff do to reduce this risk?

REFLECTION ON THE NURSING PROCESS

1. Which communication and education strategies could you use to assist people with PD who are experiencing speech difficulties?
2. Outline what you have learned from this case study that you will apply to your future nursing practice.

- *Health history:* brain trauma, stroke, infection, exposure to heavy metals or carbon monoxide, medication and drug use, incontinence, constipation, weight loss, sweating, sleep problems, muscle pain, mood.
- *Physical assessment:* affect; appearance; speech, scalp, eyelashes and skin; drooling; tremor; coordination; posture; gait; muscle rigidity; mental status.

Nursing diagnoses and interventions

People with PD have complex and, ultimately, multisystem needs. Deficits in mobility and self-care are common. Psychosocial needs may include problems related to *Ineffective coping, Powerlessness* and *Disturbed body image*. Refer to the nursing care sections throughout this chapter for discussions of fatigue, self-care deficit, ineffective airway clearance and other pertinent diagnoses. This section focuses on the nursing diagnoses related to *Impaired physical mobility*, *Impaired verbal communication*, *Imbalanced nutrition: less than body requirements* and *Disturbed sleep pattern*.

Impaired physical mobility

People with PD have impaired mobility for several reasons, including tremors, gait-pattern disturbances and alterations in body positioning, such as forwards bending of the trunk. Poor self-esteem may contribute to the person's lack of motivation and willingness to be mobile.

- Suggest referral to a physiotherapist to develop an individualised exercise program. *A program specific to the person supplies motivation as well as helping the person maintain muscle tone, flexibility and mobility.*
- Request the physiotherapist teach caregivers how to do ROM exercises at least twice a day, emphasising the trunk, neck, arms, hips and legs. *Maintaining joint mobility promotes better function and strength, improving gait pattern. Consistent ROM exercises can prevent contractures.*
- Ask caregivers to ambulate the person at least four times a day if possible. *Exercise fosters independence and self-esteem.*
- Recommend assistive devices, such as lift chairs, canes, splints or braces, as indicated. *Adaptive equipment improves balance, protects joints and promotes proper anatomical positioning.*
- To promote mobility and safety:
  - Slightly elevate the back legs of chairs and raise the toilet seat to help rise from a sitting position to a standing position.
  - Wear shoes with Velcro closures.
  - Remove potential hazards, such as unanchored throw rugs.
  - Install handrails and non-skid surfaces in bathtubs and showers.
  - Ensure adequate lighting throughout the home and in outside areas, especially in areas where transfers are common.

    Safety measures prevent potential complications that may result from falls or other accidents and promote self-esteem through self-care.

CONSIDERATION FOR PRACTICE

Parkinson's disease is a disorder common in older adults, who are at greater risk of falls resulting from orthostatic hypotension, osteoporosis, poor vision and problems causing disorientation and confusion, such as Alzheimer's disease.

Impaired verbal communication

Diminished vocal amplitude and loss of muscular control can impair the person's ability to speak. Both caregivers and family members must remember to give people enough time for self-expression; an unhurried approach is recommended. Seek input from family members when determining alternative methods of communicating with the person.

- Assess current communication abilities in speech, hearing and writing. *Communication involves both sending and receiving messages.*
- Develop methods of communication appropriate to coordination abilities, such as a magic slate, flash cards with common phrases and pointing to objects. *Individualising a method of communication decreases anxiety and isolation.*
- Suggest referral to a speech pathologist to develop oral exercises and interventions that will facilitate speaking. *The muscles of speech and swallowing are affected by the PD process.*
- Remind the person to speak more loudly, if possible. *A low, monotonous voice is characteristic of the person with PD.*

Imbalanced nutrition: less than body requirements

Tremors, altered gait, and impaired chewing and swallowing can cause nutritional problems in the person with PD. As the disorder progresses, interventions for ensuring optimal nutrition need to be adapted to the person's functional abilities. Assess the person's swallow reflex before starting any feeding program. During the initial stages of the disorder, some people may have the nursing diagnosis of *Poor nutrition related to excess intake of kilojoules manifested by kilojoule intake exceeding energy expenditure.*

- Assess nutritional status and self-feeding abilities; suggest referral to an occupational or speech therapist, if needed. *An initial assessment of abilities ensures that interventions are personalised to the person's current functional abilities.*
- Teach caregivers how to prepare foods of proper consistency as determined by swallowing function. *The person may aspirate food that is too liquid.*
- Weigh weekly. *Early recognition of weight loss allows for intervention.*
- Teach eating methods to decrease tremors, such as holding a piece of bread in the hand that is not holding an eating utensil. *Non-intentional tremor may be reduced through purposeful activity.*
- Encourage diet that is high in bulk and fluids. *Several anti-Parkinson's medications and inactivity can cause constipation.*

Disturbed sleep pattern

Rigidity and weakness can cause people with PD to lose the ability to move and change positions during sleep. The resulting discomfort causes periods of wakefulness. Medications to treat PD contribute to sleep pattern disturbance; for example, levodopa can cause vivid dreams. Nurses can help in accurately assessing the sleep pattern disturbance and in planning interventions to improve or increase sleep time.

- Assess sleep pattern and existing conditions that may affect sleep, such as depression or pain. *People experiencing anxiety, depression and dementia have a difficult time falling asleep and may wake up more at night.*

CONSIDERATION FOR PRACTICE

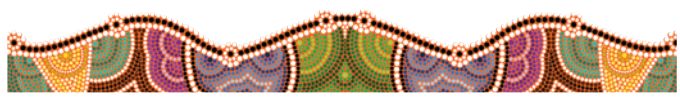
Remember to assess pain status; lack of adequate pain control may interfere with sleep.

- Explain the disease process and the effects of decreased dopamine on the sleep–wake cycle. *Depending on the dosage, levodopa causes less REM sleep and deep sleep.*
- Review the person's medication. Bromocriptine and levodopa, especially if used with an anticholinergic, can cause vivid dreams. *Other medications (diuretics, theophylline, hypnotics) also may interfere with sleep.*
- Teach how to modify lifestyle activities that affect sleep:
  - Institute a routine of activities with limited rest periods during the day; avoid napping close to bedtime. Avoid strenuous exercise in the evening. *Daytime sleeping may contribute to decreased night-time sleeping. Vigorous exercise just before bedtime may act as a stimulant.*
  - Incorporate diet modifications, such as limiting caffeine and alcohol intake. *Caffeine is a stimulant and alcohol may cause early morning awakenings, increased daytime sleepiness and nightmares.*
  - Drink a glass of milk before bedtime. *Milk contains l-tryptophan, which produces sedative effects by shortening the time taken to fall asleep (sleep latency).*
  - Adapt the environment to aid sleep (e.g. darken the room and decrease noises). *Reducing environmental stimuli decreases external sleep disturbances.*

Community-based care

It is important for both the person and their family to maintain independence and self-care as long as possible. To maintain function and quality of life, the following topics should be addressed:

- realistic expectations
- equipment suppliers
- home environment conducive to using equipment
- referrals to speech therapist, occupational therapist, physiotherapist and dietitian
- gait training and exercises for improving ambulation, speech, swallowing and self-care
- increased fluid intake of 3 L/day and increased fibre in every meal
- stool softeners or laxatives as needed for bowel elimination
- swallowing during eating and taking medications. Have suction equipment available and know first aid treatment for choking:
  - Encourage the person to relax and breathe deeply.
    - Ask the person to cough.
    - If unsuccessful, bend the person well forwards and give 5 sharp blows between shoulder blades.
    - If still unsuccessful, place the person on their left side on the floor and call 000 for an ambulance.
  - Total blockage:
    - Lie the person on their left side on the floor.
    - Give 5 sharp blows between shoulder blades.
    - If unsuccessful, give 5 quick downward lateral chest thrusts (place one hand in the middle of the person's back for support and heel of other hand in the CPR compression position and give 5 chest thrusts, slower but sharper than compressions).
    - If still unsuccessful, call 000 for an ambulance and continue alternating 5 back blows with 5 chest thrusts until medical aid arrives (St John Ambulance Australia, 2014).
- foods that can be easily swallowed (such as puréed or soft foods) and feed six small meals a day if possible
- helpful resources:
  - Parkinson's Australia: www.parkinsons.org.au
  - Brain Foundation: www.brainfoundation.org.au
  - Michael J. Fox Foundation: www.michaeljfox.org
  - The National Institute of Neurological Disorders and Stroke: www.ninds.nih.gov
  - Parkinson's Alliance: www.parkinsonalliance.org.



THE PERSON WITH HUNTINGTON'S DISEASE

Huntington's disease (HD) is a progressive, degenerative, inherited neurological disease characterised by increasing dementia and chorea (jerky, rapid, involuntary movements). It is a single-gene autosomal dominant disease that causes localised death of neurons of the basal ganglia (Grossman & Porth, 2013). The exact cause is unknown, but postmortem studies have demonstrated a decrease in gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter in the basal ganglia. There is also a decrease in acetylcholine levels, suggesting that the manifestations are the result of an imbalance in dopamine and acetylcholine. HD is a familial disease; each child of an HD parent has a 50% chance of inheriting the HD gene and, if they do, will eventually develop the disease (NINDS, 2015f). There is no cure for the disease. Huntington's disease causes progressive chorea, speech problems and dementia.

Because the person is usually asymptomatic until age 30 to 40, they may already have passed the gene to the next generation. The psychological effect is devastating to people and their families. The family not only experiences guilt from passing the disease from one generation to the next, but also is faced with the overwhelming long-term care needs of those affected. It is common for several family members to have the disease.

Pathophysiology

HD causes destruction of cells in the caudate nucleus and putamen areas of the basal ganglia. Other areas of the brain, such as the frontal lobes, may selectively atrophy. Several neurotransmitters and their receptors are decreased, including GABA and acetylcholine. The neurotransmitter dopamine is not affected in HD, but the decrease in acetylcholine results in a relative excess of dopamine in the basal ganglia. Whereas in PD a deficit of dopamine causes slow movement or lack of movement, in HD the opposite occurs: there is a relative excess of dopamine, causing excessive, uncontrolled movement.

Manifestations

Manifestations and complications primarily involve abnormal movement and progressive dementia (see the box below). The progression and sequence of manifestations varies somewhat; however, initially the psychological manifestations are more debilitating than the choreiform (rapid and jerky) movements.

Early signs of personality change include severe depression, memory loss with decreased ability to concentrate, emotional lability and impulsiveness. The person experiences frequent mood swings ranging from uncontrollable periods of anger to apathy. Eventually, signs of dementia, including disorientation, confusion and lack of sense of time, become evident and interfere with self-care.

Motor manifestations usually parallel personality and mood changes. The motor manifestations worsen with environmental stimuli and emotional stress but are absent when the person is sleeping. Initially, movement problems are described as 'fidgeting' or restlessness, followed by progressive worsening of abnormal movements. The choreiform movements, which begin in the face and arms and then involve the entire body, are manifested by facial grimaces, tongue protrusion, jerky movement of the distal arms or legs, and a rhythmic, lurching gait that almost resembles a dance. (The term *chorea* comes from *choreia*, the Greek word meaning 'dance'.) Gait changes cause uncoordinated movements and contribute to frequent falls.

The muscles of swallowing, chewing and speaking are affected, leading to dysphagia and dysarthria and associated problems with communication and nutrition. The person's constant movement and difficulty in swallowing contribute to weight loss and eventual cachexia. Breathing is impaired because the diaphragm is unable to move effectively.

The manifestations slowly progress over approximately 15 to 20 years after initial manifestations appear. Prognosis is poor, with inevitable debilitation and total dependence. Death usually results from aspiration pneumonia or another infectious process.

INTERPROFESSIONAL CARE

There is no cure for HD and treatment addresses the disease's manifestations. Nurses provide care to people with HD in a variety of community settings. Initially, people and families can manage care needs at home but, as the disease progresses, the person requires constant supervision, such as that provided in day respite facilities. Eventually, skilled long-term care is needed. People who develop acute problems may be hospitalised until the crisis is managed. Because of the inevitable total multisystem debilitation of people with Huntington's disease, nurses and other caregivers face many challenges.

MANIFESTATIONS AND COMPLICATIONS Huntington's disease

MOTOR EFFECTS

Early

- Restlessness
- 'Fidgety' feeling
- Minor gait changes—unsteady on feet
- Posture and positioning disturbances, frequent falls
- Inability to keep the tongue from protruding
- Slurred speech with poor articulation
- Complications: increasing problem with self-care activities, such as bathing, grooming, eating

Late

- Chorea—severely altered gait with irregular, uncontrollable movement; shoulders shrug arrhythmically
- Facial grimacing—raising of eyebrows, uncontrollable protrusion of the tongue
- Dysphagia
- Unintelligible speech
- Impaired diaphragmatic movement
- Complications: immobility, aspiration, choking and, eventually, total dependence, poor oxygenation, emaciation and cachexia

PSYCHOSOCIAL EFFECTS

Early

- Irritability
- Outbursts of rage alternating with euphoria
- Depression
- Complication: suicide

Late

- Decreasing memory
- Loss of cognitive skills
- Eventual dementia
- Complication: total dependence

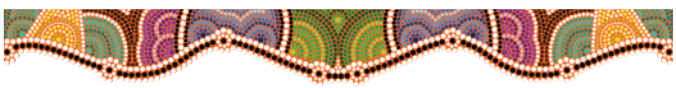
Diagnosis

Genetic testing is the only test available to diagnose people suspected of having HD. Both blood and amniotic fluid may be tested for the presence of a gene mutation on chromosome 4 using DNA analysis. The test can predict with 95% accuracy who is a carrier of the disease.

Medications

The following medications are given for the manifestations of HD:

- Antipsychotics, specifically phenothiazines and butyrophenones, are effective in HD because they block dopamine receptors in the brain. The therapeutic goal is to restore the balance between the neurotransmitters.
- Antidepressants are prescribed in the early stage of the disease; however, medications are no substitute for intense follow-up counselling for people and their families.



Nursing care

Nurses are faced with a multitude of challenges when caring for families who have Huntington's disease, including physiological, psychosocial and ethical problems. Physiological problems are related to the progressive and eventually debilitating nature of the disease. Psychosocial concerns occur as a result of the person's personality and mental changes, the family's responsibility for providing care and the guilt implicit in a genetically transmitted disease. Ethical difficulties relate to the genetic nature of the disease: DNA testing for the marker on chromosome 4 can determine whether the person is a carrier of the disease before they begin to exhibit manifestations. Children of people with HD are thus faced with the choice of finding out whether they will eventually be affected. If they choose not to be tested, they may pass the disease on to yet another generation; and if a foetus is affected they may face the decision of whether to undergo an abortion.

Nursing diagnoses and interventions

Initially, much of the nursing care focuses on teaching about the disease, psychological support and genetic counselling. As manifestations become more severe, nursing considerations centre on problems related not only to immobility and altered nutrition, but also to the increasing self-care deficits. Families and people experiencing HD face many psychosocial issues. Nurses must be prepared to listen actively as well as to provide comfort and encouragement throughout the lengthy illness. There are many possible nursing diagnoses for the person with HD; this section focuses on nursing diagnoses related to aspiration, nutrition, skin integrity and communication.

Risk of aspiration

Uncoordinated movements and swallowing and chewing problems put the person at high risk of aspiration.

- Maintain in an upright position while the person eats; support the head. *Proper positioning may prevent aspiration during meals.*
- Teach the first aid treatment for choking to caregivers and family members (see above). *Aspiration is a real possibility; caregivers must be prepared to re-establish the person's airway.*
- Provide food that is thick enough to manage, such as thick soups, mashed potatoes, stews or casseroles. *These foods are more readily tolerated and manipulated by the tongue than liquids.*
- Make sure food is swallowed before giving another spoonful of food. *The automatic phase of swallowing may be disrupted in the person with HD; providing adequate time and smaller bites may improve the ability to manipulate foods.*
- Provide a calm, relaxing eating environment. *Stress worsens choreiform movements and inappropriate behaviours.*

Poor nutrition

People with HD have unpredictable choreiform movements of the extremities and decreased ability to control muscles involved with chewing and swallowing. Families and caregivers are challenged to provide sufficient kilojoules to maintain the person in positive nitrogen balance.

- Evaluate current weight and nutritional status, including serum prealbumin and transferrin levels. *Establishing a baseline is crucial for meeting individual kilojoule, protein, vitamin and mineral needs.*
- Assess ability to swallow and manipulate eating utensils. *Aspiration is an ever-present danger that must be avoided; utensils may need to be adapted to the person's abilities, if the person is able to assist at all.*
- Continue feeding even if the person physically turns away from the meal. *Involuntary choreiform movements should not be interpreted as a refusal to eat.*
- Provide high-kilojoule, nutritious foods and sufficient snacks; request input from a dietitian. *The constant movement of HD increases kilojoule requirements.*
- Avoid milk; provide frequent oral hygiene. *Milk tends to thicken secretions. Decreasing thick secretions may improve ability to swallow and enable the person to ingest more kilojoules.*

Impaired skin integrity

Skin integrity is only one component of the person's general need for protection and avoidance of injury. Several factors increase the risk of impaired skin integrity, including poor nutritional status, eventual total immobility and incontinence.

- Evaluate the skin for actual and potential areas of breakdown. *Establishing a baseline is necessary to modify care and provide prophylactic protection of high-risk pressure areas.*
- Determine nutritional status, especially serum prealbumin level and vitamin, mineral and kilojoule intake. *Optimal nutritional status and positive nitrogen balance help prevent skin breakdown and formation of pressure ulcers.*

- Turn and inspect the skin at least every 2 hours, giving special consideration to areas that are most prone to breakdown, such as heels and coccyx. *Pressure points are particularly susceptible to skin breakdown.*
- Provide ROM exercises on a regular schedule in the daytime. *Movement stimulates circulation, which provides oxygenation and allows nutrients to reach muscles and skin.*
- Keep the skin clean and dry; pay particular attention to the perineal area if incontinent. *Skin in close proximity to the perineal area, such as the sacral area, is highly susceptible to breakdown due to exposure to wet, acidic urine and faecal material.*
- Place on an alternating-pressure mattress with foot board. *Decreasing pressure on bony prominences and preventing shearing forces serve to prevent skin breakdown.*
- Pad side rails and headrests of special chairs; have the person wear a helmet. *The person's violent movements can cause trauma to the head and extremities.*

Impaired verbal communication

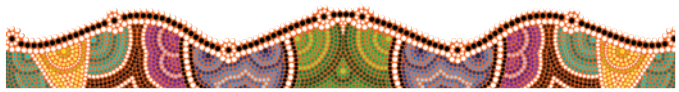
The inability to control muscles related to speech, swallowing and facial movement contributes to problems of verbal communication. Because HD affects fine motor movement, especially the distal portion of the extremities, the hands are not effective in communication. As the disease progresses, mental abilities are also compromised, making both receptive and expressive communication impossible.

- Choose alternative methods of communication while the person is able to participate. *Anticipatory planning may facilitate communication and decrease anxiety.*
- Continue to incorporate therapeutic communication techniques, even though the person is not responsive: maintain eye contact, use touch and talk directly to the person rather than to others in the room. *These techniques enhance the individual's dignity and self-worth.*
- Seek input from family about the person's usual preferences and how they are communicated; be alert for subtle cues. *Non-verbal communication techniques may be individualised and more readily recognised by the family member or caregiver who usually provides care.*
- Continue talking to the person, even though there is no apparent response. *Hearing may not be impaired, even though the person cannot speak.*

Community-based care

People with HD and their families may know how devastating the illness is because they may have cared for a parent or other close family member who had the illness. Many families are overwhelmed with just the thought of the physical and psychosocial debilitation that the disease brings. Fear, anxiety and hopelessness leading to depression are common reactions. Teaching ways to cope effectively with the psychosocial and physical changes is an integral part of the nurse's responsibilities. Referrals to appropriate agencies, such as adult day care centres, Huntington's Australia and local support groups or a psychologist, should be part of the nursing plan.

Another aspect of personal teaching concerns the genetic transmission of HD; refer people and family members to a geneticist. Nurses are frequently involved with clarifying information, especially concerning the transmission, course of illness and prognosis. A caring, sensitive approach is crucial. Information about transmission of an autosomal dominant trait is discussed in Chapter 7.



THE PERSON WITH MOTOR NEURONE DISEASE

Motor neurone disease (MND), also known as **amyotrophic lateral sclerosis (ALS)** or *Lou Gehrig's disease*, is a rapidly progressive and fatal degenerative neurological disease characterised by weakness and wasting of muscles under voluntary control, without any accompanying sensory or cognitive changes. The name is derived from the pathophysiological processes of muscle atrophy (*amyotrophy*), resulting from lower motor neuron involvement, and sclerosis of the corticospinal tract in the lateral column of the spinal cord, resulting from upper motor neuron involvement. Death results in 2 to 5 years after onset of the manifestations (although some people live 10 years or more), usually due to respiratory failure.

MND affects as many as 1900 people in Australia; on average, at least two people are diagnosed every day, and another 787 die of MND every year. In most cases, the disease occurs at random without clearly associated risk factors; however, about 10% of all cases are inherited in what is termed familial MND (Motor Neurone Disease Australia, 2013).

Most people are between 40 and 60 years of age at diagnosis; the incidence is higher in men in the earlier ages but becomes equal with women after menopause. Most of the health problems a person with MND encounters are related to swallowing and managing secretions, communication and dysfunction of the muscles used in respiration.

Pathophysiology

MND results from the degeneration and demyelination of both upper and lower motor neurons in the anterior horn of the spinal cord, brainstem and cerebral cortex. Death of the motor neurons results in axonal degeneration, demyelination, glial proliferation and scarring along the corticospinal tract. In the early stages of the disease, surviving motor neurons sprout new branches to reinnervate affected muscle fibres, preserving muscle strength. However, when more than half of the lower motor neurons are affected, reinnervation fails and weakness is evidenced.

Although the pathogenesis of MND is not clear, abnormal glutamate metabolism and hydrogen peroxide production are being studied. Echovirus RNA has also been isolated in spinal

cord tissue in some people with non-familial MND. Environmental factors, excess intracellular calcium and antibodies to calcium channels are also being researched.

Manifestations

The initial manifestations may relate to dysfunction of upper motor neurons, lower motor neurons or both. Dysfunction of upper motor neurons results in spastic, weak muscles with increased deep tendon reflexes. Dysfunction of lower motor neurons results in muscle flaccidity, paresis (weakness), paralysis and atrophy.

Weakness and paresis are common early manifestations. The weakness may initially affect only one muscle group. Manifestations vary according to the particular muscle group involved; *fasciculations* (twitching) of involved muscles are common in the early stage of the disorder. With the loss of muscle innervation, the muscles atrophy and paralysis results. Muscle mass decreases and people complain of progressive fatigue. Typically, the disease first affects the hands, then the shoulders, upper arms and, finally, the legs.

Increasing brainstem involvement causes progressive atrophy of the tongue and facial muscles with eventual dysphagia and dysarthria. Emotional lability and loss of control occur, but dementia is not part of the pathological progression of MND. Vision, hearing, sensation and cognitive ability usually remain intact. A summary of manifestations and complications is presented in the box below.

MANIFESTATIONS AND COMPLICATIONS MND

MUSCULOSKELETAL SYSTEM
- Weakness and fatigue
- 'Heaviness' of legs
- Fasciculations
- Uncoordinated movements, loss of fine motor control in hands
- Spasticity
- Paresis
- Hyperreflexia
- Atrophy
- Problems with articulation
- Complications: paralysis, loss of ability to perform ADLs, total immobility, aspiration, loss of verbal communication

RESPIRATORY SYSTEM
- Dyspnoea
- Difficulty clearing airway
- Complications: pneumonia, eventual respiratory failure

NUTRITIONAL EFFECTS
- Difficulty chewing
- Dysphagia
- Complication: malnutrition

EMOTIONAL EFFECTS
- Loss of control, lability
- Complication: depression

INTERPROFESSIONAL CARE

Because many treatable disorders may cause manifestations similar to those that appear in the initial stage of MND, a thorough evaluation is required. Once MND is diagnosed, the primary goal is to support the person and their family in meeting physical and psychosocial needs, particularly as the disease progresses.

Medical and nursing care for people with MND is primarily supportive. Referral for home health management is indicated. Occupational, physical, speech and respiratory (physiotherapy) are major supportive and rehabilitative treatments. As the disorder progresses and swallowing becomes ineffective, a gastrostomy tube may be necessary to provide adequate nutritional intake. Ventilatory assistance should be discussed with people before the need occurs.

Diagnosis

There is no specific test to diagnose MND. Rather, diagnosis is made based on manifestations and tests to rule out other diseases. A number of disorders may mimic early MND, including hyperthyroidism, hypoglycaemia, compression of the spinal cord, toxic agents, infections and neoplasms.

Medications

Riluzole (Rilutek), an antiglutamate, is the first medication developed to treat MND. It inhibits the presynaptic release of glutamic acid in the CNS and protects neurons against the excitotoxicity of glutamic acid. This oral medication is administered without food at the same time each day. People are regularly monitored for liver function, blood count, blood chemistries and alkaline phosphatase. They should be warned to report any febrile illness to their healthcare provider and to avoid alcohol.

Nursing care

Nursing care focuses on current health problems and on anticipating future difficulties. As with other disorders causing incapacitation and dependence, individualised nursing goals and interventions relate to decreasing complications, especially those associated with loss of muscular function and immobility; promoting independence to the extent possible; initiating referrals, particularly to a support group for both the person and their family; and providing physical and psychosocial support as indicated.

Of special consideration is planning for the person's eventual inability to communicate. Because the person's eye muscles and movements remain intact, signals can be prearranged before the loss of speech.

Nursing diagnoses and interventions

Two nursing diagnoses that frequently apply to people with MND are *Risk of disuse syndrome* and *Ineffective breathing pattern*.

Risk of disuse syndrome

People with MND are at risk of developing problems associated with bed rest not only because they cannot move and reposition themselves, but also because they frequently have altered nutritional and hydration status. Nursing interventions focus on preventing skin breakdown and infections such as urinary tract infections.

- Assess current condition for baseline parameters, particularly skin over bony prominences, lung sounds and vital signs. *Understanding the person's current condition allows accurate future assessment and realistic planning.*
- Assess skin; provide skin care and obtain an alternating-pressure mattress. *Pressure points are at risk of breakdown; early detection is crucial to instituting appropriate care.*
- Institute active ROM exercises, as the person is able. Perform passive ROM exercises every 2 hours, when the person is turned. *Contractures can develop within a week because extensor muscles are weaker than flexor muscles.*
- Maintain positive nitrogen balance and hydration status: monitor prealbumin levels, haemoglobin and haematocrit levels, and urine specific gravity. *Adequate protein is required to maintain osmotic pressure and prevent oedema; positive nitrogen balance promotes optimal body functioning.*
- Monitor for manifestations of infection; for example, assess urine, especially if a urinary catheter is present. *Urinary catheters place people at high risk of sepsis; bed rest places the person at greater risk of urinary stasis.*

CONSIDERATION FOR PRACTICE

Urinary tract infection is indicated by cloudy, foul-smelling urine, pain on urination, fever and general malaise.

Ineffective breathing pattern

As the muscle weakness of MND continues, people become less able to breathe. The respiratory muscles are affected and people eventually may require ventilatory assistance. The nurse must initiate measures to support the existing respiratory effort.

- Obtain a baseline assessment of breathing pattern, air movement and oxygen saturation. *Assessments indicating the person's current condition provide data to plan individualised interventions.*
- Turn at least every 2 hours. *Movement enhances the ability to move pulmonary secretions and prevents stasis.*
- Elevate the head of the bed at least 30 degrees, suction as indicated and provide oxygen. *This supports ventilation and enhances lung expansion as the person's condition changes.*
- Monitor temperature and lung sounds routinely; obtain sputum culture as indicated. *Early detection of a possible infectious process leads to prompt treatment.*

CONSIDERATION FOR PRACTICE

A pulmonary infection is indicated by respiratory difficulty, crackles and/or wheezes, cough productive of yellow or green sputum, fever and malaise.

Community-based care

Initial teaching centres on explaining the disease process, expected course and prognosis. Referral to a social worker to determine home care needs and financial assistance is helpful. Counselling and referrals to a home health agency, dietitian and physiotherapist, and speech and occupational therapists can help the family meet the person's changing needs and abilities. The realistic anticipation of needs cannot be overemphasised.

As the person becomes more debilitated, family members or other care providers focus on preventing complications. For example, family members need to know how to suction the person and perform the first aid treatment for choking to prevent aspiration. Teaching the family how to prevent problems related to immobility is a primary consideration for the nurse.

LINKS TO NATIONAL PATIENT SAFETY STANDARDS

NSQHS Standard 10:
Preventing Falls and Harm from Falls.

'The intention of this standard is to reduce the incidence of patient falls and minimise harm from falls.' (Australian Commission on Safety and Quality in Health Care (ACSQHC), 2012, p. 66)

Implementing this standard is achieved by the establishment of systems and procedures that aim to eliminate preventable falls and reduce harm resulting from falls that may occur. This includes the development of falls-related policies and procedures; thorough screening and assessment to identify people at increased risk of falls; the implementation of evidence-based falls-prevention strategies; and communicating with patients, families and carers about identified falls risks, and collaborating to develop suitable falls-prevention plans.

Individuals with degenerative neurological disorders are at risk of falls due to the progressive nature of these conditions. These conditions increase a person's risk of falls due to the development of increased confusion, disorientation, fatigue and tremors; decreased motor control; unsteady gait; and loss of balance. The implementation of systems that address prevention of falls and harm from falls is essential to the safety and wellbeing of people with degenerative neurological conditions.

Source: © Australian Commission on Safety and Quality in Health Care.

Another focus of teaching is basic care needs, such as care required to meet elimination needs. Teach families methods to establish a bowel routine, considerations related to a urinary catheter and the need to promptly report manifestations of an infection.

Throughout the early stage and continued care of the person with MND and their family, much consideration is given to psychosocial concerns. Depression, anger and denial may be initial reactions; refer the person and their family to an MND support group, social worker, psychologist or psychiatrist as indicated.

PERIPHERAL NERVOUS SYSTEM DISORDERS

Many aetiological agents are responsible for peripheral nervous system disorders. Autoimmune disorders, viruses, environmental toxins such as heavy metals, and nutritional deficiencies can affect the peripheral nervous system.

THE PERSON WITH MYASTHENIA GRAVIS

Myasthenia gravis is a chronic autoimmune neuromuscular disorder characterised by fatigue and severe weakness of skeletal muscles. People experience periods of remission and exacerbation, and mild forms of the disorder exist. Weakness may remain limited to a few muscle groups, especially the ocular muscles, or may become generalised with all muscles eventually becoming weakened.

Women are affected three times more frequently than men. The average age of onset for women is 28 and men is 42 (Muscular Dystrophy Foundation Australia, 2012). Treatment with anticholinesterase medications has greatly improved the prognosis and symptom management.

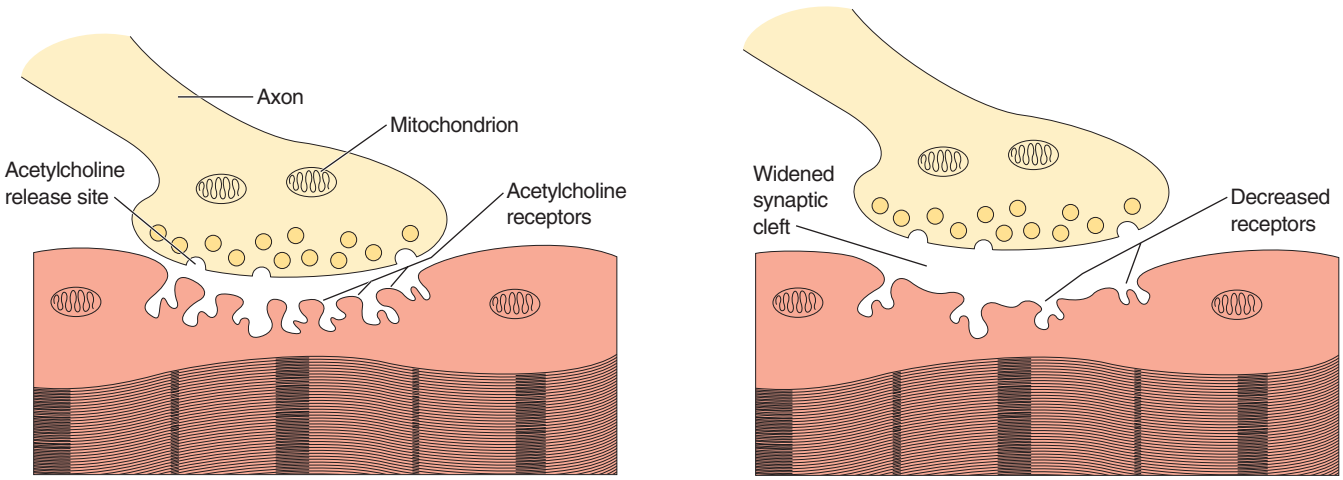
Pathophysiology

The axons of motor neurons divide as they enter skeletal muscles and each axonal ending forms a neuromuscular junction. Although the axonal ending and the muscle fibre are extremely close, they are separated by the synaptic cleft. The transmission of nerve impulses from the nerve to the muscles occurs at the neuromuscular junctions. The neurotransmitter acetylcholine is released from the axonal ending, crosses the synaptic cleft, attaches to acetylcholine receptors on the muscle fibre and stimulates the muscle.

In myasthenia gravis, antibodies destroy or block neuromuscular junction receptor sites, resulting in a decreased number of acetylcholine receptors. Structural changes also result in diminished acetylcholine uptake. The net result is a decrease in the muscle's ability to contract despite a sufficient amount of acetylcholine. A comparison of a normal neuromuscular junction and one affected by myasthenia gravis is shown in Figure 43.4.

In about 75% of people with myasthenia gravis, the thymus gland, which is usually inactive after puberty, continues to produce antibodies because of hyperplasia of the gland or because of tumours. It is believed that the thymus is a source of autoantigen that triggers an autoimmune response in myasthenia gravis. The exact mechanism and reason for the thymus gland's antibody production are unknown.

Myasthenia gravis is sometimes associated with a tumour of the thymus, thyrotoxicosis (hyperthyroidism), rheumatoid arthritis and lupus erythematosus. The disorder is often diagnosed



A Normal neuromuscular junction

B Myasthenia gravis

FIGURE 43.4 ■ *A*, A normal neuromuscular junction and *B*, one showing the changes seen in myasthenia gravis. These changes interfere with the transmission of nerve impulses to the muscle

when a person seeks treatment for a coincidental infection that exacerbates manifestations. Exacerbations may also occur before the menstrual period and during or soon after pregnancy.

Manifestations

The manifestations of myasthenia gravis correspond to the muscles involved. Initially, the eye muscles are affected and the person experiences either diplopia (unilateral or bilateral double vision) or ptosis (drooping of the eyelid) (see Figure 43.5). Next, the facial, speech and mastication muscles become weak and people may have periods of dysarthria and dysphagia. Fatigue is evident even when the person tries to eat a meal; the muscles of chewing tire and the person is forced to stop eating momentarily. A smile becomes a snarl or grimace, and the voice is weak with a muffled nasal quality. Problems performing fine motor movements of the hands, such as writing, appear early in the disease.

As the disease progresses, the muscles of the neck and extremities are affected. When the muscles of the neck become affected, the head juts forward. Deep tendon reflexes are usually normal, however, even in weak muscles. Fatigue and weakness are exacerbated with stress, fever, overexertion and exposure to heat, and are relieved by rest. Manifestations vary on a daily basis. Manifestations and complications of myasthenia gravis are listed in the box below.

Complications

Complications are directly related to the degree of muscle weakness and the specific muscles involved. For example, when the pharyngeal and palatal muscles are affected, the person cannot manage swallowing and may aspirate food or fluids. The person is at increased risk of pneumonia because weakness of the diaphragm and muscles of respiration compromises gas exchange. People with myasthenia gravis can develop life-threatening emergencies, including myasthenic crisis and cholinergic crisis.

Myasthenic crisis

Myasthenic crisis is a sudden exacerbation of motor weakness putting the person at risk of respiratory failure and aspiration. Myasthenic crisis most often is due to undermedication, missed doses of medication or a developing infection. Manifestations of myasthenic crisis include tachycardia, tachypnoea, severe respiratory distress, dysphagia, restlessness, impaired speech and anxiety.

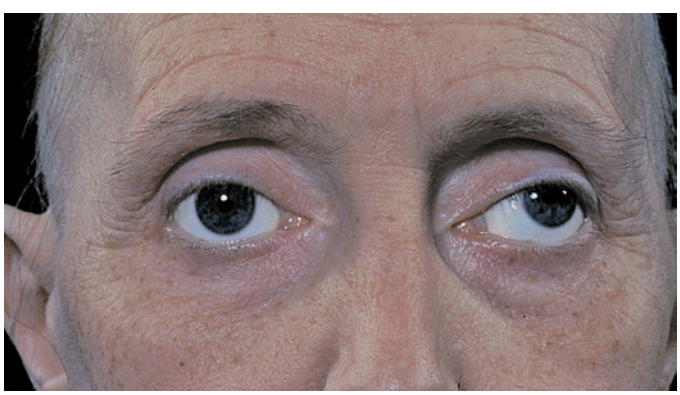


FIGURE 43.5 ■ In myasthenia gravis, the person experiences unilateral weakness of the facial muscles. Note the drooping of one eyelid

Source: Wellcome Image Library/Custom Medical Stock Photo.

MANIFESTATIONS AND COMPLICATIONS

Myasthenia gravis

OCULAR AND FACIAL
- Ptosis
- Diplopia
- Facial weakness
- Dysphagia
- Dysarthria
- Complications: difficulty closing eyes, aspiration, impaired communication and nutrition

MUSCULOSKELETAL
- Weakness and fatigue
- Decreased function of hands, arms, legs and neck muscles
- Complications: inability to perform ADLs and self-care activities, complications related to immobility, myasthenic and cholinergic crises

RESPIRATORY
- Weakening of intercostal muscles
- Decrease in diaphragm movement
- Breathlessness and dyspnoea
- Poor gas exchange
- Complications: decreasing ability to walk, eat and perform other ADLs, pneumonia

NUTRITIONAL
- Inability to chew and swallow
- Decreasing ability to move tongue
- Impairment of fine motor movements: inability to feed self
- Complications: weight loss, dehydration, malnutrition, aspiration

Cholinergic crisis

Cholinergic crisis is the result of overdosage with the anticholinesterase (cholinergic) medications used to treat myasthenia gravis. Gastrointestinal manifestations, severe muscle weakness, vertigo and respiratory distress are signs of cholinergic crisis. Both types of crises are emergency, life-threatening situations; people frequently require ventilatory assistance. Differentiation is based on the person's response to edrophonium chloride (Tensilon). In myasthenic crisis the test is positive; in cholinergic crisis the test is negative (see the discussion that follows under 'Diagnosis').

INTERPROFESSIONAL CARE

Care of the person with myasthenia gravis focuses on providing appropriate treatment, preventing complications and supporting the person and their family in meeting physical and psychosocial needs, especially as the disease progresses.

Diagnosis

Diagnostic tests are conducted following a thorough history and physical examination, with special attention to the facial, oculomotor, laryngeal and respiratory muscles. Diagnostic tests include the anticholinesterase (Tensilon) test, nerve stimulation studies and an analysis of antiacetylcholine receptor antibodies.

In the Tensilon test, the person is injected with edrophonium chloride, a short-acting anticholinesterase. People with myasthenia gravis show a significant improvement in muscle strength that lasts approximately 5 minutes. This test is also used to differentiate myasthenic crisis (caused by insufficient medication, so the person shows improvement with the drug) from cholinergic crisis (caused by overmedication, so the person does not show improvement).

Single-fibre electromyography can detect delayed or failed neuromuscular transmission in muscle fibres supplied by a single nerve fibre. Serum assay of circulating acetylcholine receptor antibodies, if increased, is diagnostic of myasthenia gravis with a sensitivity of 80–90%.

Medications

The primary group of medications used to treat myasthenia gravis is the anticholinesterases. These drugs act at the neuromuscular junction and allow acetylcholine to concentrate at the receptor sites, thus promoting muscle contraction. Pyridostigmine (Mestinon) is the most commonly used acetylcholinesterase inhibitor for myasthenia gravis. The person's decrease in manifestations guides dosage.

Immunosuppression with glucocorticoids, typically prednisone, is another pharmacological therapy aimed at improving muscle strength. People must be aware of the need to stay on the drug at the prescribed dose to determine the least amount required for efficacy. If people do not respond to prednisone alone, it may be combined with other immunosuppressive agents, such as cyclosporin or azathioprine (Imuran). Medications used to treat myasthenia gravis are discussed in the 'Medication administration' box below.

Surgery

Approximately 75% of people with myasthenia gravis have dysplasia of the thymus gland. Therefore, thymectomy is often

MEDICATION ADMINISTRATION The person with myasthenia gravis

ANTICHOLINESTERASES/CHOLINESTERASE INHIBITORS

Neostigmine (Prostigmin)
Ambenonium (Mytelase Caplets)
Pyridostigmine (Mestinon, Regonol)
For diagnosis: edrophonium chloride (Tensilon)

Cholinesterase inhibitors are used in myasthenia gravis to enhance the effects of acetylcholine at the remaining skeletal muscle receptors. Cholinesterase inhibitors do not cure or change the underlying pathophysiological processes, but they can provide effective, lifelong improvement of symptoms. Because the cholinesterase inhibitors are non-selective, the neuromuscular, muscarinic and ganglionic junctions are each affected.

Adjusting the dose to obtain maximum benefit with minimal side effects is a major consideration when administering cholinesterase inhibitors. Initially, small doses are given, followed by incremental increases until optimal muscle strength is obtained. The dose may need to be adjusted when activities result in symptoms of undermedication, such as increased ptosis. Severe undermedication results in myasthenic crisis. Although a sustained-release form of pyridostigmine is available for bedtime use, it should not be used during the day because of its inconsistent absorption.

Cholinesterase inhibitors should not be administered to people experiencing obstruction of the intestinal or urinary tract. Caution is advised when administering these drugs to people with asthma, hyperthyroidism, bradycardia or peptic ulcer disease. Cholinesterase inhibitors can cross the placenta; reproductive counselling is indicated.

Nursing responsibilities

- Obtain a baseline assessment of muscle strength and abilities, concentrating on swallowing and ptosis.
- Administer the medication parenterally if the person has dysphagia. Check the dose of the medication carefully when changing from oral to parenteral routes.
- Evaluate the effectiveness of the medication and document the response—for example, time when fatigue occurs in relation to activities.
- Promptly recognise and respond to manifestations of excessive stimulation of muscarinic receptors: excess salivation, urinary urgency, bradycardia, gastrointestinal hypermotility, diaphoresis. Atropine can be administered to combat these manifestations. Respiratory depression and failure can occur and require mechanical ventilation.
- Have a muscarinic antagonist (e.g. physostigmine) readily available to treat poisoning.

Health education for the person and family

- Balancing symptom control with dosage is crucial; record time of dose and response in a journal. Note the time of day when fatigued and any adverse effects, such as excess salivation, sweating, slow heartbeat and diarrhoea.
- Take the medication about 30 minutes prior to meals to enhance swallowing and chewing.
- Report manifestations of myasthenic crisis immediately: severe muscle weakness, fast heartbeat, restlessness, difficulty breathing and increasing difficulty swallowing or speaking.
- Report slow heartbeat, increased salivation or sweating, and/or decreased blood pressure immediately.
- Review possible causes of myasthenic crisis: physical or emotional stress, infection or reduction in the medication dosage.
- Wear or carry MedicAlert® identification.

NURSING CARE OF THE PERSON having a thymectomy

PREOPERATIVE CARE

- Reinforce the doctor's explanation of the procedure and prepare the person for chest tubes and tracheostomy. *Realistic preparation of what to expect postoperatively encourages compliance and allays anxiety.*
- Anticipate the need for alternative communication. *The person may have a tracheostomy; preoperative planning facilitates communication after surgery.*
- Allow sufficient time for questions. Thymectomy is a major surgery requiring either a thoracotomy and sternal split, or a transcervical approach. *The person is usually anxious and adequate time must be allocated to preoperative instruction.*

POSTOPERATIVE CARE

- Provide meticulous pulmonary hygiene: turning, deep breathing and coughing at least every 2 hours; use an incentive spirometer. *Regardless of surgical approach, measures are aimed at preventing pulmonary complications of atelectasis and pneumonia.*
- People with a thoracotomy and sternal split procedure will require care of the anterior chest tube. *Observe for complications, such as pneumothorax. Air may enter the thoracic cavity—be alert for sudden chest pain and dyspnoea, decreased breath sounds and early signs of shock, such as restlessness.*
- Manage pain with scheduled analgesic therapy. *Maintaining a therapeutic blood level of analgesic provides better pain control than waiting until the person requests medication, as on a prn basis.*

recommended for people younger than 60. The two surgical approaches used are the transcervical approach, which is considered less invasive, and the transsternal approach. The latter approach allows a more extensive removal of the gland; however, it also poses more potential complications because it involves splitting the sternum.

Preoperatively, people may be tapered from steroid therapy. Usually, pyridostigmine is administered to prevent muscular manifestations during the perioperative period. Postoperative nursing care focuses on preventing complications and controlling pain. Nursing implications for the person undergoing thymectomy are presented in the box above. Remission is obtained in about 40% of people but may take several years to achieve. Refer to Chapter 35 for care of the person having a thoracotomy and chest tubes. A tracheostomy may be required when the diaphragm or intercostal muscles are involved.

Plasmapheresis

Plasma exchange in myasthenia gravis may be used in conjunction with other therapies; for example, it may be performed prior to surgical intervention. The goal of therapy is to remove the antiacetylcholine receptor antibodies, thus improving severe muscle weakness, fatigue and other manifestations. The procedure is frequently performed when respiratory muscle involvement is evident. See the box below for nursing care of the person having plasmapheresis.

NURSING CARE OF THE PERSON having plasmapheresis

PREPROCEDURE CARE

- Teach about the procedure and what to expect, including what the machine looks like, the need for arterial and venous insertion sites, and the length of time of the procedure (2 to 5 hours). *Giving information, answering questions and addressing concerns decrease anxiety.*
- Check with the doctor about withholding medications until after the procedure. *Medications may be removed from the body as an incidental part of the plasmapheresis process.*
- Assess vital signs and weight. *Baseline parameters are necessary to evaluate for fluid imbalances and response to therapy.*
- Assess FBC, platelet count and clotting studies. *People undergoing plasmapheresis are at high risk of anaemia and coagulation problems secondary to haemolysis of cells.*
- Check blood type and cross-match for replacement blood products. *Hypersensitivity reactions can occur and close monitoring is important.*

CARE DURING AND AFTER THE PROCEDURE

- Observe for dizziness or hypotension. *Hypovolaemia is a complication of plasma exchange, especially during the procedure when up to 15% of the person's blood volume is in the cell separator.*
- Apply pressure dressing to access site(s). *Direct pressure helps decrease or prevent bleeding.*
- Monitor for infection and bruises at the intravenous port site. *The site of vascular access is at risk of complications and must be routinely and carefully assessed for signs of infection and bleeding or haematoma formation.*
- Monitor electrolytes and signs of electrolyte loss. Report imbalances and replace electrolytes as ordered. Observe for circumoral tingling, Chvostek's and Trousseau's signs if calcium levels are low, and cardiac arrhythmias and leg cramps if potassium levels are low. *Hypocalcaemia and hypokalaemia may occur. Hypocalcaemia occurs because the anticoagulant citrate dextrose binds with calcium.*
- Re-evaluate pre-procedure laboratory data, especially FBC, platelet count and clotting times. *The cell-separating process can damage cells; anticoagulation is part of the procedure.*

Nursing care

Because avoiding fatigue is a major part of teaching for myasthenia gravis, it is important to incorporate interventions to enhance rest and conserve energy (see Box 43.4). For example, suggest sitting while preparing meals and performing hygiene and grooming. Anticipating problems such as impaired communication, and developing alternative solutions, can be helpful in promoting independence. A nursing care plan for the person with myasthenia gravis is given below.

Nursing diagnoses and interventions

Nursing care of people with myasthenia gravis focuses not only on present problems but also on anticipated needs. Preventing myasthenic and cholinergic crises, and providing psychological support to people and families, are two important aspects of care. Individualised care depends on the specific therapy instituted. This section discusses the nursing diagnoses related to ineffective airway clearance and impaired swallowing; other nursing diagnoses that commonly apply, such as *Fatigue*, are addressed in other sections of this chapter.

Ineffective airway clearance

The underlying causes of ineffective airway clearance for the person with myasthenia gravis include poor cough mechanism, decreased rib cage expansion, diminished diaphragm movement and decreased expiratory effort. The following interventions require particular attention if the person undergoes a thymectomy.

- Assist with turning, deep breathing and coughing at least every 2 hours. Teach proper coughing techniques; use an incentive spirometer every 2 hours while the person is awake. *Position changes promote lung expansion; coughing helps clear secretions from the tracheobronchial tree.*
- Place in a semi-Fowler's position. *This position expands the lungs and alleviates pressure from the diaphragm; these are especially important considerations if the person is obese.*
- Maintain hydration status and monitor for dehydration; use a humidifier as needed. If needed, teach family how to perform percussion, postural drainage and suction. *Interventions to liquefy secretions, such as ensuring a daily fluid intake of up to 2500 mL (perhaps via feeding tube or parenteral route), help the person mobilise and expectorate sputum.*
- Monitor lung sounds, the rate and character of respirations, and pulse oximetry readings at least every 4 hours or as indicated by the person's condition. *Frequent assessments are critical to early identification of ineffective respirations and oxygenation of tissues.*

Impaired swallowing

People with myasthenia gravis have weakness of the laryngeal and pharyngeal muscles involved with swallowing. Alterations in swallowing place the person at risk of poor nutrition as well as possible aspiration. Family members need to be included in teaching, particularly the person who prepares and assists with meals.

- Assess the ability to safely manage various consistencies of foods; consult with a speech therapist for evaluation. *Dysphagic people are at risk of aspiration; matching food consistency to the person's ability to swallow enhances safety.*
- Plan meals to promote medication effectiveness. *Pyridostigmine should be given 30 minutes before the meal to provide optimal muscle strength for swallowing and chewing.*
- Have the person eat slowly, using small bites of food. Schedule meals during periods when the person is adequately rested; develop a daily schedule incorporating rest periods. *Fatigue may add to dysphagia, putting the person at greater risk of aspiration.*
- If necessary, give cues while eating, such as 'Chew your food thoroughly; swallow.' *Keeping the person focused may enhance swallowing.*
- Teach caregivers the first aid treatment for choking and how to suction. *Knowing specific measures to take in case of aspiration decreases both the person's and the family's anxiety and promotes confidence in managing potential problems.*

BOX 43.4 Personal and family teaching: myasthenia gravis

- Schedule periods of rest and avoid stress; conserve energy when possible.
- Avoid cigarette smoke, alcohol and beverages with quinine (e.g. tonic water).
- Take medications as prescribed. If manifestations change, consult the doctor; the dose may need to be adjusted.
- Avoid extremes of temperature; an environment that is too hot or too cold may cause an exacerbation of myasthenia gravis.
- Avoid people with upper respiratory infections; infections can result in an exacerbation and extreme weakness.

Community-based care

Teaching for the person and family with myasthenia gravis focuses on prevention and recognition of crisis situations, understanding the disorder and methods for coping with both physical and psychosocial problems. Setting realistic goals with the person and family provides opportunities for self-assessment and promotes active participation in rehabilitation.

Address the following topics:

- the importance of maintaining consistency in medication dosage and management
- realistic expectations

NURSING CARE PLAN A person with myasthenia gravis

Kirsten Avis, a 44-year-old homemaker and mother of two teenage sons, was diagnosed with myasthenia gravis 2 years ago. She takes an anticholinesterase medication, pyridostigmine (Mestinon), four times a day. Over the past month she has been experimenting with decreasing the dose of her pyridostigmine because she has 'felt so good'. She was prescribed 60 mg of pyridostigmine three times a day before meals and one-half of a long-acting 180 mg pyridostigmine tablet at night.

Three days ago, she began having chills and fever and her myasthenic symptoms became markedly worse. Mrs Avis is easily fatigued and has been experiencing increasing weakness, bilateral ptosis and mild dysphagia in the late afternoon and evenings. She is admitted to the hospital.

ASSESSMENT

Lela Silva, RN, is caring for Mrs Avis. Physical examination of Mrs Avis reveals severe muscle weakness bilaterally in her hands, arms and thorax. Her voice is nasal and she speaks slowly; the longer she speaks, the more difficult it becomes to understand her. She is anxious and dyspnoeic. Her complaints of weakness, dysphagia, dysarthria, problems with mobility and ptosis are more pronounced later in the day. Vital signs are as follows: BP 138/88, P 88, R 28, T 39°C.

Some improvement in muscle weakness is noted following a restful night's sleep; however, the respiratory distress is more evident and Mrs Avis is increasingly restless. She is moved to the critical care unit for advanced monitoring and possible ventilatory assistance. The medical diagnosis is myasthenic crisis secondary to pulmonary infection.

DIAGNOSES

- *Impaired gas exchange* related to ineffective breathing pattern and muscle weakness, evidenced by dyspnoea, restlessness and fatigue.
- *Risk of aspiration* related to dysphagia, manifested by difficulty swallowing.
- *Fatigue* related to increased energy needs from muscular involvement, manifested by limb weakness and mobility difficulties.

PLANNING

- Management of pulmonary infection and exacerbated symptoms of myasthenia gravis.

Expected outcomes

- Pulse oximetry readings will be maintained at 92% or above.
- No aspiration will occur.
- Will verbalise decreasing fatigue when performing ADLs.
- Will state the correct method of medication dosing and demonstrate how she will maintain schedule.

IMPLEMENTATION

Mrs Avis's manifestations improve following administration of edrophonium chloride (Tensilon) to verify myasthenic crisis. She is placed on oxygen by mask and suctioned as needed; equipment for possible intubation and ventilation is made readily available. She is placed in a semi-Fowler's position and vital signs are assessed every 5 minutes during the acute exacerbation. The nurses in the critical care unit remain in constant attendance throughout the crisis period and provide explanations to Mrs Avis in an effort to decrease her stress and to avoid further severity of manifestations.

Three days after the crisis period, Mrs Avis is moved to a progressive nursing care unit. Nurses follow up on teaching her the manifestations of both myasthenic and cholinergic crises. They discuss the need to wear MedicAlert® identification and review medication administration techniques with Mrs Avis. The nurses emphasise in particular that Mrs Avis must not split time-released medications.

Within 5 days, Mrs Avis's condition stabilises and her weakness decreases sufficiently to allow discharge home. Although her temperature has returned to normal and her respiratory status has improved, she still has a productive cough. Oral antibiotics are prescribed for 2 weeks, after which she will have a follow-up visit with her GP. She is instructed to seek treatment promptly if respiratory symptoms or temperature indicate recurrence of infection.

EVALUATION

Mrs Avis is discharged without developing aspiration pneumonia or any symptoms of aspiration. Her airway was maintained throughout the myasthenic crisis and her pulse oximetry readings remained above 92% once oxygen therapy was initiated. On discharge, pulse oximetry is above 95% without oxygen therapy. Mrs Avis states that her fatigue and weakness have significantly improved.

Both Mrs Avis and her husband are able to explain the difference between myasthenic and cholinergic crises and to identify methods to avoid both problems. Mrs Avis correctly relates her proper medication regimen and makes an appointment for a follow-up visit with her doctor.

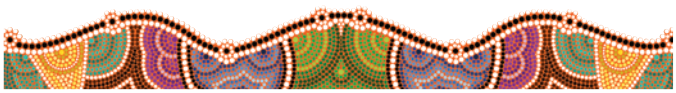
CRITICAL THINKING IN THE NURSING PROCESS

1. What is the rationale for administering Tensilon to evaluate a myasthenic crisis?
2. Develop a plan to assist Mrs Avis to avoid fatigue when preparing and eating meals.
3. Which factors contributed to Mrs Avis's diagnosis of myasthenic crisis?

REFLECTION ON THE NURSING PROCESS

1. Which communication and education strategies could you use to reduce fatigue related to the performance of ADLs and daily tasks?
2. Outline what you have learned from this case study that you will apply to your future nursing practice.

- methods to avoid fatigue and undue stress; specific measures for avoiding upper respiratory infections and exposure to extreme heat or cold
- birth control measures or referral for counselling (Pregnancy can exacerbate manifestations; also, medications used to control myasthenia gravis, such as neostigmine bromide (Prostigmin), cross the placenta.)
- referral to support groups
- helpful resources such as:
  - The Australian Myasthenic Association: www.myasthenia.org.au
  - National Institute of Neurological Disorders and Stroke (NINDS): www.ninds.nih.gov.



THE PERSON WITH GUILLAIN–BARRÉ SYNDROME

Guillain–Barré syndrome (GBS) is an acute inflammatory demyelinating disorder of the peripheral nervous system characterised by an acute onset of motor paralysis (usually ascending). The classification of Guillain–Barré subtypes includes acute inflammatory demyelinating polyradiculoneuropathy, acute axonal motor neuropathy and acute motor and sensory axonal neuropathy.

GBS is one of the most common peripheral nervous system disorders, affecting about 1 to 2 people per 100 000 in Australia (Guillain–Barré Syndrome Association of NSW, 2014). The cause is unknown, but precipitating events include a respiratory or gastrointestinal viral or bacterial infection 1 to 3 weeks prior to the onset of manifestations, surgery, viral immunisations and other viral illnesses. In 60% of cases, *Campylobacter jejuni* is identified as the cause of the preceding infection. Approximately 90% of people with GBS have a spontaneous recovery with little or no residual disabilities.

The disease is characterised by progressive ascending flaccid paralysis, accompanied by paraesthesias and numbness. About 20% of people have respiratory involvement to the point that ventilatory assistance is required. GBS is often a medical emergency.

Pathophysiology

The primary pathophysiological process in GBS is the destruction of myelin sheaths covering the axons of peripheral nerves. The demyelination is thought to be the result of both a humoral- and a cell-mediated immunological response. The loss of myelin results in poor conduction of nerve impulses, causing sudden muscle weakness and loss of reflex response. Other manifestations occur when nerve conduction to various muscles is interrupted. The stages of GBS and their usual manifestations are presented in Box 43.5.

Manifestations

Muscles, sensory nerves and cranial nerves are commonly affected in people with GBS. Most people experience symmetrical muscle weakness, initially in the lower extremities. The weakness and sensory loss then ascends to the upper extremities, torso and cranial nerves. Sensory involvement includes severe pain, paraesthesia and numbness. Cognition and level of consciousness are not affected. Facial nerve involvement results in the inability to change facial expressions and close the eyes. Muscles involved with chewing, swallowing and speaking may be affected.

Paralysis of intercostal and diaphragmatic muscles may alter respiratory function. These people require ventilatory assistance and supportive care. Involvement of the autonomic nervous system is characterised by fluctuating blood pressure, cardiac arrhythmias and tachycardia, paralytic ileus, syndrome of inappropriate antidiuretic hormone secretion and urinary retention.

The weakness usually plateaus or improves by the fourth week. Strength then improves slowly over weeks or months. Women who have had GBS are at increased risk of relapse in the first trimester of pregnancy.

BOX 43.5 Stages of Guillain–Barré syndrome

I. Acute stage
- Characterised by severe and rapid weakness, especially in the lower extremities; loss of muscle strength progressing to quadriplegia and respiratory failure; decreasing deep tendon reflexes; decreasing vital capacity; paraesthesias, numbness; pain, especially nocturnal; facial muscle involvement (inability to wrinkle forehead or change expressions).
- Involvement of the autonomic nervous system manifested by bradycardia, sweating, fluctuating blood pressure (notably hypotension) which may last for 2 weeks.

II. Stabilising/plateau stage
- Occurs 2 to 3 weeks after initial onset.
- Marks the end of changes in condition; characterised by a 'levelling off' of symptoms.
- Generally, the labile autonomic functions stabilise.

III. Recovery stage
- May take from several months to 2 years.
- Marked by improvement in symptoms.
- Generally, muscle strength and function return in descending order.

INTERPROFESSIONAL CARE

Interventions during the acute phase (1 to 3 weeks) focus primarily on ensuring oxygenation via ventilatory assistance and preventing complications from immobility. Rehabilitation time to regain muscle strength and function varies; most people return to full pre-syndrome muscle function within 6 months to 2 years.

Care of the person with GBS requires a team approach. From the initial acute phase through rehabilitation, many members of the healthcare team are involved. An accurate and rapid diagnosis is needed to ensure prompt supportive treatment, particularly if there is respiratory involvement combined with widespread paralysis.

Diagnosis

Diagnosis of GBS is made after a thorough history and clinical examination. It must be differentiated from several disorders, among them influenza, heavy metal poisoning, Lyme disease and cranial haemorrhage. Diagnosis is made based on manifestations, history of a recent viral infection, elevated CSF protein levels and electromyography studies reflecting decreased nerve conduction. Although there is no specific test to diagnose this syndrome, several findings support and confirm the diagnosis.

Medications

No medications are available for the specific treatment of GBS. Other medications may be prescribed to provide support or prophylaxis or to combat concurrent problems; for example, antibiotics may be prescribed for urinary tract or respiratory infections. Morphine is commonly administered to control muscle pain. Anticoagulation therapy is usually instituted to prevent thromboembolic complications, such as deep venous thrombosis and pulmonary embolism, which are associated with prolonged bed rest. If hypotension is a problem, vasopressors are prescribed.

Surgery

Tracheostomy is performed if respiratory failure occurs. People who need ventilatory support are usually able to be weaned after 2 to 3 weeks, but the time frame varies greatly. When the person's vital capacity reaches 8 to 10 mL/kg, they may be weaned from the ventilator (Hickey, 2013). Insertion of a temporary pacemaker may be indicated for bradycardia.

Plasmapheresis

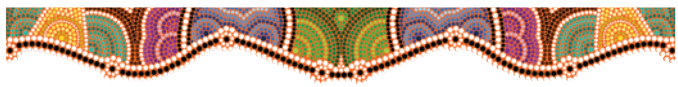
Plasma exchange has been beneficial, particularly when performed within the first 2 weeks of the syndrome's development. Antibodies are removed and immunosuppressive agents are administered concurrently. People typically have five exchanges during an 8–10-day period.

Nutrition and fluids

Nutritional support for the person who is immobilised for prolonged periods of time is crucial. Maintaining positive nitrogen balance, ensuring sufficient fluid intake and electrolyte balance, and ensuring recommended kilojoule intake are goals of therapy. When swallowing problems occur, total parenteral nutrition may be indicated if feeding via a nasogastric or gastrostomy tube is ineffective.

Physiotherapy and occupational therapy

Long-term physiotherapy and occupational therapy are crucial to recovery. People with GBS usually require prolonged rehabilitation care, which begins during the acute phase and focuses on preventing complications and limiting the effects of immobility. The severe muscle atrophy and loss of muscle tone require that people relearn many functions and skills, such as walking. Compromise in respiratory function may delay physical rehabilitation; people need positive reinforcement when they make even small gains in their progress. Continued attention to pain control is essential because paraesthesia and pain can interfere with physical therapy.



Nursing care

Many of the nursing interventions for people with Guillain–Barré syndrome involve monitoring neurological function, preventing problems of immobility, ensuring adequate hydration and nutrition, and promoting respiratory function. Anticipating needs of both the person and their family is an important aspect of care. For example, developing an alternative method of communication before it is necessary may decrease anxiety. It is important that nursing care focuses on preventing complications that may be fatal by following a rigorous predetermined schedule for turning and respiratory care (e.g. coughing, deep breathing, suctioning), using strict aseptic technique and providing continuous psychosocial support.

Nursing diagnoses and interventions

Anxiety and powerlessness are major nursing considerations. The person is almost always admitted to the critical care unit for care and is mentally alert but suddenly mute, ventilator dependent and immobile. Refer to previous nursing care sections in this chapter for interventions related to anxiety, imbalanced nutrition, impaired swallowing, impaired verbal communication and ineffective airway clearance. This section focuses on the nursing diagnoses related to pain and risk of impaired skin integrity.

Acute pain

Pain experienced with GBS varies. Frequently, there is a 'stocking–glove' pattern, with pain in the hands, feet and legs. Pain and tenderness in muscles can be severe; interventions must be individualised to personal needs. The intense pain combined with altered sensations leads to anxiety; nursing interventions can make a difference in breaking the cycle of increasing pain that leads to increased anxiety and in turn causes more pain.

- Listen to the description of pain; determine presence of triggers or a pattern. *Acknowledging the person's perception of pain is a basis for treatment; listening establishes trust.*
- Use a pain scale for determining extent of pain. *Consistent measurement is essential to evaluate degree of pain and effectiveness of intervention.*
- Use complementary therapies to help manage pain:
  - application of heat/cold
  - guided imagery
  - relaxation techniques
  - massage.

Presenting options for managing pain gives the person control over the situation and helps reduce anxiety. Non-invasive interventions may augment the therapeutic benefit of medications.

- Provide analgesics as indicated; administer on a regular schedule rather than waiting until pain becomes severe. *Anticipating and managing pain before it becomes severe decreases anxiety and averts the cycle of increased anxiety leading to increased pain.*
- Monitor for side effects of analgesics, particularly respiratory depression; assess respirations and lung sounds. Perform routine pulmonary care measures and monitor for aspiration. *People with GBS have weakened thoracic muscles; frequent respiratory monitoring is indicated.*

Risk of impaired skin integrity

During the acute and plateau stages of GBS, people are at risk of problems related to immobility and malnutrition. Impaired skin integrity is one such problem. Preventing areas of skin breakdown is important. Prophylactic interventions will help ensure that ingested protein and kilojoules are used to maintain ideal body weight and other body functions rather than to heal an avoidable problem. Implicit in interventions is maintenance of adequate nutrition.

- Inspect bony prominences and provide skin care at least every 2 hours. Reposition the person and clean, dry and lubricate the skin as needed. *These activities stimulate circulation and ensure even distribution of body weight; baseline observations allow discovery of early signs of altered integrity.*
- Pad bony prominences, such as sacral area, heels and elbows. *This decreases shearing tears on these pressure points.*
- Use an alternating-pressure mattress or water bed. *Relieving pressure stimulates circulation and promotes oxygenation of tissues.*
- Monitor for incontinence and provide thorough skin care following each episode of incontinence. *Urine is caustic to the skin and the moisture promotes skin breakdown.*

Community-based care

People and family members are frequently stunned by the rapid deterioration of function and by fear that the paralysis will be permanent. Regularly reinforce teaching because the person's high anxiety level may interfere with listening and understanding. When possible, include the person and their family in decision making; for example, seek their input when planning a daily schedule of care that incorporates various therapies.

Teaching the rationales for preventive measures reinforces the person's and family's understanding and may promote compliance during the lengthy rehabilitation. For example, because of autonomic nerve involvement, people need to be monitored for cardiac arrhythmias and taught to avoid changing position suddenly to prevent orthostatic hypotension.

Referrals to appropriate therapists are a component of anticipating needs; speech, nutritional, occupational and physical therapists are an integral part of rehabilitation. Another focus of care is teaching both the person and their family; incorporate explanations for interventions aimed at promoting self-care. For further information, refer the person and their family to the Guillain–Barré Syndrome Association in their state.

CRANIAL NERVE DISORDERS

Disorders of the cranial nerves may be caused by intracranial trauma or by pathological processes. The pairs of cranial nerves, described in Chapter 40, are numbered in the order in which they arise in the brain and are named according to their anatomical characteristic or primary function. The most common cranial nerve disorders are those affecting the trigeminal (cranial nerve V) and the facial (cranial nerve VII) nerves. These disorders, discussed in the following sections, result primarily in pain or loss of sensory or motor function.

THE PERSON WITH TRIGEMINAL NEURALGIA

Trigeminal neuralgia, also called *tic douloureux*, is a chronic disease of the trigeminal cranial nerve (V) that causes severe facial pain. The trigeminal nerve has three divisions: the ophthalmic, the maxillary and the mandibular (see Figure 43.6). The ophthalmic division supplies the forehead, eyes, nose, temples, meninges, paranasal sinus and part of the nasal mucosa. The maxillary division supplies the upper jaw, teeth, lip, cheeks, hard palate, maxillary sinus and part of the nasal mucosa. The mandibular division supplies the lower jaw, teeth, lip, buccal mucosa, tongue, part of the external ear and the meninges. Sensory fibres of the nerve conduct impulses for touch, pain and temperature; motor fibres innervate the temporal and masseter muscles used for chewing and lateral movement of the jaw. The maxillary and mandibular divisions are the divisions of the trigeminal nerve affected in almost all cases of this disorder.

Trigeminal neuralgia occurs more commonly in middle-aged and older adults, and affects women more often than men.

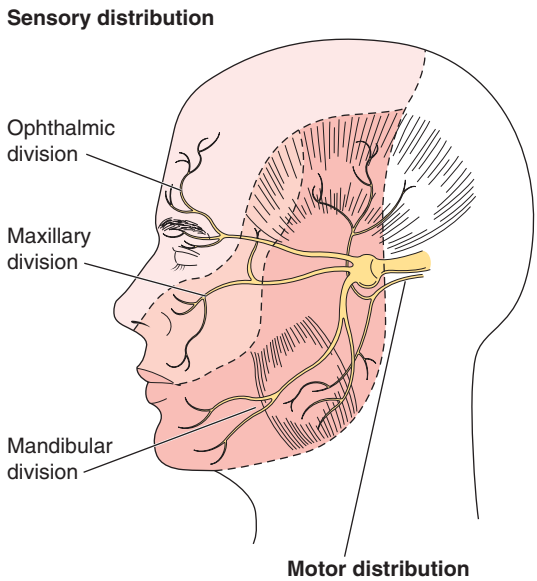


FIGURE 43.6 ■ Sensory and motor distribution of the trigeminal nerve. The three sensory divisions are ophthalmic, maxillary and mandibular

Pathophysiology

The actual cause of trigeminal neuralgia is unknown; however, contributing factors include irritation from flu-like illnesses, trauma or infection of the teeth or jaw, and pressure on the nerve by an aneurysm, a tumour or arteriosclerotic changes of an artery close to the nerve (Hickey, 2013).

Stimulating specific areas of the face, called *trigger zones*, may initiate the onset of pain. These trigger zones usually parallel the distribution of the nerve and typically follow a track leading from just over the eyebrow to the ridge of the cheekbone, along the nasolabial fold, around the corner of the mouth and down the side of the chin. The episodes of pain are initiated by many factors, including light touch, eating, swallowing, talking, sneezing, shaving, chewing gum, brushing the teeth or washing the face. Other factors that may trigger a pain episode include changes in temperature and exposure to wind. In an attempt to control the pain, people may refuse to wash, shave, eat or talk.

The episodes of pain may recur for several weeks or months. The disease then spontaneously goes into remission and the person is free of pain for periods lasting from days to years. As the person grows older, the remissions tend to become shorter and a dull ache may be present between episodes of acute pain.

Manifestations

Trigeminal neuralgia is characterised by brief (lasting a few seconds to a few minutes), repetitive episodes of sudden severe (usually unilateral) facial pain. The pain may occur as often as hundreds of times a day to as infrequently as a few times a year. The pain is experienced over the surface of the skin. It most often begins near one side of the mouth and rises towards the ear, eye or nostril on the same side of the face. People describe the pain as stabbing or lightning-like, and often respond to the pain by wincing or grimacing.

INTERPROFESSIONAL CARE

There are no specific diagnostic tests for trigeminal neuralgia. The disorder is diagnosed by the characteristic location and type of pain. The disorder is treated by pharmacological or surgical interventions.

Medications

The drug most useful in controlling the pain is the tricyclic anticonvulsant carbamazepine (Tegretol). If carbamazepine is ineffective, other medications such as the anticonvulsants phenytoin (Dilantin) or gabapentin (Neurontin) or the skeletal muscle relaxant baclofen (Lioresal) may be used. These drugs are administered to decrease paroxysmal afferent impulses and stop the pain. Drugs in this category may cause side effects of dizziness, nausea and drowsiness. Liver function, bone marrow function and blood levels of the medications should be monitored on a regular basis.

Surgery

If medications do not control the pain, surgical procedures may be performed, including various types of *rhizotomy*—the surgical severing of a nerve root. Closed surgical interventions by percutaneous rhizotomy involve inserting a needle through the cheek into the foramen ovale at the base of the brain and partially destroying the trigeminal nerve with glycerol (an alcohol), by radiofrequency-induced heat or by balloon compression of the trigeminal ganglion. These procedures carry less risk and result in shorter hospital stays than open procedures, but there is a possibility of recurrence of pain. Following surgery, the person may have some facial numbness, but there usually is no residual paralysis. The involved side of the face is insensitive to pain. The person will have some loss of facial sensation (e.g. to temperature and/or touch) and is at risk of loss of the corneal reflex. Closed procedures provide long-term pain relief and are well tolerated by the older adult. Nursing care of the person undergoing a percutaneous rhizotomy is presented in the box below.

It has been found that some structural abnormalities (such as an artery or vein compressing the nerve) may cause the neuralgia and, if so, decompression and separation of the blood vessel from the nerve root produce lasting relief of the pain (Papadakis & McPhee, 2015). The Jannetta procedure involves locating and lifting the involved vessel and placing a small piece of silicone sponge between the vessel and the nerve. Possible complications of the procedure include headache and facial pain.

Nursing care

Nursing care for the person with trigeminal neuralgia involves teaching self-management at home after medical or surgical intervention. Primary personal concerns are managing pain, maintaining nutrition and preventing injury.

NURSING CARE OF THE PERSON having a percutaneous rhizotomy

POSTOPERATIVE CARE

- Follow routine postoperative interventions for people having surgery (see Chapter 3).
- Monitor cranial nerve function every 2 to 4 hours:
  a. Assess the corneal reflex by lightly touching the cornea with a gauze square. If the reflex is intact, the person will blink. *Severing the ophthalmic division of the trigeminal nerve destroys the corneal reflex and leaves the cornea at risk of dryness and injury.*
  b. Assess the facial nerve by asking the person to blow out the cheeks, wrinkle the forehead, frown, wink and close both eyes tightly. Test taste by placing bitter, salty and sweet substances on the anterior portion of the tongue. *Facial weakness is evidenced by changes in movement in the involved side of the face. The facial nerve also innervates the anterior two-thirds of the tongue.*
  c. Assess the function of the oculomotor muscles by asking the person to follow your finger through the cardinal positions of vision (see Chapter 44). *The eyes should move together; alterations in movement indicate an abnormal response.*
  d. Assess the motor portion of the trigeminal nerve by asking the person to clench the teeth while you palpate the tightness of the contracted masseter and temporal muscles. *Loss of motor function is indicated by loss of bulk and tightness of these muscles.*
  e. Apply as prescribed an ice pack to the jaw on the operative site. *Cold decreases bleeding and swelling.*
  f. Teach the person to avoid rubbing the eye on the involved side. *Loss of the corneal reflex removes protection because the person no longer has the sensation of pain in the involved eye. Rubbing the eye could cause corneal abrasions.*

Nursing diagnoses and interventions

Interventions for managing pain and improving nutritional intake are addressed here; teaching to prevent injury following surgery is discussed in the following 'Meeting individualised needs' box.

Acute pain

The person with trigeminal neuralgia has excruciating pain and often avoids ADLs and socialising with others in an attempt to prevent the onset of pain. Pain management is fully discussed in Chapter 8. Nursing interventions for pain in people with this disorder focus on strategies for self-management.

- Identify factors that trigger an attack and discuss strategies to avoid these precipitating factors. *Most people can clearly identify trigger zones and triggering factors. Identification is the first step in pain control.*
- Determine usual response to pain. *Sensitivity and reaction to pain are influenced by previous experiences with pain and by age, gender, emotional factors and cultural background.*
- Assess factors that affect the ability to influence pain tolerance, including the knowledge and cause of the pain, the meaning of the pain, the ability to control the pain, cultural background and support systems. *Pain tolerance, which is the duration and intensity of pain a person is willing to endure, differs greatly between individuals and may also vary within particular people in different situations.*
- Monitor the effects of the medication prescribed for the neuralgia. *If the prescribed medication does not provide relief, other medications or methods of treatment may be used to control the pain.*

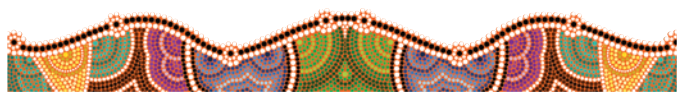
Risk of altered nutrition: less than body requirements

People often refuse to eat during periods of pain attacks, fearing that the movements of chewing may precipitate the pain. In addition, the chronic nature of the illness often causes depression, which may depress the appetite.

- Monitor dietary intake and weight at each visit and ask the person to keep a weekly weight record. *Ongoing assessments are necessary for early detection of nutritional deficiencies.*
- Discuss the temperature and consistency of foods eaten and suggest referral to a dietitian if necessary. *Hot or cold foods may trigger an attack; soft, warm or cool foods are less likely to act as triggers.*
- Suggest chewing on the unaffected side of the mouth. *Chewing on the unaffected side is less likely to trigger an attack of pain and so facilitate food intake.*
- If unable to tolerate oral food, tube feedings may be necessary. *Adequate kilojoules and nutrients for metabolic processes are essential.*

Community-based care

The person with trigeminal neuralgia who is receiving medical treatment and providing self-care at home requires teaching about the disease process, medication(s) being taken and ways to reduce the incidence of attacks or pain. Diet teaching and assistance with self-management of pain are also important. For example, if the home setting is draughty and attacks of pain are triggered by wind blowing across the face, it may be necessary to encourage the person to put weather stripping around windows and doors. To prevent injury to affected areas, the topics in the 'Meeting individualised needs' box below should be addressed.



THE PERSON WITH BELL'S PALSY

Bell's palsy, also called *facial paralysis*, is a disorder of the seventh cranial (facial) nerve, characterised by unilateral paralysis of the facial muscles. The facial nerve is

MEETING INDIVIDUALISED NEEDS Teaching for home care of trigeminal neuralgia

Eye care

- Do not rub the eyes; use artificial tears four times a day if the eyes are dry or irritated.
- Wear an eye patch at night.
- Wear protective sunglasses or goggles when outside, working in dusty areas, mowing the lawn and using any type of spray material (e.g. hair spray, cleaning materials, paint, insecticides).
- Remember to blink frequently.
- Check your eyes for redness or swelling each day.
- Schedule regular eye examinations.

Face and mouth care

- Chew on the unaffected side of the mouth.
- Avoid eating hot foods or drinking hot liquids.
- After every meal, brush your teeth and inspect the inside of your mouth for food that may collect between the gums and cheek.
- Have regular dental examinations; you will not be able to feel pain associated with gum infection or tooth decay.
- Use an electric razor to shave the face.
- Protect your face from very cold or windy conditions.

primarily a motor nerve that supplies all the muscles associated with expression on one side of the face. The sensory component innervates the anterior two-thirds of one side of the tongue.

This disorder can occur at any age but is seen most often in people aged between 15 and 60. The incidence is equal in men and women. The majority of people recover completely within 2 weeks to 6 months, and most recover without any treatment. Of those remaining, many recover some function but have some permanent facial paralysis; these people are usually older, have diabetes mellitus or have more severe manifestations, such as vertigo, a sensitivity to noise and deep head pain (NINDS, 2015b).

Pathophysiology

The exact cause of the disorder is unknown, although inflammation of the nerve and a relationship to the herpes simplex virus have been suggested (Papadakis & McPhee, 2015).

Manifestations

The onset of Bell's palsy is usually sudden and almost always involves one side of the face. Pain behind the ear or along the jaw may precede the paralysis. The person initially notices numbness or stiffness of one side of the face that distorts the appearance. As the disease progresses, the distortion becomes more obvious and the face appears asymmetrical. The facial paralysis causes the entire side of the face to droop and the person cannot wrinkle the forehead, close the eye or pucker the lips on the affected side (see Figure 43.7). When the person attempts to smile, the lower facial muscles are pulled to the opposite side of the face. Some people have only mild manifestations, whereas others have complete facial paralysis. People often believe they have had a stroke. Manifestations of Bell's palsy are listed in the 'Manifestations' box.

MANIFESTATIONS Bell's palsy

- Paralysis of the facial muscles on one side of the face
- Paralysis of the upper eyelid with loss of the corneal reflex on the affected side
- Loss or impairment of taste over the anterior portion of the tongue on the affected side
- Increased tearing from the lacrimal gland on the affected side

FIGURE 43.7 ■ The person with Bell's palsy shows the typical drooping of one side of the face

Source: © Dr P. Marazzi/Science Source.

INTERPROFESSIONAL CARE

There are no definitive laboratory or diagnostic tests for Bell's palsy, nor are there any specific treatments. Treatment includes medications and physiotherapy. Recent studies have shown that antiviral drugs such as aciclovir combined with an anti-inflammatory drug such as prednisone may be effective by limiting damage to the nerve. Physiotherapy to stimulate the facial nerve and help maintain muscle tone may help prevent permanent contractures before recovery takes place. Moist heat applied to the affected side of the face may decrease pain.

Nursing care

Although people provide self-care at home, the nurse plays a key role in teaching the person and their family about Bell's palsy and how to prevent injury and maintain nutrition. The person is often anxious about their appearance and may require counselling if any deficits in facial expression become permanent. The following topics should be addressed:

- Use artificial tears four times a day to lubricate the eye; wear an eye patch or tape the eye shut at night. Wear sunglasses or goggles when outside, working in dusty conditions and using any type of spray.
- Massage combined with warm, moist heat often is effective in relieving the pain.
- A soft diet that does not require chewing and six small meals a day are helpful. Chew slowly on the unaffected side and avoid hot foods. Clean the mouth and carefully inspect the area between the gums and cheek for food after each meal.
- As function returns, practise wrinkling the forehead, closing the eyes, blowing air out of the puckered mouth and whistling for 5 minutes three or four times a day.

DISORDERS RESULTING FROM INFECTIONS AND NEUROTOXINS

A variety of disorders of the nervous system may have infectious or toxic causes. Although these disorders are not common, those included here require significant nursing care when they do occur.

THE PERSON WITH CREUTZFELDT–JAKOB DISEASE

Creutzfeldt–Jakob disease (CJD) (also called *spongiform encephalopathy*) is a rapidly progressive, degenerative, neurological disease that causes brain degeneration without inflammation. The disease is transmissible and progressively fatal. The causative agent is believed to be an abnormal form of a cellular glycoprotein known as the prion protein. Transmission of the agent is by direct contamination with infected neural tissue, such as during eye and brain surgery. The injection of contaminated human growth hormone from cadaveric pituitaries has also been implicated.

A different form of the disease, called *variant CJD (vCJD)* is also a rare, degenerative, fatal brain disorder, but is not the same as the classic form of CJD. New variant CJD, referred to as 'mad cow disease', is believed to result from consumption of cattle products contaminated with bovine spongiform encephalopathy (BSE). This form primarily affects young adults. Because the illness is fatal and is associated with infected cattle, severe restrictions have been placed on the importation of cattle, sheep and goats, and on products from these animals, from countries in which BSE is known to exist.

Both forms of CJD occur worldwide, but clusters occur in several areas, more often in England, Chile and Italy. The incidence is approximately one case per 1 million Australians every year (NSW Health, 2012). Classic CJD affects adults over the age of 50; vCJD affects younger adults. The median age of death for people with classic CJD is 68 years. In contrast, the median age of death with vCJD is 28 years.

Pathophysiology

Creutzfeldt–Jakob disease is characterised by degeneration of the grey matter of the brain. The spongiform degeneration (involving the formation of tiny holes and resembling a sponge) produces severe dementia, myoclonus (muscle contractions) and characteristic changes in brain waves. On autopsy or biopsy of brain tissue, the brain shows loss of neurons and a proliferation of astrocytes (indicating destruction of nearby neurons).

Manifestations

The disease has characteristic stages and manifestations. The onset is characterised by memory changes, an exaggerated startle reflex, sleep disturbances and nervousness. The person then experiences rapid deterioration in motor, sensory and language function. Tremors, hyperreflexia, rigidity and a positive Babinski reflex are often present, and confusion progresses to dementia in almost all cases. People in the terminal state are comatose and exhibit decorticate and decerebrate posturing. The median duration of illness for CJD is 4 to 5 months; the median duration of illness for vCJD is 13 to 14 months (Centers for Disease Control and Prevention (CDC), 2015).

INTERPROFESSIONAL CARE

No specific treatment is available to stop or slow the progression of CJD. Collaborative interventions focus on the disease's manifestations. The disease is diagnosed by a thorough neurological examination, specific EEG changes and a CT scan. However, the final diagnosis of CJD can be made only by postmortem examination. It is often difficult to differentiate this disease from Alzheimer's disease, especially in the early stages.

Nursing care

The nurse may identify the manifestations of CJD when conducting a health history and total physical assessment. Include questions about familial history, cultural and geographical risk, and high-risk occupations or procedures in the history. Assessment of mental function, reflexes and cranial nerve function may provide information to assist in diagnosis.

Nursing care focuses on maximising comfort, preventing injury, preventing transmission and providing support. The following guidelines are useful in designing the plan of care:

- Although comfort is difficult to assess in people with impaired cognitive function, interventions that provide a quiet environment and analgesia are important.
- Communication is essential, even if the person is unable to respond.
- Institute seizure precautions and pad side rails.
- Provide skin care, changes in position and pressure-relief mattresses to decrease the risk of pressure ulcers, venous stasis and pneumonia.
- Use standard precautions for blood and body fluids when providing care. Disinfect surfaces with a solution of 5% bleach. Sterilise contaminated equipment by autoclave or soak in 5% bleach solution for 1 hour. Label all specimens as biohazardous. Teach staff members and family members guidelines for care, including careful handwashing. However, it is not necessary to place the person in isolation.
- Provide time for family members to verbalise grief and loss, which may be manifested as anger and frustration with the healthcare system.
- Provide information to family members about all procedures and the plan of care.
- Refer family members to sources of support, such as social services and the appropriate clergy.

THE PERSON WITH POSTPOLIOMYELITIS SYNDROME

Postpoliomyelitis syndrome is a complication of a previous infection by the poliomyelitis virus. This disease was epidemic in the 1940s and 1950s, but has largely been eradicated through immunisation with oral live trivalent virus vaccine. It is estimated that up to 4 million Australians were affected by poliomyelitis (Polio NSW, 2013). These people have struggled for years to rehabilitate themselves and lead productive lives. Now, as they reach retirement age, they are again experiencing manifestations which may be physically and psychologically incapacitating.

The poliomyelitis virus destroys some of the motor cells of the anterior horn cells of the spinal cord, causing neuromuscular effects that range from mild to severe flaccid paralysis and atrophy. The primary cause of death is respiratory arrest (Papadakis & McPhee, 2015).

Manifestations of motor neuron degeneration and weakness may emerge 10 to 40 years after the initial infection. Most people with postpoliomyelitis syndrome initially had a more severe case of polio and required hospitalisation, contracted the disease after the age of 10, required ventilator assistance for respiration and had paralysis in all four extremities. The incidence is slightly higher in women. As the population ages, it is projected that the number of older adults with postpoliomyelitis syndrome will increase.

Pathophysiology

The pathophysiological process in postpoliomyelitis syndrome is not known.

Manifestations

The manifestations of postpoliomyelitis syndrome include fatigue, muscle and joint weakness, loss of muscle mass, respiratory difficulties and pain. The manifestations are most often seen in muscles affected by the initial infection, but new muscle groups may also be affected. In addition to neuromuscular manifestations, the person may experience cold intolerance, dizziness, headaches, urinary incontinence and sleep disorders.

INTERPROFESSIONAL CARE

Postpoliomyelitis syndrome is diagnosed by a previous history of polio and the current manifestations. Diagnostic studies of nerve conduction, muscle strength and pulmonary function determine current physical status. Treatment addresses the manifestations and often involves physical physiotherapy and pulmonary rehabilitation programs.

Nursing care

The person with postpoliomyelitis syndrome faces the challenge of unexpected physical changes. People are often anxious about how others will react or what the future holds. Respiratory

dysfunction may result in the need for oxygen. Muscular weakness and decreased pulmonary function may make walking difficult, if not impossible. ADLs, independent self-care and careers are threatened.

Many people have not fully recovered psychologically from having polio and may respond to a recurrence of manifestations with denial and disbelief. Older people may not know they had polio as children. Nurses are responsible for assessing and identifying the manifestations of postpoliomyelitis syndrome. It is essential to question middle to older adults about a past history of polio when conducting the health history and to ask specific questions about manifestations that the person may be experiencing.

The nurse individualises teaching to meet the physical and psychosocial needs of the person and their family. The nurse should provide candid explanations and teach the person how to prevent fatigue, promote optimal respiratory function, meet self-care needs, modify ADLs and maintain safety. Follow-up care with nurses, doctors, physiotherapists, respiratory therapists and counsellors is indicated. Referral to a support group can make a positive difference in the person's and family's ability to cope with the disorder.

THE PERSON WITH RABIES

Rabies is a rhabdovirus infection of the central nervous system transmitted by infected saliva that enters the human body through a bite or an open wound. It is a fatal viral encephalitis that causes tens of thousands of deaths worldwide each year, with more than 15 million people worldwide receiving post-exposure vaccination (World Health Organization (WHO), 2015). Rabies does not currently occur in Australia, but a rabies-like virus, called the lyssavirus (which is lethal to humans), is carried by Australian animals such as bats and flying foxes; therefore, understanding the effects remains important to Australians (NSW Health, 2013). Rabies is a critical illness that almost always causes death if untreated. The rabies virus is carried by both wild and domestic animals, including bats, skunks, foxes, raccoons, cats and dogs. After an incubation period that may last from 10 days to many years (the norm is 3 to 7 weeks), the virus travels to the brain of the infected animal via the nerves. It multiplies and migrates to the salivary glands.

Pathophysiology

The person with rabies usually has a history of an animal bite but may also become infected through an abrasion or open wound that is exposed to the infected saliva. The virus spreads from the wound to local muscle cells and then invades the peripheral nerves. It eventually travels to the central nervous system. The incubation period in humans varies according to the severity and location of the bite. For example, bites on the face may result in manifestations in 10 days to a few weeks, whereas bites on the lower extremities may incubate for as long as 1 year.

Manifestations

The manifestations occur in stages. During the initial—or prodromal—stage, the site of the wound is painful and then exhibits various paraesthesias. The infected person is anxious, irritable and depressed. General manifestations of infection (such as headache, loss of appetite and sore throat) may appear. The person may also have increased sensitivity to light and sounds, and the skin is especially sensitive to changes in temperature.

The prodromal stage is followed by an excitement stage. The infected person has periods of excitement that alternate with periods of quiet. Attempts to drink cause such painful laryngospasms that the person refuses to drink (a phenomenon called *hydrophobia*). Large amounts of thick, tenacious mucus are present. The person experiences convulsions, muscle spasms and periods of apnoea. If untreated, death occurs approximately 7 days from the onset of manifestations and is usually due to respiratory failure.

INTERPROFESSIONAL CARE

Sick animals should be euthanised and their brains examined for presence of the rabies virus, which is detected by fluorescent antibody testing. The blood of an infected person can also be tested with the same diagnostic study to demonstrate the presence of rabies antibodies.

Nursing care

Nursing care for people with rabies is provided in a critical care unit, with the person in a quiet, darkened room to decrease stimulation as much as possible. The person requires interventions to maintain the airway, maintain oxygenation and control seizures. Standard precautions are essential, because the rabies virus is present in the saliva of the person. If an open wound of a healthcare provider is contaminated with infected saliva, the provider must receive post-exposure immunisations.

Health promotion

Personal and family teaching focuses on the importance of providing proper care of wounds, seeking immediate medical attention for animal bites and obtaining treatment after any suspicious bite.

Because the untreated disease is almost always fatal, the best intervention is prevention. Preventive activities include:

- Immunisation of those at risk of injury from bats (i.e. bat handlers, vets and wildlife officers).
- Local treatment of animal bites and scratches:
  - Carefully and thoroughly clean and flush wounds with soap and water to remove the saliva and dilute the viral exposure.
  - Immediately take the person with the bite for emergency treatment.

- Post-exposure care:
  - Human rabies immunoglobulin (RIG) is administered for passive immunisation. A dose of 20 units/kg of the immunoglobulin is infiltrated around the wound and the rest is administered intramuscularly. At the same time, an inactivated human diploid cell vaccine (HDCV) is administered intramuscularly, with 1 mL given on the day of exposure and on days 3, 7, 14 and 28 after exposure (WHO, 2013). HRIG and HDCV should never be given in the same syringe or at the same site. Local and mild systemic reactions include itching, tenderness, headaches, muscle aches and nausea.
  - If RIG is not available, equine rabies antiserum may be administered after testing the person for horse serum sensitivity.

THE PERSON WITH TETANUS

Tetanus, more commonly called *lockjaw*, is a disorder of the nervous system caused by a neurotoxin elaborated by *Clostridium tetani*. This anaerobic bacillus lives in the soil. Spores of the bacillus enter the body through open wounds contaminated with dirt, street dust or faeces (animal or human). The wounds may result from punctures, scratches or abrasions, bee stings, abortions, surgery, trauma, burns or intravenous drug use. Incidence is highest in people who have never been immunised, the very young who are not fully immunised and older adults whose immunity has been lost. Tetanus has a high mortality rate, with death occurring in approximately three in every 100 cases (Department of Health, 2015). Contaminated lesions of the head and face are more dangerous than those in other parts of the body.

Pathophysiology

When the spores of *C. tetani* enter the open wound, they germinate and produce a toxin called tetanospasmin. The incubation period averages 8 to 12 days but can range from 5 days to 15 weeks (Papadakis & McPhee, 2015). The toxins are absorbed by the peripheral nerves and carried to the spinal cord, where they block the action of inhibitory enzymes at spinal synapses and interfere with transmission of neuromuscular impulses. As a result, even minor stimuli cause uncontrolled muscle spasms.

Manifestations

The manifestations begin with pain at the site of the infection. The infected person has stiffness of the jaw and neck, and dysphagia. There is often profuse perspiration and drooling from increased salivation. As the infection progresses, the person experiences hyperreflexia, spasms of the jaw muscles (*trismus*) or facial muscles, and rigidity and spasms of the abdominal, neck and back muscles. Generalised tonic seizures are caused by even minor stimuli and the person assumes a typical opisthotonic position during the seizures: the head is retracted, the back is arched and the feet are extended. The muscle spasms are painful. The person may be unable to breathe from spasms of the glottis and respiratory muscles. Despite these physical effects, the person has no change in mental status.

The complications of tetanus include urinary retention and airway obstruction from the spasms. Cardiac and respiratory failure are late, life-threatening complications.

INTERPROFESSIONAL CARE

There are no specific diagnostic tests for tetanus; diagnosis is based on manifestations. Tetanus is completely preventable by active immunisation. Immunisation for children includes tetanus toxoid, administered as part of the diphtheria-pertussis-tetanus (DPT) immunisation series. In adults, immunisation is obtained by administering tetanus toxoid as two doses 4 to 6 weeks apart, with a third dose in 6 to 12 months. All individuals should have a booster dose every 10 years throughout life, or at the time of a major injury if the last booster dose was given more than 5 years prior to the injury.

If a wound is contaminated, or if the person's immunisation status is uncertain, passive immunisation with tetanus immune globulin is administered. Active immunisation with tetanus toxoid is begun at the same time. The wound is carefully and thoroughly debrided and antibiotics are administered.

The person with tetanus requires intensive care in an area of minimal stimulation. Penicillin is administered to help destroy the toxin-producing organism. Muscle spasms and seizures are controlled by chlorpromazine (Largactil) or diazepam (Valium), often combined with a sedative. Anticoagulants may be prescribed to prevent venous thrombosis. In severe cases, seizures and spasms are controlled with paralysis by a curare-like medication and airway obstruction is managed by mechanical ventilation.

Nursing care

Nursing care for the person with tetanus is intensive and focuses on assessments and interventions to promote safety, prevent injury, maintain nutrition and maintain pulmonary and cardiovascular function. The person usually requires in-hospital care for 2 to 5 weeks. The nursing care plan commonly includes the following:

- Place in a quiet, darkened room to decrease stimuli that cause muscle spasms and seizures.
- Provide only necessary physical care and do so during periods of maximal sedation to decrease tactile stimulation that causes muscle spasms.
- Maintain oxygenation through mechanical ventilator and frequent suctioning of secretions.
- Maintain intravenous access for the administration of fluids and medications.
- Administer prescribed antibiotics, anticonvulsants and sedatives. In the case of cardiovascular complications, administer prescribed beta-adrenergic blocking agents such as propranolol (Inderal).

- Provide adequate nutrition through prescribed nutritional support, such as total parenteral nutrition.
- Monitor respiratory and cardiovascular status and provide immediate interventions for respiratory or cardiovascular failure.
- Monitor fluid and electrolyte status. Ensure adequate fluid intake to maintain hydration and urinary output.
- Monitor urinary output, which should be maintained at 1.5 to 2 L per day.
- Monitor for the hazards of immobility, including constipation, pneumonia, deep venous thrombosis and pressure ulcers.

Health promotion

Tetanus is a preventable disorder and nurses have a major role in promoting immunisations for all children and for educating adults about the need for booster doses. The older population is especially at risk of never having been immunised or for letting immunisations lapse. Information for this age group can be provided through activities such as community health fairs and programs at senior citizen groups.

It is also necessary to teach the proper care of wounds. All wounds, no matter how small, should be thoroughly washed with soap and water. All foreign material should be carefully flushed out or removed from a wound, and medical care should be sought for wounds that are more extensive or contaminated.

THE PERSON WITH BOTULISM

Botulism is food poisoning caused by ingestion of food contaminated with a toxin produced by the bacillus *Clostridium botulinum*. This anaerobic spore-forming bacillus is found in the soil. Most cases of botulism occur from eating improperly canned or cooked foods, especially home-canned vegetables and fruits, smoked meats and vacuum-packed fish. The mortality rate is high if the disease is untreated.

Pathophysiology

The toxins liberated by *C. botulinum* are absorbed by the gastrointestinal tract and bound to nerve tissues. They block the release of acetylcholine from nerve endings and thus cause respiratory paralysis due to paralysis of skeletal muscles.

Manifestations

Manifestations appear 12 to 36 hours after ingestion of the contaminated food. They usually begin with visual disturbances such as diplopia, loss of accommodation and fixed, dilated pupils. Ptosis is often present. Gastrointestinal manifestations include nausea and vomiting, diarrhoea, dysphagia and dry mouth. Involvement of the larynx is manifested by dystonia (impaired muscle tone). Paralysis of all muscle groups progresses throughout the body, with respiratory paralysis causing death if the person is not placed on a mechanical ventilator. There is no effect on mental status.

INTERPROFESSIONAL CARE

Infection with the *Clostridium* toxin is verified by laboratory analysis of the serum and stool and of suspected food, if possible. If botulism is suspected, the local Public Health Unit should be notified for assistance with laboratory assays and procuring botulism antitoxin. All people who may have eaten the contaminated food must be located and observed.

Any toxins in the gastrointestinal system are removed by cathartics, enemas and gastric lavage. The person with respiratory paralysis is placed on a mechanical ventilator and may require a tracheostomy. Botulism antitoxin is administered to eradicate toxins in the circulation. Nutritional support is often provided with total parenteral nutrition. Intravenous fluids are administered to prevent dehydration and renal failure. If ventilation can be maintained, the person often recovers without further neurological deficits.

Nursing care

The person with botulism is hospitalised and interventions focus on monitoring for respiratory failure and providing ventilatory assistance if necessary. Ongoing assessments are also made for manifestations of paralytic ileus and urinary retention. The person will be nil by mouth until able to swallow and breathe; therefore, hydration and nutritional status are monitored. Teach the person and their family that fatigue and weakness may persist for up to a year. During this time, the person may need to modify ADLs and take rest periods throughout the day.

Health promotion

Education of the public to prevent botulism is important. Address the following topics at health fairs and community programs:

- Home-canned foods must be processed in a pressure cooker rather than in boiling water because the organism is difficult to kill.
- Do not eat home-processed foods that have a change in colour, are soft, contain gas bubbles or have a bad odour.
- Always heat both home-processed and commercial foods at temperatures over 120°C or boil for 10 minutes before tasting or eating them.
- Discard home-processed or commercially canned or bottled foods with defective seals.
- Discard commercially prepared canned foods that are damaged or have bulging sides or leaking contents.

CHAPTER HIGHLIGHTS

- Alzheimer's disease (AD) is a form of dementia (a disease of the brain) of older adults with progressive irreversible deterioration of general intellectual functioning. The disease is characterised by atrophy of brain tissue, loss of neurons, neurofibrillary tangles and amyloid plaques. AD finally leaves the person unable to communicate, maintain continence and recognise self or others. Caregivers need much teaching and support not only to provide care but also to avoid caregiver burden.
- Multiple sclerosis (MS) is a chronic demyelinating neurological disease of the CNS (brain, optic nerves and spinal cord). Occurring in young to middle-aged adults, it is believed to be due to an autoimmune response to a prior viral infection. The loss of myelin leads to axon dysfunction, which slows and distorts nerve impulses. Medications (ACTH, immunosuppressive agents, interferon and glatiramer acetate) are used to slow the progression of the disease, decrease the number of exacerbations and treat manifestations.
- Parkinson's disease (PD) is a progressive degenerative neurological disease characterised by tremor, muscle rigidity and bradykinesia. The loss of voluntary motor control is the result of pathological processes resulting in a decrease of dopamine (an inhibitory neurotransmitter) so that it can no longer inhibit acetylcholine (an excitatory neurotransmitter). Medications are used to treat manifestations and include MAO inhibitors, dopaminergics, dopamine agonists and anticholinergics. Other treatments include deep brain stimulation and surgery.
- Huntington's disease (HD) (chorea) is a progressive, degenerative, inherited neurological disease characterised by increasing dementia and chorea. There is an excess of dopamine, causing excessive, uncontrolled movement.
- Motor neurone disease (MND) is a rapidly progressive and fatal degenerative disease characterised by weakness and wasting of voluntary control muscles, but without sensory or cognitive changes. The person eventually loses the ability to communicate and breathe. Riluzole, antiglutamate medication, is used to treat (but not cure) manifestations.
- Myasthenia gravis (MG) is a chronic autoimmune peripheral nervous system disorder characterised by fatigue and severe skeletal muscle weakness. It results from a decreased number of acetylcholine receptors at the neuromuscular junction, so muscles are unable to contract. Life-threatening emergencies include myasthenic crisis (sudden increase in motor weakness) and cholinergic crisis (from an overdose of the anticholinesterase medications used to treat MG).
- Guillain–Barré syndrome (GBS) is an acute inflammatory demyelinating disease of the peripheral nervous system characterised by an acute onset of flaccid motor paralysis that begins in the lower extremities and ascends to involve the upper extremities, torso and cranial nerves. Paralysis of intercostal and diaphragmatic muscles often necessitates ventilatory assistance. The acute phase lasts from 1 to 3 weeks, followed by recovery, which takes from 6 months to 2 years.
- Cranial nerve disorders include trigeminal neuralgia (tic douloureux) and Bell's palsy. Trigeminal neuralgia is a chronic disorder of cranial nerve V and causes severe facial pain. Bell's palsy is an acute disorder of cranial nerve VII, characterised by unilateral paralysis of the facial muscles. People provide self-care at home and require teaching to help prevent complications.
- Neurological disorders resulting from neurotoxins or viruses include Creutzfeldt–Jakob disease, postpoliomyelitis syndrome, rabies, tetanus and botulism. Community-based health education can not only provide information but can also prevent illness in the case of rabies, tetanus and botulism.

CONCEPT CHECK

1 Which of the following statements is true of dementia? (Choose all that apply.)
1. Dementia is a general term used to describe manifestations of damage or death of neurons.
2. Dementia is the term used to describe the cognitive and behavioural manifestations of AD.
3. Dementia is an acute disorder resulting from an injury to the brain.
4. Dementia is the primary manifestation of Guillian–Barré syndrome.

2 Which manifestation is usually the first indication of the onset of AD?
1. inability to perform ADLs
2. sundowning at night
3. subtle memory deficits
4. inability to communicate

3 Which of the following nursing diagnoses is appropriate for people with MS, regardless of type or severity?
1. *Fatigue*
2. *Risk of aspiration*
3. *Acute pain*
4. *Impaired gas exchange*

4 You are preparing information to teach a person about medications for MS. Which drugs would you expect to be used?
1. antibiotics
2. antihistamines
3. interferon
4. levodopa

5 Which of the following topics is important when teaching a young adult with MS?
1. how to prevent sexually transmitted infections
2. how pregnancy can improve manifestations
3. what can be done to cure the disease
4. why it is important to avoid extremes of heat and cold

6 The manifestations of Parkinson's disease are the result of:
1. autoimmune responses to a viral infection
2. the failure of dopamine to inhibit acetylcholine
3. effects of a neurotoxin
4. a genetic defect

7 When teaching care at home for the person with PD, what would be a major area to discuss with caregivers?
1. preventing an overdose of medications
2. avoiding daily baths and showers
3. preventing falling
4. increasing appetite

8 Which drug classification is the medication used to treat MND?
1 dopamine agonist
2 anticholinergic
3 anti-inflammatory
4 antiglutamate

9 You are preparing a teaching plan for a person with Bell's palsy. What information would you include?
1 'You will experience severe facial pain during attacks.'
2 'The disease affects your muscles so you can't walk.'
3 'One side of your face will not move normally.'
4 'Be sure to boil all home-canned foods before eating them.'

10 How can the nurse prevent tetanus?
1 Teach safe food preparation techniques.
2 Promote immunisation for adults and children.
3 Demonstrate proper disposal of soiled dressings.
4 Promote immunisation of household pets.

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UNIT 11 BUILDING CLINICAL COMPETENCE

Responses to altered neurological function

CLINICAL SCENARIO

You have been assigned to work with the following people on a neurological hospital unit. Significant data obtained during report are as follows:

- Sam Kwon is a 74-year-old male who was admitted from the emergency department to the neurology ward last night. Mr Kwon was found by family in his home. He was unable to speak or move the right side of his body and continues to experience paralysis on the right side in both his arm and his leg. He is aphasic with facial drooping, but is able to nod his head to communicate. Mr Kwon has a known history of hypertension, CHF and type 2 diabetes controlled by oral hypoglycaemic medications. He has smoked a packet of cigarettes a day for 40 years. BP is now 140/105, P 98, R 24. Lungs reported to have crackles in the bases. He is receiving 2 L of oxygen via nasal prongs. A swallowing evaluation has been ordered to be done today.
- Jane Thomas is a 56-year-old female. She was admitted 4 days ago and underwent a lumbar laminectomy. She was able to ambulate with assistance on day 2 after surgery and now reports no dizziness on ambulation. Her leg strength has progressively increased since surgery. She has equal sensation and is able to wriggle her toes on both feet. Pain is controlled at her desired level of 2 to 3 on a scale of 1 to 10 with oral medications.
- Cesar Phillips is a 39-year-old male, admitted 4 days ago after experiencing paralysis and severe headache at work. He has been diagnosed with stroke following an MRI. He has a history of smoking one packet of cigarettes per day and hypertension. His wife reported that he had stopped taking his blood pressure medication due to the side effects of impotence. Mr Phillips is awake and alert, but has exhibited impulsive behaviour and agnosia. He also has a difficult time remembering words and neglects the left side of his body in ADLs. BP had been 130/85 at the beginning of last shift but has increased to 170/110. He received an additional prn blood pressure medication 1.5 hours ago.
- Tonya Walton is a 29-year-old female. She was involved in a car crash 12 hours before her admission 5 days earlier. She developed a headache, drowsiness, confusion and pupil enlargement several hours after the crash. Following an MRI, Ms Walton underwent intracranial surgery to evacuate a subdural haematoma. She has been complaining of a headache that has increased in intensity from a 2 to a 6 on a scale of 1 to 10 on the previous shift. She received two Nurofen ES tabs 1 hour ago. BP was 140–150 systolic and 90–100 diastolic for most of the shift. Her BP at the time of the Nurofen administration was 175/115. She has not experienced any changes in her neuro assessment except for the increasing headache and blood pressure elevation.

Critical thinking questions

1 In what order would you visit these people after report?

1. _____
2. _____
3. _____
4. _____

2 Which two top-priority nursing diagnoses would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Sam Kwon		
Jane Thomas		
Cesar Phillips		
Tonya Walton		

3 After completing Mr Kwon's beginning-of-shift baseline assessment, which assessment would be your next priority?

1. rhythm strip interpretation
2. intake and output
3. heart sounds
4. level of pain

4 You are completing the beginning-of-shift assessment for Mr Phillips. Which changes would best indicate that the plan initiated by the previous shift was effective?

1. Mr Phillips no longer has a craving for cigarettes.
2. Mr Phillips's respiratory rate is 18–20.
3. Mr Phillips's pain score is now 1–2.
4. Mr Phillips's blood pressure has decreased to 145/90.

5 What are the top two education priorities for Jane Thomas as you plan for discharge? (Select all that apply.)

1. appropriate exercise
2. pain management
3. weight control
4. incision care
5. importance of rest

6 You review the lab reports for Jane Thomas. You are most interested in the results for which of the following studies? (Select all that apply.)

1. white blood cell count
2. red blood cell indices
3. serum sodium
4. haemoglobin
5. haematocrit
6. osteocalcin
7. prealbumin

7 You are completing the beginning-of-shift assessment for Ms Walton. Which changes would best indicate that the plan initiated by the previous shift was effective?

1. Ms Walton has no neurological changes or deterioration.
2. Ms Walton's BP is 142/94.
3. Ms Walton's pain score is now 1–2.
4. Ms Walton's dressing is dry and intact.

8 If you re-evaluated Ms Walton and her headache pain had not decreased, what should be your next priority?

1. Reposition Ms Walton on her left side with the head of the bed flat.
2. Call the doctor about Ms Walton's report of increasing headache.
3. Decrease stimuli in the room by darkening the room and limiting noise.
4. Check the prn orders to evaluate if a stronger pain medication has been ordered.

9 If you were planning an effective team meeting with all disciplines that should be collaborating to provide care and a discharge plan for Mr Phillips, which six disciplines should be represented and why?

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____

10 Review the five steps of the abbreviated neurological assessment as outlined in the text. What are three findings you would expect when conducting a neuro check with Mr Kwon?

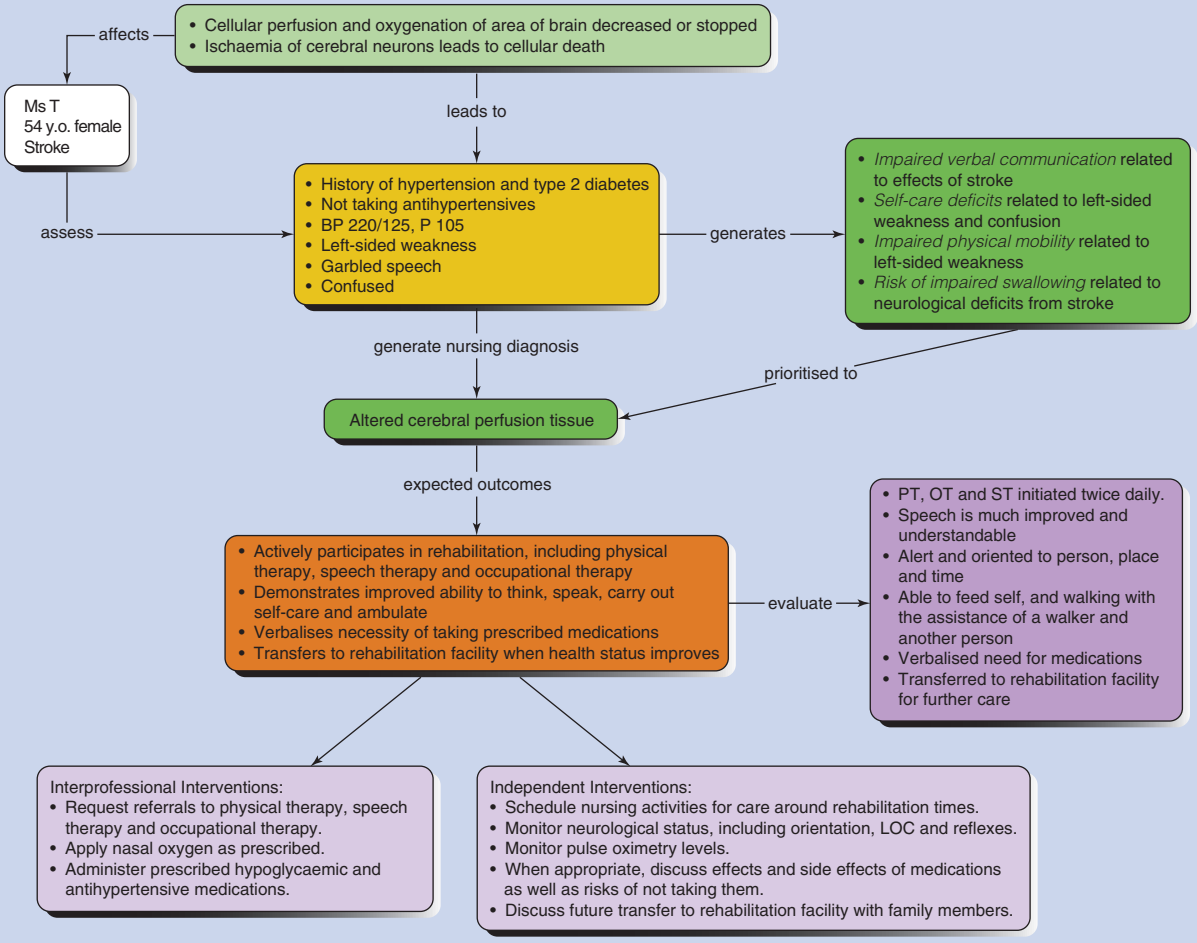
1. _____
2. _____
3. _____

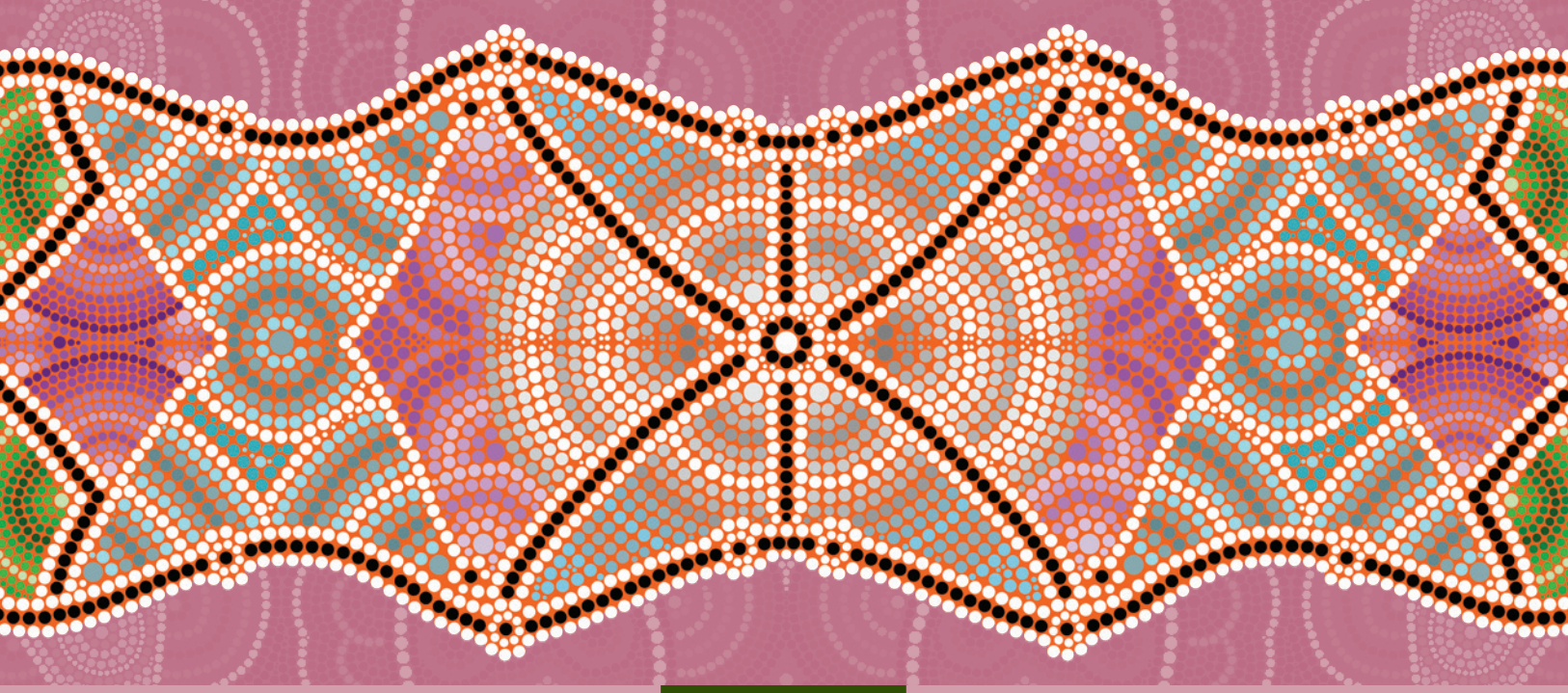
CASE STUDY

Shirley Tayler is a 54-year-old Indigenous Australian female. She was admitted 2 days earlier after being diagnosed with a stroke. She lives with her husband of 34 years and her 14-year-old grandson. Her grandson found her lying on the kitchen floor when he got home from school. When he asked her what had happened, she was unable to say words that were clear or understandable. He tried to help her stand up, but was unable to because she was extremely weak in her left side. He called 000 and an ambulance transported her to the emergency department. Her blood pressure when the ambulance arrived was 220/125, P 105, R 24, and she was very anxious and spoke garbled words loudly at the paramedics. She was diagnosed with acute stroke after evaluation at the hospital and was admitted for treatment. Mrs Tayler has a history of hypertension and type 2 diabetes. She recently stopped taking her blood pressure medication because it made her feel 'tired'.

The pathophysiology of a stroke is dependent on location of the causative factor, size of tissue affected and time of loss of cellular perfusion and oxygenation. Time is critical when it involves compromised perfusion to any organ and the brain is no exception. Cerebral neuron cellular metabolism is severely affected within 4 to 5 minutes of compromised perfusion. Compromised cellular metabolism leads to glucose, glycogen and adenosine triphosphate depletion and sodium–potassium pump failure. Wherever sodium goes, water follows. When the sodium–potassium pump fails, the sodium in the cell draws water into the cell, resulting in oedema. Additionally, cerebral vessel walls also swell, further compromising blood and oxygen supply. Severe or prolonged ischaemia leads to cellular death. The manifestations of a stroke include, but are not limited to, motor deficits, elimination disorders, sensory–perceptual deficits, communication disorders and behavioural changes. The manifestations are always sudden in onset, focal and usually unilateral. Typically, a stroke is manifested by weakness of the face, arm and leg; loss of vision in one eye; speech and swallowing problems; and difficulties with balance. There are many different complications of a stroke, including increased intracranial pressure, coma, death, chronic long-term confusion and intellectual changes.

The nursing diagnosis of *Ineffective cerebral tissue perfusion* is appropriate for implementing care for Mrs Tayler.





UNIT 12

RESPONSES TO ALTERED VISUAL AND AUDITORY FUNCTION

CHAPTER 44

A PERSON-CENTRED APPROACH TO ASSESSING THE EYE AND EAR

CHAPTER 45

NURSING CARE OF PEOPLE WITH EYE AND EAR DISORDERS

CHAPTER 44

A PERSON-CENTRED APPROACH TO ASSESSING THE EYE AND EAR

KAMAREE BERRY

KEY TERMS

accommodation 1663
cerumen 1672
convergence 1664
corneal reflex 1661
hyperopia 1668
myopia 1668
nystagmus 1668
presbyopia 1668
ptosis 1669
pupillary light reflex 1662
refraction 1663

LEARNING OUTCOMES

- Describe the anatomy, physiology and functions of the eye and ear.
- Explain the physiological processes involved in vision, hearing and equilibrium.
- Identify specific topics for consideration during a health history interview of the person with health problems of the eye or ear.
- Describe normal variations in assessment findings for the older adult.
- Identify abnormal findings that may indicate impairment in the function of the eye and the ear.

CLINICAL COMPETENCIES

- Conduct and document a health history for people having or at risk of alterations in the structure or functions of the eye and ear.
- Monitor the results of diagnostic tests and report abnormal findings.
- Conduct and document a physical assessment of the structure and/or functions of the eye and ear.

EQUIPMENT NEEDED

- Visual acuity charts
- Opaque eye cover
- Pen
- Penlight
- Cotton-tipped applicator
- Ophthalmoscope
- Otoscope
- Tuning fork

Vision and hearing allow us to experience the world in which we live. The eyes and ears provide pathways for visual and auditory stimuli to reach the brain, while specialised structures within the ear help maintain position sense and equilibrium. Deficits in vision and hearing may limit self-care, mobility, safety, independence, communication and relationships with others.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE EYES

The eyes are complex structures that contain 70% of the sensory receptors of the body.

Each eye is a sphere measuring about 2.5 cm in diameter, surrounded and protected by a bony orbit and cushions of fat. The primary functions of the eye are to encode the patterns of light from the environment through photoreceptors and to carry the coded information from the eyes to the brain. The brain gives meaning to the coded information, allowing us to make sense of what we see. Both extraocular and intraocular structures are considered parts of the eye.

Extraocular structures

Although the extraocular structures of the eye are outside the eyeball, they are vital to its protection. These structures are the eyebrows, eyelids, eyelashes, conjunctiva, lacrimal apparatus and extrinsic eye muscles (see Figure 44.1).

The eyebrows shade the eyes and keep perspiration away from them. The eyelids are thin, loose folds of skin covering the anterior eye; they protect the eyes from foreign bodies, regulate the entry of light into the eye and distribute tears by blinking. The eyelashes are short hairs that project from the top and bottom borders of the eyelids. An unexpected touch to the eyelashes initiates the blinking reflex to protect the eyes from foreign objects.

The conjunctiva is a thin, transparent membrane that lines the inner surfaces of the eyelids and folds over the anterior surface of the eyeball. The palpebral conjunctiva lines the upper and lower eyelids, whereas the bulbar conjunctiva loosely covers the anterior sclera (the white part of the eye). The conjunctiva is a mucous membrane that lubricates the eyes. The lacrimal apparatus is composed of the lacrimal gland, the puncta, the lacrimal sac and the nasolacrimal duct. Together, these structures secrete, distribute and drain tears to cleanse and moisten the eye's surface.

The six extrinsic eye muscles control movement of the eye, allowing it to follow a moving object and move precisely. The muscles also help maintain the shape of the eyeball. The cranial nerves control the extrinsic muscles (see Figure 44.2).

Intraocular structures

The intraocular structures transmit visual images and maintain homeostasis of the inner eye. Those in the anterior portion of each eyeball are the sclera and the cornea (forming the outermost coat of the eye, known as the fibrous tunic), the iris, the pupil and the anterior cavity (see Figure 44.3).

Sclera and cornea

The white sclera lines the outside of the eyeball it protects and provides its shape. The sclera gives way to the cornea over the iris and pupil which is transparent, avascular and sensitive to touch. The cornea forms a window that allows light to enter the eye and is a part of its light-bending apparatus. When the cornea is touched, the eyelids blink (**corneal reflex**) and tears are secreted.

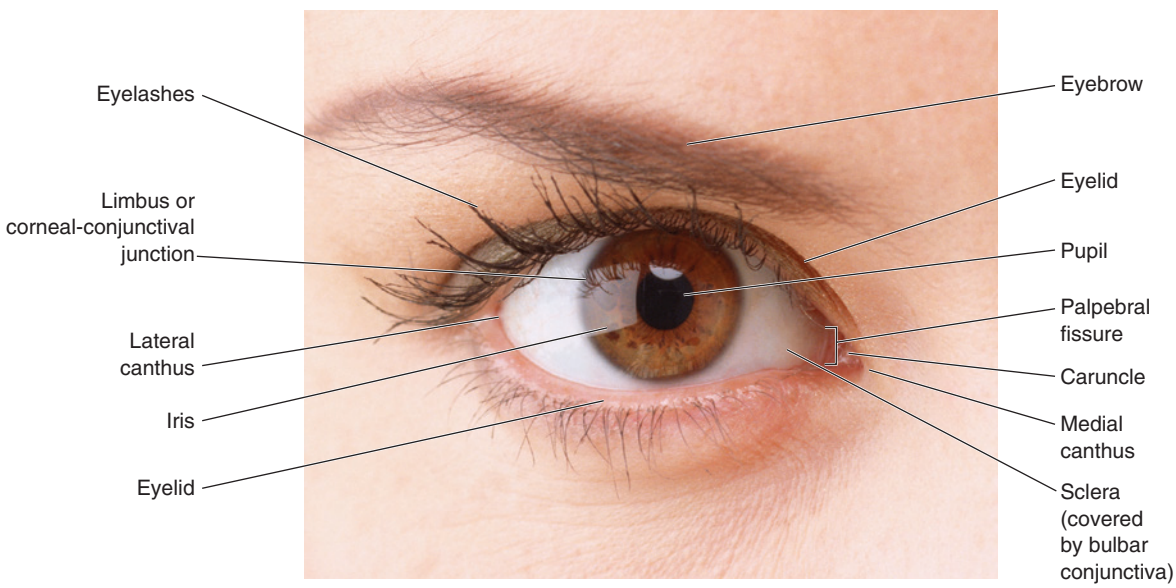
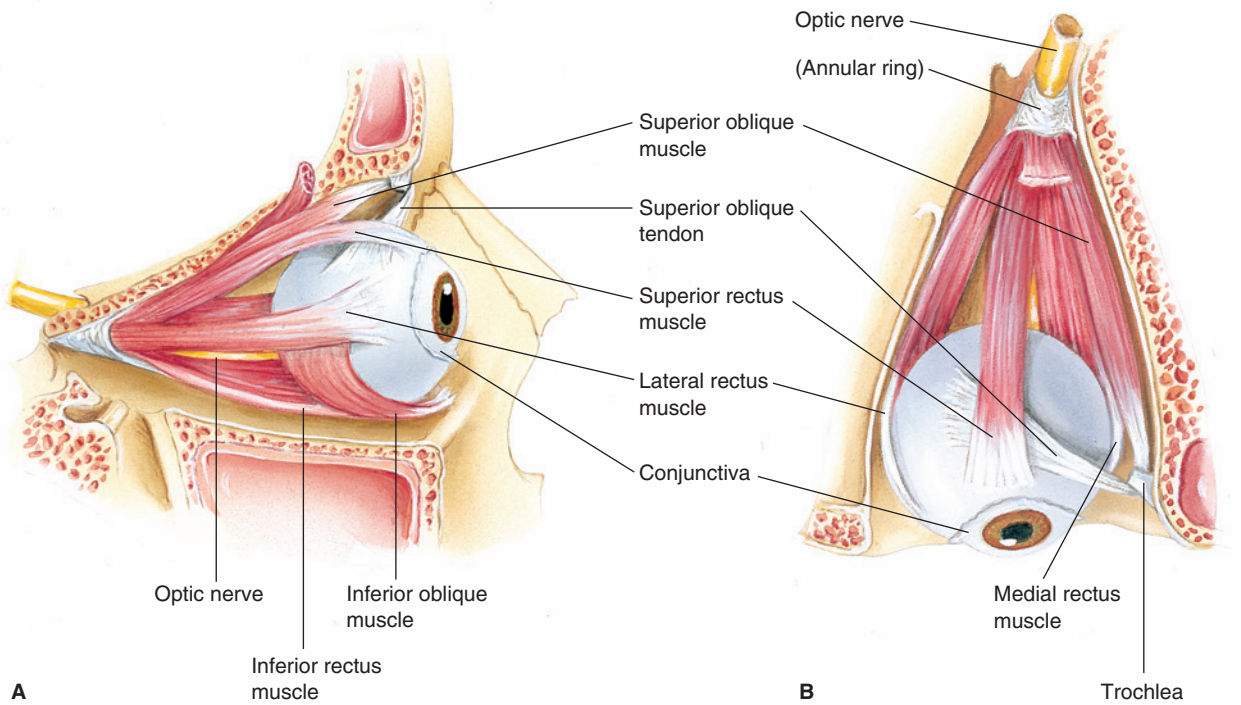


FIGURE 44.1 ■ Accessory and external structures of the eye

Source: © Sozaijiten



C

Name	Controlling cranial nerve	Action
Lateral rectus	VI (abducens)	Moves eye laterally
Medial rectus	III (oculomotor)	Moves eye medially
Superior rectus	III (oculomotor)	Elevates eye or rolls it superiorly
Inferior rectus	III (oculomotor)	Depresses eye or rolls it inferiorly
Inferior oblique	III (oculomotor)	Elevates eye and turns it laterally
Superior oblique	IV (trochlear)	Depresses eye and turns it laterally

FIGURE 44.2 ■ Extraocular muscles. *A*, Lateral view of the right eye. *B,* Superior view of the right eye. *C*, Innervation of the extraocular muscles by the cranial nerves

Iris

The iris is a disc of muscle surrounding the pupil and lying between the cornea and the lens. The iris gives the eye its colour and regulates light entry by controlling the size of the pupil. The pupil is the dark centre of the eye through which light enters. The pupil constricts when bright light enters the eye and when it is used for near vision; it dilates when light conditions are dim and when the eye is used for distance vision. In response to intense light, the pupil constricts rapidly in the **pupillary light reflex**.

Aqueous fluid

The anterior cavity is made of the anterior chamber (the space between the cornea and the iris) and the posterior chamber (the space between the iris and the lens). The anterior cavity is filled with aqueous humour, a clear fluid which constantly forms and drains to maintain a relatively constant pressure of 15 to 20 mmHg in the eye. The canal of Schlemm, a network of channels that circles the eye in the angle at the junction of the sclera and the cornea, is the drainage system for fluid moving between the anterior and posterior chambers. Aqueous humour provides nutrients and oxygen to the cornea and the lens.

Internal chamber

The intraocular structures that lie in the internal chamber of the eye are the lens, posterior cavity, vitreous humour, ciliary body, uvea and retina.

The lens is a biconvex, avascular, transparent structure located directly behind the pupil. It can change shape to focus and refract light onto the retina. The posterior cavity lies behind the lens. It is filled with a clear gelatinous substance, the vitreous humour, which supports the posterior surface of the lens, maintains the position of the retina and transmits light. The uvea, also called the vascular tunic, is the middle layer of the eyeball. This pigmented layer has three components: the iris, ciliary body and choroid. The ciliary body encircles the lens and, along with the iris, regulates the amount of light reaching the retina by controlling the shape of the lens. Most of

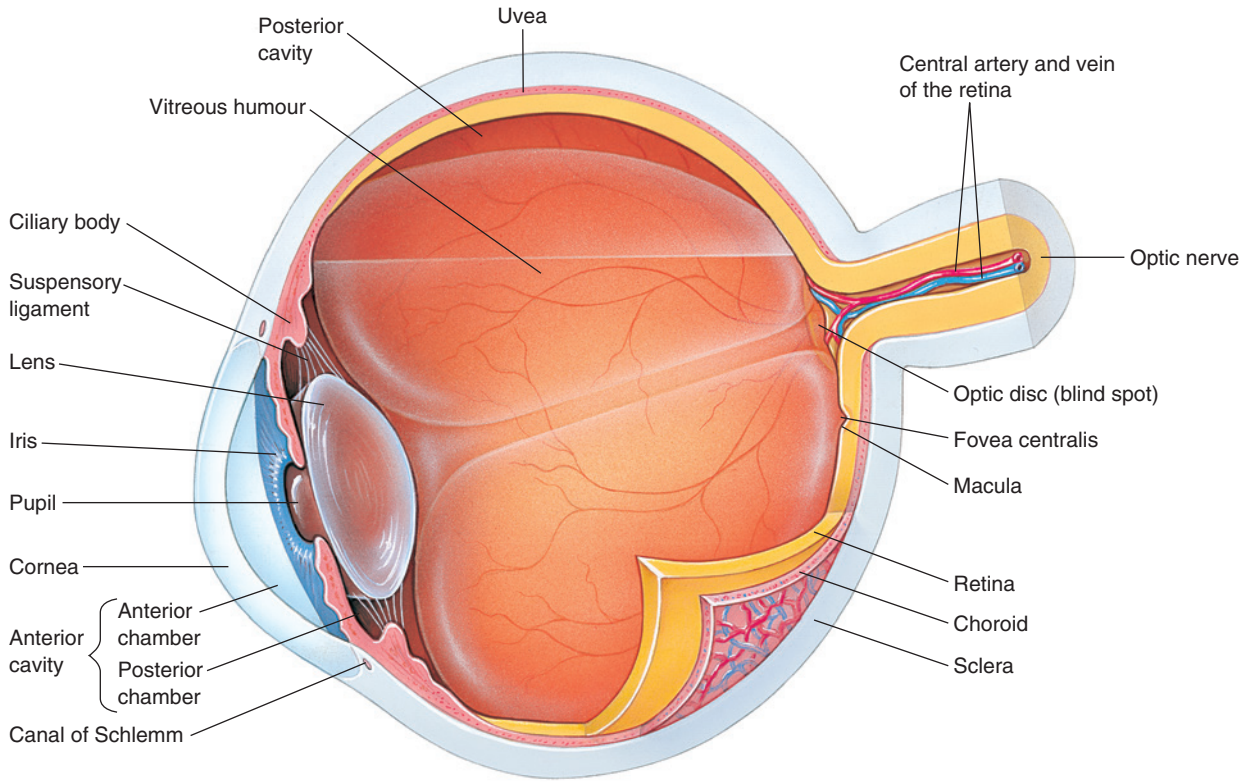


FIGURE 44.3 ■ Internal structures of the eye

the uvea is made up of the choroid, which is pigmented and vascular. Blood vessels of the choroid nourish the layers of the eyeball and its pigmented areas absorb light, preventing it from scattering within the eyeball.

The retina is the innermost lining of the eyeball. It has an outer pigmented layer and an inner neural layer. The outer layer, next to the choroid, serves as the link between visual stimuli and the brain. The transparent inner layer is made up of millions of light receptors in structures called rods and cones. Rods enable vision in dim light as well as peripheral vision and cones enable vision in bright light and the perception of colour. The optic disc, a cream-coloured round or oval area within the retina, is the point at which the optic nerve enters the eye. The slight depression in the centre of the optic disc is called the physiological cup. Located laterally to the optic disc is the macula, a darker area with no visible blood vessels and which contains primarily cones. The fovea centralis is a slight depression in the centre of the macula that contains only cones and is a main receptor of detailed colour vision.

The visual pathway

The optic nerves are cranial nerves formed of the axons of ganglion cells. The two optic nerves meet at the optic chiasma, just anterior to the pituitary gland in the brain. At the optic chiasma, axons from the medial half of each retina cross to the opposite side to form pairs of axons from each eye. These pairs continue as the left and right optic tracts (see Figure 44.4). The crossing of the axons results in each optic tract carrying information from both eyes. The left optic tract carries visual information from the lateral half of the retina of the left eye and the medial half of the retina of the right eye, whereas the right optic tract carries visual information from the lateral half of the retina of the right eye and the medial half of the retina of the left eye.

The ganglion cell axons in the optic tracts travel to the thalamus and synapse with neurons, forming pathways called optic radiations. The optic radiations terminate in the visual cortex of the occipital lobe and the nerve impulses that originated in the retina are interpreted here.

The visual fields of each eye overlap considerably and each eye sees a slightly different view. Because of this overlap and the crossing of the axons, information from both eyes reaches each side of the visual cortex, which then fuses the information into one image. This fusion of images accounts for the ability to perceive depth; however, depth perception depends on visual input from two eyes that focus well.

Refraction

Refraction is the bending of light rays as they pass from one medium to another of different optical density. As light rays pass through the eye, they are refracted at several points: as they enter the cornea; as they leave the cornea and enter the aqueous humour; as they enter the lens; and as they leave the lens and enter the vitreous humour. At the lens, the light is bent so that it converges at a single point on the retina. The focusing of the image is called **accommodation**. Because the lens is convex, the image projected on to the retina (the real image) is upside down and reversed from left to right. This real image is coded as electric signals that are sent to the brain; the brain

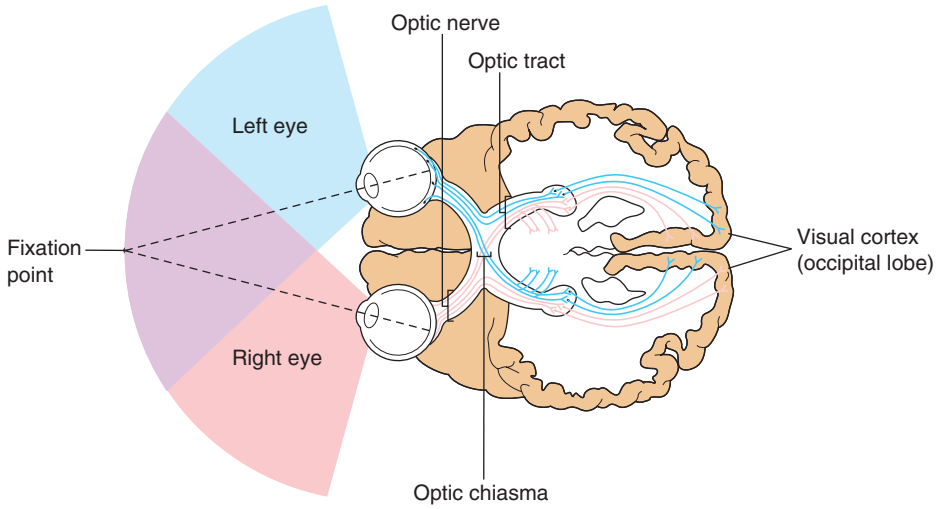


FIGURE 44.4 ■ The visual fields of the eye and the visual pathways to the brain

then decodes the image so that the person perceives it as it occurs in space.

The eyes are best adapted to see distant objects. Both eyes fix on the same distant image and do not require any change in accommodation. For people with emmetropic (normal) vision, the distance from the viewed object at which the eyes require no accommodation is 6 metres. This point is called the far point of vision. To focus for near vision, the eyes must instantly accommodate the lens, constrict the pupils and converge the eyeballs. Accommodation is accomplished by contraction of the ciliary muscles. This contraction reduces the tension on the lens capsule so that it bulges outwards to increase the curvature. This change in shape also achieves a shorter focal length, another requirement for focusing close images on the retina. The closest point on which a person can focus is called the near point of vision; in young adults with normal vision this is usually 20 to 25 cm. Pupillary constriction helps eliminate most of the divergent light rays and sharpens focus. **Convergence** (the medial rotation of the eyeballs so that each is directed towards the viewed object) allows the focusing of the image on the retinal fovea of each eye.

ASSESSING THE EYES

Structures and functions of the eyes are assessed by findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests.

Health assessment interview

To determine problems with the eyes and vision, a health assessment interview may be conducted during a health screening. It may focus on a chief complaint, such as blurred vision or an eye infection, or may be part of a complete health assessment. If the person has a health problem involving one or both eyes, analyse its onset, characteristics and course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, ask the person:

- Describe the type of pain you experience in your eyes. When did it begin? How long does it last?
- Have you noticed rings of colour around streetlights at night?
- When did you first notice having difficulty reading the paper?

Throughout the interview, be alert to non-verbal behaviours, such as squinting and/or abnormal eye movements, that may suggest problems with eye function. Explore problems such as watery, irritated eyes or changes in vision. Assess the person's use of corrective eyewear and care of eyeglasses or contact lenses. If the person uses eye medications, ask about the type, purpose, frequency, dose and duration of use. When taking the history, find out about eye trauma, surgery or infections, as well as the date and results of the last eye examination. In addition, ask the person about a medical history of diabetes, hypertension, thyroid disorders, glaucoma, cataracts and eye infections. Include questions about a family history of myopia (nearsightedness) or presbyopia (farsightedness), cancer of the retina, colour blindness and any other eye or vision disorders.

Collect information about environmental and/or work exposure to irritating chemicals, sport participation or hobbies that pose the risk of eye injury and the use of protective eyewear during dangerous activities such as sawing wood or using a lawn trimmer.

Interview questions categorised by functional health patterns are listed in the 'Functional health pattern interview' table below.

Physical assessment of the eyes and vision

Physical assessment of the eyes and of visual acuity may be performed as part of a total assessment or separately for people with known or suspected eye problems. The eyes and vision are primarily assessed through inspection of external structures and assessment of visual fields and visual acuity, extraocular muscle function and internal structures. Palpation—for example, of a blocked lacrimal duct—may be used if a problem is identified. Prior to the examination, explain the techniques to the person to decrease anxiety. The person may sit or stand

FUNCTIONAL HEALTH PATTERN INTERVIEW The eye

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Describe your vision. Rate it on a scale of 1 to 10, with 10 being excellent vision. Is it the same in both eyes? If not, which eye is better?
	■ Describe your current vision problems. How have these been treated?
	■ What eye medications do you use? What type and how often?
	■ Have you ever had eye surgery? Provide details.
	■ Describe the type of corrective lens that you wear. Are you satisfied with this appliance? How do you care for it?
	■ Describe how you care for your eyes each day.
	■ Do you wear sunglasses when you are outside?
	■ When was your last eye examination? Have you been tested for glaucoma?
Nutritional–Metabolic	■ Do you have any redness, swelling, watering or dryness of your eyes?
Activity–Exercise	■ Does your vision problem interfere with your usual activities of daily living? Provide details.
	■ Do you wear protective goggles when you take part in activities that increase the risk of injury to your eyes (such as at work or when operating machinery at home)?
Sleep–Rest	■ Does your eye problem interfere with your ability to rest or sleep (e.g. pain)? If so, what do you do?
Cognitive–Perceptual	■ Do you have any difficulty focusing on objects? If so, do you have more difficulty with near objects or distant objects?
	■ Is your vision blurry? Do you see halos around lights? Do you see flashes of light or 'floaters'? Do you see double?
	■ Do you have pain in or around your eyes? If so, describe its location, intensity, what makes it worse and how long it lasts. How do you treat it?
Self-perception–Self-concept	■ Has this problem with your eyes affected how you feel about yourself?
Role–Relationships	■ How has having this condition affected your relationships with others?
	■ Has having this condition interfered with your ability to work? Provide details.
	■ Has anyone in your family had problems with eye disease? Provide details.
Sexuality–Reproductive	■ Has this condition interfered with your usual sexual activity?
Coping–Stress–Tolerance	■ Has having this condition created stress for you? If so, does your health problem seem to be more difficult when you are stressed?
	■ Have you experienced any kind of stress that makes the condition worse? Provide details.
	■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem.
	■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem.
	■ Are there any specific treatments that you would not use to treat this problem?

during the assessment. Normal age-related findings for the older adult are summarised in Table 44.1.

Assessing visual fields

Visual fields are tested to assess the functioning of the macula and peripheral vision. The visual fields of the examiner (which must be normal to perform this assessment) are used as the standard. To measure visual fields, sit directly opposite the person at a distance of 45 to 60 cm. Ask the person to cover one eye with the opaque cover while you cover your own eye opposite to the person (e.g. if the person covers the right eye, you cover your left eye.) Ask the person to look directly at you. Move the penlight from the periphery towards the centre from right to left, above and below, and from the middle of

TABLE 44.1 Age-related changes in the eye

AGE-RELATED CHANGE	SIGNIFICANCE
The lens:	
• ↓ elasticity, decreasing focus and accommodation for near vision (presbyopia). • ↑ density and size, making lens more stiff and opaque. • Yellowing of the lens and changes in the retina affect colour perception.	Most older adults require corrective lenses to accommodate close and detailed work. Increased opacity leads to the development of cataracts. As cataracts develop, they increase sensitivity to glare and interfere with night vision.
The cornea:	
• Fat may be deposited around the periphery and throughout the cornea.	A partial or complete white circle may form around the cornea (arcus senilis). Lipid deposits in the cornea cause vision to be blurred.
• ↓ corneal sensitivity.	Decreased sensitivity increases the risk of injury to the eye.
The pupil:	
• ↓ size and responsiveness to light pupil; sphincter hardens.	Increased light perception threshold and difficulty seeing in dim light or at night means increased light is needed to see adequately.
The retina and visual pathways:	
• Visual fields narrow. • Photoreceptor cells are lost. • Rods work less effectively. • Macular degeneration is a risk. • Depth perception is distorted. • Adaptation to dark and light takes longer.	Peripheral vision is decreased and central vision may be lost from macular degeneration. Increased risk of falls as a result of changes in depth perception and adaptation to changes in light. Vision progressively declines with age.
The lacrimal apparatus:	
• ↓ reabsorption of intraocular fluid. • ↓ production of tears.	Increased risk of developing glaucoma and eyes feel and look dry.
The posterior cavity:	
• Debris and condensation become visible. • Vitreous body may pull away from the retina.	Vision is blurred and distorted and 'floaters' are often seen by the older adult.

each of these directions. Both you and the person should see the penlight enter the field of vision at the same time, if the examiner has normal peripheral vision.

The central visual field may be assessed with an Amsler grid (see Figure 44.5). The most basic form has black lines on a white grid, forming squares (boxes) that measure 5 mm with a black dot in the centre of the grid. The Amsler grid is useful for identifying early changes in vision from macular degeneration and diabetes mellitus. To use the Amlser grid, ask the person to hold the grid at normal reading distance (about 30 to 35 cm), cover one eye and stare at the centre dot. Ask the person if any of the lines look crooked or bent, if any of the boxes are different in size or shape, and if any of the lines are wavy, missing, blurry or discoloured. Repeat with the other eye. The test should be conducted before the pupils are dilated and the person should be wearing their best correction lenses.

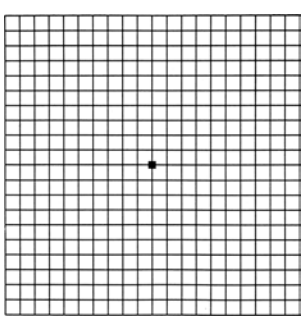


FIGURE 44.5 ■ The Amsler grid

Diagnostic tests

The results of diagnostic tests of the structure and functions of the eyes are used to support the diagnosis of a specific injury, disease or vision problem; to provide information to identify and/or modify the appropriate medications or assistive devices used to treat the disease or problem; and to help nurses monitor the person's responses to treatment and nursing care interventions. Diagnostic tests of the eye, especially for vision testing, are most often conducted in a health-care provider's office. Diagnostic tests to assess the structure and functions of the eyes are described in the 'Diagnostic tests' box below and summarised in the following list. More information is included in the discussion of specific injuries or diseases in Chapter 45.

- Refractive errors (with prescription for corrective lenses) are evaluated by retinoscopy and/or refractometry. Pupils must be dilated for accurate diagnosis.

DIAGNOSTIC TESTS Eye disorders

NAME OF TEST Refraction, retinoscopy, refractometry

PURPOSE AND DESCRIPTION Used to measure refractive error. Either a hand-held retinoscope or an instrument with multiple lenses is used; with the latter method, the person chooses lenses that provide the best vision.

RELATED NURSING CARE No special preparation is needed; advise the person that pupils will be dilated with medication and may be enlarged for several hours.

NAME OF TEST Tonometry

PURPOSE AND DESCRIPTION Used to diagnose increased intraocular pressure in glaucoma. A variety of methods are used, ranging from a hand-held instrument (tonometer) to a computerised component of the device used to evaluate refraction. The cornea is anaesthetised prior to being touched with the device.

Normal value: 10–22 mmHg.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Computed tomography (CT) scan of the eye

PURPOSE AND DESCRIPTION Radiological examination used to identify foreign objects or tumours within the eyeball or orbit.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Fluorescein angiography

PURPOSE AND DESCRIPTION Fluorescein solution is injected IV to evaluate blood vessels in the eye in conditions such as diabetes, macular degeneration or vessel occlusion.

RELATED NURSING CARE Informed consent is required for this test. Note current medications and notify the doctor if the person is pregnant. Inform the person that a hot flush and transient nausea may be experienced when the dye is injected. Monitor for anaphylactic hypersensitivity responses that may occur. Fluorescent yellow urine and a yellow skin colour may be noted for 1–2 days after the procedure; these are not considered harmful.

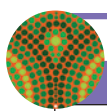
- Tonometry is used to identify and evaluate increased intraocular pressure, characteristic of glaucoma.
- A CT scan may be used to identify foreign objects or tumours of the eye.
- Fluorescein angiography is used to evaluate blood vessels in the eye in conditions such as diabetes, macular degeneration or vessel occlusion.

Regardless of the type of diagnostic test, the nurse may be responsible for explaining the procedure and any special preparation needed, assessing any medication use that might affect the outcome of the tests, supporting the person during the examination as necessary, documenting the procedures as appropriate and monitoring the results of the tests.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of the adult, as several diseases of the eyes have a genetic component. During the health assessment interview, ask about a family history of glaucoma and/or blindness.

During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the accompanying 'Genetic considerations' box). If the data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.



GENETIC CONSIDERATIONS
Eye disorders

- *Glaucoma* is a term used for a group of diseases that damage the optic nerve and cause blindness. Approximately 300 000 people in Australia may suffer from glaucoma, with nearly half of them not being aware that they suffer from this disease (Centre for Eye Research in Australia, 2014).
- Gyrate atrophy of the choroid and retina is a genetic disorder resulting in a progressive vision loss, with total blindness occurring between ages 40 and 60.
- Best disease is a familial disorder found most often in Caucasians who originated in Europe. It leads to gradual loss of vision, beginning during the teenage years.
- Retinitis pigmentosa may be transmitted in an autosomal recessive, dominant or X-linked pattern. It results in progressive night blindness, with loss of visual acuity and peripheral vision.

EYE AND VISION ASSESSMENTS

Vision assessment

Visual acuity is assessed with an eye chart such as the Snellen or the E chart for testing distance vision, and the Rosenbaum chart for testing near vision. The Snellen chart contains rows of letters in various sizes, with standardised numbers at the end of each row. The number at the end of the row indicates the visual acuity of a person who can read the row at a distance of 6 metres. If the person is unable to read or does not read English, use the E chart to test visual acuity. The top number at the end of the row is always 6, representing the distance between the person and the chart. The bottom number is the distance (in metres) at which a person with normal vision can read the line. A person with normal vision can read the row marked 6/6. To conduct the assessment, ask the person to stand 6 metres from the chart in a well-lit area. Ask the person to cover one eye with an opaque cover or with the palm of their hand (see Figure 44.6). Then ask the person to read each row of letters, moving from largest letters to the smallest ones that the person can see. Measure visual acuity in the other eye the same way and then assess visual acuity while the person has both eyes uncovered. You may test the person who wears corrective lenses with and without the lenses.

The Rosenbaum chart is held at a distance of from 30 to 35 cm from the eyes, with visual acuity measured in the same manner as with the Snellen chart (see Figure 44.7). A gross estimate of near vision may also be assessed by asking the person to read from a magazine or newspaper.

FIGURE 44.6 ■ Testing distant vision using the Snellen eye chart

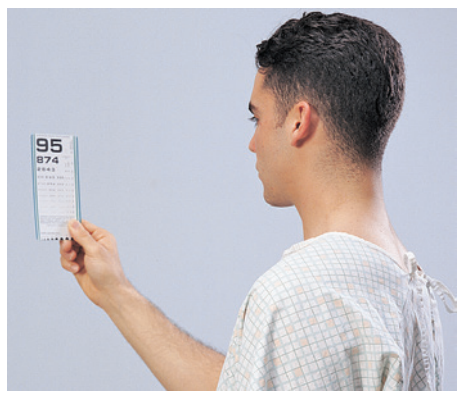
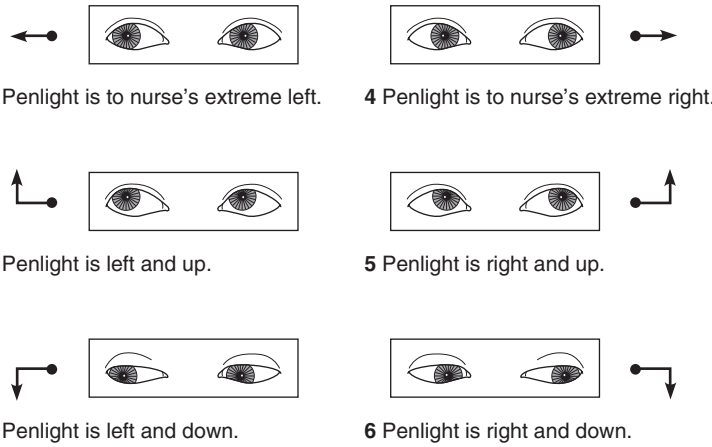


FIGURE 44.7 ■ Testing near vision using Rosenbaum eye chart

Technique/normal findings	Abnormal findings
Vision	
Assess distant vision using the Snellen or E chart. *When standing 6 metres from the chart, the person can read the smallest line of letters with or without corrective lenses (recorded as 6/6).*	■ Changes in distant vision are most commonly the result of **myopia** (nearsightedness). For example, a reading of 6/9 indicates impaired distance vision. A person has to stand 6 metres from the chart to read a line that a person with normal vision could read 9 metres from the chart.
Assess near vision, using a Rosenbaum chart or a card with newsprint held 30 to 35 cm from the person's eyes. *Normal near visual acuity is 35/35 with or without corrective lenses.*	■ Changes in near vision, especially in people over age 45, can indicate **presbyopia**, impaired near vision resulting from a loss of elasticity of the lens related to ageing. In younger people, this condition is referred to as **hyperopia** (farsightedness).
Eye movement assessment	
Assess the cardinal fields of vision to gain information about extraocular eye movements. Ask the person to follow a pen or your finger while keeping the head stationary. Move the pen or your finger through the 6 fields one at a time, returning to the central starting point before proceeding to the next field (see Figure 44.8). *The eyes should move through each field without involuntary movements.*	■ Failure of one or both eyes to follow the object in any given direction may indicate extraocular muscle weakness or cranial nerve dysfunction. ■ An involuntary rhythmic movement of the eyes, **nystagmus**, is associated with neurological disorders and the use of some medications.

Technique/normal findings	Abnormal findings
The cover–uncover test is a test for strabismus, a weakening of a muscle that causes one eye to deviate from the other when the person is focusing on an object. To conduct the test, hold a pen or your finger about 300 cm from the eyes and ask the person to focus on that object. Cover one of the person's eyes and note any movement in the uncovered eye; as you remove the cover, assess for movement in the eye that was just uncovered. Repeat the procedure with the other eye. *The uncovered eye should remain fixed straight ahead. The covered eye should remain fixed straight ahead after being uncovered.*	 FIGURE 44.8 ■ The six cardinal fields of vision
Assess convergence. Ask the person to follow an object as you move it towards the person's eyes. *Normally both eyes converge towards the centre.*	■ Failure of the eyes to converge equally on an approaching object may indicate a neuromuscular disorder or improper eye alignment.
Assess the corneal light reflex. Direct a light source onto the bridge of the nose from 30 to 40 cm. *Observe for equal reflection of the light from each eye.*	■ Reflections of the light from different sites on the eyes reveal improper alignment.
Pupils	
Observe pupil size and equality. *Pupils should be of equal size, 3 to 5 mm.*	■ Pupils that are unequal in size may indicate a severe neurological problem, such as increased intracranial pressure.
Assess direct and consensual pupil response. Ask the person to look straight ahead. Shine a light obliquely into one eye at a time. Observe for constriction of the pupil in the illuminated eye. Test both eyes. To test consensual pupil response, again shine a light obliquely into one eye at a time as the person looks straight ahead. Observe constriction of the pupil in the opposite eye. *The normal direct and consensual pupillary response is constriction.*	■ Failure of the pupils to respond to light may indicate degeneration of the retina or destruction of the optic nerve. ■ A person who has one dilated and unresponsive pupil may have paralysis of the oculomotor nerve. ■ Some eye medications may cause unequal dilation, constriction or inequality of pupil size. Morphine and narcotic drugs may cause small, unresponsive pupils and anticholinergic drugs such as atropine may cause dilated, unresponsive pupils.
Test for accommodation. Hold an object at a distance of approximately 600 cm from the person. The pupils should dilate. Ask the person to follow the object as you bring it to within a few centimetres of the person's nose. *The pupils should constrict and converge as they change focus to follow the object.*	■ Failure of accommodation along with lack of pupil response to light may signal a neurological problem. ■ Lack of response to light with appropriate response to accommodation is often seen in people with diabetes.
External eye	
Inspect the eyelids. Eyelids should be the colour of the person's facial skin, without redness, discharge or drooping. *The sclera should not be visible.*	■ Unusual redness or discharge may indicate an inflammatory state due to trauma, allergies or infection. ■ Drooping of one eyelid, called **ptosis**, may be the result of a stroke, indicate a neuromuscular disorder or be congenital (see Figure 44.9).

Technique/normal findings	Abnormal findings
	■ Unusual widening of the lids may be due to exophthalmos (protrusion of the eyeball). Exophthalmos is often associated with hyperthyroid conditions (see Chapter 18). ■ Yellow plaques noted on or near the lid margins are referred to as xanthelasma and may indicate high lipid levels. ■ An acute localised inflammation of a hair follicle is known as a *hordeolum* (sty) and is generally caused by staphylococcal organisms. ■ A *chalazion* is an infection or retention cyst of the meibomian glands.
Inspect the puncta. *The puncta should be free of redness or discharge.*	■ Unusual redness or discharge from the puncta may indicate an inflammation due to trauma, infection or allergies.
Inspect the bulbar and palpebral conjunctiva. *The conjunctiva should be clear, moist and smooth. The upper and lower palpebral conjunctiva should be clear, without redness or swelling.*	■ Increased erythema or the presence of exudate may indicate acute conjunctivitis. ■ A cobblestone appearance is often associated with allergies. ■ A fold in the conjunctiva, called a *pterygium*, may be seen as a clouded area that extends over the cornea. This is an abnormal growth of the bulbar conjunctiva, usually seen on the nasal side of the cornea. It may interfere with vision if it covers the pupil.
Inspect the sclera. *The sclera is white in Caucasians; people with darker skin normally have yellow sclera.*	■ Unusual redness may indicate an inflammatory state as a result of trauma, allergies or infection. ■ Yellow discolouration of the sclera in people with fair skin may be seen in conditions involving the liver, such as hepatitis. ■ Bright red areas in the sclera are often subconjunctival haemorrhages and may indicate trauma or bleeding disorders. They may also occur spontaneously.
Inspect the cornea. *The cornea is normally transparent.*	■ Dullness, opacities or irregularities of the cornea may be abnormal. ■ *Corneal arcus* is a thin, greyish-white arc seen towards the edge of the cornea. It is normal in older adults.
Assess corneal sensitivity. Lightly touch a wisp of cotton to the person's cornea. *This action should cause a corneal reflex (blinking the eye).*	■ Failure of the corneal reflex may indicate a neurological disorder.
Inspect the iris. *The iris is normally round, flat and evenly coloured.*	■ Lack of clarity of the iris may indicate a cloudiness of the cornea. ■ Constriction of the pupil accompanied by pain and circumcorneal redness indicates acute iritis.
Internal eye	
Assess internal structures of the eye by using the ophthalmoscope, an instrument that allows visualisation of the lens, the vitreous humour and the retina. Box 44.1 provides guidelines for using the ophthalmoscope.	
Inspect for the red reflex. *The red reflex should be clearly visible.*	■ Absence of a red reflex often indicates improper position of the ophthalmoscope, but also may indicate total opacity of the pupil by a cataract or a haemorrhage into the vitreous humour.
Inspect the lens and vitreous body. *The lens should be clear.*	■ A cataract is an opacity of the lens, often seen as a dark shadow on ophthalmoscopic examination. It may be due to ageing, trauma, diabetes or a congenital defect.

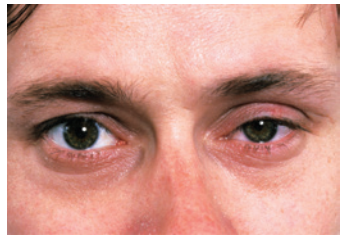


FIGURE 44.9 ■ Ptosis

Source: © Medical-on-Line/Alamy.

Technique/normal findings	Abnormal findings
Inspect the retina. *There should be no visible haemorrhages, exudate or white patches.*	■ Areas of haemorrhage, exudate and white patches may be a result of diabetes or long-standing hypertension.
Inspect the optic disc. *The optic disc should be round to oval in shape with clear, well-defined borders.*	■ Loss of definition of the optic disc, as well as an increase in the size of the physiological cup, is seen in papilloedema from increased intracranial pressure.
Inspect the blood vessels of the retina. *The retinal blood vessels should be distinct.*	■ Glaucoma often results in displacement of blood vessels from the centre of the optic disc due to increased intraocular pressure. ■ Hypertension may cause a narrowing of the vein where an arteriole crosses over. ■ Engorged veins may occur with diabetes, atherosclerosis and blood disorders.
Inspect the retinal background. *The retina should be a consistent red-orange colour, becoming lighter around the optic disc.*	■ Variations in colour or a pale colour overall may indicate disease.
Inspect the macula. *The macula should be visible on the temporal side of the optic disc.*	■ Absence of the fovea centralis is common in older adults. It may indicate macular degeneration, a cause of loss of central vision.
Palpate over the lacrimal glands, puncta and nasolacrimal duct. *There should be no tenderness, drainage or excessive tearing.*	■ Tenderness over any of these areas or drainage from the puncta may indicate an infectious process. (Wear gloves if you see any drainage.) ■ Excessive tearing may indicate a blockage of the nasolacrimal duct.

BOX 44.1 Guidelines for using the ophthalmoscope

The ophthalmoscope has a head and a handle (see figure). The head contains a focus wheel (also called a lens selector dial) located on the side, lenses of varying magnification and an opening through which the eye structures are visualised. The focus wheel adjusts the lens refraction, which is measured in diopters. The diopter measurements range from 0 to +40 when the lens is rotated clockwise and from 0 to –25 when the lens is rotated anticlockwise. By moving the focus wheel, the examiner can converge or diverge light rays to visualise the retina. The handle usually contains batteries that can be recharged.

Before the examination, explain the procedure to the person. Assemble the ophthalmoscope. Wash hands and wear disposable gloves if the person has any drainage from the eyes. Darken the room (to allow the pupils of the person to dilate) and ask the person to look straight ahead, focusing on a fixed point such as an object on the wall. Hold the ophthalmoscope in one hand, resting the index finger on the focus wheel (see the figure below left).

1. Turn on the ophthalmoscope light and set focus wheel to 0 diopters. Hold the ophthalmoscope in your right hand with your index finger on the focus wheel. Standing in front of the person, position yourself at a 15-degree angle to the person's line of vision.
2. Hold the opening of the ophthalmoscope up to your right eye and direct the light towards the person's right eye from a distance of about 30 cm.
3. As the beam of light falls on the person's pupil, observe for the red reflex which appears as a sharply outlined orange glow from within the pupil. This glow is the reflection of the light from the retina.

An ophthalmoscope

4. Move closer to the person, turning the focus wheel clockwise towards the positive numbers as needed to maintain clear focus.
5. Examine the lens and the vitreous body; both should be clear.
6. Gradually rotate the focus wheel anticlockwise towards the negative numbers as needed, focusing on a structure of the retina (such as the disc or a blood vessel). Turn the focus wheel until the image is clear. Examine the structures of the retina as follows:
   a. The optic disc (see the figure below right). Assess for size, shape, colour, distinct margins and the physiological cup. The disc is round to slightly oval and about 1.5 mm in diameter. It has a yellow to pink colour that is lighter than the retina itself. The margins should be sharp and clear. The physiological cup is a small depression that occupies about one-third of the optic disc, lying temporal to the centre of the disc.

(continued)

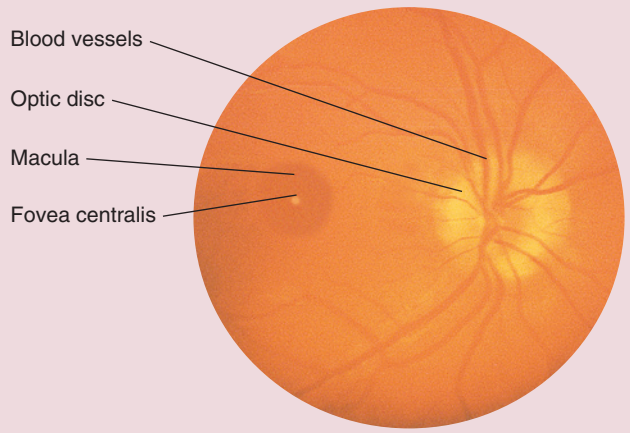
BOX 44.1 Guidelines for using the ophthalmoscope (continued)

b. The vessels of the retina. Assess for colour, arteriolar light reflex, ratio of arterioles to veins and arteriovenous crossings. The arterioles are red, brighter than the veins and about one-fourth smaller. The arterioles normally have a narrow light reflex from the centre of each vessel; veins do not have this light reflex. The ratio of arterioles to veins is usually 2:3 or 4:5. The vessels normally cross and become smaller towards the periphery.

Technique for holding an ophthalmoscope

c. The retinal background. Assess colour and changes in colour. The retina is normally reddish orange and regular in colour.
d. The macula. Assess size and colour. To assess the macula, ask the person to look directly into the ophthalmoscope light. The macula is temporal to the optic disc, appears slightly darker than the retina and has no visible vessels. The fovea centralis may be seen as a bright spot of light. Because looking directly into the light causes some discomfort, conduct this portion of the examination last. The macula is often difficult to visualise.

7. Using the same technique, examine the left eye.



The optic disc as seen through an ophthalmoscope

Source: Don Wong/Photo Researchers, Inc.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE EARS

As a sensory organ, the ears have two primary functions: hearing and maintaining equilibrium. Anatomically, each ear is divided into three areas: the external ear, the middle ear and the inner ear (see Figure 44.10). Although each area has a unique function, all three are involved in hearing, but only the inner ear is involved in equilibrium.

THE EXTERNAL EAR

The external ear consists of the auricle (or pinna), the external auditory canal and the tympanic membrane.

The auricles are elastic cartilage covered with thin skin. They contain sebaceous and sweat glands and sometimes hair. Each auricle has a rim (the helix) and a lobe that serves to direct sound waves into the ear.

The external auditory canal, which is about 2.5 cm long, extends from the auricle to the tympanic membrane. The canal is lined with skin that contains hair, sebaceous and ceruminous glands. The external auditory canal serves as a resonator for the range of sound waves typical of human speech and increases the pressure that sound waves in this frequency range place on the tympanic membrane. The canal's ceruminous glands (modified apocrine glands) secrete a yellow to brown waxy substance called **cerumen** (earwax). Cerumen traps foreign bodies; it also has bacteriostatic properties, protecting the tympanic membrane and the middle ear from infections.

The tympanic membrane lies between the external ear and the middle ear. It is a thin, semitransparent, fibrous

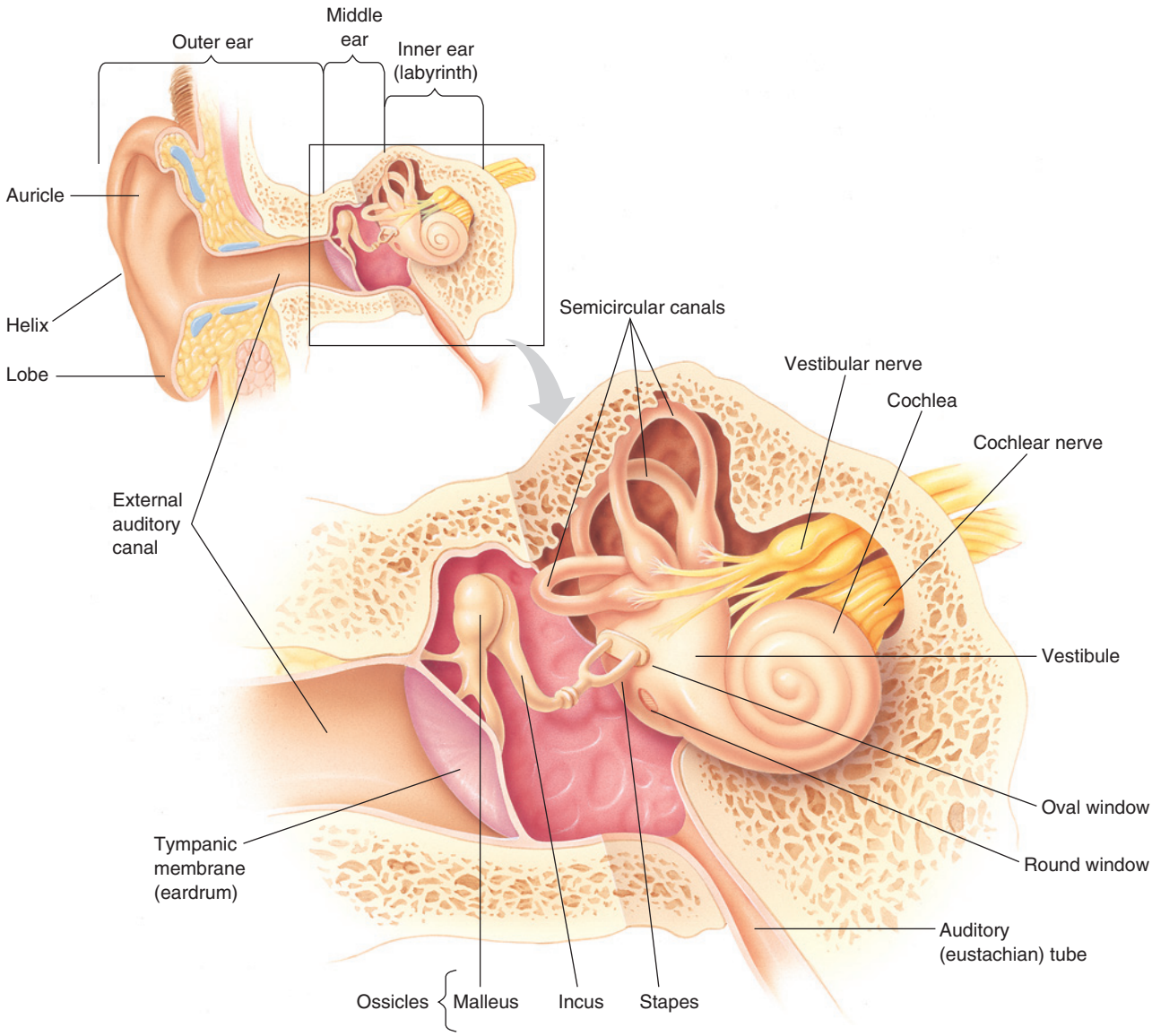


FIGURE 44.10 ■ Structures of the external ear, middle ear and inner ear

Source: ©Todd Buck, Pearson Education.

structure covered with skin on the external side and mucosa on the inner side. The membrane vibrates as sound waves strike it; these vibrations are transferred as sound waves to the middle ear.

THE MIDDLE EAR

The middle ear is an air-filled cavity in the temporal bone and contains three auditory ossicles: the malleus, the incus and the stapes. These bones extend across the middle ear. The medial side of the middle ear is a bony wall containing two membrane-covered openings: the oval window and the round window. The posterior wall of the middle ear contains the mastoid antrum. This cavity communicates with the mastoid sinuses, which help the middle ear adjust to changes in pressure. It also opens into the eustachian tube, which connects with the nasopharynx. The eustachian tube helps to equalise the air pressure in the middle ear by opening briefly in response to differences between the pressure in the middle ear and atmospheric pressure. This action also ensures that vibrations of the tympanic membrane remain adequate. The mucous membrane lining the middle ear is continuous with the mucous membranes lining the throat.

The malleus attaches to the tympanic membrane and articulates with the incus, which in turn articulates with the stapes. The stapes fits into the oval window. When the tympanic membrane vibrates, the vibrations are conducted across the middle ear to the oval window by the ossicles. The vibrations then set in motion the fluids of the inner ear, which in turn stimulate the hearing receptors. Two small muscles

attached to the ossicles contract reflexively in response to sudden loud noises, decreasing the vibrations and protecting the inner ear.

THE INNER EAR

The inner ear, also called the labyrinth, is a maze of bony chambers located deep within the temporal bone, just behind the eye socket. The labyrinth is further divided into two parts: the bony labyrinth, a system of open channels that houses the second part, the membranous labyrinth. The bony labyrinth is filled with a fluid (similar to cerebrospinal fluid) called perilymph, which bathes the membranous labyrinth. Within the chambers of the membranous labyrinth is a fluid called endolymph.

The bony labyrinth has three regions: the vestibule, the semicircular canals and the cochlea. The vestibule is the central portion of the inner ear, one side of which is a bony wall containing the oval window. Two sacs within the vestibule (the saccule and the utricle) join the vestibule with the cochlea and the semicircular canals. The saccule and the utricle contain receptors for equilibrium that respond to changes in gravity and changes in position of the head. The three semicircular canals each project into a different plane (anterior, posterior and lateral). Each canal contains a semicircular duct that communicates with the utricle of the vestibule. Each duct has an enlarged area at one end containing an equilibrium receptor that responds to angular movements of the head.

The cochlea is a tiny bony chamber that houses the organ of Corti, the receptor organ for hearing. The organ of Corti is a series of sensory hair cells, arranged in a single row of inner hair cells and three rows of outer hair cells. The hair cells are innervated by sensory fibres from the VIII (acoustic) cranial nerve. The organ of Corti is supported in the cochlea by the flexible basilar membrane, which has fibres of varying lengths that respond to different sound wave frequencies.

Sound conduction

Hearing is the perception and interpretation of sound. Sound is produced when the molecules of a medium are compressed, resulting in a pressure disturbance evidenced as a sound wave. The intensity or loudness of sound is determined by the amplitude (height) of the sound wave, with greater amplitudes causing louder sounds. The frequency of the sound wave in vibrations per second determines the pitch or tone of the sound, with higher frequencies resulting in higher sounds. The human ear is most sensitive to sound waves with frequencies of between 1000 and 4000 cycles per second; however, it can detect sound waves with frequencies of between 20 and 20 000 cycles per second.

Sound waves enter the external auditory canal and cause the tympanic membrane to vibrate at the same frequency. The ossicles not only transmit the motion of the tympanic membrane to the oval window, but also amplify the energy of the sound wave. As the stapes moves against the oval window, the perilymph in the vestibule is set in motion. The increased pressure of the perilymph is transmitted to fibres of the basilar membrane and then to the organ of Corti (directly above the basilar membrane). The up-and-down movements of the fibres of the basilar membrane pull the hair cells in the organ of Corti, which in turn generates action potentials that are transmitted to the VIII (acoustic) cranial nerve and then to the brain for interpretation.

Several brainstem auditory nuclei transmit impulses to the cerebral cortex. Fibres from each ear cross, with each auditory cortex receiving impulses from both ears. Auditory processing is so finely tuned that a wide variety of sounds of different pitch and loudness can be heard at any one time. In addition, the source of the sound can be localised.

Equilibrium

The inner ear also provides information about the position of the head. This information is used to coordinate body movements so that equilibrium and balance are maintained. The types of equilibrium are static balance (affected by changes in the position of the head) and dynamic balance (affected by the movement of the head).

Receptors called maculae in the utricle and the saccule of the vestibule detect changes in the position of the head. Maculae are groups of hair cells that have protrusions covered with a gelatinous substance. Embedded in this gelatinous substance are tiny particles of calcium carbonate called otoliths (ear stones), which make the gelatin heavier than the endolymph that fills the membranous labyrinth. As a result, when the head is in the upright position, gravity causes the gelatinous substance to bear down on the hair cells. When the head changes position, the force on the hair cells alters, bending them and changing the pattern of stimulation of the neurons. Thus, a different pattern of nerve impulses is transmitted to the brain, where stimulation of the motor centres initiates actions that coordinate various body movements according to the position of the head.

The receptor for dynamic equilibrium is in the crista, a crest in the membrane lining the ampulla of each semicircular canal. The cristae are stimulated by rotatory head movement (acceleration and deceleration) as a result of changes in the flow of endolymph and of movement of hair cells in the maculae. The direction of endolymph and hair cell movement is always opposite to the motion of the body.

ASSESSING THE EARS

The structure and functions of the ears are assessed through findings from a health assessment interview to collect subjective data, a physical assessment to collect objective data and diagnostic tests (see below for sample documentation).

Health assessment interview

To determine problems with the ear, a health assessment interview may be conducted during a health screening. It may focus on a chief complaint, such as hearing problems or pain in the ear. If the person has a problem involving one or both ears, analyse its onset, characteristics and course, severity, precipitating and relieving factors, and any associated symptoms,

SAMPLE DOCUMENTATION

Assessment of the ear

25/9/2016
NURS
0930 hrs

22-year-old male complaining of 'having some problems hearing these days'. States he often listens to music in his car 'as loud as it will go' and uses ear phones at home so as not to bother other family members. Ears are placed bilaterally; skin smooth without lesions. Small amount of dark brown cerumen present in both ear canals. Tympanic membranes grey and shiny. No bulging or retraction noted. Whisper test: unable to repeat back words spoken by examiner. Weber's test: sound lateralised to left ear. Rinne test: BC ≥ AC. No tenderness noted when mastoids palpated. Referred to ear clinic for further evaluation.

RN Fehon
O. FEHON RN

noting the timing and circumstances. For example, the following questions may be asked:

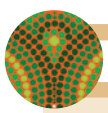
- Have you noticed any difficulty hearing high-pitched sounds, low-pitched sounds, or both?
- When did you first notice the ringing in your ears?
- Is your workplace very noisy? If so, do you wear protective ear equipment at work?

Throughout the examination, be alert to non-verbal behaviours, such as inappropriate answers or requests to repeat statements that may suggest problems with ear function. Explore changes in hearing, ringing in the ears (*tinnitus*), ear pain, drainage from the ears or the use of hearing aids. When taking a medical history, ask about trauma, surgery or infections of the ear, as well as the date of the last ear examination. In addition, ask the person about a history of infectious diseases, such as meningitis or mumps, as well as the use of medications that may affect hearing. Because ear problems tend to run in families, ask about a family history of hearing loss, ear problems or diseases. If the person has a hearing aid, ascertain the type and assess measures for its care.

Interview questions categorised by functional health patterns are found in the 'Functional health pattern interview' table below.

Physical assessment of the ears and hearing

Physical assessment of the ears and hearing may be performed as part of a total health assessment or separately for people with known or suspected problems with the ears. The ears and hearing are assessed primarily through inspection of external structures through palpation, the external auditory canal and



FUNCTIONAL HEALTH PATTERN INTERVIEW The ear

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Describe your hearing. Rate it on a scale of 1 to 10, with 10 being excellent hearing. Is it the same in both ears? If not, which ear is better? ■ Describe any current hearing problems. How have these been treated? ■ Do you use ear medications? What type, dosage and how often? ■ Have you ever had ear surgery? Provide details. ■ Describe the type of hearing aid that you use. Are you satisfied with this appliance? How do you care for it? ■ Describe how you care for your ears each day. ■ Have you ever had your hearing tested? When was your last ear examination? ■ Do you listen to loud music? Do you use ear phones when you listen to loud music?
Nutritional–Metabolic	■ Do you have any swelling or tenderness in the ears or drainage from the ears?
Activity–Exercise	■ Does your hearing problem interfere with your usual activities of daily living? Provide details. ■ Do you wear protective earplugs when you take part in activities that increase the risk of injury to your ears (such as at work or when operating machinery)?
Sleep–Rest	■ Does your ear problem interfere with your ability to rest or sleep (e.g. pain)? If so, what do you do?
Cognitive–Perceptual	■ Do you have pain in or around your ears? Have you ever had ringing in your ears? If so, describe its location, intensity, what makes it worse and how long it lasts. How do you treat it? ■ Do you have difficulty hearing conversations, either in person or on the telephone? Do you have trouble hearing the television? Do you have difficulty hearing when you are in crowds or there is background noise?

(continued)

FUNCTIONAL HEALTH PATTERN INTERVIEW The ear (continued)

	■ Have you noticed your hearing is different in each ear? ■ Do you have buzzing, ringing or crackling noises in one or both ears? Provide details. ■ Do you ever feel dizzy?
Self-perception–Self-concept	■ Has this problem with your ears affected how you feel about yourself?
Role–Relationships	■ How has having this condition affected your relationships with others? ■ Has having this condition interfered with your ability to work? Provide details. ■ Has anyone in your family had problems with ear disease? Provide details.
Sexuality–Reproductive	■ Has this condition interfered with your usual sexual activity?
Coping–Stress–Tolerance	■ Has having this condition created stress for you? If so, does your health problem seem to be more difficult when you are stressed? ■ Have you experienced any kind of stress that makes the condition worse? Provide details. ■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments that you would not use to treat this problem?

the tympanic membrane. Disorders of the middle ear may be identified with tympanometry and hearing acuity is assessed by voice and tuning fork tests. Normal age-related findings for the older adult are summarised in Table 44.2.

Prior to the assessment, collect all necessary equipment and explain the techniques to the person to decrease anxiety. During the assessment, the person should be sitting and the examiner's head should be level with the head of the person. The auditory canal and tympanic membrane are inspected with the otoscope. Guidelines for use of the otoscope are listed in Box 44.2.

Diagnostic tests

The results of diagnostic tests of the structure and functions of the ears are used to support the diagnosis of a specific injury, disease or hearing problem; provide information to identify and/or modify the appropriate medications or assistive devices used to treat the disease or problem; and help nurses monitor the person's responses to treatment and nursing care interventions. Diagnostic tests of the ear, especially for hearing, are most often conducted in a healthcare provider's office. Diagnostic tests to assess the structure and functions of the ears are described in the 'Diagnostic tests' table below

TABLE 44.2 Age-related changes in the ear

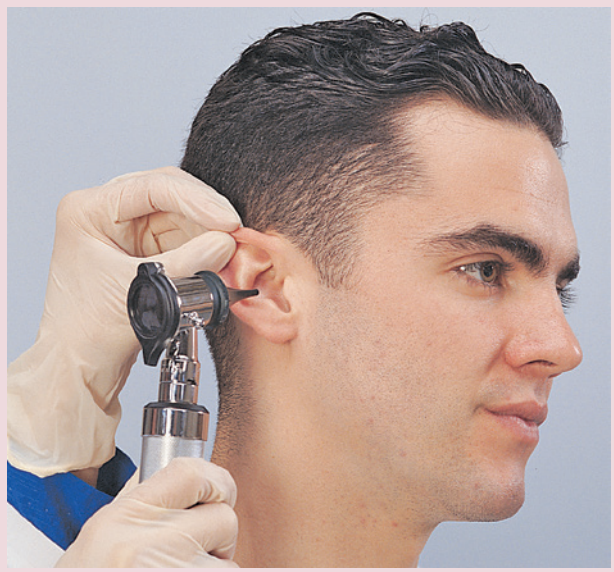
AGE-RELATED CHANGE	SIGNIFICANCE
The inner ear:	
• Loss of hair cells, ↓ blood supply, less flexible basilar membrane, degeneration of spiral ganglion cells and ↓ production of endolymph result in progressive hearing loss with age (presbycusis). • High-frequency sounds are lost; middle- and low-frequency sounds may also be lost or decreased. • Vestibular structures degenerate, organ of Corti and cochlea atrophy.	Older adults may require hearing aids to hear well. With loss of high-frequency sounds, speech may be distorted, contributing to a risk of problems with communication. Degeneration and atrophy of inner ear structures concerned with balance and equilibrium increase the risk of falls.
The middle ear:	
• Muscles and ligaments weaken and stiffen, decreasing the acoustic reflex.	Own speech and sounds are louder and may further interfere with hearing, speech and communications.
The external ear:	
• Increased cerumen in the ear canal is due to its high keratin content.	Accumulated cerumen may impair hearing.

BOX 44.2 Guidelines for using the otoscope

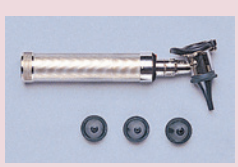
The otoscope has a handle that contains batteries for the light and various specula that fit on to the handle (see the accompanying figure). It is used to inspect the auditory canal and the tympanic membrane. A pneumatic otoscope is used to determine the mobility of the tympanic membrane. It has an attached rubber bulb that can be squeezed to inject air into the auditory canal, causing a normal tympanic membrane to move in and out.

Before the examination, explain the procedure. Assemble the otoscope using the largest speculum that will fit into the person's auditory canal without discomfort. Wash your hands; wear disposable gloves if the person has any drainage from the ears. Turn on the otoscope light. Ask the person to tip the head slightly towards the shoulder opposite the ear being examined. When they are in this position, the auditory canal is aligned with the speculum.

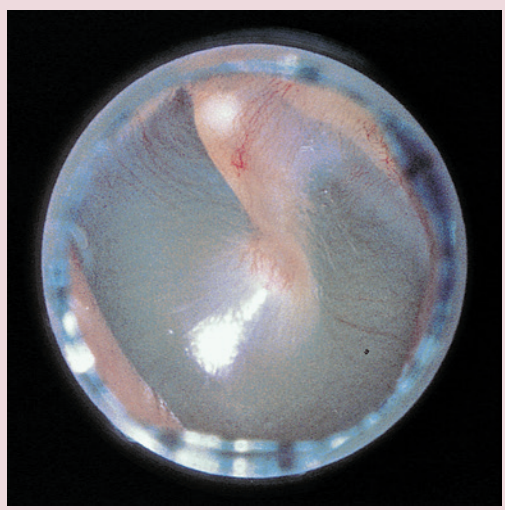
1. Hold the handle of the otoscope in your dominant hand. If the person is restless, hold the otoscope handle upward, resting the hand against the person's head. If the person is cooperative, hold the handle downward.
2. For adults, grasp the superior portion of the auricle and pull up, out and back to straighten the auditory canal (see the figure above right).
3. Insert the speculum into the ear and advance it gently. Assess the walls of the auditory canal while advancing the speculum, inspecting for colour, obstructions, hair growth and cerumen. Old cerumen is very dark and may obstruct visualisation of part or all of the tympanic membrane.
4. Move the otoscope so that you can see the tympanic membrane. You may need to realign the auditory canal by gently continuing to pull up and back on the auricle. A normal membrane is semitransparent, allowing visualisation of a portion of the auditory ossicles. The concave nature of the tympanic membrane and its oblique position in the auditory canal account for the triangular light reflex (cone of light) seen on otoscopic examination.
5. Note the colour and surface of the membrane. The normal tympanic membrane is pearly grey, shiny and semitransparent. The surface should be continuous, intact and either flat or concave.
6. Identify the landmarks on the tympanic membrane (see the figure below right):
   a. the cone of light, located over the anteroinferior quadrant
   b. the malleus, pars tensa, annulus, pars flaccida and malleolar folds.
7. Assess movement of the tympanic membrane. If the auditory tube is patent, the membrane moves in and out when air is injected or when the person performs the Valsalva manoeuvre.
8. Gently withdraw the speculum. If the speculum is soiled with drainage or cerumen, use a clean speculum for the other ear.
9. Using the same technique, examine the other ear.



Technique for using an otoscope



An otoscope



The tympanic membrane as it appears through the otoscope

and summarised in the following list. More information is included in the discussion of specific injuries or diseases in Chapter 45.

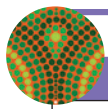
- Audiometry is used to evaluate and diagnose conductive and sensorineural hearing loss.
- Electrical activity of the auditory nerve may be evaluated by using an auditory evoked potential (AEP) or an auditory brainstem response (ABR).
- Vestibular system function is evaluated with a caloric test. If no nystagmus occurs during the test, further testing for brain lesions is conducted.

Regardless of the type of diagnostic test, the nurse may be responsible for explaining the procedure and any special preparation needed, assessing for any medication use that might affect the outcome of the tests, supporting the person during the examination as necessary, documenting the procedures as appropriate and monitoring the results of the tests.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of the adult as several diseases of the ears have a genetic component. During the health assessment interview, ask about a family history of congenital deafness or deafness associated with a thyroid goitre or with tumours of the auditory nerve.

During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see 'Genetic considerations' box below). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.



GENETIC CONSIDERATIONS
Ear disorders

- Deafness (hearing loss) is a common disorder that is seen from newborns to those of old age. About 1 in 1000 infants have a profound hearing loss, with about half being genetic in origin (National Center for Biotechnology Information (NCBI) (US), 1998). Early diagnosis is important to facilitate language and social skill development in adults.
- Penred syndrome is an inherited disorder that accounts for as much as 10% of hereditary deafness. The deafness is usually accompanied by a thyroid goitre.
- Neurofibromatosis, a rare inherited disorder, is characterised by the development of acoustic neuromas (benign tumours of the auditory nerve) and malignant central nervous system tumours.

DIAGNOSTIC TESTS Ear disorders

NAME OF TEST Audiometry

PURPOSE AND DESCRIPTION Used to evaluate and diagnose conductive and sensorineural hearing loss. Person sits in soundproof room and responds by raising a hand or pressing a button when sounds are heard.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Auditory evoked potential (AEP)

PURPOSE AND DESCRIPTION Used to identify electrical activity of the auditory nerve. Electrodes are placed on various areas of the ear and on the forehead, and a graphic recording is made.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Auditory brainstem response (ABR)

PURPOSE AND DESCRIPTION Measures electrical activity of the auditory pathway from inner ear to brain to diagnose brainstem pathology, stroke and acoustic neuroma.

RELATED NURSING CARE No special preparation is needed.

NAME OF TEST Caloric test

PURPOSE AND DESCRIPTION Used to assess vestibular system function. Cold or warm water is used to irrigate the ear canals one at a time and the person is observed for nystagmus (repeated abnormal movements of the eyes). Normally, the nystagmus occurs in the eye opposite to the ear being irrigated. If no nystagmus occurs, the person needs further testing for brain lesions.

RELATED NURSING CARE Assess person for use of alcohol, central nervous system depressants and barbiturates. These chemicals may alter the test results.

EAR AND HEARING ASSESSMENTS

Hearing assessment

Tuning forks are used to determine whether hearing loss is conductive or perceptive (sensorineural). Hold the tuning fork at the base and make it ring softly by stroking the prongs or by lightly tapping them on the heel of the opposite hand. The vibrating tuning fork emits sound waves of a particular frequency, measured in hertz (Hz). Tuning forks with a frequency of 512 to 1024 Hz are preferred for auditory evaluation because that range corresponds to the range of normal speech.

Technique/normal findings	Abnormal findings
Perform the Weber test. Place the base of a vibrating tuning fork on the midline vertex of the person's head (see Figure 44.11). Ask whether the person hears the sound equally in both ears or better in one than the other. *Sound is normally heard equally in both ears.*	■ Sound heard in, or lateralised to, one ear indicates either a conductive loss in that ear or a sensorineural loss in the other ear. The sound will be louder on the impaired side with conductive hearing loss. Conductive losses may be due to a build up of cerumen, an infection such as otitis media or perforation of the eardrum. The sound will be softer on the impaired side, suggesting sensorineural hearing loss.
Perform the Rinne test. Place the base of a vibrating tuning fork on the person's mastoid bone. Ask the person to indicate when the sound is no longer heard. When the person does so, quickly reposition the tuning fork in front of the person's ear close to the ear canal. Ask whether the person can hear the sound. If the person says yes, ask the person to indicate when the sound is no longer heard. Repeat over the opposite mastoid bone (see Figure 44.12). *The person with no conductive hearing loss will hear the sound twice as long by air conduction as by bone conduction.*	■ Bone conduction is greater than air conduction in the ear with a conductive loss. The normal pattern is air conduction greater than bone conduction (AC > BC).

FIGURE 44.11 ■ Performing the Weber test with a tuning fork

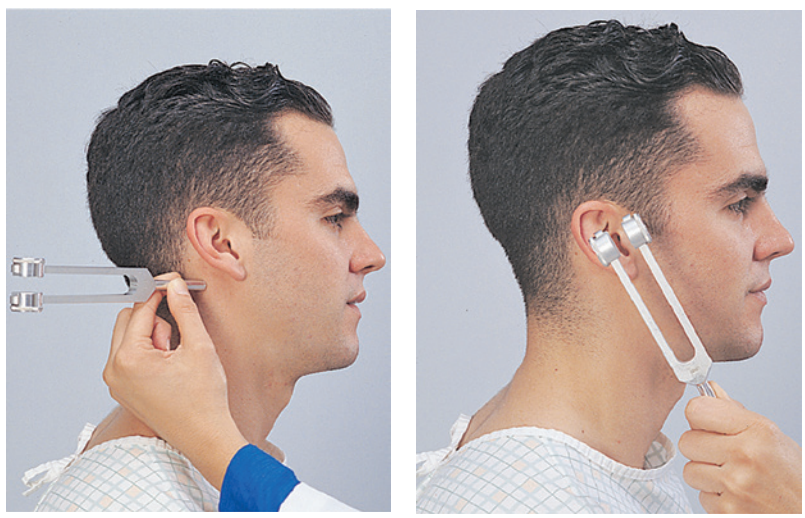


FIGURE 44.12 ■ Performing the Rinne test with a tuning fork

Technique/normal findings	Abnormal findings
Perform the whisper test. Ask the person to occlude one ear with a finger. Stand 30–60 cm away from the person, on the side of the opposite ear. Softly whisper numbers and ask the person to repeat them. Repeat the procedure, having the person occlude the other ear. Note whether you need to raise your voice or to stand closer to make the person hear you.	■ This test provides a rough estimate of hearing loss.
Use a tympanogram to measure the pressure of the middle ear and observe the tympanic membrane's response to waves of pressure. Insert the device into the ear canal. Ask the person not to speak, move, swallow or jump when hearing a sound. Tell the person they will hear a loud tone as the measurements are taken. The normal pressure inside the middle ear is a 100 daPa (a very small amount). Repeat for the other ear.	■ Abnormal findings may include fluid in the middle ear, a perforated eardrum, impacted earwax or a tumour of the middle ear.
External ear	
Inspect the auricle. *External ears are normally bilaterally equal in size, of equal colour with the person's face, and without redness or lesions.*	■ Unusual redness or drainage may indicate an inflammatory response to infection or trauma. ■ Scales or skin lesions around the rim of the auricle may indicate skin cancer. ■ Small, raised lesions on the rim of the ear are known as tophi and indicate gout.
Inspect the external auditory canal with the otoscope. *Canal walls should be pink and smooth without lesions. Cerumen is normally present in small, odourless amounts.*	■ Unusual redness, lesions or purulent drainage may indicate an infection. ■ Cerumen varies in colour and texture, but hardened, dry or foul-smelling cerumen may indicate an infection or an impaction of cerumen that requires removal. People with darker skin tend to have darker cerumen.
Inspect the tympanic membrane. *The tympanic membrane should be pearly grey, shiny and translucent without bulging or retraction.*	■ White, opaque areas on the tympanic membrane are often scars from previous perforations (see Figure 44.13). ■ Inconsistent texture and colour may be due to scarring from previous perforations caused by infection, allergies or trauma. ■ Bulging membranes are indicated by a loss of bony landmarks and a distorted light reflex. Such bulges may be the result of otitis media or malfunctioning auditory tubes. ■ Retracted tympanic membranes are indicated by accentuated bony landmarks and a distorted light reflex. Such retraction is often due to an obstructed auditory tube.
Palpate the auricles and over each mastoid process. *There should be no pain or swelling on palpation.*	■ Tenderness, swelling or nodules may indicate inflammation of the external auditory canal or mastoiditis.

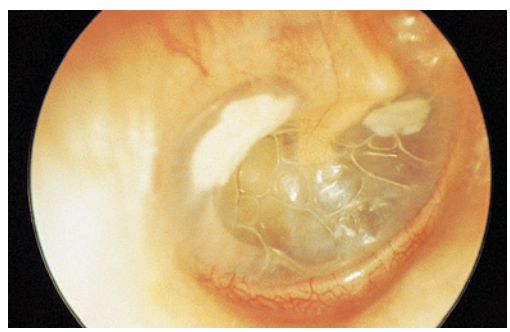


FIGURE 44.13 ■ Scarring of the tympanic membrane

Source: Professor Tony Wright/Science Source.

CONCEPT CHECK

1 During an eye assessment, you touch the part of the eye covering the iris and pupil. What normal response would you expect?
1 excess tearing
2 blinking of eyelids
3 bilateral nystagmus
4 pupil dilates

2 Which of the following statements would indicate a person has presbyopia?
1 'I am having so much trouble hearing music.'
2 'I can't seem to remember anything these days.'
3 'I think I have a lot of earwax in my ears.'
4 'My arms don't seem long enough for reading.'

3 What equipment would be necessary to test sound conduction during an assessment of the ear?
1 ophthalmoscope
2 tuning fork
3 otoscope
4 penlight

4 What occurs when light enters the lens of the eye?
1 accommodation
2 convergence
3 pupillary reflex
4 hyperopia

5 What would you tell a person before a test of refraction is done?
1 'This test is uncomfortable, but it doesn't take long.'
2 'You will be blindfolded during the test.'
3 'Are you allergic to seafood?'
4 'Your pupils will be dilated for several hours.'

6 Which function, in addition to hearing, is provided by the inner ear?
1 Coordinates visual pathways.
2 Integrates efferent neuron messages.
3 Provides information about head position.
4 Maintains middle ear structure and function.

7 Why is the Snellen eye chart used during vision assessment?
1 to test distant vision
2 to test near vision
3 to determine visual fields
4 to examine convergence

8 Of the following ear assessments, which one is a rough estimate of the ability to hear?
1 whisper test
2 Rinne test
3 Weber test
4 audiometry

9 What is a high-priority risk for the older adult with age-related changes in the vestibular structures of the ear?
1 infection
2 falls
3 medication errors
4 food intolerance

10 Which criterion is important to accurately assess visual fields?
1 Person must wear corrective lenses.
2 Person must have no less than 6/9 vision.
3 Examiner must not wear glasses.
4 Examiner must have normal visual field.

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CHAPTER 45

NURSING CARE OF PEOPLE WITH EYE AND EAR DISORDERS

KAMAREE BERRY

KEY TERMS

LEARNING OUTCOMES

- Relate knowledge of normal anatomy, physiology and sensory functions of the eye and ear to the effects of disorders of these organs on the cognitive/perceptual functional health pattern.
- Describe the pathophysiology of commonly occurring disorders of the eyes and ears, relating their manifestations to the pathophysiological process.
- Explain the risk factors for selected disorders of the eyes and ears, identifying the nursing implications for these risk factors.
- Identify diagnostic tests used for specific eye and ear disorders.
- Discuss the effects of and nursing implications for medications prescribed to treat eye and ear disorders.
- Describe surgical and other invasive procedures used to treat eye and ear disorders, identifying their implications for nursing care.
- Discuss the nurse's role in caring for a person with impaired vision or hearing loss.

CLINICAL COMPETENCIES

- Assess vision, hearing and functional health of people with eye and ear disorders.
- Using assessed data, determine priority nursing interventions and care for people with eye and ear disorders.
- Collaborate with other members of the healthcare team to provide effective care for people with eye and ear disorders.
- Plan and implement appropriate and individualised evidence-based nursing interventions and education for the person with an eye or ear disorder.
- Safely and effectively administer eye and ear medications and prescribed treatments.
- Provide appropriate care and education for the person having eye or ear surgery.
- Evaluate the effectiveness of nursing care provided for people with eye and ear disorders and revise the plan of care as indicated.

Vision and hearing provide the primary means of input for much of what we know about the world. The ability to receive and organise information orients us to our surroundings. These senses allow us to communicate easily, gain access to information and derive pleasure from the sights and sounds of the world around us.

This chapter discusses conditions affecting vision and hearing as the result of eye and ear disorders. Nursing care focuses on people with vision and hearing deficits that can result from the disorders presented.

EYE DISORDERS

Any portion of the eye and its protective structures may be affected by an acute or a chronic condition. While many disorders of the eye are minor and have little or no effect on vision, others can and often do result in permanent vision impairment. Disorders and diseases of the outer, visible portion of the eye often cause discomfort and may have cosmetic effects. Vision impairment can often be prevented or reversed with appropriate treatment.

Disorders affecting the internal structures or the function of the eye are more likely to have adverse effects on vision (e.g. disorders of the cornea present the greatest risk to vision in this group) and are more likely to affect adults over the age of 40 and the older adult. The person who has had eye surgery or minor trauma may experience either temporary or permanent visual impairment.

Although disorders that commonly affect vision cannot often be prevented or cured, some can be controlled with vision corrected to normal or near normal. Regardless of the real threat a disorder poses to vision, the person may experience anxiety related to a perceived threat. The box below discusses major causes of significantly impaired vision and nursing care for the person who is blind. These principles of nursing care may apply to people with many of the disorders discussed in this chapter.

THE PERSON WITH CONJUNCTIVITIS

The conjunctiva—the thin, transparent membrane that covers the anterior surface of the eye and lines the inner surfaces of the eyelids—is vulnerable to inflammation and infection because of its constant exposure to the environment. **Conjunctivitis**, inflammation of the conjunctiva, is the most common eye disease caused by a bacterial or viral infection. These infections can be transmitted to the eye by direct contact (e.g. hands, tissues, towels). Allergens, chemical irritants and exposure to radiant energy such as ultraviolet light from the sun or tanning devices can also lead to this common condition. Its severity can range from mild irritation with redness and tearing to conjunctival oedema, haemorrhage or a severe necrotising process with tissue destruction.

Pathophysiology and manifestations

Acute conjunctivitis

Infectious conjunctivitis may be bacterial, viral or fungal in origin. Bacterial conjunctivitis, also known as 'pink eye', is highly contagious and often is caused by *Staphylococcus* and *Haemophilus*. Adenovirus infection is the leading cause of conjunctivitis in adults. Systemic infections that may affect the eyes include herpes simplex and other viral infections. Contact with genital secretions infected with *Gonococcus* can cause gonococcal conjunctivitis, a medical emergency that can lead to corneal perforation.

Redness and itching of the affected eye are common manifestations of acute conjunctivitis (see Figure 45.1). The person may also complain of a scratchy, burning or gritty sensation. Pain is not common; however, photophobia may occur. Tearing and discharge accompany the inflammatory process. The discharge may be watery, purulent or mucoid, depending on the cause of conjunctivitis. The person may have associated manifestations such as pharyngitis, fever, malaise and swollen pre-auricular lymph nodes.

Trachoma

Trachoma, a chronic conjunctivitis caused by *Chlamydia trachomatis*, is a significant preventable cause of blindness worldwide. Trachoma is endemic in sub-Saharan Africa, the Middle East and parts of Asia. Australia is the only

FAST FACTS

- In 2007–2008, 11 million (52%) Australians had a least one long-term vision disorder (Australian Institute of Health and Welfare (AIHW), 2012).
- The economic impact of visual impairment in Australia has been estimated at $9.85 billion. Vision disorder direct spending costs are more than the combined costs associated with stroke, arthritis and depression.
- The most common, presbyopia, affected 26% of the population; myopia affected 23%. Glaucoma, cataracts and macular degeneration affected 3% of the population. Other conditions included:
  - diabetic retinopathy: 2%
  - neuro-ophthalmic conditions: 2%
  - other retinal conditions: 7%
- Over the past 4 years, the Australian Government has committed $58 billion to deal with chronic ear and eye conditions in Indigenous Australians. The prevalence of eye disease is nearly 10 times higher in Indigenous Australians than in the general population.

Sources: Eye Research Australia (2004). *Clear insight: The economic impact and cost of vision loss in Australia—An overview of the report*; Australian Bureau of Statistics (2006). *Australian demographic statistics as at 30 June 2005 (Table 7) (Report 3101.0)*, provides the individual state and territory statistics; Vision 2020 Australia (2009); AIHW (2012).

NURSING CARE OF THE PERSON who is blind

Visual impairment exists on a continuum from blindness to decreased visual acuity that can be corrected with refractive lenses, to normal or near normal vision. *Visual impairment* is defined as 6/12 vision in the better eye, even with corrective lenses. The legal definition of *blindness* is visual acuity no better than 6/60 in the better eye with optimal correction or a visual field of less than 20 degrees in diameter (compared to the normal of 180 degrees) (Prevent Blindness America, 2008). Total blindness usually indicates that the person has no light perception at all. In practical terms, a person with a visual deficit sufficient to need assistive devices or aid from other people for normal activities of daily living is considered blind.

According to the World Health Organization (2014), there are 285 million people worldwide with visual impairment. Although blindness often can be prevented or cured, it remains a significant problem worldwide because of lack of access to care, fear of surgery or treatment, poor sanitation and nutrition, and ignorance of need. In Australia, cataracts, age-related macular degeneration, glaucoma and diabetic retinopathy remain significant causes of blindness in adults.

NURSING CARE

Blind people need to cope not only with the loss of a major sense but often also with societal attitudes that may make them feel inferior, helpless and inadequate. The idea of losing the ability to see is uniformly feared, leaving sighted people often unable to understand the magnitude and impact of the loss in those who have experienced it. Because of this fear and confusion, sighted people are unsure of what the blind expect from them.

The adjustment of the person who is born blind and raised to become an independent member of society differs from that of the person who has been sighted and becomes blind. The person who has been blind from birth has developed numerous adaptive strategies that the newly blind person has yet to learn.

Although adaptation may be easier for the person who experiences a gradual loss of vision than for someone with an abrupt loss, both grieve the lost sense. The blind person needs to grieve the lost body part as well as the loss of mobility, self-sufficiency, perhaps economic security and, to a certain extent, contact with reality as it has previously been perceived. The person's self-concept and self-esteem are threatened. Anger, denial, remorse and self-pity are not uncommon in the initial period following loss of sight. Interpersonal relationships and roles are affected and communication patterns change with the loss of the ability to perceive many non-verbal cues. In addition, expressions of sexuality may be impaired.

Acceptance of the change from sighted to blind is characterised by releasing the hope that vision will be regained. Self-esteem increases as the person attempts and masters activities of self-sufficiency such as completing ADLs, cooking and becoming mobile outside the known home environment.

Health professionals often confuse the blind person with someone helpless, dependent and lacking in personal identity and control. Although nurses need to take blindness into account in planning care and maintaining the person's safety, it is vital to give the blind person the same respect and decision-making control and power that all people deserve. Nurses who have dealt with their own emotions and responses to vision loss are better prepared to help the person adapt.

Nurses can foster independence in the hospitalised person with a significant vision deficit by carrying out the following:

- Orientation to the environment verbally and physically. Describe the room using a central point such as the bed, then lead the person around the room, identifying chairs, sink, bathroom and other landmarks. Be sure that objects such as chairs, personal items and clothing remain in the same place unless moved by the person. Leave doors either fully open or closed as the person requests, but preserve safety by not leaving doors partially open. Keep the room and hallways free of clutter.
- Use verbal communication freely. Introduce yourself as you enter the room and let the person know when you are leaving, and describe activities going on around the person.
- Provide other sensory stimuli such as radio and television, as desired by the person.
- Orient to food trays by using the face of a clock to describe the position of food items on the plate and tray (unless the person has always been blind and cannot visualise a clock face).
- When assisting with ambulation, allow the person to hold your arm as you walk slightly ahead. Do not hold the person's arm. Verbally describe the environment; for example: 'There will be two steps up 1.5 metres ahead.'
- Do not hesitate to ask what assistance the person desires.

For the person with a new loss of sight, refer to available services. Counselling can help the person cope with and eventually adapt to the loss of sight. People who are blind can receive mobility training, assistance with relearning self-care activities, education in the use of Braille to communicate and vocational and other forms of rehabilitation. Local, state and national agencies such as the Association for the Blind, state and territory guide dog associations, Seeing Eye Dogs Australia, Australian Braille Authority, Vision Australia, Blind Welfare Association and Blind Citizens Australia coordinate services for the blind. They provide many assistive devices, including guide dogs, computer services, talking books and tape players, and low-vision aids.

developed country with endemic trachoma, with extremely high incidence in the Indigenous population. Trachoma is contagious, transmitted primarily by close personal contact (eye to eye, hand to eye) or by fomites such as towels, handkerchiefs and flies (Gaydos & Quinn, 2012). Certain forms of trachoma are transmitted during delivery when the newborn is exposed to contaminated genital secretions of the mother (Gaydos & Quinn, 2012).

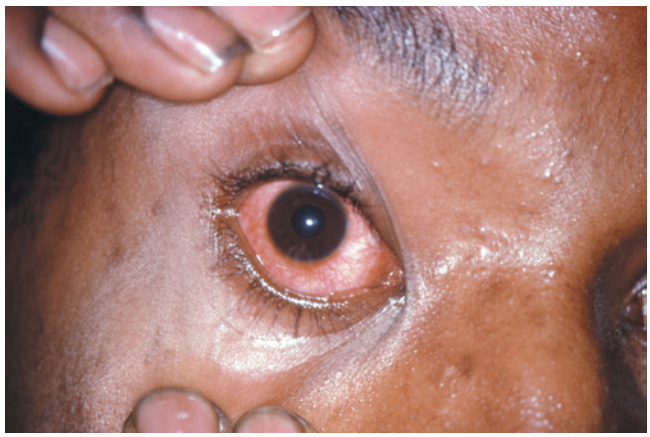


FIGURE 45.1 ■ The appearance of an eye with conjunctivitis

Source: CDC/Joe Miller.

Early manifestations of trachoma include redness, eyelid oedema, tearing and photophobia. Small conjunctival follicles develop on the upper lids. The inflammation also causes superficial corneal vascularisation and infiltration with granulation tissue. Scarring of the conjunctival lining of the lid causes entropion (see Figure 45.2). The lashes then abrade the cornea, eventually causing ulceration and scarring. The scarred cornea is opaque, resulting in loss of vision.

INTERPROFESSIONAL CARE

Management of the person with conjunctivitis focuses on establishing an accurate diagnosis and prompt treatment.

Diagnosis

Diagnosis is especially important because of other potentially vision-threatening conditions, such as acute uveitis or acute angle-closure glaucoma, that can also cause acute red eye (see Table 45.1). Diagnostic procedures may include:

- *culture and sensitivity* of exudates to determine presence of an infection and identify the infecting organism

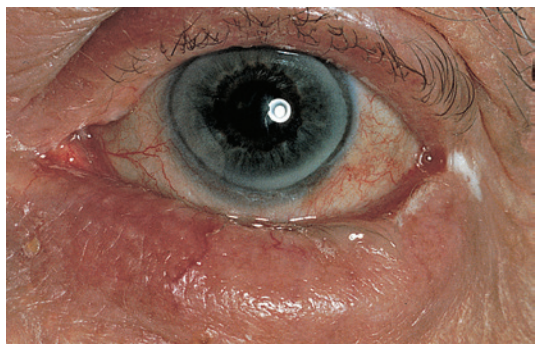


FIGURE 45.2 ■ Entropion

Source: © Science Photo Library/Science Source.

- *fluorescein stain* with slit-lamp examination to identify possible corneal ulcerations or abrasions, which appear green with staining
- *conjunctival scrapings* that are examined microscopically or cultured to identify the organisms.

Additional laboratory testing such as blood counts or antibody titres may be used to identify underlying infectious or autoimmune processes.

Medications

Conjunctivitis is treated with antibiotic, antiviral or anti-inflammatory drugs as appropriate. Topical antibiotic eye drops or ointments are usually prescribed with severe cases of conjunctivitis. Trachoma is usually treated with a single dose of oral azithromycin (Bryant & Knights, 2014). Antihistamines are used to minimise symptoms of conjunctivitis when an allergic response underlies the inflammatory process.

Complementary therapies

Frequent eye irrigations may be ordered to remove the copious purulent discharge associated with conjunctivitis. Soaking the lids with warm saline compresses prior to cleansing promotes comfort and facilitates the removal of crusts and exudate in conjunctivitis.

TABLE 45.1 Possible causes of acute red eye

	ACUTE CONJUNCTIVITIS	CORNEAL TRAUMA OR INFECTION	ACUTE UVEITIS	ACUTE ANGLE-CLOSURE GLAUCOMA
Incidence	Very common	Common	Common	Rare
Pain	Mild	Moderate to severe	Moderate	Severe
Vision	Normal	Blurred	Blurred	Markedly blurred
Discharge	May be copious	Watery, may be purulent	None	None
Conjunctival erythema	Diffuse	Primarily around cornea	Primarily around cornea	Primarily around cornea
Cornea	Clear	Depends on cause	Usually clear	Cloudy
Pupils	Normal size, response to light	Normal size, response to light	Small, minimal response to light	Moderately dilated, fixed

Nursing care

The nursing role in treating conjunctivitis is primarily one of education to prevent the disorder and its spread when it does occur.

Health promotion

Education is a vital strategy for preventing conjunctivitis. Teach all people about proper eye care, including the importance of not sharing towels, make-up or contact lenses, and avoiding rubbing or scratching the eyes. Instruct to avoid using old eye make-up, which can cause eye infections. Educate contact lens users about appropriate care (see Box 45.1). Emphasise the need to follow precise cleaning instructions to avoid bacterial contamination of lenses. If the eyes become red, irritated or develop discharge, instruct the person to avoid wearing contact lenses until the inflammatory process has cleared.

Assessment

- *Health history:* presence of redness, discomfort, tearing, photophobia and drainage; symptom onset; care measures; use of contact lenses; exposure to 'pink eye' or recent travel; allergies; previous history of conjunctivitis; presence of any chronic diseases.
- *Physical assessment:* visual acuity; inspect eyelids, conjunctiva, sclera and cornea; vital signs (temperature, heart rate, respirations, blood pressure).

Nursing diagnoses and interventions

Nursing care focuses primarily on preventing complications from the disorder. The priority nursing diagnoses include risk of infection and altered vision.

Risk of infection

Acute conjunctivitis is highly contagious. While most people experience discomfort from the disease, the infection carries a risk of scarring and damage to the delicate cornea. Preventing the spread of this infection is a vital nursing role.

BOX 45.1 Contact lens care

- Wash hands thoroughly before handling contact lenses.
- Keep storage case clean.
- Remove lenses before sleep, cleaning and storing as recommended by the manufacturer, or dispose of if single-use only.
- Use cleaning and wetting solutions recommended by the eye care professional or lens manufacturer. Do not use water or home-made solutions for wetting or cleaning lenses.
- If eye redness, tearing, vision loss or pain occurs, remove lenses and contact eye care professional as soon as possible.
- Do not share contact lenses with another person.

- Educate to wash hands thoroughly before instilling eye medications. Instruct to avoid touching or rubbing the eyes. Advise to use a new, clean cotton-tipped swab or cotton ball for cleaning each eye. *Handwashing is the single most important measure to prevent transmission of infection to the eye. Touching or rubbing the eyes increases the risk of infection and corneal trauma. Using a new swab or cotton ball prevents cross-contamination between eyes.*
- Educate person to instil prescribed eye drops as ordered. *Prescribed medications reduce inflammation and eliminate infection.*
- Discuss the importance of avoiding contact lens use until the infectious process has cleared, and of completing the prescribed treatment. *Use of contact lenses in the inflamed eyes can lead to further damage and impair healing.*

Risk of disturbed visual perception

Conjunctivitis can potentially disrupt the integrity or clarity of the cornea. Because of its vital role in focusing light on the retina, corneal damage can impair visual acuity.

- Assess vision with and without corrective lenses. *Assessment provides a baseline to evaluate possible changes in vision resulting from the infection.*
- Instruct to avoid activities requiring high visual acuity until the infection has cleared. *The inflammatory process, oedema of the conjunctiva and local antibiotic applications can decrease visual acuity and cloud vision.*
- Instruct to use dark sunglasses with appropriate UV protection when outdoors, even on cloudy days. *Photophobia, a common manifestation of conjunctivitis, causes eye pain with increased light intensity.*

Community-based care

People with conjunctivitis are typically managed in the community, reinforcing the need for effective education for home care. Emphasise to the individual and family ways to prevent transmission of infection. If the person is unable to administer eye medications, involve and educate the family in the following:

- safety and medical asepsis when cleansing the eye
- instillation of prescribed eye drops and ointments
- comfort measures such as reducing lighting intensity and wearing sunglasses
- avoiding activities such as excessive reading while the eye is inflamed.

THE PERSON WITH A CORNEAL DISORDER

The clear cornea allows light rays to enter the eye and transmits images onto the retina. It helps to focus light on the retina and protects the internal eye structures. The cornea can be affected by a variety of disorders, including infection and

trauma. While the cornea heals quickly after minor injuries or abrasions, injury to its deeper layers can delay healing or result in scarring.

Physiology review

The cornea has three main layers: the outermost epithelium, which consists of five or six layers of cells that are constantly being renewed; the stroma, which makes up 90% of corneal tissue; and the single-cell-thickness endothelium adjacent to the aqueous humour of the anterior chamber. The cornea is avascular tissue; the central cornea is dependent on atmospheric oxygen to meet its metabolic needs. Because there is no blood supply, immune defences have difficulty fending off infections of the cornea.

Pathophysiology and manifestations

When light enters the eye through the normally clear curved cornea, it is bent or *refracted* onto the lens, which then focuses the light on the sensory cells of the retina. A change in the curvature of the cornea or in its clarity affects the ability of the eye to clearly focus; as a result, vision is distorted or blurred. Refractive errors such as myopia, hyperopia and astigmatism are common. Corneal scarring or ulceration are two main causes of blindness worldwide.

Refractive errors

Refractive errors are the most common problems affecting visual acuity; they result from an abnormal curvature of the cornea or an altered shape of the eyeball. People with *emmetropia* (normal vision) can see near and far objects clearly because light rays focus directly on the retina. In myopia (nearsightedness) the curvature of the cornea is excessive or the eyeball is elongated, causing the image to focus in front of the retina instead of on it. Objects in close range are seen clearly and those at a distance are blurred. The eyeball is too short in **hyperopia** (farsightedness), causing the image to focus behind the retina. People with this condition see objects at a distance more clearly than they see those close to them.

Astigmatism develops due to an irregular or abnormal curvature of the cornea. Instead of the round, even curvature, it curves more in one direction than the other, resembling the back of a spoon. As a result, light rays focus on more than one area of the retina, distorting both near and distance vision.

Keratitis

Keratitis is inflammation of the cornea. (When the inflammatory process involves both the conjunctiva and the cornea, the term *keratoconjunctivitis* may be used.) Keratitis may be caused by infection, hypersensitivity reactions, ischaemia, tearing defects, trauma and impaired innervation of the cornea. Scarring that occurs as a result of keratitis is a leading cause of blindness worldwide (Porth & Matfin, 2014).

Keratitis is described as either non-ulcerative or ulcerative. In *non-ulcerative keratitis*, all layers of corneal epithelium are affected, but remain intact. Viral infections, tuberculosis and autoimmune disorders such as lupus erythematosus may cause non-ulcerative keratitis. *Ulcerative keratitis*, in contrast, affects the epithelium and stroma of the cornea, leading to tissue destruction and ulceration. Bacterial conjunctivitis (e.g. *Staphylococcus*, *S. pneumoniae*, *Chlamydia*) may lead to ulcerative keratitis.

Keratitis commonly causes tearing, discomfort ranging from a gritty sensation in the eye to severe pain, decreased visual acuity and *blepharospasm* (spasm of the eyelid and inability to open the eye). A discharge may be present, especially if the conjunctiva is also inflamed. Corneal ulceration may be visible on direct examination.

Corneal ulcer

A **corneal ulcer**, local necrosis of the cornea, may be caused by infection, exposure trauma or the misuse/overuse of contact lenses. Herpes viruses (e.g. herpes simplex and herpes zoster) are a leading cause of ulcerative corneal disease. Corneal ulcers may also complicate bacterial conjunctivitis, trachoma, gonorrhoea and other acute infections. People who are immunosuppressed because of disease or drug therapy are at particular risk of developing corneal ulcers due to infection.

In corneal ulceration, a portion of the epithelium and/or stroma is destroyed. Ulcers may be superficial or deep, penetrating underlying layers and posing a risk of perforation. Fibrous tissue may form during healing, resulting in scarring and opacity of the cornea. Perforation can lead to infection of deeper eye structures or extrusion of eye contents. Partial or total vision loss may result.

Corneal dystrophies

A corneal dystrophy is accumulation of cloudy material in part or parts of the normally clear cornea, potentially affecting visual acuity. Corneal dystrophies are typically inherited disorders that progress gradually and affect both eyes. *Keratoconus*, progressive thinning of the cornea, is the most common corneal dystrophy. It typically affects teenagers and young adults. In keratoconus, the centre of the cornea thins and bulges outwards, affecting its shape and ability to focus light on the lens of the eye. In most cases, the thinning stabilises over time. In Australia, there is a 98.1% success rate in reconstructive surgery for this condition in which corneal transplantation and the sclera are used.

INTERPROFESSIONAL CARE

Management of the person with a disorder of the cornea focuses on establishing an accurate diagnosis and prompt treatment to reduce the risk of permanent vision deficit. The person's history and physical assessment are key in diagnosing these disorders.

Although many eye disorders can be treated in the community, the person with a severe corneal infection or ulcer may require hospitalisation. Corneal ulcers are medical emergencies that require prompt referral to an ophthalmologist for treatment. Pressure dressings may be applied to both eyes for comfort in order to reduce the risk of perforation and possible loss of eye contents.

Diagnosis

Visual acuity is tested on all people presenting with refractory or corneal disorders. See Chapter 44 for more information about

testing visual acuity. The following tests may be ordered to identify the cause and extent of eye infections or inflammations:

- *Fluorescein stain* with slit-lamp examination allows visualisation of any corneal ulcerations or abrasions, which appear green with staining.
- *Conjunctival* or *ulcer scrapings* are examined microscopically or cultured to identify the organisms.

Additional laboratory testing such as blood counts or antibody titres may be used to identify any underlying infectious or autoimmune disease processes.

Medication

Infectious processes are treated with antibiotic or antiviral therapy as appropriate. The only topical antiviral available in Australia is aciclovir (Bryant & Knights, 2014). Corticosteroids may be prescribed for keratitis related to systemic inflammatory disorders or trauma; however, it is important to avoid their use with local infections to avoid suppressing the immune and inflammatory responses.

Corrective lenses

Corrective lenses, either in the form of eyeglasses or contact lenses, generally are prescribed to restore visual acuity for people with refractive errors such as myopia, hyperopia and astigmatism. Specially fitted contact lenses to reduce vision distortion are ordered for people with keratoconus. Because contact lenses are a risk factor for corneal infection and ulcers, providing appropriate education regarding lens care is vital (see Box 45.1).

Surgery

LASER EYE SURGERY Laser eye surgery is commonly performed to correct refractive errors such as myopia, hyperopia and astigmatism. A laser is used to permanently change the shape of the cornea, and in most cases the need to use corrective lenses is reduced or eliminated. Several surgical procedures are now available:

- laser in situ keratomileusis (LASIK)
- photorefractive keratectomy (PRK)
- laser epithelial keratomileusis (LASEK)
- laser thermokeratoplasty (LTK).

These procedures reshape the cornea using laser technology to remove a thin layer of epithelial cells or to shrink and reshape the cornea. Candidates for laser vision-correction surgery should be in good health and must have adequate corneal thickness to ensure that the risk of perforation does not occur.

Following surgery, people may experience a temporary loss of contrast sharpness (images do not appear crisp as with corrective lenses), over- or under-correction of visual acuity, dry eyes or temporarily decreased night vision with halos, glare and starbursts. Diffuse lamellar keratitis (DLK) is a rare complication of surgery that, while treatable, can lead to vision impairment if not identified and treated early.

Phototherapeutic keratectomy (PTK) provides an alternative to corneal transplant in treating corneal dystrophies, scars and some infections. In this procedure, diseased corneal tissue is vaporised and surface irregularities are corrected with little trauma to surrounding tissue; healing occurs rapidly.

CORNEAL TRANSPLANT Once the cornea has become scarred and opaque, no treatment can restore its clarity. The first successful corneal transplant (*keratoplasty*), replacement of diseased cornea by healthy corneal tissue from a donor, was performed in 1906. Current corneal transplant procedures have a success rate of approximately 90% in Australia. Data collected from the Australian Corneal Graft Registry (2012), one of the world's largest registries, show that 91.2% of such transplants in Australia survive for approximately 1 year.

Corneas are harvested from the cadavers of uninfected adults who were under the age of 65 and who died as a result of acute trauma or illness. After harvesting, the cornea can be stored in a tissue-culture medium for up to 4 weeks before being used as a graft. Corneal transplantation is usually an elective surgery, although emergency transplantation may be required for perforation of the cornea.

Corneal transplant may be either lamellar or penetrating. In a lamellar keratoplasty, the superficial layer of cornea is removed and replaced with a graft. The anterior chamber remains intact. In a penetrating keratoplasty, a button or full thickness of cornea is removed and replaced by donor tissue (see Figure 45.3). The graft is then sutured in place using suture finer than human hair and a continuous or interrupted stitch. Because the cornea is avascular, these sutures remain in place for up to a year to ensure healing.

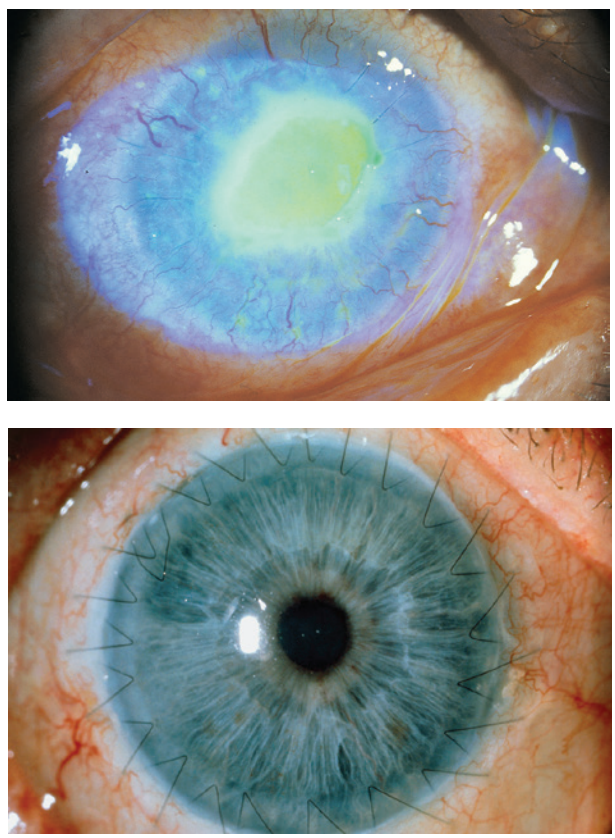
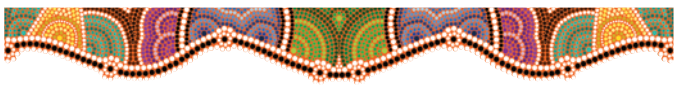


FIGURE 45.3 ■ Corneal transplant. *A*, The diseased, opaque cornea. *B*, The diseased cornea is removed and a corneal graft is sutured in place using material finer than a human hair

Source: Custom Medical Stock Photo.

Most corneal transplants do not require hospitalisation. The eye is patched for 24 hours following surgery. Narcotic analgesia may be required initially because the cornea is extremely sensitive. Corticosteroid eye drops are ordered to reduce the inflammatory response and prevent oedema of the graft, and antibiotic drops may be prescribed to prevent infection.

The risk of transplant rejection is low as the cornea is avascular and there is little exposure of the transplanted corneal tissue to the host's immune defences (Grossman & Porth, 2014). Research suggests that matching of blood type (not tissue type, as is required for major organ transplants) between the donor and the transplant recipient may reduce the risk of transplant rejection (Dana, 2011). When rejection does occur, it occurs within 3 weeks of the transplant, beginning with inflammation at the edge of the grafted tissue and spreading to involve the entire graft. In many cases, rejection can be successfully treated using corticosteroid therapy, preserving the graft. (See the accompanying box for nursing care of the person undergoing eye surgery.)



Nursing care

Nurses working in clinics and outpatient surgical settings care for people undergoing corneal transplant and surgeries to correct refractive errors. The nursing role in caring for people with corneal disorders may involve direct care; however, the focus is on prevention and education.

Health promotion

Education is a vital strategy for preventing many corneal disorders. Educate all involved about proper eye care, including the importance of not sharing towels and make-up, and avoiding rubbing or scratching the eyes, as well as preventing trauma and infection. Teach contact lens users appropriate care and cleaning techniques. Stress the importance of periodic removal of lenses, even extended-wear or single-use lenses. In general, lenses should be removed at night, even though manufacturers may claim it is safe to wear them while sleeping. Emphasise the need to follow cleaning instructions (as per the manufacturer's instructions) to avoid bacterial contamination of lenses and possible corneal infection. If the person experiences a corneal abrasion or keratitis, instruct the person to avoid wearing contact lenses until the cornea has healed completely.

Assessment

Collect the following data through a health history and physical examination (see Chapter 44). Additional focused assessments are described with the interventions below:

- *Health history:* risk factors; presence of redness, discomfort, tearing, photophobia, oedema and drainage; symptom onset; presence of pain, effect on vision.
- *Physical assessment:* visual acuity; inspect external eye, including conjunctiva, sclera and cornea; extraocular movements.

Nursing diagnoses and interventions

Nursing care focuses primarily on preventing complications and promoting healing. The priority nursing diagnosis for people with corneal disorders includes risk of altered vision, pain and injury.

Risk of disturbed visual perception

Disorders affecting the cornea may disrupt its integrity and/or clarity as the cornea plays a vital role in focusing light on the retina. Any corneal damage can affect vision, impairing visual acuity and even causing legal blindness.

- Assess vision with and without corrective lenses. *Assessment provides a baseline to evaluate possible vision changes resulting from the disorder and/or treatment.*
- Educate the person about thorough handwashing before inserting or removing contact lenses or instilling any eye medications, and about avoiding touching or rubbing the eyes. Instruct to use a new, clean cotton-tipped swab or cotton ball for cleaning each eye. *Handwashing is the single most important measure to prevent transmission of infection to the eye. Touching or rubbing the eyes increases the risk of infection and possible corneal trauma. Using a new swab or cotton ball prevents cross-contamination between eyes.*
- Emphasise the importance of proper care of contact lenses specific to the type of lens used. *People who wear hard contact lenses must remove them daily, as the central cornea requires exposure to atmospheric oxygen. Although soft and extended-wear lenses allow the cornea to 'breathe', improper cleaning carries a major risk of infection.*
- Emphasise the importance of using eye protection when engaging in potentially dangerous activities. *Trauma can increase the risk of infection and scarring of the cornea.*
- If corneal perforation is suspected, place the person in the supine position, close the eye and cover it with a dry, sterile dressing. Notify the doctor immediately. *Corneal perforation may occur without warning in people with corneal ulcers. It places the person at risk of loss of eye contents. Emergency measures are taken to reduce intraocular pressure and maintain eye integrity to preserve vision.*

CONSIDERATION FOR PRACTICE

Suspect corneal perforation with complaints of sudden, severe eye pain and photophobia.

Acute pain

The cornea of the eye is extremely sensitive; therefore, corneal disorders frequently cause significant pain. This in turn increases the stress response and interferes with rest, potentially impairing healing.

- Assess pain, using verbal and non-verbal cues. *Pain is a subjective experience and can be evaluated only by the person's response and in terms of its effect on the person.*

- Administer prescribed analgesia routinely in the first 12 to 24 hours after corneal surgery. *Routine administration of analgesics prevents pain from reaching a level of severity at which it becomes difficult to relieve.*
- Patch both eyes if necessary. *Patching both eyes reduces eye movement and irritation of the affected eye.*
- Instruct to apply warm compresses to reduce inflammation and pain. *Warm compresses for 15 minutes, three to four times a day, promote comfort for people with keratitis or corneal injury.*
- Instruct to use dark sunglasses with appropriate UV protection when outdoors, even on cloudy days. *True photophobia, often associated with corneal disorders, causes eye pain with increased light intensity.*

NURSING CARE OF THE PERSON having eye surgery

PREOPERATIVE CARE

- Review Chapter 3 for routine preoperative care.
- Assess visual acuity of the affected eye prior to surgery. *The person with limited vision in the affected eye may need additional attention and ADL assistance postoperatively to ensure safety.*
- Assess the person's support systems and the possible effect of impaired vision on lifestyle and ability to perform ADLs in the postoperative period. *Safety measures such as installing handrails and removing throw rugs from the home can help promote mobility and safety, especially if the person has limited vision in the unaffected eye.*
- Educate the person regarding measures to prevent eye injury postoperatively, such as avoiding possible vomiting, straining with stool movement, coughing, sneezing, lifting more than 2 kg and bending over at the waist. *These activities can temporarily increase intraocular pressure and may lead to postoperative complications.*
- Remove all eye make-up and contact lenses or glasses prior to surgery. Store corrective lenses and eyeglasses in a safe place and make them readily available to the person on return from surgery. *Maintaining visual acuity in the unaffected eye helps reduce fear and maintains safety.*
- Administer preoperative medications and eye drops and/or ointments as prescribed. *Mydriatic (pupil-dilating) or cycloplegic (ciliary-paralytic) drops, and/or drops to lower intraocular pressure, may be prescribed preoperatively.*

POSTOPERATIVE CARE

- Review Chapter 3 for routine postoperative care.
- Assess eye dressing for bleeding and/or drainage following surgery *as it may indicate a surgical complication.*
- Maintain the eye patch or shield in place *to prevent inadvertent injury to the operative site.*
- Place in semi-Fowler's or Fowler's position on the unaffected side. *Elevating the head of the bed and lying on the unaffected side reduces intraocular pressure in the affected eye.*
- Remind the person to avoid coughing, sneezing and/or straining, *as these activities can increase intraocular pressure.*
- Assess and medicate as necessary for complaints of pain, aching and/or a scratchy sensation in affected eye. *Complaints of sudden sharp eye pain must be reported to the doctor immediately. An abrupt increase in or onset of eye pain may indicate haemorrhage or other ocular emergency requiring immediate intervention to preserve sight.*
- Assess for potential complications:
  a. pain and/or drainage from the affected eye
  b. haemorrhage with blood in the anterior chamber of the eye
  c. indications of retinal detachment, including flashes of light, floaters or the sensation of a curtain being drawn over the eye; cloudy appearance to the cornea (corneal oedema).

  Early intervention is often necessary to preserve sight.
- Approach the person on the unaffected side *as this facilitates eye contact and communication.*
- Place personal articles and the call bell within easy reach *to prevent the person stretching and/or straining.*
- Administer antibiotics, anti-inflammatory and/or other systemic and/or topical eye medications as prescribed. *Medications are prescribed to prevent infection and/or inflammation of the operative site, maintain pupil constriction and control intraocular pressure.*
- Administer anti-emetic medication as required. *It is important to prevent vomiting, in order to maintain normal intraocular pressures.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Educate the person and family about home care:
  a. how to instil eye drops
  b. the name, dosage, schedule, duration, purpose and side effects of medications
  c. how and when to use the eye patch and eye shield
  d. the importance of avoiding scratching, rubbing, touching or squeezing the affected eye
  e. measures to avoid constipation and straining
  f. activity limitations, if ordered
  g. reporting of symptoms including eye pain or pressure, redness or cloudiness, drainage, decreased vision, floaters, or flashes of light or halos around bright objects
  h. the need to wear sunglasses with side shields when outdoors to reduce photophobia
- Remind the person that vision may not stabilise for several weeks following eye surgery. If new corrective lenses are required they will not be prescribed until vision has stabilised. *A person may be alarmed that vision seems worse after surgery and need reassurance that visual acuity usually improves with time and healing of the affected eye.*
- Emphasise the importance of keeping follow-up appointments. Provide referral to a community service for assistance with home care after discharge as required.

- Instruct how to instil prescribed eye drops/ointment as ordered. *Prescribed medications may reduce inflammation and eliminate infection, reducing discomfort.*

Risk of injury

The person who has undergone corneal transplantation has an increased risk of injury for several reasons. The eye on which surgery was performed is patched for 24 hours after surgery, changing depth perception and increasing the risk of falls. Increased intraocular pressure or trauma to the eye may damage the graft, resulting in graft rejection.

- Instruct to call for help before getting up or ambulating after surgery. Ensure access to the call bell. It may take time for the person to adjust to changes in depth perception caused by the eye patch. *Assistance helps prevent falls that may not only injure the person but also traumatise the operative site.*
- Encourage deep breathing and use the incentive spirometer to promote lung expansion. *These important postoperative measures help prevent pulmonary complications. Coughing is avoided because it increases intraocular pressure.*
- Educate the person on how to apply an eye shield at night after the eye patch is removed. *An eye shield may be recommended at night to prevent inadvertent rubbing or trauma to the eye during sleep.*
- Encourage the person not to rub or scratch the eye. *Rubbing or scratching may disrupt suture lines or damage the grafted tissue.*
- Reinforce the importance of using eye protection during hazardous activities. *Following a corneal transplant, the person has the same risk of eye injury as other people who perform hazardous activities.*

CONSIDERATION FOR PRACTICE

Administer prescribed anti-emetics and stool softeners postoperatively to prevent vomiting and straining at stool—activities that increase intraocular pressure and can damage suture lines.

Community-based care

Following treatment, educate the person to manage these conditions at home. Emphasise to the person and family ways to prevent transmission of infection. If the person is unable to administer eye medications or to perform other eye care required, involve the family in the education session. The following topics should be included:

- safety and medical asepsis when cleansing the eye
- instillation of prescribed eye drops and ointments
- application of an eye patch/shield and where to obtain supplies
- avoidance of activities such as excessive reading while eye is inflamed
- follow-up appointments after corneal transplant surgery
- signs and symptoms of graft rejection
- avoidance of activities that increase intraocular pressure, such as straining, coughing, sneezing, bending over, lifting heavy objects
- helpful resources:
  - Lions Eye Institute: www.lei.org.au
  - state organ and tissue agencies.

DISORDERS AFFECTING THE EYELIDS

The eyelids and eyelashes are constantly exposed to the environment as they protect the eye from damage. When these structures are inflamed, deformed or their function is impaired, this affects both appearance and their protective functions.

Pathophysiology and manifestations

The most common disorder affecting the eyelids is *marginal blepharitis*, an inflammation of the glands and lash follicles on the margins of the eyelids. This inflammatory disorder can be caused by a staphylococcal infection, or it may be seborrhoeic in origin; commonly, both types are present. Seborrhoeic blepharitis is usually associated with seborrhoea (dandruff) of the scalp or eyebrows. Irritation, burning and itching of eyelid margins are common manifestations of blepharitis. The eye appears red rimmed with mucus discharge and there is crusting or scaling of lid margins. Lid margins may ulcerate, resulting in a loss of eyelashes.

Infection of one or more of the sebaceous glands of the eyelid may cause a **hordeolum (sty)**. Hordeolum is a staphylococcal abscess that may occur on either the external or internal margin of the lid (see Figure 45.4). An external hordeolum is characterised initially by acute pain at the lid margin with redness, and a small tender raised area is visible. The person may also experience

FIGURE 45.4 ■ Hordeolum

Source: © Francoise Sauze/Science Source.

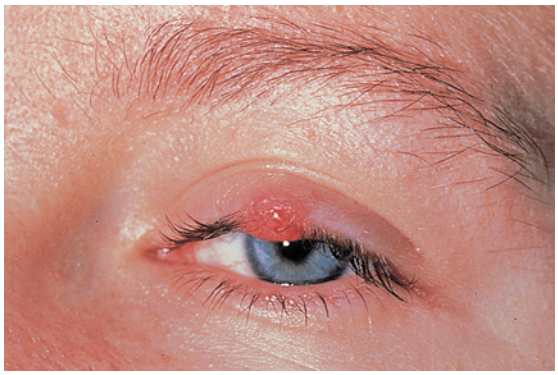


FIGURE 45.5 ■ Chalazion

Source: © SPL/Custom Medical Stock Photo.

photophobia, tearing and the sensation of a foreign body in the affected eye. Internal hordeola are seen on the conjunctival side of the lid and may have more severe manifestations.

Chronic inflammation of a meibomian gland may lead to formation of a **chalazion**, a granulomatous cyst or nodule of the lid (see Figure 45.5). It presents as a hard swelling on the lid, and the surrounding conjunctival tissue is red and inflamed. Chalazion may also follow a hordeolum that was inadequately treated. Unlike a hordeolum, a chalazion is painless; however, it may slowly increase in size and eventually require removal, although most resolve within several months.

Entropion, the inversion of the lid margin (see Figure 45.2), may be associated with the normal ageing process (senile entropion) or result from an infectious process such as trachoma. Entropion can lead to corneal irritation and, potentially, scarring as the lashes rub on the conjunctiva and cornea during blinking and sleep. *Ectropion*, or eversion of the lid margin, occurs primarily as an effect of ageing (see Figure 45.6). Other conditions may also lead to ectropion, including facial nerve paralysis or palsy (Bell's palsy), scarring or infection. Here the eye does not close effectively, increasing the risk of drying and damage to the conjunctival membrane and cornea. Both entropion and ectropion have cosmetic effects as they alter the appearance of the eyes and face.

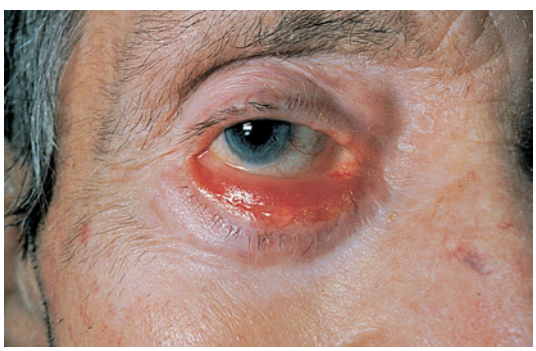


FIGURE 45.6 ■ Ectropion

Source: © Dr P. Marazzi/SPL/Science Source.

INTERPROFESSIONAL CARE

Disorders affecting the eyelids typically are managed in the community. Diagnosis is typically made through the person's history and physical examination. Diagnostic tests are rarely required except to identify corneal or conjunctival damage resulting from the condition.

Topical antibiotics (eye drops or ointments) may be prescribed for the person with hordeolum and to treat infection resulting from irritation of the eye by a deformed lid. Careful cleansing of the lid margins using a 'no-tears' baby shampoo is often recommended for marginal blepharitis. Soaking the lids with warm saline compresses prior to cleansing facilitates the removal of crusts and exudate in blepharitis. Local heat applications may be used to treat hordeolum or chalazion; excision and drainage may be required if this is not effective. In entropion or ectropion, surgery may be performed to correct the defect, reduce the risk of damage to the eye and improve cosmetic appearance.

Nursing care

The nursing role focuses on education and comfort measures. Educate people about appropriate eye care, including avoiding rubbing or scratching the eyes. Discuss the importance of not using old eye make-up, which can cause lid infections. Instruct to wash hands well before cleansing the eyelids or instilling any eye medications. Instruct to use a new, clean cotton-tipped swab or cotton ball for cleaning each eye. Instruct to instil prescribed eye drops and apply ointments as ordered. If the person is unable to administer eye medications, involve the family. Instruct to apply warm compresses to reduce inflammation and discomfort.

THE PERSON WITH EYE TRAUMA

Most eye injuries are minor but, without timely and appropriate intervention, even a minor injury can threaten a person's vision. Therefore, all eye injuries should be considered medical emergencies requiring immediate evaluation and intervention.

Pathophysiology and manifestations

Any part of the eye, especially the exposed parts, may be affected by trauma. Foreign bodies, abrasions and lacerations are the most common types of eye injury, along with traumatic injuries such as burns, penetrating objects and/or blunt force.

Corneal abrasion

Corneal abrasion is the disruption of the superficial epithelium of the cornea. Objects commonly causing corneal abrasion include contact lenses, eyelashes, small foreign bodies (dust, dirt and fingernails), drying of the eye surface and chemical irritants.

Superficial abrasions of the cornea are extremely painful but generally heal rapidly without complications or scarring. Photophobia and tearing are commonly present. When the stroma is damaged by a deep abrasion or laceration, there is an increased risk of infection, healing is slowed and scar formation occurs.

Burns

The outer surface of the eye may be subjected to burns caused by heat, radiation or explosion; however, chemical burns are the most common. Acid or alkaline substances may burn the eye. Ammonia, products that contain lye (such as oven and drain cleaners) and acids from car batteries or other sources are often implicated in eye injuries. Burns caused by alkaline substances are particularly serious as tiny particles of the chemical may remain in the conjunctival sac, causing progressive damage. Acid causes rapid damage to the eye; however, in general it causes less serious burns than alkaline substances.

Explosions and flash burn injuries pose the greatest risk of thermal burns of the eye. UV rays can also cause corneal damage ranging from mild to extensive. Depending on the source of the ultraviolet light, these burns may be known by various names such as snow blindness, welder's arc burn or flash burn.

In addition to giving a history of the face and eye contact with a caustic substance or other burning agent, the person complains of eye pain and decreased vision, and eyelids are often swollen. Burns may also affect the face or lids, and the appearance of the eye may vary, depending on the type of burn. The conjunctiva is reddened and oedematous; sloughing may be seen, particularly with chemical burns. The cornea often appears cloudy or hazy, and ulcerations may be evident.

Penetrating trauma

Perforation of the eye occurs from a variety of causes including metal flakes or other particles produced by high-speed drilling or grinding, glass shards or other substances. In a *perforating* injury, the layers of the eye do not spontaneously reapproximate, resulting in rupture of the globe and potential loss of ocular contents.

In a *penetrating* injury, the layers of the eye spontaneously reapproximate after entry of a sharp-pointed object or small missile into the globe (e.g. knives, arrows and gunshot). These injuries may not be readily apparent through inspection of the eye. Penetrating injuries may be hidden or missed due to tissue swelling or when the person has other significant injuries that command attention. When the eyelid is lacerated or has a puncture wound, inspection of the underlying eye tissue for possible damage is vital. Eye perforations/penetrations cause pain, partial or complete loss of vision, and possible bleeding or extrusion of eye contents.

Blunt trauma

Sports injuries are a common cause of blunt trauma to the eye; for example, being struck with a ball or injured in contact sports. Other causes include injury from a motor vehicle accident, falls and physical assault.

Blunt trauma may lead to a minor eye injury such as lid ecchymosis (black eye) or subconjunctival haemorrhage caused by rupture of a blood vessel in the conjunctiva. A well-defined bright area of erythema appears under the conjunctiva; however, no pain or discomfort is associated with the haemorrhage and no treatment is necessary. The blood typically reabsorbs within 2 to 3 weeks.

Hyphaema, bleeding into the anterior chamber of the eye, is a potential result of blunt eye trauma. When the highly vascular uveal tract of the eye is disrupted by blunt force, haemorrhage may result, filling the anterior chamber. The person complains of eye pain, decreased visual acuity and seeing a reddish tint. Blood is visible in the anterior chamber.

An orbital blowout fracture is another potential result of blunt eye trauma. Although any part of the eye orbit may be fractured, the ethmoid bone on the orbital floor is the most likely site. Orbital contents, including fat, muscles and the eye itself, may herniate through the fracture into the underlying maxillary sinus. The person complains of diplopia (double vision), pain with upward movement of the affected eye and decreased sensation on the affected cheek. The eye appears sunken (**enophthalmos**) and has limited movement on examination.

INTERPROFESSIONAL CARE

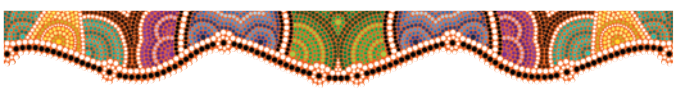
When trauma to the eye is known or suspected, a thorough examination is conducted to determine the type and extent of the injury. Unless immediate treatment is indicated, as with a chemical burn, vision is evaluated initially. If the person normally wears corrective lenses, vision assessment is performed while glasses are worn. Eye movement is evaluated unless a penetrating object is present and the lid and eye are inspected for lacerations. Inspection is performed using strong light and magnification with a headband loupe or slit lamp. Topical anaesthesia may be used prior to inspection if eye pain and photophobia make eye opening difficult. Fluorescein staining can help identify foreign bodies and abrasions. Any conjunctival or anterior chamber haemorrhage is noted, as is the presence or absence of the red reflex. Ophthalmoscopic examination is used to detect haemorrhage or trauma to the interior chamber. Facial x-rays and computed tomography (CT) scans are used to identify orbital fractures or foreign bodies within the globe. Ultrasonography may be employed to detect a detached retina or vitreous haemorrhage.

Foreign bodies are removed using irrigation, a sterile cotton-tipped applicator or a sterile needle or other instrument. Antibiotic ointments can be applied once objects have been removed. In a person with corneal abrasion and large foreign bodies, an eye patch is applied firmly after the antibiotic application to keep the eye closed for approximately 24 hours.

The immediate priority of care for people with chemical burns is flushing the affected eye with copious amounts of fluid. Normal saline is preferred; however, water may be used if saline is not available. A special contact lens irrigating unit (Morgan lens), normal saline eye bath or bag of irrigation with intravenous tubing held to flush all eye surfaces may be useful. The eyelid is everted to identify and remove material from the conjunctival sac. A topical anaesthetic helps relieve pain, making inspection and irrigation easier. During irrigation, fluid is directed from the inner canthus of the eye to the outer. Tipping the person's head slightly to the affected side prevents contamination of the unaffected eye. Irrigation is continued until the pH of the eye is normal (in the range 7.2 to 7.4). Following irrigation, a topical antibiotic ointment may be applied.

Penetrating wounds of the eye generally require surgical intervention by an ophthalmic surgeon. Immediate care focuses on relieving pain and protecting the eye from further injury. To prevent loss of intraocular contents, pressure should not be placed directly on the eye, but gently cover with a sterile gauze or an eye pad. If a foreign body is embedded in or sticking out of the eye, no attempt should be made to remove it. The object should be immobilised and the eye protected with a metal eye shield until the surgeon can review the person. A paper cup or other protective device may be used if the object is too large to use an eye shield. Patching the unaffected eye decreases ocular movement. Pain is managed using narcotic analgesics such as morphine. The person may also require sedation (e.g. diazepam), anti-emetic medications to prevent vomiting and antibiotics to prevent infection.

Interventions for the person with blunt trauma to the eye include placing the person on bed rest in semi-Fowler's position and protecting the eye from further injury with an eye shield. The unaffected eye is also patched to minimise eye movement. A carbonic anhydrase inhibitor may be prescribed to reduce intraocular pressure.



Nursing care

The nursing role involves educating people about prevention and providing direct care regarding eye injuries.

Health promotion

Educating individuals and groups about how to prevent eye injuries, the use of protective devices and first aid measures is an important nursing role, especially for people involved in hazardous occupations and high-risk sports. Emphasise the importance of using seat belts and air bags to prevent eye injury in motor vehicle accidents. Instruct people to immediately flush the eye with copious amounts of water if a chemical splash occurs. Loose, visible foreign bodies can be removed using a clean, moistened cotton-tipped swab. If an abrasion, penetrating or blunt injury is suspected, the eye should be covered loosely with sterile gauze and medical attention sought immediately; people should not try to remove objects.

Nursing diagnoses and interventions

Ocular injuries require immediate interventions simultaneously with assessment and accurate history collection, including pre-existing visual problems. The time, type and extent of injury, and the circumstances under which it occurred, must be determined.

Impaired ocular tissue integrity

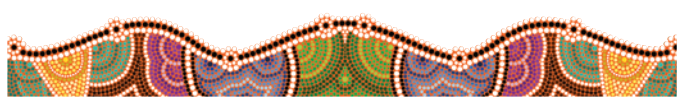
All types of eye trauma pose the risk of violating the integrity of the eye and threatening vision. The goals of nursing care are to preserve vision and the integrity of the eye, and prevent further damage.

- Assess vision in each eye followed by both eyes together, with and without corrective lenses, on entry into the emergency department or primary care setting. *An initial assessment provides valuable information about the effect of the injury on the person's vision and a baseline for future comparisons.*
- Inspect eye(s) carefully for evidence of foreign bodies, burns, penetrating injury or blunt trauma. Note if lacerations, burns or other trauma are evident in tissues surrounding the eye. *Eye trauma may be hidden by other injuries and, as a result, remain untreated.*
- If a burn or foreign body is present, anaesthetic drops may be instilled and the eye irrigated either before or after the doctor evaluates the person. *Blepharism and eye pain may impair assessment of the injured eye. Irrigation to remove the chemical is of higher priority than assessment of the eye.*
- Loose foreign bodies may be removed using a moist, sterile cotton-tipped applicator. *Prompt removal of foreign bodies may prevent corneal abrasion.*
- For a severe or penetrating injury, promote rest and stabilise the injured eye by applying an eye pad or gauze dressing loosely over both the affected and unaffected eye. Stabilise any penetrating object if possible. *These measures reduce eye movement and can help preserve the person's vision.*
- Following treatment, apply eye drops or ointment as prescribed and apply an eye pad or shield as required. *An eye pad is applied to the affected eye to reduce pain and photophobia and to promote healing.*

Community-based care

Following an injury, discuss the following topics with the person and family:

- prescribed medications and possible adverse effects
- strategies to prevent further trauma
- application of the eye pad or shield
- avoidance of activities that increase intraocular pressure
- importance of restricting activities.



THE PERSON WITH UVEITIS

The middle vascular layer of the eye, including the choroid, the ciliary body and the iris, is known as the uvea and uveal tract. *Uveitis* is inflammation of all or part of this vascular layer; *iritis*, inflammation of the iris, occurs more commonly than uveitis.

Uveitis is a disease limited to the eye; it may be idiopathic or caused by an autoimmune process, infection, parasitic disease or trauma. Many cases can be linked to a systemic disease, often an arthritic or autoimmune disorder such as ankylosing spondylitis, Reiter's syndrome, rheumatoid arthritis or sarcoidosis (see Chapter 39). Uveitis has also been linked with tuberculosis and syphilis. Manifestations of uveitis include pupillary constriction and erythema around the limbus, severe eye pain and photophobia and blurred vision.

Immunosuppressive therapy may be used to suppress the inflammatory response in people with severe uveitis. Atropine may also be prescribed for associated inflammation of the iris. The person may require analgesics for pain management. Nursing care is supportive, focusing on promotion of comfort and education regarding the disorder and its management.

THE PERSON WITH CATARACTS

A **cataract** is an opacification (clouding) of the lens of the eye which can significantly interfere with light transmission to the retina and the ability to perceive images clearly.

Incidence and risk factors

Age is the greatest single risk factor for cataract; however, genetics may contribute to the risk, although the link is unclear. Environmental and lifestyle factors also play a role, such as long-term exposure to sunlight (UVB rays); cigarette smoking and heavy alcohol consumption are associated with earlier cataract development. Although senile cataracts are by far the most common, cataracts may also be congenital or acquired in origin. Eye trauma, including injury to the lens capsule by a foreign body, blunt trauma or exposure to heat or radiation, can precipitate cataract formation. Diabetes mellitus is associated with earlier development of cataracts, especially when the blood glucose level is not carefully controlled at or near normal levels. Certain drugs such as systemic or inhaled corticosteroids also prompt the formation of cataracts.

Pathophysiology

The majority of cataracts are known as senile and are formed as a result of the ageing process. As the lens ages, its fibres and proteins change and degenerate. In addition, the proteins clump, clouding the lens and reducing light transmission to the retina. This process generally begins at the periphery of the lens, gradually spreading to involve the central portion. As the cataract continues to develop, the entire lens may become opaque. An immature cataract occurs when only a portion of the lens is affected; however, a mature cataract affects the entire lens. In addition to clouding, the lens may discolour over time, affecting the ability to accurately discriminate colours.

FIGURE 45.7 ■ A scene as viewed by a person with cataracts

Source: Courtesy of National Eye Institute, National Institutes of Health (NEI/NIH).

Manifestations

Cataracts tend to occur bilaterally unless they are related to eye trauma. Fortunately, they tend to develop at different rates and one cataract generally matures more rapidly than the other. As a cataract interferes with light transmission through the lens, visual acuity decreases, affecting both close and distance vision (see Figure 45.7). Light rays are scattered as they pass through the lens, causing complaints of glare, which affects the ability to adjust between light and dark environments. Colour discrimination is impaired, particularly in the blue to purple range. When the cataract is mature, the pupil may appear cloudy grey or white, rather than black.

INTERPROFESSIONAL CARE

The diagnosis of a cataract is made based on the person's history and eye examination. Ophthalmoscopic examination confirms the diagnosis by identifying the location and extent of a cataract. As the cataract matures, ophthalmoscopy reveals a dark area instead of the red reflex.

Surgery

Surgical removal is the only treatment used for cataracts, as no medical treatment is available to prevent or treat them. If the person presents with bilateral cataracts, surgery is performed on one eye at a time. If an intraocular lens (an artificial lens to replace the diseased lens of the eye) is to be implanted during surgery, the corneal curvature and anteroposterior diameter of the eye are measured prior to surgery to determine the lens power needed for the intraocular lens implant.

Surgical removal of the cataract and lens is indicated when the cataract has developed to the point that vision and ADLs

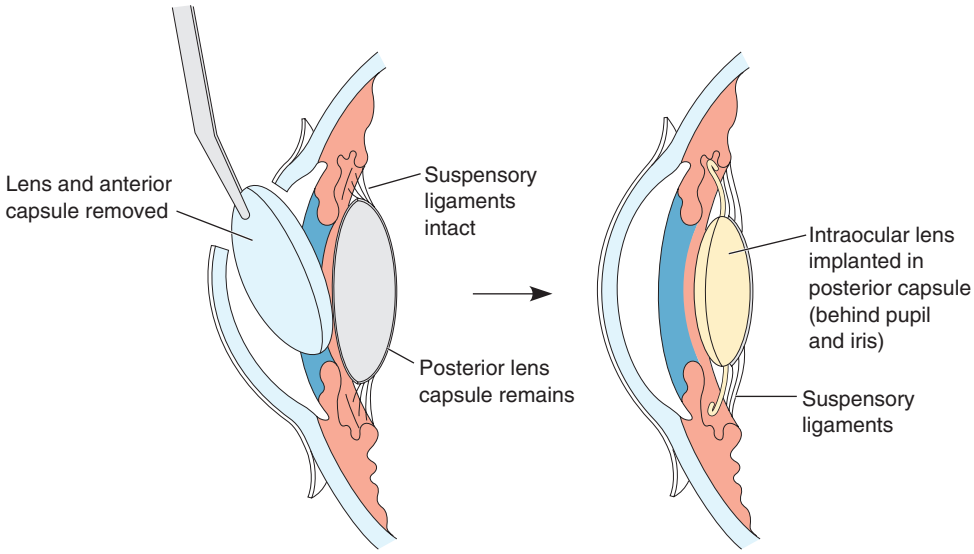


FIGURE 45.8 ■ Extracapsular cataract extraction with removal of the lens and anterior capsule, leaving the posterior capsule intact. The intraocular lens is implanted within the posterior capsule

are affected. A mature cataract may also be removed when it causes a secondary condition such as glaucoma or uveitis.

Cataract surgery is usually done on a day surgery basis, using local anaesthesia. If general anaesthesia and/or sedation is required, the person may be hospitalised overnight. Extracapsular extraction, in which the anterior capsule, nucleus and cortex of the lens are removed, leaving the posterior capsule intact, is the procedure of choice (see Figure 45.8). Using an operating microscope, the surgeon makes a small incision at the edge of the cornea and extracts the lens intact or via emulsification and aspiration. In the latter technique, ultrasound vibrations are used to break the lens material into fragments (phacoemulsification), which are then suctioned out of the eye. The remaining capsule supports the lens implant and protects the retina.

After removal of the lens, the eye can no longer focus light on the retina and vision is seriously affected. Usually a polymethylmethacrylate (PMMA or Plexiglas) intraocular lens is implanted at the time of surgery. This implant rapidly restores binocular vision and depth perception. Following extracapsular lens removal, the intraocular lens is positioned in the posterior capsule behind the iris (see Figure 45.8).

If an intraocular lens cannot be implanted, convex corrective glasses or contact lenses may be used to correct vision after cataract removal. Although contact lenses can provide excellent vision correction following cataract surgery, they may be difficult for some people to adapt to or manipulate. The person with a pre-existing refractive error may continue to require corrective lenses and often needs a prescriptive change even after surgery.

Complications of cataract surgery are unusual and occur in less than 1% of the surgeries. Loss of vitreous humour, corneal oedema, increased intraocular pressure, haemorrhage, inflammation or infection, retinal detachment and displacement of the implanted lens are considered potential complications. Up to 35% of people who undergo extracapsular extraction may develop opacification of the remaining posterior capsule. Vision can be restored using laser capsulotomy (creating an opening for light to pass through the opacified capsule) or surgical incision into the posterior capsule to allow light to reach the retina (Rothrock, 2011).

Nursing care

Health promotion

Advise all people about the importance of protecting the eyes from UVB rays by wearing eye protection during activities such as welding and when outdoors. Discuss the link between heavy smoking and cataract development, and provide necessary education and resources to young people regarding the additional effects of smoking.

Assessment

- *Health history:* effect of vision changes on lifestyle and activities (e.g. ability to read, watch television, participate in work and recreational activities); history of smoking, diabetes, use of prescription drugs associated with increased risk of cataract.
- *Physical examination:* general health; visual acuity (using corrective lenses and Snellen chart) in each eye; presence of red reflex.

Nursing diagnoses and interventions

The person with cataracts has few physical care nursing requirements. Patient advocacy, psychological and emotional

support, and education are typically of higher priority for these people. (See 'Nursing care of the person having eye surgery' in the earlier box.)

Decisional conflict: cataract removal

With the initial diagnosis of a cataract, the nurse often becomes an important information resource for the person.

- Explain the non-emergent nature of the condition and help the person determine the extent to which the cataract is affecting daily life. *Providing information about cataracts and their surgical removal assists with decision making and helps the person decide whether to proceed with the surgery.*
- Attend to verbalised concerns about surgery and its outcome. Address questions factually and completely. Fear of blindness is second only to fear of cancer for many people. *Careful listening, education and a caring, understanding attitude can help the person deal with possible fear prior to surgery.*

Risk of ineffective therapeutic regimen management

- Assess for factors that may interfere with the person's ability to be self-caring in the postoperative phase. *A chronic condition such as arthritis may affect the ability to administer eye drops and may indicate the need to include the education and support of a family member.*
- Assess for additional care needs that may be necessitated by vision changes in the early postoperative period. *Additional needs, such as insulin injections, may suggest the requirement for additional home or nursing care postoperatively.*

Community-based care

Following the initial diagnosis, the focus is on education, indications for surgery and vision restoration following cataract removal. Provide adaptive strategies to deal with effects of the cataract on vision and depth perception. When surgery is scheduled, provide pre- and postoperative education and include a significant other in the sessions. Reinforce the following information with written instructions:

- limitations such as avoiding reading, lifting, strenuous activity and sleeping on operative side
- importance of not disturbing the eye dressing
- importance of not rubbing or scratching the eye
- prescribed medications and side effects
- importance of follow-up appointments
- manifestations of postoperative complications such as eye pain, decreased visual acuity or other change in vision, headache, nausea or itching and redness of the affected eye
- instillation of eye drops and application of eye patch or shield
- care, insertion and removal of contact lenses as appropriate
- visual changes associated with thick-lensed eyeglasses as appropriate.

THE PERSON WITH GLAUCOMA

Glaucoma is a condition characterised by optic neuropathy with gradual loss of peripheral vision and, usually, increased intraocular pressure. Glaucoma is a silent thief of vision in that the person typically experiences no manifestations other than narrowing of the visual field, which occurs so gradually that it often goes unnoticed until late in the disease process.

Incidence and risk factors

Glaucoma affects over 300 000 Australians aged over 60 and is the leading cause of blindness worldwide. The limited data available to date highlight the prevalence of glaucoma in the older generation of Indigenous Australians.

Glaucoma is usually a primary condition without an identified cause. Primary glaucoma is most common in adults over the age of 60; however, it may be a congenital condition in infants and children. Secondary glaucoma can develop as a result of infection or inflammation of the eye, cataract, tumour, haemorrhage or eye trauma.

Pathophysiology

The aqueous humour occupies both the anterior and posterior chambers of the eye. The normal intraocular pressure of approximately 10 to 20 mmHg is maintained by a balance between the production of aqueous humour in the ciliary body, its flow through the pupil from the posterior to the anterior chamber of the eye and its outflow or absorption through the trabecular meshwork and canal of Schlemm (see Figure 44.3). If the balance is disrupted, due to a decrease in the outflow or absorption of aqueous humour, the intraocular pressure increases. Although the exact relationship is unclear, it appears that increased intraocular pressure injures the optic nerve. Axons in the periphery of the optic disc are damaged first. As optic fibres are destroyed, the rim of the optic disc shrinks and the normal depression in its centre (the *optic cup*) becomes larger and deeper (called optic 'cupping'). These changes to the optic disc are visible before visual field changes can be detected. As the disease progresses, there is a painless, progressive narrowing of the visual field (see Figure 45.9) and eventual blindness. Vision loss is often significant before the person seeks treatment and glaucoma is diagnosed.

Primary glaucoma in adults has two main forms: open-angle and angle-closure glaucoma. Both terms refer to the angle formed at the point where the iris meets the cornea in the eye's anterior chamber (see Figure 45.10). Forms of primary glaucoma are compared in Table 45.2.

Open-angle glaucoma

Open-angle glaucoma, often called chronic simple glaucoma, is the most common form of glaucoma in adults, accounting for approximately 90% of all glaucoma. Its cause is unknown; it is thought to have a hereditary component, but no clear inheritance pattern can be identified.

In open-angle glaucoma, the anterior chamber angle between the iris and cornea is normal (see Figure 45.10A);

FIGURE 45.9 ■ Narrowing of visual fields typical of untreated glaucoma

Source: Courtesy of National Eye Institute, National Institutes of Health (NEI/NIH).

hence the term *open angle*. However, the flow of aqueous humour through the trabecular meshwork and into the scleral venous sinus is relatively obstructed: the cause of this obstruction is unknown. Restricted outflow leads to an increased amount of fluid in the eye and increased intraocular pressure. Open-angle glaucoma tends to be a chronic, gradually progressive disease. The trabecular meshwork increasingly inhibits the outflow of aqueous humour and the intraocular pressure gradually increases. The result is neuronal ischaemia and optic nerve degeneration, leading to gradual loss of vision.

Open-angle glaucoma typically affects both eyes, although the pressures and progression may not be symmetrical.

MANIFESTATIONS Open-angle glaucoma is painless, with gradual loss of visual fields. The loss of peripheral vision generally is so gradual that the person is often unaware of it until it is detected through a comprehensive vision examination. Intraocular pressure is usually, but not always, elevated (Horton, 2012).

Angle-closure glaucoma

Acute angle-closure (also called narrow-angle or closed-angle) glaucoma is a less common form of primary glaucoma in adults. It accounts for approximately 5–10% of all cases of glaucoma (Grossman & Porth, 2014). Approximately 1% of people over the age of 40 have narrowed anterior chamber angles; the incidence is higher in older adults and in people of Far Eastern, Asian or Inuit ancestry (Riordan-Eva, 2012).

Narrowing of the anterior chamber angle (see Figure 45.10A for an illustration of the normal anterior chamber angle) occurs because of corneal flattening or bulging of the iris into the anterior chamber. When the lens thickens during accommodation, or the iris thickens during pupil dilation, this angle can close completely. Closure of the angle blocks the outflow of aqueous humour through the trabecular meshwork and scleral venous sinus, and the intraocular pressure rises abruptly (see Figure 45.10B). This abrupt increase in intraocular pressure damages the neurons of the retina and the optic nerve, leading to a rapid and permanent loss of vision if not treated promptly.

Episodes of angle-closure glaucoma are typically unilateral. However, a history of angle-closure glaucoma of one eye increases the risk that it will occur in the other eye.

Because of the effect of pupil dilation on aqueous outflow in angle-closure glaucoma, episodes often occur in association with darkness, emotional upset/stress or other factors that cause the pupil to dilate. People may have intermittent episodes

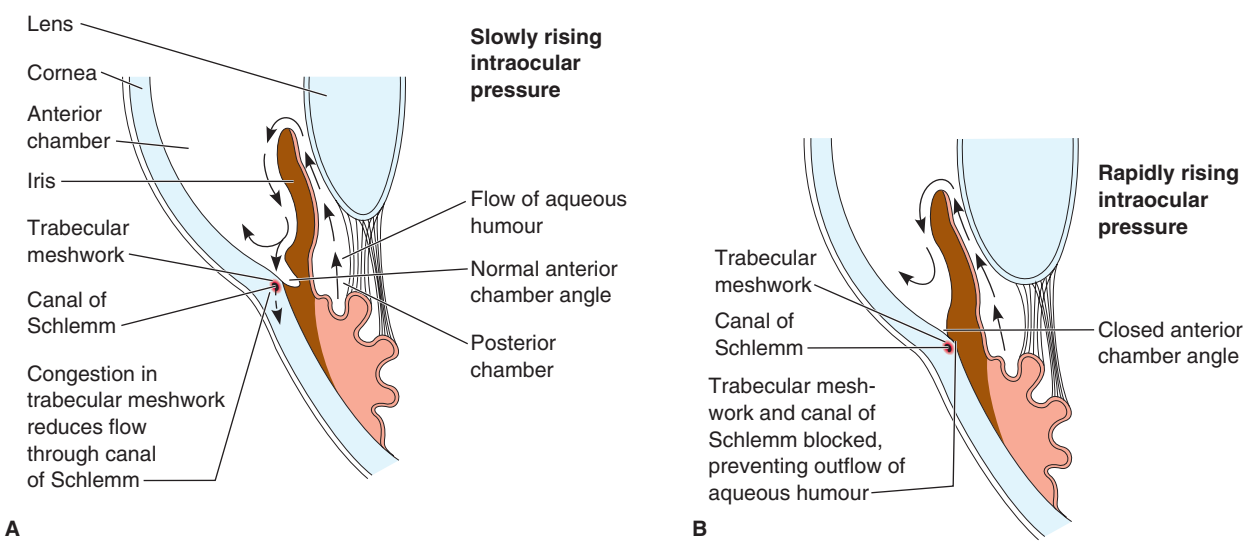


FIGURE 45.10 ■ Forms of primary adult glaucoma. *A*, In chronic open-angle glaucoma, the anterior chamber angle remains open, but drainage of aqueous humour through the canal of Schlemm is impaired. *B*, In acute angle-closure glaucoma, the angle of the iris and anterior chamber narrows, obstructing the outflow of aqueous humour

TABLE 45.2 A comparison of open-angle and angle-closure glaucoma

	OPEN-ANGLE GLAUCOMA	ANGLE-CLOSURE GLAUCOMA
Incidence	• Common • Accounts for 90% of all cases of glaucoma	• Uncommon
Risk factors	• Over age 40 • Genetic link	• Narrow anterior chamber angle • Ageing • Asian ancestry
Pathophysiology	• Impaired aqueous outflow through the scleral venous sinus • Damage to axons of retinal ganglion cells with optic nerve atrophy • Gradual, consistent increase in intraocular pressure • Usually bilateral	• Pupil dilation or lens accommodation causes already narrowed angle to close, blocking aqueous outflow • Rapid rise in intraocular pressure • Usually unilateral
Manifestations	• No initial manifestations • Frequent lens changes in glasses • Impaired dark adaptation • Halos around lights • Gradual reduction of visual fields with preservation of central vision until late in the disease • Mild to severe increased intraocular pressure	• Abrupt onset of eye pain, headache • Decreased visual acuity • Nausea and vomiting • Reddened conjunctiva • Cloudy cornea • Fixed pupil • Rapid, significant increase in intraocular pressure
Management	• Topical medications such as miotics, beta-blockers, prostaglandin analogues • Carbonic anhydrase inhibitors • Laser trabeculoplasty, trabeculectomy	• Topical miotics or beta-blockers • Systemic osmotic agents, carbonic anhydrase inhibitors • Laser iridotomy or peripheral iridectomy

lasting several hours before having a more typically prolonged attack of angle-closure glaucoma. For people with a history of the condition, it is vital to avoid medications such as atropine and other anticholinergics, which have a mydriatic or pupil-dilating effect.

MANIFESTATIONS Symptoms such as severe eye and face pain, general malaise, nausea and vomiting, seeing coloured halos around lights and an abrupt decrease in visual acuity are associated with acute episodes of angle-closure glaucoma. The conjunctiva of the affected eye may be reddened and the cornea clouded with corneal oedema, and the pupil may be fixed (non-reactive to light) at midpoint. Some people may experience periodic mild attacks, usually in the evening, with eye discomfort, impaired vision and coloured rings around lights.

INTERPROFESSIONAL CARE

Although glaucoma cannot be predicted, prevented or cured, in most cases it can be controlled and vision can be preserved if diagnosed early. Because open-angle glaucoma, the most prevalent type of glaucoma, has few symptoms, routine eye examinations are recommended for early detection. Measurement of intraocular pressure, fundoscopy to assess the optic disc and visual field testing are used for diagnosis and monitoring of treatment effectiveness.

Diagnosis

The following diagnostic studies are used to detect and evaluate for the presence, severity, type and effects of glaucoma:

- *Tonometry* indirectly measures intraocular pressure (see Figure 45.11). Contact or non-contact tonometry may be used. Routine tonometry screening is recommended for all people over the age of 60. A single elevated pressure reading does not warrant a diagnosis of glaucoma; variations in intraocular pressure occur throughout the day. See Chapter 44 for more information about tonometry.
- *Fundoscopy* (visual inspection of the optic fundus using an ophthalmoscope) identifies pallor and an increase in the size and depth of the optic cup on the optic disc. These changes are significant for diagnosing glaucoma.
- *Gonioscopy* uses a gonioscope to measure the depth of the anterior chamber. This test differentiates open-angle from angle-closure glaucoma.
- *Visual field testing* (see Figure 45.12) identifies the degree of central visual field narrowing and peripheral vision loss. The person with glaucoma may retain 6/6 central vision even though there is severe peripheral vision loss.

Medications

Although medications cannot cure glaucoma, many people with open-angle glaucoma can control intraocular pressure and preserve vision indefinitely with medications. Medications are used alone or in combination, with the timing and

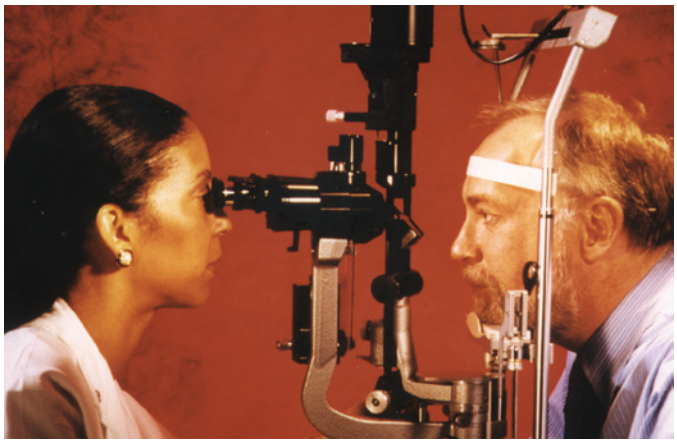


FIGURE 45.11 ■ An eye professional uses a tonometer to indirectly measure the intraocular pressure of a person's eye

Source: Courtesy of National Eye Institute, National Institutes of Health (NEI/NIH).

FIGURE 45.12 ■ Visual field testing. Peripheral vision or visual fields are assessed by testing the person's ability to detect an object brought into the line of vision from the periphery. The person's peripheral vision is compared to the nurse examiner's. Each eye is tested separately

dosage individually determined by pressure measurements. The primary pharmacological agents used to treat glaucoma are topical beta-adrenergic blocking agents, adrenergics (mydriatics), prostaglandin analogues or carbonic anhydrase inhibitors. An oral carbonic anhydrase inhibitor also may be used.

Topical beta-adrenergic blocking agents decrease the production of aqueous humour in the ciliary body. Beta-adrenergic blockers can be used once or twice a day, depending on the drug and dosage form. When administering beta-blockers or teaching about their use, it is important to remember that ophthalmic preparations can produce systemic effects, including bronchospasm, bradycardia and heart failure.

Prostaglandin analogues are a newer class of ophthalmics prescribed to increase aqueous outflow. They are similar to beta-blockers in their longer duration of action, thus requiring only a daily dose. Although they have fewer systemic effects, these drugs may cause conjunctival hyperaemia and permanent changes in the colour of the iris and eyebrows.

An adrenergic agonist may be prescribed along with a beta-blocker, or, if beta-blockers are contraindicated (e.g. in a person with heart failure, asthma or chronic obstructive pulmonary disease (COPD)), another adrenergic agonist may be prescribed when other drugs do not sufficiently reduce intraocular pressure, but adverse effects make it inappropriate for long-term use (Riordan-Eva, 2012).

A carbonic anhydrase inhibitor decreases the production of aqueous humour and reduces intraocular pressure. It is used with other drugs to control pressures and in people for whom beta-blockers are contraindicated because of heart failure or reactive airway disease. A systemic carbonic anhydrase inhibitor also may be used for some people.

Nursing implications for the medications used to control chronic glaucoma are outlined in the 'Medication administration' box below.

In acute angle-closure glaucoma, diuretics may be administered intravenously to achieve a rapid decrease in intraocular pressure prior to surgical intervention. Carbonic anhydrase inhibitors and osmotic diuretics are used. Fast-acting miotic drops, such as acetylcholine, are also administered to constrict the pupil and draw the iris away from the angle and the scleral venous sinus.

Surgery

Surgical intervention is indicated for people with acute angle-closure and chronic open-angle glaucoma that is not effectively controlled by medication.

Surgical management of chronic open-angle glaucoma involves improving the drainage of the aqueous humour from the anterior chamber of the eye. Trabeculoplasty and trabeculectomy filtration surgery are the most commonly used procedures.

In a *laser trabeculoplasty*, an argon laser is aimed through a gonioscope to create multiple laser burns spaced evenly around the trabecular meshwork. As the burns heal, the scars they create cause tension, stretching and opening the meshwork. This non-invasive technique is the treatment of choice because it requires no incision and can be performed as an outpatient procedure.

Trabeculectomy is a type of filtration surgery in which a permanent fistula is created to drain aqueous humour from the anterior chamber of the eye. A portion of trabecular meshwork is removed and a flap of sclera is left unsutured to create a channel or fistula between the anterior chamber and the subconjunctival space. Aqueous humour is able to drain into the space under the conjunctiva, where it can be absorbed into the systemic circulation. A trabeculectomy is usually performed under general anaesthesia and requires hospitalisation.

MEDICATION ADMINISTRATION The person with glaucoma

ADRENERGIC AGONISTS (MYDRIATICS)

Brimonidine
Apraclonidine

Adrenergic agonists dilate the pupil, reduce the production of aqueous humour and increase its absorption, effectively reducing intraocular pressure in open-angle glaucoma.

Nursing responsibilities

- Assess the person for contraindications and adverse reactions to adrenergic agonists, including acute angle-closure glaucoma, hypertension, cardiac arrhythmias and coronary heart disease.
- Assess for central nervous system side effects of anxiety, nervousness and muscle tremors. If these side effects are severe, notify the doctor.
- Assess for a hypersensitivity reaction, including itching, lid oedema and discharge from the eyes. Notify the doctor if you notice these signs.

Health education for the person and family

- Report any change in visual acuity or eye pain. (Eye pain may indicate an attack of angle-closure glaucoma and must be reported to the doctor immediately.)
- Avoid over-the-counter sinus and cold medications containing pseudoephedrine and phenylephrine, as they may accentuate the side effects of this drug.

BETA-ADRENERGIC BLOCKERS

Betaxolol
Carteolol
Levobunolol
Metipranolol
Timolol

Selected beta-adrenergic blockers reduce intraocular pressure by decreasing the production of aqueous humour. Because beta-blockers do not affect pupil size and lens accommodation, they do not have the adverse effects on visual acuity that adrenergic agonists do. Their systemic effects, however, may limit their usefulness for certain people.

Nursing responsibilities

- Assess the person for allergies or contraindications to beta-blocker therapy, including asthma, COPD, heart block and heart failure.
- Maintain pressure over the lacrimal sac after administration to prevent systemic absorption.
- Assess for side effects such as bradycardia, hypotension and depression.
- Educate about the drug, its dose, administration and desired and side effects.

Health education for the person and family

- After instilling the eye drops, put pressure on the lacrimal sac, at the corner of the eye near the bridge of the nose, to keep the drug from entering your system.
- Your vision may be blurred during the initial period of therapy, but it will improve as you continue to use the drug.
- Report adverse effects, including worsening vision, difficulty breathing, reduced exercise tolerance and sweating or flushing, to the doctor.

CARBONIC ANHYDRASE INHIBITORS

Dorzolamide
Brinzolamide
Acetazolamide (most common)

The carbonic anhydrate inhibitors lower intraocular pressure and are used primarily as adjunctive therapy. Dorzolamide and brinzolamide are administered as eye drops; acetazolamide may be given PO, IM or IV in glaucoma emergencies.

Nursing responsibilities

- Assess for allergies or other contraindications to the use of carbonic anhydrase inhibitors, including known allergy to sulfur or severe kidney or hepatic disease.
- Monitor for increased drug interactions of amphetamines, procainamide, quinidine, tricyclic antidepressants, and ephedrine and pseudoephedrine.
- Assess daily weight, intake and output, serum electrolytes and vital signs in people taking oral or parenteral carbonic anhydrase inhibitors.
- Administer PO in the morning to prevent sleep disruption because of the diuretic effect.
- If used with another topical ophthalmic, administer 10 minutes apart.
- Educate the person about the drug, its dose, administration and desired and side effects.

Health education for the person and family

- For oral medications, maintain a fluid intake of 2 to 3 L per day and rise slowly from lying or sitting positions because you may feel dizzy when you first stand (orthostatic hypotension).
- For topical medications, notify the doctor if you have prolonged eye irritation.

PROSTAGLANDIN ANALOGUES

Bimatoprost
Latanoprost
Travoprost

The prostaglandin analogue drugs relax the ciliary muscle, improving the outflow of aqueous humour and reducing intraocular pressure. These drugs have the advantage of requiring only a single daily dose; however, they do have some adverse effects such as blurred vision and stinging and, when used long term, cause permanent darkening of the iris of the eye and eyebrows, increased growth of eyelashes and conjunctival hyperaemia (redness).

Nursing responsibilities

- Assess and note eye colour, presence of inflammation, exudates or pain.
- Note vital signs and most recent liver function test results because these may be altered by the drug.

Health education for the person and family

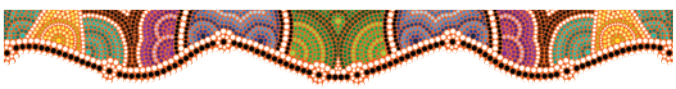
- Use once daily at bedtime as directed. This drug may blur vision; use at bedtime minimises associated safety risks.
- Remove contact lenses before administering this drug.
- Minor eye discomfort, including burning and tearing, may occur with this drug. Notify your doctor if adverse effects are severe or intolerable.
- This drug may cause darkening of your iris, the skin around the eyes and the eyebrows, as well as increased growth of the eyelashes. These colour changes are permanent and will not progress if the drug is discontinued by your doctor.

If these procedures are not fully effective, either photocoagulation using an argon laser (heat) or cyclocryotherapy using a probe to freeze tissue may be employed to destroy portions of the ciliary body. This tissue destruction reduces the production of aqueous humour, subsequently reducing intraocular pressure. Another surgical procedure involves insertion of a glaucoma drainage device that regulates the outflow of aqueous humour.

Surgical procedures used in the treatment of acute angle-closure glaucoma include gonioplasty, laser iridotomy and peripheral iridectomy. Because of the high risk of a future attack of angle-closure glaucoma in the unaffected eye, these procedures are often performed prophylactically.

In *gonioplasty*, the healing and scarring of microscopic lesions created at the periphery of the iris draws the iris away from the cornea, widening the anterior chamber, increases the angle and opens drainage channels for the aqueous humour.

Laser iridotomy is a non-invasive procedure using a laser to create multiple small perforations in the iris of the eye. These perforations allow aqueous humour to drain from the posterior chamber to the anterior chamber, and out through the trabecular meshwork and the scleral venous sinus. During an *iridectomy*, a small segment of the iris is removed to facilitate the flow of aqueous humour between the posterior and anterior chambers and to open the anterior chamber angle.



Nursing care

When planning and providing nursing care for the person with glaucoma, both the specific form of the disease and its actual or potential effects on the person's vision, lifestyle, safety and psychosocial wellbeing must be considered. In the hospitalised person, glaucoma is typically a concurrent diagnosis rather than the primary reason for seeking care, unless the diagnosis is acute angle-closure glaucoma. For additional nursing activities for the person with glaucoma, see the accompanying nursing care plan.

Health promotion

Although glaucoma cannot be prevented, its severity and potentially deleterious permanent effects can be limited with early visual screening. The nurse assumes an important role in educating the public about the risk factors for glaucoma, such as increased age and the higher incidence in certain cultures. All people over the age of 40 are encouraged to receive an eye examination every 2 to 4 years, including tonometry screening. Those with a predominant family history should be evaluated more frequently, every 1 to 2 years. After the age of 65, yearly ophthalmological examinations are recommended.

Assessment

Collect the following data through a health history and physical examination (see Chapter 44).

- *Health history:* family history; presence of altered vision, halos and excessive tearing; sudden, severe eye pain; use of corrective lenses; most recent eye examination.
- *Physical examination:* distant and near vision, peripheral fields and retina for optic nerve cupping.

Nursing diagnoses and interventions

Nursing care planning focuses on problems associated with the temporary or permanent visual impairment, the resultant increased risk of injury and the psychosocial problems of anxiety and coping.

Disturbed visual perception

Whether glaucoma and resulting impaired vision is the person's primary problem or a pre-existing condition in a person with another disorder, it must be a primary consideration in nursing care planning.

- Establish rapport with the person. Orient to time, place, person and situation as indicated. State the purpose of your visit. *The person with impaired vision must rely on input from the other senses. A lack of visual cues increases the importance of verbal ones. For example, the person with impaired vision cannot see the nurse checking an intravenous infusion and needs a verbal explanation of who is in the room and why. When the person's normal daily routine is disrupted by illness or hospitalisation, additional sensory input such as a radio or television, and explanations of the routine and activities, are useful to maintain the person's orientation.*
- Provide any visual aids that are routinely used. Keep them close, making sure that the person knows where they are and can reach them easily. *Easy access encourages the person to use these items and enhances the ability to provide self-care.*
- Orientate the person to the environment. Explain the location of the call bell, personal items and the furniture in the room. If able, tour the person's room, including the bathroom and sink. *People with visual impairments are usually very capable of providing self-care in a known environment.*
- Provide other tools or items that can help compensate for diminished vision:
  a. bright, non-glare lighting
  b. books, magazines and instructions in large print
  c. books on tape, CD or MP3 player
  d. telephones with oversized pushbuttons
  e. a clock with numbers and hands that can be felt.
- Assist with meals by:
  a. reading menu selections and marking choices
  b. describing the position of foods on a meal tray according to the clock system—for example, 'On the plate, the peas are at 9 o'clock, the mashed potatoes at 1 o'clock and the chicken breast at 6 o'clock. The water glass is at 2 o'clock on the tray above the plate and coffee is at 11 o'clock'
  c. placing the utensils in a readily accessible position
  d. removing lids from containers, buttering bread and cutting meat, as needed
  e. if the visual impairment is new or temporary, the person may need feeding or continued assistance during the meal.

NURSING CARE PLAN A person with glaucoma and cataracts

Lila Rainey is an 80-year-old widow who lives alone in the house she and her late husband built 50 years ago. She has worn glasses for myopia since she was a young girl and now wears bifocals to correct her near vision as well. She was diagnosed 4 years ago with chronic open-angle glaucoma, for which she takes timolol maleate 0.5%—one drop in each eye twice a day. Recently she has noticed difficulty reading and watching television despite a new lens prescription. She has stopped driving at night because the glare of oncoming headlights makes it difficult for her to see. Mrs Rainey's ophthalmologist has told her that she has cataracts but that they do not need to come out until they bother her. Although her glaucoma is still controlled by the medication, her intraocular pressure measurements have been gradually increasing. Mrs Rainey has taken 325 mg of aspirin daily since a transient ischaemic attack 8 years ago. She is being admitted to the outpatient surgery unit for a cataract removal and intraocular lens implant in her right eye.

ASSESSMENT

Mrs Rainey is admitted to the eye surgery unit by Susan Schafer, RN. In her assessment, Ms Schafer finds Mrs Rainey to be alert and oriented, though apprehensive about her upcoming surgery. Assessment findings include BP 134/72, P 86 and R 18. Mrs Rainey's neurological, respiratory, cardiovascular and abdominal assessments are essentially normal. Her pupils are round, equal and react briskly to light and accommodation. Her conjunctiva are pink; sclera and corneas, clear. Using the ophthalmoscope, Ms Schafer notes that the red reflex in Mrs Rainey's right eye is diminished. Ophthalmic examination shows visual acuity of 6/45 OD (right eye) and 6/15 OS (left eye) with corrective lenses. Her intraocular pressures are 21 mmHg OD and 17 mmHg OS. On fundoscopic exam, no disease of the blood vessels, retina, macula or disc is found. Ms Schafer reviews the operative procedure with Mrs Rainey, answering her questions and advising what to expect after surgery. Following preoperative protocols, Mrs Rainey is prepared and transported to surgery.

NURSING DIAGNOSIS

- *Risk of disturbed visual perception* related to myopia and lens extraction.
- *Risk of anxiety* related to anticipated surgery.
- *Risk of knowledge deficit* related to lack of information regarding postoperative care.
- *Risk of impaired home maintenance* related to activity restrictions and impaired vision.

PLANNING

- Provide a safe environment, placing the call bell and personal care items within easy reach.
- Encourage Mrs Rainey to express her fears about surgery and its potential effect on vision.
- Explain all procedures related to surgery and recovery.

Expected outcomes

- To regain sufficient visual acuity to maintain ADLs, including reading and watching television for enjoyment.
- Demonstrate the procedure for instilling eye drops postoperatively.
- Demonstrate knowledge of the home care she will require after surgery, signs of complications and actions to take if complications occur.
- Use appropriate resources to assist with home maintenance until vision stabilises and activity restrictions are lifted.
- Demonstrate a reduced level of anxiety.

IMPLEMENTATION

- Instruct her to avoid shutting her eyelids tightly, sneezing, coughing, laughing, bending over, lifting or straining to have a bowel movement. Educate her to wear glasses during the day and an eye shield at night to prevent injury to the surgical site.
- Explain and demonstrate the procedure for administering eye drops.
- Provide verbal and written instructions about postoperative care, including a schedule of follow-up examinations, potential complications and actions to take in response.
- Refer Mrs Rainey to a discharge planner or social worker to help establish a plan for home maintenance as required.

EVALUATION

Mrs Rainey is discharged the morning after her surgery. She is visibly relieved when the eye patch is removed because her vision in the operated eye is better than before surgery, even without her glasses. She is able to relate the recommended activity restrictions. Mrs Rainey administers her own eye drops before discharge and relates an understanding of the prescribed postoperative care and safety precautions. Mrs Rainey's daughter plans to visit her mother two to three times a week to help with laundry and vacuuming until she is able to resume all her household activities. Mrs Rainey says that she will not 'be so scared when I need my other eye done'. She understands the chronic nature of her glaucoma and states that her vision is too important for her to neglect her timolol drops and routine eye exams.

CRITICAL THINKING IN THE NURSING PROCESS

1. Why did it become more difficult to control Mrs Rainey's intraocular pressure as her cataract matured?
2. Identify medications that are commonly prescribed following cataract surgery. What are the risks of interactions between these medications and Mrs Rainey's timolol drops?
3. Develop a care plan for the nursing diagnosis *Self-care deficit: dressing/grooming*, related to visual impairment and restricted bending.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from the case study and how this applies to your future practice.
2. What communication strategies could you use when caring for a person experiencing issues with their vision?

Providing assistance during meals is important to maintain the person's nutritional status. The person may be ashamed of needing help or embarrassed to request it, and may respond by not eating or by claiming not to be hungry.

- Assist with mobility and ambulation as needed:
  a. Have the person hold your arm or elbow and walk slightly ahead as a guide. Do not hold the person's arm or elbow.
  b. Describe the surroundings and progress as you proceed. Warn in advance of potential hazards, turns and steps.
  c. Educate on how to feel the chair, bed or commode with the hands and the back of the legs before sitting.

  These measures help ensure the person's safety while providing for mobility and helping prevent complications associated with immobility.
- If the vision loss is unilateral and recent, provide instructions related to this loss and change in depth perception:
  a. Caution about the loss of depth perception and educate about safety precautions, such as reaching slowly for objects and using visual cues for distance, especially when driving.
  b. Educate the person to scan their environment, turning the head fully towards the affected side to identify potential hazards and looking up and down to compensate for the loss of depth perception.

  The person with a unilateral vision loss is often unaware of its effects on peripheral vision and depth perception.

Risk of injury

Whether the person is experiencing a sudden loss of vision due to acute angle-closure glaucoma or significant visual impairment due to inadequately managed chronic glaucoma, both are at an increased risk of injury. People who have had surgical interventions for glaucoma are at even greater risk.

- Assess ability to perform ADLs. People may be reluctant to request assistance, believing that they should be able to perform these familiar tasks. *Careful assessment and provision of needed assistance help prevent injury and maintain the person's self-esteem and confidence.*
- Notify all staff and place a sign on the person's door to alert all personnel not to change the arrangement of the person's room. *The person with impaired vision is at high risk of falling when in an unfamiliar environment. It is important to maintain a safe, familiar room when the person is hospitalised.*
- Raise the person's side rails on their bed. *Raised rails remind people to ask for assistance before ambulating in an unfamiliar environment.*
- Discuss possible adaptations in the home to help the person remain as independent as possible and prevent falls or other injuries. *Often minor changes in the home environment, such as removing scatter rugs and small items of furniture, allow the person to navigate safely in this already familiar environment.*

CONSIDERATION FOR PRACTICE

Keep traffic areas free of clutter to reduce the risk of injury in people with impaired vision.

Anxiety

The actual or potential loss of sight threatens the person's self-concept, role functioning, patterns of interaction and, potentially, environment. The person with impaired vision who functions well in a familiar environment will feel anxious in the unfamiliar setting of a hospital or care facility.

- Assess for verbal and non-verbal indications of anxiety levels and for normal coping mechanisms. Repeated expressions of concern or denial that the vision change will affect the person's life indicate anxiety. Non-verbal indicators include tension, difficulty concentrating or thinking, restlessness, poor eye contact and changes in vocalisation (rapid speech, voice quivering). Physical indicators include tachycardia, dilated pupils, cool and clammy skin, and tremors. *The person may not recognise this feeling as anxiety. Identifying and acknowledging the anxiety state can help the person recognise and deal with it.*
- Encourage to verbalise fears, anger and feelings of anxiety. *Verbalising helps externalise the anxiety and allows fears to be addressed.*
- Discuss perception of the eye condition and its effects on lifestyle and roles. *Discussion provides an opportunity to correct misperceptions and introduce alternative activities and assistive devices for people with visual impairments.*
- Establish rapport with the person by explaining all procedures fully before and as they are being performed, and use touch to convey proximity and caring. *The person with impaired vision must rely on the other senses to make up for the loss of sight. Because the person cannot see what you are doing, complete explanations of even simple tasks such as refilling a water glass help to relieve anxiety.*
- Identify coping strategies that have been useful in the past and adapt these strategies to the present situation. *Previously successful coping strategies may be employed to increase the person's sense of control.*

Community-based care

People with glaucoma require education about lifetime strategies for managing the disease at home. They need to understand the importance of lifetime therapy to control the disease and prevent blindness. If a permanent visual impairment has resulted, the person needs information on achieving the maximum possible independence while maintaining safety. The following topics should be discussed with the person and family:

- prescribed medications, including proper way to instil eye drops
- importance of not taking certain prescription and over-the-counter medications without consulting a doctor
- periodic eye examinations with intraocular pressure measurement

- risks, warning signs and management of acute angle-closure glaucoma
- possible surgical options
- available state and territory community resources.

THE PERSON WITH AGE-RELATED MACULAR DEGENERATION

In Australia, age-related **macular degeneration** (AMD) is the second most common eye disease that causes visual impairment after cataracts.

FAST FACTS

The total cost of vision loss associated with AMD in Australia was estimated at $5 billion in 2010. In 2010, there were approximately 1 million Australians with AMD, equivalent to one in seven people over the age of 50. This could reach almost 2 million by 2030 (Deloitte Access Economics & Macular Degeneration Foundation, 2011).

Although the exact cause of AMD is unknown, factors associated with it include ageing, smoking, race and, possibly, genetic factors and cultural background. The destructive changes in the macula occur most often as a response to the ageing process. AMD affects males and females equally. Recent evidence suggests that inflammation plays a role in the development of AMD (Horton, 2012) as does the interaction of certain genes with the immune system (NEI, 2012c). Evidence suggests that the risk of developing AMD may be reduced by consumption of omega-3 fatty acids in fish, and vegetables high in lutein and zeaxanthin (carotenoids found in vegetables such as spinach, kale and broccoli).

Pathophysiology

The macula is the area of the retina that provides sharp central vision, receiving light from the centre of the visual field. Two forms of AMD identified are non-exudative (dry) and exudative (wet). Although both are progressive disorders, their manifestations and management differ.

Non-exudative or *dry macular degeneration* is the more common form of AMD. It is a gradual process that begins with accumulation of deposits called *drusen* beneath the pigment epithelium of the retina. Over time, these deposits enlarge and become more numerous. The pigment epithelium detaches in small areas and becomes atrophic, interfering with sensory function of the macula. Vision loss typically is not significant and the disorder progresses slowly. There is, however, a risk that the disorder will progress to the exudative stage of the disease.

Exudative macular degeneration is characterised by the formation of new, weak blood vessels in the potential space between the choroid (vascular layer of the eye) and the retina (neurosensory layer). These new vessels are prone to leak, elevating the retina from the choroid and distorting vision. Although exudative macular degeneration typically is a gradual process, bleeding can lead to acute vision loss in some cases. With significant or repeated bleeding episodes, scar tissue forms and central vision is permanently lost (Horton, 2012).

Manifestations

When the macula is damaged, central vision becomes blurred and distorted, but peripheral vision remains intact. Distortion of vision in one eye is a common initial manifestation; straight lines appear wavy or distorted. With the loss of central vision, activities that require close central vision, such as reading and sewing, are particularly affected (see Figure 45.13).

INTERPROFESSIONAL CARE

Age-related macular degeneration is diagnosed through vision and retinal examination. The Amsler grid (see Figure 44.5) may be used to identify distortion of central vision caused by AMD. If treatment for wet AMD is planned, a *fluorescein angiogram* may be done. Pictures are taken as the dye passes through the blood vessels of the retina, allowing detection of leaks.

In its early or intermediate stages, the progress of dry AMD can be slowed through the use of high-dose antioxidants and zinc. Research demonstrated a benefit when vitamins C and E, beta-carotene (vitamin A), zinc and copper were administered daily.

Wet AMD is treated with laser surgery or photodynamic therapy. Although these treatments do not cure the disease, they may slow the rate of vision loss. In laser surgery, fragile blood vessels are destroyed, preventing bleeding. There is a risk, however, of damage to surrounding healthy tissue, some vision loss and continued growth of new vessels. In photodynamic therapy, verteporfin, a drug that tends to adhere to the surface of new blood vessels, is injected systemically. Light is

FIGURE 45.13 ■ Loss of central vision with advanced age-related macular degeneration

Source: Courtesy of National Eye Institute, National Institutes of Health (NEI/NIH).

then shined into the affected eye, activating the drug and destroying new blood vessels. This treatment is relatively fast and painless, but does require avoidance of exposure to direct sunlight or bright indoor light for 5 days following treatment (NEI, 2013a).

Large-print books and magazines, the use of a magnifying glass and high-intensity lighting can help the person to cope with the reduced vision of macular degeneration.

Nursing care

Nurses should be alert for people demonstrating new and rapid onset manifestations of macular degeneration and promptly refer these people for ophthalmological evaluation. Early intervention may preserve a greater degree of vision and slow the progress of the disease. For people with slowly progressive manifestations, the nursing focus is on helping the person and family members adapt to the gradual decline in vision by recommending visual aids and other coping strategies. Person education materials should be in a large-print format. See also the accompanying 'Translation to practice' box.

THE PERSON WITH DIABETIC RETINOPATHY

Diabetic retinopathy is a vascular disorder affecting the capillaries of the retina. The capillaries become sclerotic and lose their ability to transport sufficient oxygen and nutrients to the retina.

The risk of developing diabetic retinopathy is related to the duration of the diabetes and the degree of glycaemic control. Hypertension also is a risk factor (Powers, 2012). Retinopathy is seen in both type 1 and type 2 diabetes. Nursing care of the person with diabetes is discussed in Chapter 19.

FAST FACTS

- In Australia, diabetic retinopathy is the third most common eye disease that causes visual impairment after cataracts and age-related macular degeneration.
- People with diabetes are 25 times more likely to become legally blind than people unaffected by the disease (Powers, 2012).

Pathophysiology and manifestations

Diabetic retinopathy progresses through four stages: (1) mild *non-proliferative* or background retinopathy, (2) moderate non-proliferative retinopathy, (3) severe non-proliferative retinopathy, and (4) *proliferative* retinopathy (NEI, 2013b). Non-proliferative retinopathy is typically the initial form seen. The venous capillaries of the eye dilate and develop microaneurysms that may then leak,

TRANSLATION TO PRACTICE Evidence-based practice: the person with impaired vision

Age-related macular degeneration is the leading cause of significant visual impairment among older adults. However, little research has been done to study the effect of visual impairment on the lives of older adults. Moore and Miller (2003) studied the lived experience of severe visual impairment for a group of older, community-dwelling men with AMD. While the lives of the men in the study tended to be somewhat defined by what they could and could not do, these men tended to express hope and optimism, focusing on remaining abilities and developing strategies to cope with vision loss. However, in contrast to the results of a similar study of older women with AMD, the men in this study expressed scepticism about their disorder and its prognosis, critically questioning treatment and consistency of the care received.

IMPLICATIONS FOR NURSING

Older adults with significant vision deficit continue to cherish their independence and focus on their remaining abilities (as opposed to their disabilities). While it is important to present clear, accurate and realistic information to people with irreversible vision impairment, maintenance of hope and optimism also is important to the older adult. Many people faced with impaired vision make conscious choices to maintain a positive attitude and lifestyle, developing strategies that allow them to continue with activities that are important to them or provide pleasure. The nurse can help support and encourage these choices and selection of positive behaviours to maintain independence and self-worth.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. The men in this study cherished their freedom and found many ways to maintain it despite impaired vision. Discuss ideas to suggest for a person with impaired vision to continue to engage in activities such as shopping, attending cultural events and playing golf or participating in other sports.
2. The men participating in this study expressed more scepticism about their diagnosis and prognosis and criticism of their treatment than did older women with AMD who had participated in an earlier study. What factors might account for this difference? What implications could this difference have for nursing assessment and care?
3. Develop a teaching plan for a person with newly diagnosed AMD using the nursing diagnosis of *Deficient diversional activity* related to recent change in visual acuity.

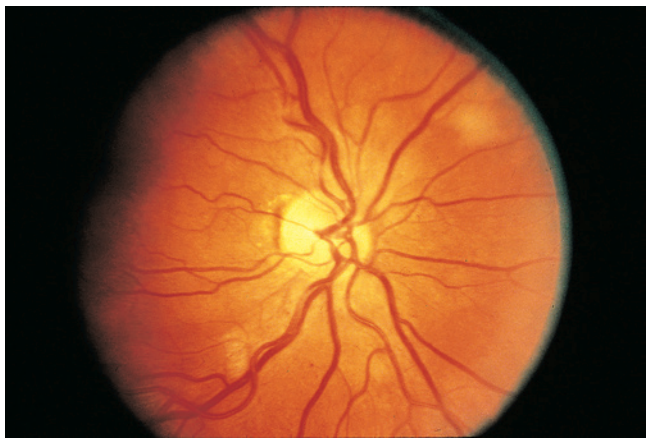


FIGURE 45.14 ■ Appearance of the ocular fundus in diabetic retinopathy

Source: Courtesy of National Eye Institute, National Institutes of Health (NEI/NIH).

causing retinal oedema; or they may rupture, causing small haemorrhages into the retina. On ophthalmoscopic examination, yellow exudates, cotton-wool patches indicative of retinal ischaemia and red-dot haemorrhages are observed (see Figure 45.14). When the peripheral retina is involved, the person may experience few symptoms other than light glare. Oedema of the macula or a large haemorrhage may cause vision loss.

Diabetic retinopathy may progress to the proliferative form. This disease is marked by large areas of retinal ischaemia and the formation of new blood vessels (neovascularisation) spreading over the inner surface of the retina and into the vitreous body. These vessels are fine and fragile, making them permeable and easily ruptured. Blood and blood protein leakage contribute to retinal oedema, and haemorrhage into the vitreous body may occur. The vessels gradually become fibrous and firmly attached to the vitreous body, increasing the risk of retinal detachment.

INTERPROFESSIONAL CARE

People with diabetes should be examined yearly by an ophthalmologist. The development of any new visual manifestations is an additional indication for prompt ophthalmological examination and possibly retinal angiography.

Laser photocoagulation is used to treat both the nonproliferative and proliferative forms of diabetic retinopathy. Leaking microaneurysms are sealed and proliferating vessels destroyed, reducing the risk of haemorrhage, retinal oedema and retinal detachment. This treatment also slows the progress of aneurysms and new vessel formation; however, it does not cure the disorder. People with severe proliferative retinopathy may undergo vitrectomy to remove vitreous haemorrhage or treat associated retinal detachments (Papadakis & McPhee, 2015). Although conclusive research is lacking, the diabetic person with retinopathy may be advised to avoid physical activity associated with the Valsalva manoeuvre (e.g. weight training) (Longo et al., 2012).

Nursing care

As with many other eye disorders, the nursing care focus for diabetic retinopathy is primarily educational. The newly diagnosed diabetic person needs to understand the importance of regular eye examinations, beginning approximately 5 years after the onset of type 1 diabetes and at the time of onset of type 2 diabetes. Changes of diabetic retinopathy may already be present when type 2 diabetes is diagnosed.

Educate the person to report promptly any new visual manifestation, including blurred vision; black spots (floaters), cobwebs or flashing lights in the visual field; or a sudden loss of vision in one or both eyes. Emphasise to the person that careful blood glucose control may help prevent diabetic retinopathy from developing; it may also slow its progress. The person's blood pressure should also be maintained within normal limits to prevent further damage to retinal vessels. Although diabetic retinopathy cannot be halted or cured, its progress can be slowed with aggressive management. Much of the burden for this management falls on the person, increasing the importance of good teaching.

THE PERSON WITH A RETINAL DETACHMENT

The retina contains the photoreceptors of the eye, which allow the perception of light and initial processing of images and stimuli for transmission to the optic centre of the brain. Disruption of this neural layer of the eye by trauma or disease interferes with light perception and image transmission, potentially resulting in blindness.

Both primary eye conditions and systemic diseases can affect the retina and interfere with vision.

Pathophysiology and manifestations

Separation of the retina or sensory portion of the eye from the choroid, the pigmented vascular layer, is known as a **retinal detachment**. Although retinal detachment may be precipitated by trauma, it usually occurs spontaneously. The vitreous humour normally adheres to the retina at the optic disc, the macula and the periphery of the eye. With ageing, the vitreous humour shrinks and may pull the retina away from the choroid. Ageing therefore is a common risk factor, as are myopia and aphakia—absence of the lens (e.g. following lens removal for cataracts) (Porth & Matfin, 2014; Papadakis & McPhee, 2015).

The retina may actually tear and fold back on itself, or may remain intact but no longer adhere to the choroid. A break or tear in the retina allows fluid from the vitreous cavity to enter the defect. This, along with fluid that escapes from choroid

vessels, the pull of gravity and traction exerted by the vitreous humor, separates the retina from the choroid. The detached area may rapidly increase in size, increasing loss of vision. Unless contact between the retina and choroid is re-established, the neurons of the retina become ischaemic and die, causing permanent vision loss. For this reason, retinal detachment is a true medical emergency, requiring prompt ophthalmological referral and treatment.

The person experiences floaters, 'spots', lines or flashes in the visual field when the retina detaches. Often the person describes the sensation of having a curtain drawn across the vision, much like a curtain being drawn over a window. The area of the visual field affected is directly related to the area of detachment. For example, because light rays cross as they pass through the lens, a retinal tear in the superior portion of the eye results in a deficit in the lower part of the visual field. The person feels no pain and the eye appears normal to visual inspection. Common manifestations of retinal detachment are listed in the box below.

MANIFESTATIONS Retinal detachment

- Floaters: irregular dark lines or spots in the field of vision.
- Flashes of light.
- Blurred vision.
- Progressive deterioration of vision.
- Sensation of a curtain or veil being drawn across the field of vision.
- If the macula is involved, loss of central vision.

INTERPROFESSIONAL CARE

Retinal detachment is a medical emergency; prompt treatment is necessary to preserve vision. The manifestations and examination of the ocular fundus by ophthalmoscopy establish the diagnosis of retinal detachment, as early diagnosis and intervention are vital. If the condition is left untreated, the detached portion will become necrotic because of separation from the vascular supply of the choroid. The result is permanent blindness in that portion of the eye. If an ophthalmologist is not readily available, the person's head is positioned so that gravity pulls the detached portion of the retina into closer contact with the choroid.

Interventions are directed towards bringing the retina and choroid back into contact and re-establishing the blood and nutrient supply to the retina. Either cryotherapy, using a supercooled probe, or laser photocoagulation may be used to create an area of inflammation and adhesion to 'weld' the layers together.

A surgical procedure called *scleral buckling* also may be used. In this procedure, an indentation or fold is created in the sclera, bringing the choroid into contact with the retina. Contact is maintained with a local implant on the sclera or an encircling strap or 'buckle'. Air may also be injected into the vitreous cavity, a procedure called pneumatic retinopexy. The person is positioned so that the air bubble pushes the detached portion of the retina into contact with the choroid.

With a retinal tear, it may be necessary to use surgical instruments to manipulate the detached section of retina into place. Air or a liquid is then injected into the vitreous to maintain retinal contact with the choroid or laser therapy is used to create a bond.

Nursing care

The nursing focus for the person with a detached retina is on early identification and treatment. Because early intervention is vital to preserve the person's sight, nurses must recognise early manifestations of retinal detachment and intervene appropriately to obtain definitive treatment for the person. Retinal detachment can be successfully treated on an outpatient basis, often in an ophthalmologist's office. For these people, the nursing focus is on education.

Ineffective retinal tissue perfusion

Restoring contact between the retina and choroid is a priority of nursing and medical care for the person with retinal detachment. Vitreous humour may leak through a retinal tear and fluid exudate may collect behind the tear, causing further detachment. If the macula is detached, central vision is lost and the prognosis for full vision restoration is poorer.

CONSIDERATION FOR PRACTICE

Carefully assess anyone who complains of a sudden rapid loss of vision because this often signals a medical emergency.

- Assess for other manifestations of eye disease. Retinal detachment is painless and has no outward manifestations. *The person with a red eye or cloudy cornea may be experiencing acute angle-closure glaucoma rather than retinal detachment.*
- Notify the doctor and ophthalmologist immediately. *Immediate medical intervention is required in people with retinal detachment to preserve vision.*
- Position so the area of detachment is inferior. For instance, for a superior temporal retinal detachment of the right eye (with corresponding vision loss in the inferior medial visual field of that eye), place supine with the head turned to the right. *Correct positioning allows the contents of the posterior portion of the eye to place pressure on the detached area, bringing the retina in closer contact with the choroid.*

Anxiety

Retinal detachment causes a rapid decline in vision in the affected eye, often occurring spontaneously and without pain. Unless previous episodes have occurred, the person usually does not know what is causing the problem. Anxiety and fear of complete vision loss are common, expected reactions.

- Maintain a calm, confident attitude while carrying out priority interventions. *Administering care in a calm, although urgent, manner helps reassure the person that the problem is treatable and that appropriate measures are being taken.*
- Reassure that most retinal detachments are successfully treated, usually on an outpatient basis. *Reassurance can help allay the person's fear of permanent vision loss.*
- For spontaneous detachments, assure the person that they did not cause the detachment to occur. *The person may believe that the detachment is related to a specific activity and feel guilty for 'causing' this loss of vision.*
- Explain all procedures fully, including the reason for positioning. *Explanations facilitate understanding and help relieve anxiety in unfamiliar settings.*
- Allow supportive family members or friends to remain with the person as much as possible. *Additional support helps lower the person's anxiety level.*

Community-based care

Teaching for the person undergoing surgical repair of retinal detachment is similar to that for people experiencing other types of eye surgery. If the retina remains detached, provide instructions about the change in peripheral vision or other visual fields, and changes in depth perception.

Discuss the following topics with the person and family to prepare for home care:

- limitations on positioning the head before or following repair
- activity restrictions such as no bending or straining at stool movement
- use of eye shield
- early manifestations and the importance of seeking immediate treatment
- follow-up treatment with the ophthalmologist.

THE PERSON WITH RETINITIS PIGMENTOSA

Retinitis pigmentosa is a hereditary degenerative disease characterised by retinal atrophy and loss of retinal function progressing from the periphery to the central region of the retina. It is inherited as an autosomal dominant, autosomal recessive or X-linked trait, and may be associated with other genetic defects (Longo et al., 2012; Porth & Matfin, 2014).

In retinitis pigmentosa, the genetic defect appears to cause production of an unstable form of rhodopsin, the receptor protein of rod cells in the retina. Rod cells degenerate, initially at the periphery of the retina. The areas of degeneration and cell death slowly expand, causing vision to narrow. Central vision is finally lost as well.

The initial manifestation of retinitis pigmentosa, difficulty with night vision, is often noted during childhood. As the disease progresses, there is slow loss of visual fields, photophobia and disrupted colour vision. The progression to tunnel vision and blindness is gradual; the person may be totally blind by age 40.

Currently, there is no effective treatment for retinitis pigmentosa. Research into defective rhodopsin holds future promise for the development of therapy that may at least slow its progress.

People with retinitis pigmentosa may benefit from low-vision aids, much like those for the person with macular degeneration. Additionally, information about the disease and its progress is vital so the person can plan for the eventual total loss of sight. People should be referred for genetic counselling prior to starting a family to determine the risk of transmitting the disease to their children.

THE PERSON WITH HIV INFECTION

More than 50% of people infected with the human immunodeficiency virus (HIV) develop an infectious or non-infectious ocular condition, generally as a late manifestation of the disease (Longo et al., 2012).

HIV retinopathy, seen as cotton-wool spots around the optic nerve, is the most common non-infectious ophthalmic lesion in AIDS. Cotton-wool spots indicate areas of retinal ischaemia. Microaneurysms and dot-, blot- or flame-shaped haemorrhages may also be seen in HIV retinopathy.

Neoplasms common in the person with AIDS can also affect the eye. Kaposi's sarcoma may affect the external surface or anterior segment of the eye or the eyelids. Kaposi's lesions vary in colour (red, brown or purple) and in size, shape and location. Conjunctival lesions resemble a benign subconjunctival haemorrhage. Kaposi's lesions of the lid may cause ptosis (drooping of the lid) and abnormal lid function. Vision or eye position and movement may be affected by the tumour or by the effect of increased intracranial pressure on the cranial nerves.

The most serious and frequent opportunistic eye infection associated with HIV infection is cytomegalovirus (CMV) retinitis. CMV retinitis generally develops when CD4 cell counts drop below 50 mL. Initially unilateral, CMV retinitis commonly progresses to become bilateral because of the systemic nature of the infection. CMV invades the retina of the eye directly, producing exudate and cotton-wool spots, haemorrhage, cell death and necrosis. Visual field deficits develop and can progress to eventual blindness.

Corneal ulcers from opportunistic bacterial, fungal, protozoal or viral infections are also associated with HIV infection. Toxoplasmic and fungal retinal infections may occur.

The person with an HIV-associated eye disorder may complain of a change in visual acuity, blurring, floaters or gaps in the field of vision. Extensive retinal damage may cause retinal detachment and symptoms of flashing lights, multiple floaters and a loss of vision. Because the observed changes in the retina are non-specific, it is important for the examining healthcare provider to know that the person is HIV positive in order to make an accurate diagnosis.

In addition to the general treatment of HIV infection with retroviral medications, specific therapies may be directed towards the ocular manifestations of the disease. CMV retinitis is commonly treated with the antivirals ganciclovir and foscarnet sodium.

Although treatment of ocular Kaposi's sarcoma is usually not indicated, conjunctival lesions may be excised for comfort or cosmetic reasons. Lid lesions may be treated with radiation or intralesional chemotherapy.

THE PERSON WITH AN ENUCLEATION

Occasionally, surgical removal of an eye is necessary because of trauma, infection, glaucoma, intractable pain or malignancy. This procedure is known as **enucleation**.

Enucleation is performed under local or general anaesthesia. After the globe is removed, the conjunctiva and eye muscles are sutured to a round implant inserted into the orbit to maintain its shape. A pressure dressing is left in place for 24 to 48 hours. The person is permitted out of bed on the day of surgery. Haemorrhage and infection are the most commonly seen complications.

Postoperative nursing care includes education, psychological support and observation for potential complications. The person may be instructed to apply warm compresses and instil antibiotic ointment or drops postoperatively.

Within 1 week, a temporary prosthesis called a conformer is fitted into the empty socket. The permanent prosthesis is individually designed to closely resemble the person's other eye. The prosthesis can be fitted 1 to 2 months after surgery. Often it is difficult to discern which eye is functional and which is the prosthesis. Procedure 45.1 outlines the proper way to remove and reinsert an eye prosthesis when the person is unable to do so.

PROCEDURE 45.1 Removing and reinserting a prosthetic eye

GATHER SUPPLIES

- Gloves
- Clean basin or plastic denture cup
- Sterile normal saline or soap and water for cleaning the prosthesis
- Gauze squares or cotton cloth for cleaning the socket
- A bulb syringe for irrigation if necessary

BEFORE THE PROCEDURE

Most people who have an artificial eye provide self-care and require little assistance. However, it may be necessary for the nurse to remove an eye prosthesis from the unconscious or debilitated person. If the person is conscious, explain the procedure and provide for privacy.

PROCEDURE

- Follow standard precautions.
- Wash the hands and put on clean exam gloves.
- To remove the prosthesis, do one of the following:
  - Pull down the lower lid and gently exert outward and upward pressure on the lower edge of the prosthesis. This pressure usually causes the prosthesis to slip out.
  - Pull down the lower lid and apply a moistened suction cup to the prosthesis by squeezing the device. Twist gently to remove the prosthesis from the socket.
- Wash the prosthesis using mild soap and water or normal saline. Rinse thoroughly. Do not use abrasives or chemicals for cleaning.
- If the prosthesis is not immediately replaced in the eye socket, store it in a clearly labelled plastic container lined with a soft cloth or gauze squares. Avoid scratching or damaging the prosthesis. Store it in a safe place to prevent loss.
- If irrigation of the eye socket is ordered, have the person lean over a sink or basin if possible or position on the affected side with a clean emesis basin to hold the irrigant as it flows out of the socket. Gently hold the lids open and irrigate the socket using a bulb syringe and clean warm water.
- Reinsert the prosthesis.
  a. Moisten the prosthesis with warm normal saline or water.
  b. Gently hold the lids open. Insert the upper edge of the prosthesis under the upper lid first, then the lower edge under the lower lid using slight pressure.
  c. If a suction device is used, attach it to the cleaned prosthesis over the pupil. Holding the lids open, insert the prosthesis using the above procedure, then remove the suction cup by squeezing it gently and exerting slight pressure on the edge of the cup with the lower lid.

AFTER THE PROCEDURE

- Ensure that the person is comfortable. Chart the procedure and any abnormal findings, such as drainage or inflammation.

EAR DISORDERS

For a person to hear, sound waves must enter the external auditory meatus and travel through the ear canal to vibrate the tympanic membrane and bony structures of the middle ear, which in turn activate the receptors of the cochlea. Trauma or disease involving any portion of this pathway can affect hearing. **Tinnitus**, the perception of sound such as ringing, buzzing or roaring in the ears, is another potential result of problems affecting the auditory system.

Disorders of the external ear, including the auricle, auditory meatus and ear canal, can affect the conduction of sound waves and hearing. Obstruction of the external auditory canal or damage to the tympanic membrane, which separates the outer from the middle ear, may lead to conductive hearing loss. Infection or inflammation, trauma and obstruction of the ear canal with cerumen (wax) or a foreign body are the most common conditions affecting the external ear.

Disorders of the middle ear may be either acute or chronic. Unless these disorders are treated promptly and effectively, damage and scarring of middle ear structures can result in a permanent conductive hearing loss. Infectious or inflammatory disorders such as otitis media and mastoiditis are the most common conditions affecting the middle ear. Otosclerosis, a genetic condition, may also affect the structures of the middle ear.

THE PERSON WITH OTITIS EXTERNA

Otitis externa is inflammation of the ear canal. Commonly known as *swimmer's ear*, it is most prevalent in people who spend significant time in the water. Competitive athletes, including swimmers, divers and surfers, are particularly prone to otitis externa. Wearing a hearing aid or ear plugs, which hold moisture in the ear canal, is an additional risk factor. Although *Pseudomonas aeruginosa* or other bacterial infection is the most common cause, external otitis may also be due to fungal infection, mechanical trauma (such as cleaning the ear with a toothpick) or a local hypersensitivity reaction.

Pathophysiology and manifestations

Disruption of the normal environment within the external auditory canal typically precedes the inflammatory process. Retained moisture, cleaning or drying of the ear canal removes the protective layer of cerumen, an acidic, water-repellent substance with antimicrobial properties. Its removal leaves the skin of the ear canal vulnerable to invasion and infection. For surfers, the presence of *exostoses*, bony growths in the ear canals resulting from prolonged exposure to cold, predisposes to impaction and retained moisture within the canal.

The person with otitis externa often complains of a feeling of fullness in the ear. Ear pain typically is present and may be severe. The pain of otitis externa can be differentiated from that associated with otitis media by manipulation of the auricle. In external otitis, this manoeuvre increases the pain, whereas the person with otitis media experiences no change in pain perception. Odourless watery or purulent drainage may be present. The ear canal appears inflamed and oedematous on examination.

INTERPROFESSIONAL CARE

Management of the person with an external ear disorder focuses on restoring the normal balance of the external ear and canal and teaching the person how to prevent future problems.

For otitis externa, the following steps are recommended in treatment:

- thorough cleansing of the ear canal, particularly if drainage or debris is present
- treatment of the infection with local antibiotics; if cellulitis is present, systemic antibiotics may be necessary
- medication to relieve the pain and itching
- education on the prevention of future episodes of swimmer's ear.

Topical antimicrobials used to treat otitis externa include chloramphenicol, framycetin, neomycin, gramicidin, ciprofloxacin and bacitracin. Nystatin, an antifungal agent, is also used (Bryant & Knights, 2014).

A topical antibiotic is often prescribed for the treatment of otitis externa. A topical corticosteroid may be ordered in combination with the antibiotic to provide immediate relief of the pain, swelling and itching. Chloramphenicol ear drops (broad-spectrum antibiotic) are effective against *Pseudomonas*, *Staphylococcus aureus* and *Enterobacter*. Other ear drop preparations include aminoglycoside antibiotics (framycetin and neomycin); however, it is important to identify known sensitivity to any of the drugs in this preparation prior to initiating therapy. People who are sensitive to neomycin may develop dermatitis, in which case the drug must be stopped.

Nursing care

External otitis can cause severe pain and discomfort. Although the disorder is rarely serious enough to require hospitalisation, the nurse teaches the person about the disorder, comfort measures and prevention of future episodes.

Nursing diagnoses and interventions

Impaired tissue integrity

External otitis may result from attempts to clean the ear canal with a toothpick, cotton-tipped applicator or other implement that damages the skin, allowing an infectious organism to invade the tissue. Even if the canal is not damaged by attempts

to clean it, the cleaning process often interrupts normal mechanisms, causing cerumen and debris to collect in the canal. The collected debris, in turn, tends to trap water within the canal, causing maceration of the skin.

- Inform that ear canals rarely need cleansing beyond washing of the external meatus with soap and water. Educate people of all ages not to clean ear canals with any implement. '*Cleaning' increases the risk of tissue damage and impairs the normal mechanism that clears the canal of accumulated cerumen and debris.*
- Educate person (and, if necessary, a family member) about how to instil prescribed ear drops:
  a. Wash hands.
  b. Warm the medication briefly by holding the container in the hand or placing it in a pocket for approximately 5 minutes before instilling the drops. *Warming the medication promotes comfort.*
  c. Lie on the unaffected side; if sitting, tilt the head towards the unaffected side. *This position allows gravity to assist in moving the medication to the inner portion of the ear canal.*
  d. Partially fill the ear dropper with medication.
  e. Using the non-dominant hand, straighten the ear canal by pulling the pinna of the ear up and back. *Straightening helps the medication travel along the length of the canal.*
  f. Administer the prescribed number of drops into the ear canal. *It is important that the full amount of prescribed medication be administered to penetrate the length of the canal and achieve full effectiveness.*
  g. Remain in the side-lying position for approximately 5 minutes after the instillation of drops. *This position allows the medication to penetrate into deeper portions of the canal and prevents it from running out when the head is moved upright.*
  h. Loosely place a small piece of cotton in the auditory meatus for 15 to 20 minutes. *The cotton helps keep the medication in the canal.*
- Educate to avoid getting water in the affected ear until it is fully healed. Cotton balls may be used while showering to prevent water from entering the ear canal. *The person should refrain from water sports and activities until approved by the primary care provider. Retained moisture in the ear canal can further impair skin integrity, increasing inflammation.*

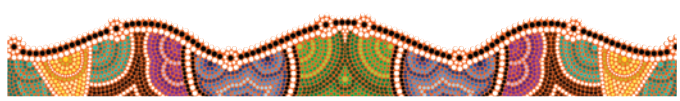
Community-based care

The person is ultimately responsible for carrying out the prescribed treatment regimen in external otitis and for implementing measures to prevent future episodes. Education is vital. Provide verbal and written instructions on use of the prescribed medications. Educating the person about care measures to prevent recurrent episodes is especially important in swimmers, divers and surfers (see Box 45.2).

Cellulitis of the surrounding tissue is a possible complication of external otitis. Instruct the person to report to the primary care provider any increase in pain, swelling or redness of surrounding tissues; fever; or other manifestations of infection such as malaise or increased fatigue.

BOX 45.2 Teaching to prevent otitis externa

- Stay out of the water until the acute inflammatory process is completely resolved. Ideally, allow 7 to 10 days before resuming water activities.
- Take precautions to keep the ear canal dry while in the water:
  a Use silicone ear plugs, which can keep water out of the ear without reducing hearing significantly.
  b Wear a tight-fitting swim cap or wetsuit hood, especially in cold ocean water. Although these do not prevent water from entering the ear, they protect the ear from the cold and possibly slow the formation of bony growths in the ears. They also protect the ear from sand and other water debris.
- Immediately after swimming, dry the ear canal. Allow water to drain by tilting the head and jumping to shake water out of the ear. Dry the outer ear with a towel, then use a hair dryer on the lowest setting several centimetres from the ear to dry the canal.
- Do not insert cotton swabs or other objects into the ear canal to dry it. This removes the protective layer of cerumen and may damage the skin of the canal, increasing the risk of bacterial infection. In addition, if debris such as sand is present, the swab may actually push debris further into the canal, forming an impacted mass.
- Consult primary care provider about using a drying agent in the ear canal after swimming. A 2% acetic acid solution or 2% boric acid in ethyl alcohol is effective in drying the canal and restoring its normal acidic environment.
- If it is necessary to remove impacted debris from the ear canal, irrigate the ear with warm tap water. A bulb syringe available over the counter or a 20 mL syringe attached to a short Teflon intravenous catheter (with the needle removed) is effective. With the head tilted towards the affected side, direct a stream of warm water towards the upper wall of the ear canal, allowing the water to run out into a bowl or sink. Repeated instillations may be necessary to break up and flush out impacted wax and debris.
- Follow manufacturer's directions for cleaning and disinfecting pools and spas (hot tubs). Use a pool test kit to check for adequate disinfectant and pH levels before entering.



THE PERSON WITH IMPACTED CERUMEN OR A FOREIGN BODY

The external auditory canal can be obstructed by cerumen or foreign bodies. The curved shape and narrow lumen of the canal make it particularly vulnerable to obstruction.

Pathophysiology and manifestations

As cerumen dries, it moves down and out of the ear canal. In some individuals it tends to accumulate, narrowing the canal.

Ageing is a risk factor for impaction because less cerumen is produced and it is harder and drier. The accumulation of cerumen is often aggravated by attempting to remove it using cotton-tipped swabs or hair pins, which pack it more deeply into the ear canal.

A variety of objects become foreign bodies in the ear canal. In adults, implements used to clean the ear canal may break and become lodged. Insects also may enter the ear canal and be unable to exit.

When the ear canal becomes occluded with either cerumen or a foreign body, the person experiences a conductive hearing loss in the affected ear. Manifestations include a sensation of fullness, along with tinnitus and coughing due to stimulation of the vagal nerve. The foreign body or impacted cerumen may be visualised on otoscopy. Impacted cerumen appears as a yellow, brown or black mass in the canal.

INTERPROFESSIONAL CARE

Treatment focuses on clearing the canal. If there is no evidence of tympanic membrane perforation, irrigation of the canal is often the initial therapy.

Impacted wax, objects or insects may require physical removal using an ear curette, forceps or right-angle hook inserted via an otoscope and ear speculum. Mineral oil or topical lignocaine drops are used to immobilise or kill insects prior to their removal from the ear. When an organic foreign body such as a bean or an insect is suspected, water should not be instilled into the ear canal because it may cause the object to swell, making its removal more difficult. Smooth, round objects present the biggest challenge to remove from the ear canal. Suction applied using a piece of soft intravenous tubing may be effective.

Nursing care

Nurses are often involved in identifying and relieving obstructions of the ear canal, especially in outpatient and community settings. Any person with evidence of a new conductive hearing loss or complaints of discomfort and fullness in one ear should be evaluated for possible obstruction. Inability to visualise the tympanic membrane or observation of a dark, shiny mass obstructing the canal may indicate a need for irrigation or other procedure to clear the canal. It is important to determine that the tympanic membrane is intact before irrigating; assessment by a doctor or advanced practitioner may be necessary if a ruptured membrane is suspected.

Because obstruction of the ear canal with cerumen or a foreign body is generally preventable, education is a key component of nursing care. People need to know appropriate care measures for the external ear. Although the ear canal rarely needs cleaning, the person prone to cerumen impaction requires education regarding the use of mineral oil or commercial products to soften wax, and irrigation to remove it. All people should understand the importance of not inserting anything smaller than a finger wrapped with a washcloth into the ear canal to avoid trauma to the canal or eardrum. Stress the risk of impacting cerumen against the tympanic membrane when using cotton-tipped swabs to clean the ear canal. Additionally, the swab may break and lodge in the canal. If ear drops have been prescribed, educate the person and a family member about how to instil them.

THE PERSON WITH OTITIS MEDIA

Otitis media, inflammation or infection of the middle ear, primarily affects infants and young children but may also occur in adults. It can persist from infancy through adolescence and adulthood (Burrow, Galloway & Weissofner, 2009). The tympanic membrane, which separates the middle ear from the external auditory canal, protects the middle ear from the external environment. The eustachian (auditory) tube connects the middle ear with the nasopharynx to help equalise the pressure in the middle ear with the atmospheric pressure. Unfortunately, this connecting tube also provides a route by which infectious organisms enter the middle ear from the nose and throat, causing otitis media, the most common disease of the middle ear. This is the most common cause of hearing loss in the Australian Indigenous population.

Pathophysiology

There are two primary forms of otitis media: (1) serous, and (2) acute or suppurative. Both forms are associated with upper respiratory infection and eustachian tube dysfunction. The eustachian tube is narrow and flat, normally opening only during yawning and swallowing. Allergies or upper respiratory tract infections can cause oedema of the tube lining, impairing its function. Air within the middle ear is trapped and gradually absorbed, creating negative pressure in this space.

Serous otitis media

Serous otitis media (also called *otitis media with effusion*) occurs when the eustachian tube is obstructed for a prolonged time, impairing equalisation of air pressure in the middle ear. Air within the middle ear space is gradually absorbed; the tube obstruction prevents more air from entering the middle ear. The resulting negative pressure in the middle ear causes sterile serous fluid to move from the capillaries into the space, forming a sterile effusion of the middle ear.

Upper respiratory infections or allergies such as hay fever predispose the person to serous otitis media. In addition, people with narrowed or oedematous eustachian tubes may also be subject to barotrauma or barotitis media. In these people, the middle ear cannot adapt to rapid changes in

barometric pressure such as those that occur during air travel or underwater diving. Barotrauma tends to occur during descent in an aeroplane because negative pressure within the middle ear causes the eustachian tube to collapse and lock. However, underwater diving places even greater stress on the eustachian tube and middle ear.

MANIFESTATIONS Typical manifestations of serous otitis media include decreased hearing in the affected ear and complaints of 'snapping' or 'popping' in the ear. On examination, the tympanic membrane demonstrates decreased mobility and may appear retracted or bulging. Fluid or air bubbles are often visible behind the drum. Severe pressure differences such as those occurring with barotrauma may cause acute pain, haemorrhage into the middle ear, rupture of the tympanic membrane or even rupture of the round window with sensory hearing loss and severe **vertigo** (a sensation of whirling or rotation). *Haemotympanum*, bleeding into or behind the tympanic membrane, may be observed on otoscopic examination.

Acute otitis media

The eustachian tube also provides a route for the entry of pathogens into the normally sterile middle ear, resulting in acute or suppurative otitis media. Acute otitis media typically follows an upper respiratory infection. Oedema of the eustachian tube impairs drainage of the middle ear, causing mucus and serous fluid to accumulate. This fluid is an excellent environment for the growth of bacteria, which may enter from the oronasopharynx via the eustachian tube. Although a viral upper respiratory infection may predispose the person to a middle ear infection, the bacteria *Streptococcus pneumoniae*, *Haemophilus influenza* and *Streptococcus pyogenes* account for most cases of otitis media in adults. Invasion and colonisation of the middle ear by bacteria and the resultant migration of white blood cells cause pus formation. Accumulated pus can increase middle ear pressure sufficiently to rupture the tympanic membrane. The bacterial infection may also migrate internally, causing mastoiditis, brain abscess or bacterial meningitis. A more common complication of otitis media is a persistent conductive hearing loss, which typically resolves when the middle ear effusion clears.

MANIFESTATIONS The person with acute otitis media experiences mild to severe pain in the affected ear. The person's temperature is often elevated. Diminished hearing, dizziness, vertigo and tinnitus are common associated complaints. Pus within the mastoid air cells often causes mastoid tenderness in acute otitis media. On otoscopic examination, the tympanic membrane appears red and inflamed or dull and bulging (see Figure 45.15). Decreased movement of the membrane is demonstrated by tympanometry or air insufflation. Spontaneous rupture of the tympanic membrane releases a purulent discharge. **Myringotomy** (an incision of the tympanic membrane) may be performed to relieve the pressure.

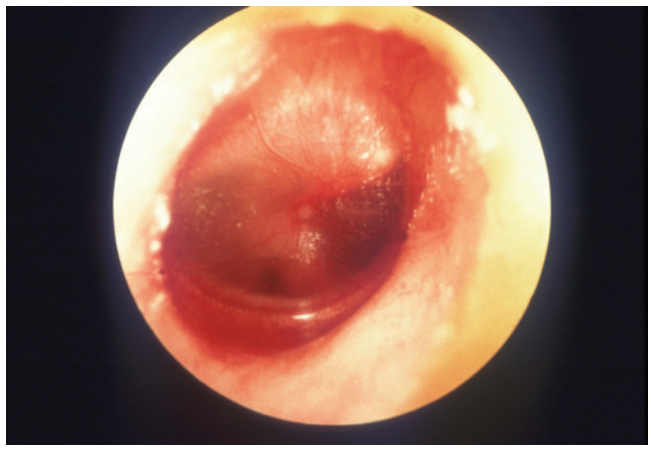


FIGURE 45.15 ■ A red, bulging tympanic membrane of otitis media

Source: © Medical-on-Line/Alamy..

INTERPROFESSIONAL CARE

The diagnosis of otitis media is usually based on the person's history and the physical examination. The tympanic membrane can be visualised and its mobility evaluated using a pneumatic otoscope that allows a puff of air to be instilled into the ear canal. Generally, the tympanic membrane moves slightly when air is instilled or the person performs the Valsalva manoeuvre. Less movement is seen in people with eustachian tube dysfunction and acute otitis media with effusion.

Diagnosis

- *Impedance audiometry*, also known as tympanometry, is an accurate diagnostic test for otitis media with effusion. A continuous tone is delivered to the tympanic membrane by an audiometer with a sealed probe tip. Compliance of the tympanic membrane and middle ear is measured by recording energy reflected from the membrane surface. With middle ear effusion, compliance is reduced.
- A *full blood count (FBC)* may be done to assess for an elevated WBC count and increased numbers of immature cells indicative of acute bacterial infection.
- If the tympanic membrane has ruptured or a tympanocentesis or myringotomy is performed, drainage is cultured to determine the infecting organism.

Medications

When eustachian tube dysfunction and serous otitis media do not spontaneously resolve or lead to hearing loss, a short course of an anti-inflammatory drug (e.g. oral prednisone for 7 days) is prescribed to reduce mucosal oedema of the tube and improve its patency.

Although a decongestant or antihistamine may be used, there is little evidence of their effectiveness in treating serous otitis media. Antibiotic/corticosteroid ear drops or ointments can be prescribed. See Chapter 12 for the nursing implications of corticosteroid medications.

The person with auditory tube dysfunction may be taught to auto-inflate the middle ear by performing the Valsalva manoeuvre or by forcefully exhaling against closed nostrils. Additionally, the person is advised to avoid air travel and underwater diving.

Acute otitis media usually is treated with antibiotic therapy, especially amoxicillin, trimethoprim-sulfamethoxazole, cefaclor or azithromycin for 5 to 10 days. This course of treatment is long enough to ensure eradication of the infective organism, yet short enough to reduce the incidence of bacterial resistance. (See Chapter 11 for further discussion of antibiotics.) Symptomatic relief may be provided by analgesics, antipyretics, antihistamines and local application of heat.

Surgery

A myringotomy or tympanocentesis may be performed to relieve excess pressure in the middle ear and prevent spontaneous rupture of the eardrum. To perform a tympanocentesis, the doctor inserts a 20-gauge spinal needle through the inferior portion of the tympanic membrane, allowing aspiration of fluid and pus from the middle ear to relieve pressure and, if necessary, obtain a specimen for culture. Myringotomy may be performed to relieve severe pain or when complications of acute otitis media, such as mastoiditis, are present. As soon as the pressure is released, pain subsides and hearing improves.

People who do not respond to antibiotic therapy may require myringotomy with insertion of ventilation (tympanostomy) tubes. Small tubes are inserted into the inferior portion of the tympanic membrane, providing for ventilation and drainage of the middle ear during healing. The tube is eventually extruded from the ear and the tympanic membrane heals. While the tube is in place, it is important to avoid getting any water in the ear canal because it may then enter the middle ear space.

Nursing care

People with otitis media are commonly treated in outpatient and community settings. The nursing role is primarily one of support and education. A comprehensive intersectoral and multidisciplinary approach specifically addressing prevention and early intervention will assist in improving awareness in the Australian Indigenous population, to reduce the incidence of this condition.

Health promotion

Health promotion for otitis media focuses on educating people about the importance of seeking medical care for prolonged, severe ear pain with or without drainage combined with an upper respiratory tract infection. Untreated or repeated attacks of otitis media can progress to a chronic form of otitis media, acute mastoiditis or eardrum perforation.

Assessment

Collect assessment data through a health history and physical examination (Chapter 44).

- *Health history:* recent upper respiratory infection; presence, intensity and nature of pain in affected ear; sense of fullness or pressure in the ear; change in hearing; snapping or popping sensation in the affected ear; presence of vertigo.
- *Physical examination:* temperature; hearing test; inspect tympanic membrane.

Nursing diagnoses and interventions

Pain can be a significant problem for people with otitis media, as can the risk of damage to delicate tissues of the middle ear by the infectious and inflammatory processes.

Pain

Tissue oedema, effusion of the middle ear and the inflammatory response can affect the pain-sensitive tissues of the middle ear in otitis media, causing acute discomfort. This discomfort is increased by pressure changes, such as those that occur during air travel or underwater diving.

- Assess pain for severity, quality and location. A thorough assessment is important to determine the source of the pain. *Unlike that of external otitis, the pain of otitis media is not aggravated by movement of the external ear.*
- Encourage the use of mild analgesics such as aspirin or paracetamol every 4 hours as needed to relieve pain and fever. *These non-prescription medications are effective in reducing the perception of pain. Aspirin also has anti-inflammatory properties that may help relieve the inflammation of the ear.*
- Advise to apply heat to the affected side unless contraindicated. *Heat dilates blood vessels, promoting the reabsorption of fluid and reducing swelling.*
- Instruct to avoid air travel, rapid changes in elevation or diving. *A rapid change in barometric pressure can increase the person's pain significantly.*
- Instruct to report promptly an abrupt relief of pain to the primary care provider. *Pain that subsides abruptly may indicate spontaneous perforation of the tympanic membrane with relief of pressure within the middle ear.*

Community-based care

The person who has otitis media needs educating regarding the disorder, its causes and prevention, and any specific treatment recommended or prescribed. Discuss the following topics with the person and family:

- antibiotic therapy and potential side effects
- importance of completing all ordered doses
- follow-up examinations in 2 to 4 weeks
- avoiding swimming, diving or submerging the head while bathing if ventilation tubes are in place.

If surgical intervention is necessary, educate the person and family members about the surgery and postoperative care. Provide instructions regarding any special postoperative precautions, such as avoiding water in the ear canals or avoiding sudden changes in air pressure.

THE PERSON WITH ACUTE MASTOIDITIS

The mastoid process is a portion of the temporal bone of the skull lying adjacent to the middle ear. It is full of air cavities called mastoid air cells or mastoid sinuses. The infection of acute otitis media generally extends into the mastoid air cells; effective treatment of acute otitis media eliminates the infection from the mastoid cells as well. When treatment is ineffective, pus remains in the mastoid air cells and acute **mastoiditis**, bacterial infection of the mastoid process, may develop.

The incidence of acute mastoiditis is low in countries where the prescription and use of antibiotics to treat acute otitis media is widespread. Its incidence is higher in countries where antibiotics are less likely to be prescribed.

Pathophysiology and complications

In acute mastoiditis, the bony septa between mastoid air cells are destroyed and cells coalesce to form large spaces. Portions of the mastoid process are eroded. With chronic infection, an abscess may form or bony sclerosis of the mastoid may result. Acute mastoiditis increases the risk of meningitis because only a very thin bony plate separates mastoid air cells from the brain. Fortunately, this complication is rare since the advent of effective antibiotic therapy for treating otitis media.

Manifestations

Manifestations of acute mastoiditis usually develop approximately 2 to 3 weeks after an episode of acute otitis media and include recurrent earache and hearing loss on the affected side. The pain is persistent and throbbing; tenderness is present over the mastoid process (behind the ear). It may also be red and inflamed. Swelling of the process can cause the auricle of the ear to protrude more than normal. Fever may be accompanied by tinnitus and headache, and profuse drainage from the affected ear may be noted.

INTERPROFESSIONAL CARE

In addition to the manifestations of acute mastoiditis, loss of septa between mastoid air cells may be noted on radiological examination. Acute mastoiditis is treated aggressively with antibiotic therapy tailored to the infecting organism. Antibiotics are continued for at least 14 days. Infections that do not respond to medical therapy or that pose a high risk of spreading to the brain may necessitate a *mastoidectomy*, the surgical removal of the infected mastoid air cells, bone and pus, and inspection of the underlying dura for possible abscess. The extent of tissue destruction determines the extent of surgery required. In a modified mastoidectomy, as much tissue is preserved as possible to avoid disruption of hearing. A radical mastoidectomy involves removal of middle ear structures, including the incus and malleus, as well as the diseased portions of the mastoid process. Unless reconstruction is performed at the time of surgery, this surgery results in conductive hearing loss. **Tympanoplasty**, surgical reconstruction of the middle ear, can restore or preserve hearing.

Nursing care

Prevention is the primary focus of collaborative and nursing care related to mastoiditis. Adequate, effective antibiotic treatment of acute otitis media prevents mastoiditis in nearly all instances.

Following surgical intervention, carefully assess the wound and drainage for evidence of infection or other complications. The person's hearing may be temporarily or permanently affected, depending on the extent of the surgery. If the person has impaired hearing in the unaffected ear as well, develop a means of communication with the person prior to surgery. If the hearing is preserved in the unaffected ear, position the person with that ear towards the door. Speak slowly and clearly; do not shout or speak unusually loudly. Be sure that family and staff know about the person's hearing loss and use appropriate communication techniques. Assist the person with ambulation initially because dizziness and vertigo are not unusual following surgery. Nursing care of the person having ear surgery is discussed in the box below.

Community-based care

When educating about acute mastoiditis, stress the importance of complying with the prescribed antibiotic therapy and recommendations for follow up. Instruct the person and family to report any adverse reactions to the primary care provider so that therapy can be adjusted. Educate the person and family about how to change the surgical dressing using aseptic technique. Provide referrals to appropriate community agencies for the person with a new hearing loss resulting from mastoiditis or its treatment.

THE PERSON WITH CHRONIC OTITIS MEDIA

Chronic otitis media involves permanent perforation of the tympanic membrane, with or without recurrent pus formation. Changes in the mucosa and bony structures (ossicles) of the middle ear often accompany chronic otitis media. It usually is the result of recurrent acute otitis media and eustachian tube dysfunction, but may also result from trauma or other diseases.

Marginal perforations, which usually occur in the posterior–superior portion of the tympanic membrane, are associated with more complications than central perforations. With marginal perforations, squamous epithelium may migrate from the ear canal into the middle ear, where it begins to desquamate and accumulate, forming a *cholesteatoma* (a cyst or mass filled with epithelial cell debris). Its incidence is highest in children and young adults. The desquamating epithelium continues to accumulate and remains infected, producing collagenases (enzymes) that destroy adjacent bone. The inflammatory process

NURSING CARE OF THE PERSON having ear surgery

PREOPERATIVE CARE

- Review Chapter 3 for routine preoperative care.
- Assess hearing and/or verify documentation of preoperative hearing assessment. *This data is important in evaluating the results of the surgical procedure.*
- Establish a means of communication to be used after surgery, *when hearing may be impaired*.
- Explain that blowing of the nose, coughing and sneezing need to be restricted postoperatively to prevent pressure changes in the middle ear and potential disruption of the surgical site. *Keeping the mouth open during a cough or sneeze minimises pressure changes in the middle ear. Providing education and the opportunity to practise before surgery promotes cooperation in the postoperative period.*

POSTOPERATIVE CARE

- Review Chapter 3 for routine postoperative care.
- Assess for bleeding and/or drainage from the affected ear. *Infection and haemorrhage are possible complications.*
- Administer anti-emetics as ordered to prevent vomiting. *Vomiting may increase the pressure in the middle ear, disrupting the surgical site.*
- Elevate the head of the bed and position the person on the unaffected side. *This position minimises the pressure in the middle ear.*
- Assess for vertigo or dizziness, especially with ambulation or movement in bed. Avoid unnecessary movements such as turning. *Take measures to ensure safety during ambulation. Surgery on the ear may disrupt equilibrium, increasing the risk of falling.*
- Assess hearing postoperatively. Stand on the unaffected side to communicate and use other measures such as written messages as needed for effective communication with the person with impaired hearing. Reassure the person that decreased hearing acuity immediately after surgery is expected. *Hearing improvement is an expected result of the ear surgery and typically does not occur until ear plugs are removed and oedema and drainage at the operative site have resolved. If no reconstruction of the middle ear is carried out or the cochlea is involved, permanent hearing loss in the affected ear may be an expected result.*
- Remind to avoid coughing, sneezing or blowing the nose. *These increase pressure in the middle ear.*

HEALTH EDUCATION FOR THE PERSON AND FAMILY

- Provide education and instructions for home care:
  a. To prevent contamination of the ear canal, avoid showers, shampooing and immersing the head until the doctor says you can do so.
  b. Keep the outer earplug clean and dry, changing it as needed. Do not remove inner ear dressing until instructed to do so by the doctor.
  c. Avoid blowing the nose; if you need to cough or sneeze, keep the mouth open.
  d. Do not swim or dive without doctor approval. Check with your doctor regarding air travel.
  e. Meclizine hydrochloride or other anti-emetic and/or antihistamine medication may be necessary for up to 1 month following surgery.
  f. Fever, bleeding, increased drainage, increased dizziness or decreased hearing after discharge may indicate a complication. Notify the doctor if any of these occur.

impairs the blood supply to the stapes, causing its destruction and conductive hearing loss. Cholesteatomas are benign and slow-growing tumours which can enlarge to fill the entire middle ear. Untreated, the cholesteatoma can progressively destroy the ossicles and erode into the inner ear, causing profound hearing loss.

Systemic antibiotics are prescribed for exacerbations of purulent otitis media. Tympanic membrane perforation is repaired with a tympanoplasty to restore sound conduction and the integrity of the middle ear. A cholesteatoma may require delicate surgery for its removal. If at all possible, radical mastoidectomy with removal of the tympanic membrane, ossicles and tumour is avoided.

As with other complications of acute otitis media, a priority of nursing care is prevention of chronic otitis media and cholesteatoma. People with chronic otitis media need to understand various treatment options and their risks and benefits, as well as the long-term risk of not treating a perforated tympanic membrane. They are also educated on how to instil ear drops, to clean the external auditory meatus and to not irrigate the ear when the tympanic membrane is perforated or if they think it might be.

If surgical treatment of chronic otitis media will affect the person's hearing, include this information in preoperative teaching. Teach the person and family how to use alternative means of communication if this will be necessary postoperatively. When an assistive device is ordered, teach the person and a family member about its use.

THE PERSON WITH OTOSCLEROSIS

Otosclerosis is a common cause of conductive hearing loss. Abnormal bone formation in the osseous labyrinth of the temporal bone causes the footplate of the stapes to become fixed or immobile in the oval window. The result is a conductive hearing loss.

Otosclerosis is a hereditary disorder with an autosomal dominant pattern of inheritance. It occurs most commonly in Caucasians and females. The progressive hearing loss typically begins in adolescence or early adulthood, and seems to be accelerated by pregnancy. Although both ears are affected, the rate of hearing loss is asymmetrical. Because bone conduction of sound is retained, the person may be able to use the telephone but have difficulty conversing in person. Tinnitus may also be associated with this condition.

On examination, a reddish or pinkish-orange tympanic membrane may be noted because of increased vascularity of the middle ear. The Rinne test (Chapter 44) shows bone sound conduction to be equal to or greater than air conduction, an abnormal finding.

People with otosclerosis may choose conservative treatment, relying on a hearing aid to improve their ability to hear and interact with others. Sodium fluoride may be prescribed to slow bone reabsorption and overgrowth. Surgical treatment involves a stapedectomy and middle ear reconstruction, or a stapedotomy. A *stapedectomy* is a microsurgical technique for removing the diseased stapes. A metallic prosthesis is then inserted, with one end connected to the incus and the other inserted into the oval window. *Stapedotomy* involves creation of a small hole in the footplate of the stapes and insertion of a wire or platinum ribbon prosthesis. An argon, KTP or CO_2 laser may be used for surgery. Surgery usually restores hearing for the person with otosclerosis.

Education and referral of the person to appropriate community agencies are important nursing care priorities for the person with otosclerosis. For the person who chooses surgical treatment, nursing care is similar to that for other people undergoing ear surgery. The following may be appropriate:

- *Risk of injury* related to hearing loss or postoperative vertigo.
- *Risk of disturbed auditory perception* related to bony sclerosis of the stapes.
- *Risk of impaired verbal communication* related to hearing loss.
- *Risk of anxiety* related to concern about transmission of a genetic disorder to children.

THE PERSON WITH AN INNER EAR DISORDER

Disorders affecting the inner ear are much less common than disorders of the outer or middle ear. Inner ear disorders affect equilibrium and may also affect sensorineural hearing, the perception of sound. Labyrinthitis and Ménière's disease are the most common diseases of the inner ear. Vertigo may be a disorder of the inner ear itself or a manifestation of other disorders.

Pathophysiology and manifestations

The labyrinth (the inner ear) contains the cochlea and the semicircular canals. The hair cells and neurons that allow sound perception and transmission to the auditory centre of the brain are in the cochlea. The semicircular canals filled with endolymph are the primary organs involved in maintaining equilibrium. Disruption of this portion of the ear by an inflammatory process or excess endolymph not only affects balance but may also result in permanent hearing loss.

Vertigo

Normally, the integration of input from the labyrinths, eyes, muscles, joints and neural centres maintains balance and posture. This input and integration can be affected by disorders of the labyrinth, vestibular nerve or nuclei, eyes, cerebellum, brainstem or cerebral cortex, causing vertigo. Vertigo, the sensation of movement when there is none, is a disorder of equilibrium. The sensation of whirling, rotation or movement is described as either subjective or objective.

People with subjective vertigo report the sensation of being in motion in a stable environment. This is not always a sense of spinning; the person may have a sense of tumbling or falling forwards or backwards. The sensation is reversed in objective vertigo; people report a sensation of stability in a moving environment. This motion may be perceived as the room spinning around the person or the ground rocking beneath the person's feet. Dizziness, which may be mistaken for vertigo, is a sensation of unsteadiness, lack of balance, light headedness or movement within the head. The person who is dizzy does not have the rotational sensation felt with vertigo.

Vertigo may be disabling, resulting in falls, injury and difficulty walking. Attacks of vertigo are often accompanied by nausea and vomiting, nystagmus and autonomic symptoms such as pallor, sweating, hypotension and salivation.

Labyrinthitis

Labyrinthitis, also called otitis interna, is inflammation of the inner ear. It is an uncommon disorder because the bony protection of the membranous labyrinth makes it difficult for organisms to enter the inner ear. However, bacteria, viruses and other organisms may enter and infect the inner ear through the oval window during acute otitis media, the cochlear aqueduct during meningitis, or the blood. Viral labyrinthitis is suspected when the person has a sudden onset of symptoms after an upper respiratory infection or when there is no evidence of concurrent otitis media. Labyrinthitis also may result from an autoimmune process of unknown aetiology.

MANIFESTATIONS Inflammation of the labyrinth typically causes vertigo, sensorineural hearing deficit and nystagmus (rapid involuntary eye movements).

Vertigo is the hallmark manifestation of inner ear disorders. The vertigo of labyrinthitis is severe and often accompanied by nausea and vomiting. Any movement can aggravate the vertigo, and falling is a significant risk if the person attempts to stand. Vertigo lasts days to weeks in labyrinthitis, making education a vital component of care.

Hearing loss in the ear affected by labyrinthitis may be temporary or permanent. If inflammation destroys tissue of the membranous labyrinth, the hearing loss may be complete and permanent.

The involuntary rhythmic eye movements of nystagmus may not be present in all people with labyrinthitis. When present, the eye movement is typically horizontal. Applying positive or negative pressure to the tympanic membrane of the affected ear may stimulate nystagmus, as will caloric testing (irrigating the ear canal with warm or cool water). Although nystagmus may also be a symptom of brainstem or cerebellar dysfunction, vertigo and hearing loss are not typically associated with those disorders.

Ménière's disease

Ménière's disease, also known as endolymphatic hydrops, is a chronic disorder characterised by recurrent attacks of vertigo with tinnitus and a progressive unilateral hearing loss.

This disorder affects men and women equally, with adults between the ages of 35 and 60 at highest risk. The cause of Ménière's disease is unclear, although the most common form of the disease is thought to result from viral injury to the fluid transport system of the inner ear. Other factors that may increase the risk of Ménière's disease include trauma, bacterial infections such as syphilis, autoimmune processes, vascular disorders, selected drugs and toxins (Grossman & Porth, 2014). A family history of the disease increases risk, suggesting a possible genetic link in some people.

Ménière's disease results from an excess of endolymph, the fluid in the membranous labyrinth of the inner ear. Although the precise pathophysiological mechanism leading to accumulation of endolymph is unclear, it is thought to result from impaired filtration and excretion of the fluid by the endolymphatic sac (Grossman & Porth, 2014). Excessive pressure resulting from the increased fluid volume causes neural organs of the cochlea to degenerate.

COURSE AND MANIFESTATIONS The onset of Ménière's disease may be gradual or sudden. It is characterised by recurrent attacks of vertigo, gradual loss of hearing and tinnitus. Attacks may be preceded by a feeling of fullness in the ears and a roaring or ringing sensation. The sensorineural hearing loss and tinnitus are usually unilateral but can become bilateral. Attacks of severe rotary vertigo occur abruptly and often unpredictably, lasting from minutes to hours. An attack may be linked to increased sodium intake, stress, allergies, vasoconstriction or premenstrual fluid retention. As the disease continues, hearing loss progresses and the vertigo can be severe enough to cause immobility, nausea and vomiting. Attacks are often accompanied by hypotension, sweating and nystagmus.

INTERPROFESSIONAL CARE

The manifestations associated with inner ear disorders are similar, making testing necessary to establish a diagnosis. Once the diagnosis is determined, collaborative care is directed towards managing symptoms and preventing permanent hearing loss. People with labyrinthitis or an acute attack of Ménière's disease may require hospitalisation to manage the vertigo and its effects.

Diagnosis

The following diagnostic studies may be ordered:

- *Caloric testing (electronystagmography)* evaluates the vestibulo-ocular reflex by identifying eye movements (nystagmus) in response to caloric testing. In people with impaired vestibular function, the normal nystagmus response is blunted or absent. This portion of the test is contraindicated in people who have a perforated tympanic membrane.
- *Rinne* and *Weber tests* of hearing (Chapter 44) show decreased air and bone conduction on the affected side if a sensorineural hearing loss is present. In Ménière's disease, audiology shows sensorineural hearing loss involving the low tones.
- *X-rays* and *CT scans* of the petrous bones are used to evaluate the internal auditory canal. In people with Ménière's disease, the vestibular aqueducts may be shorter and straighter than normal.
- *Glycerol test* is conducted by giving the person oral glycerol to decrease fluid pressure in the inner ear. An acute temporary hearing improvement is considered diagnostic for Ménière's disease.

Medications

A scopolamine patch may be used for people with recurrent vertigo, although adverse effects such as dry mouth, blurred vision and urinary retention may limit its use. In Ménière's disease, a diuretic such as hydrochlorothiazide may be prescribed to reduce endolymphatic pressure. A central nervous system depressant such as diazepam or lorazepam may halt an attack of vertigo. Parenteral droperidol provides both a sedative and an anti-emetic effect, making it a useful drug for acute attacks. Anti-vertigo/anti-emetic medications such as meclizine, prochlorperazine or hydroxyzine hydrochloride are prescribed to reduce the whirling sensation and nausea. If the nausea and vomiting are severe, intravenous fluids may be necessary to maintain fluid and electrolyte balance.

Treatments

Bed rest in a quiet, darkened room with minimal sensory stimuli and minimal movement provides the most comfort for the person experiencing an acute attack of vertigo.

Between acute attacks, management of the person with Ménière's disease is directed at preventing future attacks and preserving hearing. A low-sodium diet helps reduce labyrinthine pressure. The Furstenberg diet, a salt-free neutral ash diet, may be prescribed if moderate sodium restriction is ineffective in controlling attacks. People should avoid tobacco, which causes vasoconstriction and can precipitate an attack, along with alcohol and caffeine.

Surgery

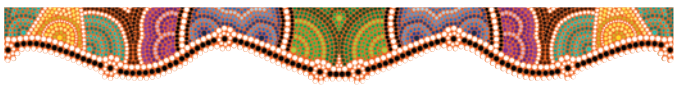
When episodes of vertigo are not controlled through medical interventions, surgery may be necessary. Surgical *endolymphatic decompression* relieves the excess pressure in the labyrinth; a shunt is then inserted between the membranous labyrinth and the subarachnoid space to drain excess fluid away from the labyrinths and maintain lower pressure. This procedure preserves hearing for most people. Vertigo is relieved in approximately 70% of people, but about half of people undergoing this procedure continue to experience sensations of fullness and tinnitus.

Destruction of a portion of the acoustic nerve is an alternative to shunting procedures. In a *vestibular neurectomy*, the portion of cranial nerve VIII that controls balance and sensations of vertigo is severed. This procedure relieves vertigo for up to 90% of people. Although there is a risk of damage to the cochlear portion of the nerve and resultant hearing loss, for most people hearing loss stabilises after neurectomy, even improving for some.

The surgery of last resort for Ménière's disease is a *labyrinthectomy*. The labyrinth is completely removed, destroying

cochlear function. This procedure is used only when hearing loss is nearly complete and vertigo is persistent. Although labyrinthectomy relieves vertigo in nearly all cases, the person may remain unsteady and have continued problems with balance.

After surgery on the inner ear, the person is positioned to minimise ear pressure and vertigo. Movement is restricted and assistance is provided when the person gets up. Anti-emetics and anti-vertigo medications are used to manage symptoms resulting from disruption of the inner ear. Complications include infection and leakage of cerebrospinal fluid.



Nursing care

The person with an inner ear disorder has multiple nursing care needs related to the manifestations of the disorder.

Health promotion

Health promotion focuses on identifying people with potential inner ear disorders. Persistent episodes of dizziness, ringing in the ears, balance problems or loss of hearing should be reported to a healthcare provider. People diagnosed early may have a lower risk of injury and can be taught strategies for maintaining, as near normal as possible, their work and social life.

Assessment

In addition to the following, assess the older person for other medical causes of imbalance and dizziness, such as neurological dysfunction, musculoskeletal and cardiovascular disorders, and endocrine problems.

- *Health history:* medication use; presence of vertigo, tinnitus, nausea and vomiting and hearing loss; balance problems; frequency and duration of symptoms; precipitating factors for an attack.
- *Physical examination:* vital signs, general health; hearing, nystagmus, balance.

Nursing diagnoses and interventions

The risk of trauma in people with inner ear disorders is great. Attacks of vertigo may occur without warning and can be so severe that the person is unable to remain upright. If frequent attacks are accompanied by nausea, nutrition may be compromised. Constant or intermittent tinnitus can interfere with sleep and rest. Finally, because nearly all inner ear disorders are associated with some degree of hearing loss, which may be progressive, the person has significant psychosocial needs.

Risk of trauma

Because of the unpredictable nature of attacks, the person with vertigo due to an inner ear disorder needs to learn strategies for dealing with an acute episode. Because vertigo tends to be chronic except in acute labyrinthitis, the emphasis is on helping the person develop strategies to reduce the frequency of attacks and the risk of injury.

- Monitor for vertigo, nystagmus, nausea, vomiting and hearing loss. *Monitoring is important to determine the severity of impairment, the duration of attacks and the person's ability to predict an impending attack.*
- Instruct to not get up without assistance during episodes of vertigo. *During attacks of vertigo, assistance reduces the risk of falling.*

CONSIDERATION FOR PRACTICE

During an acute attack of vertigo, keep on bed rest with the side rails raised and the call bell readily accessible.

- Educate to avoid sudden head movements or position changes *as sudden movement may precipitate an attack of vertigo.*
- Administer prescribed medications as ordered, including anti-emetics, diuretics and sedatives. *These medications may reduce the frequency, severity and duration of vertigo attacks.*
- Instruct to take the prescribed medication and lie down in a quiet, darkened room when an impending attack is sensed. *These measures help protect the person from injury and may shorten the duration and reduce the severity of the attack.*
- Advise to pull to the side of the road and wait for the symptoms to subside if an attack occurs while driving. *Perception and judgment necessary for safe driving may be impaired during an acute attack; pulling off the road is vital to protect the safety of the person and others.*
- Discuss the effect of unilateral hearing loss on the ability to identify the direction of sounds. To ensure safety, encourage the person to use other senses (e.g. when crossing the street). *Sound perception and differentiation of direction change when hearing is lost unilaterally, just as depth perception changes when vision is lost in one eye.*

Disturbed sleep pattern

The tinnitus often associated with inner ear disorders may be loud and continuous, interfering with the person's ability to concentrate, relax and sleep. It may be perceived as a continuous high-pitched whine, buzzing, ringing or humming sound. In some people, it may have a pulsatile quality.

- Refer for a complete hearing and ear examination if one has not been done. *Although most tinnitus is associated with hearing loss, often due to noise exposure, it may also be associated with treatable conditions such as impacted cerumen, hypertension, cerebrovascular disorders and other conditions.*
- Discuss options for masking tinnitus to promote concentration and sleep:
  a. ambient noise from a radio or sound system
  b. masking device or white-noise machine
  c. hearing aid that produces a tone to mask the tinnitus
  d. hearing aid that amplifies ambient sound.

  These techniques or devices help mask the subjective perception of tinnitus, allowing the person to focus on something other than the sound.

- Discuss the possible risks and benefits of medications to treat tinnitus. *Many medications have been used to treat tinnitus; oral antidepressants such as nortriptyline taken at bedtime have been shown to be most effective.*

Community-based care

Because disorders of the inner ear disrupt balance, safety is a primary focus of education. Assist the person to identify possible hazards in the home environment. Discuss the following points during the teaching session:

- Change positions slowly, especially when ambulating.
- Turn the whole body rather than just the head.
- Sit down immediately with the onset of vertigo and lie down if possible.
- Take prescribed anti-emetic and anti-vertigo medications.
- Wear MedicAlert® identification.
- If appropriate, discuss the surgical procedure, the immediate postoperative period and the long-term effects of the surgery.
- Discuss alternative communication techniques and associated community resources as needed.

THE PERSON WITH AN ACOUSTIC NEUROMA

An **acoustic neuroma**, or schwannoma, is a benign tumour of cranial nerve VIII. It typically occurs in adults between the ages of 40 and 50. They are common and account for about 9% of primary brain tumours. Although usually unilateral, people with neurofibromatosis type 2, a genetic disorder, frequently develop bilateral schwannomas (DeAngelis & Wen, 2012).

These tumours usually occur in the internal auditory meatus, compressing the auditory nerve where it exits the skull to the inner ear. Both the vestibular and the cochlear branches are affected; however, the tumour arises from the vestibular division of the auditory nerve twice as often. If allowed to grow, the tumour eventually destroys the labyrinth, including the cochlea and vestibular apparatus. As the tumour expands, it erodes the wall of the internal auditory meatus. The tumour may eventually impinge on the inferior cerebellar artery, which provides blood to the lateral pons and medulla, the brainstem and the cerebellum. An obstructive hydrocephalus can also occur. Cranial nerves VII (facial) and V (trigeminal) are often affected by the expanding tumour; the tumour frequently wraps around the facial nerve.

Early manifestations of an acoustic neuroma are those associated with disorders of the inner ear: tinnitus, unilateral hearing loss and nystagmus. Dizziness or vertigo may occur. As the tumour expands and occupies increasing amounts of space in the closed cranium, the person experiences neurological signs related to the area of the brain affected.

The presence of the tumour can generally be identified on CT or MRI scans. X-ray films of the petrous pyramid of the temporal bone may show erosion caused by the tumour. The treatment of choice for an acoustic neuroma is surgical excision. In surgery, every effort is made to preserve this nerve and its function as well as other cranial nerves that may be affected.

Postoperative nursing care focuses on preserving cerebral function. Position the person to minimise cerebral oedema and monitor frequently for signs of increased intracranial pressure. Because the gag reflex may be affected, assess the person carefully before food and fluids are allowed by mouth. Speech therapy is often prescribed for the person after surgery. Because deficits may not resolve for a long time after surgery, education and support are vital components of nursing care for the person. (See Chapter 41 for care of the person undergoing craniotomy.)

THE PERSON WITH HEARING LOSS

One in every six people in Australia is reported to be affected by hearing loss. In 2007–2008 the ABS National Health Survey showed that 3 million (13%) Australians had at least one long-term hearing disorder, the most common being complete or partial deafness (10%), followed by tinnitus (ringing in the ears), which affected 2% of the population (AIHW, 2012). Ear disease and/or hearing loss is exceptionally high (up to 70% in adults) in Australian Indigenous communities, especially in remote areas (Burrow et al., 2009).

Hearing loss impairs the ability to communicate in a world filled with sound and hearing individuals. A hearing deficit can be partial or total, congenital or acquired. It may affect one or both ears. In some types of hearing loss, the ability to perceive sound at specific frequencies is lost. In others, hearing is diminished across all frequencies.

People with a hearing loss often display signs that caregivers can recognise. The voice volume of the person with impaired hearing frequently increases and the person positions the head with the better ear towards the speaker. The person frequently may ask people to repeat what they have said or respond inappropriately to questions or statements. A question may elicit a blank look if the person has not heard or understood its content.

Pathophysiology and manifestations

Lesions in the outer ear, middle ear, inner ear or central auditory pathways can result in hearing loss. The process of ageing also can affect the structures of the ear and hearing. Hearing loss is classified as conductive, sensorineural or mixed, depending on which portion of the auditory system is affected. Profound deafness is often a congenital condition.

Conductive hearing loss

Anything that disrupts the transmission of sound from the external auditory meatus to the inner ear results in a conductive hearing loss. The most common cause of conductive hearing loss is obstruction of the external ear canal. Impacted cerumen, oedema of the canal lining, stenosis and neoplasms all may lead to canal obstruction. Other causes of conductive loss include a perforated tympanic membrane, disruption or fixation of the ossicles of the middle ear, fluid, scarring or tumours of the middle ear.

With conductive hearing loss, there is an equal loss of hearing at all sound frequencies. If the level of sound is greater than the threshold for hearing, speech discrimination is good. Because of this, the person with a conductive hearing loss benefits from amplification by a hearing aid.

Sensorineural hearing loss

Disorders that affect the inner ear, the auditory nerve or the auditory pathways of the brain may lead to a sensorineural hearing loss. In this type of hearing loss, sound waves are effectively transmitted to the inner ear. In the inner ear, however, lost or damaged receptor cells, changes in the cochlear apparatus or auditory nerve abnormalities decrease or distort the ability to receive and interpret stimuli.

A significant cause of sensorineural hearing deficit is damage to the hair cells of the organ of Corti. Damage may result from either loud impulse noise (e.g. an explosion) or loud continuous noise (e.g. machinery). Exposure to a high level of noise (e.g. standing close to the stage or speakers at a rock concert) on an intermittent or continuing basis damages the hair and supporting cells of the organ of Corti. Ototoxic drugs also damage the hair cells; when combined with high noise levels, the damage is greater and resultant hearing loss more profound. Ototoxic drugs include aspirin, furosemide, aminoglycosides, streptomycin, vancomycin, antimalarial drugs and chemotherapy such as cisplatin. Other potential causes of sensory hearing loss include prenatal exposure to rubella, viral infections, meningitis, trauma, Ménière's disease and ageing.

Tumours such as acoustic neuromas, vascular disorders, demyelinating or degenerative diseases, infections (bacterial meningitis, in particular) or trauma may affect the central auditory pathways and produce a neural hearing loss.

Sensorineural hearing losses typically affect the ability to hear high-frequency tones more than low-frequency tones. This loss makes speech discrimination difficult, especially in a noisy environment. Hearing aids are often not useful because they amplify both speech and background noise. The increased sound intensity may actually cause discomfort for the person.

Presbycusis

With ageing, the hair cells of the cochlea degenerate, producing a progressive sensorineural hearing loss. In **presbycusis**, gradual hearing loss associated with ageing, hearing acuity begins to decrease in early adulthood and progresses as long as the individual lives. Higher-pitched tones and conversational speech are lost initially. Hearing aids and other amplification devices are useful for most people with presbycusis.

Because the hearing loss of presbycusis is gradual, the person and family may not realise the extent of the deficit. The individual with a hearing impairment may be described as unsociable or paranoid. The family may worry that the person is becoming increasingly forgetful, absentminded or perhaps 'senile'. Depression, confusion, inattentiveness, tension and negative attitudes and/or behaviours have been noted in older adults with hearing impairments. Functional problems such as poor general health, reduced mobility and impaired interpersonal communication are also associated with hearing loss. Caregivers need to be alert for signs of impaired hearing such as cupping an ear, difficulty understanding verbal communication when the person cannot see the speaker's face, difficulty following conversation in a large group and withdrawal from social activities.

Tinnitus

Tinnitus is the perception of sound or noise in the ears without stimulus from the environment. The sound may be steady, intermittent or pulsatile and is often described as a buzzing, roaring or ringing.

Tinnitus is usually associated with hearing loss (conductive or sensorineural); however, the mechanism producing the sound is poorly understood. It is often an early symptom of noise-induced hearing damage and drug-related ototoxicity. Tinnitus is especially associated with salicylate, quinine or quinidine toxicity. Other aetiologies include obstruction of the auditory meatus, presbycusis, middle or inner ear inflammations and infections, otosclerosis and Ménière's disease. Most tinnitus, however, is chronic and has no pathological importance.

Tinnitus that is intermittent or slight enough to be masked by environmental sounds is often well tolerated. When it is loud, continuous and not responsive to treatment, tinnitus can be a significant stressor. It can interfere with activities of daily living, sleep and rest.

INTERPROFESSIONAL CARE

The best treatment for hearing loss is prevention. People need to know the risk of hearing damage and how to prevent it. Awareness of the effects of noise exposure, especially when combined with the ototoxic effects of aspirin or other drugs, is important to prevent sensorineural hearing loss.

Diagnosis

Hearing evaluation includes gross tests of hearing (such as the whisper test), the Rinne and Weber tests, and audiometry.

- *Rinne* and *Weber tests* compare air and bone sound conduction. When bone conduction of sound is better than air conduction, the hearing deficit is a conductive loss. The Rinne test can identify even mild conductive hearing losses. If both air and bone conduction are impaired, a sensorineural loss is indicated (see Chapter 44).
- *Audiometry* identifies the type and pattern of hearing loss. Specific sound frequencies are presented to each ear by either air or bone conduction.
- *Speech audiometry* identifies the intensity at which speech can be recognised and interpreted. *Speech discrimination* evaluates the ability to discriminate between various speech sounds.
- *Tympanometry* is an indirect measurement of the compliance and impedance of the middle ear to sound transmission. The external auditory meatus is subjected to neutral, positive and negative air pressure while the resultant sound energy flow is monitored.
- *Acoustic reflex testing* uses a tone presented at various intensities to evaluate movement of the structures of the middle ear.

Amplification

A hearing aid or other amplification device can help many people with hearing deficits. These assistive devices do nothing to prevent, minimise or treat the hearing loss itself. They amplify the sound presented to the hearing apparatus of the ear, which may bring the level of sound above the hearing threshold, allowing more accurate perception and interpretation of its meaning. When sound perception is distorted, a hearing aid may be less helpful because it simply amplifies the distorted sound.

Unfortunately, fewer than one-fifth of older people with a hearing deficit have and/or use a hearing aid. Denial of the deficit, other health problems, poor visual acuity, decreased manual dexterity and cost all contribute to this low usage. Hearing aids must be individually prescribed by an audiologist. Proper design, proper fit and regular maintenance are necessary for their effectiveness.

All hearing aids include a microphone, amplifier, speaker, earpiece and volume control. Most allow volume control, reduce background noise and can be adjusted for the person's pattern of hearing loss. Behind-ear and in-ear aids often include a telecoil, which amplifies sound from the telephone without feedback. The models also may allow direct audio input (e.g. MP3 player) or include Bluetooth capability for hands-free telephone use. Hearing aids are available in a variety of styles, each with advantages and disadvantages:

- Canal hearing aids (in-the-canal and completely-in-canal) are the least noticeable style, fitting in the ear canal. They are appropriate for mild to moderately severe hearing loss. These small and unobtrusive devices allow use of the telephone and can be worn during exercise. Because of their small size, the person must have good manual dexterity to insert, clean and change the batteries on canal hearing aids. For this reason, older people or people with impaired dexterity may be unable to use them.
- The in-ear style of hearing aid fits into the external ear and is used for mild to severe hearing loss (see Figure 45.16). Its larger size makes manipulation somewhat easier, although it still may be difficult for less dexterous individuals. A greater degree of amplification is possible with the in-ear aid. Many have a toggle switch for telephone usage.
- The behind-ear hearing aid allows finer adjustment of the level of amplification and is easier for the person to manipulate (see Figure 45.17). It can be used by people with mild to profound hearing loss. For the person who wears glasses, this style can be modified, with all components fitting into the temple of the eyeglasses.
- People with profound hearing loss may require a body hearing aid. The microphone and amplifier of this aid are contained in a pocket-sized case that the person clips on to clothing, slips into a pocket or carries in a harness. The receiver is attached by a cord to the case and clips on to the ear mould, which delivers the sound to the ear canal.

With both the in-canal and in-ear style, cleaning is important. Small portals may become plugged with cerumen, interfering with sound transmission.

For the person who does not have a hearing aid, an *assistive listening device* or 'pocket talker', with a microphone and ear buds, is useful. Pocket talkers are available over the counter or through an audiologist and are relatively inexpensive. The earpiece requires no special fitting and the external microphone allows the person to focus on the desired sound rather than simply amplifying all sounds. Assistive listening devices may also be used in conjunction with a hearing aid.

People with tinnitus may find a white-noise masking device helpful to promote concentration and rest. These devices conduct a pleasant sound to the affected ear, allowing the person to block out the abnormal sound.

Surgery

Reconstructive surgeries of the middle ear, such as a stapedectomy or tympanoplasty, may help restore hearing with a conductive hearing loss. Stapedectomy is the removal and replacement of the stapes. This procedure is used to treat hearing loss related to otosclerosis.

In a tympanoplasty, the structures of the middle ear are reconstructed to improve conductive hearing deficits. Chronic otitis media with necrosis and scarring of the middle ear is a common indication for this type of surgery.

For the person with a sensorineural hearing loss, a *cochlear implant* may be the only hope for restoring sound perception.

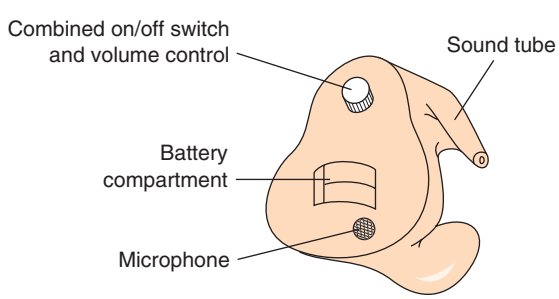


FIGURE 45.16 ■ An in-ear hearing aid

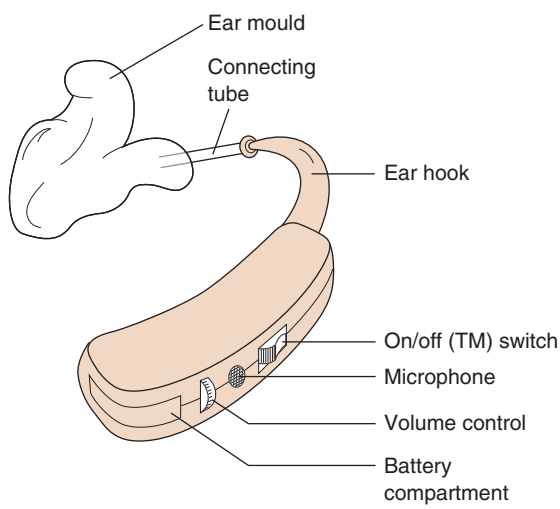


FIGURE 45.17 ■ A behind-ear hearing aid

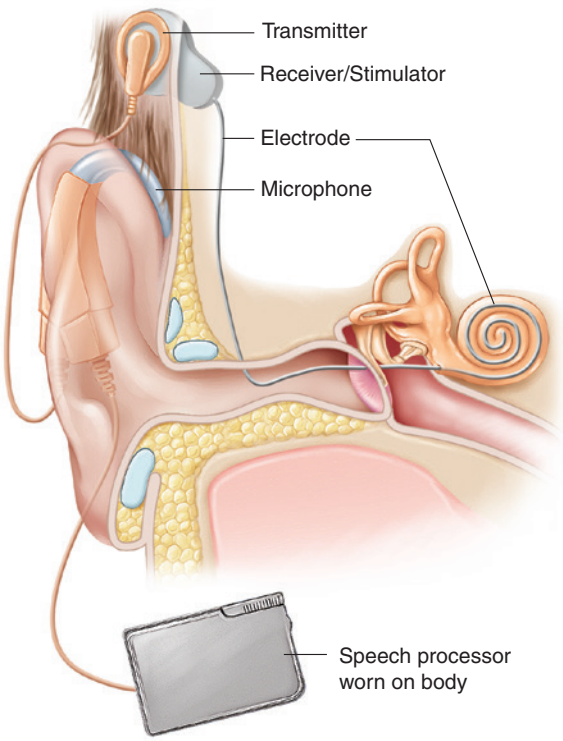


FIGURE 45.18 ■ A cochlear implant for hearing loss

The cochlear implant consists of a microphone, speech processor, transmitter and receiver/stimulator, and electrodes (see Figure 45.18). Its function is more similar to the way the ear normally receives and processes sounds than it is to a hearing aid. The microphone picks up sounds, sending them to the speech processor, which selects and processes useful sounds. The transmitter and receiver/stimulator receive signals from the speech processor, convert them to electrical impulses and send these impulses to the electrodes for transmission to the brain.

Cochlear implants provide sound perception but not normal hearing. The person is able to recognise warning sounds such as cars, sirens, telephones and doors opening or closing. They also receive stimuli to alert them to incoming communication so they can focus on the person speaking. Many people learn to interpret perceived sounds as words, especially when the hearing loss is acquired as an adult.

Nursing care

In planning and implementing nursing care for the person with a hearing deficit, the type and extent of hearing loss, the person's adaptation to the loss and the availability of assistive hearing devices are considered, as well as the person's ability and willingness to use assistive devices.

Health promotion

Healthcare personnel can be instrumental in preventing hearing loss through education. It is important to promote environmental noise control and the use of ear protection. Workplace Health and Safety legislation requires ear protection for work environments that consistently exceed 85 decibels. Education for primary prevention focuses on the following:

- care of the ears and ear canals, including cleaning and treatment of infection
- not placing hard objects into the ear canal
- use of plugs to protect the ears during swimming or diving
- avoiding intermittent or frequent exposure to loud noise
- monitoring for side effects with ototoxic medications
- hearing evaluation when hearing difficulty is present.

Assessment

- *Health history:* perceived ability to hear; effect of hearing loss on function and lifestyle; risk factors such as use of ototoxic medications; upper respiratory tract or frequent ear infection; noise exposure; presence of vertigo, tinnitus, unsteadiness or imbalance.
- *Physical examination:* apparent perception of normal speech; inspection of external ear, tympanic membrane; whisper, Rinne and Weber tests; tests of balance and cranial nerve function.

Nursing diagnoses and interventions

This section focuses on the problems of having a hearing deficit, impaired communication and social isolation for the person who is hearing impaired.

Disturbed auditory perception

Whether the person's hearing deficit is partial or total, impaired sound perception is the primary problem. The person needs to understand what causes the deficit and what to expect for the future. Nursing interventions focus on maximising available hearing and preventing further deterioration to the extent possible.

- Encourage the person to talk about the hearing loss and its effect on activities of daily living. *Hearing loss affects each individual in a different way. The person may be denying the extent of the deficit or grieving the loss. Listening and providing support encourage the person to develop coping strategies.*
- Provide information about the type of hearing loss. Refer the person to an audiologist for evaluation of the hearing loss and possible exploration of amplification devices. *With improved understanding of the deficit, the person can plan ways to compensate.*
- Replace batteries in hearing aids regularly and as needed. *Hearing aid batteries last approximately 1 week. If a battery is old or has been improperly stored, the life may be reduced further.*
- If the hearing aid has a toggle switch for microphone/telephone, be sure it is in the appropriate position. *This ensures proper amplification with the hearing aid.*

CONSIDERATION FOR PRACTICE

Check hearing aids for patency, cleaning out cerumen as necessary.

Impaired verbal communication

A hearing deficit impairs the person's ability to receive and interpret verbal communication. A hearing loss affects the person's ability to follow conversations, use the telephone and enjoy television or other forms of entertainment.

- Use the following techniques to improve communication:
  a. Wave the hand or tap the shoulder before beginning to speak.
  b. If the person wears corrective lenses, ensure that they are clean and encourage the person to wear them.
  c. When speaking, face the person and keep your hands away from your face.
  d. Keep your face in full light.
  e. Reduce the noise in the environment before speaking.
  f. Use a low voice pitch with normal loudness.
  g. Use short sentences and pause at the end of each sentence.
  h. Speak at a normal rate and do not overarticulate.
  i. Use facial expressions or gestures.
  j. Provide a magic slate for written communication.

  Individuals with hearing impairments often lip read, making good visibility of the speaker's face necessary. Excessive environmental noise interferes with the ability to perceive the message. Higher tones are typically lost with presbycusis and other types of hearing loss. Using short sentences and pausing give the person time to interpret the message. Overarticulating makes it more difficult to follow the flow and to lip read. Non-verbal cues and written messages enhance the person's understanding.
- Be sure the hearing aid is properly placed, is turned on and has fresh batteries. *The person may not be aware that the hearing aid is not functioning well.*
- Do not place intravenous catheters in the dominant hand. *The person may need to use that hand to write in order to communicate.*
- Rephrase sentences when there is difficulty understanding. *Hearing losses may affect different sound tones, making some words more difficult to comprehend. Using alternative words and phrases may increase the person's ability to perceive the message.*
- Repeat important information. *The nurse makes sure that the person understands the information.*
- Inform other staff about the person's hearing deficit and effective strategies for communication. *Consistent use of effective strategies for communication decreases the person's frustration.*

Social isolation

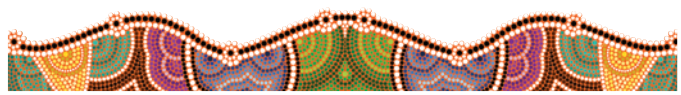
The person with impaired hearing often becomes socially isolated. This isolation may be self-imposed because of difficulty communicating, especially in a group. Often, however, the isolation comes about gradually and without intention. The person finds social settings such as family dinners or community gatherings increasingly difficult. Friends and family become frustrated trying to communicate with someone who has a hearing impairment and invitations to participate in social activities dwindle.

- Identify the extent and cause of the social isolation. Help to differentiate the reality of the isolation and its cause from the person's perception of isolation. *People with impaired hearing may be unaware that they are isolated. Identifying factors that contribute to isolation may provide the needed impetus to remedy the hearing loss. People may also experience paranoid thinking as a result of impaired communication and believe that friends and family have purposely begun to avoid interactions.*
- Encourage the person to interact with friends and family on a one-to-one basis in quiet settings. *People with impaired hearing are more successful in understanding conversations that take place in small groups and quiet settings.*
- Treat the person with dignity and remind friends and family that a hearing deficit does not indicate loss of mental faculties. *Inappropriate responses due to a hearing deficit can cause others to perceive the person as 'stupid' or demented.*
- Involve the person in activities that do not require acute hearing, such as draughts and chess. *The person has an opportunity to interact socially without the stress of straining to hear.*
- Obtain a pocket talker or encourage the person and family to do so.
- Refer the person to an audiologist for evaluation and possible hearing-aid fitting.
- Refer the person to resources such as support groups and senior citizen centres. *These groups provide new social outlets.*

Community-based care

Educating for home and community-based care for the person with hearing loss and family focuses on managing the deficit and developing coping strategies. Referral to an audiologist for evaluation of the deficit and the usefulness of a hearing aid may be appropriate. In addition, discuss the following topics as appropriate for each person:

- use, care and maintenance of a hearing aid
- strategies for coping with the hearing deficit
- voicing a preference for individual visits and small-group interactions rather than large social functions
- highlighting relevant resources available within the state and/or territory.



CHAPTER HIGHLIGHTS

- Structures of the external eye are vulnerable to trauma and infection. While usually minor, these problems can cause significant pain, scarring and clouding of the cornea, and loss or impairment of vision.
- Cataracts, glaucoma, age-related macular degeneration and diabetic retinopathy are leading causes of visual impairment in Australia. While these conditions cannot, in most cases, be prevented, they often can be treated or their progress slowed, preserving vision.
- Age, smoking, diabetes and long-term use of certain drugs are risk factors for cataract development. Removal of the clouded lens with insertion of an intraocular lens is the treatment of choice for cataracts. Surgery is elective, performed only when the cataract significantly impairs the ability to maintain ADLs and recreational activities.
- Glaucoma is progressive loss of visual fields associated with increased intraocular pressure and impaired aqueous humour drainage. Open-angle glaucoma, the predominant form of the disorder, can be controlled using medications and, as needed, laser surgery to promote aqueous humour drainage.
- Angle-closure glaucoma is a medical emergency requiring immediate treatment to lower intraocular pressure to preserve vision. Angle-closure glaucoma usually affects only one eye; however, the person is at risk of future attacks affecting the other eye.
- Age-related macular degeneration, a leading cause of blindness, cannot be effectively treated, although its progress may be slowed or halted through use of high-dose antioxidant vitamins and zinc if it is identified early. Macular degeneration affects the macula, the area of high-acuity central vision.
- Diabetic retinopathy eventually affects nearly all people with diabetes. It is a disease of the small blood vessels of the retina, leading to formation of aneurysms, retinal ischaemia and growth of fragile new vessels (neovascularisation) that easily rupture leading to haemorrhage. It is treated with laser surgery to seal fragile vessels.
- Otitis media is related to eustachian tube dysfunction, with impaired pressure equalisation of the middle ear. Otitis media may be either serous (sterile) or infectious (suppurative). Both cause acute discomfort with diminished hearing, snapping, popping and possible vertigo and systemic symptoms. The risk of complications, including rupture of the tympanic membrane, damage to structures of the middle ear and spread of infection to surrounding tissues, is greater with acute suppurative otitis media.
- Potential complications of acute otitis media include mastoiditis, chronic otitis media with tympanic membrane perforation and cholesteatoma formation. Hearing loss in the affected ear is a possibility with these disorders. The primary treatment is prevention through adequate treatment of acute otitis media.
- The primary manifestations of disorders of the inner ear are vertigo and possible hearing loss. Severe vertigo can interfere with safety, nutrition and the person's ability to maintain ADLs and life roles.
- The two main types of hearing loss are conductive and sensorineural. Presbycusis, hearing loss associated with ageing, is a type of sensorineural hearing loss. Hearing loss may be accompanied by tinnitus, the perception of sound without an environmental stimulus. Amplification devices (hearing aids) are the primary treatment for hearing loss.

CONCEPT CHECK

1 A nurse is working with a group of residents in a long-term care facility. All of the residents have moderate to severe hearing or vision impairment. Which of the following does the nurse identify as the highest priority of care?
1 preventing sensory deprivation
2 encouraging social interaction
3 promoting family relationships
4 maintaining resident safety

2 The nurse teaching a person with newly diagnosed glaucoma emphasises which of the following instructions?
1 turning the head side to side to compensate for impaired peripheral vision
2 using the prescribed eye drops as directed on a continuing basis
3 contacting the doctor if further decline in vision is noticed
4 avoiding coughing, sneezing or straining to have a bowel movement

3 A person with glaucoma has a history of heart failure. Which medication should the nurse discuss with the doctor before administering it?
1 brimonidine
2 dorzolamide
3 timolol
4 latanoprost

4 A person with Ménière's disease experiences frequent attacks of vertigo and tinnitus. Of the following teaching points, which one has the highest priority for this person?
1 Follow a low-sodium diet.
2 Stop smoking.
3 Take prescribed anti-emetic medications.
4 Sit down when an attack develops.

5 On a person's return from cataract surgery, the nurse in the ambulatory surgery recovery unit places the person:
1 in semi-Fowler's position
2 on the affected side
3 in a private room
4 in proximity to the nurses' station

6 A person calls her primary care provider's office with complaints of bright flashing lights to the side of her vision. The appropriate response by the nurse is to:
1 recommend that she lie down until the sensation has passed
2 advise her to make an appointment to have her blood pressure checked
3 initiate immediate referral to an ophthalmologist
4 reassure her that this is not unusual and should resolve without treatment

7 A person presents at the urgent care clinic with complaints of right ear pain. Which of the following should be included in physical assessment of this person? (Select all that apply.)
1 vital signs with temperature
2 inspection of the oral pharynx
3 manipulation of the ear pinna
4 palpation of cervical lymph nodes
5 inspection of the ear canal and tympanic membrane

8 An elderly resident in an assisted living facility complains to the nurse that his head 'feels stuffy' and he has ringing in his ears. The appropriate response by the nurse would be to
1 make an appointment with the resident's primary care provider

2 refer the person for evaluation by a local audiologist
3 provide non-prescription ear drops for daily use
4 inspect the ear canals for patency

9 The nurse caring for a person with a severe hearing deficit identifies which of the following as an appropriate goal towards improving the person's social interactions?
1 Will plan to have dinner with one or two friends weekly.
2 Will participate in senior centre communal lunches at least twice per week.
3 Will engage in activities such as card tournaments and dancing.
4 Will attend religious services of choice.

10 When assessing a person, the nurse notes absence of the red reflex in the person's right eye. On questioning, the person responds: 'Oh yes, my doctor told me I have cataracts. When do you think I should have them removed?' How should the nurse respond?
1 'It appears that the right eye is due for surgery.'
2 'Are you having difficulty reading or doing activities you enjoy?'
3 'Are you starting to experience pain in your right eye or frequent headaches?'
4 'Cataracts can be removed any time that it is convenient for you.'

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UNIT 12 BUILDING CLINICAL COMPETENCE

Responses to altered sensory function

CLINICAL SCENARIO

You have been assigned to work with the following four people for the 0700 shift on a medical–surgical unit. Significant data obtained during report are as follows:

- Andrew Hardy, a 50-year-old type 1 diabetic with hypertension, is a new admission during the night shift with blood glucose of 33.4 mmol/L. On assessment, the person reveals that he has been seeing black spot floaters and flashing lights at times. He is now complaining of blurred vision.
- Gladys Harvey is an 84 year old who had right eye cataract surgery yesterday. Her vital signs have been stable since surgery. She has begun complaining of itching and slight discomfort in the right eye.
- Georgia Stanley is a 45 year old admitted with complaints of feeling like she is spinning or falling, and has ringing and a fullness feeling in her left ear. Her vital signs are T 37°C, P 78, R 16, BP 102/68. She is diaphoretic and complains of nausea. She is scheduled for x-rays and a CT scan of her head at 0800.
- Kenneth Koch, a 30 year old, is a postoperative person who had a right ear tympanoplasty yesterday because of hearing loss due to chronic otitis media. Vital signs are T 37.6°C, P 90, R 20, BP 136/86. The person is requesting medication for ear pain. Pain scale is 9 out of 10 on a scale of 10 being the highest.

Critical thinking questions

1 In what order would you visit these people after handover?

1. _____
2. _____
3. _____
4. _____

2 Which top two priority assessments would you choose for each of the people presented above? Can you explain, if asked, the rationale for your choices?

	Priority Assessment #1	Priority Assessment #2
Andrew Hardy		
Gladys Harvey		
Georgia Stanley		
Kenneth Koch		

3 Which is the correct treatment for a person such as Mr Hardy who has symptoms of diabetic retinopathy?

1. scleral buckling
2. enucleation
3. photorefractive keratectomy
4. laser photocoagulation

4 After cataract surgery, Ms Harvey is placed in which position to reduce intraocular pressure?

1. Sims' position on the unaffected side
2. semi-Fowler's position on the unaffected side
3. supine with the head elevated 10 degrees
4. prone position on the affected side

5 A low-sodium diet is ordered for Ms Stanley, who has Ménière's disease. Ms Stanley understands this diet when she picks which meal plan?

1. hot dog on a roll with tomato sauce and mustard
2. ham and cheese sandwich with potato salad
3. grilled chicken sandwich with lettuce and tomato
4. hamburger on a roll with potato chips

6 Which assessment data would be most indicative of hyphema, a potential result of blunt eye trauma?

1. eye pain, decreased visual acuity, seeing a reddish tint
2. hypertension, headache, facial pain
3. white eye reflex, pressure and blindness in affected eye
4. double vision, sunken eye, limited eye movement

7 Which techniques may improve communication with the person who has a hearing impairment? (Select all that apply.)

1. Speak loudly to enhance hearing.
2. Speak at a normal rate and avoid overarticulating.
3. Stand in front of a window so person can see the face.
4. Face the person to enhance lip reading.
5. Turn down the television or radio.
6. Use short sentences and pause frequently.

8 A prescription for gentamicin is ordered for a person with conjunctivitis. The nurse understands that a serious adverse effect of aminoglycosides is which effect?

1. damage to the eighth cranial nerve resulting in hearing loss
2. vasodilation leading to migraine headaches
3. leg pain caused by thromboembolism
4. stomach irritation leading to gastric ulcers

9 When evaluating for Ménière's disease, which diagnostic studies would you expect to be ordered? (Select all that apply.)

1. caloric testing
2. Rinne and Weber tests
3. troponins
4. complete blood cell count
5. glycerol test
6. blood glucose test

10 After eye surgery, which measures does the nurse instruct the person in?

1. Avoid lifting more than 7 kg.
2. Avoid coughing or sneezing.
3. Lie on the affected side.
4. Remove the eye patch when sleeping.

11 Which is the correct way to instil ear drops in the adult person with otitis media?

1. Have person hang head over side of bed, pull pinna of the ear straight up, instil ear drops and have person turn to instil drops to other ear.
2. Have person tilt head towards unaffected side, pull pinna of the ear down and back, instil ear drops and have person remain still for 3 minutes.

3. Have person lie on unaffected side, pull the pinna of the ear up and back, instil ear drops and have person remain still for 5 minutes.
4. Have person lie in Sims' position on unaffected side, pull the pinna of the ear back, instil ear drops and have the person turn to other side after 1 minute.

12 Which is the appropriate primary prevention to teach Mr Koch to prevent hearing loss?

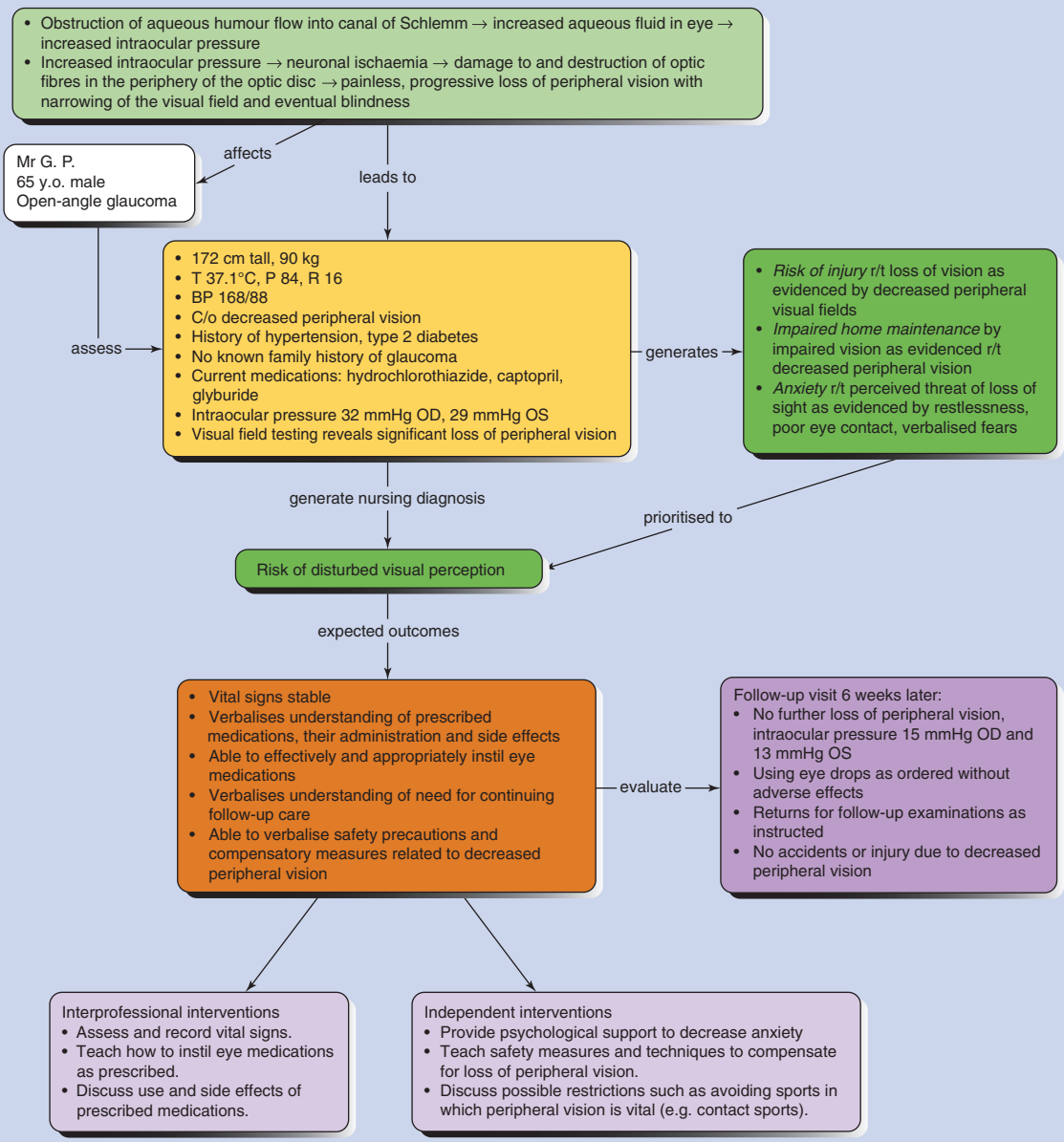
1. Cleanse the ear with ear swabs to prevent earwax build-up.
2. Avoid getting water in the ears when showering.
3. Monitor for ringing in the ears when taking paracetamol.
4. Wear ear protectors when exposed to loud noises.

CASE STUDY

George Panzarin is a 65-year-old Greek–Australian male who is seen in the eye clinic for a routine eye examination. He states he has not had any recent eye infections or eye injuries. He denies pain in the eyes. Vital signs are T 37.1°C, P 84, R 16, BP 168/88. His height is 172 cm and weight is 90 kg. His medical history indicates that he has hypertension and type 2 diabetes. He is taking hydrochlorothiazide, captopril and glyburide. He states that he is on an 8400 kilojoule diabetic diet but he is often non-compliant with the diet. He sees his medical doctor about once a year but has not had an eye exam in about 10 years. He is married with five grown children and six grandchildren. Mr Panzarin states he does not know if there is any family history of glaucoma because his father died of a heart attack at age 50 and his mother died of cancer at age 60.

The ophthalmologist performs a tonometry, which indicates an increase in intraocular pressure. A fundoscopic examination indicates pallor and increase in the size and depth of the optic cup on the optic disc. Visual field testing indicates significant peripheral vision loss. The results of these tests indicate a diagnosis of glaucoma.

Based on the medical diagnosis of open-angle glaucoma and the person's decreased peripheral vision, the nursing assessment *Risk of disturbed visual perception* is appropriate for planning care for Mr Panzarin.



UNIT 13

RESPONSES TO ALTERED REPRODUCTIVE FUNCTION

CHAPTER 46

A PERSON-CENTRED APPROACH TO ASSESSING THE MALE AND FEMALE REPRODUCTIVE SYSTEMS

MOIRA STEPHENS

LEARNING OUTCOMES

- Describe the anatomy, physiology and functions of the male and female reproductive systems, including the breasts.
- Explain the functions of the male and female sex hormones.
- Identify specific topics for consideration during a health history interview and assessment of a person with health problems involving the reproductive system and breast structures and/or functions.
- Describe normal variations in assessment findings for the older adult.
- Identify manifestations of impairment in the male and female reproductive system and breast structure or function.

CLINICAL COMPETENCIES

- Conduct and document a health history for men and women having or at risk of alterations to the reproductive system, including the breasts.
- Conduct and document a physical assessment of male and female reproductive system structures and functions, including the breasts.
- Monitor and interpret the findings of diagnostic tests and report abnormal findings.

EQUIPMENT NEEDED

- Disposable gloves
- Water-soluble lubricant
- A good light source
- Sterile cotton swabs (for culture)
- Culture media (for culture)
- A spatula, cotton swab or endocervical brush, slides and cytological fixative (for Pap smear)
- Vaginal speculum of appropriate size

KEY TERMS

Although the reproductive organs in men and women are very different, they share common functions: enabling sexual pleasure and reproduction. The reproductive organs, in conjunction with the neuroendocrine system, produce hormones important in biological development and sexual behaviour. Parts of the reproductive organs also enclose and are integral to the function of the urinary system. Assessment of the reproductive and urinary systems is often difficult for both the nurse and the individual, and requires sensitivity on the part of the nurse when asking questions about topics that the person may be hesitant to talk about. Skill and sensitivity in conducting physical examinations of an area of the body usually considered private is also required.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE MALE REPRODUCTIVE SYSTEM, INCLUDING THE BREASTS

The male reproductive system comprises the paired testes, scrotum, ducts, glands and penis (see Figure 46.1). The breasts are part of the male reproductive system and are also assessed. The location and functions of the male reproductive organs are summarised in Table 46.1.

The breasts

The male breast is comprised primarily of an areola (circular pigmented area) and a small nipple. These lie over a thin disk of undeveloped breast tissue that may not be overtly different from surrounding tissue. Approximately one in three men has a firm area of breast tissue 2 cm or larger; the limits of the normal size of this area have not been established (Bickley, 2012).

The penis

The penis is the genital organ that encloses the urethra (see Figure 46.1). It is homologous to the clitoris of the female. The penis is composed of a shaft and a tip called the glans, which is covered in the uncircumcised man by the foreskin (or prepuce). The shaft contains three columns of erectile tissue: the two lateral columns are called the corpora cavernosa and the central mass is called the corpus spongiosum.

Erection occurs when the penile masses become filled with blood in response to a reflex that triggers the parasympathetic nervous system to stimulate arteriolar vasodilation. The erection reflex may be initiated by touch, pressure, sights, sounds, smells or thoughts of a sexual encounter. After ejaculation, the arterioles vasoconstrict and the penis returns to a flaccid state.

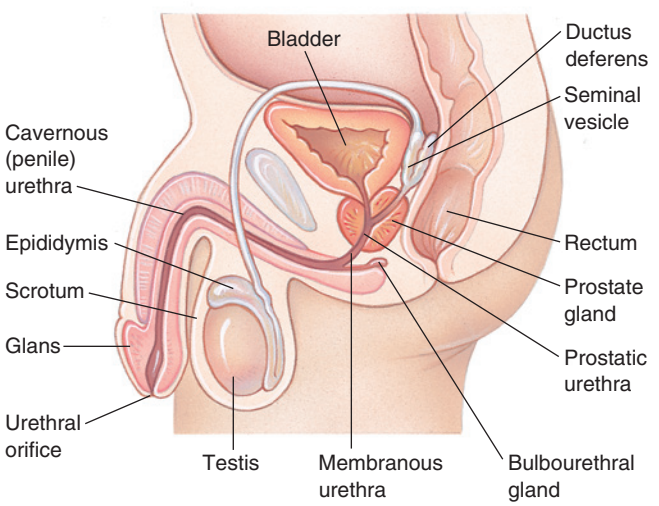


FIGURE 46.1 ■ The male reproductive system

The scrotum

The scrotum is a sac or pouch made of two layers. The outer layer is continuous with the skin of the perineum and thighs. The inner layer is made of muscle and fascia. The scrotum hangs at the base of the penis, anterior to the anus, and regulates the temperature of the testes. The optimum temperature for sperm production is about 2 to 3°C below body temperature. When the testicular temperature is too low, the scrotum contracts to bring the testes up against the body. When the testicular temperature is too high, the scrotum relaxes to allow the testes to lie further away from the body.

The testes

The testes develop in the abdominal cavity of the foetus and then descend through the inguinal canal into the scrotum. Approximately 30–45% of premature and 1–4.5% of full-term infant boys are born with at least one undescended testicle (Ashley, Barthold & Kolon, 2010), a condition that usually corrects itself within 1 year. This is cryptorchidism (the absence of one or both testes from the scrotum) and is distinct from monorchism (the condition of having one testicle). The testes are homologous to the female's ovaries. These paired organs are each about 4 cm long and 2.5 cm in diameter. They are suspended in the scrotum by the spermatic cord. Each is surrounded by two coverings: an outer tunica vaginalis and an inner tunica albuginea. Each testis is divided into 250 to 300 lobules, with each lobule containing one to four seminiferous tubules. The testes produce sperm and testosterone.

The seminiferous tubules are responsible for sperm production. Leydig's cells (or interstitial cells) lie in the connective tissue surrounding the seminiferous tubules and produce testosterone.

The ducts and semen

The seminiferous tubules lead into the efferent ducts and become the rete testis. From the rete testis, 10 000 to 20 000 efferent ducts join the epididymis, a long coiled tube that lies over the outer surface of each testis. The epididymis is the final

TABLE 46.1 Location and function of the male reproductive organs

MALE REPRODUCTIVE ORGAN	LOCATION	FUNCTION
Penis	Attached to front and sides of the pubic arch. Proximal, ventral surface is directly continuous with the scrotum.	Excretes semen and urine. Deposits sperm.
Scrotum	Hangs from body at root of penis.	Contains testes, epididymis and portions of the vas (ductus) deferens.
Testes	In the scrotal sac.	Produce sperm and testosterone.
Epididymis	Posterolateral to upper aspect of each testis.	Stores sperm. Promotes sperm maturation. Transports sperm to vas deferens.
Vas deferens (ductus deferens)	Between the epididymis and the seminal vesicle forming the ejaculatory duct.	Stores sperm. Transports sperm.
Urethra	Begins at bladder and passes through prostate and penis.	Serves as passageway for urine or semen.
Prostate gland	Encircles the urethra at the neck of the bladder.	Contributes to ejaculatory volume. Enhances sperm motility and fertility.
Seminal vesicles	Lie on posterior bladder wall.	Contribute to ejaculatory volume. Contain nutrients to sustain sperm and prostaglandins to facilitate sperm motility.
Bulbourethral (Cowper's) glands	Inferior to the prostate.	Secrete mucus into urethra. Neutralise traces of acidic urine in urethra.

area for the storage and maturation of sperm. When a man is sexually excited, the epididymis contracts to propel the sperm through the vas deferens to the ampulla, where the sperm are stored until ejaculation.

The seminal vesicles at the base of the bladder produce about 60% of the volume of seminal fluid. Seminal fluid is also made of secretions from the accessory sex organs, the epididymis, the prostate gland and Cowper's glands. Seminal fluid nourishes the sperm, provides bulk and increases its alkalinity. (An alkaline pH is essential to mobilise the sperm and ensure fertilisation of the ova.) Sperm mixed with this fluid is called **semen**. Each seminal vesicle joins its corresponding vas deferens to form an ejaculatory duct, which enters the prostatic urethra. During ejaculation, seminal fluid mixes with sperm at the ejaculatory duct and enters the urethra for expulsion.

The total amount of semen ejaculated is 2 to 4 mL, although the amount varies. The total ejaculate of a healthy male contains from 100 to 400 million sperm.

The prostate gland

The prostate gland is about the size of a walnut. It encircles the urethra just below the urinary bladder (see Figure 46.1). It is made of 20 to 30 tubuloalveolar glands surrounded by smooth muscle. Secretions of the prostate gland make up about one-third of the volume of the semen. These secretions enter the urethra through several ducts during ejaculation.

Spermatogenesis

Spermatogenesis is the series of physiological events that generate sperm in the seminiferous tubules. This process begins with puberty and continues throughout a man's life, with several hundred million sperm produced each day.

The inner layer of the seminiferous tubules consists of sustentacular cells (or Sertoli's cells), which contain the spermatocytes and sperm in different stages of development. Sertoli's cells secrete a nourishing fluid for the developing sperm, as well as enzymes that help convert spermatocytes to sperm. The events in spermatogenesis, which take 64 to 72 days, are as follows:

1. The spermatogonia (sperm stem cells) undergo rapid mitotic division. As these cells multiply, the more mature spermatogonia divide into two daughter cells. These daughter cells grow and become the primary spermatocytes (and eventually become sperm).
2. Primary spermatocytes divide by meiosis to form two smaller secondary spermatocytes, which in turn divide to form two spermatids. This process occurs over several weeks.
3. The spermatids elongate into a mature sperm cell with a head and a tail. The head contains enzymes essential to the penetration and fertilisation of the ova. The flagellar motion of the tail allows the sperm to move. The sperm cells then move to the epididymis to mature further and develop motility.

Functions of the male sex hormones

The male sex hormones are called **androgens**. Most androgens are produced in the testes, although the adrenal cortex also produces a small amount. **Testosterone**, the primary androgen produced by the testes, is essential for the development and maintenance of sexual organs and secondary sex characteristics

and for spermatogenesis. It also promotes metabolism, growth of muscles and bone, and libido (sexual desire).

ASSESSING THE MALE REPRODUCTIVE SYSTEM

The structures and functions of the male reproductive system are assessed by findings from a comprehensive assessment, which includes both a health assessment interview to collect subjective data and a physical assessment to collect objective data. In addition, a number of diagnostic tests may provide further information.

Health assessment interview

A health assessment interview to determine health issues with the male reproductive system may be conducted for different reasons; for example:

- during a health screening
- as part of a comprehensive health assessment
- during a focused assessment for a specific health problem (such as a discharge from the penis).

Men may be embarrassed to discuss health problems or concerns involving their reproductive organs so it is important for the nurse to ask questions in a non-judgmental, non-threatening and matter-of-fact manner. Consider the psychological, social and cultural factors that affect sexuality and sexual activity. Use words that the man can understand and do not be embarrassed or offended by the words he uses. The man may perceive the interview as less threatening if the discussion begins with more general questions and then progresses to specific questions. Ask questions in a way that gives the man permission to describe behaviours and manifestations. For example, rather than asking a man if he has difficulty achieving or maintaining an erection, ask him to describe any changes he has noticed in his erections.

If the man has identified a health problem, the nurse needs to analyse its onset, characteristics and course, severity, precipitating and relieving factors, and any associated symptoms, noting the timing and circumstances. For example, you may ask the man:

- When did you first notice that you were having difficulty urinating?
- Did you use a different brand of condoms before you noticed the rash on your penis?
- Describe the changes that occurred in your ability to have an erection after you started taking medicine for high blood pressure.

In questioning the man about past medical history, ask about chronic illnesses such as diabetes, chronic kidney failure, cardiovascular disease, multiple sclerosis, spinal cord tumours or trauma, or thyroid disease. The effects of these illnesses, as well as the treatment of the illnesses, may cause impotence (inability to achieve or maintain an erection). The following drugs may cause sexual function problems: antihypertensives, antidepressants, antispasmodics, tranquillisers, sedatives, antipsychotics and histamine$_2$-receptor antagonists. Psychosocial stressors also may contribute to impotence.

If the man was born to a woman treated during pregnancy with diethylstilbestrol (DES), a drug used in the 1940s and 1950s to prevent miscarriage, he may have congenital deformities of the urinary tract as well as decreased semen levels. If the man had mumps as a child, sterility is possible. Testicular cancer is a rare cancer with 76% of diagnoses occurring in men between 20 and 44 years of age. In Australia in 2011, there were 732 new cases diagnosed and 15 testicular-cancer-related deaths (Australian Institute of Health and Welfare (AIHW), 2015). There are two kinds of testicular cancer: testicular teratoma or non-seminoma, and seminoma. Non-seminoma is a cancer of the mature germ cells, mostly affecting men between the ages of 15 and 35, whereas seminoma develops in the immature germ cells, primarily affecting slightly older men between the ages of 25 and 55. Both kinds of testicular cancer can be treated successfully (cured), especially if detected early. The risk of testicular cancer is greatest in men who have a history of an undescended testicle, an inguinal hernia, testicular swelling with mumps, a history of maternal use of DES or oral contraceptives, and a family history of testicular cancer.

Explore the lifestyle and social history of the man; the use of alcohol, cigarettes or illicit drugs may affect sexual function. Unprotected sexual intercourse increases the potential for sexually transmitted infections, including hepatitis B and HIV infection. Ask about sexual preference. Unprotected sexual intercourse with same-sex partners further increases the risk of hepatitis B and HIV infection. Other questions about sexual activity may include number of sexual partners, history of premature ejaculation, impotence, any history of sexual trauma, use of condoms or other contraceptives, and current level of sexual satisfaction.

Interview questions categorised by functional health patterns are listed in the 'Functional health pattern interview' table below.

Physical assessment

Physical assessment of the male reproductive system may be performed as part of a comprehensive assessment or separately for men with identified sexual health problems. If conducted as part of a total physical assessment, this is usually the final

SAMPLE DOCUMENTATION

Assessment of the male reproductive system

12/10/2015 NURS 1000 hrs	23-year-old male, first visit to health department for problems he described as 'an unusual lump in his testicle'. He states that his girlfriend noticed a lump in his left testicle 2 weeks ago, which is still there. He also states that it is not painful. Palpation of his testicles confirms a 1 cm nodule in his left testicle. Palpation of inguinal lymph nodes revealed no enlarged nodes. Referred for testicular ultrasound ———— RN P.Tone

P.TONE RN

FUNCTIONAL HEALTH PATTERN INTERVIEW The male reproductive system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Have you ever had problems with your reproductive organs (penis, testicles, prostate gland)? Explain. If so, how was the problem treated? ■ Have you ever had surgery on your reproductive organs? If so, what type, when, and what was the outcome? ■ Have you ever noticed any pain or swelling in your breasts? Explain. ■ Do you practise testicular self-examination? How often? ■ Have you ever noticed any pain or swelling in your testicles? Explain. ■ Do you smoke? If so, how much and for how long?
Nutritional–Metabolic	■ Describe your usual intake of food and fluids in a 24-hour period.
Elimination	■ Do you now or have you ever had a discharge from your penis? If so, describe the colour, odour, consistency, amount and frequency. ■ Have you ever had any bleeding from your penis? Explain. ■ Have you noticed any change in your urination, such as burning, frequency, urgency, difficulty starting the stream, size of the stream, dribbling or getting up frequently at night? Explain.
Activity–Exercise	■ Describe your usual activity in a 24-hour period. ■ Do you participate in sports or heavy lifting? If so, do you wear a protective cup or athletic support?
Sleep–Rest	■ Describe the quality of your rest and sleep.
Cognitive–Perceptual	■ Describe any pain you have had in the groin area, testicles, penis or scrotum. Where is it? Do you experience it in other parts of your body? How long does it last? What makes it worse or relieves it? ■ Has there been a change in the condition or colour of the skin on your scrotum or penis? Explain.
Self-perception–Self-concept	■ Has this problem affected how you feel about yourself? ■ Do you feel that your needs for intimacy and affection are being met?
Role–Relationships	■ Has having this condition affected your relationships with others? ■ Has having this condition interfered with your ability to work? Explain. ■ Has anyone in your family had problems with prostate cancer? Explain.
Sexuality–Reproductive	■ Are you currently in a sexual relationship? If so, has this condition interfered with your usual sexual activity? ■ How long have you been with your current partner? Have you had any other partners during this time? ■ What is your sexual preference? ■ Has having this problem affected your relationship with your spouse or sexual partner? ■ Are you satisfied with your current level of sexual functioning? ■ Have you ever had any problem with achieving or maintaining an erection or ejaculation? ■ Do you use any medications to facilitate your sexual ability? Describe. ■ Do you use condoms every time you have sexual contact?
Coping–Stress–Tolerance	■ Has having this condition created stress for you? If so, does your health problem seem to be more difficult when you are stressed? ■ Have you experienced any kind of stress that makes the condition worse? Explain. ■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments that you would not use to treat this problem?

TABLE 46.2 Age-related changes in the male reproductive system

AGE-RELATED CHANGE	SIGNIFICANCE
Prostate gland	
• A significant number of older men have some degree of benign prostatic hyperplasia.	Although ageing does not cause prostate cancer, its incidence does increase with age. The following 2011 data describe incidence per 100 000 men (AIHW, 2015): 55–59 years of age—377 60–64 years of age—608 65–69 years of age—895 70–74 years of age—883
Penis, testes and scrotum	
• Epithelial tissue and mucosa of seminal vesicles are thinner and have reduced capacity to hold fluid. • Sclerosis of penile arteries and veins may occur.	Although men may father children throughout life, the sperm count is reduced in some men. Changes in the vascular system of the penis may mean the ageing man takes longer to achieve an erection and ejaculation or may be impotent.

'system' to be assessed. Problems of the male reproductive system may involve the urinary system, making an assessment of both systems important. (See Chapter 25 for assessment of the urinary system). The nurse must feel comfortable with the examination of people of the opposite gender; if either the nurse or the individual is not comfortable, a nurse of the same gender should be asked to conduct this part of the assessment. Normal age-related findings for the older man are summarised in Table 46.2.

The male reproductive system is assessed by inspection and palpation. Explain the procedures for the examination thoroughly and in a matter-of-fact way to decrease anxiety and embarrassment. If the man is unfamiliar with his internal genitalia, charts may be used to illustrate the parts that will be examined. Ask the man to empty his bladder (to be more comfortable during the examination), remove his clothing and put on a gown. The assessment may be done with the man sitting or standing. Expose only those body parts being examined to preserve modesty. Ensure that the examining room is warm and private. Put on gloves before beginning and wear them throughout the examination.

Diagnostic tests

The results of diagnostic tests of the structures and functions of the male reproductive system are used to support the diagnosis of a specific sexual problem, injury or disease; to provide information to identify or modify the appropriate medications or treatments used to treat the condition; and to help nurses monitor the man's responses to treatment and nursing care interventions. Diagnostic tests used to assess the male reproductive system are described in the 'Diagnostic tests' table below and summarised in the bulleted list that follows. More information is included in the discussion of specific health problems or diseases in Chapter 47.

- Hormone changes and syphilis are diagnosed with blood tests, discussed in Chapters 47 and 49. Gonorrhoea, as well as other sexually transmitted infections, is diagnosed by cultures and smears of discharge or mucous membranes.
- Prostate cancer may be diagnosed following a raised PSA reading. Relapse is monitored by measuring prostate specific antigen (PSA). PSA is a glycoprotein secreted by

DIAGNOSTIC TESTS The male reproductive system

NAME OF TEST Prostate specific antigen (PSA)

PURPOSE AND DESCRIPTION The PSA level is raised in prostate carcinoma, benign prostatic hypertrophy and following prostate examination. PSA is used to monitor recurrence of prostate cancer. PSA as a screening test is unproven and the predictive value of a raised PSA in healthy men is low.

Normal value: There is no specific normal level but most doctors consider below 4 ng/mL as normal and would recommend a biopsy of the prostate if the result was greater than 0.4 ng/mL

RELATED NURSING CARE No special physical preparation is needed but psychological care is always a consideration, particularly because of the potential implications of a raised PSA result.

NAME OF TEST Prostate ultrasound

PURPOSE AND DESCRIPTION Conducted to identify testicular torsion or masses and to evaluate prostate enlargement. Uses high-frequency sound waves, passed through tissues of various densities, to produce a visual graphic of tissue being examined.

RELATED NURSING CARE A full bladder may be required for the study. Note that if the man has frequency, urgency of micturition or urinary incontinence he may be most anxious about this requirement.

DIAGNOSTIC TESTS The male reproductive system (continued)

NAME OF TEST Prostate biopsy
- Transrectal biopsy
- Transurethral biopsy

PURPOSE AND DESCRIPTION Conducted to diagnose prostate cancer—35–45% of men undergoing the procedure are diagnosed with prostate cancer and approximately 5% of people suffer complications which are commonly associated with post-procedure infection. A transrectal ultrasound (TRUS) is often used to guide the placement of the needle during the procedure.

- A transrectal biopsy is performed with a spring-loaded needle, inserted through the rectal wall and into the prostate gland to remove one or more tissue samples.
- A transurethral biopsy is performed by inserting a cystoscope through the urethra and using a cutting loop to remove small samples of prostate tissue.

RELATED NURSING CARE Prior to the procedure, the man should be advised to stop taking any medications with anticoagulant properties—such as warfarin, heparin, aspirin, Cartia, for example—and antibiotics may be prescribed to commence up to 3 days prior to the procedure. The procedure takes 15 to 20 minutes and may be performed either under a local anaesthetic or with sedation. Advise the man to avoid strenuous activity for 4 hours post procedure and to avoid heavy lifting and sexual activity for the following 24 hours. Explain that there may be some discomfort in the biopsy area for 1 to 2 days, there may be some blood in the urine or from the rectum, and semen may appear dark. Following a transurethral biopsy, a urinary catheter may remain in place for a few hours after the procedure and antibiotics will be prescribed. Excess bleeding, pain or signs of infection should be reported.

NAME OF TEST Gonorrhoea culture

PURPOSE AND DESCRIPTION A culture is performed to evaluate for gonorrhoea. A swab is used to collect a sample of discharge from the infected area (urethra, penis, anus or throat), smeared on a slide and a Gram stain is conducted to identify the organism (*Neisseria gonorrhoeae*). A urine sample is used in some tests.

RELATED NURSING CARE No special physical preparation is needed but psychological care is required. If the test is positive, request the names of all sexual partners and emphasise the need for treatment to eradicate the infection.

NAME OF TEST Venereal disease research laboratory (VDRL); Rapid plasma reagin (RPR); Fluorescent treponemal antibody absorption (FTA-ABS)

PURPOSE AND DESCRIPTION These blood tests are conducted to screen for syphilis. Positive findings can be made within 1 to 2 weeks after the primary lesion appears or 1 to 4 months after the initial infection. The FTA-ABS test is used to detect antibodies to the syphilis-causing bacteria *Treponema pallidum.* It is considered the most accurate and is often used if findings from the VDRL or RPR are questionable, but it remains positive after treatment and so cannot be used to monitor treatment efficacy.

RELATED NURSING CARE No special physical preparation is needed but psychological care is required. If the test is positive, request the names of all sexual partners and emphasise the need for treatment to eradicate the infection.

NAME OF TEST Semen analysis

PURPOSE AND DESCRIPTION Evaluates volume; liquefaction time; sperm count, morphology and motility; pH; white blood cell count and fructose level. Antisperm antibody testing may also be performed.

Normal values:
Volume: > 2 mL
pH: 7.2–7.8
Sperm count (concentration): > 20 x 10^6/mL
Motility: > 50% motile in a forwards direction and/or > 25% with rapid forwards motility
% normal sperm: > 30% with 'normal' morphology
WBC: < 1 x 10^6/mL
Sperm antibodies: less than 50% antibodies detected (World Health Organization, 2010)

RELATED NURSING CARE The man is asked to bring in a fresh specimen of semen within 1 hour of ejaculation and following 4–5 days of abstinence.

the cells of the prostatic ductal epithelium and is present in all men and increases with age over 50.
- The prostate may be examined by ultrasound to identify testicular torsion or masses, by digital rectal examination and by a prostate biopsy to accurately diagnose cancer or benign hypertrophy of the prostate.
- Semen analysis is done to evaluate semen volume, sperm count and motility, and percentage of abnormal sperm.

Regardless of the type of diagnostic test, and after the person gives informed consent, the nurse is responsible for fully explaining the procedure and any special preparation needed, assessing for any medication use that might affect the outcome of the tests, supporting the man during the examination as necessary, documenting the procedures as appropriate and monitoring the results of the tests.

MALE REPRODUCTIVE SYSTEM ASSESSMENTS

Technique/normal findings	Abnormal findings
Breast and lymph nodes	
Inspect and palpate both breasts, including areola and nipple. *Breast tissue should not be swollen, tender or enlarged (although soft, fatty and enlarged breast tissue does occur with obesity in men).*	■ A smooth, firm, mobile, tender disc of breast tissue behind the areola indicates gynaecomastia, overdevelopment of breast tissue in men. Gynaecomastia requires additional investigation to determine the cause but is generally caused by an alteration in the testosterone/oestrogen ratio precipitated by high levels of sex-hormone-binding globulin (SHBG). There are a number of health conditions and also medications which may cause gynaecomastia. ■ A hard, irregular nodule in the nipple area suggests cancer.
Palpate the axillary and supraclavicular lymph nodes. *Lymph nodes should not be palpable.*	■ Enlarged axillary nodes are common with infections of the hand or arm but may be caused by cancer. ■ Enlarged supraclavicular nodes may indicate breast cancer or lymphoma.
External genitalia Inspect and palpate the inguinal and femoral area for bulges. Ask the man to bear down or cough as you palpate (see Figure 46.2). *There should be no bulging with coughing or bearing down.*	■ A bulge that increases with coughing or straining suggests a hernia.
Inspect the penis. If the man is uncircumcised, retract the foreskin, or ask him to do so. When non-erect, the penis is normally soft, flaccid and non-tender. *The foreskin should be without lesions, of colour equal with the penis and should retract easily. The glans is normally free of lesions.*	■ Priapism is the presence of a prolonged, painful erection of the penis that is not related to sexual stimulation. It is a urological emergency as it may lead to necrosis and/or erectile dysfunction if untreated. It may be idiopathic in origin or associated with health conditions such as leukaemia, sickle cell disease, pelvic tumours, spinal cord injury or some medications. ■ Phimosis (tightness of prepuce that prevents retraction of foreskin) may be congenital or due to recurrent balanoposthitis (generalised infection of glans penis and prepuce). ■ Narrow or inflamed foreskin can cause paraphimosis, retraction of the foreskin that causes painful swelling of the glans. ■ Balanitis (inflammation of the glans) is associated with bacterial or fungal infections. ■ Ulcers, vesicles or warts suggest a sexually transmitted infection. ■ Nodules or sores seen in uncircumcised men may be cancer.

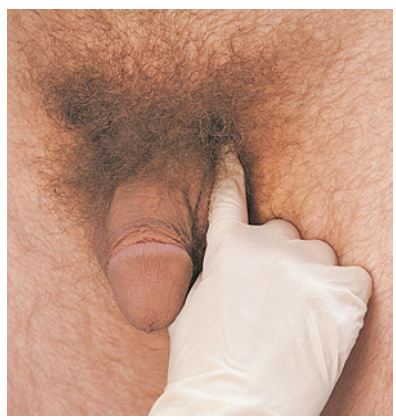


FIGURE 46.2 ■ Palpating the male inguinal area for bulges

Technique/normal findings	Abnormal findings
Inspect the external urinary meatus. Press the glans between the thumb and forefinger (see Figure 46.3). Replace the foreskin if appropriate. *The external urinary meatus is normally in the centre of the glans, without redness or discharge.*	■ Erythema or discharge indicates inflammatory disease or infection. Further assessment is required.

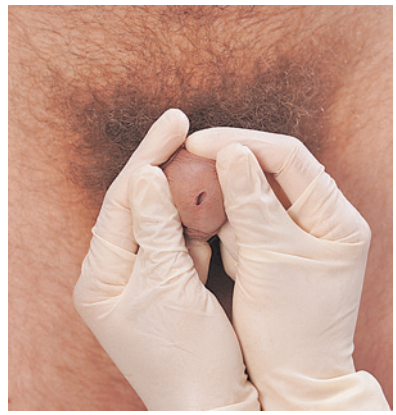


FIGURE 46.3 ■ Inspecting the external urinary meatus of the male

Inspect the skin on the shaft of the penis. *The skin on the shaft of the penis should be free of redness or lesions.*	■ Excoriation or inflammation suggests lice or scabies.
Palpate the shaft of the penis. *The shaft of the penis should not be tender.*	■ Induration with tenderness along the ventral surface suggests urethritis or urethral stricture with inflammation.
Inspect the scrotum. Further assess any swelling in the scrotum using transillumination: darken the room and place a lighted flashlight against the skin of the scrotum. *The normal scrotum and epididymis appear as dark masses with regular borders.*	■ A unilateral or bilateral poorly developed scrotum suggests cryptorchidism (failure of one or both testes to descend into the scrotum). ■ Swelling of the scrotum may indicate indirect inguinal hernia, hydrocoele (accumulation of fluid in the scrotum) or scrotal oedema. Swellings containing serous fluid will transilluminate. Swellings containing blood or tissue will not transilluminate.
Palpate each testis and epididymis. *The testes should not be tender or swollen.*	■ Tender, painful scrotal swelling occurs in acute epididymitis, acute orchitis, torsion of the spermatic cord and strangulated hernia. ■ A painless nodule in the testis is associated with testicular cancer.
Prostate The prostate gland is assessed by digital rectal examination (DRE). (See Chapter 20, Figure 20.20 for the technique used to palpate the prostate through the rectal wall.)	
With a gloved index finger, palpate the posterior rectal wall for the rounded, two-lobed structure of the posterior prostate. *The prostate is normally non-tender, with two lateral lobes that are divided, smooth and about 2.5 cm long.*	■ Enlargement (1 cm protrusion into the rectum) with obliteration of the median sulcus suggests benign prostatic hypertrophy. ■ Enlargement with asymmetry and tenderness suggests prostatitis. ■ A hard irregular nodule is suspicious of carcinoma.

GENETIC CONSIDERATIONS Male reproductive system disorders

- Although the exact genetic predisposition for some men to have prostate cancer is unknown, the findings of a number of studies have suggested that a family history may increase the relative risk. Risk appears to be two to three times greater for men with an affected brother, rather than an affected father, or who are diagnosed before the age of 65 and have a first-degree relative with the disease (Kiciński, Vangronsveld & Nawrot, 2011).
- A family history of testicular cancer is a risk factor for cancer of the testes.
- Men who have XX chromosomes (instead of XY) often have altered testicular development because they are missing a gene called the sex-determining region Y gene (SRY), which is responsible for the development of secondary sex characteristics in men.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of an adult. Several diseases of the male reproductive system have a genetic component. During the health assessment interview, it is especially important to ask about a family history of testicular or prostate cancer. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the box above). If data are found to indicate genetic risk factors or alterations, ask about genetic testing and refer for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

ANATOMY, PHYSIOLOGY AND FUNCTIONS OF THE FEMALE REPRODUCTIVE SYSTEM, INCLUDING THE BREASTS

The female reproductive system consists of the external genitalia (mons pubis, labia, clitoris, vaginal and urethral openings and glands) and the internal organs (vagina, cervix, uterus, fallopian tubes and ovaries). The breasts are a part of women's reproductive organs. In women, the urethra and urinary meatus are separated from the reproductive organs; however, they are so close to each other that a health problem with one often affects the other. The location and function of the female reproductive organs are summarised in Table 46.3.

TABLE 46.3 Location and function of the female reproductive organs

FEMALE REPRODUCTIVE ORGAN	LOCATION	FUNCTION
Mons pubis (mons veneris)	Anterior and superior to the pubis.	Enhances sexual sensations. Protects and cushions pubic symphysis during intercourse.
Labia majora	Extend from mons pubis to perineum.	Protect labia minora, urethral and vaginal openings. Enhance sexual arousal.
Labia minora	Enclosed by the labia majora.	Protect clitoris. Inferiorly, merge to form posterior ring of vaginal introitus (fourchette). Lubricate vulva. Enhance sexual arousal.
Vestibule	Area enclosed by labia minora.	Contains openings for urethra, vagina, Bartholin's glands and Skene's glands.
Bartholin's (greater vestibular) glands	Posterior on each side of the vaginal orifice. Open onto the sides of the vestibule in the groove between the labia minora and hymen.	Secrete clear, viscid mucus during intercourse.
Skene's (lesser vestibular, paraurethral) glands	Open onto the vestibule on each side of the urethra.	Drain urethral glands. Produce lubricating mucus.
Clitoris	Small bud of erectile tissue just below the superior joining of the labia minora.	Stimulates and elevates levels of sexual arousal.
Perineum	Skin-covered muscular area between vaginal opening and anus.	Provides support for pelvic organs.
Mammary glands	Contained within breasts. Anterior to pectoral muscles of thorax.	Produce human milk. Play a role in sexual arousal.
Ovaries	Lie on each side of the uterus below and behind the uterine tubes.	Produce and secrete ova. Produce the hormones oestrogen and progesterone.
Fallopian tubes (uterine tubes, oviducts)	One tube extends medially from the area of each ovary and empties into the upper portion (fundus) of the uterus.	Transport ova.
Uterus (adnexa of the uterus are composed of the uterine tubes and ovaries)	Anterior to the rectum and posterior/superior to the bladder.	Receives, retains and nourishes the fertilised ovum. Contracts rhythmically to expel infant. Cyclically sheds lining when ovum is not fertilised.
Cervix	Lower portion of uterus extending into the vagina.	Connects uterine cavity with vagina. Opens to allow passage of menstrual flow and infant.
Vagina	Extends from the external orifice in the vestibule to the cervix.	Receives penis and semen during intercourse. Passageway for menstrual flow and expulsion of infant at birth.

The breasts

The breasts (or mammary glands) are located between the third and seventh ribs on the anterior chest wall. They are supported by the pectoral muscles and are richly supplied with nerves, blood and lymph (see Figure 46.4). A pigmented area called the areola is located slightly below the centre of each breast and contains sebaceous glands and a nipple. The nipple is usually protrusive and becomes erect in response to cold and stimulation.

The breasts are made of adipose tissue, fibrous connective tissue and glandular tissue. Cooper's ligaments support the breast and extend from the outer breast tissue to the nipple, dividing the breast into 15 to 25 lobes. Each lobe is made of alveolar glands connected by ducts that open to the nipple.

The external genitalia

The external genitalia collectively are called the vulva. They include the mons pubis, the labia, the clitoris, the vaginal and urethral openings, and glands (see Figure 46.5).

The mons pubis is a pad of adipose (fat) tissue covered with skin. It lies anterior to the symphysis pubis. After puberty, the mons is covered with hair.

The labia are divided into two structures. The *labia majora* are folds of skin and adipose tissue covered with hair following puberty. The labia majora are outermost and begin at the base of the mons pubis and end at the anus. The *labia minora*, located between the clitoris and the base of the vagina, are enclosed by the labia majora. They are made of skin, adipose tissue and some erectile tissues. They are usually light pink and hairless.

The area between the labia is called the vestibule and contains the openings for the vagina and the urethra as well as the Bartholin's glands. Skene's glands open on to the vestibule on each side of the urethra. Bartholin's and Skene's glands secrete lubricating fluid during the sexual response cycle prior to menopause.

The clitoris is an erectile organ analogous to the penis in the male. It is formed by the joining of the labia minora. Like the penis, it is highly sensitive and distends during sexual arousal.

The vaginal opening, called the introitus, is the opening between the internal and the external genitals. Prior to rupture from intercourse or trauma, the introitus is surrounded by a connective tissue membrane called the hymen.

The internal organs

The vagina, cervix, uterus, fallopian tubes and ovaries are the internal organs of the female reproductive system (see Figure 46.6). The ovaries are the primary reproductive organs in women and also produce female sex hormones. The vagina, uterus and fallopian tubes serve as accessory ducts for the ovaries and a developing foetus.

The vagina

The vagina is a fibromuscular tube about 8 to 10 cm in length located posterior to the bladder and urethra, and anterior to the rectum. The walls of the vagina are membranes that form folds, called rugae. These membranes are composed of mucus-secreting stratified squamous epithelial cells. The vagina serves as a route for the excretion of secretions, including menstrual fluid; an organ of sexual response; and as a passageway for the birth of an infant.

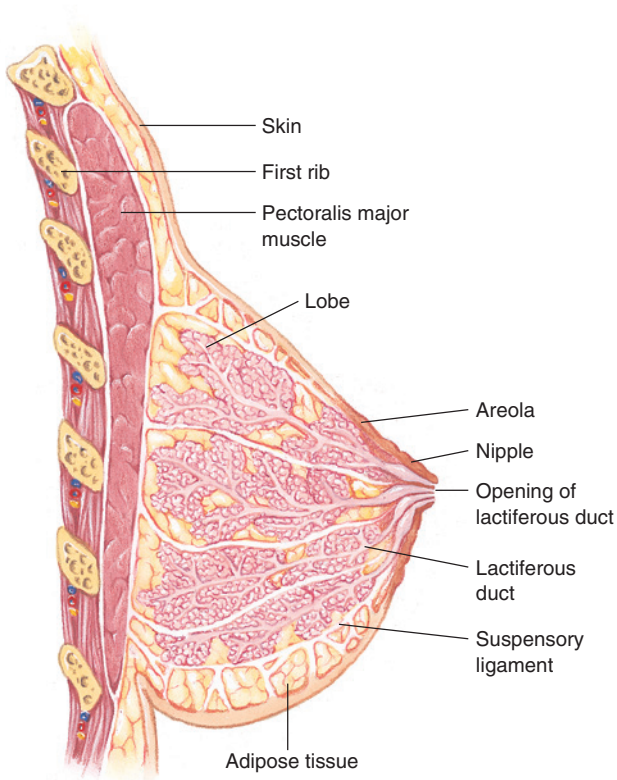


FIGURE 46.4 ■ Structure of the female breast

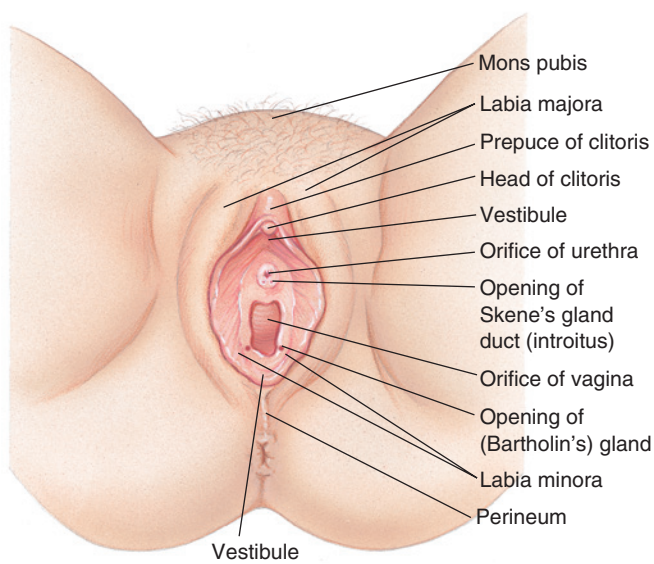


FIGURE 46.5 ■ The external organs of the female reproductive system

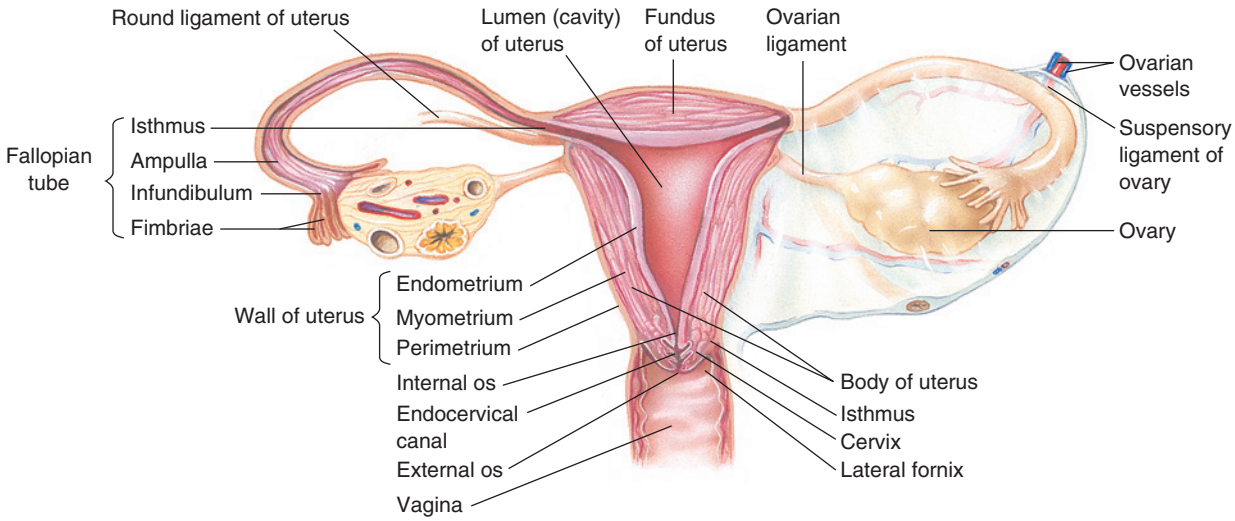


FIGURE 46.6 ■ The internal organs of the female reproductive system

The walls of the vagina are usually moist and maintain a pH ranging from 3.8 to 4.2. This pH is bacteriostatic and is maintained by the action of oestrogen and normal vaginal flora. **Oestrogen** stimulates the growth of vaginal mucosal cells so that they thicken and have increased glycogen content. The glycogen is fermented to lactic acid by Döderlein's bacilli (lactobacilli that normally inhabit the vagina), slightly acidifying the vaginal fluid. The upper end of the vagina contains the uterine cervix in an area called the fornix.

The cervix

The cervix is essentially the 'neck of the uterus' and is the lower, narrow part of the uterus which is cylindrical in shape and protrudes through the upper anterior vaginal wall forming a conduit between the uterus and the vagina. The cervix is a firm structure, protected by mucus that changes consistency and quantity during the menstrual cycle and during pregnancy. The uterine opening of the cervix is called the internal os; the vaginal opening is called the external os. The space between these openings, the endocervical canal, serves as a route for the discharge of menstrual fluid, the entrance for sperm and expulsion of the infant during birth.

The uterus

The uterus is a hollow, pear-shaped muscular organ with thick walls located between the bladder and the rectum. It has three parts: the fundus, the body and the cervix. It is supported in the abdominal cavity by the broad ligaments, the round ligaments, the uterosacral ligaments and the transverse cervical ligaments. The uterus receives the fertilised ovum and provides a site for growth and development of the foetus.

The uterine wall has three layers. The perimetrium is the outer serous layer that merges with the peritoneum. The myometrium is the middle layer and makes up most of the uterine wall. This layer has muscle fibres that run in various directions, allowing contractions during **menstruation** (the periodic shedding of the uterine lining in a woman of childbearing age who is not pregnant) or childbirth and expansion as the foetus grows. The endometrium lines the uterus; its outermost layer is shed during menstruation.

The fallopian tubes

The fallopian tubes are thin cylindrical structures about 10 cm long and 1 cm in diameter. They are attached to the uterus on one end and are supported by the broad ligaments. The lateral ends of the fallopian tubes are open and made of projections called fimbriae that drape over the ovary. The fimbriae pick up the ovum after it is discharged from the ovary.

The fallopian tubes are made of smooth muscle and are lined with ciliated, mucus-producing epithelial cells. The movement of the cilia and contractions of the smooth muscle move the ovum through the tubes towards the uterus. Fertilisation of the ovum by the sperm usually occurs in the outer portion of a fallopian tube.

The ovaries

The ovaries in the adult woman are flat, almond-shaped structures located on either side of the uterus below the ends of the fallopian tubes. They are homologous to the man's testes. They are attached to the uterus by a ligament and are also attached to the broad ligament. The ovaries store the female germ cells and produce the female hormones oestrogen and progesterone. A woman's total number of ova is present at her birth as oogenesis, the formation and development of a female reproductive cell ovum, occurs during gestation.

Each ovary contains many small structures called ovarian follicles. Each follicle contains an immature ovum, called an oocyte. Each month, several follicles are stimulated by follicle-stimulating hormone (FSH) and luteinising hormone (LH) to mature. The developing follicles are surrounded by layers of follicle cells, with the mature follicles called graafian follicles. The graafian follicles produce oestrogen, which stimulates the development of endometrium. Each month in the menstruating woman, one or two of the mature follicles eject an oocyte in a process called ovulation.

The ruptured follicle then becomes a structure called the corpus luteum. The corpus luteum produces both oestrogen and progesterone to support the endometrium until conception occurs or the cycle begins again. The corpus luteum slowly degenerates, leaving a scar on the surface of the ovary.

Functions of the female sex hormones

The ovaries produce oestrogens, progesterone and androgens in a cyclical pattern. Oestrogens are steroid hormones that occur naturally in three forms: oestrone (E1), oestradiol (E2) and oestriol (E3). Oestradiol is the most potent and is the form secreted in greatest amount by the ovaries. Although oestrogens are secreted throughout the menstrual cycle, they are at a higher level during certain phases of the cycle.

Oestrogens are essential for the development and maintenance of secondary sex characteristics. In conjunction with other hormones, they stimulate the female reproductive organs to prepare for growth of a foetus. Oestrogens are responsible for the normal structure of skin and blood vessels. They also decrease the rate of bone resorption, promote increased high-density lipoproteins, reduce cholesterol levels and enhance the clotting of blood. Oestrogens also promote the retention of sodium and water.

Menopause, a normal physiological process, occurs as a result of the gradual decrease and final cessation of oestrogen production by the ovaries. Menstruation ceases and the tissues that had been supported by oestrogen change. Long-term effects of oestrogen deprivation increase the risk of osteoporosis and cardiovascular disease. Menopause is discussed in Chapter 48.

Progesterone primarily affects the development of breast glandular tissue and the endometrium. During pregnancy, progesterone relaxes smooth muscle to decrease uterine contractions. It also increases body temperature.

Androgens are responsible for normal hair growth patterns at puberty and may also have metabolic effects.

Oogenesis and the ovarian cycle

Oogenesis is the process of maturation of oocytes. It begins before birth and is completed after puberty, continuing until menopause. During prenatal oogenesis, the total number of a woman's ova develop and are present as primary oocytes in ovarian follicles. Each month from puberty until menopause, the remaining events of oogenesis, known as postnatal oogenesis, occur. Collectively, these events are known as the **ovarian cycle**.

The ovarian cycle has three consecutive phases that occur cyclically each 28 days (although the cycle may be longer or shorter), as follows:

- The follicular phase lasts from the 1st to the 10th day of the cycle.
- The ovulatory phase lasts from the 11th to the 14th day of the cycle and ends with ovulation.
- The luteal phase lasts from the 14th to the 28th day.

During the follicular phase, the follicle develops and the oocyte matures. These processes are controlled by the interaction of FSH and LH. On day 1 of the cycle, gonadotropin-releasing hormone (GnRH) from the hypothalamus increases and stimulates increased production of FSH and LH by the anterior pituitary. FSH and LH stimulate follicular growth and the oocyte increases in size. The structure, now called the primary follicle, becomes a multicellular mass surrounded by a fibrous capsule, the theca folliculi. As the follicle continues to increase in size, oestrogen is produced and a fluid-filled space (the antrum) forms within the follicle. The oocyte is enclosed by a membrane, the zona pellucida. By about day 10, the follicle is a mature graafian follicle and bulges out from the surface of the ovary. There are always follicles at different stages of development in each ovary, but usually only one follicle becomes dominant and matures to ovulation, while the others degenerate.

The ovulatory phase begins when oestrogen levels reach a level high enough to stimulate the anterior pituitary and a surge of LH is produced. The LH stimulates meiosis in the developing oocyte and its first meiotic division occurs. The LH also stimulates enzymes that act on the bulging ovarian wall, causing it to rupture and discharge the antrum fluid and the oocyte. The oocyte is expelled from the mature ovarian follicle in the process called ovulation.

During the luteal phase, the surge in LH also stimulates the ruptured follicle to change into a corpus luteum and then stimulates the corpus luteum to begin immediately producing progesterone and oestrogen. The increase of progesterone and oestrogen in the blood has a negative feedback effect on the production of LH, inhibiting the further growth and development of other follicles.

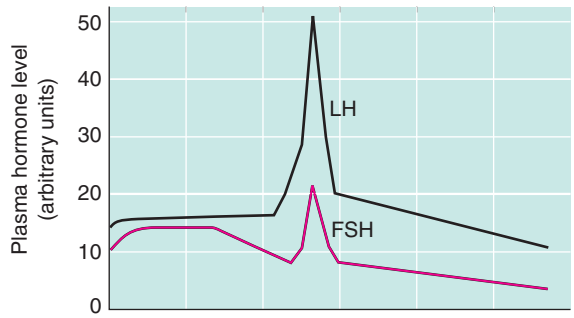
If pregnancy does not occur, the corpus luteum begins to degenerate and its hormone production ceases. The declining production of progesterone and oestrogen at the end of the cycle allows the secretion of LH and FSH to increase and a new cycle begins. The ovarian cycle is compared with the menstrual cycle in Figure 46.7.

The menstrual cycle

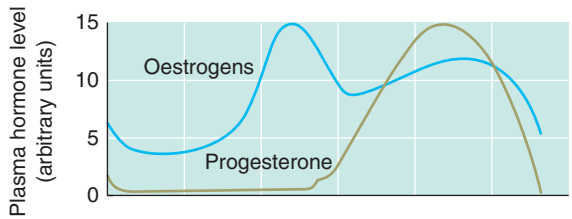
The endometrium of the uterus responds to changes in oestrogen and progesterone during the ovarian cycle to prepare for implantation of the embryo. The endometrium is receptive to implantation of the embryo for only a brief period each month, coinciding with the time when the embryo would normally reach the uterus from the uterine tube (usually 7 days).

The **menstrual cycle** begins with the *menstrual phase*, lasting from days 1 to 5. The inner endometrial (functionalis) layer detaches and is expelled as menstrual fluid (fluid and blood) for 3 to 5 days. As the maturing follicle begins to produce oestrogen (days 6 to 14), the proliferative phase begins. In response, the functionalis layer is repaired and thickens, while spiral arteries increase in number and tubular glands form. Cervical mucus changes to a thin, crystalline substance, forming channels to help the sperm move up into the uterus.

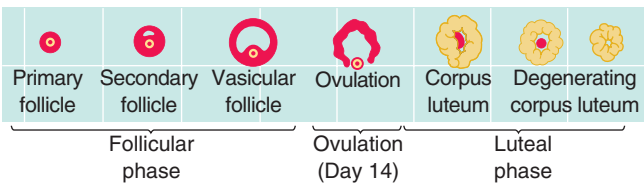
The final phase, lasting from days 14 to 28, is the secretory phase. As the corpus luteum produces progesterone, the rising levels act on the endometrium, causing increased vascularity, changing the inner layer to secretory mucosa, stimulating the secretion of glycogen into the uterine cavity and causing the



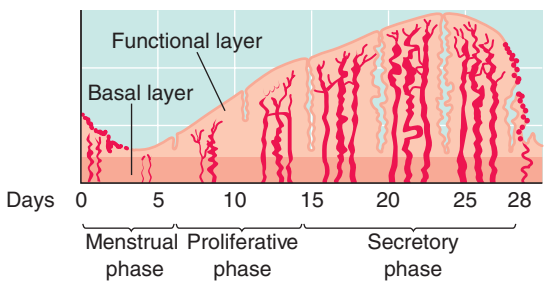
A Fluctuation of gonadotropin levels



B Fluctuation of ovarian hormone levels



C Ovarian cycle



D Menstrual cycle

FIGURE 46.7 ■ Comparison of the ovarian and menstrual cycles. *A*, Fluctuating levels of follicle-stimulating hormone (FSH) and luteinising hormone (LH), the pituitary gonadotropins regulating the ovarian cycle. *B*, Fluctuating levels of ovarian hormones that cause endometrial changes during the menstrual cycle. *C*, Changes in the ovarian follicles during the 28-day menstrual cycle. *D*, Corresponding changes in the endometrium during the menstrual cycle

cervical mucus again to become thick and block the internal os. If fertilisation does not occur, hormone levels fall. Spasm of the spiral arteries causes hypoxia of the endometrial cells, which begin to degenerate and slough off. As with the ovarian cycle, the process begins again with the sloughing of the functionalis layer.

ASSESSING THE FEMALE REPRODUCTIVE SYSTEM

The structures and functions of the female reproductive system are assessed by findings from a comprehensive assessment which includes both a health assessment interview to collect subjective data and a physical assessment to collect objective data. In addition, a number of diagnostic tests may provide further information.

Information from the health assessment interview is used to individualise the questions that are asked; for example, a woman who is postmenopausal would not be asked specific questions about her menstrual cycle, but it would be important to ask about vaginal dryness. Sample documentation of an assessment of the female reproductive system is included in the box below.

Health assessment interview

A health assessment interview to determine problems with the female reproductive system may be conducted for different reasons, for example:

- during a health screening
- as part of a comprehensive health assessment
- during a focused assessment for a specific health problem (such as severe menstrual cramping).

Women may be embarrassed to discuss health problems or concerns involving their reproductive organs; therefore it is important for the nurse to ask questions in a non-judgmental, non-threatening, matter-of-fact and sensitive manner. Consider the psychological, social and cultural factors that affect sexuality and sexual activity. Use words that the woman can understand and do not be embarrassed or offended by the words that she uses. The woman may perceive the interview as less threatening if the discussion begins with more general questions and then progresses to specific questions, and if questions are asked in a way that gives the woman permission to describe behaviours and manifestations. For example, ask about menstrual and childbirth histories before asking questions about sexually transmitted infections.

The focused interview for the female reproductive system is usually extensive. However, the questions may in many instances be tailored to the specific health problem of the woman. As with the assessment of other body systems, analyse and document the onset of the problem, its duration, frequency, precipitating and relieving factors, any associated symptoms, treatment, self-care and outcome. For example, ask the woman:

- Have you noticed vaginal bleeding after intercourse that isn't related to menstruation?
- Does anything relieve the vaginal itching and discharge?
- Have you had any fever or abdominal pain with this vaginal infection?

Ask about menstrual history, obstetric history, use of contraceptives, sexual history, use of medications and reproductive system examinations. Ask about the use of condoms during intercourse because unprotected sexual intercourse increases the risk of sexually transmitted infections, including

SAMPLE DOCUMENTATION

Assessment of the female reproductive system

2/10/2015
NURS
1000 hrs

30-year-old female, first visit to health department for problems she described as 'pain and burning down below' and 'It really hurts to pee'. Also states she had unprotected sex on a date about 2 weeks ago. Oral temperature 38.5°C. Vaginal examination findings of vesicles and red ulcerations on labia majora and vaginal mucosa. Inguinal lymph nodes enlarged and tender to palpation. Culture of ulcerations taken and specimen sent to the laboratory for analysis.——————— RN T. Tunn

T. TUNN. RN

hepatitis B and HIV infection. Also ask about smoking because a history of smoking increases the risk of circulatory problems in the woman taking oral contraceptives and increases the risk of cancer of the cervix.

Chronic conditions may affect the function of the female reproductive system. Diabetes mellitus increases the risk of vaginal infections and vaginal dryness, both of which interfere with sexual pleasure. Chronic heavy menstrual flow may result in anaemia. Thyroid and adrenal conditions may affect secondary sex characteristics, the menstrual cycle and the ability to become pregnant.

Obtaining any family history of cancer is important. The risk of endometrial cancer is higher in women with a family history of endometrial, breast or colon cancer; the risk of ovarian cancer is higher in women with a family history of ovarian or breast cancer; and the risk of breast cancer is higher in women with a family history of breast cancer. Exposure to DES (diethylstilbestrol, a synthetic form of oestrogen) in utero increases the risk of cancer of the cervix and vagina. Exposure to asbestos poses a risk of cancer of the ovary. The risk of breast cancer is also greater if the woman has a history of fibrocystic disease.

Carefully explore with the woman any history of vaginal bleeding and vaginal discharge. Ask about the onset of vaginal bleeding, any related factors, the colour (pink, red, dark red, brown), the character (thin, watery, presence of mucus, size and number of clots), the amount (spotting, how many pads or tampons in a specific amount of time) and relationship to her menstrual cycle. Ask about the onset of any vaginal discharge; the colour (white, green, grey), character (thin, thick, curd-like), odour, itching and if a rash is present.

Questions about sexual activity may include sexual preference, number of sexual partners, use of condoms, femidoms or other contraceptives, and current level of sexual satisfaction. Ask about any experiences or history of **anorgasmia** (absence of orgasm), **dyspareunia** (painful intercourse) or other problems with intercourse, and any history of sexual trauma.

Interview questions categorised by functional health patterns are listed in the following 'Functional health pattern interview' table.

Physical assessment

Physical assessment of the female reproductive system may be performed as part of a comprehensive assessment or separately for women with identified sexual health problems. If conducted as part of a total physical assessment, this is usually the final system to be assessed. The nurse must feel comfortable with the examination of people of the opposite gender. If either the nurse or the individual

FUNCTIONAL HEALTH PATTERN INTERVIEW The female reproductive system

FUNCTIONAL HEALTH PATTERN	INTERVIEW QUESTIONS AND LEADING STATEMENTS
Health perception–Health management	■ Have you ever had problems with your reproductive organs (ovaries, tubes, uterus, vagina) or with menstruation or menopause? Explain. If so, how was this problem treated? ■ Do you routinely take any prescribed or herbal medications for symptoms of menopause? If so, what and when do you take it? ■ Did you ever take hormone replacement therapy for menopausal symptoms? ■ Do you practise breast self-examination? When and how often do you do this? ■ Have you noticed any lumps in your breasts or discharge from your nipples? If so, describe. ■ Have you ever had a breast examination or mammogram? When was your last one? How often do you have these? ■ When was your last gynaecological examination? Pap smear? How often do you have these done? ■ Do you use birth control? If so, what do you use? ■ What do you do to provide self-care if you have mood swings or menstrual cramps? ■ Have you ever had a sexually transmitted infection or an infection of the reproductive organs? What was it? How was it treated? ■ Do you use douches or vaginal sprays? If so, what type and how often? ■ Do you smoke? If so, how much and for how long?

(continued)

FUNCTIONAL HEALTH PATTERN INTERVIEW The female reproductive system (continued)

Nutritional–Metabolic	■ Do you notice a change in your appetite right before your menstrual period? ■ Have you gained weight recently? If so, why do think this happened? ■ Describe your usual food intake for a 24-hour period.
Elimination	■ When was your last menstrual period? ■ At what age did you start/end having menstrual periods? ■ Describe the length, amount of flow and clotting with your menstrual periods. ■ Do you ever have bleeding between your menstrual periods? If so, describe the type and amount. ■ Describe any unusual vaginal discharge you have had (colour, consistency, odour, itching or rash). ■ Have you noticed any changes in urination (frequency, urgency, burning)? ■ Have you noticed changes in bowel elimination during your menstrual periods?
Activity–Exercise	■ Describe your usual activities of daily living. ■ Have you noticed any change in activity or energy during your menstrual period? ■ Have you noticed any change in activity or energy since menopause (if applicable)? If so, how?
Sleep–Rest	■ How long do you sleep at night? Is your sleep restful? ■ Do night sweats wake you? ■ Do menstrual cramps ever wake you at night?
Cognitive–Perceptual	■ Do you have pain or other symptoms (such as headache, mood swings, irritability, bloating, constipation, diarrhoea and/or breast tenderness) before your menstrual period? Describe. What do you do about this? ■ Do you have cramping before or during your menstrual period? Describe the type of cramping, how long it lasts and what you do to be more comfortable. ■ Do you ever have vaginal itching, pain, burning or dryness? If so, is it affected by sexual intercourse? Does dryness interfere with intercourse?
Self-Perception–Self-Concept	■ Has this problem affected how you feel about yourself as a woman? ■ Do you believe your needs for intimacy and affection are being met?
Role–Relationships	■ How has having this condition affected your relationships with others? ■ Has having this condition interfered with your ability to work? Explain. ■ Has anyone in your family had problems with breast or ovarian cancer? Explain.
Sexuality–Reproductive	■ Are you currently in a sexual relationship? If so, has this condition interfered with your usual sexual activity? ■ How long have you been with your current partner? Have you had any other partners during this time? ■ What is your sexual preference? ■ Has having this problem affected your relationship with your spouse or sexual partner? ■ Have you ever been pregnant? How many times? Have you ever had a miscarriage? ■ Do you practise birth control? If so, what do you use? ■ Do you ensure that your partner of the opposite gender uses a condom every time you have intercourse? ■ Do you use a vaginal condom?
Coping–Stress–Tolerance	■ Has having this condition created stress for you? If so, does your health problem seem to be more difficult when you are stressed? ■ Have you experienced any kind of stress that makes the condition worse? Explain. ■ Describe what you do when you feel stressed.
Value–Belief	■ Describe how specific relationships or activities help you cope with this problem. ■ Describe specific cultural beliefs or practices that affect how you care for and feel about this problem. ■ Are there any specific treatments that you would not use to treat this problem?

is not comfortable, a nurse of the same gender should be asked to conduct this part of the assessment. The female reproductive system is assessed by inspection and palpation. Ask the woman to void before having the examination. Prior to the examination, collect all necessary equipment and explain the techniques to the woman to decrease anxiety. Put on disposable gloves before beginning the examination and wear them throughout the examination. Ask the woman to remove her clothing and put on a gown. Ensure that the examining room is private and warm.

Explain the procedures for the examination thoroughly and in a matter-of-fact way to decrease anxiety and embarrassment. If the woman is unfamiliar with her reproductive organs, charts may be used to illustrate the parts that will be examined. Carefully explain the procedure for the examination and show the speculum to the woman. The assessment may be done with the woman in the sitting or supine position to examine the breasts and in the lithotomy position to assess the external genitalia and internal organs. Expose only those body parts being examined to preserve modesty. Normal age-related findings for the older woman are summarised in Table 46.4.

The examination usually begins with examination of the breasts with the woman in the sitting and supine positions. The nurse then helps the woman move to the lithotomy position on the examining table, with the feet in the stirrups and the buttocks level with the foot of the table. This position may induce feelings of vulnerability for the woman and anxiety related to past experiences. Older or frail women may not be able to tolerate this position. In this case, the woman may be examined in the supine position. Although the entire examination is described here, the internal examination is conducted only by a nurse with advanced practice in the procedure. However, nurses are often asked to assist with the examination and should be able to explain the examination to a woman and answer her questions about the procedure.

Diagnostic tests

The results of diagnostic tests of the structures and functions of the female reproductive system are used to monitor the health of female reproductive structures; to support the diagnosis of a specific sexual problem, injury or disease; to provide information to identify or modify the appropriate medications or treatments used to treat the condition; and to help monitor the woman's responses to treatment and nursing care interventions. Diagnostic tests to assess the female reproductive system are described in the 'Diagnostic tests' table below and summarised in the following bulleted list. More information is included in the discussion of specific health problems or diseases in Chapter 48.

- Blood tests are used to diagnose a variety of hormone changes and sexually transmitted infections. These tests are discussed in Chapters 48 and 49.
- Sexually transmitted infections are often diagnosed with cultures and smears of a discharge or mucous membranes.
- A mammogram is used to detect breast tumours, often followed by an ultrasound and breast biopsy for a definitive diagnosis. The type of biopsy conducted depends on many factors, including the size, location, appearance and characteristics of the breast abnormality.
- The Papanicolaou (Pap) smear and the HPV DNA test are conducted to diagnose premalignant and malignant

TABLE 46.4 Age-related changes in the female reproductive system

AGE-RELATED CHANGE	SIGNIFICANCE
Breasts	
• Atrophy, with sagging of breast tissue. • Linear strands may appear from shrinkage and fibrotic changes.	Although ageing does not cause breast cancer, the incidence rises in older women; age-related changes may make finding tumours more difficult.
External genitalia	
• Labia flatten and vulvar adipose tissue and hair decreases. • ↓ collagen and adipose tissues in the vaginal canal, resulting in loss of rugae, shortening and narrowing of vaginal canal. • ↓ vaginal lubrication, epithelium becomes thinner and avascular. • More alkaline pH of vagina. • Cervix becomes smaller.	Vagina is more easily irritated, increasing the risk of vaginal infections. Lubricants are necessary for comfortable intercourse.
Internal organs	
• Uterus shrinks. • Fallopian tubes shrink and shorten. • Ovaries are smaller and thicker. • With menopause, hormone production of oestrogen decreases. • Loss of oestrogen may cause pelvic floor muscles to weaken. • Loss of oestrogen causes changes throughout the body, including loss of skin tone (wrinkling) and growth of facial hair.	With the completion of menopause, the menstrual cycles end and the woman is infertile. Weakening of the pelvic floor muscles may contribute to involuntary incontinence with increased intra-abdominal pressure (as with coughing and sneezing). Skin is dry and thin.

DIAGNOSTIC TESTS The female reproductive system

Screening tests, smears and cultures

NAME OF TEST Papanicolaou smear (Pap test)

PURPOSE AND DESCRIPTION Conducted to diagnose malignant and premalignant lesions of the cervix; to assess the effects of hormone replacement therapy; to identify viral, bacterial, fungal and parasitic conditions; and to evaluate response to chemotherapy or radiation therapy to the cervix. Cells are obtained during a pelvic examination, with a wooden spatula, a cotton swab or an endocervical brush. The sample collected may be smeared on a glass slide or put into a special liquid preservative and then the cells in suspension are processed onto a slide. The cells are then stained and examined.

RELATED NURSING CARE Explain that the test should be done during a time when the woman is not menstruating and that she should not have intercourse, douche or use vaginal medications for 36 hours prior to the examination. Ask the woman to void prior to the examination. Reassure the woman, explain the procedure and what to expect.

NAME OF TEST HPV test (HPV DNA test, genital human papilloma test)

PURPOSE AND DESCRIPTION Routinely used as a screening tool for human papillomavirus (HPV) in women after the age of 30. Conducted in conjunction with a pelvic examination and Pap smear. A finding of 'low-grade changes' on the Pap smear with HPV indicates the likely presence of HPV and the need for further testing. A positive test for HPV indicates the presence of a high-cancer type of HPV, but does not specify which type is present.

RELATED NURSING CARE Explain that the test should be done during a time when the woman is not menstruating and that she should not have intercourse, douche or use vaginal medications for 36 hours prior to the examination. Ask the woman to void prior to the examination.

NAME OF TEST Chlamydia culture

PURPOSE AND DESCRIPTION Performed to screen for or diagnose chlamydial infections. A swab of cells from the infected area is taken and either smeared on a slide and analysed or cultured. Although usually taken from the urethra, vagina or cervix, cultures may also be taken from the throat and rectum.

RELATED NURSING CARE Assess if the woman is pregnant or has enlargement of inguinal lymph nodes. Withhold antibiotics (if prescribed) until after obtaining the specimen. Instruct not to douche before the examination. If the test is positive, request the names of all sexual partners and emphasise need for treatment to eradicate the infection.

NAME OF TEST Gonorrhoea culture

PURPOSE AND DESCRIPTION A culture is performed to evaluate for gonorrhoea. A swab is used to collect a sample of discharge from the infected area (cervix, urethra, anus or throat), smeared on a slide and a Gram stain is conducted to identify the organism (*N. gonorrhoeae*). A urine sample is used in some tests.

RELATED NURSING CARE No special physical preparation is needed but psychological care is required. Instruct not to douche before the examination. If the test is positive, request the names of all sexual partners and emphasise the need for treatment to eradicate the infection.

NAME OF TEST Trichomonas, bacteria, candidae (yeast)

PURPOSE AND DESCRIPTION A culture is performed to identify vaginal organisms or blood cells. A specimen of vaginal discharge is obtained with a swab, placed in solution and examined under the microscope immediately after it is collected (referred to as a wet mount).

RELATED NURSING CARE Request not to douche before the examination.

NAME OF TEST Venereal disease research laboratory (VDRL); Rapid plasma reagin (RPR); Fluorescent treponemal antibody absorption (FTA-ABS)

PURPOSE AND DESCRIPTION These blood tests are conducted to screen for syphilis. Positive findings can be made within 1 to 2 weeks after primary lesion appears or 1 to 4 months after the initial infection. The FTA-ABS test is used to detect antibodies to the syphilis-causing bacteria *Treponema pallidum.* It is considered the most accurate and is often used if findings from the VDRL or RPR are questionable, but it remains positive after treatment and so cannot be used to monitor treatment efficacy.

RELATED NURSING CARE No special physical preparation is needed but psychological care is required. If the test is positive, request the names of all sexual partners and emphasise the need for treatment to eradicate the infection.

NAME OF TEST Syphilis (dark-field examination)

DIAGNOSTIC TESTS The female reproductive system (continued)

PURPOSE AND DESCRIPTION A specimen is obtained from a lesion believed to be caused by syphilis (*T. pallidum*) and examined under the microscope.

RELATED NURSING CARE No special physical preparation is needed but psychological care is required. If the test is positive, request the names of all sexual partners and emphasise the need for treatment to eradicate the infection.

Breast examinations

NAME OF TEST Mammogram

PURPOSE AND DESCRIPTION Used to detect tumours in the breast. Breasts are flattened in the mammography machine and low-dose x-rays are taken.

RELATED NURSING CARE Ask the woman not to apply body powder or underarm deodorant prior to the test.

NAME OF TEST Breast ultrasound

PURPOSE AND DESCRIPTION This examination uses high-frequency sound waves passing through tissues to detect masses in the breast. May be performed if lesions are identified in a mammogram.

RELATED NURSING CARE No special physical preparation is needed but psychological care is required.

NAME OF TEST Breast biopsy

- Fine-needle aspiration
- Core-needle biopsy
- Vacuum-assisted mammotome
- Large-core surgical biopsy
- Open surgical biopsy

PURPOSE AND DESCRIPTION

- A fine-needle aspiration is conducted to withdraw fluid from cysts and may be used to sample cells from masses in the breast. A 22- to 25-gauge needle is used to collect 5 to 6 samples of fluid or cells.
- A core needle biopsy is conducted to obtain a sample of tissue from a solid mass or calcium deposits in the breast. A 10-, 11- or 12-gauge needle is used to collect 5 to 6 tissue samples.
- A vacuum-assisted mammotome is primarily used to evaluate calcifications. An 11- or 14-gauge needle is inserted through a small (6 mm) incision and 8 to 10 samples are removed.
- A large-core surgical biopsy is performed to evaluate breast masses or calcification identified with a mammogram, but that are non-palpable. An incision is made and a 5 to 20 mm cylinder of breast tissue (about the size of a wine cork) is removed.
- An open surgical biopsy is performed to evaluate breast masses, hard-to-reach lesions, multiple lesions and masses with calcifications. A 3.8 to 5 cm incision is made and a golf ball size (or larger) area of tissue is removed.

RELATED NURSING CARE For all types of test, wearing a well-fitted, comfortable bra, applying ice packs and mild analgesics decrease discomfort post procedure.

- Explain that, depending on the medical officer, some procedures may be performed with or without a local anaesthetic.
- Explain that for a core-needle biopsy or a mammotome a local anaesthetic is used, but no stitches are required.
- Explain that for a large-core biopsy, a local anaesthetic will be administered and stitches will be used to close the incision.
- Explain that for an open surgical biopsy, a general anaesthetic is usually used and the incision will require stitches and leave a scar.

Tests of the internal reproductive system

NAME OF TEST Ultrasound (abdominal, vaginal)

PURPOSE AND DESCRIPTION Used to detect the presence of space-occupying lesions, such as fibroid tumours, cysts, abscesses and neoplasms. The abdomen is coated with transducing gel and a graphical visualisation is made. For a vaginal ultrasound, a transducer is covered with a condom or vinyl glove coated with transducer gel and then introduced into the vagina.

RELATED NURSING CARE Explain need to increase intake of fluids and tell the woman not to void until the test is completed to ensure a full bladder for the abdominal ultrasound. (This lifts the pelvic organs higher in the abdomen and improves visualisation.) An empty bladder is required for the vaginal ultrasound.

NAME OF TEST Hysterosalpingogram

PURPOSE AND DESCRIPTION Used to diagnose causes of infertility and abnormalities of the uterus or fallopian tubes. A contrast medium is instilled through the cervix and its passage through the uterus and the fallopian tubes is monitored using serial x-rays.

RELATED NURSING CARE Assess for allergy to seafood (iodine) or previous contrast media. Explain that the procedure is briefly painful.

(continued)

DIAGNOSTIC TESTS The female reproductive system (continued)

NAME OF TEST Colposcopy

PURPOSE AND DESCRIPTION Conducted to further study abnormal Pap tests and as screening for women exposed to intrauterine DES. A binocular microscope is used to directly visualise the cervix.

RELATED NURSING CARE No special physical preparation is needed but psychological care is required.

NAME OF TEST Conisation, Loop electrosurgical excision of transformation zone (LEETZ), Loop electrosurgical excision procedure (LEEP)

PURPOSE AND DESCRIPTION A conisation, LEETZ or LEEP is performed to remove cervical tissue for evaluation (most often for cervical cancer). A cone-shaped area of tissue surrounding the cervical os is removed.

RELATED NURSING CARE Explain that the procedure requires general anaesthesia; however, local anaesthesia may also be used for the procedure. Postoperative self-care includes rest for 2 to 3 days. Explain that minor vaginal bleeding and discharge are expected for several days after the procedures; perineal pads (not tampons) should be used. Sexual intercourse should be avoided until discharge stops. Notify medical officer of increased bleeding or signs of infection (pain, foul-smelling discharge, fever) occur.

NAME OF TEST Endometrial biopsy

PURPOSE AND DESCRIPTION Performed to identify endometrial hyperplasia or endometrial cancer. The cervix is cleaned and tissue is obtained transcervically from the endometrium, either by curettage or vacuum aspiration.

RELATED NURSING CARE Explain that the procedure is briefly painful and causes vaginal bleeding. Advise to use perineal pads and avoid tampons and sexual intercourse while bleeding.

NAME OF TEST Cervical biopsy

PURPOSE AND DESCRIPTION Performed for women when Pap test results indicate possible cervical cancer or cervical intraepithelial neoplasia (CIN), and for screening women at high risk of vaginal and cervical cancers from intrauterine exposure to DES. Cervix is cleaned and a sample of tissue is taken for analysis.

RELATED NURSING CARE Explain that minor vaginal bleeding and discharge are expected for several days after the procedures; perineal pads (not tampons) should be used. Sexual intercourse should be avoided until discharge stops. Notify the healthcare provider if increased bleeding or signs of infection (pain, foul-smelling discharge, fever) occur.

NAME OF TEST Laparoscopy

PURPOSE AND DESCRIPTION This examination is conducted to visualise the organs in the peritoneal cavity (uterus, fallopian tubes, ovaries); to withdraw fluid for analysis; and to perform a tubal ligation. A fibre-optic scope is inserted through small abdominal incisions and carbon dioxide is inserted into the peritoneal cavity for better visualisation.

RELATED NURSING CARE Ask the woman to void prior to the examination and explain that a general anaesthetic will be used. Explain that shoulder pain is common after the procedure (referred pain from the retained carbon dioxide gas); that some vaginal bleeding may occur and the woman should use a perineal pad; and to report excess bleeding, pain or signs of infection to the healthcare provider.

conditions of the cervix and to monitor positive HPV tests. These tests may also be used to assess the effects of hormone replacement, identify other infective organisms and evaluate response to therapy.

- Space-occupying lesions and abnormalities of the vagina, cervix or uterus may be evaluated with ultrasound, a hysterosalpingogram, a colposcopy, a cervical biopsy, a laparoscopy and/or an endometrial biopsy.

Regardless of the type of diagnostic test, the nurse is responsible for explaining the procedure and any special preparation needed, assessing for any medication use that might impact on findings of the tests, supporting the woman during the examination, documenting the procedures as appropriate and following up with regard to the results of the tests.

Genetic considerations

When conducting a health assessment interview and a physical assessment, it is important for the nurse to consider genetic influences on the health of an individual. Several conditions that affect the female reproductive system have a genetic component. During the health assessment interview it is especially important to ask about a family history of ovarian or breast cancer. During the physical assessment, assess for any manifestations that might indicate a genetic disorder (see the box below). If findings indicate genetic risk factors or conditions, discuss genetic testing with the woman and refer her for appropriate genetic counselling and evaluation. Chapter 7 provides further information about genetics in medical–surgical nursing.

GENETIC CONSIDERATIONS Female reproductive system disorders

- There is a clear genetic link in some types of breast and ovarian cancers. Two breast-cancer-susceptibility genes have been identified: BRCA1 and BRCA2. If a woman has either of these genes, she is at increased risk of having breast or ovarian cancer at some point in her life.
- A family history of endometrial, colon or breast cancer increases a woman's risk of endometrial cancer.
- Turner's syndrome is a disorder in a female caused by complete or partial absence of one of the two X chromosomes. The disorder is characterised by short stature and the lack of sexual development at puberty. Other physical effects include a webbed neck, heart defects and kidney abnormalities.

FEMALE REPRODUCTIVE SYSTEM ASSESSMENTS

Technique/normal findings	Abnormal findings
Breasts	
Inspect both breasts simultaneously with the woman seated in the following positions: arms at sides, arms overhead, hands pressed on hips, leaning forwards. Inspect breast size, symmetry, contour, skin colour, texture, venous patterns and lesions. Lift the breasts and inspect the lower and lateral aspects. *Breasts normally vary in size and shape and one breast may normally be larger than the other. Colour should be consistent with the skin tone and texture smooth. There should be no redness, swelling, prominent veins or lesions.*	■ Retractions, dimpling and abnormal contours suggest benign lesions, but may also suggest malignancy. ■ Thickened, dimpled skin with enlarged pores (called peau d'orange, orange peel or pig skin) and unilateral venous patterns are also associated with malignancy. ■ Redness may be seen with infection or cancer.
Inspect the areolae and nipples. *The colour of the areolae should be consistent with the woman's skin colour (ranging from dark pink to dark brown) and Montgomery tubercles may be present. The nipples should be equal bilaterally in size, centrally located in each breast and free of lesions or discharge. Nipples are usually everted, but may normally be inverted or flat.*	■ Peau d'orange may be noted first in the areola. ■ Recent unilateral inversion of the nipple or asymmetry in the directions in which the nipples point suggests cancer.
Palpate both breasts, axillae and supraclavicular areas. Figure 46.8 illustrates a possible pattern for breast palpation. Various palpation patterns may be used as long as every part of each breast is palpated, including the axillary tail (also called tail of Spence), which is the breast tissue that extends from the upper outer quadrant towards and into the axillae.	■ Tenderness may be related to premenstrual fullness, fibrocystic disease or inflammation. Tenderness may also indicate cancer. ■ Nodules in the tail of the breast may be enlarged lymph nodes. ■ Hard, irregular, fixed unilateral masses that are poorly delineated suggest cancer. ■ Bilateral, single or multiple, round, mobile, well-delineated masses are consistent with fibrocystic breast disease or fibroadenoma. ■ Swelling, tenderness, erythema and heat may be seen with mastitis.

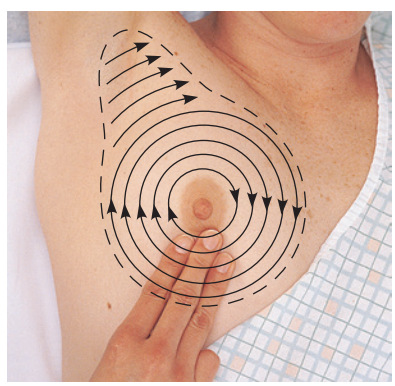


FIGURE 46.8 ■ Possible pattern for palpation of the breast

Technique/normal findings	Abnormal findings
Ask the woman to assume a supine position with a small pillow under the shoulder and the arm over the head, and repeat the systematic palpation sequence. Describe identified masses by location, size, shape, consistency, tenderness, mobility and delineation of borders. *Breasts should feel smooth, firm and elastic, without palpable masses. Prior to the menstrual cycle, there may be increased nodularity and tenderness.*	
Palpate the nipple, then compress it between the thumb and index finger. Note the colour of any discharge. *Nipples should be firm and elastic, normally without discharge (although some women normally have a clear discharge, and a milky substance may be expressed during pregnancy and lactation).*	■ Loss of nipple elasticity is seen in cancer. ■ Bloody or serous discharge is associated with intraductal papilloma. ■ Milky discharge not due to prior pregnancy and found on both sides suggests galactorrhoea (lactation not associated with pregnancy or breastfeeding), which is sometimes associated with a pituitary tumour. ■ Unilateral discharge from one or two ducts can be seen in fibrocystic breast disease, intraductal papilloma or carcinoma.
Axillae Inspect the skin of the axillae. *There should be no redness, irritation, lesions or enlarged lymph nodes on palpation.*	■ Rash may be due to allergy or other causes. ■ Signs of inflammation and infection may be due to infection of the sweat glands. ■ Palpate all sections of both axillae for palpable nodes (see Figure 46.9). ■ Enlarged axillary nodes are most often due to infection of the hand or arm but can be caused by malignancy. ■ Enlarged supraclavicular nodes are associated with lymphatic metastases from abdominal or thoracic cancer.
External genitalia Help the woman to the lithotomy position with the knees flexed and separated.	
Inspect and palpate the labia majora. *The labia majora should be equal in size and free of lesions or bulging.*	■ Excoriation, rashes or lesions suggest inflammatory or infective processes. ■ Bulging of the labia that increases with straining suggests a hernia. ■ Varicosities may be present on the labia.
Inspect the labia minora. Separate the labia majora for better visualisation. *The labia minora should be symmetrical, dark pink and moist, without redness or lesions.*	■ Inflammation, irritation, excoriation or caking of discharge in tissue folds suggests vaginal infection or poor hygiene. ■ Ulcers or vesicles may be symptoms of sexually transmitted infection.

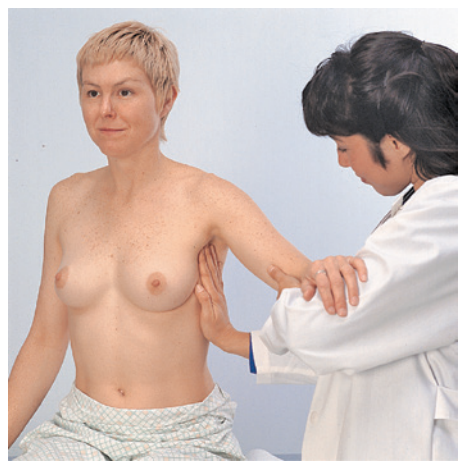


FIGURE 46.9 ■ Palpating the axillary lymph nodes

Technique/normal findings	Abnormal findings
Palpate the inside of the labia minora between thumb and forefinger. *There should be no nodules, ulcers or lesions.*	■ Small, firm, round cystic nodules in labia suggest sebaceous cysts. ■ Wart-like lesions suggest condylomata acuminata (genital warts). ■ Firm, painless ulcers suggest chancre of primary syphilis. ■ Shallow, painful ulcers suggest herpes infection. ■ Ulcerated or red raised lesions in older women suggest vulvar cancer.
Inspect the clitoris. *The clitoris is normally not enlarged.*	■ Enlargement may be a symptom of a masculinising condition.
Inspect the vaginal opening. *There should be no swelling, discolouration, lacerations, discharge or lesions visible in the vaginal opening.*	■ Swelling, discolouration or lacerations may be caused by trauma. ■ Discharge or lesions may be symptoms of infection. ■ Fissures or fistulas may be related to injury, infection, spreading of a malignancy or trauma.
Palpate Skene's glands. Using the index finger, 'milk' Skene's glands on both sides and over the urethra and inspect for possible discharge (see Figure 46.10). *There should be no discharge or tenderness present.*	■ Discharge from Skene's glands and/or tenderness suggests infection.

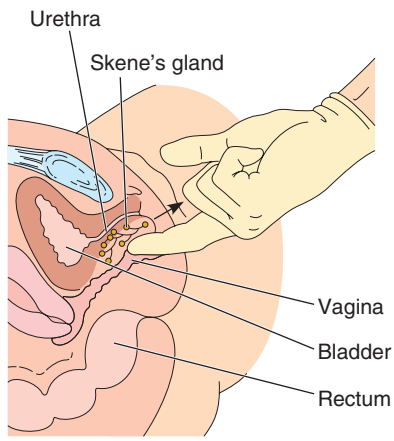


FIGURE 46.10 ■ Palpating Skene's glands

Palpate Bartholin's glands at the posterior labia majora (see Figure 46.11). *There should be no masses, redness, swelling or tenderness on palpation.*	■ A non-tender mass in the posterolateral portion of the labia majora is indicative of a Bartholin's cyst. ■ Swelling, redness or tenderness, especially if unilateral, may indicate abscess of Bartholin's glands.

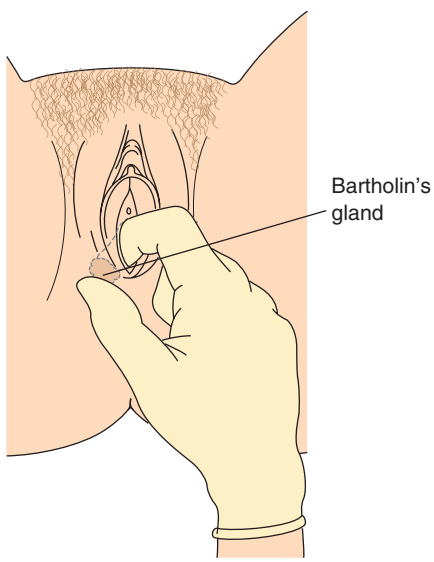


FIGURE 46.11 ■ Palpating Bartholin's glands

Inspect the vaginal orifice for bulging and urinary incontinence. Ask the woman to strain or 'bear down'. *No bulging should be visible with straining.*	■ Bulging of the anterior vaginal wall and urinary incontinence suggest a cystocele. ■ Bulging of the posterior wall suggests a rectocele. ■ Protrusion of the cervix or uterus into the vagina indicates uterine prolapse.

Technique/normal findings	Abnormal findings
Inspect and palpate the perineum. The perineum should be free of redness or lesions. *Episiotomy scars are a normal finding.*	■ Inflammation, lesions and growths may be seen in infections or cancer. ■ Fistulas may be the result of injury, trauma, infection or spreading of a malignancy.
Vagina and cervix	
Use a vaginal speculum to inspect the vaginal walls and cervix. See the guidelines in Box 46.1. *The vaginal opening varies, depending on age, sexual history and vaginal births. Vaginal mucosa is normally pink and moist, without discharge or odour. There should be no bulging or loss of urine. The cervix is normally smooth and pink, without lesions, and has a consistency similar to the tip of the nose.*	■ Bluish colour of the cervix and vaginal mucosa may be a sign of pregnancy. ■ A pale cervix is associated with anaemia. ■ A cervix to the right or left of the midline may indicate a pelvic mass, uterine adhesions or pregnancy. ■ Projection of the cervix more than 3 cm into the vaginal canal may indicate a pelvic or uterine mass. ■ Transverse or star-shaped cervical lacerations reflect trauma causing tearing of the cervix. ■ An enlarged cervix is associated with infection. ■ Nabothian cysts (small, white or yellow raised, round areas on the cervix) are considered normal but may become infected. ■ Cervical polyps may be cervical or endometrial in origin.
Palpate the cervix, uterus and ovaries. See the guidelines in Box 46.2. *The cervix can be moved slightly without discomfort. The uterus is normally at the level of the pubis, moves freely and is non-tender. The ovaries (about the size of a walnut) are firm, smooth, mobile and slightly tender on palpation. The ovaries are not usually palpable 3 to 5 years after menopause. A small amount of clear drainage is normal.*	■ The uterus may be retroverted (tilted backwards) or retroflexed (angled backwards). ■ Pain on movement of the cervix during manual examination suggests pelvic inflammatory disease (PID). ■ Softening of the uterine isthmus (Hegar's sign), softening of the cervix (Goodell's sign) and uterine enlargement may be objective signs of pregnancy. ■ Firm, irregular nodules that vary greatly with size and are continuous with the uterine surface are likely to be myomas (fibroids). ■ Unilateral or bilateral smooth, compressible adnexal masses are found in ovarian tumours. ■ Profuse menstrual bleeding is seen with endometrial polyps, dysfunctional uterine bleeding (DUB) and use of an intrauterine device. ■ Irregular bleeding may be associated with endometrial polyps, DUB, uterine or cervical carcinoma, or oral contraceptives. ■ Postmenopausal bleeding is seen with endometrial hyperplasia, oestrogen therapy and endometrial cancer.

BOX 46.1 Guidelines for intravaginal assessment and use of the vaginal speculum

The size of the speculum that is used for an internal examination of the female reproductive system depends on the age of the woman and the size of her vagina. Two types of specula are available. The Graves speculum, used most often for examinations of adult women, is available in lengths of 9 to 13 cm and widths of 2 to 4 cm. The Pederson speculum, which is narrower, may be used to examine adolescents or adult women who are virgins, who have never had a baby or who are postmenopausal with vaginal atrophy. The speculum should be warm. If cultures or smears are to be obtained, neither water nor gel should be used to warm or to lubricate the speculum.

If cells are to be taken for cytological studies, the woman should not douche, use vaginal medications or take a tub bath for 24 hours before the examination. Finally, the examination is usually deferred if the woman is menstruating or has a vaginal infection.

The general procedure is as follows:

1. Place the index and middle finger of one hand into the vagina, just inside the introitus, and press the fingers towards the rectum. Hold the speculum in the other hand.
2. Ask the woman to bear down and insert the closed blades of the speculum into the vagina at an oblique angle until the ends of the blades reach the fingertips (see the accompanying figure). Withdraw the fingers and rotate the speculum to a transverse position.
3. Continue to insert the speculum until it reaches the end of the vagina. Depress the lever of the speculum to open the blades. If the cervix is not in full view, try closing the blades, withdrawing the speculum about halfway and inserting it again at a more downward angle. When the cervix is in full view, fix the depressed lever to an open position.
4. Inspect the cervix. The normal cervix is pink and midline. Assess colour, position, size, projection into the vagina, surface and shape, and any discharge.

If a Pap smear to collect cervical cells for cytological studies is done, the following procedure may be used:

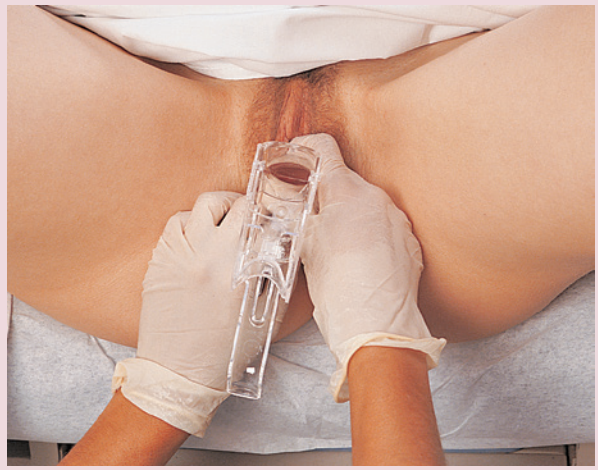
1. To collect cells from the vaginal pool, roll a sterile cotton-tipped applicator on the vaginal wall below the cervix. Paint the smear on the slide and spray the slide with fixative.

BOX 46.1 Guidelines for intravaginal assessment and use of the vaginal speculum (continued)

2. To collect endocervical cells, place the groove of the spatula snugly against the cervical os and rotate it 360 degrees. In a single stroke, spread the material from both sides of the spatula on a slide and immediately spray with fixative. Note: some clinics/practices may use a contour tip brush to collect endocervical cells instead of the spatula.

If cultures are to be done, take a specimen from the vagina and/or cervix with a sterile, cotton-tipped applicator and then either spread the specimen on a culture plate or place it in a culture container. Follow institutional protocols for preparing specimens for vaginal infections from suspected organisms.

At the end of the examination, loosen the lever control and slowly withdraw the speculum, closing the blades slowly and rotating the speculum while observing all areas of the vaginal wall. Assess the colour of the mucosa and the colour and appearance of any discharge.



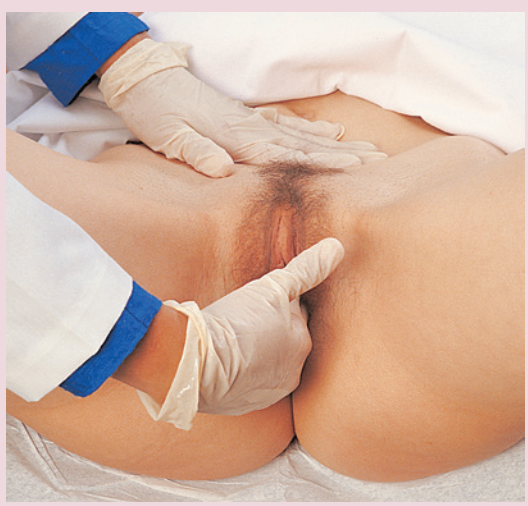
Inserting the vaginal speculum

BOX 46.2 Guidelines for bimanual pelvic examination

The bimanual pelvic examination is done to palpate the cervix, uterus and ovaries. The examiner's hand that will be used intravaginally and which is gloved is held with the index and middle fingers extended, the thumb abducted and the fourth and fifth fingers folded on the palm of the hand. The extended fingers are lubricated.

The general procedure is as follows:

1. Spread the labia with the thumb and finger of the opposite hand and insert the lubricated fingers into the vagina with the palm upwards.
2. Place the opposite hand on the abdomen; it is used to press on the abdomen and gently move the internal genitals towards the intravaginal fingers (see the accompanying figure).
3. Ask the woman to take deep breaths to relax the abdominal wall.
4. Palpate the cervix, assessing size, contour, position, surface, consistency, tenderness and mobility. The cervix should be freely movable and non-tender.
5. Palpate the uterus by pressing downwards on the abdomen while placing the intravaginal fingers in the anterior fornix and gently lifting against the abdominal hand. Assess the size, shape, surface, consistency, position, mobility and tenderness of the uterus. The normal uterus is freely movable and non-tender.
6. Palpate the adnexal areas, which surround the uterus and contain the fallopian tubes and ovaries. Because these structures are small, palpation may not be possible. If the ovaries are palpable, they should be smooth and firm. The normal ovary is sensitive to touch, firm and highly movable.
7. Withdraw the fingers. Provide tissues for the woman to wipe her genital area.



Bimanual pelvic examination

CONCEPT CHECK

1 In the male, sex hormones are called:
   1 androgens
   2 steroids
   3 prostaglandins
   4 testerones

2 When assessing the breasts of an obese man, the nurse may notice what physical change?
   1 phimosis
   2 gynaecomastia
   3 enlarged axillary nodes
   4 absent areola

3 You are assessing an 82-year-old woman. She tells you that she is suddenly having menstrual bleeding. What should you tell her?
   1 'This is normal, and sometimes happens even at your age.'
   2 'Do you have menstrual cramps with the bleeding?'
   3 'You need to see a doctor because you should not be bleeding.'
   4 'Are you sexually active?'

4 Which blood test may be used to monitor prostate cancer?
   1 PSA
   2 VDRL
   3 CBC
   4 WBC

5 Atrophy and sagging of breast tissue is associated with:
   1 poor nutrition
   2 smoking
   3 normal ageing
   4 breast cancer

6 Oogenesis is the process of oocyte development and continues until which biological event?
   1 birth
   2 adolescence
   3 menopause
   4 death

7 Cessation of menstruation in young women is a normal response to which biological event?
   1 implantation of an embryo
   2 onset of menopause
   3 onset of puberty
   4 beginning spermatogenesis

8 Which of the following diagnostic tests may be used to detect HPV?
   1 colposcopy
   2 mammogram
   3 culture
   4 Pap smear

9 Which assessment technique is primarily used to determine abnormalities of the breast?
   1 inspection
   2 auscultation
   3 palpation
   4 percussion

10 At which anatomical location would you palpate Bartholin's glands?
   1 above the clitoris
   2 posterior to the labia majora
   3 inferior to the urinary meatus
   4 internal vaginal wall

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CHAPTER 47

NURSING CARE OF MEN WITH REPRODUCTIVE SYSTEM AND BREAST DISORDERS

REBEKKAH MIDDLETON, PETER THOMAS

LEARNING OUTCOMES

- Explain the pathophysiology, manifestations, complications, interprofessional care and nursing care of men with disorders of the male reproductive system, including disorders of sexual function, the penis, the testes and scrotum, the prostate gland and the breast.
- Compare and contrast the risk factors for cancer of the penis, testes and prostate gland.
- Discuss the purposes, nursing implications and person-centred education required for medications and treatments used to treat disorders of sexual function, the penis, the testes and scrotum, the prostate gland and the breast.
- Describe the various surgical procedures used to treat men with disorders of the male reproductive system.

CLINICAL COMPETENCIES

- Assess functional health status of men with reproductive system and breast disorders, and monitor, document and report abnormal manifestations.
- Use evidence-based research to provide information and education to men having a radical prostatectomy.
- Determine nursing priorities, based on assessed data, to select and implement individualised nursing interventions for men with disorders of the reproductive system and breast.
- Administer or teach men how to knowledgeably and safely administer topical, oral and injectable medications used to treat disorders of the male reproductive system.
- Provide skilled care to men undergoing prostate surgery.
- Revise care plans as needed to provide effective interventions to promote, maintain or restore functional health status to men with disorders of the reproductive system and breast.

KEY TERMS

Men are subject to disorders of the penis, scrotum and testes, prostate gland and breast. These disorders may be inflammatory, structural, benign or malignant. Young men are at increased risk of testicular cancer (Russell, 2014). As men age, both benign and malignant conditions of the prostate gland become common. Many of the disorders pose significant risk to the man's fertility and sexual and urinary function, and some are life threatening. This chapter discusses disorders of the male reproductive system, including disorders of sexual expression and the male breast. Because many of the treatments and disorders of the male reproductive system have the potential to affect erection and ejaculation, these problems are discussed first.

DISORDERS OF MALE SEXUAL FUNCTION

THE MAN WITH ERECTILE DYSFUNCTION

Erectile dysfunction (ED) is the inability of the male to attain and maintain an erection sufficient to permit satisfactory sexual intercourse. **Impotence**, a term often used synonymously with erectile dysfunction, may involve a total inability to achieve erection, an inconsistent ability to achieve erection or the ability to sustain only brief erections. ED has many possible causes (see Table 47.1) and may or may not be associated with a loss of **libido** (sexual desire).

Erectile dysfunction is a common problem. In Australia, one in five men over the age of 40 experience significant erectile problems and about one in 10 men are completely unable to have erections (Andrology Australia, 2014). The incidence of the problem increases with age. Most problems with erection are the result of a disease, injury or chemical substance (such as prescribed medications, alcohol, nicotine, cocaine, barbiturates, amphetamines, methadone, opiates or marijuana) that decreases blood flow in the penis (WebMD, 2015). Because this is a problem primarily of ageing men, the discussion of pathophysiology focuses on this age group.

TABLE 47.1 Causes of erectile dysfunction

MAJOR PATHOLOGICAL CAUSES

Neurogenic
- Spinal cord injury
- Stroke
- Parkinson's disease
- Multiple sclerosis
- Epilepsy
- Alzheimer's disease
- Guillian–Barré syndrome

Endocrinological
- Diabetes mellitus
- Hypogonadism
- Hypothyroidism

Inflammatory
- Prostatitis
- Cystitis

Activity intolerance
- Pulmonary problems
- Anaemias
- Myocardial infarction
- Congestive heart failure
- Hepatic diseases
- Kidney failure

Substance dependency
- Alcohol
- Marijuana
- Narcotics
- Sedatives
- Tobacco

Arterial
- Atherosclerosis
- Hypertension
- Aortic aneurysm
- Sickle cell anaemia

Mechanical
- Decreased penile distensibility
- Congenital disorders
- Morbid obesity
- Hydrocoele
- Hip or pelvic fractures

Psychogenic
- Depression
- Stress
- Fatigue
- Fear of failure

Compulsive food disorders
- Compulsive overeating
- Anorexia nervosa
- Bulimia

MAJOR IATROGENIC CAUSES

Medications

Antihypertensives
- Hydrochlorothiazide
- Spironolactone
- Methyldopa
- Clonidine
- Prazosin
- Propranolol

Psychotropic agents
- Phenothiazines
- Butyrophenones
- Tricyclic antidepressants
- MAO inhibitors
- Diazepam

Endocrinological agents
- *Endocrinological agents*
- LHRH agonists
- Oestrogen compounds
- Progesterone

Other
- Antiparkinsonian agents
- Anticholinergic agents
- Immunosuppressive agents
- Antihistamines
- Non-steroidal anti-inflammatory drugs

Procedures and infections

Surgery
- Coronary artery bypass
- Pelvic lymphadenectomy
- Radical prostatectomy
- Radical cystectomy
- Abdominal perineal resection
- Sympathectomy
- Aortic aneurysm repair
- Transplant surgeries
- Spinal surgery

Other
- Severe healthcare-associated infection
- Radiation therapy to pelvis

Pathophysiology

Age-related changes in sexual function involve cellular and tissue changes in the penis, decreased sensory activity, hypogonadism and the effects of chronic illness. In the penis, a change from elastic collagen to a more rigid collagen results in decreased distensibility (a less rigid erection). This, in turn, interferes with the veno-occlusive mechanism, which prevents blood from 'leaking' out of the penis into the general vasculature prematurely. Problems with this mechanism result in incomplete erections. Vibrotactile sensation over the skin of the penis declines with age. This decline may explain why some older men require longer stimulation to achieve an erection. Hypogonadism, common in ageing men, results in decreased testosterone levels. There may be a relationship between lower androgen levels and erectile function.

Many illnesses affect erectile function. Damage to arteries, smooth muscles and fibrous tissues are the most common causes of impotence. Diseases such as diabetes, kidney disease, chronic alcohol misuse, liver disease, atherosclerosis, hypertension and vascular disease are responsible for organic ED. Innervation and blood flow to the penis may be damaged during surgery—prostate surgery, in particular, but also by surgery to the bladder, lower bowel and/or spine. Given the effects of ageing on the vasculature of the penis, the increased incidence of chronic illness and the multiple medications and treatments required to manage those illnesses, it is not surprising that many older men have problems with ED.

INTERPROFESSIONAL CARE

The management of men with ED is growing in importance and scale. Because the population as a whole is ageing, so the incidence is increasing proportionately. Another factor is the gradual change in the willingness of men and their partners to discuss sexual concerns. Although sexuality is still a very sensitive and private area for most people, the knowledge that help is available is causing men to seek answers. Many older men are coming to believe that loss of erectile function is not an inevitable part of ageing.

Diagnosis

The diagnostic tests that may be ordered for the man include blood studies, penile monitoring and penile blood flow.

Blood chemistry, testosterone, prolactin, thyroxin and prostate-specific antigen (PSA) levels are measured to identify metabolic and endocrine problems that may be causing the dysfunction. Nocturnal penile tumescence and rigidity (NPTR) monitoring helps differentiate between psychogenic and organic causes. These tests can be performed in a sleep laboratory, although home testing with portable devices is an alternative. The number and quality of erections occurring during REM sleep can be determined. Cavernosometry and cavernosography of the corpora are used to evaluate arterial inflow and venous outflow of blood in the penis.

Medications

ED can be treated with medications taken orally, injected directly into the penis or inserted into the urethra at the tip of the penis.

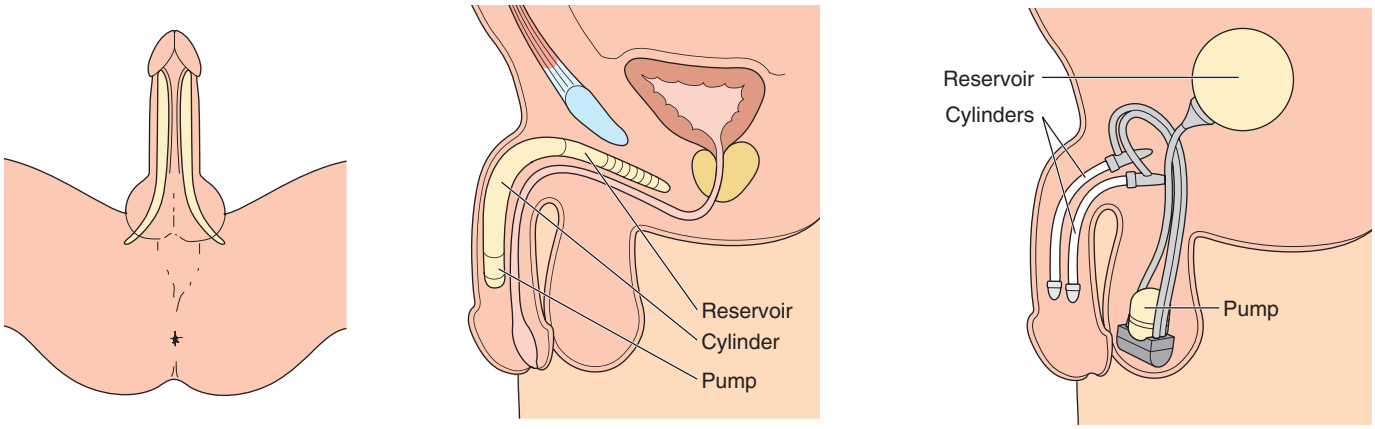
- *Oral medications:* oral medications used to treat ED include sildenafil citrate (Viagra), vardenafil hydrochloride (Levitra) or tadalafil (Cialis). Viagra and Levitra are taken an hour before sexual activity; they enhance the effects of nitrous oxide to facilitate relaxation of the smooth muscle in the penis during sexual stimulation to increase blood flow. Both drugs should be taken no more than once a day and should not be taken by men who are also taking nitrate-based drugs (for health problems) or alpha-blockers (used to treat hypertension and prostate enlargement). Cialis is a selective phosphodiesterase type-5 inhibitor that allows smooth muscle relaxation to facilitate inflow of blood into the penis. Its action lasts for 36 hours, but an erection only occurs with sexual stimulation. Cialis should not be taken if the man is also taking nitrates, alpha-blockers, erythromycin or rifampicin (antibiotics), ketoconazole or itraconazole (antifungals) or protease inhibitors (for HIV).
- *Injectable medications:* hormone replacement therapy with testosterone injections (250 mg IM every 3 weeks) or topical patches (Testogel) may be used for men with documented androgen deficiency and who do not have prostate cancer. Injectable medications, including papaverine and prostaglandin E injections (Alprostadil/Caverject), may be used. When injected directly into the penis, papaverine relaxes the arterioles and smooth muscles of the cavernosum, thus inducing tumescence (swelling). An erection usually develops that lasts from 30 minutes to 4 hours. Prostaglandin E functions much as papaverine does, but has fewer side effects. One problem with this treatment is its mode of delivery. There is a high attrition rate and men report dissatisfaction with lack of spontaneity, loss of interest in sex, physical limitations, cost and, occasionally, pain.

Mechanical devices

A frequently prescribed mechanical device for ED is the vacuum constriction device (VCD) (vacuum pump). The VCD draws blood into the penis with a vacuum, trapping it there with a constricting band at the base of the penis. After the device is removed for intercourse, a single small band, often called an O-ring, is left at the base of the penis to maintain the erection. If the man can attain an erection but cannot maintain it, an O-ring alone can be used.

Surgery

Surgical treatment for ED involves either revascularisation procedures or implantation of prosthetic devices. Venous or arterial procedures are generally not successful. The result is often temporary because the underlying cause of the vascular insufficiency is usually not corrected. Implantation of penile prostheses is now common (see Figure 47.1). Men are generally satisfied with their prostheses and they rank the inflatable type highest. Partners are also more likely to report satisfaction with the penile implant, although not to the same degree as men. Some partners report that the implanted penis is harder than a normal erect penis and therefore causes pain. Also, the man can have intercourse for a prolonged period of time and some partners do not find prolonged penetration enjoyable. Education for both the man and their partner is mandatory. Counselling by a sex therapist may be needed to facilitate adaptation to the implant.



A Semi-rigid **B** Self-contained **C** Inflatable

FIGURE 47.1 ■ Types of penile implants. *A*, With semi-rigid rods implanted in the corpora cavernosa, the penis is always in a state of semi-erection, which may not be acceptable to the man. *B*, With a self-contained penile implant, the penis remains flaccid until the man compresses a pump at the head of the penis, which transfers fluid from a reservoir to a cylinder within the penis to achieve an erection. The man presses a release valve to return the fluid to the reservoir. *C*, With an inflatable penile implant, the penis remains flaccid until the man compresses a pump in the scrotum, which transfers fluid from an abdominal reservoir to cylinders in the corpora cavernosa to achieve an erection. Pressing a release valve returns the fluid to the reservoir

Nursing care

Nurses in any healthcare setting may encounter men with ED, either through routine examinations or through careful assessment of men's conditions and treatments that may incidentally cause ED. Nurses employed in clinics, operating rooms and surgical units with urological services commonly encounter men being treated for ED. Nurses in a variety of settings, including long-term and residential care, encounter men who have had surgical interventions, such as penile implants.

Nursing diagnoses and interventions

Because nurses often complete the man's health history, they are most likely to discover problems of ED. (See the 'Functional health pattern interview' in Chapter 46 for appropriate questions to elicit information.) Once a problem is known, nurses are involved in giving information, providing emotional support and referring men to doctors or counsellors. Although there are many possible nursing interventions, this section focuses on nursing care related to sexual dysfunction and self-esteem.

Sexual dysfunction

Many men who lose erectile function are not aware of the cause. Often the man blames the loss on unrelated factors, such as age, a medication for an illness, a dangerous illness or his sexual partner. Not knowing causes anxiety, which may disrupt the relationship with his partner or lead him to discontinue an important medication.

- Assess for risk factors for ED. Be especially alert to men who have recently begun medications or had recent surgery that could cause ED. *Awareness of risk factors helps the nurse to prioritise care, although nurses must remember that almost all ageing men have at least one risk factor for ED.*
- Assess for sexual dysfunction. Men have shown increasing willingness and comfort to discuss sexual concerns, and expect nurses to be aware of the physiological effects of their disease and side effects of treatment on all aspects of their health. *If a problem exists, information obtained in a sexual assessment guides the nurse in deciding if the next step should be health education, referral or both.*
- Perform a detailed assessment of current sexual practices. *It is essential for healthcare providers to understand the man and his partner's sexual pattern in order to provide appropriate, individualised care.*
- Discuss previous methods of coping with ED. *Awareness of coping strategies can provide insight for the nurse and guide health education.*
- Provide information about treatment options. *The man needs to know the details of the intervention, the chances for success and the possible complications.*

CONSIDERATION FOR PRACTICE

Many men will not volunteer information about sexual function unless asked, but when the concept is raised, which should be done in a sensitive, non-judgmental but matter-of-fact way, are open about concerns and appreciate being asked.

Situational low self-esteem

The man with ED often believes himself to be 'less than a man'. In addition, the insertion of a penile implant with a semi-rigid prosthesis may result in disturbances in body image related to changes in sexual activity, as well as the appearance and embarrassment of a permanent semi-erection.

- Collect data during the health history, in a non-judgmental manner, about physiological function, other chronic illnesses and feelings about sexual inadequacy. *This information is necessary to establish the database for individualised interventions.*
- If the man has had a penile implant, teach him and his partner how to use the pump, including how to inflate and deflate the device. Suggest he practise inflation and deflation during the postoperative period. Suggest wearing snug-fitting underwear with the penis placed in an upright position on the abdomen, and loose trousers. Provide information about length of healing and that sexual activity may resume within 6 to 8 weeks post surgery. *Practising using the pump will maintain the pump position and promote tissue growth around the implant. The type of clothing worn can improve the ability to conceal a semi-rigid prosthesis and decrease embarrassment. Recovery from surgery is necessary before resuming sexual activity.*

Community-based care

Many nurses find that men with ED and their partners have lived in isolation with the problem for many years. The partner may even be unaware of the problem. The partner may believe that the man is seeing someone else or that he has lost his attraction to the partner. The man may have kept his problem a secret because an intense feeling of shame makes him unable to admit that he cannot perform sexually. Many men greet the information about the high incidence of ED with a sense of relief that they are not alone in having this problem. All men and their partners need to be aware of support services available to them. Referral sources include:

- Andrology Australia: www.andrologyaustralia.org
- Impotence Australia: www.impotenceaustralia.com.au/site/
- The Australian and New Zealand Infertility Counsellors Association: www.fertilitysociety.com.au/anzica/

THE MAN WITH EJACULATORY DYSFUNCTION

There are many types of ejaculatory dysfunction. **Retrograde ejaculation** (seminal fluid discharged into the bladder) may develop in ageing men but is usually related to treatment of prostate conditions or testicular cancer. *Premature ejaculation* is usually psychogenic in origin, although diabetes can cause the problem as well. *Delayed ejaculation* can be related to ageing changes, such as decreased vibrotactile sensation over the penis or decreased libido secondary to hypogonadism. Delayed ejaculation and inability to ejaculate at all may be caused by certain medications, such as antihypertensives, antidepressants, anxiolytics and narcotics.

Of these problems, premature ejaculation has proved most responsive to intervention. The man can experiment with ways to decrease sensitivity (such as wearing condoms). Using relaxation and guided imagery can delay orgasm. Mechanical devices, such as constrictive rings around the base of the penis, can help the man delay ejaculation and sustain an erection.

Nursing care focuses on assessment of the problem and health education for all types of ejaculatory dysfunction. The man's partner can be taught how to avoid excessive stimulation that would result in premature ejaculation. If the problem persists, the man should be referred to a specialist.

DISORDERS OF THE PENIS

THE MAN WITH PHIMOSIS OR PRIAPISM

Two less common disorders of the penis are phimosis and priapism. Although uncommon, these disorders can cause problems with urination and sexual activity. In some cases, they are considered a medical emergency because decreased blood flow to the penis may result in tissue ischaemia and necrosis.

Pathophysiology

Phimosis is constriction of the foreskin in uncircumcised men so that it cannot be retracted over the glans penis. Phimosis may be congenital or it may be related to chronic infections under the foreskin that lead to adhesions. The main problem with this condition is that it prevents adequate hygiene, which may lead to malignant changes of the penis. It also may interfere with urinary elimination and intercourse. In a related disorder, called *paraphimosis*, the foreskin is tight and constricted and is not able to cover the glans penis. The glans becomes engorged and oedematous and is painful. Paraphimosis may result from long-term retraction of the foreskin, such as occurs in placement of an indwelling catheter in the uncircumcised male (Grossman & Porth, 2013). The tight foreskin can result in ischaemia of the glans.

Priapism is an involuntary, sustained, painful erection that is not associated with sexual arousal. The prolonged erection may result in ischaemia and fibrosis of the erectile tissue with high risk of subsequent impotence (Grossman & Porth, 2013). The disorder, classified as either primary or secondary, is caused by impaired blood flow in the corpora cavernosa. Primary priapism results from conditions such as tumours, infection or trauma. Secondary priapism is caused by blood disorders (e.g. leukaemia, thalassaemia and thrombocytopenia), neurological disorders (e.g. spinal cord injury or stroke), kidney failure and some medications (see Box 47.1). Men who use intracavernous injection therapy or Cialis for ED are at risk of priapism.

BOX 47.1 Factors implicated in the aetiology of priapism

Illnesses/conditions
- Thalassaemia
- Metastatic cancer
- Leukaemia
- Spinal cord trauma

Drugs
- Papaverine
- Alcohol
- Psychotropic drugs
- Marijuana
- Warfarin
- Cocaine

INTERPROFESSIONAL CARE

Severe phimosis or paraphimosis may require surgical circumcision. If infection is present, the appropriate antibiotic is administered.

Treatment of priapism includes iced saline enemas, intravenous ketamine (Ketalar) administration to induce anaesthesia and spinal anaesthesia. Blood may be aspirated from the corpus through the dorsal glans, followed by catheterisation and pressure dressings to maintain decompression. If necessary, more aggressive surgery to create vascular shunts to maintain blood flow is performed. When priapism is prolonged, it increases the risk of subsequent ED.

Nursing care

Nursing care for men with priapism focuses on assessing the penis, monitoring urinary output and providing pain control. Assessment of the penis includes inspection for degree of erection and changes in colour due to ischaemia and palpation of the penis for firmness and degree of rigidity. Monitor urine output, assessing for oliguria or signs of acute urinary retention. Pain is treated with analgesics.

The man usually has moderate to severe anxiety related to pain, the treatment and the threat to his sexual function. The treatment may sound bizarre and painful, especially since the area is already extremely sensitive. The man may be acutely embarrassed by the erection and needs reassurance that the nurse understands that the erection is not within his control.

THE MAN WITH CANCER OF THE PENIS

Cancer of the penis is a rare cancer in Australia, occurring in 108 men in 2011 (Australian Institute of Health and Welfare (AIHW), 2015f). It most commonly affects men between the ages of 45 and 60. The cause is unknown. Penile cancer is rare in Jewish and Muslim men, populations in which routine circumcision is practised, although the correlation between circumcision and this cancer is unclear. Phimosis and poor genital hygiene are risk factors, as are human papillomavirus (HPV) and HIV infection. Ultraviolet light exposure (such as that used to treat psoriasis) also may play a role (Grossman & Porth, 2013).

Pathophysiology

Squamous cell carcinoma accounts for 95% of all penile cancers (Grossman & Porth, 2013). The tumour usually develops as a nodular or wart-like growth or a red velvety lesion on the glans or foreskin. The tumours tend to grow slowly. Penile cancer spreads to the superficial or deep inguinal nodes and very late in the disease may spread to the bone, liver or lungs. If the lesion is treated before inguinal node involvement, chances for a cure are good. Most of these lesions are painless but there may be significant ulceration and bleeding. Purulent, foul-smelling discharge may be evident under the foreskin. Occasionally, men with penile cancer may present with enlarged inguinal lymph nodes.

INTERPROFESSIONAL CARE

Cancer of the penis is diagnosed by a biopsy of the lesion, including any suspicious inguinal lymph nodes. The cancer is staged according to the size of the tumour, extent of invasion, status of inguinal lymph nodes and the presence or absence of distant metastasis. Small, localised lesions may be treated with fluorouracil cream, external-beam radiation, laser therapy or surgical excision. Larger lesions with superficial or deep infiltration of penile structures require partial or total amputation of the penis. Chemotherapy (discussed in Chapter 13) may be administered to men with distant metastasis.

Nursing care

Health education can help prevent this disease or provide early detection and intervention. Teach men about genital hygiene (including retraction of the foreskin if uncircumcised and washing the glans penis while bathing or showering) and the risks of unprotected sex, and encourage condom use. Encourage men to shield their genitals when having ultraviolet light therapy. Discuss the importance of seeking prompt treatment for any lesion or abnormal drainage noted on the penis.

If the man has a penile amputation (*penectomy*), nurses help cope with challenges associated with a shortened or absent

penis, including the potentially devastating effect on body image and self-concept. If a total penectomy is performed, the surgeon creates a perineal urethrostomy, preserving urinary continence. However, the man must void in the sitting position, reinforcing the feeling of loss. Dribbling of urine after voiding may be a problem for a few weeks. The man should be taught to perform careful perineal hygiene following surgery, using mild soap and water. Sitz baths may be helpful to relieve pain and to promote healing. If an inguinal lymph node dissection is performed, the man may experience persistent lymphoedema of the lower extremities.

DISORDERS OF THE TESTIS AND SCROTUM

THE MAN WITH A BENIGN SCROTAL MASS

Most scrotal masses are benign and can be managed in a manner that is satisfactory to the man. The most common are hydrocoeles, spermatocoeles and varicocoeles (see Figure 47.2).

Pathophysiology

A **hydrocoele**, the most common cause of scrotal swelling, is a collection of fluid within the tunica vaginalis. The swelling ranges from slightly larger than the testicle to larger than a grapefruit. The cause of chronic hydrocoele in men over the age of 40 years is an imbalance between production and reabsorption of fluid within the layers of the scrotum. Hydrocoeles also may occur secondary to trauma, infection or a tumour. A hydrocoele may be differentiated from a solid mass by transillumination or ultrasound of the scrotum. If the hydrocoele becomes large enough to cause embarrassment or significant pain, the fluid is aspirated and an agent is injected into the scrotal sac to sclerose the tunica vaginalis. Hydrocoeles are not associated with infertility.

A **spermatocoele** is a mobile, usually painless mass that forms when efferent ducts in the epididymis dilate and form a cyst. It is thought to result from leakage of sperm due to trauma or infection. Treatment is usually not necessary. Spermatocoeles are not associated with infertility.

A **varicocoele** is an abnormal dilation of a vein within the spermatic cord. It is caused by incompetent or congenitally missing valves that allow blood to pool in the spermatic cord veins. The dilated vein forms a soft mass that may be painful. Most varicocoeles occur on the left side after puberty. A major concern with this condition is that it can decrease blood flow through the testis, interfere with spermatogenesis and cause infertility. Varicocoeles can be felt by scrotal palpation. Scrotal ultrasound is also frequently used for diagnosis. If infertility is a concern, the spermatic vein may be ligated or occluded with a sclerosing agent or balloon catheter. If the varicocoele is small and infertility is not a concern, a scrotal support is recommended.

Nursing care

Nursing care focuses on reducing anxiety and providing health education about comfort measures. Almost all men are aware of the possible pain associated with scrotal manipulation. They need information and reassurance about pain management if surgical treatment is necessary. External bleeding is minimal after surgery; however, some men do develop scrotal haematomas, manifested by scrotal oedema and a purple discolouration.

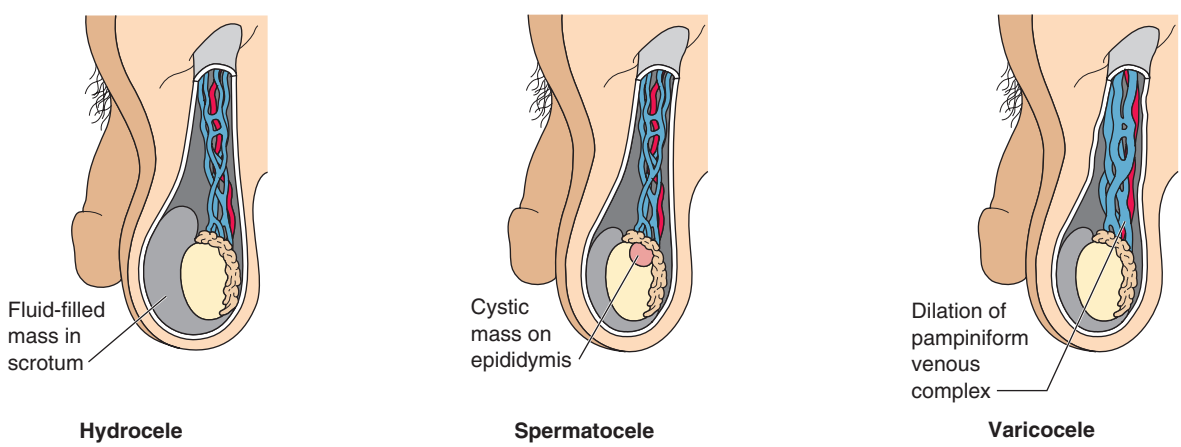


FIGURE 47.2 ■ Common disorders of the scrotum. Hydrocoeles and spermatocoeles do not usually require treatment unless they become large and cause pain. Varicocoeles are usually treated to prevent infertility

THE MAN WITH EPIDIDYMITIS

Epididymitis is an infection or inflammation of the epididymis, the structure that lies along the posterior border of the testis. This disorder is more often seen in sexually active men who are less than 35 years of age.

Sexually transmitted urethritis caused by *Chlamydia trachomatis* or *Neisseria gonorrhoeae* is the usual precipitating factor for epididymitis in younger men. Men who practise unprotected anal intercourse may acquire sexually transmitted epididymitis from *Escherichia coli*, *Haemophilus influenzae*, *Cryptococcus* or tuberculosis. In men older than age 35, epididymitis is usually associated with a urinary tract infection or prostatitis. Chemical epididymitis is associated with an inflammatory response to the reflux of urine into the ejaculatory ducts from urethral strictures, congenital structural anomalies or increased abdominal pressure from excessive heavy lifting. This type is usually self-limiting and does not require treatment.

Infectious epididymitis spreads by ascending the vas deferens from an already infected urethra or bladder. Early manifestations include pain and local oedema, which can progress to erythema and oedema of the entire scrotum, especially on the side of the involved epididymis. Complications of the disorder include abscess formation, infarction of the testis and infertility.

INTERPROFESSIONAL CARE

The infection is diagnosed with a specimen culture from a urethral swab or epididymal aspiration. Severe epididymitis may be treated with intravenous antibiotics and hospitalisation. Less acute forms of the disease are treated with antibiotic therapy. The man's sexual partner should also be treated with antibiotics if the causative organism is sexually transmitted.

Nursing care

Nursing care involves symptomatic relief and health education. Ice packs and a scrotal support may be applied to the scrotum to relieve pain. Ensure the man knows that complete resolution of the infection may take weeks to months and that treatment should continue until the infection is gone. Provide information about the possibility of infertility because the man may wish to seek evaluation for this problem at a later date.

THE MAN WITH ORCHITIS

Orchitis is an acute inflammation or infection of the testes. It most commonly occurs as a complication of a systemic illness or as an extension of epididymitis. Infection may reach the testes through the vas deferens and the lymphatic and vascular channels. Trauma, including vasectomy and other scrotal surgeries, may cause inflammation of the testes.

The most common infectious cause of orchitis in postpubertal men is mumps. Other causes include scarlet fever or pneumonia. The manifestations have a sudden onset, usually within 3 to 4 days after the swelling of the parotid glands. Manifestations include a high fever, increased WBCs and unilateral or bilateral scrotal redness, swelling and pain. If both testes are involved, permanent sterility may result, but this is rare (Grossman & Porth, 2013).

INTERPROFESSIONAL CARE

Treatment is supportive and symptomatic, including antibiotic therapy if urine cultures are positive. Bed rest, scrotal support and elevation, hot or cold compresses, and analgesics for pain are prescribed. If a hydrocoele occurs, it is aspirated. Nursing care is similar to that of the man with epididymitis and other scrotal disorders.

THE MAN WITH TESTICULAR TORSION

Testicular torsion, twisting of the spermatic cord with scrotal swelling and pain, is a potential medical emergency. The condition occurs most often in males between birth and age 20, but can occur at any age. Testicular torsion may occur spontaneously or it may follow trauma or physical exertion. The torsion of the arteries and veins decreases or stops testicular circulation, with resultant vascular engorgement and ischaemia.

Testicular torsion is usually diagnosed by history and physical examination. Testicular scanning may be used to determine if blood flow to the testicle is reduced, or a prostate ultrasound may be done to identify masses or torsion. Surgical treatment, which involves detorsion of the testicle and fixation to the scrotum, must begin as quickly as possible. If the testicle is necrotic or has sustained significant damage, an *orchidectomy* (surgical removal of a testis) is performed.

THE MAN WITH TESTICULAR CANCER

Testicular cancer accounts for only 1% of all cancers in men; however, it most commonly affects men between 15 and 50 years of age (Cancer Council South Australia, 2012). In 2011, an estimated 732 men in Australia were diagnosed with this cancer (AIHW, 2015c). Survival from testicular cancer has improved dramatically as a result of treatment with effective combination chemotherapy.

The cause of testicular cancer is unknown, but both congenital and acquired factors have been associated with tumour development. About 5% of cases develop in a man with a history of undescended testicle (*cryptorchidism*). Testicular cancer is more common on the right side, which parallels the incidence of cryptorchidism (Papadakis & McPhee, 2015).

Risk factors

Risk factors for testicular cancer are listed below:

- age
- cryptorchidism
- genetic predisposition, especially in identical twins and brothers
- Klinefelter's syndrome
- cancer of the other testicle
- other risk factors under investigation, including occupational risks, presence of multiple atypical naevi, HIV infection, cancer in situ, body size and maternal hormone use (American Cancer Society, 2012).

Pathophysiology

Approximately 95% of testicular malignancies are germ-cell tumours (Grossman & Porth, 2013). Germ-cell tumours are classified, depending on their origin and ability to differentiate, as seminomas and non-seminomas. Seminomas are the most common type and are believed to arise from the seminiferous epithelium of the testes. Non-seminomas contain more than one cell type; they include embryonal carcinoma, teratoma, choriocarcinoma and yolk cell carcinoma. The most common type in men ages 20 to 30 is embryonal carcinomas. Testicular cancer may also arise from specialised cells of the gonadal stroma. These tumours are named for the cells from which they originate: Leydig cell, Sertoli cell, granulosa cell and theca cell tumours.

Manifestations

The first sign of testicular cancer may be a slight enlargement of one testicle with some discomfort. The man may also have an abdominal ache and a feeling of heaviness in the scrotum. Local spread of the cancer to the epididymis or spermatic cord is inhibited by the outer covering of the testicles, the tunica albuginea. Therefore, spread by lymphatic and vascular channels to other organs often causes distant disease before large masses develop in the scrotum. Lymphatic dissemination usually leads to disease in retroperitoneal lymph nodes, whereas vascular dissemination can lead to metastasis in the lungs, bone or liver. Bilateral presentation of testicular cancer is unusual. Manifestations of testicular cancer are summarised in the box below. Manifestations of metastasis include lower extremity oedema, back pain, cough, haemoptysis or dizziness. Human chorionic gonadotropin (hGC)-producing tumours may cause breast enlargement (*gynaecomastia*).

MANIFESTATIONS Testicular cancer

COMMON
- Painless swelling on one testicle

OCCASIONAL
- Dull ache in pelvis or scrotum
- Painless nodule on one testicle

UNCOMMON
- Acute pain in scrotum

RARE
- Infertility
- Gynaecomastia

METASTATIC SYMPTOMS
- Neck mass
- Respiratory symptoms
- Gastrointestinal disturbance
- Lumbar back pain

INTERPROFESSIONAL CARE

Care focuses on diagnosis, elimination of the cancer and prevention or treatment of metastasis. Once testicular cancer is suspected, the man undergoes a number of screening tests to help identify the disease and its stage. If the disease is confined to the testicle, it is classified as stage I. Stage II disease is limited to the testicle and regional lymph nodes. Stage III disease involves metastasis above the diaphragm or extensive visceral involvement. Often, the man does not undergo biopsy before the beginning of treatment, but instead receives a definitive diagnosis after orchidectomy. Most men treated for testicular cancer will live a normal lifespan.

Diagnosis

Diagnosis may be made by various laboratory tests. Serum studies are done to identify tumour markers. Germ-cell tumours produce biochemical markers such as beta-hCG and alpha-fetoprotein (AFP) that can be measured using radioimmunoassay techniques. Elevated levels provide strong evidence of testicular cancer. These markers are also measured after surgery to help determine the presence of residual disease that remains undetected by other means. Persistent elevation may indicate the need for further therapy. Serum lactic acid dehydrogenase (LDH) levels are elevated in testicular cancer and may be significantly elevated when metastatic disease is present. LDH is a less specific indicator of testicular cancer than beta-hCG and AFP.

Medications

Progress in chemotherapy to treat testicular cancer is one of the chief reasons why most men survive the disease. The man who has an advanced disease receives platinum-based combination chemotherapy. Two frequently used combinations are: (1) cisplatin, bleomycin and etoposide (BEP); and (2) etoposide plus cisplatin (EP). Toxicity from the BEP regimen can be significant, with nausea, vomiting, hair loss, bone marrow suppression, nephrotoxicity, ototoxicity and peripheral neuropathy. Decreasing the number of BEP cycles to three (rather than four) or using the EP regimen reduces both the mortality and morbidity associated with chemotherapy. Chemotherapy is discussed in Chapter 13.

Surgery

Radical orchidectomy is the treatment used in all forms and stages of testicular cancer. A modified retroperitoneal lymph node dissection that preserves the nerves necessary for ejaculation often is performed at the same time.

Radiation therapy

Radiation therapy is used for stage I seminoma to treat cancer in the retroperitoneal lymph nodes, the most frequent site for distant metastasis. The man may experience temporary diarrhoea, nausea or a decline in bone marrow function, such as thrombocytopenia or leucopenia. These problems are usually mild and respond well to symptomatic treatment or time. Damage to the contralateral testicle is minimised by careful shielding. Pre-treatment and post-treatment analysis of sperm number and function is necessary. The most common long-term complication is dyspepsia or ulcer disease. Radiation therapy is discussed in Chapter 13.

Nursing care

Health promotion

Unfortunately, most men who develop testicular cancer do not have overt risk factors. Therefore, beginning at about the age of 15, all men should perform monthly testicular self-examination, as described in Box 47.2.

Nursing diagnoses and interventions

Nursing care of the man with testicular cancer is complex. The nurse must consider the reactions to the diagnosis, the change in body image accompanying treatment and sexual and reproductive issues. Although chances of a cure are excellent, the long-term effect on quality of life may be extensive, requiring a change in life goals.

Deficient knowledge

The nurse often initiates and reinforces health education about what to expect after radical orchidectomy. The man's knowledge about surgery is assessed and postoperative routines such as early ambulation are explained (see Chapter 3).

- Explain pain-control methods. In addition to the usual analgesics used to control postoperative incisional pain, ice bags may be applied to the scrotum. A scrotal support provides relief, especially when the man ambulates. *Surgery results in incisional pain and the scrotum is tender and slightly swollen.*

BOX 47.2 Testicular self-examination

- Examine your testicles when you are taking a warm shower or bath, or just after if you prefer to use a mirror to compare size.
- The scrotum, testicles and hands should be soapy to allow easy manipulation of the tissue.
- Gently roll each testicle between the thumb and fingers of each hand. If one testicle is substantially larger than the other or if you feel any hard lumps, consult your doctor immediately.
- Normal scrotal contents may be confusing. Just above and behind the testicle is the epididymis. It feels soft and tender overall, although parts of it may be rather firm. This is normal. The spermatic cord, a small, round, movable tube, extends up from the epididymis. It feels firm and smooth. Of greatest concern is any hard lump felt directly on the testicle, even if it is painless.
- Choose a day out of each month on which to examine yourself. Most men choose an easy day to remember, such as the first or last day of the month. Note this day on your calendar to help you remember.
- Start examinations young so that you become aware of what your testicles normally feel like. This way you will detect changes more readily.

- Educate the man about the manifestations of complications. The incision is closed with steri-strips or staples, and, although rare, wound dehiscence is possible. If the incision gapes open or if there is bleeding beyond slight oozing after 24 hours, the man should contact the surgeon. Another rare complication is a haematoma in the scrotum caused by bleeding from the spermatic cord stump. *Rapid onset of scrotal oedema is a sign of this problem. Because the man is usually discharged early, complications may not become apparent until he is at home.*

Ineffective sexuality patterns

The effect of testicular cancer and its treatment on sexual and reproductive function is varied. If the man has a retroperitoneal lymph node dissection, severing of the sympathetic plexus may result in retrograde ejaculation or failure to ejaculate. Infertility may be caused by ejaculation disorders, surgery, chemotherapy or radiation therapy.

- Assess the man's pre-diagnosis sexual function. To assess this, the nurse must establish an atmosphere of trust, openness and permission to discuss sexual concerns. After the initial shock of the diagnosis, men report intense concern about sexual and reproductive issues, which can be relieved only by information. *Knowledge of the man's usual sexual function can guide health education.*
- Discuss the possibility of preserving sperm in a bank prior to treatment. *This option may help relieve the man's fears about his ability to father children in the future, but must be completed prior to initiating treatment with surgery, chemotherapy or radiation therapy.*
- Help coping with feelings about altered sexual function and appearance. Explain that testicular implants can be inserted to preserve appearance. *Many men, regardless of whether they are in a significant relationship, deeply grieve the loss of the ability to father children. It is important to maintain body image despite disfiguring surgery.*

Community-based care

Families need to be included in health education for a variety of reasons. If the man is of reproductive age, his partner will have significant anxiety and will also require information. For the teenager, parents need information about the effect on sexual function and are often very involved in postoperative care. The man needs the support of the people he loves, and knowledgeable loved ones can give effective support.

Provide health education and reinforcement of the need for follow up, especially if the retroperitoneal lymph nodes were not surgically explored. For men with a risk of recurrence, surveillance with periodic physical examinations, chest x-ray films, tumour markers and computed tomography (CT) scans of the retroperitoneal nodes could continue for a minimum of 5 years, and possibly 10 years, after orchidectomy.

DISORDERS OF THE PROSTATE GLAND

THE MAN WITH PROSTATITIS

Prostatitis refers to different types of inflammatory disorders of the prostate gland. *Prostatodynia* is a condition where the man experiences the symptoms of prostatitis but shows no evidence of inflammation or infection. Manifestations of prostatitis and prostatodynia are summarised in the box below.

Pathophysiology and manifestations

The National Institutes of Health in the United States has defined four types of prostatitis. To gain a consistent approach to classification, Australia and New Zealand have adopted these definitions. The types are known as: acute bacterial prostatitis, chronic bacterial prostatitis, chronic prostatitis/pelvic pain syndrome and asymptomatic inflammatory prostatitis. Men with asymptomatic inflammatory prostatitis have no subjective symptoms, but are diagnosed when a biopsy or prostatic fluid examination is conducted.

Acute bacterial prostatitis

Acute bacterial prostatitis is most often caused by an ascending infection from the urethra or reflux of infected urine into the ducts of the prostate gland. *E. coli* is the most common causative organism; other causative organisms include *Pseudomonas, Klebsiella* and *Chlamydia*.

Symptoms of acute bacterial prostatitis include fever, malaise, muscle and joint pain, urinary frequency and urgency, dysuria and urethral discharge. The man often experiences dull, aching pain in the perineum, rectum or lower back. On rectal examination, the prostate is enlarged and painful.

Chronic bacterial prostatitis

Men with chronic bacterial prostatitis often present with a history of recurrent urinary tract infections. The causative organisms are most often *E. coli*, *Proteus* or *Klebsiella*. Calculi may form in the prostate and contribute to the chronicity of the problem.

The manifestations of chronic bacterial prostatitis include urinary frequency and urgency, dysuria, lower back pain and perineal discomfort. Epididymitis may be associated with the prostatitis.

Chronic prostatitis/chronic pelvic pain syndrome

This type of prostatitis is both the most common and the least understood of the syndromes (Yuan et al., 2014). The two types (inflammatory and non-inflammatory) are based on the presence of white blood cells in the prostatic fluid.

- *Inflammatory prostatitis* is believed to be an autoimmune disorder, but the actual cause is unknown. Men with this type of prostatitis have lower back pain; urinary manifestations; pain in the penis, testicles, scrotum, lower back and rectum; decreased libido; and painful ejaculations. They do not have bacteria in their urine, but have leukocytes in the urine and abnormal inflammatory cells in prostatic secretions.

MANIFESTATIONS Prostatitis and prostatodynia

ACUTE BACTERIAL PROSTATITIS
- Onset (may be abrupt): obstruction, irritation or pain upon voiding; frequency; and urgency
- Positive cultures of infectious organism
- Non-urinary symptoms: chills, fever, low back and pelvic floor pain

CHRONIC BACTERIAL PROSTATITIS
- Urinary symptoms sometimes similar to those of the acute form, except less sudden, less dramatic or even absent
- Positive cultures of causative organism not always obtainable

CHRONIC PROSTATITIS
- Perineal, suprapubic, lower back or genital pain
- Irritation upon voiding
- Post-ejaculatory pain
- Negative cultures of organisms

PROSTATODYNIA
- Pelvic, lower back or perineal pain
- Irritation or obstruction upon voiding
- No evidence of inflammation in the prostate
- No urinary tract infection
- Normal prostatic secretions

- *Non-inflammatory prostatitis* (prostatodynia) has manifestations that imitate those of inflammatory prostatitis, but no evidence of urinary or prostatic infection or inflammation can be found. The cause is not known, but is believed to be the result of a problem outside the prostate gland, such as obstruction of the bladder neck. Treatment is mainly symptomatic.

Asymptomatic inflammatory prostatitis

This type of prostatits is generally diagnosed during investigation of other genitourinary complaints. No symptoms are evident in the man. Leukocytes are found in the seminal fluid from the prostate.

INTERPROFESSIONAL CARE

Diagnosis

It is often difficult to diagnose prostatitis. Urine and prostatic secretion examination and cultures are obtained to determine the presence and type of blood cells and/or bacteria. X-ray studies and ultrasound may be useful to visualise pelvic structures.

Medications

Bacterial prostatitis is treated with antibiotics appropriate for the causative organism. Oral antibiotics are usually administered up to 4 weeks for acute bacterial prostatits, but men with the chronic form must take antibiotics for a much longer period. This can

be up to 4 months, and they may still relapse as soon as the antibiotic is discontinued. Non-bacterial prostatitis does not usually respond satisfactorily to drug therapy, although relief from symptoms is possible. Non-steroidal anti-inflammatory drugs are often useful for pain but it can be difficult to manage pain in chronic prostatitis where moderate relief may be obtained only. At times opioid analgesia may be necessary to control chronic pain but need careful monitoring and evaluation. Anticholinergics may assist in reducing voiding symptoms. Prostatodynia is treated symptomatically to relieve muscle tension, usually with alpha-adrenergic blocking agents or muscle relaxants.

Nursing care

Educating men with prostatitis focuses on symptom management. Men with acute and chronic bacterial prostatitis should be taught to increase fluid intake to approximately 3 L daily and to void every few hours. These measures help decrease irritation when voiding and assist in minimising the effects of increased fluid loss due to fever and infection. Management of fever is important in reducing fluid loss and minimising discomfort. Regular bowel movements help ease potential pain associated with defecation. It is important to educate the man on the need to complete the course of antibiotic therapy. Men with chronic prostatitis/chronic pelvic pain syndrome need to know that the condition is not contagious and does not cause cancer (Grossman & Porth, 2013). Referral sources for information include Andrology Australia <www.andrologyaustralia.org/> and HealthInsite <www.healthinsite.gov.au/>.

THE MAN WITH BENIGN PROSTATIC HYPERPLASIA

Benign prostatic hyperplasia (BPH), a non-malignant enlargement of the prostate gland, is a common disorder of the ageing male, thought to be associated with endocrine changes although its exact cause is unknown. The prostate, very small at birth, grows at puberty and reaches adult size around age 20. Almost one in four Australian men aged 40–49 years receive treatment for prostate problems, and this increases to three in every four men aged 70 years and older (Andrology Australia, 2015a). Some men remain asymptomatic even though their prostate may have begun to grow larger. The problem that brings men to a healthcare provider is usually the associated urinary dysfunction.

Risk factors

Although the exact cause of BPH is unknown, risk factors include:

- age
- family history
- risk factors similar to heart disease (i.e. obesity, high blood pressure, type 2 diabetes mellitus)
- diet high in meat and fats (Andrology Australia, 2015a).

Pathophysiology

The two necessary preconditions for BPH are age of 50 or greater and the presence of testes. Men who are castrated before puberty do not develop BPH. The androgen that mediates prostatic growth at all ages is dihydrotestosterone (DHT), which is formed in the prostate from testosterone. Although androgen levels decrease in ageing men, the ageing prostate appears to become more sensitive to available DHT. Oestrogen, produced in small amounts in men, appears to sensitise the prostate gland to the effects of DHT. Increasing oestrogen levels associated with ageing or a relative increase in oestrogen related to testosterone levels may contribute to prostatic hyperplasia.

FAST FACTS

Urinary problems with BPH:

- A hesitant, interrupted weak stream.
- Urgency with leaking or dribbling of urine.
- More frequent urination in small amounts, especially at night (nocturia).

BPH begins as small nodules in the periurethral glands, which are the inner layers of the prostate. The prostate enlarges through formation and growth of nodules (hyperplasia) and enlargement of glandular cells (hypertrophy). These changes occur over a long period of time. The pathophysiological effects result from a combination of factors, including urethral resistance to the effects of BPH, intravesical pressure during voiding, detrusor muscle strength, neurological functioning and general physical health.

Manifestations

The expanding prostatic tissue compresses the urethra (see Figure 47.3) and causes partial or complete obstruction of the outflow of urine from the urinary bladder. The detrusor muscles hypertrophy to compensate for increased resistance to urinary flow; however, eventually decreased bladder compliance and bladder instability result.

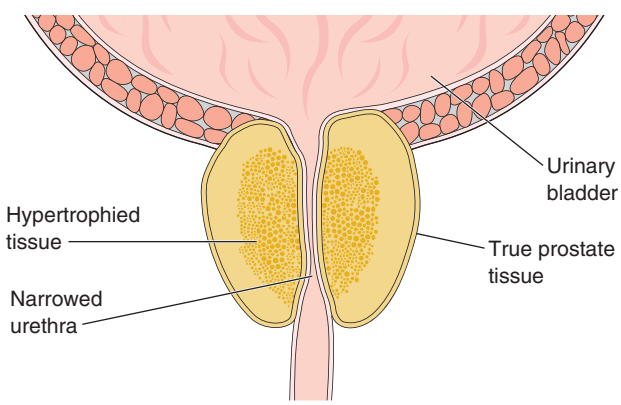


FIGURE 47.3 ■ Benign prostatic hyperplasia

MANIFESTATIONS Benign prostatic hyperplasia

- Diminished force of urinary stream
- Hesitancy in initiating voiding
- Post-void dribbling
- Sensation of incomplete emptying
- Urinary retention
- Nocturia
- Frequency
- Urgency
- Urge incontinence
- Dysuria

As a result, the man with BPH has manifestations from obstruction (decreased force of stream, increased time to initiate and complete void, hesitancy, incomplete bladder emptying and post-void dribbling) and irritation (frequency, urgency, incontinence, nocturia, dysuria and bladder pain). Urinary retention may become chronic, resulting in overflow incontinence with any increase in intra-abdominal pressure. There is little correlation between the size of the prostate gland and the urinary manifestations. Manifestations of BPH are summarised in the box above.

Complications

Unless the enlarging mass is reduced, multiple complications may occur. Acute urinary retention is quite common in men with BPH (van Vuuren, Heyns & Zarrabi, 2011; Eifler, 2014). As urine is retained in the bladder, increasing bladder distension occurs. Diverticula (outpouchings) on the bladder wall result from the distension. The distension may also obstruct the ureters. Infection, more common in retained urine and in diverticula, may ascend from the bladder to the kidneys. Hydroureter, hydronephrosis and renal insufficiency are possible complications. Renal calculi risk is increased in men with BPH, due to the alkalisation of the retained or residual urine.

INTERPROFESSIONAL CARE

Care of men with BPH focuses on diagnosing the disorder, correcting or minimising the urinary obstruction and preventing or treating complications. There is no way to reverse BPH. Treatment is often determined by the severity of the manifestations and the presence of complications. Mild cases are often monitored over time, and may remain stable or improve.

Diagnosis

A diagnosis of BPH involves both physical examination and laboratory tests not only to diagnose the disease but also to differentiate it from prostate cancer. A digital rectal examination (DRE) is done to examine the external surface of the prostate; in BPH it is asymmetrical and enlarged. Examination of the creatinine levels of the blood is conducted to assess for kidney damage.

The urine is examined for WBCs, RBCs and bacteria. Urinary function is assessed by measuring residual urine (amount of urine remaining in the bladder after voiding) with ultrasonography or post-voiding catheterisation (more than 100 mL is considered high) and through uroflowmetry, which measures urine flow rate. Normal is greater than 14 mL/sec. A finding of less than 10 mL/sec indicates obstruction.

PSA levels are obtained to rule out prostate cancer. PSA may be slightly elevated, but cannot be used to determine BPH. PSA is a glycoprotein produced only in the cytoplasm of benign and malignant prostate cells; the serum level corresponds with the volume of both benign and malignant prostate tissue. Further information is provided in the next section under diagnosis of prostate cancer.

In addition, the man's own subjective experiences with BPH are included in the diagnosis and treatment. For example, the International Prostate Symptom Score uses a scale of 0 (not at all) to 5 (almost always) to collect data about areas such as feeling as though the bladder did not empty with urinating, the need to urinate within 2 hours after urinating, starting and stopping the stream several times while urinating and straining to urinate. This questionnaire also asks how many times during the night the man gets up to urinate and how he feels about having the disorder.

Medications

Treatment with medications is based on two considerations: the hyperplastic tissue is androgen dependent and smooth muscle contraction within the prostate can exacerbate urinary obstruction. The first consideration is usually addressed by treatment for mild prostate enlargement with an anti-androgen agent such as finasteride (Proscar) that inhibits the conversion of testosterone to DHT and causes the enlarged prostate to shrink in size. These agents may cause impotence, decreased libido and decreased volume of ejaculate. Individual and family education includes the information that crushed tablets should not be handled by pregnant women because the drug may be absorbed through the skin and be harmful to a male foetus.

Excessive smooth muscle contraction in BPH may be blocked with the alpha-adrenergic antagonists such as terazosin (Hytrin) and tamsulosin (Flomaxtra). These medications relieve obstruction and increase the flow of urine. They may cause orthostatic hypotension. Individual and family education includes advice about making position changes slowly to avoid dizziness and accidental falls, how to take and record blood pressure, and to check with the healthcare provider before taking any medication for coughs, colds or allergies (because these over-the-counter medications may contain an adrenergic agent).

Surgery

Men who have urinary retention, recurrent urinary tract infection, haematuria, renal calculi or renal insufficiency secondary to BPH are candidates for surgical intervention. Surgical treatment may be performed by minimally invasive surgery or through transurethral surgery, open surgery or by laser surgery.

MINIMALLY INVASIVE SURGERY Because medications are not effective for all men, a number of procedures have been developed to relieve the manifestations of BPH that are less invasive than traditional surgery.

Transurethral microwave thermotherapy uses a transurethral probe to deliver microwaves directly to the prostate. It uses heat to destroy excess prostate tissue. During the procedure, a cooling system protects the urinary tract. The procedure takes about an

hour and can be performed on an outpatient basis. Although microwave procedures do not cure BPH, they do reduce urinary manifestations. The procedures do not cause impotence or incontinence.

The *transurethral needle ablation* (TUNA) system uses low-level radio frequency through twin needles to burn away a region of the enlarged prostate. Shields protect the urethra. TUNA improves the flow of urine through the urethra. It does not cause impotence or incontinence.

TRANSURETHRAL SURGERY A *transurethral resection of the prostate (TURP)* is the most common surgical procedure used. Obstructing prostate tissue is removed using the wire loop of a resectoscope and electrocautery, inserted through the urethra (see Figure 47.4). No external incision is necessary. During the procedure a resectoscope is used to remove obstructing tissue one piece at a time. The tissue is flushed into the bladder with fluid and then flushed out at the end of the operation. This surgery has potential risks, however, including postoperative haemorrhage or clot retention, inability to void and urinary tract infection. Other possible complications are incontinence, impotence and retrograde ejaculation.

In the *transurethral incision of the prostate* (TUIP) procedure, small incisions are made in the smooth muscle where the prostate is attached to the bladder. The gland is split to reduce pressure on the urethra. No tissue is removed, so this procedure is most appropriate for men with smaller prostate glands. TUIP can be done on an outpatient basis and has the additional advantage of less risk of postoperative retrograde ejaculation than is associated with TURP or other prostatectomy procedures.

LASER SURGERY Another minimally invasive procedure used to treat BPH is laser therapy. In laser surgery, a laser beam is delivered via a cystoscope transurethrally to cut, coagulate and vaporise excessive prostatic tissue with several short bursts of energy. An advantage of laser surgery is decreased blood loss and a more rapid recovery time. However, this method may not be as effective for larger prostates.

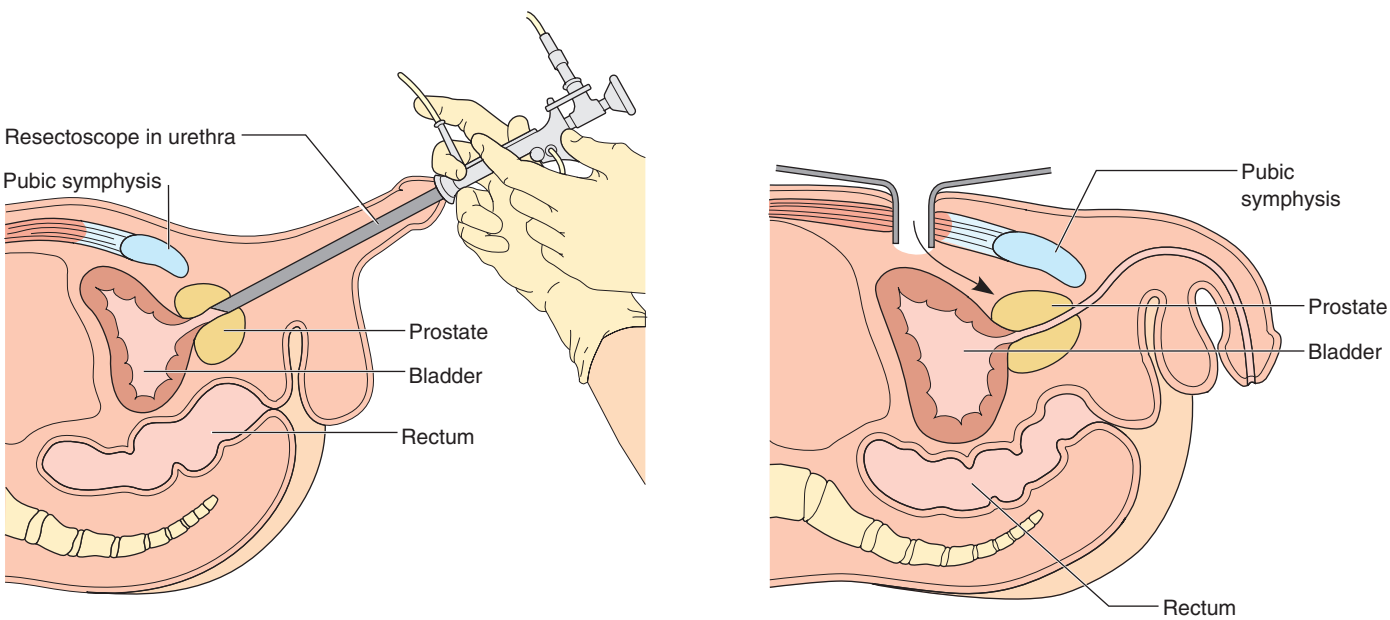
OPEN SURGERY When the prostate gland is very large, an open prostatectomy may be used. These procedures are discussed in the section on prostate cancer that follows. Nursing care for the man having prostate surgery (prostatectomy) is outlined in the box below.

New treatments

Newer treatments for BPH include minimally invasive procedures such as balloon urethroplasty and placement of intraurethral stents to maintain patency of the urethra. Balloon urethroplasty is a simple procedure in which a balloon-tipped catheter is inserted into the narrowed portion of the urethra and inflated. Inflation of the balloon widens the urethra, relieving obstruction. These procedures can be done as outpatient surgery.

Alternative and complementary therapies

Phytotherapy is the use of plants or plant extracts for medical treatment. Several plant extracts have been used for years in Europe to treat BPH, including *Serenoa* (saw palmetto berry), the bark of *Pygeum africanum*, the roots of *Echinacea*, *purpurea* and *Hypoxis rooperi* and the leaves of the trembling poplar. The mechanism of action of these extracts is unknown, but men report they are effective in relieving manifestations (Papadakis & McPhee, 2015). A Cochrane review has found that *Serenoa* has no clinical benefit over placebo (Tacklind et al., 2011).



A Transurethral resection of the prostate

B Retropubic prostatectomy

FIGURE 47.4 ■ *A*, In a transurethral resection of the prostate, a resectoscope inserted through the urethra is used to remove excess prostate tissue. *B*, In a retropubic prostatectomy, prostate tissue is removed through an abdominal incision

NURSING CARE OF THE MAN having a prostatectomy

PREOPERATIVE CARE

- Assess the man's and family's knowledge about the surgery. *Some men are confused about the surgical approach because there are several, quite different methods.*
- Inform the man that he will have a urinary catheter when he returns from surgery and he may have a drain(s) in his incision. He also will be wearing sequential pneumatic compression stockings. *This knowledge can reduce anxiety postoperatively and increase cooperation with postoperative care.*
- Ensure that a signed consent form is in the chart and that all other preoperative tasks outlined in Chapter 3 are completed.
- Bowel preparation with oral neomycin sulfate (Neosulf) may be ordered. *This cleanses the bowel if a perineal approach will be used.*
- Communicate willingness to address any concerns or anxiety. *Men may be anxious about the outcome of their surgery and potential long-term effects of the surgery on their sexuality. When a prostatectomy is performed for prostate cancer, additional fears include the extent of the cancer and surgery, chances for cure and possible end-of-life issues.*

POSTOPERATIVE CARE

- Maintain the usual postoperative assessments (see Chapter 3) and follow aseptic techniques in urinary drainage and irrigation care. Monitor vital signs closely for the first 24 hours and regularly thereafter. *The man who has had prostate surgery is at risk of haemorrhage and infection. Vital sign changes may be early manifestations and can assist in recognising deterioration.*
- Maintain an accurate fluid balance chart, including amounts of irrigating solution used. Frequently assess patency of any catheters and drains. Monitor colour and character of urine. *Catheters may become occluded by blood clots or kinks, interfering with urinary drainage and increasing the risk of haemorrhage.*
- Assess and manage the man's pain. *The man may have at least three types of pain: incisional pain, bladder spasms and abdominal cramps due to intestinal gas. Analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) are administered on a routine and prn basis to control incisional pain. Bladder spasms may be accompanied by strong urges to void and urine leakage around the catheter. Oral oxybutynin hydrochloride may be used to relieve bladder spasms.*
- Maintain anti-embolic stockings and pneumatic compression devices as ordered. Assist with leg exercises and ambulation as ordered. *The man who has had prostate surgery is at risk of developing thromboemboli; these are important preventive measures.*
- Encourage the man to maintain a liberal fluid intake of 2 to 3 L a day. *Increased fluids reduce burning on urination after catheter removal and the risk of urinary tract infection.*

THE MAN WITH A TRANSURETHRAL RESECTION OF THE PROSTATE (TURP)

- For the first 24 to 47 hours, monitor for haemorrhage, evidenced by frankly bloody urinary output, presence of large blood clots, decreased urinary output, increasing bladder spasms, decreased haemoglobin and haematocrit, tachycardia and hypotension. Notify the doctor if any of these manifestations occur. *Postoperative haemorrhage may be either arterial or venous and may be precipitated by movement, bladder spasms or an obstructed urinary drainage system.*
- Instruct the man with a three-way indwelling catheter with traction to keep his leg straight while the traction is applied. An 18 Fr. to 22 Fr. three-way catheter with a 30 to 45 mL balloon usually is inserted following a TURP. *The inflated balloon is pulled down into the prostatic fossa and the catheter tubing is pulled down and taped to the man's leg to apply pressure against the operative site, preventing bleeding.*
- Explain that the presence of a urinary catheter will cause the sensation of needing to void, but it is important not to strain to try to void around the catheter or when having a bowel movement. Explain that bladder spasms—experienced as lower abdominal pressure or pain—and a desire to urinate may occur. Ensure that the man understands that this is an expected sensation and that medications can help alleviate this discomfort. *Pressure on the urethra by the large catheter and on the internal sphincter by the catheter's balloon stimulate the micturition reflex. Straining to void or to have a bowel movement may stimulate bladder spasms and increase pain; it also may increase the risk of bleeding. Administer pain medications at regular intervals.*
- If the man has continuous bladder irrigation (CBI), assess the catheter and the drainage tubing at regular intervals. Maintain the rate of flow of irrigating fluid to keep the output light pink or colourless. Assess the urinary output every 1 to 2 hours for colour, consistency, amount and presence of blood clots; assess for bladder spasms. *CBI is used to prevent the formation of blood clots which could obstruct urinary output. Bladder distension resulting from output obstruction increases the risk of bleeding. Irrigating fluids are continuously infused and drained at a rate to keep urine light pink or colourless. Indicators of obstruction and bleeding are: urine has frank blood; urine contains multiple blood clots, decreased urinary output, or the man has bladder spasms.*
- Assess for fluid volume excess and hyponatraemia, called TURP syndrome, manifested by hyponatraemia, decreased haematocrit, hypertension, bradycardia, nausea and confusion. If these manifestations occur, notify the doctor. *TURP syndrome results from the absorption of irrigating fluids during and after surgery. Untreated, it may result in arrhythmias, seizures or both.*
- If the man does not have CBI, follow organisational policy and protocols for irrigating the indwelling catheter (usually when the urine contains frank blood or has numerous larger blood clots, or when bladder spasms increase). In most instances, using sterile technique, the catheter is gently irrigated with 50 mL of irrigating solution at a time, until the obstruction is relieved or the urine is clear. Ensure equal input and output of irrigating fluid. *Intermittent irrigation may be used to prevent obstruction of urinary drainage.*

(continued)

NURSING CARE OF THE MAN having a prostatectomy (continued)

- Following catheter removal, assess the amount, colour and consistency of urine. Explain to the man that he may experience burning on urination, that dribbling after urination is a common experience and that the urine may contain small blood clots after catheter removal. *The CBI and catheter usually are removed in the 24 to 48 hours following surgery. Urinary control may be improved by teaching the man to start and stop the urine stream several times during each voiding and by practising pelvic floor exercises. Regaining full control may take up to 1 year.*

THE MAN WITH A RETROPUBIC PROSTATECTOMY

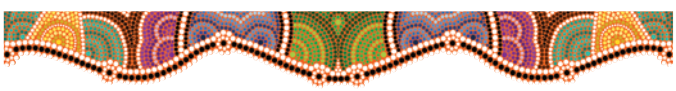
- Assess the abdominal incision for the presence of urine. *Because the bladder is not entered during a retropubic prostatectomy, no urine should be found on the dressing.*
- Assess the abdominal incision for increased or purulent drainage and the man for an increased temperature and pain. *These manifestations indicate the presence of infection.*

THE MAN WITH A SUPRAPUBIC PROSTATECTOMY

- Assess urinary output from both the suprapubic and the urethral catheters. *The man with a suprapubic prostatectomy often has two separate closed drainage systems: one from the suprapubic incision and one from a urethral catheter.*
- Assess the abdominal dressing for urinary drainage and change saturated dressings frequently. Consult with a wound care specialist if necessary. *Urine is highly irritating to the skin.*
- Following removal of the urethral catheter (usually 2 to 4 days after surgery) and based on protocols, clamp the suprapubic catheter and encourage the man to void. Assess residual urine by unclamping the suprapubic catheter and measuring urinary output after voiding. *If residual urine is 75 mL or less with several voidings, the suprapubic catheter is removed.*

THE MAN WITH A PERINEAL PROSTATECTOMY

- Assess perineal incision for drainage and manifestations of infection. *Location of the incision in the perineum increases the risk of infection.*
- Do not take rectal temperatures or administer enemas. *Insertion of a thermometer or enema tubing into the rectum may precipitate bleeding.*
- Use a T-binder or padded scrotal support to hold the dressing in place. Following removal of the dressing and perineal sutures, heat lamps or sitz baths may be used. *The location of the dressing makes application difficult: heat lamps or sitz baths provide heat and promote healing.*
- Teach the man to perform perineal irrigations with sterile normal saline as ordered and after each bowel movement. *Because of the proximity of the incision to the anus, special wound care is necessary to prevent infection.*



Nursing care

Most men are unsure of the function of the prostate gland and even of the prostate's exact location, though its relationship to sexual and urinary function is at least generally known. This lack of knowledge, coupled with the growing number of treatment options, is confusing to many men. There are many similarities between the nursing care of men with BPH and that of men with prostate cancer (see the prostate cancer section that follows).

Nursing interventions

This section provides interventions related to deficient knowledge, urinary retention, risk of infection and risk of imbalanced fluid volume.

Deficient knowledge

- Explain the anatomy and physiology of the prostate gland, as well as normal changes that occur with ageing. *Men must know about their bodies in order to make accurate decisions about treatment.*
- Discuss treatment options, including information about effects on erectile function, ejaculation and fertility. *Counsel the man to discuss specific concerns with his urologist. Many different treatment options are available; the choice should be a mutual decision between the man, his partner and the urologist.*
- Discuss effects of prostate surgery, including urinary retention and urinary incontinence. *These common transient postoperative effects are related to the surgical procedure and the postoperative indwelling catheter.*
- Explain to the man having a TURP that a catheter will be placed into the bladder, with the tubing taped to his inner thigh, and that irrigation fluid will be infusing into and out of the catheter for the first 36 to 72 hours following surgery. *The catheter and irrigation are necessary to remove blood clots from the bladder and allow drainage of urine. Gentle traction is applied to the catheter to apply pressure to the operative site (prostatic fossa) and prevent excessive bleeding.*
- Explain that, following removal of the catheter, he will most likely have urinary frequency and urgency. He may also experience dribbling of urine after voiding. Stress the importance of increasing oral fluid intake and regular pelvic floor exercises. *Urinary manifestations are related to the surgical procedure and the indwelling catheter. Increased fluid intake helps decrease dysuria. Pelvic floor exercises strengthen periurethral muscles and decrease post-voiding urine leakage.*

Urinary retention

- Educate the man about the manifestations of acute urinary retention: dysuria, overflow incontinence, bladder pain and

distension, no urine output. *Acute urinary retention is a potential complication of BPH, requiring immediate medical attention.*

- Provide health education about how the risk of developing urinary retention increases when the man with BPH takes over-the-counter (OTC) decongestant medications or prescription medications such as antidepressants, anticholinergics, calcium channel blockers, antipsychotics and medications to treat Parkinson's disease. *OTC decongestants may contain alpha-adrenergic agonists that increase smooth muscle tone of the prostate, bladder neck and proximal urethra. The prescribed medications may relax detrusor muscle contractions. Both actions may increase the risk of urinary retention (Keller et al., 2012; Barkin, 2011).*
- Suggest avoiding intake of large volumes of liquid at any one time. *A single intake of a large volume of liquid results in rapid bladder filling and increases the risk of urinary retention.*
- Teach how to use the double-voiding technique: urinate, then sit on the toilet for 3 to 5 minutes, then urinate again. *This technique may relieve mild to moderate urinary retention.*

CONSIDERATION FOR PRACTICE

In addition to avoiding a large amount of fluids at one time, it is also important to teach the man to limit liquids that stimulate voiding, such as coffee, caffeine-containing beverages and alcoholic beverages.

Risk of infection

- Monitor WBC and vital signs. *Infection is indicated by an increase in WBCs, body temperature and pulse rate.*
- Maintain sterile procedures when changing irrigation fluids and emptying Foley catheter draining bag. *Sterile procedures are necessary to prevent infection.*

Risk of imbalanced fluid volume

A prostatectomy brings increased risk of imbalanced fluid volume as a result of excessive bleeding from the operative site (prostatic fossa) as well as absorption of irrigating fluid. Report manifestations indicating hypovolaemic shock, excess bleeding and/or TURP syndrome immediately.

- Monitor pulse and blood pressure. *Manifestations of hypovolaemic shock include an increasing pulse and a decreasing blood pressure.*
- Monitor colour of drainage in urinary drainage bag (see Table 47.2). *The appearance of dark or red-coloured urine and irrigation fluid in the urinary drainage bag is an excellent indicator of bleeding after a prostatectomy.*
- Monitor for manifestations of transurethral resection (TURP) syndrome: nausea and vomiting, confusion, hypertension, bradycardia and visual disturbances. *The absorption of isotonic bladder irrigating fluids during and after surgery may cause this hypervolaemic, hyponatraemic state. Treatment includes diuresis and, in severe cases, hypertonic saline administration (Papadakis & McPhee, 2015).*

TABLE 47.2 Significance of character of urine after prostatectomy and related nursing care

URINE COLOUR	NURSING IMPLICATIONS
Light red to red	Normal on day of surgery and first postoperative day.
Very dark red	May indicate increased venous bleeding or inadequate dilution. Catheter at risk of occlusion. Increase flow rate of irrigant. If urine does not clear, notify doctor.
Bright red	May indicate arterial bleeding. Increase flow rate of irrigant, monitor vital signs and notify doctor.
Contains blood clots	Occasional blood clot normal. If clots are frequent, catheter may become obstructed. Increase flow rate of irrigant.
Clear to light pink	Normal throughout hospitalisation.

Community-based care

Depending on the man's choice of treatment, the procedure may be performed on an outpatient basis. The man having a TURP, although hospitalised for the surgery, may be discharged quite quickly after surgery if there are no complications. Discharge instructions after prostate surgery are provided in the 'Meeting individualised needs' box below. Home care often involves care of an indwelling urinary catheter. The nurse should educate the man how to care for the catheter and drainage bag, including the following information:

- Change from the daytime leg drainage bag to a larger night drainage bag. A larger bag suspended from the bed frame at night permits gravity drainage of urine and prevents reflux of urine back into the bladder.
- Avoid strapping the leg bag on too tightly, which can decrease venous return and increase risk of thrombophlebitis and embolic complications such as pulmonary emboli.
- Place a soft cloth between the leg bag and thigh to decrease friction and absorb dampness under the bag, reducing the risk of skin irritation.
- Empty the leg bag every 3 to 4 hours during waking hours to prevent overfilling.
- Promptly report any unexpected changes in urine colour, consistency or odour, haematuria, evidence of frank bleeding or large blood clots, as well as a lack of or significant decrease in urine output.

THE MAN WITH PROSTATE CANCER

Cancer of the prostate is the most common form of cancer present in Australian men over 50 (excluding some forms of skin cancer), with over 19 000 men diagnosed annually, accounting for one-third of all new diagnoses of cancer in

MEETING INDIVIDUALISED NEEDS **Discharge instructions for men after prostate surgery**

Activity
The healing period lasts from 4 to 8 weeks. Avoid strenuous activity and heavy lifting. Do not drive for 2 weeks. Take long walks; take stairs slowly and carefully. Continue exercises that you did in the hospital to prevent blood clots in the legs. You can take showers; avoid baths while the catheter is in place.

Bleeding
Bleeding can occur any time after surgery. It is fairly common after a bowel movement, coughing or increased exercise. If you notice blood in the urine, increase fluids and rest until the urine is clear. If heavy bleeding blocks the catheter, call the doctor immediately. Avoid aspirin and NSAIDs (e.g. Nurofen) for at least 2 weeks.

Bowel movements
Keep bowel movements regular and soft to avoid pressure on the prostate area. Drink fruit juices and take mild laxatives or stool softeners as ordered.

Diet
Resume your normal diet. Increase fluids to 10 glasses daily. Avoid alcohol unless otherwise advised by your doctor.

Sexual intercourse
Do not have sex for 6 weeks after surgery to avoid bleeding. You may still have erections even with the catheter in place. When you resume sex, ejaculate flows back into the bladder, so you will express little or no semen.

Urination
After your catheter is removed, you may experience some burning, stinging or leakage for several weeks and you may pass small blood clots occasionally. These symptoms will disappear as the area heals. It is best to use pads to control leakage.

Work
If work is not strenuous, you may return in 4 weeks; otherwise, wait 6 to 8 weeks.

Please call immediately if:
- You are unable to urinate.
- Bleeding is not controlled by fluids and rest, or is excessive.
- You have chills and fever or severe abdominal pain.
- Your scrotum becomes swollen and tender.
- You have pain in one calf, chest pain or difficulty breathing.

men (AIHW, 2015b). It is the second most common cause of deaths from cancer in men, with one in nine men developing prostate cancer in their lifetime (Prostate Cancer Foundation of Australia (PCFA) 2015). Men living in rural and regional areas are at greater risk of prostate cancer, where the mortality rate is 21% higher than in capital cities (AIHW & Australasian Association of Cancer Registries, 2012). This is attributed to lack of awareness and education about prostate cancer, along with distances from testing and treatment, poor general practitioner awareness and limited access to specialists. The number of deaths annually from prostate cancer in men is equivalent to the number of women who die from breast cancer, approximately 3300 (PCFA, 2015). It is projected that prostate cancer will remain the most common cancer in men, representing the biggest burden in 2025 (AIHW, 2015d).

When diagnosed early, prostate cancer is curable. If the man presents with metastatic prostate disease, it is incurable, so control of disease progression is key. The incidence of prostate cancer increases with age. Most diagnoses (85%) are made in men over 65 years of age (Cancer Council Australia 2015), with 1 in 7 men at risk up to age 75 (AIHW, 2015e). In New Zealand, the incidence of prostate cancer is lower, with 1 in 13 men being diagnosed up to age 75 (Prostate Cancer Foundation of New Zealand, 2015). It is more prevalent in men in Western countries and less common in men from South-East Asia. This may be due to the extent to which PSA and digital rectal examination screening are used; hence, caution should be taken when interpreting variations in prevalence. Many men are found to have prostate cancer on autopsy; usually the cancer has produced no manifestations or complications.

Risk factors

In addition to age, other risk factors being investigated are as follows:
- genetic and hereditary factors, with risk increased in men who have a family history of the disease
- ethnicity—more common in Western countries and less common in Asian countries, particularly South-East Asia
- having a vasectomy, believed to increase the levels of circulating free testosterone
- dietary factors, including a diet high in animal fat and excessive supplemental vitamin A.

FOCUS ON CULTURAL DIVERSITY
Risk and incidence of prostate cancer

- The incidence of prostatic cancer is lower for Indigenous Australian men than it is for non-Indigenous men (Condon et al., 2013).
- Prostatic cancer is the second most common cancer for Indigenous males, following lung cancer (Australian Bureau of Statistics, 2012; Diaz et al., 2015).

Pathophysiology

The prostate gland consists primarily of glandular epithelial cells. The exact aetiology of prostate cancer is unknown, although androgens are believed to have a role in its development. Almost all primary prostate cancers are adenocarcinomas and develop in the peripheral zones of the prostate gland. This location increases the risk of local spread to the prostatic

capsule. Despite its proximity to the rectum, metastasis to the bowel is uncommon because a tough sheet of tissue, Denonvilliers' fascia, acts as an effective physical barrier.

As the tumour enlarges, it may compress the urethra, obstructing urinary flow. The tumour may metastasise and involve the seminal vesicles or bladder by direct extension. Metastasis by lymph and venous channels is common.

Manifestations

Men with early-stage prostate cancer are often asymptomatic. Pain from metastasis to bones is often the initial manifestation noted. Urinary manifestations depend on the size and location of the tumour and the stage of the malignancy. They are often much like manifestations of BPH: urgency, frequency, hesitancy, dysuria and nocturia. The man may also notice haematuria or blood in the ejaculate (Grossman & Porth, 2013). Manifestations of prostate cancer are summarised in the box below.

MANIFESTATIONS Prostate cancer

GENITOURINARY
- Urinary symptoms—frequency, urgency, poor flow, difficulty commencing voiding, incomplete emptying
- Dysuria
- Haematuria
- Abnormal prostate on digital rectal examination
- Nocturia
- Haematospermia
- Erectile dysfunction

MUSCULOSKELETAL
- Bone or joint pain
- Migratory bone pain
- Back, hip and pelvic pain

NEUROLOGICAL
- Nerve pain
- Bilateral lower extremity weakness
- Bowel or bladder dysfunction
- Muscle spasms

SYSTEMIC
- Weight loss
- Fatigue

Complications

Death usually occurs secondary to debility caused by multiple sites of skeletal metastasis, especially to the vertebrae. Compression fractures of the spine are common, resulting in the possible loss of mobility and bowel and bladder function. Tumours may eventually involve bone marrow, resulting in severe anaemias and impaired immune function.

INTERPROFESSIONAL CARE

Care of the man with prostate cancer focuses on diagnosis, elimination or containment of the cancer, and prevention or treatment of complications. There are currently no clinical strategies to prevent the development of prostate cancer. Therefore, early detection remains the main emphasis for intervention related to this disease.

Diagnosis

Although an increasing number of men are now diagnosed with asymptomatic prostate cancer, many men with prostate cancer have either locally advanced cancer or distant metastasis at the time of diagnosis. The definitive diagnosis can be made only by biopsy (prostate biopsy is discussed in Chapter 46); however, other tests may suggest the presence of prostate cancer.

A digital rectal examination (DRE) is useful in assessing the prostate gland. Any hardness in the gland may indicate cancer; however, hardness may be a result of calcification due to prolonged inflammation or prostatic stones. DRE only allows the posterior surface of the gland to be palpated. Anterior lesions are not able to be detected using DRE. PSA levels are used to diagnose and stage prostate cancer and to monitor response to treatment. PSA is not a true tumour marker, but measurement of PSA has made dramatic differences to earlier diagnosis of prostate cancer in men. PSA levels rise in the presence of benign prostatic enlargement, prostatitis and other non-malignant conditions due to the fact that PSA is organ specific rather than cancer specific. Levels depend on age, and there is no specific normal or abnormal level. An increase over time is more significant than one reading. The PSA test is used with a DRE to help detect prostate cancer in men age 50 or older, and is also used to monitor effects of treatment.

While the normal range for PSA is 0–4.0 ng/mL, normal ranges for serum PSA for men of different ages in Western countries are as follows:
- 40–49 years: average 0.65 ng/mL, upper limit of normal 2.0 ng/mL
- 50–59 years: average 0.85 ng/mL, upper limit of normal 3.0 ng/mL
- 60–69 years: average 1.39 ng/mL, upper limit of normal 4.0 ng/mL
- 70–79 years: average 1.64 ng/mL, upper limit of normal 5.5 ng/mL (Andrology Australia, 2013).

Transrectal ultrasonography (TRUS) may be used when the DRE is abnormal or if the PSA is elevated. In this test, a small probe is inserted into the rectum. The probe gives off sound waves that create a picture of the prostate on a video screen. Guided by this picture, the doctor inserts a narrow needle through the rectal wall into the prostate gland and the needle removes a sample of tissue for examination. Other tests that may be ordered include a urinalysis or cystoscopy. Bone scan, magnetic resonance imaging (MRI) or CT scans may be performed to determine the presence of tumour metastasis.

Grade and stage help to determine prognosis and guide treatment decisions. Grade (cancer cell differentiation) is determined by the pathologist. Prostate cancer is staged with a variety of tests. Table 47.3 outlines treatment options according to the stage of the cancer.

Diagnosis is reached following a combination of history taking, DRE, PSA testing and prostate biopsy or alternative.

TABLE 47.3 Prostate cancer staging and treatment

STAGE	DESCRIPTION	TREATMENT
Localised	Cancer is confined to prostate, non-palpable, focal involvement; well differentiated	Close observation and follow up Interstitial or external-beam radiation therapy Prostatectomy Brachytherapy (radioactive seeds implanted in prostate)
Locally advanced	Cancer has broken through the capsule of the prostate, palpable, involves one or both lobes; poorly differentiated	Careful observation in selected people Hormone therapy Prostatectomy Interstitial or external-beam radiation therapy Ultrasound-guided percutaneous cryosurgery
Advanced	Extension of the tumour outside the prostate capsule, possible seminal vesicle involvement, cancer has metastasised, often to bone or lymph nodes	Adjunctive hormone therapy Chemotherapy/interstitial radiation Steroids External-beam radiation therapy Radical prostatectomy Palliative surgery (TURP)

Research for prevention

Toremifene (Fareston) to treat men with abnormal prostate growth might help prevent the growths from becoming malignant. The drug, which blocks some of the effects of oestrogen, had previously been used to treat advanced breast cancer in women. Men who have prostate intraepithelial neoplasia (PIN) have about a 30% chance of developing prostate cancer in 1 year and about a 65% chance within 2 years. A larger study is now in progress. In addition, other studies reported that men who took statins (to treat high cholesterol) were less likely to have prostate cancer.

In May 2012, the National Institute for Health and Clinical Excellence in the United Kingdom recommended the use of abiraterone (Zytiga). This drug was developed by Cancer Research UK and has been found to extend the lives of some people with advanced prostatic cancer following chemotherapy.

Treatments

The treatment of prostate cancer is complex and depends on the grade and stage of the cancer, as well as the age, general health and preference of the man. In some cases—for example, when the man with a slow-growing tumour is elderly or has a limited life expectancy—watchful waiting is the treatment of choice. Treatments for prostate cancer include surgery, radiation therapy and hormone manipulation.

SURGERY Surgery for prostate cancer includes several types of prostatectomies. For very early disease in older men, cure may be achieved with a simple prostatectomy (such as TURP), discussed in the section on benign prostate hyperplasia.

- *Radical prostatectomy* involves removal of the prostate, prostate capsule, seminal vesicles and a portion of the bladder neck. The entire prostate gland with that component of the urethra within the gland and seminal vesicles is removed, so the urethra below the prostate is then connected to the bladder. The surgery can be performed by open surgery or by using laparoscopic surgery, where small incisions are made in the abdomen and a laparoscope is inserted and used to remove the prostate. Varying degrees of urinary incontinence are experienced by men following this surgery, with approximately 10% having ongoing incontinence 12 months on. Erectile dysfunction is commonly experienced by men following this surgery, with up to 85% of men having problems with getting and maintaining an erection. There are strategies to reduce this incidence and treatment if ED persists (Andrology Australia, 2014) (see Table 47.4). Some surgeons do the surgery from an area other than the operating room by using a robotic interface. Nursing research reporting men's satisfaction with a discharge program following a radical prostatectomy is discussed in the 'Translation to practice' box below.
- *Retropubic prostatectomy* may be performed because it allows adequate control of bleeding, visualisation of the prostate bed and bladder neck, and access to pelvic lymph nodes.
- *Perineal prostatectomy* is often preferred for older men or those who are poor surgical risks. This approach requires less time and involves less bleeding.
- *Suprapubic prostatectomy* is rarely used, usually when problems with the bladder are expected. Control of bleeding is more difficult because the surgical approach is through the bladder.

TABLE 47.4 Potential complications related to radical prostatectomy and radiation therapy

RADICAL PROSTATECTOMY	RADIATION THERAPY
Erectile dysfunction	Erectile dysfunction*
Urethral stricture	Urethral stricture
Fistula/rectal injury	Rectal/anal stricture*
Urinary incontinence	Cystitis
Surgical/anaesthetic risk	Diarrhoea
	Proctitis
	Rectal ulcer
	Bowel obstruction*
	Urinary incontinence

*Delayed complications; may appear months or years after completion of therapy.

TRANSLATION TO PRACTICE Evidence-based practice: improve discharge teaching

The period immediately after discharge for a radical prostatectomy is one that is often a difficult time for men and their families as they cope with the emotional and physical demands of cancer surgery. Knowledge deficits about how long it will take to recover and how to provide care at home can significantly affect recovery. Nurses must recognise the need to provide discharge teaching prior to hospital discharge, and make it part of actual practice for every man they treat.

IMPLICATIONS FOR NURSING

Discharge education for men is essential and needs to include how to manage potential side effects following surgery, including urinary incontinence, ED and other side effects associated with radiotherapy and hormone therapy, such as bladder irritation and cystitis. Early discussion of side effects and clear management strategies are vital for men as they prepare to go home. Use of printed information sheets about preoperative and postoperative radical prostate surgery, along with a health education checklist and a discharge bag containing a urinary leg bag, urinary collection bag, wound supplies, incontinence product samples and a community resources brochure, are good strategies to ensure information is clear. Catheter care is one of the most valuable types of information required by men. Nurses are now, and will continue to be, challenged to provide the type and amount of information needed for self-care at home. Essential information that needs discussion and advice with the man following surgery is outlined below:

- pelvic floor exercises to promote continence and discussion about regular undertaking of the exercises
- education on preventing constipation and ensuring adequate fluid intake
- awareness and avoidance of bladder irritants such as tea, coffee, alcohol and diet soft drinks
- advice on how to source and use incontinence pads for men
- discussion with the doctor about medication to assist erections is important from a psychological, as well as a physical, perspective
- referral to support organisations.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. You are caring for a 75-year-old man who has had a radical prostatectomy for prostate cancer. His wife tells you they have always had an active sex life and she hopes this surgery will not change that. What would you say to her?
2. Why is the risk of infection high for a man providing self-care at home following a radical prostatectomy? Which interventions would you suggest during health education to reduce this risk?
3. If you were developing a list of community resources for men following prostate surgery, what would you include? How would your list vary for the following situations?
   - a 64-year-old man with a wife and four married children who all live close
   - a 77-year-old man who lives alone and has no family
   - a 90-year-old man who will go to a residential care facility after discharge
   - a 56-year-old homosexual man who lives with his partner.

For men with locally advanced (beyond the prostatic capsule) cancer, surgery is controversial because of the likelihood of hidden lymph node metastasis and relapse. TURP is not performed as curative therapy but may be used to relieve urinary obstruction for men with advanced disease.

Surgical intervention is now available for men with urinary sphincter insufficiency, which is the main cause of incontinence after prostatectomy. An artificial urinary sphincter is surgically implanted (see Figure 47.5). To be eligible, the man must be able to manipulate the pump placed in the scrotum and have adequate cognitive function to know when a problem with the appliance occurs.

RADIATION THERAPY Radiation therapy may be used as a primary treatment for prostate cancer. Long-term problems of impotence and urinary incontinence may be avoided and survival rates often are comparable. Radiation may be delivered either by external beam or interstitial implants of radioactive seeds of iodine, gold, palladium or iridium (*brachytherapy*). Brachytherapy is used when there are no major or acute lower urinary tract symptoms. Interstitial radiation has a lower risk of impotence and rectal damage than external-beam radiation. See Chapter 13 for nursing care of the person receiving radiation therapy.

Radiation therapy has a palliative role for men with metastatic prostate cancer, reducing the size of bone metastasis, therefore controlling symptoms of controlling pain and restoring some function, such as continence or the ability to ambulate for men with spinal cord compression.

HORMONAL MANIPULATION Androgen deprivation therapy is used to treat advanced prostate cancer by aiming to achieve castration and hence increase survival rate by suppressing testosterone, since prostate cancer is androgen dependent. Many cells in the growing tumour are androgen dependent and either cease to grow or die if deprived of androgens. Unfortunately, other cancer cells thrive without androgen and are unaffected by therapy to reduce circulating androgens. Therefore, the effects of hormone manipulations vary from complete but temporary regression of the tumour to no response at all. Strategies to induce androgen deprivation vary from orchidectomy to oral administration of hormonal agents. Table 47.5 compares surgical and hormone therapies. In addition, new drugs are being developed that block the effects of male hormones and research is being conducted to demonstrate what mix of hormones is best and at what time in the perioperative period they are most effective.

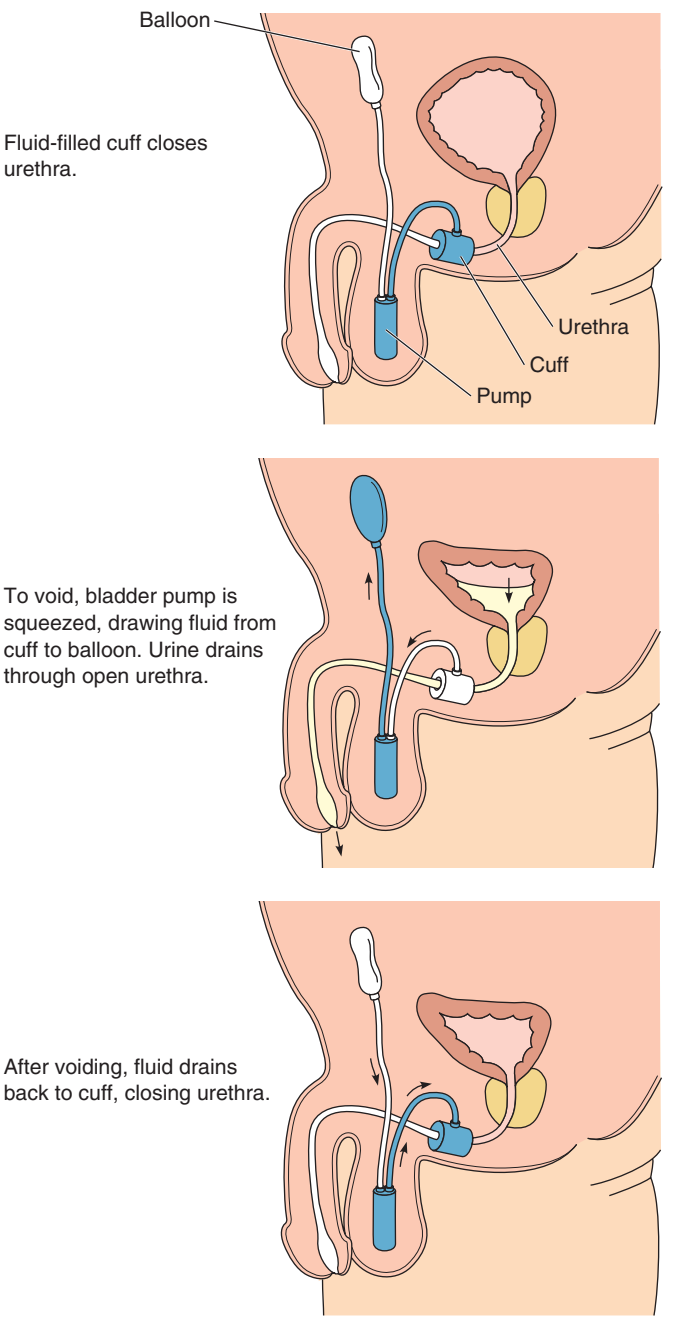


FIGURE 47.5 ■ Method of operation of an artificial urinary sphincter

Nursing care

Nurses plan and provide interventions to help prevent prostate cancer and to facilitate a return to functional health status. Interventions may range from health education to using knowledge and skill in physical care following a radical prostatectomy. A nursing care plan for a man with prostate cancer is given below.

Health promotion

Nurses are in a unique position to increase public awareness about early detection of prostate cancer. Every encounter with men and their families—in clinics, hospital units or in the home—is an opportunity to provide information about early detection and identify needs. Several studies have shown a positive correlation between increased awareness of and participation in prostate cancer screening procedures. Organisations such as Cancer Council Australia and Andrology Australia have information about early detection of prostate cancer, which are useful in educating the public.

One risk factor that can be easily changed is diet. Men should know that they can lower their risk of prostate cancer by eating less red meat and fat, and increasing their intake of fruit and vegetables, particularly tomatoes, pink grapefruit and watermelon, as these are high in lycopenes, which help prevent damage to DNA and may help lower prostate cancer risk. Other substances that may help lower the risk are vitamin E and selenium.

All men should be given information about the limitations and benefits of testing for early detection and of treatment so they can make an informed decision. Currently, routine screening of PSA as a diagnostic tool for prostatic cancer is not recommended by Australian cancer authorities. An elevated PSA may indicate that there is a problem with the prostate; however, it may not be cancer. Factors such as the man's level of concern about having cancer, age and family history should be discussed prior to having a PSA level taken. Diagnosis of cancer is made following physical assessment and a transrectal ultrasound and biopsy of the prostate (Andrology Australia, 2015b).

Assessment

Collect the following data through the health history and physical examination (see Chapter 46). Note that a digital rectal examination (DRE) is an advanced nursing assessment.

- *Health history:* risk factors, urinary elimination patterns and manifestations, haematuria, pain.
- *Physical assessment:* DRE to assess prostate size, symmetry, firmness and nodules.

Nursing diagnoses and interventions

The nursing care of men with prostate cancer must be holistic, sensitive and individualised. Nursing interventions discussed for the man with BPH may also be appropriate. This section focuses on problems with urinary incontinence, sexual function and pain.

Urinary incontinence (reflex, stress, total)

Urinary incontinence is a disturbing complication following treatment for prostate cancer. Both radical prostatectomy and external-beam radiation therapy can cause urinary incontinence, ranging from a few drops when the man lifts a heavy object (*stress incontinence*) to no control at all. Older men may experience *urge incontinence*, the involuntary passage of urine soon after a strong sense of urgency to void. Total and unpredictable loss of urine is classified as total incontinence. The man's reaction to incontinence may be severe even if the incontinence is not great. Many men have significant anxiety at the prospect of an incontinent episode in

TABLE 47.5 Surgical and hormone* therapy in the management of advanced prostate cancer

TREATMENT	ADVANTAGES	DISADVANTAGES
Orchidectomy	Inexpensive Immediate effect (i.e. men report diminished pain from metastasis in the OR recovery room)	Body image issues due to loss of testicles
Oestrogen compounds (diethylstilbestrol)	Inexpensive Effects reversible	Increased risk of cardiovascular problems (e.g. DVT) More likely to cause gynaecomastia, hypertrophy of breast tissue
Luteinising hormone-releasing hormone agonist (LHRH) (leuprolide)	Effects reversible No cardiovascular risk Monthly administration	Very expensive Subcutaneous injection route Slow onset: up to 3 weeks Can lead to exacerbation of clinical symptoms
Steroidal anti-androgens (megestrol acetate (Megace))	Effects reversible No cardiovascular risk Inexpensive Less risk of ED Less risk of osteoporosis	May not drop testosterone levels sufficiently Weight gain Increased risk of hepatotoxicity
Non-steroidal anti-androgens (flutamide; often used in conjunction with LHRH)	Do not alter circulating androgens Block some side effects of LHRH May be effective if other methods fail	Very expensive

*All hormonal manipulations have the potential disadvantage of loss of libido, erectile dysfunction, hot flushes and gynaecomastia.

public because they feel shame and often guilt about the loss of control.

- Assess the degree of incontinence and its effects on lifestyle. *The nurse needs to determine previous urinary patterns and the type of incontinence currently being experienced to plan appropriate interventions.*
- Teach pelvic floor exercises to help restore continence and discuss regularity of performing the exercises. *Pelvic floor or Kegel exercises can almost always improve stress incontinence, with the possibility of eliminating it.*
- Teach methods to control dampness and odour from stress incontinence:
  - Do not attempt to prevent accidental voiding by restricting fluids. *Not only will the man continue to have incontinent episodes, but also his urine will become concentrated, exacerbating the problem with odour and increasing risk of UTI.*
  - Manage occasional episodes (one to three small-volume accidents per day) with an absorbent pad worn inside the underwear and changed as needed. Most pads are made with a polymer gel that controls odour. *Appropriate measures help promote good hygiene, decrease anxiety and increase comfort.*
- Refer to a physiotherapist or a continence specialist for additional measures to promote continence. *Special exercises, restricting some types of fluids and other measures such as bladder training can help the man deal with incontinence.*
- Explore options such as an external collection device (external catheter or suprapubic catheter) for the man with total incontinence. *This device may improve the man's self-esteem and allow resumption of social activities.*
- Encourage verbalising feelings about the impact of incontinence on quality of life. The degree of incontinence does not necessarily correlate with the perceived level of suffering. *Listening to these concerns with sensitivity can help the man work through these feelings and may allow him to move towards a healthy adaptation to his disability.*

Sexual dysfunction

Surgical treatment for prostate cancer may cause erectile dysfunction and changes in ejaculatory function. Hormone therapy for advanced prostate cancer lowers libido and may also cause ED. The diagnosis of cancer and body image changes caused by hormone therapy may lower self-esteem, which in turn can diminish sexual desire and willingness to interact sexually with a partner. Most older men are active sexually and fully capable of sustaining an erection. They are likely to fear the effect of treatment on their sexual health. They may allow their anxiety to guide their decision about treatment or they may refuse all interventions because of this fear. Reactions vary greatly and the nurse must maintain a non-judgmental approach to education and support.

- Assess the man's pre-treatment sexual function. *Knowledge of previous sexual function is necessary to plan appropriate interventions.*
- Teach the man about the actual or potential effects of therapy on sexual function. *The incidence of ED varies with different therapies for prostate cancer.*
- Provide an opportunity for the man and his partner to discuss the implications of and their concerns about the diagnosis and treatment of sexual function. *The treatments for prostate cancer often affect the physiology of erection. The man and his partner need support and counselling during the period of adjustment. Sexual activity should not be resumed until 6–8 weeks following surgery. Stress and anxiety can exacerbate ED, so management of these need to be discussed and strategies put in place to manage them.*

NURSING CARE PLAN A man with prostate cancer

Bill Burns, a 76 year old, lives with his wife in a small retirement community in Victoria. His wife had a stroke 2 years ago and Mr Burns does all the cooking and housework. He has been in good health for most of his life, having only mild osteoarthritis in his knees and hands. He has noticed a gradual onset of urinary urgency and frequency over the past 2 years, but has never had incontinence. During a routine checkup, the doctor at the local general practice performs a digital rectal examination and palpates a hard nodule on the surface of Mr Burns's prostate. Following discussion with Mr Burns and his wife, a PSA is performed. After his PSA is found to be elevated, Mr Burns is referred to a urologist, who diagnoses prostate cancer. Mr Burns chooses to have surgery, and a radical retropubic prostatectomy and lymph node dissection are performed. The lymph nodes are negative for metastasis. Following surgery, Mr Burns' recovery is uncomplicated. However, the nurse caring for him is concerned about his ability to care for his indwelling catheter because of his arthritis and his wife's physical disabilities from the stroke. The nurse makes a referral to community health to ensure he can manage his care at home. An initial home health assessment is scheduled for the day after Mr Burns is discharged from the hospital.

ASSESSMENT

The community health nurse notes that the house is clean and neat. Mr Burns is dressed, but still wearing his night urinary drainage bag, even though it is 1300 hrs. Mr Burns tells the nurse that his main problem is going to get groceries because he is embarrassed to be seen with the drainage bag. He says he has not been able to remove the drainage bag and attach the leg bag because of his arthritis. Physical assessment findings include the pelvic incision healing without signs of infection. There is no tenderness in his calves, chest pain or shortness of breath. The urine is yellow, without odour. Mr Burns states that he sees no need for the pelvic exercises since he is no longer in the hospital. He also expresses the belief that he is cured of cancer and questions the need for follow-up care.

NURSING DIAGNOSES

- *Risk of stress urinary incontinence* related to surgical procedure.
- *Ineffective health maintenance* related to inability to care for the urinary drainage system, not understanding the need for postoperative exercises and questions about follow-up care.

PLANNING

Establish realistic outcomes for Mr Burns through education and support.

Expected outcomes

- Regain urinary continence after catheter removal.
- Change the urinary drainage bag with the appropriate assistance.
- Verbalise the rationale for performing postoperative exercise.
- Verbalise the need for continued follow-up care.

IMPLEMENTATION

- Discuss the possibility of stress incontinence after the catheter is removed.
- Reinforce the need for ongoing pelvic floor exercises while the catheter is still in place.
- Explore Mr Burns's support system to identify people who could assist him with catheter care and arrange a teaching session with them.
- Teach Mr Burns the importance of follow-up care, relating the care to the history of the disease.

EVALUATION

Good friends from Mr Burns's bowling club have assisted him with care of his drainage bag and have reminded him to do his pelvic floor exercises several times a day while the catheter is in place. When the catheter is removed, Mr Burns has only a small amount of leaking of urine after voiding. He understands that it may take several weeks for this to resolve. Efforts to help him understand the need for continued medical care are less successful. He continues to state that he is cured, his wife needs him and he sees no need to see his doctor.

CRITICAL THINKING IN THE NURSING PROCESS

1. Outline a health education plan for Mr Burns related to the *Risk of altered skin integrity* related to urinary incontinence.
2. As a result of Mr Burns's refusal to have ongoing medical care, he might be labelled as non-compliant. Would you make this assumption? Why or why not?
3. If you were the community health nurse making a home visit and found that Mr Burns had no urinary drainage for 16 hours, which assessments would you make? How would you handle this problem?

REFLECTION ON THE NURSING PROCESS

1. Consider some strategies you might use to engage Mr Burns in understanding the need for ongoing medical care.
2. How would you know that your education strategies with Mr Burns were successful for the long term?

- Discuss medical and surgical treatments for ED (see the first section of this chapter). *Many men are as devastated by the loss of erectile function as they are by the diagnosis of cancer. Information about achieving erection and maintaining sexual intimacy is essential to quality of life.*
- Refer for sexual counselling as appropriate. Refer to support groups. *The man and his partner may require therapy beyond that provided by nurses.*

CONSIDERATION FOR PRACTICE

A therapeutic approach to assessing how the man feels is to use an opening statement such as, 'Some men are very concerned about the effects of [type of treatment] on their ability to have an erection. Tell me how you feel about it.'

Acute/chronic pain

There are many causes of pain in men with advanced prostate cancer. It is not unusual for a man to have three or four distinct pains simultaneously, all from different sources. The most common cause of pain is metastasis to the spinal column, usually the thoracic spine. Other sources of pain include fractures, lymphoedema of the lower extremities and muscle spasms. Because most men with prostate cancer are over the age of 65, many also have pain associated with pre-existing conditions, such as osteoarthritis, unrelated to the cancer.

- Assess the intensity, location and quality of the pain. A cardinal rule of successful pain management is the importance of reducing or eliminating the cause of pain. *Appropriate interventions are based on a careful assessment of the man's pain.*
- Provide optimal pain relief with prescribed analgesics. *It is important that the man and his family understand that pain medications should be used on a regular basis to maintain comfort and should not be delayed until pain is severe.*
- Teach the man and his family non-invasive methods of pain control. *Various modalities can be successful in alleviating pain or reducing its perception, thus enhancing the comfort of the person (see Chapter 8).*

Community-based care

Depending on the type of treatment, the following topics should be addressed in preparing the man and his family for home care:

- for the man having a surgical procedure: manifestations of infection and excessive bleeding, catheter care, wound care, pain management
- for the man receiving radiation therapy:
  - danger of radiation damage to others (sleep in a room alone for a week; and avoid close contact with pregnant women, infants and children)
  - condom use during sexual contact (ejaculate may be discoloured, distressing the sexual partner)
- the importance of keeping appointments with healthcare providers and having yearly PSA and rectal examinations
- if appropriate, community services, such as support groups, community-based nurses and hospice
- helpful resources:
  - Andrology Australia: www.andrologyaustralia.org
  - Cancer Council Australia: www.cancer.org.au/home.htm
  - Prostate Cancer Foundation of Australia: www.prostate.org.au/articleLive.

MALE BREAST DISORDERS

THE MAN WITH GYNAECOMASTIA

Gynaecomastia, the abnormal enlargement of the glandular tissue in the male breast, is thought to result from a change in the ratio of testosterone to oestrogen, where the level of oestrogen is elevated. It is common during puberty, affecting one breast in as many as 50% of adolescent males, but usually resolves within 1 to 2 years. Any condition that increases oestrogen activity or decreases testosterone production can contribute to gynaecomastia. Conditions that increase oestrogen activity include obesity, testicular tumours, kidney and liver disease, and adrenal carcinoma. Conditions that decrease testosterone production include chronic illness such as tuberculosis or Hodgkin's disease, injury, orchitis and some genetic conditions (such as Klinefelter's syndrome). Drugs can contribute to gynaecomastia, particularly certain antidepressants, antihypertensives, anabolic steroids, marijuana, opiates, extreme amounts of alcohol and chemotherapeutic agents. Gynaecomastia after adolescence is usually bilateral. If it is unilateral, biopsy may be necessary to rule out breast cancer.

No treatment is necessary for the transient gynaecomastia of puberty. If the condition becomes chronic, however, creating psychological discomfort, surgery may be necessary to remove the subcutaneous breast tissue. When related to an underlying disorder, treatment of that disorder is required. If the cause is due to drugs, ceasing that drug usually decreases the breast tissue within a month. Where there is no hormonal problem and in severe cases, tamoxifen can be given to decrease oestrogen activity. Tamoxifen is used in treating breast cancer and is not approved for management and treatment of gynaecomastia, although improvements have been reported by men taking the drug.

Nursing care for the man with gynaecomastia includes health education about the cause and treatment of the condition, and emotional support for the psychosocial implications of this feminising condition.

THE MAN WITH BREAST CANCER

Although male breast cancer is rare, accounting for about 0.2% of all male cancer cases in Australia, it is as serious to the men who have it as it is to the women. Breast cancer accounts for about 0.1% of all male cancer deaths in Australia (AIHW, 2015a).

The aetiology of male breast cancer is unclear; hormonal, genetic and perhaps environmental factors appear to be important.

Male breast cancer is clinically and histologically similar to female breast cancer, although lobular cancer is rare in males. Most tumours are oestrogen-receptor positive. Because many men believe that breast cancer is only a woman's disease, they often delay seeking medical attention for symptoms, and thus may present with advanced disease.

Treatment of male breast cancer is much like the treatment of female breast cancer, beginning with modified radical mastectomy, node dissection and staging to determine the

therapeutic options. Radiation, chemotherapy and hormonal therapy (usually tamoxifen) are the conventional adjuncts to surgery. Castration (surgical removal of the testes) is the most successful palliative measure in men with advanced breast cancer, resulting in tumour regression and prolonging life.

Nursing care for the man with breast cancer is essentially the same as for the woman with breast cancer (see Chapter 48). The nurse has an opportunity to help the man and his family cope with the psychosocial effects of having breast cancer. He may feel embarrassment or shame about his condition, as well as fear about the life-threatening nature of the disease. His family may share those feelings. By listening with understanding and empathy, the nurse can help the man and his family resolve their feelings and move towards healing.

CHAPTER HIGHLIGHTS

- Disorders of male sexual function include erectile dysfunction (ED) and ejaculatory dysfunction. Many different illnesses, medications and surgical procedures may affect male sexual function. Treatments include medications, mechanical devices and surgical procedures. It is important for nurses to initiate a discussion of sexual concerns during assessments and to recognise that many male reproductive treatments and surgeries may result in sexual dysfunction.
- Phimosis and priapism are disorders of the penis that can cause problems with urination and sexual activity, and may in some cases be considered medical emergencies. The risk of cancer of the penis, although rare, is increased by phimosis, poor genital hygiene and viral HPV and HIV infections.
- Benign scrotal masses include hydrocoele, spermatocoele and varicocoele. Epididymitis may be associated with a urinary tract infection, prostatitis, urethral strictures or a sexually transmitted infection.
- The testes may be infected (orchitis), twisted (testicular torsion) or develop cancer. Testicular cancer is the most common cancer in men between the ages of 15 and 40. Monthly testicular self-examination is critical to early detection and treatment of cancer.
- The prostate gland may be inflamed or infected (prostatitis), enlarged (benign prostatic hyperplasia (BPH)) or develop cancer. BPH is a common disorder of the ageing male that causes problems with urination as the enlarging prostate gland constricts the urethra. Treatments include medications and various types of surgery, depending on the size of the prostate and the age and health status of the man.
- Cancer of the prostate is the most common type of cancer in Australian men. When diagnosed early, prostate cancer is curable. Diagnosis is often based on an increasing level of PSA and an abnormal DRE. The cancer is treated with surgery, radiation or hormonal manipulation.
- The male breast may become enlarged (gynaecomastia) or develop cancer.

CONCEPT CHECK

1 When conducting a health assessment, which of the following statements would most likely elicit information about sexual concerns?
1. 'Following your prostate surgery, when did you first notice you had problems with sexual intercourse?'
2. 'Why do you think you should be sexually active at your age?'
3. 'Do you miss having sex?'
4. 'Tell me about your experience with sexual function since you developed prostate enlargement.'

2 You are conducting a health education session for young men. Which topic would be appropriate to teach them about reducing the risk of cancer of the penis?
1. wearing a condom during sexual intercourse
2. retracting the foreskin of the penis when showering
3. avoiding tight pants and very hot showers
4. maintaining a regular testicular self-examination schedule

3 Which disease of the male reproductive system is a risk if a man also has a sexually transmitted infection (gonorrhoea)?
1. epididymitis
2. hydrocoele
3. erectile dysfunction
4. gynaecomastia

4 Which of the following statements is true of testicular cancer?
1. The incidence increases with age.
2. It occurs most between ages 15 and 40.
3. It rarely occurs in brothers.
4. Severe pain is the initial manifestation.

5 You are educating a man with chronic prostatitis how to care for himself at home. Which simple measures can be used to decrease discomfort?
1. take cold showers and restrict oral fluids
2. wear a scrotal support and take anti-inflammatory drugs
3. increase oral fluid intake to 3 L/day and void often
4. increase fibre intake and avoid sexual activity

6 Which diagnostic tests are used to differentiate BPH from prostate cancer? (Select all that apply.)
1. pelvic ultrasound
2. digital rectal examination
3. blood chemistry
4. PSA level
5. sperm count

7 The enlarging prostate in BPH typically is manifested by assessment of problems with:
1. bowel elimination
2. urinary elimination
3. peripheral vascular function
4. skin integrity

8 You are caring for a man who has returned to the unit from the recovery room following a TURP. His urinary drainage bag is filled with dark-red fluid with obvious clots. He is having painful bladder spasms. What would you do first?
1. assess his intake and output since surgery
2. administer pain medication in the form of oral oxybutynin hydrochloride
3. report your assessments to his urologist
4. nothing, because these manifestations are expected following a TURP

9 Which cancer is the most common malignancy in Australian men?
1 prostate cancer
2 testicular cancer
3 lung cancer
4 colon cancer

10 Which nutritional information should be included in a community program to reduce the risk of prostate cancer?
1 increase fibre intake
2 decrease lycopene intake
3 avoid foods high in sodium
4 decrease red meat and fat intake

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CHAPTER 48

NURSING CARE OF WOMEN WITH REPRODUCTIVE SYSTEM AND BREAST DISORDERS

MOIRA WILLIAMSON

KEY TERMS

amenorrhoea 1791
dysfunctional uterine bleeding (DUB) 1790
dysmenorrhoea 1789
endometriosis 1799
fibrocystic changes (FCC) 1813
leiomyoma 1798
menopause 1810
menorrhagia 1791
metrorrhagia 1791
premenstrual syndrome (PMS) 1786

LEARNING OUTCOMES

- Explain the pathophysiology, symptoms, complications, interprofessional care and nursing care of disorders of female sexual function, menstrual disorders, structural disorders and reproductive tissue disorders.
- Describe the surgical procedures used to treat female reproductive system disorders.
- Discuss alternative and complementary therapies used by women to relieve symptoms associated with menstrual disorders.
- Compare and contrast the risk, incidence, pathophysiology, symptoms, diagnosis, treatment and nursing care for cancer of the cervix, endometrium, ovary and vulva.
- Describe the physiological process of menopause.
- Discuss treatment, including alternative and complementary therapies, used by women to relieve symptoms associated with menopause disorders.
- Discuss the health education for women and their families for women with disorders of the reproductive system and breast.
- Discuss cancer screening, the purposes, nursing implications and medications and treatments.

CLINICAL COMPETENCIES

- Assess the functional status of women with reproductive system and breast disorders, and report, monitor and document abnormal symptoms.
- Use evidence-based research to design interventions to promote early diagnosis and treatment of all Australian women with cervical and breast cancer, with particular focus on the health disparities for women from areas of social disadvantage; for example, women living in remote and rural areas of Australia or Indigenous women.
- Determine nursing priorities based on assessed data to select and implement individualised nursing interventions for women with reproductive system and breast disorders.
- Administer medications used to treat female reproductive system and breast disorders knowledgeably and safely.
- Provide skilled care for the woman having a cervical dilation and curettage (D&C), laparoscopy, hysterectomy, mastectomy and breast reconstruction.
- Integrate an interdisciplinary approach into the care of women with reproductive system and breast disorders.
- Provide health education appropriate for community-based self-care of female reproductive and breast disorders.
- Revise the plan of care as needed to provide effective interventions to promote, maintain or restore functional health status to women with reproductive system and breast disorders.

Disorders of the female reproductive system range from a minor discomfort of menstrual cramps to life-threatening diseases such as cancer. Many of these disorders can occur at any point in a woman's adult life. They may affect her sexuality, ability to bear children and sense of wellbeing as a woman.

A holistic approach to meet the needs of women who experience reproductive system changes and disorders is required to ensure that their physical, emotional and educational needs are met. The ability to reproduce affects self-esteem, feelings of femininity and general health; both sensitivity and understanding from caregivers is essential. Often women are required to disclose personal information when providing a medical and family history, and undergoing diagnostic tests. Women may find providing intimate information embarrassing and uncomfortable. When planning and implementing care, nurses must consider the woman within the context of her culture, socioeconomic and educational level, and lifestyle. It is also important that the nurse not make assumptions or judgments about sexual orientation.

This chapter summarises disorders of female sexual function and discusses the physiological process of menopause, menstrual disorders, structural disorders and disorders of female reproductive tissue, including the breast. Sexually transmitted infections, including vaginal infections and pelvic inflammatory disease, are discussed in Chapter 49. Many of the disorders result in actual or potential health problems requiring nursing care that is evidence based. To avoid repeating strategies and interventions for each disorder, they have been divided between the nursing care discussions as appropriate. Treatment of cancer with chemotherapy and radiation is discussed in Chapter 13.

DISORDERS OF FEMALE SEXUAL FUNCTION

Women maintain the capacity for sexual activity and orgasm long after menopause (see the 'Meeting individualised needs' box below). In a typical sexual event, two physiological sexual responses occur: vasocongestion and myotonia. Sexual stimulation results in vasocongestion of the blood vessels surrounding the vagina, causing engorgement, increased lubrication and genital swelling and enlargement. Arousal, or myotonia, increases muscular tension, resulting in voluntary and involuntary muscle contraction.

The sexual response cycle has four phases: excitement, plateau, orgasm and resolution. These phases always occur in the same sequence; however, the duration of each phase may vary. Sexual arousal typically ends in orgasm (climax), but does not always do so. A refractory period, or period in which the sexual organs are incapable of responding to stimulus, does not occur in the female. Multiple orgasms are physically possible in all women.

Factors affecting women's sexuality are often not discussed or raised by health professionals. Although nurses may not conduct sexual counselling (unless trained to do so), they should be able to obtain a sexual history without embarrassment, discuss sexual concerns with women and make appropriate referrals.

MEETING INDIVIDUALISED NEEDS Sexual function in the ageing woman

The belief that older women are no longer interested in expressing their sexuality may occur within society. This view results from myths, taboos and stereotypical views held by some within society. Two commonly held myths are that menopause causes the demise of a woman's sexuality and that hysterectomy results in the inability of a woman to function sexually. Loss of sexual function is not an inevitable result of ageing, although physical changes related to ageing do affect the female sexual response. These physical changes, along with chronic conditions common in ageing women, may alter a woman's sexual function. In addition, the sexual response can be altered by some medications used to treat the chronic conditions associated with ageing. The role of the nurse is to provide information about ways to achieve optimal sexual health, including educating women about the myths and misinformation around changes in sexual functioning.

Physiological changes

Changes in ageing women's sexual function begin in the perimenopausal period as oestrogen levels decrease. Oestrogen-sensitive cells are found throughout the central nervous system and the cardiovascular system. These cells are involved in the female sexual response. With menopause comes a decrease in the levels of oestradiol, which affects nerve transmission and the response in the peripheral vascular system. As a result, the timing and degree of vasocongestion during the sexual response are affected.

Specific changes in the female sexual response occur in all phases. During the plateau phase, the capacity for vasocongestion decreases, as does muscle tension. In the orgasmic phase, the contractions are fewer and less intense. During the resolution phase, vasocongestion subsides more quickly.

Nursing care

The nurse's role centres on educating women about the physiological and psychological changes associated with menopause and assisting ageing women to reach optimal sexual functioning. Provide education on how the effects of chronic illness and the medications used to treat these illnesses affect sexual functioning. Educating about the importance of maintaining a healthy lifestyle, which includes a balanced diet, weight-bearing and aerobic exercises, stress management and routine health examinations is important.

For problems related to vaginal dryness and dyspareunia, water-soluble vaginal lubricants or vaginal gels can be used before intercourse. Intercourse on a regular basis and oestrogen replacement therapy (ordered by a medical practitioner) can also be recommended. Women who experience joint pain or other musculoskeletal pain due to conditions such as arthritis can benefit from education on how to adapt positions for intercourse.

Pathophysiology

Disorders of sexual function include dyspareunia, inhibited sexual desire and orgasmic dysfunction.

Dyspareunia

The woman with dyspareunia (pain during intercourse) may find it difficult to express her feelings to her partner. This condition may manifest itself as decreased desire or inhibited orgasm. The causes of dyspareunia range from organic to psychogenic.

Physical conditions, such as imperforate hymen, vaginal scarring or vaginismus, may cause dyspareunia. *Vaginismus* is a rare condition in which the vaginal muscles at the introitus contract so tightly that an erect penis cannot be inserted. An early traumatic event, such as sexual abuse, fear of men or rape, may contribute to this disorder. However, it is estimated that most dyspareunia is psychogenic in origin. The woman develops an anxiety–fear–guilt cycle in which negative thoughts become associated with the act of vaginal penetration, initiating a conditioned involuntary reflex. However, other sexual activity may be pleasurable. The woman's partner needs to be included in any discussion or education sessions to assist with their understanding of the issues affecting their partner's responsiveness.

Inhibited sexual desire

Inhibited sexual desire is complex and may be a result of pathophysiological processes or may be psychogenic in origin. Inhibited sexual desire often is rooted deeply in childhood education or personal experiences that may be too painful to recall. Cultural and religious values can also affect the processing of sexual stimuli. Fear of pregnancy or sexually transmitted infections (STIs) and depression can also contribute to decreased libido.

Orgasmic dysfunction

Female orgasm inhibition (anorgasmia) is the most prevalent sexual problem in women. However, fewer than 20% of cases are physiological in origin. It is estimated that between 8% and 15% of women have never experienced an orgasm in the waking state. Psychogenically induced anorgasmia may result from unresolved conflicts about sexual activity. Organic causes of anorgasmia include the presence of disease that results in general debilitation or that affects the sexual response cycle, or the use of medications that depress the central nervous system (CNS).

Primary anorgasmia exists when a woman has never experienced an orgasm during the waking state, either through self-stimulation, partner stimulation or intercourse. Secondary anorgasmia exists when a woman who previously experienced orgasms is no longer able to do so.

Nursing care

Nursing care focuses on identifying the type of sexual dysfunction with a thorough history, including the onset, duration, frequency and context or situation in which the problem occurs. The woman's partner should be included in discussions.

The woman and her partner may require education about varied normal sexual responses. The goal is to increase self-awareness and understanding of communication and how this impacts on sexual desire. The differences in the behaviours that men and women consider sexually stimulating may need to be discussed. Sex therapists may provide training in stimulation techniques (masturbation) after inhibitions about this practice are discussed. Group therapy may be encouraged to help the woman discuss her problem and to decrease the sense of isolation it gives her.

MENSTRUAL DISORDERS

Monthly menstruation normally involves some minor discomfort, including breast tenderness, a feeling of heaviness and congestion in the pelvic area, uterine cramping and lower backache. Many women, however, experience more serious effects, both physiological and psychological. This section discusses premenstrual syndrome, dysmenorrhoea and abnormal uterine bleeding. (The menstrual cycle is discussed in Chapter 46.)

THE WOMAN WITH PREMENSTRUAL SYNDROME

Premenstrual syndrome (PMS) is a complex group of symptoms. Women can present with a range of these (e.g. mood swings, breast tenderness, fatigue, irritability, food cravings and depression) that are limited to 2 to 14 days before menstruation and relieved by the onset of menses. However, the usual time frame for symptoms to be present for most women who experience PMD is approximately 7 days a month. For a small number of women, PMS is so disabling that it is given the psychiatric label of premenstrual dysphoric disorder (PMDD).

The syndrome is seen less frequently during the teens and twenties, and is more common in women aged between 30 and 45 years of age. The cause is unknown; however, it is linked to stress.

Pathophysiology

Although the pathophysiology of PMS is not clearly understood, hormonal changes such as altered oestrogen–progesterone ratios, increased prolactin levels and rising aldosterone levels during the luteal phase of the menstrual cycle may contribute to the problem. Increased aldosterone results in sodium retention and oedema. Decreased levels of monoamine oxidase in the brain are associated with depression, and reduced levels of serotonin can lead to mood swings.

Manifestations

Symptoms of PMS occur during the luteal phase of the menstrual cycle (7 to 10 days prior to the onset of the menstrual flow), abating when the menstrual flow begins. The multisystem effects of PMS are shown below. Although PMS may produce a variety of physiological and psychological symptoms, the exact nature of these symptoms and their intensity are individualised for each woman with this disorder. The symptoms may even differ from month to month in the same woman.

INTERPROFESSIONAL CARE

If no organic cause can be identified, the goals of care are to relieve symptoms and develop self-care patterns that will help the woman anticipate and cope more effectively with future episodes of PMS. There are no definitive diagnostic tests for PMS. The regular recurrence of symptoms preceding the onset of menses for at least 2 to 3 months leads to a diagnosis of PMS. The treatment of PMS integrates a self-monitored record of symptoms. Women can then be educated on ways of relieving symptoms, such as undertaking regular exercise, ensuring healthy eating patterns and reducing the level of stress in their lives. However, there are a variety of treatments available (Women's Health & Research Institute of Australia, 2014).

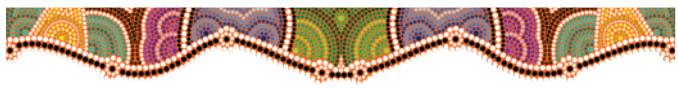
Medications

If the symptoms of PMS are severe or incapacitating, ovulation may be suppressed by the use of gonadotropin-releasing hormone (GnRH) agonists, oral contraceptives or danazol. Progesterone and antiprostaglandin agents such as non-steroidal anti-inflammatory drugs (NSAIDs) may help relieve cramping. Diuretics may be prescribed to relieve bloating. Selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine (Prozac), sertraline (Zoloft) and paroxetine (Aropax) may be used to manage mood and some physical symptoms of PMS.

Alternative and complementary therapies

Alternative and complementary therapies the woman with PMS may find helpful focus on diet, exercise, relaxation and stress management.

- A diet high in complex carbohydrates with limited simple sugars and alcohol is recommended to minimise reactive hypoglycaemia, which can contribute to the symptoms of PMS.
- Reduced sodium intake helps minimise fluid retention. Increased intake of calcium (1200 mg per day), magnesium (360 mg per day) and vitamin E (400 international units per day) may be helpful (Mayo Foundation for Medical Education and Research, 2014).
- Caffeine should be restricted to reduce irritability.
- Herbal remedies include ginger, chasteberry, St John's wort and evening primrose oil (Mayo Foundation for Medical Education and Research, 2014). However, there is limited research to support the use of alternative therapies. Woman may feel psychologically, however, that these herbal remedies are of benefit. Therefore, discussion about alternative therapies with the healthcare provider is recommended.
- Exercise is beneficial, but adequate rest also is necessary.
- Techniques for relaxation and stress management include deep abdominal breathing, meditation, muscle relaxation and guided imagery.



Nursing care

Nursing diagnoses and interventions

Nursing care for the woman with PMS focuses on relieving symptoms. Most women experiencing PMS require interventions to manage pain and enhance coping.

Acute pain

The woman with PMS may have pain from headache (including migraine), menstrual cramps, excessive fluid retention, breast swelling, joint and muscle pain, and backache.

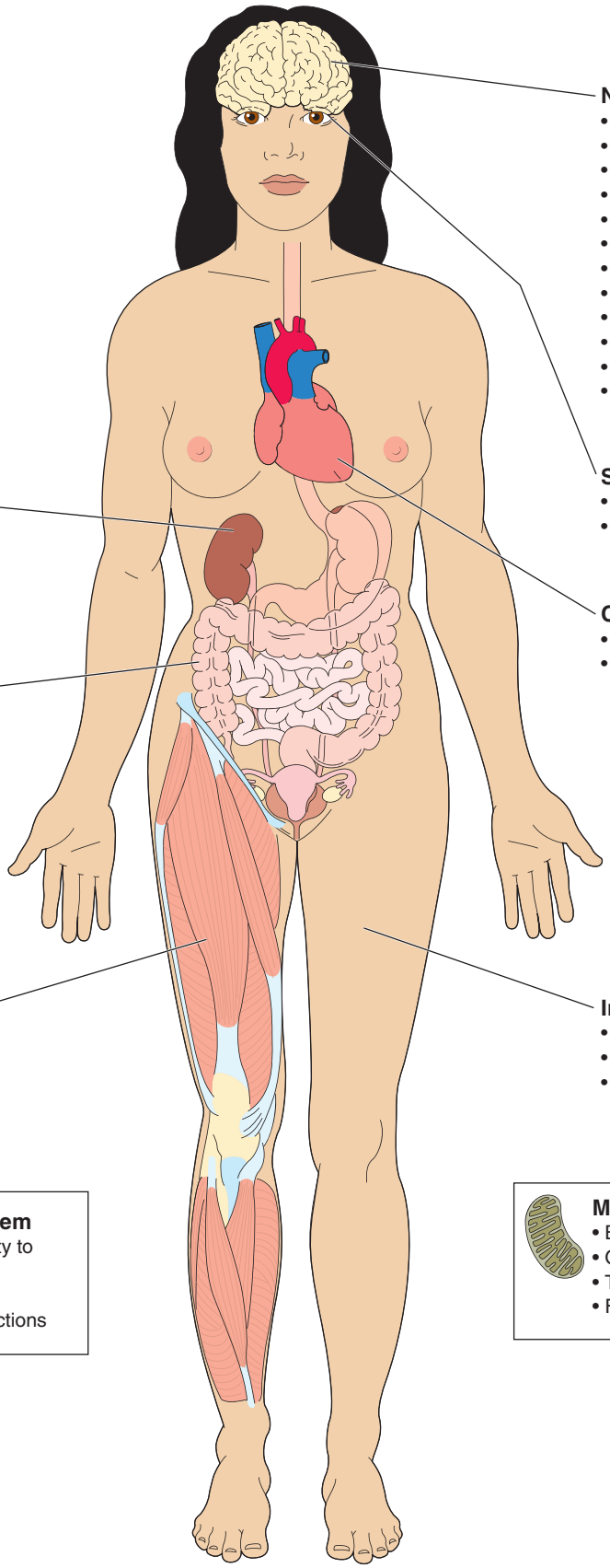
- Teach effective pharmacological and non-pharmacological self-care measures to relieve pain: application of heat, relaxation techniques (such as breathing exercises, imagery techniques or meditation) and exercise. *Heat relieves muscle spasms and dilates blood vessels, increasing blood supply to the pelvis and uterine muscles. Relaxation and exercise aid the release of naturally produced pain relievers called endorphins.*
- Review daily activities and suggest ways to balance rest periods and activity. *During rest periods, energy and oxygen requirements decrease, increasing the amount of energy and oxygen available to muscles.*
- Review symptoms and, if possible, correlate these with dietary patterns and activity levels. Encourage the woman to keep a diary of PMS symptoms. *Maintaining a diary of PMS symptoms, activity and foods eaten can provide data to identify modifiable causes of discomfort.*
- If appropriate, suggest sexual activity as a way to lessen menstrual cramps. *Orgasm may help relieve dysmenorrhoea.*

Ineffective coping

Many women experience wide mood swings during episodes of PMS, sometimes exhibiting self-destructive or aggressive behaviours towards others. These mood swings can interfere with a woman's ability to manage her responsibilities at home, in the community or at work.

- Encourage the woman to keep a journal of her menstrual cycle and to document her mood changes in the 7 to 10 days prior to menstruation. *Recognising the signs and timing of PMS is the first step in developing methods to cope with the problem.*
- Explore possible ways to rearrange or reschedule activities when experiencing PMS. *Planning ahead enables the woman to assume more control and promotes coping methods.*
- Explore what, if any, self-care measures have helped with mood alterations in the past. *Encourage healthful coping mechanisms, such as relaxation techniques and exercise. Some women may rely on alcohol or other medications during PMS, which only exacerbate the symptoms.*

MULTISYSTEM EFFECTS OF PMS



Neurological
- Syncope
- Vertigo
- Dizziness
- Paraesthesia
- Headache
- Inability to concentrate
- Depression
- Irritability
- Anxiety
- Mood swings
- Anger
- Aggressive behaviour

Sensory
- Conjunctivitis
- Visual disturbances

Cardiovascular
- Bruising
- Palpitations

Urinary
- Cystitis
- Oliguria

Gastrointestinal
- Constipation
- Nausea
- Vomiting

Musculoskeletal
- Backache
- Pelvic stiffness

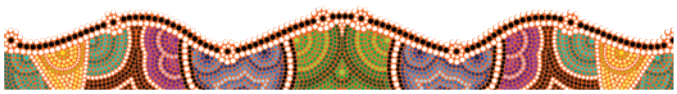
Integumentary
- Acne
- Herpes recurrence
- Urticaria

Immune system
- ↑ Susceptibility to infection
- Asthma
- ↑ Allergic reactions

Metabolic processes
- Breast tenderness
- Oedema
- Transient weight gain
- Food cravings

Community-based care

Provide health education that enables the woman and her family to understand that PMS is not caused by a pathological process but is a physiological response to hormonal changes of the menstrual cycle. With an understanding of the condition, the woman is better able to manage anxiety and to become actively involved in techniques to reduce the symptoms. Education should also include dietary measures, relaxation techniques and exercise, stress-reduction techniques, mental health promotion and support systems.



THE WOMAN WITH DYSMENORRHOEA

Dysmenorrhoea (pain or discomfort associated with menstruation) is experienced by a significant number of menstruating women. *Primary dysmenorrhoea* occurs without specific pelvic pathology and is most often seen in girls who have just begun menstruating, becoming less severe after the mid twenties or giving birth. *Secondary dysmenorrhoea* is related to identified pelvic disease.

Pathophysiology

In primary dysmenorrhoea, excessive production of prostaglandins stimulates uterine muscle fibres to contract. As the muscles contract, uterine circulation is compromised, resulting in uterine ischaemia and pain. These contractions can range from mild cramping to severe muscle spasms. Psychological factors, such as anxiety and tension, may contribute to dysmenorrhoea. Secondary dysmenorrhoea is related to underlying organic conditions that involve scarring or injury to the reproductive tract. Endometriosis, fibroid tumours, pelvic inflammatory disease or ovarian cancer may result in painful menses.

Manifestations

Symptoms of primary dysmenorrhoea (see the box below) may be severe enough to disrupt activities of daily living, sexual function and even fertility.

MANIFESTATIONS Primary dysmenorrhoea

- Abdominal pain beginning with onset of menses and lasting 12 to 48 hours
- Pain radiating to lower back and thighs
- Headache
- Nausea
- Vomiting
- Diarrhoea
- Fatigue
- Breast tenderness

INTERPROFESSIONAL CARE

Care of the woman with menstrual pain focuses on identifying the underlying cause, re-establishing functional capacity and managing pain.

A careful history is taken and a physical assessment is performed to rule out any underlying organic cause of dysmenorrhoea. If no organic cause can be found, the diagnosis is primary dysmenorrhoea. In addition, attitudes and expectations about menstruation and lifestyle disruption are identified and explored.

Diagnosis

Various diagnostic tests are performed to identify structural abnormalities, hormonal imbalances and pathological conditions that could cause menstrual pain. Diagnostic tests are described in Chapter 46.

Diagnosis is made based on a comprehensive history of the signs and symptoms. As the onset of primary dysmenorrhoea usually occurs following the onset of menarche, the age group for this presentation is normally between 11 and 13 years of age. Therefore, it is not appropriate for a vaginal examination and pelvic ultrasound to be undertaken unless there are physical signs on abdominal palpation of ovarian masses. For this group of young teenagers, treatment with non-steroidal anti-inflammatory drugs (NSAIDs) is recommended (Davis & de Costa, 2011).

The second group of women who experience primary dysmenorrhea is older teenage women who may be sexually active. Performing a vaginal examination for women in this age group needs to be carefully assessed on an individual basis. For this age group, treatment may be offered in the form of the combined oral contraceptive pill (Davis & de Costa, 2011). If the young woman is sexually active, it is important to discuss the importance of cervical screening, contraception and sexually transmitted infections (STIs). If symptoms persist after commencing treatment, a pelvic examination and diagnostic procedures (including a Papanicolaou (Pap) smear and cervical and vaginal cultures; ultrasound of the pelvis and vagina; and computed tomography (CT) scan or magnetic resonance imaging (MRI) to detect structural abnormalities, malignancy or infections) are undertaken. Laboratory tests used to assess possible causes of dysmenorrhoea are as follows:

- *follicle-stimulating hormone (FSH)* and *luteinising hormone (LH) levels* to assess the function of the pituitary gland. The results are correlated with the time of the menstrual cycle
- progesterone and oestradiol levels to assess ovarian function
- thyroid function tests (T_3 and T_4) to assess thyroid function.

Laparoscopy is used to diagnose structural defects and blockages caused by scarring, endometriosis, tumours and cysts (see Figure 48.1). See the box below for nursing care of the woman having a laparoscopy. A dilation and curettage (D&C) of the uterus may be performed to obtain tissue for evaluation or to relieve dysmenorrhoea and heavy menstrual bleeding. (This procedure is discussed later in this chapter.)

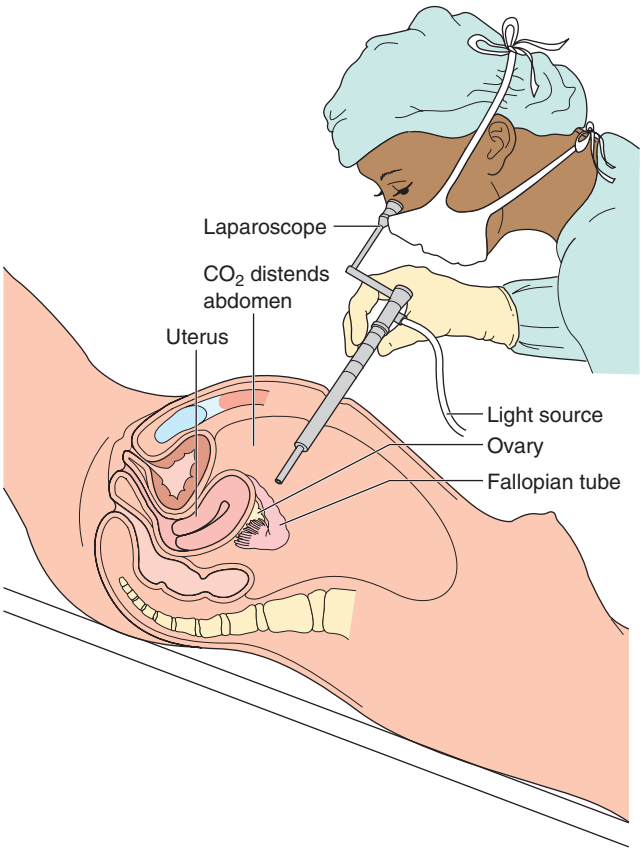


FIGURE 48.1 ■ Laparoscopy. In this surgical procedure, a flexible, lighted instrument (laparoscope) is inserted through a periumbilical incision. Laparoscopy allows visualisation of the pelvic cavity

Medications

Dysmenorrhoea may be treated with analgesics, prostaglandin inhibitors such as NSAIDs or oral contraceptives (see the 'Medication administration' box below).

Alternative and complementary therapies

The complementary therapies listed for the woman with PMS may also be useful for the woman with dysmenorrhoea. Other helpful activities are regular physical exercise, supplementing the diet with zinc and calcium, and using herbal remedies such as *Viburnum prunifolium*, black cohosh, evening primrose oil and blue cohosh. Using a heated pad on the abdomen or taking a warm bath also helps reduce pain.

Nursing care

Nursing care for the woman with primary dysmenorrhoea focuses on controlling symptoms and providing health education about the normal physiology of the menstrual cycle and self-care measures. Care of the woman with secondary dysmenorrhoea varies according to the underlying cause and is discussed in this chapter within sections on specific disorders. Nursing interventions previously described for the woman with PMS are also appropriate for the woman with dysmenorrhoea.

THE WOMAN WITH DYSFUNCTIONAL UTERINE BLEEDING

Dysfunctional uterine bleeding (DUB) refers to vaginal bleeding that is usually painless but is abnormal in amount, duration or time of occurrence. The types of DUB include primary and secondary amenorrhoea, oligomenorrhoea, menorrhagia, metrorrhagia and postmenopausal bleeding.

A number of factors may predispose a woman to DUB. These factors include stress, extreme weight changes, use of

NURSING CARE OF THE WOMAN having a laparoscopy

PREOPERATIVE CARE

- Orient the woman to the environment.
- Instruct the woman to empty the bladder prior to the surgical procedure.
- Explain to the woman that referred shoulder pain or expulsion of gas through the vagina may occur postoperatively. *During the procedure, the woman's abdomen is insufflated with carbon dioxide gas to distend the abdomen and facilitate visualisation of the pelvic organs. The examination table is then tilted so that the intestines will fall away from the pelvic organs. Some carbon dioxide gas may remain in the abdomen after the procedure.*
- Explain that pain should be minimal. However, pain relief is available if required. Instruct the woman to report excessive pain immediately. *Excessive pain signals infection or other postoperative complication.*

POSTOPERATIVE CARE

- Routine postoperative observations include pulse, blood pressure, respiratory rate, oxygen saturation level and consciousness.
- It is important to monitor vaginal bleeding. *Minor bleeding is normal; excessive bleeding may indicate haemorrhage.*
- Change the perineal pad, recording blood loss. Teach the woman proper perineal hygiene, emphasising the need to change pads at least every 4 hours. Keep a pad count to monitor blood loss. Proper perineal hygiene reduces the risk of postoperative infection. *Pad count is an indication of blood loss.*

MEDICATION ADMINISTRATION The woman with dysmenorrhoea

EXAMPLES OF ORAL CONTRACEPTIVES

Norethisterone and ethinylestradiol (Brevinor)
Ethinylestradiol (Microgynon 30)

Oral contraceptives inhibit ovulation and help reduce cramping and bleeding. Side effects of oral contraceptives include breast tenderness, weight gain, nausea, midcycle bleeding, mood swings, depression, chloasma (skin discolouration) on the face and chest, hypertension, vascular complications, vaginal candidiasis, migraines and glucose intolerance. Oral contraceptives are contraindicated in women with personal or family history of breast cancer in first-degree relatives, hypertension, history of stroke or transient ischaemic attack (TIA), smoking, history of oestrogen-dependent cancer, pregnancy, liver disease or thrombophlebitis.

Nursing responsibilities

- Assess the woman for potential contraindications to medication therapy.

Health education for the woman and family

- Take the medication as prescribed until otherwise indicated by a health professional or until side effects prevent you from continuing to take it.
- If you are taking oral contraceptives, be sure to take them at the same time every day.
- Report any suspected pregnancy and any side effects such as nausea, rash, drowsiness, stomach pain, ringing in the ears, tenderness in the calf or shortness of breath.
- Do not smoke while taking oral contraceptives.
- Wear TED stockings when travelling long distances when you may not be very active, such as long plane flights or car travel.

oral contraceptive agents or intra-uterine devices (IUDs), and postmenopausal status. DUB is usually related to hormonal imbalances or pelvic neoplasms, either benign or malignant.

Pathophysiology

The types of DUB outlined above will now be discussed.

- **Amenorrhoea** is the absence of menstruation. *Primary amenorrhoea*, absence of menarche by age 16 (or by age 14 if secondary sex characteristics fail to develop), may be caused by structural abnormalities, hormonal imbalances, polycystic ovary disease or an imperforate hymen. Because a certain percentage of body fat is required for menstruation to occur, anorexia nervosa, bulimia or excessive athletic training can also cause primary amenorrhoea. *Secondary amenorrhoea*, absence of menses for at least 6 months in a previously menstruating female, may also be caused by anorexia nervosa, excessive athletic activity or training, or a large weight loss. Other causes include hormonal imbalances and ovarian tumours. Normal (physiological) secondary amenorrhoea occurs during pregnancy, breastfeeding and menopause.
- *Oligomenorrhoea* (scant menses) usually is related to hormonal imbalances.
- **Menorrhagia** (excessive or prolonged menstruation) may result from thyroid disorders, endometriosis, pelvic inflammatory disease, functional ovarian cysts or uterine fibroids or polyps. Clotting disorders and anticoagulant medications also can cause menorrhagia.
- **Metrorrhagia** (bleeding between menstrual periods) may be caused by hormonal imbalances, pelvic inflammatory disease, cervical or uterine polyps, uterine fibroids or cervical or uterine cancer. Because cancer is a possible cause of metrorrhagia, early evaluation and treatment are extremely important. *Mittelschmerz* (midcycle spotting associated with ovulation) occurs in many women and is not considered metrorrhagia.
- *Postmenopausal bleeding* may be caused by endometrial polyps, endometrial hyperplasia or uterine cancer. The possibility of cancer makes early evaluation and treatment essential.

Hormonal imbalances, especially progesterone deficiency with relative oestrogen excess, result in endometrial hyperplasia. Oestrogen stimulates endometrial proliferation. However, without the support provided by progesterone, sloughing occurs, resulting in vaginal bleeding that may be irregular, prolonged or profuse. Defects in the follicular phase shorten the proliferative phase of the menstrual cycle, resulting in spotting and breakthrough bleeding. Defects during the luteal phase result in excessive amount or duration of flow due to persistence of the corpus luteum. This leads to a deficiency of progesterone, resulting in vaginal bleeding. *Anovulation*, absence of ovulation, is associated with both oestrogen and progesterone deficiencies. Emotional upsets or stress can cause hormonal imbalances and thus affect menstruation. Pelvic neoplasms, discussed later, also cause abnormal bleeding.

INTERPROFESSIONAL CARE

The care of the woman with DUB focuses on identifying and treating the underlying disease. A careful history is taken and a physical examination is performed. Abdominal and pelvic examinations are performed to rule out abdominal masses. The woman may need to keep a menstrual history and basal body temperature chart for several months to determine whether ovulation is occurring.

Diagnosis

A variety of diagnostic tests are used to diagnose the cause of DUB. Diagnostic tests are discussed in Chapter 46 and include a Pap smear to rule out or identify cervical carcinoma, a pelvic ultrasound to identify luteal cysts, a hysteroscopy to detect abnormalities of the uterine cavity or an endometrial biopsy to obtain endometrial tissue for histological examination.

Laboratory studies may include:

- a *full blood count (FBC)* to rule out systemic disease as a contributing factor to DUB and to evaluate its effects.

- *thyroid function studies*, including measurement of triiodothyronine (T_3), thyroxine (T_4) and thyroid-stimulating hormone (TSH) levels, to rule out hyper- or hypothyroidism as a cause of DUB.
- *endocrine studies* to evaluate pituitary and adrenal function. Pituitary dysfunction may first be manifested by menstrual irregularities.
- *serum progesterone levels* to determine the level of progesterone deficiency.

Medications

For many women, hormonal agents can correct menstrual irregularities. For anovulatory DUB, oral contraceptives may be prescribed for 3 to 6 months. Progesterone or medroxyprogesterone also may be prescribed to regulate uterine bleeding.

Ovulatory DUB may be treated with progestins during the luteal phase. Oral iron supplements may be prescribed to replace iron lost through menstrual bleeding.

Surgery

Surgical intervention emphasises the least invasive method that provides effective relief, beginning with a therapeutic D&C, then endometrial ablation and, finally, hysterectomy.

THERAPEUTIC D&C In a therapeutic D&C, the cervical canal is dilated and the uterine wall is scraped. D&C, the most frequently performed minor gynaecological surgical procedure, is used to diagnose and treat DUB and other disorders of the female reproductive system. It may be performed to correct excessive or prolonged bleeding. D&C is contraindicated in any woman who has been taking anticoagulant medications or whose condition precludes the use of regional or general anaesthesia. Nursing care of the woman having a D&C is described in the box below.

ENDOMETRIAL ABLATION In an endometrial ablation, the endometrial layer of the uterus is permanently destroyed using laser surgery or electrosurgical resection. It is performed in women who do not respond to pharmacological management or D&C. The woman needs to understand that this procedure ends menstruation and reproduction.

HYSTERECTOMY Hysterectomy, or removal of the uterus, may be performed when medical management of bleeding disorders is unsuccessful or malignancy is present, particularly if the woman no longer wishes to bear children. In premenopausal women, the ovaries are usually left in place; in postmenopausal women, a total hysterectomy, or panhysterectomy, may be performed; this procedure involves removal of the uterus, fallopian tubes and ovaries. However, removal of the ovaries may significantly impact on the woman's future 'cardiovascular, psychosexual, cognitive and mental health' and, therefore, this needs to be considered before removing the ovaries (Hickey, Ambekar & Hammond, 2010).

FAST FACTS

- Hysterectomy is a commonly performed elective surgery for Australian women.
- This surgery is most often performed in women who are between the ages of 40 and 44.
- The three conditions most associated with hysterectomy are uterine leiomyomas (fibroids), endometriosis and uterine prolapse (Women's Health Queensland Wide Inc., 2011).

Hysterectomy may involve an abdominal, vaginal or laparoscopic approach. The choice depends on the underlying disorder, the need to explore the abdominal cavity and the preference of the surgeon and woman. Nursing care of the woman undergoing a hysterectomy is described in the box below.

Abdominal hysterectomy is performed when a pre-existing abdominal scar is present, when adhesions are thought to be present or when a large operating field is necessary. The surgical incision may be either longitudinal, made in the midline from umbilicus to pubis, or a *Pfannenstiel incision*, also known as the bikini cut.

Vaginal hysterectomy, removal of the uterus through the vagina, is desirable when the uterus has descended into the vagina or if the urinary bladder or rectum have prolapsed into the vagina. Vaginal

NURSING CARE OF THE WOMAN having a dilation and curettage (D&C)

PREOPERATIVE CARE

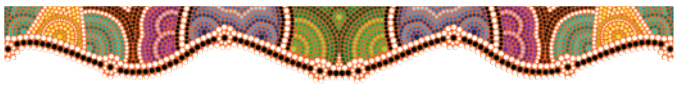
- Orient the woman to the environment.
- Provide education and reassurance.
- If indicated and possible, ask the woman to come in 24 hours before surgery for insertion of a laminaria tent. *This device absorbs cervical secretions and slowly dilates the cervix.*
- Instruct the woman to remain NBM (nil by mouth) after midnight on the day of surgery.

POSTOPERATIVE CARE

- Monitor circulation and sensation in the legs and avoid compression of the popliteal area. *The lithotomy position requires the woman's legs to be elevated, which can impair circulation.*
- Asses and monitor blood loss.
- Instruct the woman to use perineal pads and avoid using tampons for 2 weeks. *This reduces the risk of infection and allows tissues to heal.*
- Explain that the onset of the next menstrual period may be delayed.
- Explain that intercourse and anything inserted into the vagina should be avoided until after the postoperative checkup and after vaginal discharge has ceased. *This precaution reduces the risk of infection.*
- Instruct the woman to rest for several days after surgery, avoid heavy lifting and report any bleeding that is bright red or exceeds that of a normal menstrual period. *Vigorous activity, lifting or straining interferes with healing and may cause haemorrhage.*

hysterectomy leaves no visible abdominal scar. Laparoscopy-assisted vaginal hysterectomy (LAVH) is most often performed.

Laparoscopic hysterectomy is when a hysterectomy is performed by making three to four incisions in the abdomen. The organs can then be viewed by inserting the laparoscope through one of the incisions; the surgical instruments are then inserted through the remaining incisions. The benefits of this procedure are a shorter hospital stay and a quicker recovery (Women's Health Queensland Wide Inc., 2011).



Nursing care

DUB usually causes the woman anxiety. Her self-image, sexuality or reproductive capacity may be threatened, and she may fear the possibility of cancer. She may be embarrassed to discuss her menstrual history and hygiene practices.

Nursing diagnoses and interventions

Interventions for the woman with DUB commonly address problems with anxiety and sexual function.

Anxiety

The anxiety associated with abnormal uterine bleeding can be intense. Until the cause of the bleeding is identified and has been addressed, the woman may fear cancer or other life-threatening conditions.

- Discuss the results of tests and examinations with the woman. *This allows for open exchange of information.*
- Provide information about the causes, treatments, risks, long-term effects of treatments and prognosis. *This allows the woman to assume responsibility for her own health and become involved in her own treatment plan.*
- Evaluate coping strategies and psychosocial support systems. Teach coping strategies if indicated. The possibility of surgery or cancer represents a crisis for the woman and her support system. *Support groups can provide assistance for the woman through crisis intervention.*

Sexual dysfunction

The woman with DUB may be unwilling to express herself sexually, particularly if bleeding is frequent or heavy. Offer information about engaging in sexual activity during menstruation.

NURSING CARE OF THE WOMAN having a hysterectomy

PREOPERATIVE CARE

- Assess the woman's understanding of the procedure. Provide explanation, clarification and emotional support as needed. Reassure that the anaesthesia will eliminate any pain during surgery and that medication will be administered postoperatively to minimise discomfort. *The woman who understands the procedure to be performed and what to expect after surgery will be less anxious.*
- Check the chart to ensure that the consent form has been signed.
- Perform preoperative procedures as per policy.
- Administer preoperative medications as indicated.

POSTOPERATIVE CARE

- Assess for signs of haemorrhage. *Haemorrhage is more common after vaginal hysterectomy than after abdominal hysterectomy.*
- Monitor vital signs every 4 hours and measure intake and output. *These data are important indicators of haemodynamic status and complications.*
- Once the catheter has been removed, measure the amount of urine voided.
- Assess for complications, including infection, ileus, shock or haemorrhage, thrombophlebitis and pulmonary embolus.
- Assess vaginal discharge; provide health education about perineal care.
- Assess incision and bowel sounds regularly.
- Encourage turning, coughing, deep breathing and early ambulation.
- Encourage fluid intake.
- Teach to splint the abdomen and cough deeply. Instruct to restrict physical activity for 4 to 6 weeks. Heavy lifting, stair climbing, douching, tampons and sexual intercourse should be avoided. The woman should shower, avoiding tub baths, until bleeding has ceased. *Infection and haemorrhage are the greatest postoperative risks; restricting activities and preventing the introduction of any foreign material into the vagina helps reduce these risks.*
- Explain to the woman that she may feel tired for several days after surgery and needs to rest periodically.
- Explain that appetite may be depressed and bowel elimination may be sluggish. *These are after-effects of general anaesthesia, handling of the bowel during surgery and loss of muscle tone in the bowel while empty.*
- Teach the woman to recognise signs of complications that should be reported:
  a temperature greater than 37.7°C
  b vaginal bleeding that is greater than a typical menstrual period or is bright red
  c urinary incontinence, urgency, burning or frequency
  d severe pain.
- Encourage the woman to express feelings that may signal a negative self-concept. Correct any misconceptions. *Some women believe that hysterectomy means weight gain, the end of sexual activity and the growth of facial hair.*
- Provide information on risks and benefits of hormone replacement therapy (HRT), if indicated. *If the ovaries have also been removed, the woman is immediately thrust into menopause and may want or need HRT.*
- Reinforce the need to obtain gynaecological examinations regularly, even after hysterectomy.

Explain that conception is possible during this time (therefore birth control measures are required) and that orgasm may help relieve symptoms. *Orgasm causes a release of tension and vascular congestion, and frequently provides at least temporary relief of symptoms.*

- Provide an opportunity for the expression of concerns related to alterations in lifestyle and sexual functioning. Some women have had a prolonged period of sexual abstinence related to DUB. *Allowing women to verbalise concerns can assist them in working collaboratively with the healthcare provider to minimise the impact of illness and optimise function.*

CONSIDERATION FOR PRACTICE

If the nurse is not comfortable with frank discussions about sexual activities, referral is indicated. Nurses, however, should be open to discussing sexuality and sexual activities with women. If the nurse is not comfortable they cannot expect the woman to be.

Community-based care

Provide support, appropriate reassurance and information to help the woman and her family better understand her disorder and the therapeutic interventions indicated. Education on the following topics should be included:

- administration and side effects of prescribed medications, including iron
- the need to maintain a balanced diet, increasing iron-rich foods such as eggs, beans, liver, beef and shellfish. (Inform the woman that while orange juice may improve the absorption of iron, foods high in calcium and oxalic acid, such as spinach, may reduce its absorption.)
- importance of maintaining a fluid intake of 2 to 3 L a day
- the need to immediately report recurring episodes of DUB, particularly in postmenopausal women, to the healthcare provider
- the importance of the woman looking after her mental health.

STRUCTURAL DISORDERS

Structural disorders of the female reproductive system include displacement disorders and fistulas.

THE WOMAN WITH A UTERINE DISPLACEMENT

The uterus may be displaced within the pelvic cavity or may descend into the vaginal canal. Displacement of the uterus within the pelvic cavity is classified according to the direction of the displacement (see Figure 48.2):

- *Retroversion* of the uterus is a backwards tilting of the uterus towards the rectum.
- *Retroflexion* involves a flexing or bending of the uterine corpus in a backwards manner towards the rectum.
- *Anteversion* is an exaggerated forwards tilting of the uterus.
- *Anteflexion* is a flexing or folding of the uterine corpus upon itself.

Prolapse of the uterus into the vaginal canal can vary from mild to complete prolapse outside of the body. First-degree, or mild, prolapse involves a descent of less than half the uterine corpus into the vagina. Second-degree, or marked, prolapse involves the descent of the entire uterus into the vaginal canal, so that the cervix is at the introitus to the vagina. Third-degree prolapse, or *procidentia*, is complete prolapse of the uterus outside the body, with inversion of the vaginal canal (see Figure 48.3). Prolapse of the uterus is often accompanied by *cystocoele* (herniation of the bladder into the vagina) or *rectocoele* (herniation of the rectum into the vagina).

Pathophysiology

Displacement or prolapse of the uterus, bladder or rectum can be a congenital or an acquired condition. Congenital tilting or flexion of the uterus is rare. More commonly, tilting or flexion disorders in which the uterus remains within the pelvic cavity are related to the scarring and inflammation of pelvic inflammatory disease, endometriosis, pregnancy and tumours.

Downwards displacement of the pelvic organs into the vagina results from weakened pelvic musculature, usually attributable to stretching of the supporting ligaments and muscles during pregnancy and childbirth. Unrepaired lacerations from childbirth, rapid deliveries, multiple pregnancies, congenital weakness or loss of elasticity and muscle tone with ageing may contribute to these disorders.

Manifestations

The symptoms of displacement disorders are listed below.

MANIFESTATIONS Displacement disorders

UTERINE DISPLACEMENT WITHIN THE PELVIC CAVITY

- Dysmenorrhoea
- Backache
- Dyspareunia
- Infertility

UTERINE PROLAPSE

- Backache
- Urinary incontinence
- Bearing-down sensation
- Haemorrhoids
- Constipation
- Dyspareunia

CYSTOCOELE/RECTOCOELE

- Bearing-down sensation
- Haemorrhoids
- Constipation
- Urinary incontinence
- Faecal incontinence

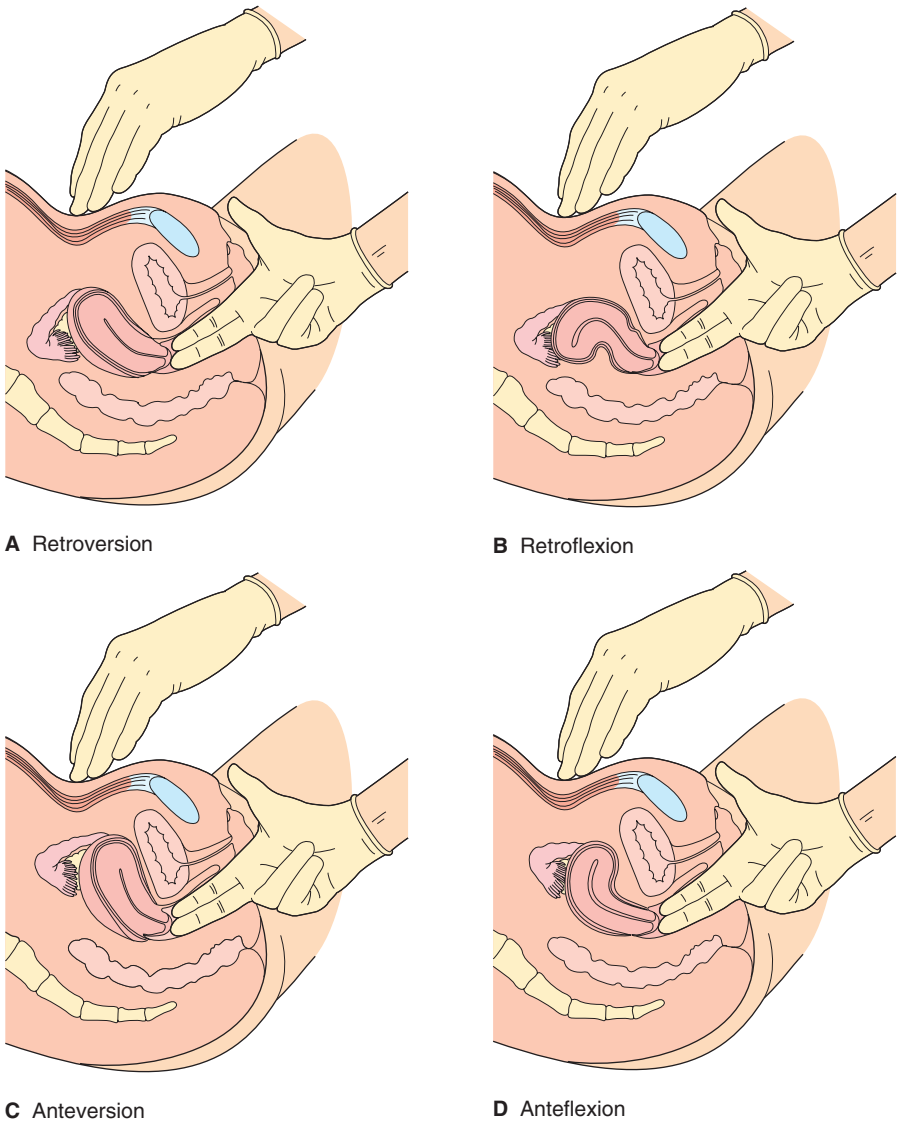


FIGURE 48.2 ■ Displacements of the uterus within the uterine cavity. *A*, Retroversion is a backwards tilting. *B*, Retroflexion is a backwards bending. *C*, Anteversion is a forwards tilting. *D*, Anteflexion is a forwards bending

INTERPROFESSIONAL CARE

Interprofessional care focuses on identifying the cause of the structural disorder, correcting or minimising the condition, relieving pain, preventing or treating infection, and supporting and educating the woman.

A careful history is taken and a physical examination is performed. Diagnosis of uterine displacement is made after physical examination. If herniation of the rectum or bladder is suspected, the woman is asked to bear down or cough during the examination so the prolapse can be palpated and any leakage of urine or faeces visualised. A history of infections, multiple pregnancies in rapid succession and rapid labours support this diagnosis.

Treatment may include Kegel (pelvic floor) exercises to strengthen weakened pelvic muscles. Kegel exercises can be useful in the early stages of downwards displacement. These exercises are discussed in Chapter 26.

Surgery

Several surgical procedures are used to repair structural disorders. For women presenting with a cystocoele, *anterior colporrhaphy* (repair of the cystocoele) is the most common procedure. The anterior repair shortens the pelvic muscles, providing tighter support for the bladder. The *Marshall–Marchetti–Krantz procedure* involves resuspension of the urinary bladder in correct anatomical position. A rectocoele is repaired with a posterior colporrhaphy, which shortens the pelvic muscles, providing a tighter support for the rectum.

A prolapsed uterus may be surgically repositioned and the supporting muscles shortened to provide greater support. In postmenopausal women or women with procidentia, hysterectomy is the preferred treatment.

Pessary

When surgery is contraindicated, a *pessary* (a removable device) may be inserted into the vagina to provide temporary support for the uterus or bladder. At regular intervals, the pessary is removed, cleaned and reinserted.

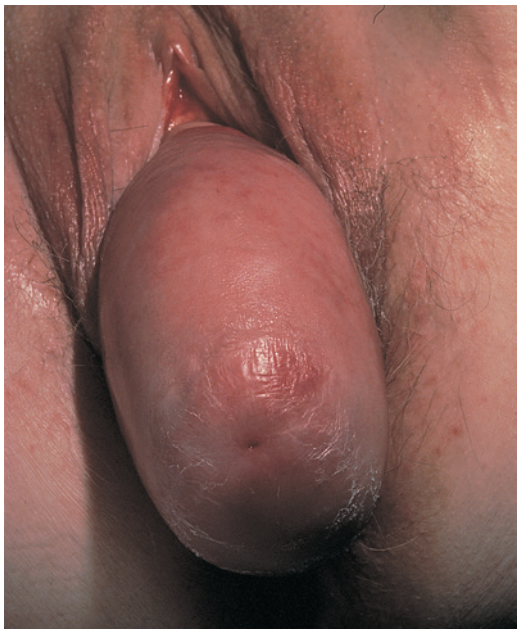


FIGURE 48.3 ■ Prolapse of the uterus can vary from mild to complete. In third-degree uterine prolapse, or procidentia, the uterus prolapses completely outside the body, with inversion of the vagina

Source: © M. English, MD/Custom Medical Stock Photo.

Nursing care

Nursing care focuses on education about the disorder, proposed treatments and self-care measures for relief of symptoms.

Nursing diagnoses and interventions

Nursing interventions for the woman with a displacement disorder address problems with urinary incontinence and anxiety.

Stress incontinence

Relaxation of the pelvic floor can lead to stress incontinence. This can prove both troublesome and embarrassing, and can increase the incidence of urinary tract infection.

- Teach Kegel exercises. These exercises strengthen perineal muscle tone, minimise urinary leakage and minimise descent of the bladder and rectum into the vagina. *In postmenopausal women, oestrogen supplements also can improve muscle tone in the perineal area.*
- Suggest the use of perineal pads (ranging from thin panty liners to full-thickness incontinence pads) or special underwear (such as Depend) to absorb urine leakage. *Using pads or undergarments often allows the woman to once again take part in her usual social activities.*
- Explain perineal care and proper use of perineal pads. Cleansing the perineum from front to back and applying and removing perineal pads the same way minimises cross-infection from the anus to the vaginal and urethral openings. *Incontinence pads need to be changed frequently to minimise surface bacterial counts.*
- Suggest reducing or eliminating caffeine intake. *Reducing caffeine intake can reduce urinary frequency and urgency.*
- Stress the importance of cleaning the perineal area. *Urine is very irritating to the skin.*

Anxiety

Anxiety is common in women with a displacement disorder. Many women have a limited understanding of their reproductive anatomy. This lack of knowledge often compounds the anxiety. The nurse can use models to explain structural disorders and treatment options available.

- Encourage questions from the woman and her partner. *This helps assess the level of understanding so that health education can be more effective.*
- Explain that the relief from discomfort and fatigue may positively influence sexual expression and reassure the woman that the capacity for orgasm will not be affected. *Many women and their partners have major concerns about the effects of the disorder and its treatment on their sex life and capacity for sexual pleasure.*
- Explore coping mechanisms that have been previously successful. *This can help relieve anxiety and boost selfesteem.*

Community-based care

If surgery is the treatment of choice, health education centres on what to expect in the preoperative and postoperative periods. If medical treatment is used initially, education focuses on measures to relieve the symptoms, such as Kegel (pelvic floor) exercises, use of incontinence pads or the use, care and insertion of a pessary.

As obesity is a risk factor associated with relaxation of the pelvic and abdominal muscles, dietary counselling may be indicated. Preoperatively, a diet high in fibre may alleviate constipation, a particular concern during the postoperative period.

THE WOMAN WITH A VAGINAL FISTULA

A fistula is an abnormal opening or passage between two organs or spaces that are normally separated, or an abnormal passage to the outside of the body. Vaginal fistulas may be vesicovaginal or rectovaginal. A *vesicovaginal fistula* is an abnormal opening between the urinary bladder and the vagina, leading to incontinent leakage of urine through the vagina. A *rectovaginal fistula* (less common) is an abnormal opening between the rectum and vagina, causing incontinent leakage of stool or flatus through the vagina.

Vesicovaginal or rectovaginal fistulas may develop as a complication of childbirth, gynaecological or urological surgery, or radiation therapy for gynaecological cancer. Cancer of the bladder is sometimes involved. The woman with a vaginal fistula often presents with a complaint of involuntary leakage of urine or flatus and symptoms of infection.

INTERPROFESSIONAL CARE

Fistulas are diagnosed by pelvic examination. Diagnosis of a vesicovaginal fistula can be made by instilling dye into the urinary bladder through a catheter and observing the vagina for leakage. If no leakage is detected, a tampon or vaginal pack is inserted into the vagina and the woman is asked to ambulate. If an abnormal opening is present, the tampon will absorb the dye. Dye may also be injected intravenously because it is excreted by the kidneys. Urine and vaginal cultures may be performed to rule out infections. Antibiotics are administered if infection is present.

A small vaginal fistula may resolve spontaneously. Otherwise, surgery is performed after inflammation has subsided—often a period of several months. Rarely, in the presence of a large, highly inflamed rectovaginal fistula, a temporary colostomy is performed, allowing inflammation and irritation to subside (see Chapter 23).

Nursing care

Nursing care for the woman with repair of a vaginal fistula is similar to that for the woman with a displacement disorder. Health education is an important component of nursing care. Stress the importance of careful perineal cleansing to reduce irritation and prevent further tissue breakdown. Perineal pads or special underwear may be used to absorb urine or faecal drainage. For the woman with a rectovaginal fistula, provide information about avoiding gas-forming foods to minimise embarrassment from odour.

DISORDERS OF FEMALE REPRODUCTIVE TISSUE

Both benign and malignant tissue disorders affect the female reproductive system. Benign tumours and cysts include Bartholin's gland cysts, cervical polyps, endometrial cysts and polyps, ovarian cysts and uterine leiomyomas (fibroids). Endometriosis is a condition in which endometrial tissue implants outside the uterus in various locations in the pelvic cavity. Malignant tumours of reproductive tissue include cervical cancer, endometrial cancer, ovarian cancer and vulvar cancer.

THE WOMAN WITH CYSTS OR POLYPS

A *cyst* is a fluid-filled sac. A *polyp* is a highly vascular solid tumour attached by a pedicle or stem. Cysts or polyps of the female reproductive system can occur in the vulva, cervix, endometrium or ovaries.

Pathophysiology

Following are different types of female reproductive tissue cysts and polyps:

- *Bartholin's gland cysts* are the most common cystic disorder of the vulva. These cysts are caused by the infection or obstruction of Bartholin's gland.
- *Cervical polyps* are the most common benign cervical lesion in women of reproductive age. These polyps tend to occur in women over age 40 who have borne several children and have a history of using oral contraceptives. It is possible that cervical polyps develop from endocervical hyperplasia. The polyp develops at the vaginal end of the cervix, has a stem and is highly vascular.
- *Endometrial cysts and polyps* are caused by endometrial overgrowth and are often filled with old blood. (The dark colour leads to the label 'chocolate cysts'.) Endometrial cysts are the result of endometrial implants on the ovary and are associated with endometriosis. Endometrial polyps, in contrast, are intrauterine overgrowths, similar to cervical polyps, and usually have a stalk.
- *Ovarian cysts* are classified as follicular cysts and corpus luteum cysts. Follicular cysts develop as a result of failure of the mature follicle to rupture or failure of an immature follicle to reabsorb fluid after ovulation. Corpus luteum cysts develop as a result of increased hormone secretion by the corpus luteum after ovulation. Most functional cysts regress spontaneously within two or three menstrual cycles.
- *Polycystic ovarian syndrome* (POS, also known as *Stein–Leventhal syndrome*) is an endocrine disorder characterised by an excess of androgens and a long-term lack of ovulation. The exact cause is unknown. As a part of the disease, as many as 8 to 10 cysts form in the ovaries from a failure to release ova. Symptoms include amenorrhoea or irregular menses, hirsutism, obesity, acne, hypertension, sleep apnoea and infertility. Women with POS often have insulin resistance and are at increased risk of early-onset type 2 diabetes, as well as heart disease, breast cancer and endometrial cancer.

Manifestations and complications

The causes and symptoms of benign cysts and polyps of the female reproductive system are presented in Table 48.1. Complications associated with these disorders include infection, rupture, infertility, haemorrhage and recurrence.

INTERPROFESSIONAL CARE

Care focuses on identifying and correcting the disorder and preventing its recurrence. A careful history is taken and a physical examination is performed, including inspection and visualisation. Examination of the reproductive tract reveals the

TABLE 48.1 Benign cysts and polyps of the female reproductive system

SITE	TYPE	AETIOLOGICAL ORIGIN	SYMPTOMS
Ovary	Functional cysts	Ovulation—include follicular cysts and corpus luteum cysts	May resolve spontaneously; can cause pain, menstrual irregularity or amenorrhoea
	Polycystic ovarian syndrome	Unknown; possible hypothalamic–pituitary dysfunction	Hirsutism, obesity; amenorrhoea or irregular menses; hyperinsulinaemia; infertility
Vulva	Bartholin cysts	Obstruction or infection of Bartholin's gland	Pain, redness, perineal mass, dyspareunia
Endometrium	Chocolate cysts	Endometrial overgrowth; filled with old blood	
	Endometrial polyps	Unknown	Bleeding between periods
Cervix	Cervical polyps	Unknown	Bleeding after intercourse or between periods

presence of most cysts and polyps. The menstrual history may reveal menstrual irregularities.

Diagnosis

Diagnostic tests that may be used to diagnose cysts and polyps of the female reproductive system include laparoscopy to visualise ovarian cysts, ultrasound or x-ray to differentiate cysts from solid tumours and a pregnancy test when luteal cysts are suspected. Laboratory analysis will demonstrate elevated LH and testosterone levels, as well as a reverse in FSH/LH in the woman with POS.

Medications

Antibiotics are used to treat infection or abscess, and oral contraceptives are used to promote regression of functional ovarian cysts. Clomiphene (Clomid, Serophene) may be prescribed to stimulate ovulation in the woman with POS who wishes to become pregnant. Dexamethasone suppresses ACTH and adrenal androgens and may be added to increase the likelihood of ovulation.

Surgery

Cervical polyps are visible through a vaginal speculum and usually are removed with a clamp, using a twisting motion. To remove endometrial cysts or polyps, a transcervical approach is used. The specimen is sent to the laboratory for evaluation and chemical or electrical cauterisation is applied after cyst removal. For Bartholin's gland cysts and any abscesses, the lesion is incised and drained and a drainage device is left in place. Follicular cysts may be punctured through laser surgery or a wedge resection of the ovary may be performed to restore ovulation. Rarely, *oophorectomy* (removal of the ovary) is performed if the cysts are very large.

Nursing care

Nursing care focuses on relieving pain and preventing recurrence and complications. Address the following topics for self-care at home:

- the condition, its treatment and measures to relieve pain
- the importance of keeping follow-up appointments
- symptoms of infection (for post-surgical care) and the need to notify the health professional should they occur
- if cervical polypectomy is performed, advise use of external pads for 1 week. The woman must be able to state the signs of excessive bleeding and recognise that saturating more than one pad in an hour indicates the need for immediate follow up
- the importance of long-term follow-up care for the woman with POS.

THE WOMAN WITH LEIOMYOMA

Leiomyomas (*fibroid tumours*) are benign tumours that originate from smooth muscle of the uterus. They are the most common form of pelvic tumour, believed to occur in 60% of women older than 45 years of age (Okolo, 2008).

Pathophysiology

The actual cause of fibroid tumours is not clearly understood, but there is a strong association with oestrogen stimulation. Fibroid tumours usually develop in the uterine corpus and may be intramural, subserous or submucous (see Figure 48.4):

- *Intramural fibroid tumours* (the most common type) are embedded in the myometrium. They usually present as an enlargement of the uterus.
- *Subserous fibroid tumours* lie beneath the serous lining of the uterus and project into the peritoneal cavity. They may become pedunculated (on a stem) and displace or compress other tissues, such as the ureter or bladder.
- *Submucous fibroid tumours* lie beneath the endometrial lining of the uterus. They displace endometrial tissue and are more likely to cause bleeding, infection and necrosis than the other types.

Manifestations

Small tumours may be asymptomatic. The rate of growth varies, but they may increase in size during pregnancy or with use of oral contraceptives or HRT. Large fibroid tumours can crowd other organs, leading to pelvic pressure, pain, dysmenorrhoea, menorrhagia and fatigue. Depending on the location of the tumour, constipation and urinary urgency and frequency may occur. Most fibroid tumours shrink with menopause.

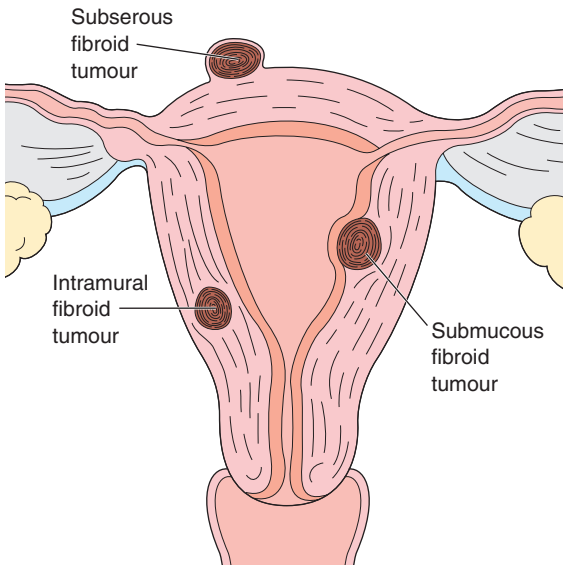


FIGURE 48.4 ■ Types of uterine fibroid tumours (leiomyomas). Intramural fibroid tumours lie within the uterine wall. Subserous fibroid tumours lie beneath the serous lining of the uterus and project into the peritoneum. Submucous fibroid tumours lie beneath the endometrial lining of the uterus

INTERPROFESSIONAL CARE

Treatment of the woman with uterine fibroids depends on the size and location of the tumours, the severity of the symptoms and her age and childbearing status. Tests used to diagnose uterine fibroids may include ultrasound to differentiate leiomyoma from endometriosis, and laparoscopy to visualise subserosal leiomyomas.

In asymptomatic women who wish to bear children, the fibroid tumours are monitored. Follow up is recommended two to three times per year to monitor growth.

Medications

Leuprolide acetate (Lupron) is used to decrease the size of the tumour if surgery is contraindicated or not desired. Gonadotropin-releasing hormone (GnRH) agonists are also administered.

Surgery

Myomectomy, removal of the tumour without removing the entire uterus, is the surgical procedure of choice for young women who wish to retain reproductive capability. Laparoscopic laser technique is used for many women. A hysterectomy is performed if tumours are large and if bleeding or other problems continue in perimenopausal women. A hysterectomy usually requires a hospital stay of 3 to 4 days and a 6-week recovery time. A non-surgical method of treatment is a *uterine fibroid embolisation*. In this procedure, a catheter is guided through the femoral artery to the uterus, where tiny particles are injected into the artery supplying the fibroid to cut off the fibroid's blood supply.

Nursing care

If surgery is deferred, health education emphasises the importance of regular follow-up assessments to monitor tumour growth. If a hysterectomy is performed, education emphasises appropriate preoperative and postoperative care. Dietary modifications to increase iron intake, prevent constipation and promote healing are important.

THE WOMAN WITH ENDOMETRIOSIS

Endometriosis is a condition in which multiple, small, usually benign implantations of endometrial tissue develop most commonly in the pelvic cavity, but also occasionally in other areas of the body, such as the lungs. Endometriosis affects 10–15% of women of childbearing age and is more common in women who postpone childbearing. Risk factors for endometriosis include early menarche, regular periods with a cycle of less than 27 days, menses lasting more than 7 days, heavier flow, increased menstrual pain and a history of the condition in first-degree female relatives.

Pathophysiology

The cause of endometriosis is unclear, but several theories have been proposed. The metaplasia theory asserts that endometrial tissue develops from embryonic epithelial cells as a result of hormonal or inflammatory changes. The theory of retrograde menstruation suggests that menstrual tissue backs up through the fallopian tubes during menses, implants on various pelvic structures and survives. The transplantation theory asserts that endometrial implants spread via lymphatic or vascular routes.

The abnormally located endometrial tissue responds to cyclical ovarian hormone stimulation, and bleeding at the time of menstruation occurs at the sites of implantation. Scarring, inflammation and adhesions may develop. Endometriosis is a slowly progressive disease, responsive to ovarian hormone stimulation. Thus, the implants regress during pregnancy and atrophy at menopause unless the woman is receiving HRT. Because progressive scarring may interfere with the ability to conceive, women with significant endometriosis are encouraged to have children early if they wish to do so.

Manifestations

Symptoms of endometriosis, which usually occur during the luteal phase of the menstrual cycle, are summarised below.

INTERPROFESSIONAL CARE

Endometriosis may be difficult to diagnose, but a history of dysmenorrhoea, dyspareunia and infertility strongly suggests this diagnosis. Interventions depend on the severity of

MANIFESTATIONS Endometriosis

- Heavy, throbbing pain of the lower abdomen and pelvis, radiating down the thighs and around the back. (The degree of pain, however, is not indicative of the severity of the disease.)
- Feeling of rectal pressure and discomfort when having a bowel movement.
- Dyspareunia.
- Dysfunctional uterine bleeding.
- Infertility.

symptoms, the extent of the disease and the woman's age and desire for childbearing. Treatment goals focus on pain management and restoring fertility.

Diagnosis

Diagnostic tests are ordered to rule out other medical conditions and identify the endometrial implants. The tests include pelvic ultrasound and laparoscopy (see Chapter 46), as well as an FBC with differential to rule out pelvic abscesses and infectious processes. A low haemoglobin and haematocrit may be noted if menorrhagia accompanies endometriosis or tissue implants bleed significantly during menses. A definitive diagnosis can be made when a laparoscopy is performed and a biopsy of tissue is undertaken (Endometriosis Australia, 2014).

Medications

Medications include analgesics to control pain and prostaglandin synthesis inhibitors such as NSAIDs. Hormone therapy may include oral contraceptives or progesterone to induce pseudopregnancy or danazol (Azol) to induce amenorrhoea and involution of endometrial tissue. Prolonged use of danazol, however, may result in masculinising effects. GnRH is used to elevate levels of oestrogen and progesterone and minimise bleeding.

Surgery

Surgical interventions include laparoscopy with laser ablation (excision or removal) of endometrial implants. Refractory endometriosis may be treated with total hysterectomy.

Nursing care

Nursing care includes providing pain relief, engaging the woman in health education about her condition and the treatment options, and helping her cope with treatment outcomes. The severity of the disease and its symptoms are not necessarily related. Advanced disease may exhibit few symptoms, whereas early disease may be quite painful. A nursing care plan for a woman with endometriosis is found below.

Nursing diagnoses and interventions

Interventions for pain, discussed previously, are also appropriate for the woman with endometriosis. A priority for care for women with this disorder is anxiety related to the risk of loss of reproductive function.

Anxiety

Anxiety about the unsure prognosis related to infertility is a particular problem for young women who plan to have a family in the future.

- Encourage expression of fears and anxiety about infertility and answer questions honestly and based on evidence. *Knowledge helps relieve anxiety and fear.*
- Provide information on fertility awareness methods, including measurement of basal body temperature and other techniques for recognising ovulation. *Understanding these techniques helps the woman optimise the conditions for conception.*

NURSING CARE PLAN A woman with endometriosis

Joy Smith is a 29-year-old school teacher. She has been in a permanent relationship with her partner for the past 4 years. Joy describes a history of severe dysmenorrhoea and menorrhagia, a feeling of pelvic heaviness and pain that radiates down her thighs. Joy is seeking assistance with the above symptoms, as she is not coping with the ongoing effect of the symptoms on her health and lifestyle. She also discusses her own and her partner's desire to have children. However, at the current time she is reluctant to have sex as she experiences severe discomfort and pain. Her partner has been supportive. However, it is affecting their relationship. Following review, endometriosis is suspected and a diagnostic laparoscopy has been scheduled.

ASSESSMENT

Registered Nurse and Nurse Practitioner Christian Bright interviews Joy and makes the following assessments: BP 110/70, P 68, R 18, T 36.7°C. Joy's weight is 59 kg and within normal limits for her height. Review of laboratory findings indicates a haemoglobin level of 98 g/L (normal range: 115 to 165 g/L) and a haematocrit of 33.1% (normal range: 38% to 47%). Physical examination reveals pelvic tenderness on manipulation of the cervix and small masses that are palpable on abdominal/pelvic examination.

DIAGNOSES

- *Chronic pelvic pain* related to the endometriosis and endometrial pelvic implants.
- *Anxiety* related to effect of endometriosis on fertility.
- *Deficient knowledge* related to diagnosis and treatment options.
- *Ineffective sexuality pattern* related to the symptoms of endometriosis.

NURSING CARE PLAN A woman with endometriosis (continued)

PLANNING

- Identify the location, type, duration and history of the pain.
- Recommend analgesics and heat therapy.
- Provide information on biofeedback, relaxation and imagery to lessen pain.
- Discuss with Joy and her partner, David, the causes of endometriosis and its symptoms.
- Encourage Joy and David to discuss their feelings about the effect of the disease on their sex life, lifestyle and fertility.
- Refer the couple to appropriate counselling if required.
- Refer the couple to support organisations such as Endometriosis Australia www.endometriosisaustralia.org/.

Expected outcomes

- Develop effective self-care measures to deal with the pain and discomfort.
- Verbalise decreased anxiety.
- Demonstrate understanding of the disease and treatment options.
- Verbalise an improvement in sexual functioning and a decrease in interpersonal stress between Joy and her partner.

IMPLEMENTATION

- Initiate each of the steps outlined in the planning section detailed above.
- Monitor the effectiveness of the plan by monitoring Joy's response to:
  - analgesics, heat therapy and biofeedback
  - relaxation and imagery to lessen pain.
- Ongoing discussion with Joy and David about their sexual relationship and infertility issues.

EVALUATION

Two years after the initiation of treatment, Joy and David have become parents of a baby girl. Joy states that the discomfort and other symptoms of endometriosis have eased. Relaxation and imagery have effectively minimised her pain and brought about improvement in her function as a mother and sexual partner. Counselling has improved the interpersonal and sexual relations between Joy and David. Dietary management has improved her anaemia, although the menorrhagia persists. Joy and David are trying to have a second baby. They will be followed up and referred to an infertility clinic if conception does not occur within 1 year.

CRITICAL THINKING IN THE NURSING PROCESS

1. Explain the pathophysiological basis for Joy's anaemia.
2. How would you handle the situation if Joy and David were extremely uncomfortable and embarrassed about discussing their sexual problems?
3. Develop a plan of care for Joy for *Situational low self-esteem* related to the symptoms of endometriosis.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from Joy's case study. How would you incorporate this into your future nursing practice?
2. Which strategies would you use in your future nursing practice to assist women who are experiencing endometriosis and subsequent infertility?

Community-based care

Explain the cause of the disorder and the various treatment options, including their side effects. Discuss fertility awareness methods and the risks and benefits of long-term use of oral contraceptives. Stress the importance of regular exercise, smoking cessation and weight control. If surgical treatment is chosen, provide preoperative and postoperative education.

THE WOMAN WITH CERVICAL CANCER

Cancer of the cervix is the second most common cancer in women worldwide and the fourteenth most common in women in Australia (Cancer Australia, 2015a). The lower incidence in Australia is primarily the result of screening with Pap tests and the introduction of the HPV vaccine Gardasil, which has been shown to be 100% effective against HPV types 6, 11, 16 and 18, which are responsible for 70% of cervical cancer cases and 90% of genital warts in women (Australian Institute of Health and Welfare (AIHW), 2011).

The incidence of diagnosis with cervical cancer prior to 85 years of age is one in 62 women (Cancer Council Australia, 2015a) However, it is important to remember that cervical cancer begins to appear in women in their twenties. According to the AIHW (2011), effective screening with the Pap smear test and treatment have reduced the death rate in women as a result of cervical cancer. However, the mortality rate remains high for Aboriginal and Torres Strait Islander women (see 'Fast facts' box), and health professionals need to provide health education and work towards lowering this unacceptable mortality rate (AIHW, 2013).

Risk factors

Risk factors for cervical cancer include first intercourse before 16 years of age, multiple sex partners or male partners with multiple sex partners, a history of sexually transmitted infections and infection with HIV. The most important risk factor is infection with HPV. Other risk factors include smoking and poor nutritional status, family history of cervical cancer and exposure to diethylstilbestrol (DES) in utero.

Pathophysiology

Most cervical cancers (90%) are squamous cell carcinomas that begin as neoplasia in the cervical epithelium. *Precancerous dysplasia* (*cervical intraepithelial neoplasia (CIN)*, *cervical carcinoma in*

FAST FACTS

- The Australian Institute of Health and Welfare (AIHW) (2011) reports that cervical cancer rates in Australia are at a historic low. Incidence rates have decreased since the introduction in 1991 of the National Cervical Screening Program (NCSP). However, for people from Aboriginal and Torres Strait Islander heritage, the incidence rates are double that of the non-Indigenous population. The mortality rate for cervical cancer is five times higher in Aboriginal and Torres Strait Islander women in comparison with the non-Indigenous population.
- Nearly 100% of women with cervical cancer have evidence of cervical infection with human papillomavirus (HPV) (see Chapter 49).
- The National Cervical Screening Program for Australian women recommends that all women aged 18 to 69 years who have ever been sexually active, whether vaccinated or unvaccinated, should have cervical screening by Pap smear every 2 years (Department of Health, 2012).

situ) is estimated to occur in one of eight women before the age of 20, and is often associated with HPV infection. Studies have also found a strong association with reproductive infections with *Chlamydia trachomatis*. (These infections are discussed in Chapter 49.) The precursor lesions may spontaneously regress (60%), persist (30%) or progress and undergo malignant change (10%). Only about 1% become invasive. Systems of grading of dysplastic changes in the cervix use the term *cervical intraepithelial neoplasia (CIN)* or the Bethesda system (see Table 48.2). Carcinoma in situ is localised; invasive cancer spreads to deeper layers.

Cancer in situ most often develops in the transformation zone where the columnar epithelium of the cervical lining meets the squamous epithelium of the outer cervix and vagina. Squamous cell cancers spread by direct invasion of accessory structures, including the vaginal wall, pelvic wall, bladder and rectum. Although metastasis is most frequently confined to the pelvic area, distant metastasis may occur through the lymphatic system.

Manifestations

Pre-invasive cancer is limited to the cervix and rarely causes symptoms. Invasive cancer causes vaginal bleeding after intercourse or between menstrual periods, and a vaginal discharge that increases as the cancer progresses. These changes are subtle and may be more readily noticed by the postmenopausal woman. Symptoms of advanced disease include referred pain in the back or thighs, haematuria, bloody stools, anaemia and weight loss.

INTERPROFESSIONAL CARE

The goals of treatment are to eradicate the cancer and minimise complications and metastasis. The type of treatment depends on the degree of malignant change, the size and location of the lesion and the extent of metastasis.

Diagnosis

Diagnostic tests used to diagnose cervical cancer include a Pap smear, colposcopy and cervical biopsy. (See Chapter 46 for further information about these procedures.) A loop diathermy technique (loop electrosurgical excision procedure (LEEP)) allows simultaneous diagnosis and treatment of dysplastic lesions found on colposcopy. This procedure is performed in the healthcare provider's office, using a wire for both cutting and coagulation during excision of the dysplastic region of the cervix. An MRI or CT of the pelvis, abdomen or bones may be performed to evaluate the spread of the tumour.

Medications

Chemotherapy is used for tumours not responsive to other therapy, tumours that cannot be removed or as adjunct therapy if metastasis has occurred (see Chapter 13).

Surgery

When combined with colposcopy, laser surgery is a viable treatment method provided that the cancer is limited to the cervical epithelium. Cryosurgery, which involves the use of a probe to freeze tissue, causing necrosis and sloughing, is also used for non-invasive lesions. Conisation (see Figure 48.5) is performed to treat microinvasive carcinoma when colposcopy

TABLE 48.2 Classification systems for Pap smears

DYSPLASIA/NEOPLASIA	CIN (CERVICAL INTRAEPITHELIAL NEOPLASIA)	BETHESDA SYSTEM	NUMERICAL
Benign	Benign	Normal	1
Benign with inflammation	Benign with inflammation	Normal	2
		Atypical squamous cells of undetermined significance (ASC-US)	
Moderate dysplasia	CIN I	Low-grade squamous intraepithelial lesion (SIL)	3
Severe dysplasia	CIN II	High-grade SIL	3
Carcinoma in situ	CIN III	High-grade SIL	4
Invasive cancer	Invasive cancer	Invasive cancer	5

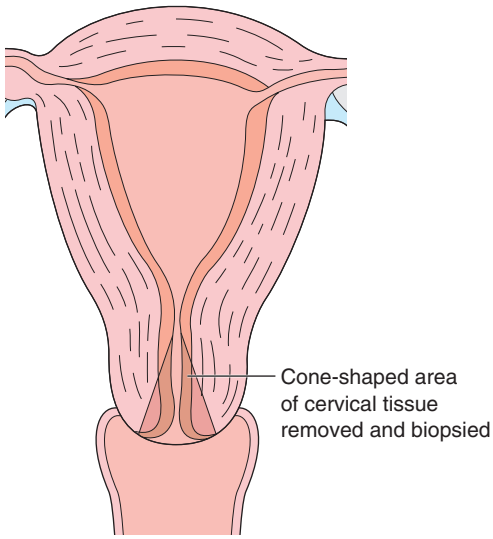


FIGURE 48.5 ■ Conisation, the surgical removal of a cone-shaped section of the cervix, is used to treat microinvasive carcinoma of the cervix

cannot define the limits of the invasion. For invasive lesions, hysterectomy or radical hysterectomy (removal of the uterus, fallopian tubes, lymph nodes and ovaries) is performed.

A *pelvic exenteration*, the removal of all pelvic contents including the bowel, vagina and bladder, is performed if the cancer recurs without involvement of the lymphatic system. An anterior exenteration is the removal of the uterus, ovaries, fallopian tubes, vagina, bladder, urethra, and lymphatic vessels and nodes. An ileal conduit is created for excretion of urine. A posterior exenteration is the removal of the uterus, ovaries, fallopian tubes, bowel and rectum. A colostomy is created for excretion of faeces (see Chapter 23).

Radiation therapy

Radiation therapy is used to treat invasive cervical cancer. External radiation beam therapy and intracavity cesium irradiation can be used. Radiation is discussed in Chapter 13.

Nursing care

Nursing care involves helping the woman deal with the physical and psychological effects of a potentially life-threatening illness, providing information needed to make informed decisions and minimising the adverse effects of therapy. Pain relief measures are important, as is counselling for the woman and her family. The woman should be educated and encouraged to perform self-care activities and to resume normal everyday activities and sexual functioning to the extent possible. A nursing care plan for a woman with cervical cancer is found below.

NURSING CARE PLAN A woman with cervical cancer

Kay Young is a 45-year-old divorced mother of two children aged 15 and 13. She was married at age 28 and had several sexual partners prior to her marriage. She has had three sexual partners since her marriage ended. Last year she was treated with cryosurgery for genital warts. A Pap smear taken 2 weeks ago showed atypical cells and she has come in for a repeat test.

ASSESSMENT

Laura Jones, Registered Nurse and Nurse Practitioner, the admitting nurse, interviews Kay and records the following assessment findings: BP 130/80, P 72, R 18, T 37.3°C. Kay weighs 64.5 kg. Examination of the cervix reveals a large necrotic lesion at the 7 o'clock position. She has reduced her smoking to less than 10 cigarettes per day and she does not drink alcohol.

Kay is extremely fearful and anxious, and has told no one about her abnormal Pap smear. She reveals that she has had back pain radiating down her thighs for several months and a foul vaginal discharge that increases after intercourse. Until 2 weeks ago, she had not had a Pap smear for 5 years. Laura Jones performs a repeat Pap smear, which is positive for squamous cell carcinoma of the cervix. A CT scan and lymphangiography are scheduled. Laparoscopy shows the disease to be widespread in the pelvic cavity.

DIAGNOSES

- *Personal conflict* related to Kay having to make a decision about the various treatment options that she could undertake.
- *Chronic and acute pain* related to disease progress and the management of the metastasis, including recovering from surgery.
- *Risk of impaired skin integrity* related to side effects of the treatment options such as radiation.
- *Fear for her wellbeing and future* related to diagnosis and management of cervical cancer.
- *Anticipatory grieving* related to potential loss of life and the effect of this on her children.
- *Anxiety* related to her diagnosis and prognosis, and the future wellbeing of her two children.

PLANNING

- Discuss treatment alternatives, including the prognosis with each option.
- Administer pain medications as prescribed.
- Examine skin surfaces daily before and after radiation therapy.
- Provide information on biofeedback training and relaxation techniques for control of moderate pain.
- Refer to a local cancer support group so that she can interact with cancer survivors. (If in a rural or remote

(continued)

NURSING CARE PLAN A woman with cervical cancer (continued)

area provide resources and/or online and/or telephone contacts.)
- Refer Kay to a social worker in preparation for her altered level of functioning and for assistance with finding care for her two children while she is undergoing treatment. (Kay lives in an urban area in Australia. Social worker services might not be immediately available if Kay lived in a rural or remote rural area.)

Expected outcomes
- Gain knowledge to make informed decisions about treatment options.
- Develop strategies for pain control.
- Maintain skin and tissue integrity during radiation treatment.
- Express her feelings about the fear of cancer and death, and her concerns related to her children.
- Develop effective coping strategies for dealing with life-threatening illness and pain.

IMPLEMENTATION
- Initiate each of the steps outlined in the planning section detailed above.
- Monitor the effectiveness of the plan by monitoring Kay's response to:
  - analgesics
  - radiation therapy.
- Ongoing discussion with Kay about the implementation of the plan and her psychosocial wellbeing.

EVALUATION
Kay has begun radiation therapy following pelvic exenteration. She controls her pain with relaxation and imagery techniques, requiring only occasional analgesics. She uses a water-based lotion to soothe the skin surface and is careful not to remove the skin markings. She seems optimistic and has quit smoking. She and her family have continued to attend the cancer support group meetings. Kay is planning for the future and has talked with her children and extended family about what it means to live with cancer.

CRITICAL THINKING IN THE NURSING PROCESS
1. Compare and contrast your education plan for health promotion interventions to decrease the risks of cervical cancer for a young woman aged 17 and an older woman aged 70. Would they differ and, if so, how?
2. Develop an education plan to help Kay cope with the effects of radiation.
3. During a home visit, Kay informs the nurse that she has been so tired since beginning radiation treatments that all she can do is sit in her chair. Design a plan of care for assisting Kay to cope with *Fatigue*.

REFLECTION ON THE NURSING PROCESS
1. Outline what you have learned from Kay's case study. How would you incorporate this into your future nursing practice?
2. Which strategies would you use in your future nursing practice to assist women who are undergoing treatment for cervical cancer?

Health promotion

The National Cervical Screening Program in Australia recommends that women should begin annual screening for cervical cancer with the Pap test every 2 years from age 18 (or 2 years after first sexual intercourse—whichever is later) and to continue throughout their life until age 69. Women who are 70 years of age and older and who have had three or more normal Pap smears in the past 10 years may be advised by their general practitioner that it is safe to stop having Pap smears (Department of Health, 2012). Screening should be done every year with regular Pap tests, or every 2 years using liquid-based tests. Alternately, cervical cancer screening with HPV DNA tests and Pap tests may be performed every 3 years. Screening for women who have had a total hysterectomy (including the cervix) is not recommended unless the surgery was done as a treatment for cancer. Women who have had a hysterectomy without removal of the cervix should continue to follow National Cervical Screening Program guidelines.

It is vital that nurses educate women of all ages about controlling risk factors for cervical cancer and about the importance of screening for this cancer throughout the lifespan. Educate young women about the relationship between early sexual activity, multiple partners and risk of STIs and cervical cancer. Discuss safer-sex alternatives and using condoms for protection. Emphasise the importance of continued screening examinations for the older woman who may not see a health professional on a regular basis. Encourage women under the age of 26 to receive Gardasil, the vaccine developed to prevent cervical cancer, precancerous genital lesions and genital warts due to HPV types 6, 11, 16 and 18. Gardasil is given as three injections over a 6-month period and does not protect against HPV in a woman already infected. For this reason, the Therapeutic Goods Administration in Australia recommends that the vaccine be routinely given to girls aged 12 and 13 (Department of Health, 2011).

Assessment

Collect the following data through a health history and physical examination (see Chapter 46):
- *Health history:* history of STIs, sexual history, partner's sexual history, family history of cervical cancer, vaginal bleeding or discharge, smoking history, maternal treatment with DES.
- *Physical assessment:* pelvic examination, abdomen, lymph glands.
- *Psychological assessment:* anxiety, coping strategies and stress levels.

Nursing diagnoses and interventions

This section discusses nursing interventions for the woman who has been diagnosed with cervical cancer and requires surgical and/or radiation treatment. Other interventions that may be appropriate for the woman with cervical cancer are discussed in the sections on other female reproductive system cancers.

Fear

Many people believe that cancer equals death; however, this is no longer true in many cases, especially with early diagnosis. For cervical cancer that is diagnosed at an early stage, the 5-year survival rate is 92%. If the disease is in situ, the rate is nearly 100% (Department of Health, 2009).

- Explain that 92% of all women with cervical cancer survive for 5 years or more and that the earlier the cancer is detected, the better the prognosis. *This gives the woman hope, an essential ingredient in recovery.*
- Allow adequate time for the woman and her family to express their concerns and ask questions. *Unexpressed feelings and fears and lack of understanding may cause the woman to view the situation as worse than it is.*
- Refer to cancer counsellor or support groups for additional information. *Cancer survivors who visit people in the hospital provide proof that people can survive the diagnosis and treatment of cancer and lead normal, productive lives.*

Impaired tissue integrity

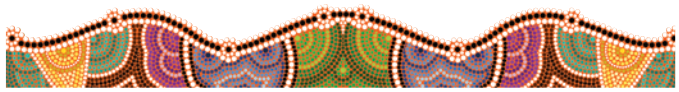
Surgery interrupts the integrity of the skin surface, providing a potential portal of invasion for bacteria. Radiation therapy causes an inflammatory response in the skin and mucous membranes within the field of radiation, creating further risk of tissue reaction and breakdown.

- Provide health education on wound and skin care, particularly if pelvic exenteration is performed. Irrigations with saline or other prescribed solutions can be performed at intervals. *Open and damaged tissue increases the risk of infection. Meticulous skin and wound care is necessary to prevent infection and further tissue destruction.*
- If appropriate, provide education on stoma care and care for the skin surrounding the stoma. (These procedures are discussed in Chapter 23.) *Urine and stool are irritating to the skin. Without proper care, the skin surrounding the stoma can become excoriated.*
- Apply non-oil-based lotions to skin to help minimise itching and maintain integrity. *Oil-based lotions are not recommended for tissue undergoing radiation.*
- Educate the woman about the importance of the markings used to localise the radiation beam to the target area and not to remove them. *Markings are used in future radiation treatments.*
- Monitor for evidence of fistula formation and teach the woman to do the same. *Fistula formation is a potential complication of radiation to the pelvic or abdominal cavities.*

Community-based care

Health education varies according to the stage of the cancer and the treatment selected. Provide information concerning radiation, chemotherapy or surgery, as indicated. Preoperative health education focuses on postoperative expectations, including management of urinary or faecal diversion, if indicated (see Chapters 23 and 26) and psychological impacts of the diagnosis and surgery. Help the woman and family recognise signs of infection and understand the importance of follow-up care. In addition, suggest the following resources:

- ABC Health & Wellbeing: www.abc.net.au/health/conditions/cancer/default.htm
- Cancer Council Australia Support: www.cancer.org.au/about-cancer/patient-support/



THE WOMAN WITH ENDOMETRIAL CANCER

Endometrial cancer is the most common invasive gynaecological cancer in Australia. The incidence rate of endometrial cancer is increasing due to the increased rate of obesity and the aging population. Presently, one in 69 women are diagnosed prior to being 75 years of age (Cancer Council Australia, 2012). When diagnosed and treated early in the disease, the 5-year survival rate is about 82.5% (Cancer Australia, 2015b).

Risk factors

A significant risk factor for endometrial cancer is prolonged oestrogen stimulation. Other factors that increase risk are obesity, anovulatory menstrual cycles, decreasing ovarian function (as from menopause), oestrogen-secreting tumours and unopposed oestrogen (e.g. oestrogen therapy without progesterone). Medical conditions that may alter oestrogen metabolism and increase the risk of endometrial cancer are diabetes mellitus, hypertension and polycystic ovarian syndrome (Porth & Matfin, 2014). Tamoxifen, a medication that blocks oestrogen receptor sites and is used to treat breast cancer, has a weak oestrogenic effect on the endometrium and is also a risk factor.

Endometrial cancer is the most commonly inherited gynaecological cancer. A family history of hereditary non-polyposis colon cancer (HNPCC) may mean that a woman has an inherited mutation that is a mismatch of repair genes and has a 60% risk of endometrial cancer.

Pathophysiology

Most endometrial malignancies are adenocarcinomas that are slow to grow and metastasise. These cancers develop in the glandular cells or endometrial lining of the uterus (the same tissue that is shed each month during a normal menstrual period). Endometrial hyperplasia (excessive growth) is a precursor of endometrial cancer. These tumours tend to grow slowly in the early stages.

Tumour growth usually begins in the fundus, invades the vascular myometrium and spreads throughout the female reproductive tract. Metastasis occurs by means of the lymphatic system, through the fallopian tubes to the peritoneal cavity and to the rest of the body via the bloodstream. Target areas for metastasis include the lungs, liver and bone. The International Federation of Gynecology and Obstetrics (FIGO) classification of endometrial cancer is presented in Table 48.3.

TABLE 48.3 FIGO staging classification for endometrial cancer

STAGE	DESCRIPTION
I	Tumour limited to endometrium or myometrium
II	Endocervical glandular involvement or invasion of cervical stroma
III	Metastasis or invasion of serosa, adnexae, vagina and pelvic or para-aortic lymph nodes
IV	Tumour invasion of bladder or bowel mucosa; distant metastases

Manifestations

The main manifestation of endometrial hyperplasia or overt endometrial cancer is abnormal, painless vaginal bleeding. In menstruating women, this bleeding is manifested as menorrhagia or metrorrhagia. In postmenopausal women, any bleeding is abnormal. Later symptoms include pelvic cramping, bleeding after intercourse and lower abdominal pressure. In advanced disease, lymph node enlargement, pleural effusion, abdominal masses and ascites may be present.

INTERPROFESSIONAL CARE

The goals of care for the woman with endometrial cancer are to eradicate the cancer and minimise complications and metastasis.

Diagnosis

Tests used to diagnose cancer of the endometrium include a vaginal or transvaginal ultrasound to determine endometrial thickening, which may indicate hypertrophy or malignant changes, or an endometrial biopsy or a dilation and curettage (D&C) to provide a definitive diagnosis (see Chapter 46 for further information and nursing care). Other tests to determine the extent of the disease include chest x-ray, intravenous urography, cystoscopy, barium enema, sigmoidoscopy, MRI and bone scans.

Medications

Although the treatment of choice for primary endometrial carcinoma is surgery, progesterone therapy may be used for recurrent disease. About one-third of women respond favourably, primarily those with well-differentiated tumours. Chemotherapy is less effective than other forms of therapy, although cisplatin or combination chemotherapy may be used for women with disseminated disease.

Surgery

After the diagnosis is confirmed, a total abdominal hysterectomy and bilateral salpingo-oophorectomy is performed for stage I cancer. A radical hysterectomy with node dissection is performed if the disease is stage II or beyond.

Radiation therapy

Treatment with external and internal radiation may be performed as a preoperative measure or as adjuvant treatment in advanced cases.

Nursing care

Health promotion

All women, including perimenopausal and postmenopausal women, need regular pelvic examinations. At the time of menopause all women should be informed of the risks and symptoms of endometrial cancer and strongly encouraged to report any unexpected bleeding or spotting to their healthcare provider. Those in high-risk groups are advised to have endometrial biopsies every 2 years, beginning at age 35. In addition, control of diseases such as diabetes mellitus and hypertension decreases the risk of endometrial hyperplasia.

Assessment

Undertake a health history and physical examination (see Chapter 46):

- *Health history:* abnormal vaginal bleeding, menstrual history, use of oestrogen (without progesterone) to treat menopausal symptoms, breast cancer treated with tamoxifen, childbearing status, presence of chronic illnesses, family history of hereditary non-polyposis colon cancer.
- *Physical assessment:* height and weight, pelvic examination, abdomen, lymph glands.
- *Mental health assessment:* anxiety, coping strategies, emotional wellbeing.

Nursing diagnoses and interventions

Nursing care involves helping the woman deal with the physical and psychological effects of a potentially life-threatening illness, make informed decisions and minimise the adverse effects of therapy. Pain relief is a key component of care, as is counselling for the woman and family. Encourage the woman to perform self-care and resume normal activities of daily living.

Acute pain

Total abdominal hysterectomy can involve severe and prolonged pain, not only from the surgical incision but also from the manipulation of internal organs during surgery. Abdominal viscera are highly vascular and are easily bruised by handling.

- Encourage the woman to report her pain. *This will allow a nursing assessment to be undertaken and effective pain relief to be administered.*
- Administer analgesics. *Analgesics provide pain relief and promote early ambulation.*
- Encourage ambulation. *Ambulation facilitates the expulsion of flatus, which can cause distension as well as discomfort.*

Disturbed body image

For many women, the side effects of cancer treatment can be almost as difficult and painful as the disease itself. Although side effects of the different therapies vary between individuals, the woman's body image and quality of life are always

affected. Such side effects as alopecia (hair loss), nausea, vomiting, fatigue, diarrhoea, stomatitis and surgical scarring disturb body image.

- Encourage the woman to verbalise her feelings. *This allows the nurse to work with the woman on her emotional wellbeing.*
- Review the side effects of the proposed treatment regimen and assist the woman to develop a plan to deal with these effects. *This promotes a sense of control.*
- Suggest to the woman and family that side effects are usually manageable and may be temporary. *Over-the-counter agents can be used to alleviate stomatitis. Frequent rest periods can relieve fatigue. Medications can be prescribed for nausea, vomiting and diarrhoea.*

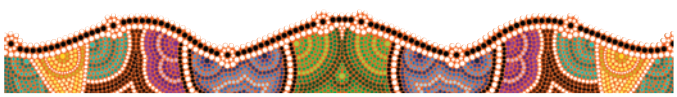
Ineffective sexuality pattern

Altered sexuality may result from a feeling of unattractiveness, fatigue or pain, and discomfort. The woman's partner may fear that sexual activity will be harmful.

- Encourage expression of feelings about the effect of cancer on their lives and sexual relationship. *Verbalising feelings helps relieve stress and maximises relaxation.*
- Suggest that the couple explore alternative sexual positions and coordinate sexual activity with rest periods and times that are relatively free from pain. *This creates a more favourable environment for satisfying sexual activity.*

Community-based care

Provide information about the specific treatment and prognosis for the cancer. Explain the expected side effects of radiation implant therapy (see Chapter 13). Pain control measures are also an essential part of the health education plan (see Chapter 8). The resources listed for the woman with cervical cancer are also appropriate for the woman with endometrial cancer.



THE WOMAN WITH OVARIAN CANCER

Ovarian cancer is the ninth most common cancer diagnosed in women in Australia. There is a one in 78 chance of being diagnosed with ovarian cancer before the age of 85. More than 1200 women are diagnosed each year (Cancer Council Australia, 2013).

Risk factors

Family history is a significant risk factor, with a 50% risk of developing the disease if two or more first- or second-degree relatives have site-specific ovarian cancer. Other types of inherited risk are breast–ovarian cancer syndrome (first- and second-degree relatives have both breast and ovarian cancer) and family cancer syndrome (Lynch syndrome II), in which male or female relatives have a history of colorectal, endometrial, ovarian, pancreatic or other types of cancer. The breast cancer susceptibility genes BRAC1 and BRAC2 are implicated in 5–10% of hereditary ovarian cancers (Cancer Council Australia, 2013).

Risk factors also include having no children or giving birth after age 30, exposure to talc powder prior to 1973 or asbestos, endometriosis, pelvic inflammatory disease and living in a Western industrialised country (Cancer Council Western Australia, 2013). Protective factors include long-term contraceptive use, having a child before the age of 25, tubal ligation, breastfeeding and hysterectomy.

Pathophysiology

There are several types of ovarian cancers: epithelial tumours, germ-cell tumours and gonadal stromal tumours. Most ovarian cancers are epithelial tumours, originating from the surface epithelium of the ovary. Ovarian cancer usually spreads by local shedding of cancer cells into the peritoneal cavity and direct invasion of the bowel and bladder. Cancer cells in peritoneal fluid can implant in the intestines, bladder and mesentery. Tumour cells also spread through the lymph and blood to such organs as the liver and across the diaphragm to involve the lungs. Both pelvic and para-aortic lymph nodes may be involved and tumour cells can block lymphatic drainage from the abdomen, resulting in ascites. Staging for ovarian cancer is based on surgical and histological evaluation (see Table 48.4).

Manifestations

In early stages, ovarian cancer generally causes no warning signs or symptoms. When symptoms do develop, they are often vague and mild, such as indigestion, urinary frequency, abdominal bloating and constipation. Abnormal vaginal bleeding may occur if the endometrium is stimulated by a hormone-secreting tumour or if the tumour erodes the vaginal wall. Pelvic pain sometimes occurs. An enlarged abdomen with ascites signals later-stage disease.

Complications

The complications of advanced ovarian cancer, with related nursing assessments and treatment, are outlined in Table 48.5.

TABLE 48.4 FIGO staging classification for ovarian cancer

STAGE	DESCRIPTION
I	Growth limited to the ovaries
II	Growth involving one or both ovaries with pelvic extension
III	Tumour involving one or both ovaries, with peritoneal implants outside the pelvis or positive retroperitoneal or inguinal nodes
IV	Growth involving one or both ovaries with distant metastasis

TABLE 48.5 Complications of advanced ovarian cancer

COMPLICATION	ASSESSMENTS	TREATMENT
Ascites (accumulation of fluid in the abdominal cavity; a form of third spacing)	• Abdominal distension • Shiny abdominal skin • Dullness on percussion of dependent areas • Dyspnoea, constipation • Abdominal pain	Paracentesis (removing fluid from the abdomen)
Intestinal obstruction	• Abdominal distension • Abdominal pain • Projectile vomiting • Constipation • Hyperactive bowel sounds	Nasogastric tube insertion, NBM
Deep venous thrombosis	• Leg oedema • Leg pain • Redness, warmth	Anticoagulants
Lymphoedema (leg)	• Oedema of leg • Decreased range of motion • Tight, shiny skin on leg	Skin care, range-of-motion (ROM) exercises, massage or physical therapy, compression bandaging

INTERPROFESSIONAL CARE

As with other malignancies, care of the woman with ovarian cancer is focused on surgery to determine the stage of the tumour and to remove as much of the tumour as possible. Unfortunately, because there are no early symptoms, the disease is often well advanced prior to diagnosis. In younger women an ovarian mass may be monitored for several menstrual cycles, but any ovarian mass must immediately be investigated in a postmenopausal woman.

Diagnosis

Tests used in the diagnosis of ovarian cancer may include transvaginal or abdominal ultrasound and a CT scan of the abdomen and pelvis. (See Chapter 46 for further information about diagnostic tests.)

The blood test most useful is a CA-125 antigen level. CA-125 is a tumour marker that is highly specific to epithelial ovarian cancer. Transvaginal or transabdominal ultrasonography is used to measure ovarian size and detect small masses. These tests, however, are not appropriate screening measures because they cannot differentiate between cystic or benign ovarian masses and malignancy.

Medications

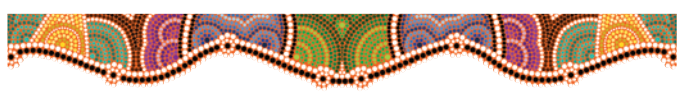
While surgery is the treatment of choice for ovarian cancer, chemotherapy may be used to achieve remission of the disease. Chemotherapy is not curative for ovarian cancer. Combination chemotherapy regimens using cyclophosphamide and cisplatin or other agents may be employed. Combination therapy with a platinum compound (cisplatin or carboplatin) is superior to treatment with a single medication, with paclitaxel/carboplatin the preferred regimen (Martin, 2005). Chemotherapy with paclitaxel (Taxol) may prolong survival. Close monitoring of bone marrow and renal function is vital while the woman is on chemotherapy because these medications have significant toxic effects.

Surgery

In young women with stage I disease who wish to have children, treatment may be limited to removal of one ovary. Usually, however, total hysterectomy with bilateral salpingo-oophorectomy (removal of the ovaries and fallopian tubes) and removal of the omentum are performed.

Radiation therapy

Radiation therapy using external-beam or intracavitary implants is performed for palliative purposes only and is directed at shrinking the tumour at selected sites.



Nursing care

Nursing care for the woman with ovarian cancer is similar to the nursing care for women with other gynaecological cancers. The side effects of treatment of cancer and generally poor prognosis diminish the woman's quality of life and involve major psychosocial implications (see Chapter 13).

Community-based care

Address the following topics in preparing the woman and her family for self-care:

- If a positive family history of the disease or previous breast cancer exists, stress the importance of obtaining regular pelvic examinations. Inform women in this risk group that annual screening with transvaginal ultrasound and CA-125 measurements may be recommended.

- Long-term use of oral contraceptives may reduce the risk of developing ovarian cancer.
- It is crucial not to ignore symptoms such as indigestion, nausea or urinary frequency, because these seemingly unrelated symptoms may be early signs of ovarian tumours. Emphasise, however, that ovarian cancer usually is asymptomatic in early stages.
- Discuss treatment options and their side effects and provide information on ways to minimise or manage side effects.
- Refer to hospice services when appropriate. The resources suggested for the woman with cervical cancer are also appropriate for the woman with ovarian cancer.

THE WOMAN WITH CANCER OF THE VULVA

Cancer of the vulva is rare, occurring in two in 100 000 women in Australia. It usually occurs in postmenopausal women. However, there has been an increase of vulvar cancer in women under 50 years of age. It has been identified that for Indigenous women living in remote rural areas it is 50 times more common than it is for the non-Indigenous population (Condon, 2010). The prognosis of vulvar carcinoma depends on the degree of invasion, general health status of the woman, presence of chronic diseases and ability to withstand treatment.

Pathophysiology

The cause of vulvar cancer is unknown, but there is evidence to associate it with STIs, particularly HPV. Nearly 85% of malignant and premalignant cervical and vulvar lesions have been found to contain HPV DNA, HPV structural antigens or both. Herpes simplex type 2 (HSV2) infection has also been associated with vulvar cancer. Other risk factors include advanced age, diabetes and a history of leucoplakia (a precancerous lesion on the vulvar mucous membranes characterised by raised white patches).

Most vulvar cancers are epidermoid or squamous cell carcinomas. The primary site is usually the labia majora, but vulvar cancer is also found on the labia minora, clitoris, vestibule and occasionally in multiple locations. Metastasis occurs by direct extension into the vagina, perineal skin, anus and urethra. The cancer also spreads through the lymphatic system via the superficial and deep inguinal and femoral nodes and to the pelvic lymph nodes.

Manifestations

The woman with vulvar cancer is often asymptomatic and lesions are discovered on routine examination or self-examination. Discolouration can vary from white macular patches to red painless sores. Lesions may be *exophytic* (proliferating outwardly), *endophytic* (proliferating inwardly), *ulcerative* or *verrucous* (resembling a wart).

Pruritus (itching) is the most common manifestation and the woman often has had a history of prolonged vulvar irritation. Perineal pain and bleeding indicate large tumours and advanced disease. In very advanced disease, dysuria related to urethral involvement may be the presenting symptom.

INTERPROFESSIONAL CARE

The report of itching, burning or a sore on the vulva merits careful investigation and biopsy of any lesions found. Inguinal lymph nodes may be enlarged. The goal of care is to eradicate the lesion and reduce the risk of recurrence. Surgical resection is the preferred treatment. If lymph nodes are involved, radiation therapy is used postoperatively. Chemotherapy is reserved for distant metastases.

Diagnosis is based on the results of an excisional biopsy of the lesion. Metastasis, if suspected, can be evaluated by chest x-ray examination, barium enema, intravenous pyelogram, cystoscopy, CT and MRI scans, and proctoscopy. Lymphangiography can also be used.

Surgery is the most common treatment for vulvar cancer. The specific procedure depends on the stage of the cancer. Early, non-invasive lesions may be treated with laser surgery, cryosurgery or electrocautery. For more advanced disease, vulvectomy may be performed (see Figure 48.6). A simple vulvectomy involves the removal of the vulva, labia majora and minora, clitoris and prepuce. A radical vulvectomy is performed if invasion is suspected. This procedure involves removal of all the tissue in a simple vulvectomy, as well as the subcutaneous tissue and regional lymph nodes.

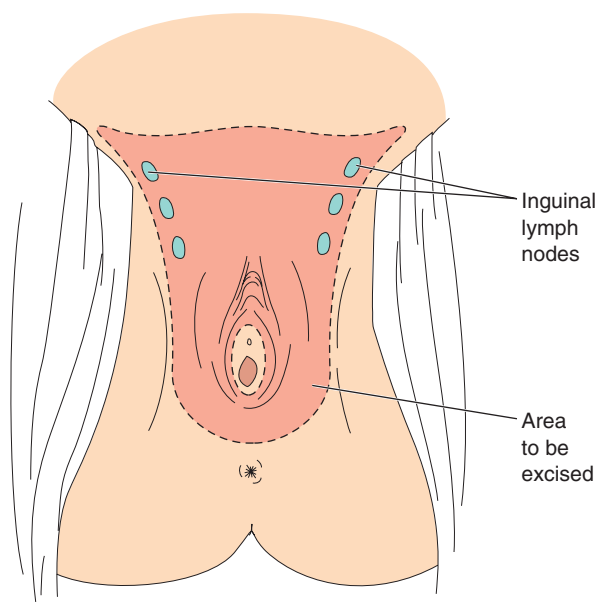


FIGURE 48.6 ■ Vulvectomy for vulvar carcinoma. A radical vulvectomy involves removal of the vulva, labia majora, labia minora, clitoris, prepuce, subcutaneous tissue and regional lymph nodes

Nursing care

Nursing care is similar to that for the woman with endometrial cancer. The woman fears death as the ultimate outcome, as well as the possible pain and suffering that surgery and other treatments may cause. Radical surgery represents a great emotional loss to women of all ages.

Nursing diagnoses and interventions

- Women who have been diagnosed with cancer of the vulva and are undergoing treatment will need to be supported. A mental health assessment for anxiety, coping strategies and emotional wellbeing will need to be undertaken. The disruption of perineal tissues is a priority issue for these women. Pain, discomfort and altered body image would also impact on the woman's wellbeing.

Impaired tissue integrity

The woman who has undergone a vulvectomy is at high risk of infection and impaired healing because of the proximity of the surgical site to urinary and anal orifices. In addition, the women are often older and may have age-related changes in healing and immune function.

- Educate the woman and/or her partner or other family member on the procedure for irrigation of the vulvectomy. *If neither is able to perform this procedure, arrange for home health nursing. Irrigation helps prevent skin breakdown and infection.*
- After irrigation, dry the area; a hairdryer on low heat may be used (Cancer Council NSW, 2014). *Dry heat helps promote healing and comfort.*
- Provide information on maintaining a diet high in protein, iron and vitamin C. *These nutrients promote collagen formation and wound healing.*

Community-based care

Health education for the woman undergoing a vulvectomy should emphasise the potential for skin breakdown, particularly with radiation therapy. Explain that removal of lymph nodes leads to lymphoedema and that recurrent cellulitis and sexual dysfunction are common complications of vulvar cancer.

THE PERIMENOPAUSAL WOMAN

MENOPAUSE

Menopause is the permanent cessation of menses. The *climacteric*, or *perimenopausal*, period denotes the time during which reproductive function gradually ceases. For most women, the perimenopausal period lasts several years. It begins with a decline in the production of the hormone oestrogen, includes the permanent cessation of menstruation due to loss of ovarian function, and extends for 1 year after the final menstrual period, at which time a woman is said to be *postmenopausal*. On average, women live one-third of their lives after menopause.

Menopause is a normal physiological process. It is not a disease or a disorder. It is included here because it does increase the risk of physical disorders, as well as affecting various aspects of women's health. Many women welcome the freedom from monthly menstrual periods and have relatively minor physical effects from the reduction in oestrogen. However, the hormonal changes that occur can be accompanied by side effects. There is wide variation in how individual women experience these side effects. In Australia, most women cease menstruating at between 48 and 55 years of age, with the average being about 50 or 51 years. Early menopause is when a woman stops menstruating before 45 years of age. In 50% of cases of early menopause there are no known causes. However, early menopause is associated with surgical removal of the ovaries, chemotherapy and radiotherapy (Jean Hailes Foundation, 2014). After menopause certain health risks increase, including heart disease, osteoporosis, macular degeneration, cognitive changes and breast cancer.

The physiology of menopause

The menopausal period marks the natural biological end of reproductive ability. *Surgical menopause* occurs when the ovaries are removed in premenopausal women, dramatically reducing the production of oestrogen and progestins. *Chemical menopause* often occurs during cancer chemotherapy, when cytotoxic medications arrest ovarian function.

As ovarian function decreases, the production of oestradiol (E2) decreases, and is ultimately replaced by oestrone as the main ovarian oestrogen. Oestrone is produced in small amounts and has only about one-tenth the biological activity of oestradiol. With decreased ovarian function, the second ovarian hormone, progesterone, is also markedly reduced.

Manifestations

As oestrogen decreases, various tissues are affected, and breast tissue, body hair, skin elasticity and subcutaneous fat decrease. The ovaries and uterus become smaller and the cervix and vagina also decrease in size and become pale in colour. These changes may result in problems with vaginal dryness, dyspareunia, urinary stress incontinence, urinary tract infections (UTIs) and vaginitis. Hot flushes, palpitations, dizziness and headaches are often caused by vasomotor instability. Other problems resulting from vasomotor instability include insomnia, frequent awakening and perspiration (night sweats). The woman may experience irritability, anxiety and depression as a result of these events.

MANIFESTATIONS The perimenopausal period

- Menstrual cycles become unpredictable. Menstrual flow varies widely in amount and duration and eventually ceases.
- Vaginal, vulval and urethral tissues begin to atrophy.
- Vaginal pH rises, predisposing the woman to bacterial infections.
- Vaginal lubrication decreases and vaginal rugae decrease in number. This may result in dyspareunia, injury and fungal infections.
- Vasomotor instability due to a decrease in oestrogen may result in hot flushes and night sweats. A hot flush starts in the chest and moves upwards towards the face, and may last from seconds to several minutes.
- Psychological symptoms may include moodiness, nervousness, insomnia, headaches, irritability, anxiety, inability to concentrate and depression.

Long-term oestrogen deprivation results in an imbalance in bone remodelling and osteoporosis, leading to fractures and kyphosis. The risk of cardiovascular diseases increases in response to an increase in atherosclerosis (from an increase in the LDL-to-HDL cholesterol ratio). Symptoms of the perimenopausal period are listed in the following box. These symptoms vary widely. Some women experience few or no symptoms, others experience moderate symptoms and some women experience severe symptoms.

INTERPROFESSIONAL CARE

Care of the woman experiencing menopausal symptoms focuses on relieving symptoms and minimising postmenopausal health risks.

Diagnosis

As oestrogen secretion diminishes, levels of FSH and LH rise and remain elevated. A woman who has not menstruated for 1 full year or who has an increased FSH blood level is considered menopausal.

Medications

Hormone replacement therapy may be prescribed to alleviate severe symptoms of menopause, but only for a limited amount of time and only after a woman has been provided with information about known risks. HRT may include oestrogen alone for women who have had a hysterectomy, or a combination of oestrogen and progestin for women who still have their uterus. The addition of progestin stimulates monthly shedding of the interuterine lining, decreasing the risk of uterine cancer. HRT relieves hot flushes and night sweats, and decreases problems of vaginal dryness and urogenital tissue atrophy, which can lead to painful intercourse and urinary incontinence. Long-term HRT may increase the risk of breast cancer, ovarian cancer, stroke and venous thrombosis (Mayo Foundation for Medical Education and Research, 2015). However, women who have had a hysterectomy and take oestrogen alone do not have an increased risk of breast cancer (Lancet Oncology, 2012).

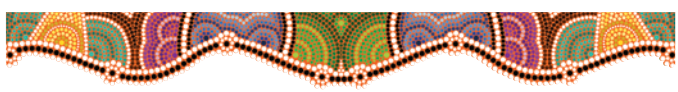
Selective oestrogen receptor modulators (SERMs), such as raloxifene (Evista) and tamoxifen, bind to oestrogen receptors and exert site-specific effects in different target tissues. Tamoxifen and toremifene (a derivative of tamoxifen) have a beneficial effect on bone mineral density, and serum lipids and decrease the risk of invasive breast cancer in women at high risk. They also provide an alternative to HRT for preventing osteoporosis.

Alternative and complementary therapies

Non-traditional or alternative therapies have become popular as a result of the controversy surrounding the use of HRT. The following complementary therapies are examples of those used by menopausal women to reduce associated discomfort:

- acupuncture
- biofeedback
- massage
- herbs such as *Cimcifuga racemosa* (black cohosh), *Vitex agnus castii* (chasteberry), *Rehmannia*, ginseng, the Chinese tonics he shou wu and dong quai, golden seal, flaxseed and evening primrose
- supplements such as vitamin E and soy protein (soy is high in plant oestrogens)
- meditation and yoga.

However, there is little reliable research available on the effect of soy, primrose oil, black cohosh or other herbs on decreasing hot flushes or sleep disturbances. However, exercise such as yoga or walking will assist women to maintain a healthy lifestyle and may combat some of the side effects of menopause (Women's Health Queensland Wide Inc., 2013).



Nursing care

Nursing care focuses on educating the woman about implementing lifestyle changes that are important to her health and wellbeing. These will assist women to minimise the symptoms associated with hormonal changes, reducing the risk of cardiovascular disease, cancer and osteoporosis.

Health promotion

National screening programs are available in Australia for various cancers, such as breast, bowel and cervical (Department of Health, 2015). It is a good idea, especially as people age, to have regular health assessments. A health assessment includes examination for cancers of the thyroid, ovaries, lymph nodes, oral cavity and skin. Other important health assessments include screening for cervical, breast and colorectal cancer. Health counselling should also include information about alcohol and tobacco use, sun exposure, diet and nutrition, exercise, risk factors, sexual practices, mental and emotional wellbeing, and environmental and occupational exposures. It is important to discuss the benefits of rest and exercise, as well as a diet that includes fruit, vegetables and

fibre. In addition, suggest the following resources for further information:

- Women's Health Australia: www.alswh.org.au
- Australasian Menopause Society: www.menopause.org.au
- Jean Hailes Foundation for Women's Health: https://jeanhailes.org.au/health-a-z/menopause
- Women's Health Queensland Wide Inc. (women's health):
  - *About menopause fact sheet:* www.womhealth.org.au/conditions-and-treatments/about-menopause-fact-sheet
  - *Alternatives to HRT fact sheet:* www.womhealth.org.au/conditions-and-treatments/alternatives-hrt-fact-sheet
  - Women's Health Queensland Wide Inc. (middle years): www.womhealth.org.au/resources/book-library/30-middle-years

Assessment

Undertake a thorough health history and physical examination. When assessing the older woman, be aware of normal changes with ageing, as outlined in Chapter 46.

- *Health history:* problems with urinary frequency, urgency or incontinence; menstrual history; sexual history; dyspareunia; use of alcohol, nicotine and illicit drugs; medications, sleep patterns, hot flushes, night sweats, changes in emotional responses.
- *Physical assessment:* height and weight; posture; vital signs; abdominal assessment; with verbal consent, breast examination and pelvic examination.

Nursing diagnoses and interventions

Although each nursing care plan must be individualised, interventions often focus on physical problems. There is often a lack of information on sexuality, self-esteem, emotional well-being and body image.

Deficient knowledge

Menopausal symptoms vary widely. However, the well-informed woman is better prepared to deal with whatever symptoms she experiences.

Discuss physiological symptoms, such as hot flushes and night sweats. The underlying cause of hot flushes is not known. As previously discussed, many physiological effects of menopause are responsive to non-pharmacological methods of relief, such as lifestyle changes.

CONSIDERATION FOR PRACTICE

When hot flushes occur at night and are accompanied by perspiration, they are called night sweats. Night sweats often interfere with normal sleep patterns, leading to increased tiredness and irritability.

- Provide information to the woman about dietary recommendations. The recommended daily intake of calcium for women over 50 is 1200 mg. *Some women need to use calcium supplements or calcium-containing antacid tablets to meet this requirement.*
- Emphasise the importance of weight-bearing exercise. *Weight-bearing exercise reduces the rate of bone loss, helps maintain optimum weight and reduces cardiovascular risk.*
- Provide information about the benefits and risks of HRT. Not every woman will need or want it. *Every woman needs to understand both the risks and the benefits before deciding whether to use HRT.* To assist health professionals and individuals to whom they provide care to make informed choices regarding HRT, there are various web-based resources available.
- Encourage the woman to perform monthly breast self-examination (BSE) on the same day each month, and to have a mammogram and a cervical screening every 2 years (Department of Health, 2015). In May 2017, a new cervical screening program will be introduced in Australia. A primary human papillomavirus (HPV) test will replace the Pap smear test for cervical screening. It is recommended that a HPV test be performed every 5 years on women between the ages of 25 and 74 years of age (Department of Health, 2015).

Ineffective sexuality pattern

Vaginal dryness and atrophy, together with the emotional effects of menopause, can interfere with sexual expression and satisfaction. Suggesting measures to help the woman and her partner cope with these changes can enable them to continue or resume a mutually satisfying sexual relationship.

- Encourage expression of feelings and concerns about how menopause is changing the woman's sex life. *The woman may not be comfortable in discussing their intimate sexual behaviour.*
- Suggest ways to increase vaginal lubrication, such as spending more time in foreplay and/or using water-soluble gels (e.g. K-Y jelly) for vaginal lubrication. A more leisurely approach to sexual activity can be mutually gratifying for both the woman and her partner. *Use of water-soluble gels can prevent vaginal pain and irritation, and improve the quality of the sexual experience.*
- Explain that as women age, it may take longer for vaginal lubrication and orgasm to occur. *This information is important to prevent the woman from believing something is wrong with her, or her partner believing she is no longer interested or sexually exciting.*
- Inform about changes in the sexual responsiveness of male partners. Difficulty in achieving an erection or maintaining an erection occurs in one in five Australian men over the age of 40 (Andrology Australia, 2015).

Situational low self-esteem

Each woman responds to the ageing process in her own way. Most women have coping skills that adequately equip them to deal with the gradual changes associated with ageing. Factors that may provoke a lowered self-esteem are the loss of youth, a sense of emptiness as children leave home and the need to redefine one's self-concept and roles as parenting becomes less important. Women who place a high value on their physical attractiveness may experience a painful psychological response to the physical changes of menopause.

- Encourage expression of fears and concerns related to changes in interpersonal and family functions. *Many women associate ageing with 'uselessness' and unattractiveness.*
- Suggest volunteer activities or employment for the woman who has extra time. *This enables the woman to feel that she is still a contributing member of society.*
- Discuss the importance of a healthy lifestyle in maintaining physical attractiveness. Identify risk factors and high-risk behaviours. *Lifestyle habits and behaviours affect many body systems and physical appearance. For example, cigarette smoking and overexposure to the sun make the skin age faster, contributing to wrinkles.*

Disturbed body image

As a woman progresses through the perimenopausal period, changes in her appearance and the loss of childbearing ability may combine to make her feel vulnerable to community stereotyping of the 'older woman' as less attractive and unproductive. Although this is far from the truth, with women living at least one-third of their lives after menopause in productive careers and activities, it nevertheless is the perception of women as well as society. The physical changes the woman often experiences include growth of facial hair, excessive perspiration and flushing of the face, and weight gain.

- Encourage the woman to describe her perceptions of her own body. *This information is necessary to obtain data to establish an individualised plan of care.*
- Encourage verbalisation of feelings of concern, anger, anxiety, loss and fear over body changes. *Expressing these emotions can facilitate the grieving process and acceptance of change.*
- Stress that certain physical characteristics of a person cannot be changed; emphasise the importance of learning to recognise and appreciate one's own special strengths. *These help the woman gain acceptance and have a realistic appraisal of self.*
- Refer, as appropriate, for dietary management, exercise, sleep studies, stress management and cosmetic assistance (e.g. for aggravating facial hair). *These actions increase wellness and a positive sense of self.*

DISORDERS OF THE BREAST

Breast disorders are common conditions that primarily affect women. (Disorders of the male breast are discussed in Chapter 47.) When a woman discovers a breast lump, her first response is often fear: of breast cancer, of losing her breast and perhaps of losing her life. As many societies view the breast as a significant component of feminine sexuality, any problem that threatens the breast often strikes at the core of a woman's self-image.

Nurses play a critical role in the care of women experiencing breast disorders by providing education, support and advocacy. Part of the nurse's role is educating women about normal breast tissue, common benign breast disorders, available screening techniques and risk factors for breast cancer, and breast self-examination (BSE).

THE WOMAN WITH A BENIGN BREAST DISORDER

Benign breast disorders occur frequently in women and may be a source of anxiety. Changes in a woman's breast tissue often correspond to hormonal changes of the menstrual cycle. Most women notice increased tenderness and lumpiness prior to menses. (For this reason, it is best to perform BSE 7 to 10 days after the beginning of the menstrual period.) Breast tissue changes in response to hormonal, nutritional, physical and environmental stimuli. Benign breast disorders include fibrocystic breast changes, fibroadenomas, intraductal papillomas, duct ectasia, fat necrosis and mastitis (see Table 48.6).

Pathophysiology and manifestations

Fibrocystic changes

Fibrocystic changes (FCC) (*fibrocystic breast disease*) are the physiological nodularity and breast tenderness that increase and decrease with the menstrual cycle. An estimated 60% of all women experience some of these changes, which include fibrosis, epithelial proliferation and cyst formation. FCC is most common in women 30 to 50 years of age (Centre of Health, 2015) and is rare in postmenopausal women who are not taking HRT.

FCC includes many different lesions and breast changes. The more common non-proliferative form does not increase the risk of breast cancer. The proliferative form, accompanied by giant cysts and proliferative epithelial lesions, does increase the risk of breast cancer.

Non-proliferative changes may be cystic or fibrous. Cystic change refers to the dilation of ducts in the subareolar, lobular or lobe areas. Cysts often go unnoticed unless pain and tenderness is associated with menses. Fibrous changes are infrequent but can occur during the menstrual years. A firm, palpable mass, 2–3 cm in size, is typically located in the upper outer breast quadrant following an inflammatory response to ductal irritation.

Women with fibrocystic changes experience bilateral or unilateral pain or tenderness in the upper, outer quadrants of their breasts and report that their breasts feel particularly thick and lumpy the week prior to menses. Nipple discharge may be present. Pain is due to oedema of the connective tissue of the

TABLE 48.6 Summary of common breast disorders

CONDITION	AGE	PAIN	NIPPLE DISCHARGE	LOCATION	CONSISTENCY AND MOBILITY	DIAGNOSIS AND TREATMENT
Duct ectasia	35 to 55 years; median age 40	Burning around nipple	Sticky; multicoloured; usually bilateral	No specific location	Retroareolar mass with advanced disease	Open biopsy; local excision of diseased portion of breast
Fibroadenoma	15 to 39 years; median age 20	No	No	No specific location	Mobile, firm, smooth, well-delineated mass	Mammography; surgical or needle biopsy; excision of the tumour
Fibrocystic changes (FCC)	20 to 49 years; median age 30 (may subside with menopause)	Yes	May occur	Upper outer quadrant	Bilateral multiple lumps influenced by the menstrual cycle	Needle aspiration; observation; biopsy if there is an unresolved mass or mammographic changes
Intraductal papilloma	35 to 55 years; median age 40	Yes	Serous or sanguineous; usually unilateral from one duct	No specific location	Usually soft, poorly delineated mass	Pap smear of nipple discharge; biopsy; wedge resection
Mastitis, acute	Childbearing years	Tenderness; pain	No	No specific location	Generalised redness of overlying skin	Antibiotic therapy; incision and drainage if mastitis progresses to an abscess
Mastitis, chronic	Any age	Tenderness, pain; headache; high fever	No	No specific location	Generalised redness and swelling	Antibiotics, usually penicillin
Fat necrosis	Any age	Tenderness	No	No specific location	Firm; irregular; palpable	Surgical biopsy to rule out cancer

breast, dilation of the ducts and some inflammatory response; some women report an increase in breast size. Multiple, mobile cysts may form, usually in both breasts (see Figure 48.7). Fluid aspirated from these cysts ranges in colour from milky white to yellow, brown or green. If the fluid is tinged with blood, there is reason to suspect malignancy.

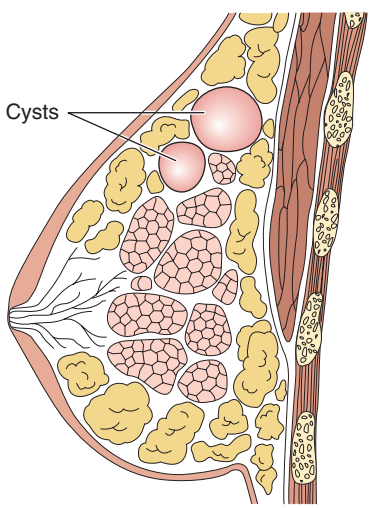


FIGURE 48.7 ■ Fibrocystic breast changes

Intraductal disorders

An *intraductal papilloma* is a tiny, wart-like growth on the inside of the peripheral mammary duct that causes discharge from the nipple. The discharge may be clear and sticky or bloody. When more than one of these growths is present, the condition is called *intraductal papillomatosis*. This condition is most common in women in their 40s (Breast Cancer Care, 2015). The lesion must be investigated to rule out malignancy.

Mammary duct ectasia (*plasma cell mastitis*) is a palpable lumpiness found beneath the areola. Duct ectasia involves periductal inflammation, dilation of the ductal system and accumulation of fluid and dead cells that block the involved ducts. The condition usually occurs in perimenopausal women and is difficult to differentiate from cancer.

Symptoms of mammary duct ectasia include sticky, thick nipple discharge with burning and itching around the nipple and inflammation. The discharge may be green, greenish brown or bloody. Nipple retraction often is associated with duct ectasia in postmenopausal women.

INTERPROFESSIONAL CARE

Diagnosis of FCC is based on complete history, physical examination and imaging studies. A biopsy may be required for diagnosis.

Analysis of nipple discharge, mammography and possibly ductography may be used to diagnose ductal disorders. The affected duct is excised in an open biopsy procedure. Nursing care for the woman is similar to that for any person with an open biopsy. It also is important to reassure the woman that these disorders are not breast cancer.

The treatment is usually symptomatic. Cyst aspiration may relieve pain and also allows examination of fluid to confirm the cystic nature of the disease. A well-fitting bra, often one without underwire, that provides good support worn day and night helps relieve discomfort. Some women report that eliminating xanthines (found in coffee, tea, cola and chocolate) from the diet decreases symptoms. Aspirin, mild analgesics, local heat or cold, and vitamin E may help relieve breast pain. Hormone therapy is controversial because of the benign nature of the disease and potential adverse effects of therapy. Danazol, a synthetic androgen, may be prescribed for women with severe pain.

Nursing care

When a woman presents with a breast mass, nursing responsibilities include taking a careful physical and mental health history and facilitating follow-up care. If a palpable mass is present, it is important to ask how long the lesion has been present and whether the woman has noticed any pain associated with the mass, any change in its size and any changes in association with the menstrual cycle.

In many cases, definitive diagnosis of the breast disorder requires surgical biopsy to rule out cancer. During the diagnostic process, the nurse should provide emotional support and health education about diagnostic and therapeutic procedures, self-care and comfort measures, and resources to help the woman discuss her feelings and cope with the experience.

THE WOMAN WITH BREAST CANCER

Breast cancer is the unregulated growth of abnormal cells in breast tissue. Breast cancer is the third most common invasive cancer in Australia. The Australian Government estimated that more than 15 740 new cases would be diagnosed in 2015, consisting of 145 males and 15 600 women. One in eight women and one in 719 men under the age of 85 are at risk of being diagnosed with breast cancer (Cancer Australia, 2015c). There are racial differences in the incidence and mortality of breast cancer (see the 'Focus on cultural diversity' box).

FOCUS ON CULTURAL DIVERSITY Incidence and mortality for breast cancer in women

- Breast cancer is more prevalent in non-Indigenous Australian women than Indigenous Australian women.
- Breast cancer is more prevalent in Caucasian women over the age of 40 years.
- Indigenous Australian women are more likely to die from breast cancer because they are often diagnosed at an advanced stage as they are less likely to participate in breast screening (Cancer Australia, 2015d).

Risk factors

Of the various kinds of risk factors for breast cancer, some can be changed and some cannot. Those that cannot be changed are:

- *Age and gender.* Women are 100 times more likely to have breast cancer than men, with the risk increasing with age. See the 'Nursing care of the older woman' box below.
- *Genetic risk factors* (discussed below).
- *Family history of breast cancer*. Relatives from either the maternal or the paternal side of the family. The risk is greater if two or more relatives have been diagnosed with breast cancer. Although women who have a first-degree relative who has experienced breast cancer are at an increased risk, most do not develop breast cancer. If these women do develop breast cancer, they are likely to be aged over 50. Having a male family member with breast cancer also poses an increased risk.
- *Personal history of breast cancer.* A woman with cancer in one breast has a three- to five-fold increase in risk of developing a new cancer in the other breast or in a different part of the same breast.
- *Previous breast biopsy.* If earlier breast biopsies were diagnosed as proliferative, then breast disease without atypical hyperplasia increases risk by 1.5 to 2 times. A previous biopsy of atypical hyperplasia increases risk by 4 to 5 times.
- *Previous chest irradiation.* Radiation of the chest as a child or young woman for other cancer (such as Hodgkin's disease) significantly increases the risk.
- *Menstrual history.* Women who begin menstruating before the age of 12 or who have menopause after the age of 50 are at a slightly higher risk.

Lifestyle-related factors and breast cancer risk include using oral contraceptives, not having children or having them after the age of 30, using HRT for more than 5 years, not breastfeeding, drinking alcohol (especially two to five drinks daily), obesity, high-fat diets, physical inactivity and (possibly) environmental pollution. Lower risk factors for breast cancer include breastfeeding, moderate or vigorous physical activity, and maintaining a healthy body weight.

Pathophysiology

Possible causes of breast cancer include environmental, hormonal, reproductive and hereditary factors. Two breast cancer susceptibility genes have been identified: BRCA1 on chromosome 17 and BRCA2 on chromosome 13. These genes may be responsible for the approximately 10% of women with hereditary breast cancer,

NURSING CARE OF THE OLDER WOMAN with breast cancer

- Although the incidence of breast cancer is increasing in premenopausal women, it is still primarily a disease of older women. However, the needs of older women with breast cancer have been inadequately addressed in the professional literature and in the popular media.
- Women between the ages of 50 and 65 are the group most likely to benefit from screening mammography, yet many women in this age group have never had a mammogram. Failure of health professionals to refer older women for mammography is the reason most frequently cited for this statistic; nurse practitioners and female medical officers are more likely to refer women for mammography.
- For too long, mastectomy was perceived as the only treatment option open to most older women with breast cancer, even those with early-stage disease. Slowly that perception is changing as breast-conservation treatment gains greater acceptance. The choice of surgical treatment, particularly for older women, is highly individual. Many older women wish to preserve their breasts.
- Although older women with breast cancer may experience coexisting chronic illnesses and impaired physical function, research suggests that they show lower levels of emotional distress than younger women. Obviously the need for services such as personal care, shopping, housekeeping and transportation increases as the ages of the woman and the caregiver increase.

with genetic mutations causing up to 80% of breast cancer in women younger than 50 years of age. A woman with identified mutations in BRCA1 (known to be involved in tumour suppression) has a lifetime risk of 56–85% for breast cancer and also has an increased risk of ovarian cancer. Mutations of a tumour suppressor gene are also linked to increased risk of breast cancer.

Cancer of the breast begins as a single transformed cell and is hormone dependent. Cancers of the breast are classified as non-invasive (in situ) or invasive, depending on the penetration of the tumour into surrounding tissue. Breast cancer may remain a non-invasive disease or an invasive disease without metastasis for long periods of time.

Breast cancer may be categorised as carcinoma of the mammary ducts, carcinoma of mammary lobules or sarcoma of the breast. Most breast cancers are adenocarcinomas and appear to arise in the terminal section of the breast ductal tissue. There are many histological types of breast cancer and only examples are described here. The most common type is *infiltrating ductal carcinoma*. Two atypical types of breast cancer are inflammatory carcinoma and Paget's disease. Inflammatory carcinoma of the breast, a systemic disease, is the most malignant form of breast cancer. Oedema with dimpling of the skin that results in the skin looking like the peel of an orange (*peau d'orange*) is usually present. *Paget's disease* is a rare type of breast cancer involving infiltration of the nipple epithelium.

Breast cancer can metastasise to other sites through the bloodstream or lymphatic system. The common sites of metastasis of breast cancer are bone, brain, lung, liver, skin and lymph nodes. Staging is a system of classifying cancer according to the size of the tumour, involvement of lymph nodes and metastasis to distant sites, and the presence/absence of distant metastasis (see Table 48.7). The staging of

TABLE 48.7 Staging of breast cancer

STAGE	TUMOUR	NODE INVOLVEMENT	METASTASIS
0	Tis—Carcinoma in situ or Paget's disease of the nipple	N0—No regional lymph node metastasis	M0—No evidence of distant metastasis
I	T1—Tumour no larger than 2 cm	N0	M0
IIA	T0—No evidence of primary tumour T1 T2—Tumour no larger than 5 cm	N1—Metastasis to movable ipsilateral axillary nodes N0	M0 M0
IIB	T2 T3—Tumour larger than 5 cm	N1 N0	M0 M0
IIIA	T0 T1 T2 T3	N2—Metastasis to ipsilateral fixed axillary nodes N1 N2	M0 M0 M0
IIIB	T4—Tumour of any size with direct extension to chest wall or skin	Any N	M0
	Any T	N3—Metastasis to ipsilateral internal mammary lymph nodes	M0
IV	Any T	N0 and N1	M1—Distant metastasis

the breast cancer provides important information for making decisions about treatment options and is also used as a basis for prognosis.

Manifestations

The symptoms of breast cancer may include a non-tender lump in the breast (most often in the upper outer quadrant, the area with the most glandular tissue), abnormal nipple discharge, a rash around the nipple area, nipple retraction, dimpling of the skin or a change in the position of the nipple (see the box below). There may also be nipple pain, scaliness, ulceration, skin irritation or discharge. Breast cancer is usually painless, but some women report a burning or stinging sensation. Many women with breast cancer have no symptoms and their tumours are detected by mammography. However, most breast cancers are found by the women themselves (during BSE or a shower) or by their partners during sexual activity.

MANIFESTATIONS Breast cancer

- Breast mass or thickening
- Unusual lump in the underarm or above the collarbone
- Persistent skin rash near the nipple area
- Flaking or eruption near the nipple
- Dimpling, pulling or retraction in an area of the breast
- Nipple discharge
- Change in nipple position
- Burning, stinging or pricking sensation

INTERPROFESSIONAL CARE

Diagnosis of breast cancer begins with detection, either detection of asymptomatic lesions discovered through screening or of symptomatic lesions discovered by the woman. Any palpable mass requires evaluation. Once the diagnosis is made, a number of treatment options are available. The choice of treatment depends on several factors, such as the stage of the cancer, the age of the woman, geographical location of the woman and related health services and the woman's preferences.

Diagnosis

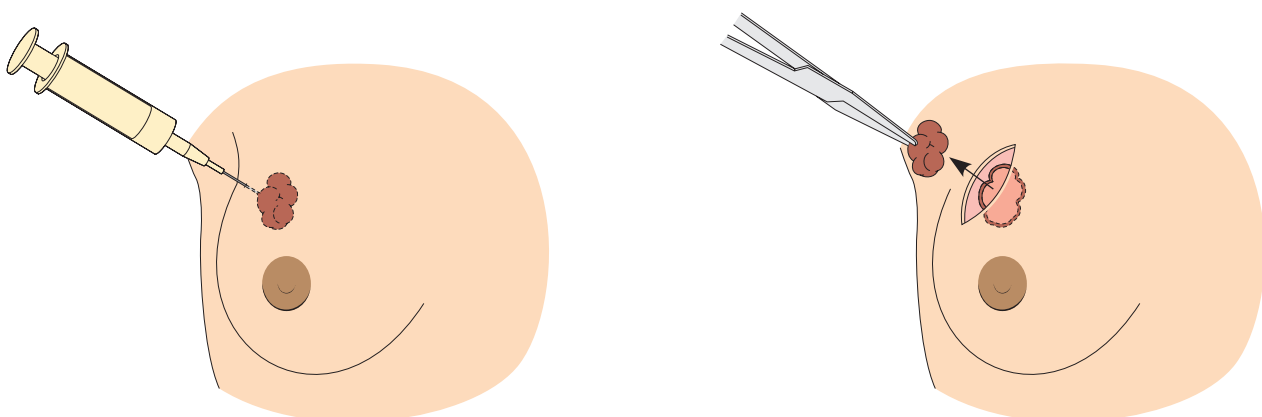
Early detection of breast cancer is possible with clinical breast examination (CBE) and mammogram (see Chapter 46 for further information). Mammography can detect breast tumours 2 years before they reach palpable size; most of these tumours have been present for 8 to 10 years. Cancer Council Australia recommends mammograms every two years for women aged between 50 and 69 years of age, although women from 40 years of age onwards can have free mammograms (Cancer Council Australia, 2015b). Magnetic resonance imaging (MRI) is used for young women who are at risk of breast cancer (Cancer Council Australia, 2015b).

Other diagnostic tests include a percutaneous needle biopsy to define a cystic mass or fibrocystic changes and provide specimens for cytological examination and a breast biopsy. In aspiration biopsy or fine-needle aspiration biopsy, a needle is used to remove cells or fluid from the breast lesion (see Figures 48.8A and 48.8B). The types of breast biopsy and related nursing care are described in Chapter 46. In many facilities, fine-needle aspiration biopsies are performed using a stereotactic biopsy device; mammography and a computer are used to guide the needle.

Medications

Tamoxifen citrate (Nolvadex) is an oral medication that interferes with oestrogen activity. It is used to treat advanced breast cancer, as an adjuvant for early-stage breast cancer and as a preventive treatment for women at high risk of developing breast cancer. Nursing implications for tamoxifen are presented in the 'Medication administration' box below.

Immunotherapy, using trastuzumab (Herceptin), is used to stop the growth of breast tumours that express the HER2/neu



A Aspiration biopsy

B Excisional biopsy

FIGURE 48.8 ■ Types of breast biopsy. *A*, In an aspiration biopsy, a needle is used to aspirate fluid or tissue from the breast. *B*, In an excisional biopsy, tissue from the breast lesion is removed surgically

MEDICATION ADMINISTRATION Tamoxifen

TAMOXIFEN (NOLVADEX)

Tamoxifen is the most widely prescribed breast cancer medication, commonly given to prevent recurrence of oestrogen-positive breast cancer in postmenopausal women. It inhibits tumour growth by blocking the oestrogen receptor sites of cancer cells. Tamoxifen increases a woman's risk of developing endometrial cancer, deep venous thrombosis (DVT) and pulmonary embolism.

Nursing responsibilities

- Assess for potential contraindications to therapy.
- Assess liver function tests; tamoxifen may interfere with liver function.

Health education for the woman and family

- If in childbearing years, use a non-hormonal, barrier form of contraception; tamoxifen has adverse effects on the developing foetus.
- Take the medication as prescribed until the doctor indicates otherwise.
- Side effects such as hot flushes, vaginal dryness, irregular periods and weight gain are commonly experienced by women taking tamoxifen.
- If you are a smoker, stop. Smoking further increases the risk of DVT and is linked to the development of cancer.
- Promptly report any abnormal vaginal bleeding (non-menstrual bleeding, bleeding after menopause) to your primary care provider.

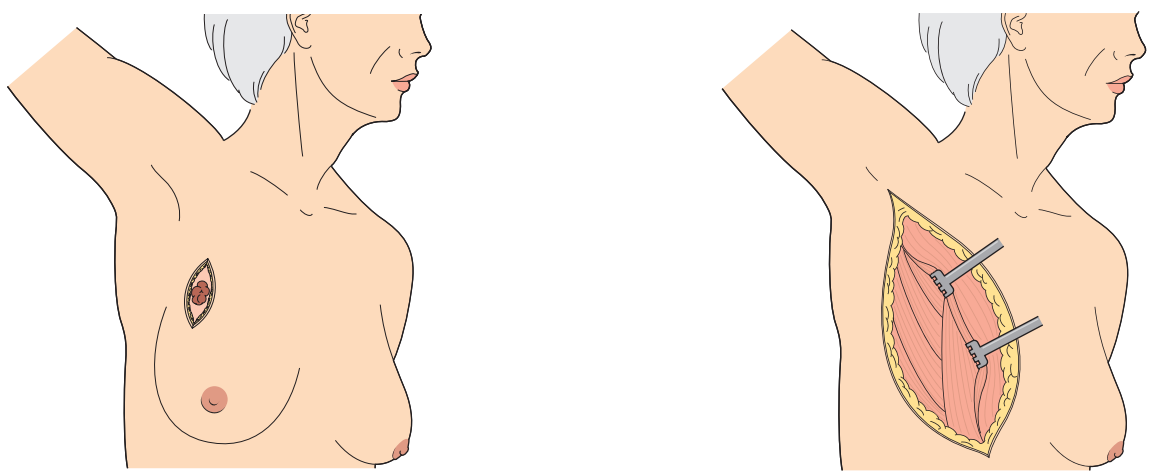
receptor (which binds an epidermal growth factor that contributes to cancer cell growth) on their cell surface. This medication is a recombinant DNA-derived monoclonal antibody that binds to the receptor, inhibiting tumour cell proliferation.

Chemotherapy has become the standard of care for the majority of breast cancer cases with axillary node involvement. In late metastatic disease, chemotherapy becomes the primary treatment to prolong the woman's life. Chemotherapy is discussed in Chapter 13. Adjuvant (additional) systemic therapy following primary treatment for early-stage breast cancer refers to the administration of chemotherapy and other pharmacological agents. This type of therapy has been widely studied; its use reduces the rates of recurrence and death from breast cancer. For example, bevacizumab (Avastin), when combined with chemotherapy to treat metastatic breast cancer, has extended cancer-free survival; and letrozole (Femara) (an aromatase inhibitor) has reduced the risk of recurrence after surgery (in some cases, more effectively than tamoxifen).

Surgery

Until recently, the treatment of choice for breast cancer was a radical mastectomy. The trend now is towards more conservative surgery combined with chemotherapy, hormone therapy or radiation, depending on the stage of the tumour and the age of the woman.

MASTECTOMY There are various types of mastectomy for breast cancer. *Radical mastectomy* is the removal of the entire affected breast, the underlying chest muscles and the lymph nodes under the arms. *Simple mastectomy* is the removal of the complete breast only. *Segmental mastectomy* or *lumpectomy* (see Figure 48.9A) is the removal of the tumour and the surrounding margin of breast tissues. *Modified radical mastectomy*



A Lumpectomy

B Modified radical mastectomy

FIGURE 48.9 ■ Types of mastectomy. *A*, In a lumpectomy, only the tumour and a small margin of surrounding tissue are removed. *B*, In a modified radical mastectomy, all breast tissue and the underarm lymph nodes are removed, but the underlying muscles remain

is the removal of the breast tissue and lymph nodes under the arm (axillary node dissection), leaving the chest wall muscles intact (see Figure 48.9B). See below for the nursing care of a woman having a mastectomy.

Axillary node dissection is generally performed during surgery for all invasive breast carcinoma to stage the tumour. This surgery can cause lymphoedema (accumulation of fluid in the soft tissues of the arm caused by removal of lymph channels), nerve damage and adhesions, and because of the role of the lymph nodes in immune system function, non-surgical methods of detecting lymph node involvement are used. Sentinel node biopsy prior to a node dissection is conducted by injecting a radioactive substance or dye into the region of the tumour. The dye is carried to the first (sentinel) lymph node to receive lymph from the tumour and would therefore be the node most likely to contain cancer cells if the cancer had metastasised. If the sentinel node is positive, more nodes are removed. If it is negative, further node evaluation is usually not indicated.

LUMPECTOMY Breast conservation surgery (*lumpectomy*) may be defined as excision of the primary tumour and adjacent breast tissue followed by radiation therapy. Many women are candidates for this procedure; however, women who have multicentric breast neoplasms and those who have large tumours in relation to their breast size are examples of unsuitable candidates. Selection of women for this procedure is guided by the need for local control of the lesion, cosmetic results and personal preference.

BREAST RECONSTRUCTION After a mastectomy, some women may choose to have their breast reconstructed. They report that surgical reconstruction of the breast simplifies their lives and restores a sense of body integrity. Other women choose to use a removable breast prosthesis and some women are comfortable without reconstruction or a prosthesis.

Breast reconstruction may be performed at the time of the mastectomy or at any time thereafter, depending on the woman's preference. A number of procedures may be used for the breast reconstruction (see Figure 48.10). These include placement of a submuscular implant, the use of a tissue expander followed by an implant, the transposition of muscle and blood supply from the abdomen or back, or using (most often) the transverse rectus abdominis myocutaneous (TRAM) free-tissue flap. Nursing implications for the care of women undergoing breast reconstruction surgery are summarised in the box below.

Radiation therapy

Radiation therapy is typically used following breast cancer surgery to destroy any remaining cancer cells that could cause recurrence or metastasis. If a tumour is unusually large, radiation may be used to shrink the tumour prior to surgery. Radiation therapy is most commonly used in combination with lumpectomy for early stage (I or II) breast cancer. Palliative radiation therapy is also used to treat chest wall recurrences and some bone metastases to help

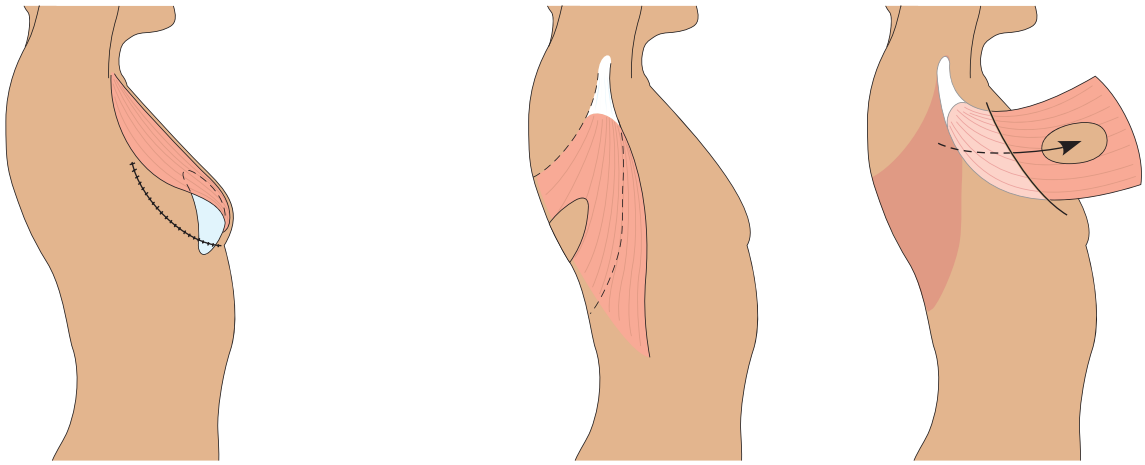
NURSING CARE OF THE WOMAN having a mastectomy

PREOPERATIVE CARE

- Ensure that the woman or family member signs an informed consent form.
- See Chapter 3 for preoperative preparation.

POSTOPERATIVE CARE

- Deep-breathing exercises are important because after general anaesthesia it is difficult for air to reach the lungs, particularly with the restrictive surgical dressing that decreases chest expansion.
- A suction apparatus will be placed in the wound to allow drainage of excess body fluids that accumulate when the lymph nodes are removed. This device is usually removed 3 to 5 days after surgery.
- An IV line may be in place for fluid replacement and antibiotics to reduce the risk of postoperative infection.
- Control pain by using the patient-controlled analgesia device or requesting analgesics before pain becomes severe. Take analgesics as needed before performing recommended exercises to facilitate full movement.
- Note any signs of bleeding on the dressing or on the bedding.
- Numbness or feelings of 'pins and needles' in the axillary area are common.
- Lying on one's back or on the side not operated on helps fluid drain from the site.
- Moving the arm on the operated side helps regain mobility; specific exercises will be prescribed for increasing mobility after the incisions have healed.
- If fluid builds up after the drains have been removed, it can be aspirated by the surgeon.
- Use caution if lifting heavy objects with the arm on the operated side.
- Be careful about injury and infection on the affected side; wear rubber gloves when washing dishes, garden gloves when working outside. Request that caregivers not perform blood pressures or venipunctures on the operative side to reduce the risk of injury and infection.
- Feelings of anxiety, sadness and fear of looking at the incision are normal; mastectomy means abrupt change in body image. It is normal to mourn the loss of a breast and to fear the loss of one's life after a cancer diagnosis.
- Sexual intimacy can be affected by mastectomy; it often helps to be able to discuss potential sexual problems with one's partner, a counsellor or a breast cancer support group.



A Implant **B** Latissimus dorsi musculocutaneous flap

FIGURE 48.10 ■ Types of breast reconstruction surgeries. *A*, A breast implant is inserted under the pectoris muscle. *B*, Autogenous procedures transfer a flap of skin, muscle and fat from the donor site on the woman's body to the mastectomy site. The most frequently used donor muscle sites are the latissimus dorsi and the rectus abdominis (the TRAM flap)

control pain and prevent fractures. Radiation therapy is administered by means of an external beam or tissue implants.

A new radiation treatment (*intraoperative radiotherapy*) is provided by a single, concentrated dose of radiation. During surgery, a probe is inserted into the cavity created by the lumpectomy and radiation equivalent to 6 weeks of doses is emitted for about 25 minutes. If this proves successful, the treatment could make lumpectomy available to more women and prevent the woman from having 6 weeks of daily radiation treatments following surgery.

Nursing care

Breast cancer is not one disease entity, but many, depending on the affected breast tissue, the tissue's oestrogen dependency and the age of the person at onset. The psychological and social impact of breast cancer extends beyond the fear and threat of death. The diagnosis may transform the woman's sense of self and lead to reintegration or negotiation of family relationships. A nursing care plan for a woman with breast cancer is found below.

NURSING CARE OF THE WOMAN having breast reconstruction

HEALTH EDUCATION FOR THE WOMAN AND FAMILY

- Controversy exists about the health effects of silicone. While there is no conclusive evidence that silicone implants induce cancer or autoimmune disease, they are associated with hardening and pain due to contracture of the capsule around the implant. The implant may rupture, releasing silicone gel, or infection may occur. Saline-filled breast implants may be an alternative.
- Reconstruction can be done immediately after a mastectomy or at any time later on. Some surgeons believe that delayed reconstruction offers better cosmetic results.
- Reconstructive surgery can create a natural-looking breast that makes clothes fit better. Since it has no nerve endings, however, the reconstructed breast has no feeling or sensations.
- If a simple mastectomy is done, an implant approximately the same size as the other breast is placed under the pectoral muscle on the operative side. This creates a breast mound that closely resembles the natural breast in shape and softness. If the implant is placed over the pectoral muscle, a high degree of firmness may occur.
- With a simple mastectomy or modified radical mastectomy, a tissue expander may be used to replace the breast. The tissue expander is placed under the pectoral muscle and gradually expanded with saline injections every 2 to 3 weeks to stretch the overlying skin and create a pocket. After a period of time, usually 1 to 2 months, the tissue expander is exchanged for a saline implant.
- With more extensive surgery such as radical mastectomy, a flap of skin, fat or muscle is transferred from a donor site to the operative area. A new nipple may be created by using tissue from the opposite nipple or from the inner thigh.
- Reconstructive surgery may require multiple surgeries, including all the risks associated with anaesthesia. As the complexity of the procedures increases, so does the risk of complications such as infection.
- To decrease the risk of a fibrous capsule forming around the implant, it is important to perform breast massage as instructed.

NURSING CARE PLAN A woman with breast cancer

Vanessa Cole is a 46-year-old mother of three—Sarah, aged 20, Rory, aged 18, and Jennifer, aged 16. Due to a family history of breast cancer, she has been closely monitored (annual mammograms and clinical breast examination, monthly BSE, a needle aspiration biopsy with negative findings) for 4 years prior to her diagnosis. Vanessa discovers a lump in her left breast during her monthly BSE. An incisional biopsy reveals invasive lobular carcinoma in the left breast. Vanessa is debating whether to have reconstructive breast surgery. One of her greatest concerns is how her illness will affect her ability to support and care for her family. The breast cancer diagnosis seems part of the family legacy. She wonders, 'When will it happen to Jennifer? To Sarah?'

ASSESSMENT

During the history, Marc Acut, RN, the admitting nurse, learns from Vanessa that her mother, two of her aunts and one sister had been diagnosed with breast cancer. Her mother and one of the aunts died before age 45. Physical assessment findings include T 37.0°C, BP 110/62, P 65, R 14. Her weight is 54 kg; she is 168 cm tall. Modified radical mastectomy is performed; histological examination shows a 3 cm tumour; axillary node dissection shows that 4 of 16 lymph nodes are positive.

DIAGNOSES

- *Infection risk* related to surgical incision.
- *Acute pain* related to surgery and postoperative recovery.
- *Body image altered* in response to loss of her breast.
- *Personal conflict* about treatment options, related to concerns about risks and benefits.
- *Personal fear* related to current and future wellbeing related to the disease process/prognosis.
- *Anxiety.*

PLANNING

- Educate Vanessa about handwashing and wound care.
- Assess her pain tolerance and administer analgesics as prescribed.
- Educate her to use caution when moving the arm on the operated side, to avoid lifting heavy objects and to wear gloves when gardening.
- Encourage her to discuss her thoughts and feelings about her body changes.
- The Breast Cancer Network www.bcna.org.au or her state/territory Cancer Council can also be a good source of support; also social work or community health referral to assist with care of her children while undergoing treatment. If Vanessa lives in a rural or remote rural area of Australia, her family may need to be temporarily relocated to be with her.
- Assess her interest in spiritual/religious support and refer if appropriate.
- Discuss the use of a temporary prosthesis and later the fitting of a permanent prosthesis (6 to 8 weeks after surgery), the need to be fitted by an experienced person and health insurance reimbursement for the prosthesis.
- Discuss the possibility of attending a breast cancer support group where she can draw on the experiences of other women who have undergone mastectomy, chemotherapy or radiation.
- Encourage her to verbalise her fears about her own prognosis and about her daughters' future risk of breast cancer; assess the need/interest for referral to psychological counselling in the future. However, these services may not readily available in some geographical regions of Australia. Registered Nurses as health professionals can provide information, education and reassurance.

Expected outcomes

- Remain free of infection.
- Experience minimal pain or discomfort during her recovery.
- Maintain a positive body image, regardless of her decision about reconstruction.
- Evaluate the treatment options in relation to personal values and decide on a course of action.
- Identify the sources of her fear and demonstrate behaviours that may reduce fears.

IMPLEMENTATION

- Initiate each of the steps outlined in the planning section detailed above.
- Monitor the effectiveness of the plan by monitoring Vanessa's response to:
  - surgery
  - pain management.
- Engage in ongoing discussion with Vanessa about the implementation of the plan and her psychosocial wellbeing.

EVALUATION

At discharge, Vanessa has no signs of physical complications and is looking forward to being at home with her daughters as temporary caregivers. Vanessa contacted a Breast Cancer Network volunteer, who sent her a temporary prosthesis and booklets about postmastectomy exercises, chemotherapy and breast reconstruction. The volunteer also referred her to a local cancer support group. Vanessa has talked about her concerns related to breast reconstruction. 'I want to avoid anything that would increase the risk of complications. The possibility of recurrence and my fear for my daughters' future health are more than enough to worry about.'

CRITICAL THINKING IN THE NURSING PROCESS

1. What role could genetic counselling play in helping Vanessa and her daughters better understand the daughters' risk of breast cancer?
2. Describe the types of mastectomies and their implications for nursing care.
3. Which medications might help minimise the side effects of chemotherapy?
4. Develop a plan of care for Vanessa for her *Disturbed sleep pattern*.

REFLECTION ON THE NURSING PROCESS

1. Outline what you have learned from Vanessa's case study. How would you incorporate this into your future nursing practice?
2. Which strategies would you use in your future nursing practice to assist women who are undergoing treatment for breast cancer?

Health promotion

All women should be taught to perform BSE monthly (see Box 48.1). Premenopausal women should perform BSE between 7 and 10 days from the first day of their menstrual period, because hormonal changes increase breast tenderness and lumpiness prior to menses. Postmenopausal women should choose one date of the month (e.g. the first day of the month) for BSE.

Educational messages about breast cancer screening need to be culturally and age sensitive to the intended audience. Media campaigns promoting mammography often show young white women, an approach that has proved ineffective for Aboriginal and Torres Strait Islander women (see the 'Translation to practice' box below). By working with women from culturally and linguistically diverse (CALD) backgrounds, nurses can help make breast cancer education more meaningful to women in these groups.

Assessment

Undertake a through health history and physical examination (see Chapter 46). Further focused assessments are described with nursing interventions.

- *Health history:* family history of breast cancer, breast changes, nipple discharge, use of HRT, personal history of breast cancer, previous diagnostic tests and treatment for cancer, menstrual history, pregnancies, alcohol intake, physical activity, dietary history.
- *Physical assessment:* height and weight, breasts, lymph glands.
- *Mental health assessment:* anxiety, coping strategies, emotional wellbeing.

BOX 48.1 Breast self-examination (BSE)

- Observe your breasts for any visual changes.
- Lie down on your back and place your right arm behind your head. (BSE should be done while lying down because this position spreads breast tissue evenly over the chest wall, making it easier to feel all the breast tissue.)
- Use the finger pads of the middle fingers on your left hand to feel for lumps in the right breast. Use overlapping 20-cent-sized circular motions of the finger pads to feel the breast tissue.
- Use three different levels of pressure to feel all the breast tissue. Light pressure is needed to feel the tissue closest to the skin; medium pressure to feel a little deeper; and firm pressure to feel the tissue closest to the chest and ribs. A firm ridge in the lower curve of each breast is normal. Use each pressure level to feel the breast tissue before moving on to the next spot.
- Move around the breast in an up and down pattern starting at an imaginary line drawn straight down your side from the underarm and moving across the breast to the middle of the chest bone (sternum, breastbone). Be sure to check the entire breast area before going down until you feel only ribs and up to the neck or collar bone.
- Repeat the exam on your left breast, using the finger pads of your right hand.
- Stand in front of the mirror with your hands pressing firmly down on your hips. Look at your breasts for any changes in size, shape, contour or dimpling.
- Examine your underarm while sitting or standing and with your arm only slightly raised.
- If you find any changes, see your healthcare provider as soon as possible.

Nursing diagnoses and interventions

Although each woman has individual needs, nursing priorities prior to surgery are concerned with *Anxiety*, *Decisional conflict*, *Grief*, *Risk of infection*, *Risk of injury* and *Disturbed body image* over the loss of a breast. As the typical hospital stay is short, usually 2 to 3 days, preoperative education is often done on an outpatient basis.

Anxiety

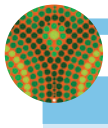
The woman with breast cancer is often anxious about the diagnosis, the surgery, the outcome of surgery if nodal involvement is found and the possible changes in sexual and family relationships. Studies show that young women with breast cancer, a growing population, are particularly vulnerable to anxiety and other psychosocial effects, as are their spouses and their children.

- Provide opportunities to express thoughts and feelings. In this process, the woman can name her fears. *Once the fears are named, the nurse may simply listen, educate or dispel fears that stem from lack of understanding.*
- Discuss with the woman her knowledge of breast cancer. *Assessing the woman's knowledge of breast cancer helps the nurse plan more effective health education.*
- Encourage discussion relating to immediate concerns about resuming her life at home and the changes she must make. *Anticipatory guidance can help plan for and cope with changes in her life and relationships.*
- Explain the surgical procedure, including information about preoperative medications, anaesthesia and recovery. *Knowing what to expect helps to decrease anxiety.*
- Explain that it is normal to have decreased sensation in the surgical area. *Severed or damaged nerves reduce sensation.*

Decisional conflict

The woman with breast cancer must make life-changing decisions about treatment within a relatively brief and highly stressful time. Her age, menopausal status, relationships and stage of cancer are only some of the factors that affect her decisions. Culture, values, lifestyle, socioeconomic status and self-esteem also are considered.

- Provide an opportunity for the woman to ask questions; answer them as simply and directly as possible. Make eye contact as appropriate and pay attention to body language. *During this time, the woman can process information and make informed decisions.*
- Focus on immediate concerns and provide up-to-date written material for the woman to review. *Written material provides easy reference to information not processed immediately because of anxiety and stress.*
- Listen to the woman in a non-judgmental and respectful manner during her decision-making process.



TRANSLATION TO PRACTICE Evidence-based practice: breast and cervical cancer in Aboriginal and Torres Strait Islander women

Despite efforts to improve both the diagnosis and treatment of women with breast and cervical cancer, Aboriginal and Torres Strait Islander women experience a higher incidence of cervical cancer, and have poorer outcomes for breast and cervical cancer than non-Aboriginal and Torres Strait Islander women (Shannon et al., 2011). Data show that Aboriginal and Torres Strait Islander women experience less incidence of breast cancer than non-Aboriginal and Torres Strait Islander women, yet the survival outcome is poorer. It is believed that this statistic is the result of Aboriginal and Torres Strait Islander women's advanced stage of disease at diagnosis, primarily due to a delay in seeking treatment. Seeking treatment is difficult in rural and remote areas of Australia where health services are few or non-existent (Sabesan et al., 2012).

Reath and Carey (2008) conducted a study that explored whether improved service coordination and access to healthcare, general practitioner knowledge of cultural issues and health promotion and a recall system improved detection of cervical and breast cancer in Aboriginal and Torres Strait Islander women. The researchers found that partnership with the Aboriginal and Torres Strait Islander community and Aboriginal and Torres Strait Islander health workers when planning and delivering healthcare services was the key to improving Aboriginal and Torres Strait Islander women's participation in cervical and breast screening processes. In addition, these researchers found that the availability of a female general practitioner also enhanced Aboriginal and Torres Strait Islander women's attendance at healthcare clinics.

IMPLICATIONS FOR NURSING

Nurses must consider multiple factors when considering what may or may not influence Aboriginal and Torres Strait Islander women to delay diagnosis and treatment for cervical and breast cancer, including intra- and intercultural differences and similarities, as well as perceptions and health beliefs. Nurses need to conduct accurate assessments and design interventions based on considerations of both individual and group cultural differences. Culturally safe and appropriate healthcare is paramount.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Which barriers to cervical and breast cancer screening in women of all cultures and ethnicities can you identify? Do you think these barriers differ based on culture, race or socioeconomic level? Why or why not?
2. Are there barriers to cervical or breast cancer screening that might be unique to Aboriginal and Torres Strait Islander women?
3. What type of questions would you include in a health assessment to identify if an Aboriginal and Torres Strait Islander woman may be worried about cervical or breast cancer, but has not sought diagnosis?

Non-judgmental, empathic listening helps the woman process information and make informed decisions. Only she knows the context of her life.

- If the woman wishes, provide opportunities for her to meet with other women who have had breast cancer surgery. *Not all women are ready to meet others in their situation, but opening the door to this resource is appropriate. The woman may choose to talk with these women after the surgery.*
- Facilitate a team approach with the surgeon, anaesthetist, oncologist, plastic surgeon and other health professionals. *Being the woman's advocate during this time of anxiety and decision making reduces the stress of coordinating multiple healthcare provider schedules.*

Anticipatory grieving

Breast surgery, even lumpectomy, alters the appearance of the breast. This loss is expressed through grief and anger.

- Listen attentively to expressions of grief and watch for nonverbal cues (failure to make eye contact, crying, silence, anger, bargaining). *Not all women will express grief clearly; sometimes unspoken grief is the most painful. Grief is best relieved only when expressed in a non-threatening environment.*
- Allow time to interact and do not rush interactions. *Taking time to be with the woman communicates caring.*
- Explain that it is normal to have periods of depression, anger and denial after breast surgery. *All these feelings are appropriate expressions of grief.*
- If the woman wishes to do so, involve the partner in helping her cope with her grief. Remember that the partner may also be grieving. *Not all women want to share their grief and not all partners are interested and supportive.*

Risk of infection

Like any person undergoing surgery, the woman who has breast surgery is at risk of infection. Removal of lymph nodes and the presence of a draining wound increase the risk.

- Assess the surgical dressings for bleeding, drainage, colour and odour every 4 hours for 24 hours and document your findings. Circle any visible bleeding and drainage on the dressing as a baseline for subsequent assessment. *Excessive bleeding or drainage signals postoperative complications that may require emergency attention.*
- Observe the incision and IV sites for pain, redness, swelling and drainage. Assess the drainage system for patency and adequate suction; note the colour and amount of drainage. *Careful observation for any signs of infection is essential because the woman's immune system is compromised. IV catheters should be placed on the uninvolved side only.*
- Change dressings and IV tubing using aseptic technique. Moist dressings and intravenous tubing provide sites for bacterial growth. *Routine dressing and IV tubing changes using aseptic technique reduce the risk of infection.*
- Encourage a protein-rich diet. Discuss the woman's nutritional status with the dietitian and request a

consultation for the woman. *Adequate nutrition promotes healing and boosts the immune system.*
- Teach the woman how to care for the drainage system, if present. (Clean the site, empty the device and record the amount, colour and type of drainage.) *The woman is often discharged prior to removal of the drainage system and dressings, and needs health education to provide self-care.*
- At discharge, educate the woman how to recognise and report to her healthcare provider the symptoms of infection: fever, redness or hardness at the surgical site or purulent drainage. Any of these symptoms should be reported to the physician/surgeon. *Knowing the signs and symptoms of infection prepares the woman to seek prompt treatment if infection occurs.*
- Explain that she may experience scaling, flaking, dryness, itching, rash or dry desquamation of the skin, particularly after radiation therapy. *Impaired skin integrity increases the risk of infection.*
- Tell the woman to avoid deodorants and talcum powder on the affected side until the incision is completely healed. *These substances may irritate the skin and impede healing.*

Risk of injury

Removal of the lymph nodes puts the woman at risk of injury and long-term complications such as lymphoedema and infection.
- When obtaining blood pressure and starting IVs, use the non-surgical side. *Compression of the arm on the surgical side may cause lymphoedema.*
- Elevate the affected arm higher than the shoulder on a pillow, but do not abduct it; the hand should be higher than the elbow. *Elevating the arm permits drainage, prevents swelling and promotes circulation.*
- Encourage ROM exercises in the affected arm. *Exercise helps develop collateral drainage.*
- Explain that lymphoedema massage and an elastic compression bandage may help control the swelling after she has recovered from surgery. *It is important that women know about the resources available after recovery.*

Disturbed body image

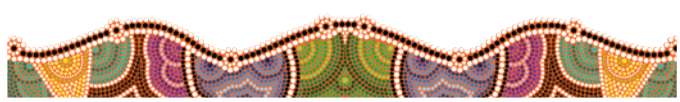
Breast surgery can change the woman's body image. The surgical changes may be compounded by weight gain and other side effects of chemotherapy or hormone therapy. Self-esteem also affects adjustment to a changed body image.
- Assess how the woman views her body. Discuss with the woman what image of herself she had prior to surgery. *Self-image is related to self-esteem. Discuss whether her self-image has changed.*
- Explain that redness and swelling in the scar will fade with time. *The knowledge that the scar will fade may give the woman a more realistic view of the changes.*
- Include the partner and family if possible when discussing the plan of care and activities of daily living (ADLs). Request consultation with a mental health professional if the woman is interested. *Discussion with the partner and family can facilitate the woman's emotional healing process.*
- Offer pamphlets and suggest books and DVDs and appropriate websites that might increase knowledge about what lies ahead. *Knowing what to expect can help the woman cope.*
- Encourage the woman to look at her incision when she feels ready; often the reality is not as frightening as she had imagined. Explain that it is normal to be afraid to look. *Reassurance that her behaviour is normal decreases anxiety.*
- If the woman is interested in breast reconstruction, provide written material and encourage her to talk (in person, telephone or online) with a plastic surgeon and with women who have had reconstruction. *It is important for the woman to be fully informed about available options to make an informed decision.*

CONSIDERATION FOR PRACTICE

Offer referral to support groups with women experiencing similar problems. Some women may prefer one-on-one counselling. However, these services may not readily available in some geographical regions of Australia. Registered Nurses as health professionals can provide information, education and reassurance.

Community-based care

The woman with breast cancer and her family have much to learn to provide self-care at home. Address the following topics in preparation for home care:
- Symptoms of infection and the need to report any that occur to her healthcare provider.
- The importance of ADLs, such as eating properly, combing her hair and washing her face.
- Postmastectomy exercises and lymphoedema care (see Figure 48.11), as discussed with physicians and physical therapists.
- The need for adequate rest and emotional support.
- Participation in a breast cancer support group and online information services and bulletin boards for sources of education and support.
- Prosthesis management, if this option is chosen. (A temporary lightweight prosthesis may be worn immediately after the drains and sutures have been removed from the surgical site. Due to prostheses being expensive, a permanent one should not be purchased until the wound has completely healed. Prostheses are available at medical stores and many larger department stores. Most health insurance policies pay for the first prosthesis.)
- Helpful resources:
  - Breast Cancer Network Australia: www.bcna.org.au
  - Cancer Council Australia—*After a diagnosis*: www.cancer.org.au/about-cancer/after-a-diagnosis/
  - Cancer Council NSW—*Understanding breast cancer*: www.cancercouncil.com.au/wp-content/uploads/2014/09/Breast-Booklet_-NSW-lversion.pdf



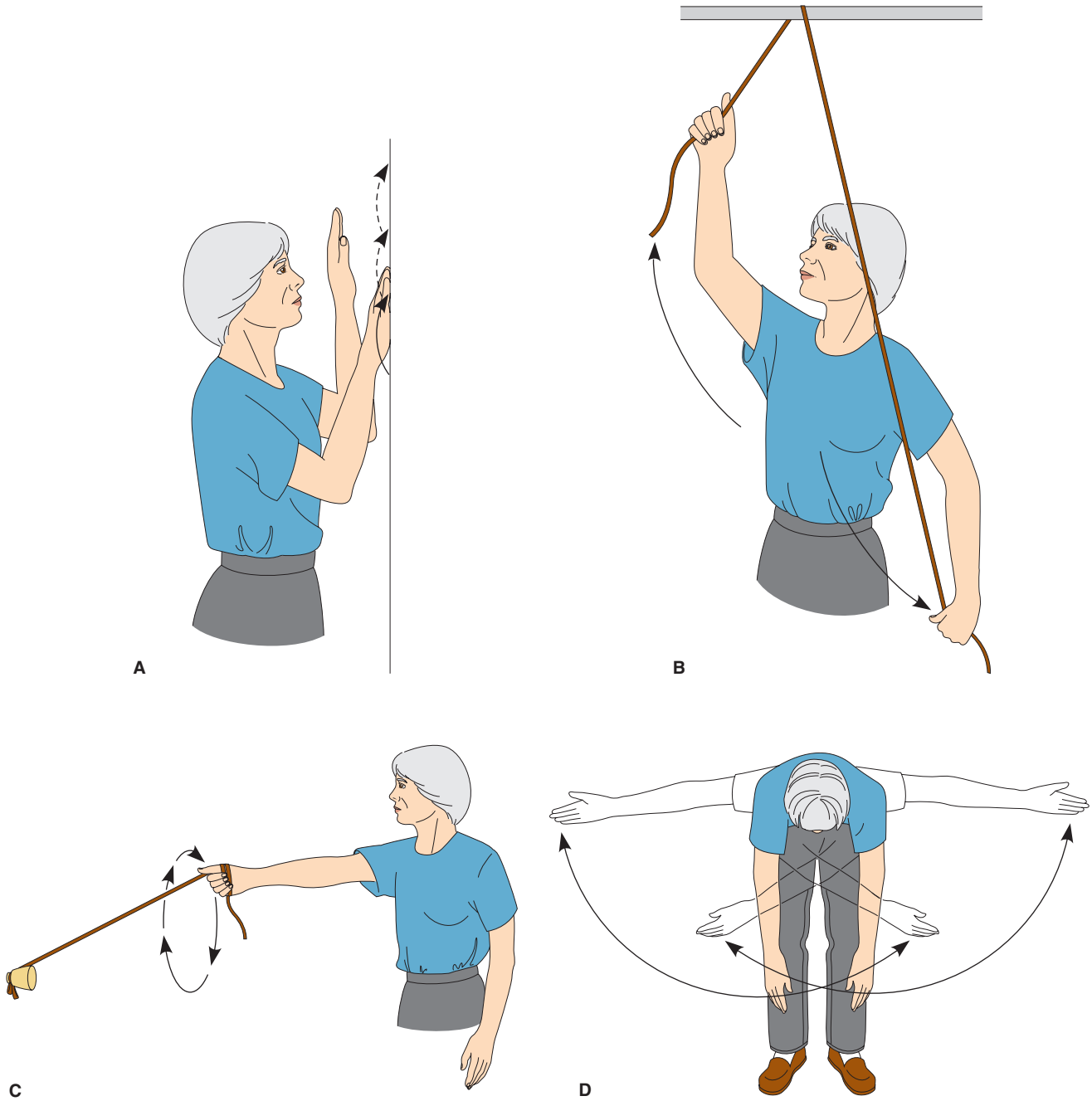


FIGURE 48.11 ■ Postmastectomy exercises. *A*, Wall climbing: stand facing wall with toes 15 to 30 cm from wall. Bend elbows and place palms against wall at shoulder level. Gradually move both hands up the wall parallel to each other until incisional pulling or pain occurs. (Mark that spot on wall to measure progress.) Work hands down to shoulder level. Move closer to wall as height of reach improves. *B*, Overhead pulley: using operated arm, toss 1.8 metre rope over shower curtain rod (or over top of a door that has a nail in the top to hold the rope in place for the exercise). Grasp one end of rope in each hand. Slowly raise operated arm as far as comfortable by pulling down on the rope on opposite side. Keep raised arm close to your head. Reverse to raise unoperated arm by lowering the operated arm. Repeat. *C*, Rope turning: tie rope to door handle. Hold rope in hand of operated side. Back away from door until arm is extended away from body, parallel to floor. Swing rope in as wide a circle as possible. Increase size of circle as mobility returns. *D*, Arm swings: stand with feet 20 cm apart. Bend forwards from waist, allowing arms to hang towards floor. Swing both arms up to sides to reach shoulder level. Swing back to centre, then cross arms at centre. Do not bend elbows. If possible, do this and other exercises in front of a mirror to ensure even posture and correct motion

CHAPTER HIGHLIGHTS

- Disorders of female sexual function include dyspareunia, inhibited sexual desire and orgasmic dysfunction. Nurses should be able to obtain a sexual history, discuss sexual concerns and make appropriate referrals without embarrassment.
- Menopause, a normal physiological event in the lifespan of a woman, is the permanent cessation of menses. Loss of oestrogen results in widespread tissue changes and increases the risk of osteoporosis, fractures and cardiovascular disease.
- Menstrual disorders encompass PMS, dysmenorrhoea and abnormal uterine bleeding. Nursing interventions are focused on providing health education about good health practices and interventions to relieve symptoms. Interprofessional care includes a therapeutic D&C and hysterectomy.
- Uterine displacement and vaginal fistulas are structural disorders. Uterine displacements may be treated surgically or with a pessary and may include education about Kegel exercises to minimise urinary leakage. Fistulas may spontaneously resolve if small or may be surgically repaired.
- There are both benign and malignant disorders of female reproductive tissue, including cysts or polyps, leiomyomas, endometriosis and cancers of the cervix, endometrium, ovaries and vulva.
- Leiomyomas (fibroid tumours) are benign tumours that originate from smooth muscle of the uterus. Treatment depends on the size and location of the tumours. Both leiomyomas and endometrial implants (benign implants of endometrial tissue in the pelvic cavity) may interfere with the ability to have a child and are reduced in size after menopause.
- Cervical cancer is a common female reproductive system cancer, but the incidence and mortality has been greatly reduced by the Pap smear for early diagnosis.
- Endometrial cancer is the most common invasive gynaecological cancer in Australia, with one in 69 women being affected by the age of 75 years. The main manifestation is abnormal, painless vaginal bleeding (menorrhagia or metrorrhagia in menstruating women). Postmenopausal women should have annual pelvic examinations and report any unexpected vaginal bleeding to their healthcare provider.
- Ovarian cancer increases in incidence with ageing; one in 78 women will be diagnosed prior to the age of 85. In early stages, there are generally no warning signs.
- Benign breast disorders in women include fibrocystic changes and intraductal disorders. Both treatment and nursing care are primarily symptomatic.
- Breast cancer is the most commonly invasive cancer in Australian women. A strong genetic link has been identified, as have a large number of risk factors. Early diagnosis is possible with BSE, clinical breast examination and mammograms. Treatment includes surgery (one of several types of mastectomy, lumpectomy), radiation therapy and chemotherapy. Both combination therapies and immunotherapy are proving to be effective in suppressing tumour growth and facilitating longer life. Along with other problems, mastectomy and radiation therapy interfere with lymph drainage on the affected side, leading to lymphoedema of the arm.
- Appropriate preoperative nursing diagnoses for the woman with breast cancer include anxiety and fear, decisional conflict and anticipatory grieving. Following surgery, interventions focus on risk of infection, risk of injury, pain and disturbed body image.

CONCEPT CHECK

1 During a health assessment, a woman in her fifties tells you that she is having some pain with intercourse. You are uncomfortable discussing this topic with her. What would you say?
1. 'I know this can be a problem; please discuss it with your doctor.'
2. 'I don't know anything about that; you'll have to ask someone else.'
3. 'Do you normally enjoy sexual activity?'
4. 'What do you think is causing your problem?'

2 Long-term oestrogen deprivation results in an increased risk of physical disorders. What are these? (Select all that apply.)
1. colon cancer
2. osteoporosis
3. cardiovascular disease
4. fractures
5. cervical cancer

3 You are conducting a health education seminar for postmenopausal women. When discussing calcium intake, you recommend _____ mg per day.

4 An intervention for the woman with a uterine displacement disorder is to teach Kegel exercises. These exercises may help reduce:
1. stress incontinence
2. menorrhagia
3. vaginal discharge
4. retroversion

5 Which of the following topics would you include in a health-promotion seminar to reduce the risk of cervical cancer?
1. weight loss
2. safer-sex methods
3. yearly mammograms
4. a diet high in iron

6 When discussing dietary guidelines with a woman with PMS, you recommend she reduce her sodium intake. What is the rationale for this recommendation?
1. Sodium increases reactive hypoglycaemia, increasing physical symptoms.
2. Sodium increases thirst, thereby facilitating increased oral fluid intake.
3. In and of itself, sodium is not harmful, but it may increase cancer risk.
4. Sodium restriction helps minimise fluid retention.

7 What happens to endometrial implantations after menopause?
1. They tend to become malignant.
2. They tend to atrophy and disappear.
3. The number of implants increases.
4. Each implant enlarges.

8 You are designing an education plan for home care for a woman who had an abdominal hysterectomy. Which interventions should be included? (Select all that apply.)
1. Restrict heavy lifting for 4 to 6 weeks.
2. Take tub baths until bleeding has stopped.
3. Report an increase in temperature or severe pain.

4 Take regular rest periods.
5 Avoid coughing and deep breathing at home.

9 Of the following women, which one would be most at risk of breast cancer?
1 age 23, two children
2 age 33, never pregnant
3 age 45, very thin
4 age 64, positive family history

10 You are caring for a woman who is scheduled to have a mastectomy for breast cancer. She is crying. Why might this be so?
1 *Disturbed body image*
2 *Fatigue*
3 *Anticipatory grieving*
4 *Risk of injury*

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CHAPTER 49

NURSING CARE OF PEOPLE WHO HAVE SEXUALLY TRANSMITTED INFECTIONS

LORRAINE FIELDS & LORNA MOXHAM

KEY TERMS

LEARNING OUTCOMES

- Explain the incidence, prevalence, characteristics and prevention/control of sexually transmitted infections (STIs).
- Compare and contrast the pathophysiology, manifestations, interprofessional care and nursing care of genital herpes, genital warts, vaginitis, chlamydia, gonorrhoea, syphilis and pelvic inflammatory disease.
- Explain the risk factors for and complications of STIs.
- Discuss the effects and nursing implications of medications and treatments used to treat STIs.

CLINICAL COMPETENCIES

- Assess the functional health status of people living with an STI and monitor, document and report clinical indicators.
- Determine nursing priorities and select and implement individualised nursing interventions for people living with an STI.
- Administer treatment, including medications, knowledgeably and safely.
- Integrate interprofessional care for people living with an STI.
- Provide education appropriate for prevention, management and self-care of STIs.
- Revise plans of care as needed to provide effective interventions to promote, maintain or restore functional health status for people living with an STI.

Infections transmitted by vaginal, oral and anal intimate contact and intercourse are referred to as **sexually transmitted infections (STIs)**. Infections transmitted by sexual intercourse are also sometimes known as *sexually transmitted diseases (STDs)* or *venereal diseases.* The most contemporary and acceptable term is STI. A person can have an STI before having symptoms of the disease. STIs also include systemic diseases (such as tuberculosis, hepatitis and HIV/AIDS) that can be transmitted from one infected person to another. This chapter discusses STIs that involve the urogenital system, as well as vaginal infections.

OVERVIEW OF SEXUALLY TRANSMITTED INFECTIONS

Sexually transmitted infections include those caused by bacteria, *Chlamydiae*, viruses, fungi, protozoa and parasites. Portals of entry for these agents of transmission include the mouth, genitalia, urinary meatus, anus, rectum and skin. STIs have many consequences and nurses have the responsibility to provide health education to people on how to prevent STIs, regardless of their gender, age or sexual orientation. Nurses have a critical role in the prevention of STIs by providing accurate information about these diseases, their prevention, treatment and potential complications. Nurses should be aware of policies, protocols and strategies. Australia (Department of Health, 2014a) has guidelines within the *Third National Sexually Transmissible Infections Strategy 2014–2017* available at www.health.gov.au/internet/main/publishing.nsf/Content/ohp-bbvs-sti. The World Health Organization (WHO) (2007) has a publication called *Global strategy for the prevention and control of sexually transmitted infections: 2006–2015. Breaking the chain of transmission.* This book is available from www.who.int/reproductivehealth/publications/rtis/9789241563475/en/.

Incidence and prevalence

Sexually transmitted infections are a major public health issue. STIs have reached epidemic proportions in many countries and are on the increase worldwide. The WHO estimates that 500 million new infections of curable sexually transmitted infections (syphilis, gonorrhoea, chlamydia and trichomoniasis) occur globally each year (WHO, 2013).

Accurate information about infection rates is difficult to obtain, especially for many developing countries, and the available information is often at least 3 to 4 years old. There is no one organisation that regularly collates all global STI statistics, although the WHO does try. To complicate matters further, different countries have different types and levels of reporting. It is thought that many reports substantially underestimate the number of new STI cases because of social stigma and other factors, such as poverty, that prevent people seeking healthcare.

Women and infants are disproportionately affected by STIs. Many STIs are more easily transmitted from a man to a woman than they are from a woman to a man. Women often experience few early manifestations of the infection, delaying diagnosis and treatment. Furthermore, women are at greater risk of complications of STIs such as pelvic inflammatory disease (PID) and genital cancers.

Several factors may help explain the escalating incidence of STIs. The 'sexual revolution' of the 1960s and 1970s, fuelled by 'the pill' and the freedom this gave women from unplanned pregnancy, led to more permissive attitudes about sexual freedom and increases in sexual activity and the number of sexual partners. When hormonal contraceptives became available in Australia in the 1960s, they replaced the condom as the predominant means of birth control. However, unlike condoms, oral contraceptives do not protect against STIs, a fact of increasing public health importance.

STIs affect men and women of all ages, backgrounds and socioeconomic levels. Individual factors related to the contraction of STIs are risky behaviours such as drug abuse, sex with multiple or new partners, sex with high-risk partners, unprotected sex, sex while intoxicated and sex in exchange for money. A further factor contributing to the increasing incidence of STIs is that people are becoming sexually active at an earlier age and committing to one partner later. As a result, sexually active people today are more likely to have multiple sex partners in their lifetime and are potentially at higher risk of STIs.

The emergence of HIV/AIDS has created a kind of 'epidemiological synergy' between all STIs. Other STIs, such as syphilis, herpes simplex virus (HSV) and chancroid, facilitate the transmission of HIV/AIDS, and the immunosuppression caused by HIV potentiates the infectious process of other STIs. People who are infected with STIs are at greater risk of acquiring HIV if they are exposed to the virus. This is the result of genital ulcers creating a portal of entry for HIV, non-ulcerative STIs increasing the concentration of cells in genital secretions that can be targets for HIV and infection with both an STI and HIV resulting in an increased likelihood of having HIV in genital secretions and semen.

Pathophysiology, manifestations and nursing care

Although STIs are caused by various organisms, they have several common characteristics:

- Most can be prevented by the use of latex condoms.
- They can be transmitted during sexual activities, including non-penetrating intimate exposure.
- For treatment to be effective, sexual partners of the infected person must also be treated.
- Two or more STIs frequently coexist in the same person.

FAST FACTS

- The majority of STIs are present without symptoms.
- More than 1 million people acquire an STI every day.
- Each year, an estimated 500 million people become ill with one of four STIs: chlamydia, gonorrhoea, syphilis and trichomoniasis.
- More than 530 million people have the virus that causes genital herpes (HSV2).
- More than 290 million women have a human papillomavirus (HPV) infection. HPV causes 530 000 cases of cervical cancer and 275 000 cervical cancer deaths each year.
- Some STIs can increase the risk of HIV acquisition three-fold or more.
- STIs can have serious consequences beyond the immediate impact of the infection itself, through mother-to-child transmission of infections and chronic diseases.
- Drug resistance, especially for gonorrhoea, is a major threat to reducing the impact of STIs worldwide.
- In developing countries, STIs and their complications rank in the top five disease categories for which adults seek healthcare.
- In women of childbearing age, STIs, excluding HIV, are second only to maternal factors as causes of disease, death and healthy life lost.
- According to estimates from the WHO (2015a), around 36.9 million people were living with HIV in 2014. The number of people newly infected with HIV in 2014 was 2 million.
- AIDS deaths globally in 2013 accounted for 1.5 million people.
- AIDS incidence in Australia (0.9 per 100 000 population) is similar to that in the UK and Canada (1.4 and 0.8, respectively) but much lower than in the US (12.8).
- As of December 2011, an estimated 24 731 people were living with an HIV diagnosis in Australia.
- From the start of the epidemic until the end of 2011, there have been 31 645 diagnoses of HIV and 10 796 diagnoses of AIDS. Australia has recorded 6843 AIDS deaths.
- New South Wales had the highest incidence of AIDS diagnosis followed by Victoria, Queensland, Western Australia, South Australia, the Australian Capital Territory, Tasmania and the Northern Territory.
- Chlamydia was the most common STI notification in Australia, with 82 707 new cases in 2012.
- Indigenous Australians are over-represented in STI notification data.
- One in two sexually active people will contract an STI by age 25.

Sources: World Health Organization (2013). *Health topics: Sexually transmitted infections.* Fact sheet 110; Strobel, N. A. & Ward, J. (2012). *Education programs for Indigenous Australians about sexually transmitted infections and bloodborne viruses*. Resource sheet no. 14. Produced for the Closing the Gap Clearinghouse. Canberra: Closing the Gap Clearinghouse; Department of Health (2014b). *Sexually transmissible infections.* Retrieved from www.sti.health.gov.au/internet/sti/publishing.nsf.

The complications of STIs in women include pelvic inflammatory disease, ectopic pregnancy, infertility, chronic pelvic pain, neonatal illness and death, and genital cancer. Some bacterial STIs can be treated through appropriate early intervention with antibiotics. Others, like genital herpes, are chronic conditions that can be managed but not cured because they are caused by viruses. The most serious STI is AIDS, which currently does not have a cure. HIV/AIDS is discussed in Chapter 12. Treatment guidelines for STIs are updated regularly and are available from the World Health Organization and the Centers for Disease Control and Prevention (CDC) websites.

Prevention and control

Prevention and control of STIs are based on the principles of education, early detection, diagnosis and treatment of infected people, and evaluation, treatment and counselling of sexual partners of people who are infected. The skill of the healthcare provider to be able to obtain an accurate sexual history is essential to prevention and control efforts. One approach to collecting accurate information about key areas has been summarised by the CDC (2015). It includes the '5 Ps' partners, prevention of pregnancy, protection from STIs, practice and past history of STIs. Suggested questions to use are found on the CDC website: www.cdc.gov/std/tg2015/clinical.htm.

The most effective way to prevent STIs is to avoid sexual intercourse with an infected partner. It is recommended that both partners be tested for STIs, including HIV, before beginning to have sexual intercourse. If a person chooses to have intercourse with an infected partner or one whose infection status is unknown, a new condom should be used for each act of intercourse (CDC, 2015). See Table 49.1 for STI barrier guidelines.

Health education for the person who is an injecting-drug user includes:

- Enrol or continue in a drug treatment program.
- Do not use injection equipment that has been used by another person. If equipment is shared, first clean the syringe and needle with bleach and water (to reduce the rate of transmission).
- If needles can be legally obtained, use clean needles.
- Dispose of used needles safely and appropriately.

Syringe disposal containers are located in many public places including shopping centres, public toilets, airports, cinemas and universities.

Eliminating further transmission and reinfection of STIs is critical for control. For treatable STIs, this means that referral of sexual partners for diagnosis, treatment and counselling is essential. HIV/AIDS is a reportable disease in every state and territory in Australia. When an infected person is referred, every effort is made to identify and contact sexual partners. Reports of STI and HIV infections are maintained in the strictest confidence and are protected by law from subpoena. Suggested resources for people with STIs are listed in Box 49.1.

TABLE 49.1 STI barrier guidelines

BARRIER PROTECTION	HEALTH EDUCATION
Male condoms	• Use a new condom for each act of sexual intercourse. • Handle carefully to avoid damaging the condom. Condoms are affected adversely by heat—keep them in a cool place. • Be sure no air is trapped in the end of the condom. • Put the condom on when the penis is erect and before genital contact with partner. • Ensure adequate lubrication exists during intercourse, using only water-based lubricants (e.g. K-Y® jelly) and latex condoms. Oil-based lubricants, such as petroleum jelly, massage oil, mineral oil or body lotions can weaken latex and should not be used. • Ensure adequate lubrication during vaginal and anal sex. • Withdraw while the penis is erect and hold the condom firmly against the base of the penis during withdrawal.
Female condoms	• The female condom is a lubricated polyurethane sheath with a ring on each end that is inserted into the vagina. It is an effective mechanical barrier to viruses.
Vaginal spermicides, sponges, diaphragms	• Spermicides used alone without condoms do not reduce the risk of cervical gonorrhoea, chlamydia or HIV infection. • Diaphragms protect against cervical gonorrhoea, chlamydia and trichomoniasis, but not HIV.

BOX 49.1 Resources for people who have STIs

- Sexual Health and Family Planning Australia: www.shfpa.org.au
- Queensland Health Government (sexual health website): http://www.qld.gov.au/health/staying-healthy/sexual-health/index.html
- Australian Government: http://www.sti.health.gov.au/internet/sti/publishing.nsf
- healthdirect Australia: http://www.healthdirect.gov.au/sexual-health-overview Australian Federation of Aids Organisations (AFAO): www.afao.org.au
- Closing the Gap (education programs for Indigenous Australians about sexually transmitted infections and blood-borne viruses): http://www.aihw.gov.au/uploadedFiles/ClosingTheGap/Content/Publications/2012/ctgc-rs14.pdf

THE PERSON WITH GENITAL HERPES

Genital herpes is caused by the herpes simplex viruses HSV1 and HSV2. Like most STIs, genital herpes is most commonly found in young, sexually active people and is associated with early onset of sexual activity and multiple sexual partners. In Australia, one in eight adults have genital herpes. It is twice as common in adult women (one in six) as in adult men (one in 12) and is most prevalent in women aged 35–44 (Sexual Health Australia, 2013). Currently there is no cure and the treatments are primarily symptomatic.

Pathophysiology

Both HSV1 and HSV2 are transmissible via direct contact. HSV1 is associated with cold sores, but may be transmitted to the genital area by oral intercourse or by self-inoculation through poor handwashing practices. HSV2 is the virus that causes genital herpes and is transmitted by sexual activity or during childbirth. HSV infections begin with exposure to the virus by contact with infectious lesions or secretions. The virus then moves into the stratified squamous epithelium, stimulating the replication of the epithelium and infecting the neurons that innervate the area. HSVs are neurotropic viruses, meaning that they grow in neurons and can maintain their disease potential even when there are no manifestations. The virus ascends through the peripheral nerves to the dorsal root ganglia, where it can remain dormant. For unknown reasons, the virus may reactivate and return to the nerve root of the skin, causing lesions. During dormancy, the virus is impervious to treatment. The incubation period ranges from 6 weeks to 8 months. Genital HSV2 infection is more common in women (approximately one in four women) than in men (almost one in five) (CDC, 2015).

Manifestations

Within 2 to 10 days after exposure to the herpes virus, painful red papules appear in the genital area. In men, the lesions generally occur on the glans or shaft of the penis. In women, the lesions commonly occur on the labia, vagina and cervix. Anal intercourse or oral–anal sex may also result in lesions in and around the anus.

Soon after the papules appear, they form small painful blisters filled with clear fluid containing virus particles (see Figure 49.1). The blisters break, shedding the highly infectious virus and creating patches of painful ulcers that last approximately 6 weeks (or longer if they become infected). Touching these blisters and then rubbing or scratching in another place can spread the infection to other areas of the body (*autoinoculation*).

The first outbreak of herpes lesions is called *first-episode infection*, with an average duration of 12 days. Subsequent occurrences, usually less severe, are termed *recurrent infections* (average duration of 4–5 days). The period between episodes is

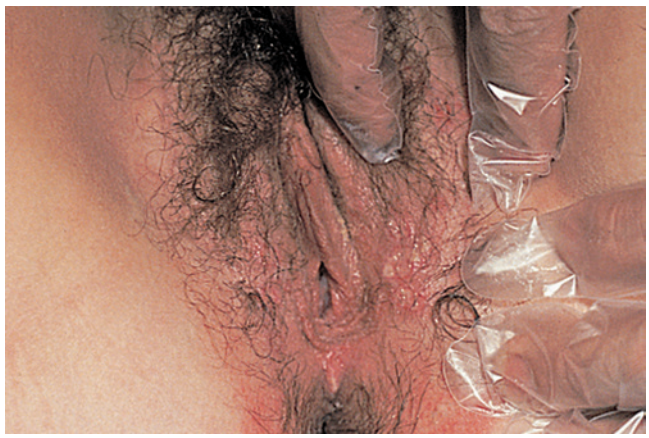


FIGURE 49.1 ■ Genital herpes blisters as they appear on the labia

Source: © Biophoto Associates/Science Source.

called *latency*, during which time the person remains infectious even though no symptoms are present. During latency, the virus withdraws into the nerve fibres that lead from the infected site to the lower spine, remaining dormant until recurrence, at which time it retraces its path to the genital area.

The manifestations of genital herpes are presented below. Prodromal symptoms of recurrent outbreaks of genital herpes can include burning, itching, tingling or throbbing at the sites where lesions commonly appear. These sensations may be accompanied by pain in the legs, groin or buttocks. Some research suggests that prodromal symptoms signal increased levels of infectiousness, during which sexual contact should be avoided.

MANIFESTATIONS Genital herpes

- Herpetic lesions
- Regional lymphadenopathy
- Headache
- Fever
- General malaise
- Dysuria
- Urinary retention
- Vaginal discharge
- Urethral discharge (men)

INTERPROFESSIONAL CARE

Presumptive diagnosis of genital herpes is based on history and physical examination of the person, including lesions and patterns of recurrence. Because there is no cure for genital herpes, treatment focuses on relieving symptoms and preventing the spread of the infection. Health education is essential to prevent further transmission of the disease and to help the person integrate chronic disease management into their life.

Diagnosis

Definitive diagnosis requires isolation of the virus in tissue culture. Ideally, tissue specimens should be obtained within 48 hours of the appearance of the blisters. Diagnostic tests are described in Chapter 46.

Medications

Aciclovir is an antiviral drug. It slows the growth and spread of the herpes virus so that the body can fight off the infection. Aciclovir will not cure herpes, but it can help reduce the length and severity of the first episode and lessen the symptoms of the infection. The oral form is considered most effective for first-episode infections as well as for recurrent infections and is given for 7 to 10 days or until lesions heal. It may also be administered intravenously. Some strains of HSV are becoming resistant to aciclovir, particularly in HIV-positive people. In those cases, foscarnet is used. Other antivirals used for treatment and prevention are valaciclovir and famciclovir.

Nursing care

Planning and implementing nursing care for the person who has genital herpes requires consideration of short- and long-term implications. Although the immediate priority is symptom relief and prevention of further transmission, the person will need help to formulate strategies to deal with the life-changing diagnosis of living with a chronic disease. Stigma is also an important consideration; therefore, nurses need to be aware of the person's psychological, as well as physical, needs.

Nursing diagnoses and interventions

Nursing interventions in this section focus on pain and sexual dysfunction.

Acute pain

Herpetic lesions are very painful and can become infected. Because the virus resides in the nerve ganglia, pain may also occur in the legs, thighs, groin or buttocks. Although aciclovir diminishes the pain of herpes and accelerates the healing process, additional measures can relieve the discomfort further.

- Educate the person on how to keep herpes blisters clean and dry. A solution of warm water, soap and hydrogen peroxide (if lesions are not open) can be used to cleanse the lesions two to three times daily. Burow's solution (a liquid containing aluminium sulfate, acetic acid, precipitated calcium carbonate and water) can also be used. Lesions can be dried using a hair dryer turned to a cool setting. It is important to wear loose cotton clothing that will not trap moisture and avoid wearing items such as lycra, pantyhose and tight jeans. *Keeping the lesions clean and dry reduces the possibility of secondary infection and speeds the healing process.*
- For dysuria, suggest the person pours water over their genitals while urinating. Drinking additional fluids

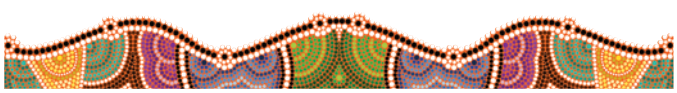
(particularly water) also helps dilute the acidity of the urine. Fluids that increase acidity, such as cranberry juice, should be avoided. *These measures dilute the acid content of urine and thereby reduce the burning sensation.*
- Suggest the person has sitz baths (with tepid water) for 15–30 minutes several times a day. *The warm water is soothing and decreases pain from ulcers and an irritated urethral meatus. It also facilitates wound healing.*

Sexual dysfunction

Some people who learn that they are infected with an incurable STI may believe they can no longer have a normal sex life. Fortunately, many people have learned to live with and manage genital herpes without infecting their partners and/or children.
- Provide a supportive, non-judgmental environment so feelings can be discussed and questions asked freely about what this diagnosis means for future sexual relations. *Feelings of guilt, shame and anger are natural responses and can lead to a total avoidance of sexual intimacy.*
- Provide information about support groups and other resources for people with herpes. *Information about how others cope with this disease can offset feelings of shame and hopelessness.*

Community-based care

Health education for people who are living with genital herpes involves supporting them to manage this chronic disease with the least possible disruption to their lifestyle and relationships. Understanding the disease process and factors that affect it helps the person regain a sense of control and see the potential for future sexual intimacy without transmission of infection. The following topics should be discussed:
- how to recognise prodromal symptoms of recurrence and factors that seem to trigger recurrences (e.g. emotional stress, acidic food, sun exposure)
- the need for abstinence from sexual contact from the time prodromal symptoms appear until 10 days after all lesions have healed
- if lesions become infected, use of topical aciclovir (painful lesions can be protected with sterile petroleum jelly or aloe vera gel)
- use of latex condoms, due to viral shedding at any time, and careful hygiene practices (such as not sharing towels or other personal items) even during latency periods.



THE PERSON WITH GENITAL WARTS

Genital warts (*condylomata acuminata*), caused by the human papillomavirus (HPV), are one of the most common transmissible genital infections and are considered epidemic. Genital warts are chronic and, in many people, largely asymptomatic. Currently, they are incurable.

Genital warts are often described as fleshy growths or bumps seen mostly in areas around the genitals and anus. HPV is spread through direct skin-to-skin contact with a person infected with HPV. This occurs most commonly through sexual contact. HPV may be passed from person to person where there is skin-to-skin contact of the genital area. This can occur even when there are no visible warts, explaining why genital HPV infection spreads easily. HPV may also be passed from mother to baby during childbirth. The virus can live on the skin for many years and during that time can be passed on through sexual contact.

Warts that occur elsewhere on the body are caused by different types of HPV. Contact with these warts is not known to cause genital warts.

Prevention

Some types of HPV infection can be prevented by new vaccines which have been registered for use in Australia. One of the vaccines licensed for use can prevent HPV infection that causes genital warts. The use of male and female condoms for all sexual contact can reduce the transmission of HPV.

Women are at greater risk of HPV genital infections because they have a larger mucosal surface area exposed in the genital area. Most HPV infections are asymptomatic or unrecognised.

HPV infection rates vary greatly between geographical regions and population groups. Almost all cervical cancer is caused by HPV. HPV 16 and 18 are the two most common forms and are known to cause over 70% of cervical cancers. Two vaccines are available that prevent infection from both HPV 16 and 18; however, these vaccines do not treat HPV and it is recommended they are administered prior to first sexual contact (WHO, 2015).

HPV and cervical cancer

All women who have ever had sexual contact should commence having Pap smears from 2 years after first sexual contact. This includes male-to-female and female-to-female contact. Thereafter, Pap smears are routinely done every 2 years, or more frequently if abnormalities are detected. The Gardasil® vaccine, developed by a team led by Australian scientist Professor Ian Frazer, protects against four HPV strains: HPV genital types 6, 11, 16 and 18. HPV types 16 and 18 are linked to 70% of cervical cancers in Australia; types 6 and 11 are linked to approximately 90% of genital warts cases. The National HPV Vaccination Program is ongoing for 12- to 13-year-old girls and boys and provides free vaccination through a school-based

FAST FACTS

- One of the most common STIs is HPV.
- Almost all sexually active people in the US acquire genital HPV infection at some point in their lives.
- Most people with a genital HPV infection do not know they are infected; most women are diagnosed by abnormal Pap tests.

Source: CDC (2015).

program. For information about the program, see the Cancer Council Victoria website www.hpvvaccine.org.au.

The vaccine will not prevent all types of HPV that cause cervical cancer, nor can it 'cure' an HPV infection if it has been acquired previously. There are many different types of the HPV virus that can affect various parts of the body.

Although the majority of infected people are asymptomatic, some experience frequent recurrences. Other than recurrences, men are not likely to experience serious physical complications from genital warts. Women, however, have an increased risk of cervical cancer, with HPV DNA having been identified in almost all cervical cancers worldwide, and in approximately 50–80% of vaginal, vulvar and anogenital cancers.

Pathophysiology

Genital warts, sometimes called venereal warts, are caused by HPV and are transmitted by vaginal, anal or oral–genital/anal contact. The incubation period is between 3 weeks and 8 months (Sexual Health Australia, 2012).

Manifestations

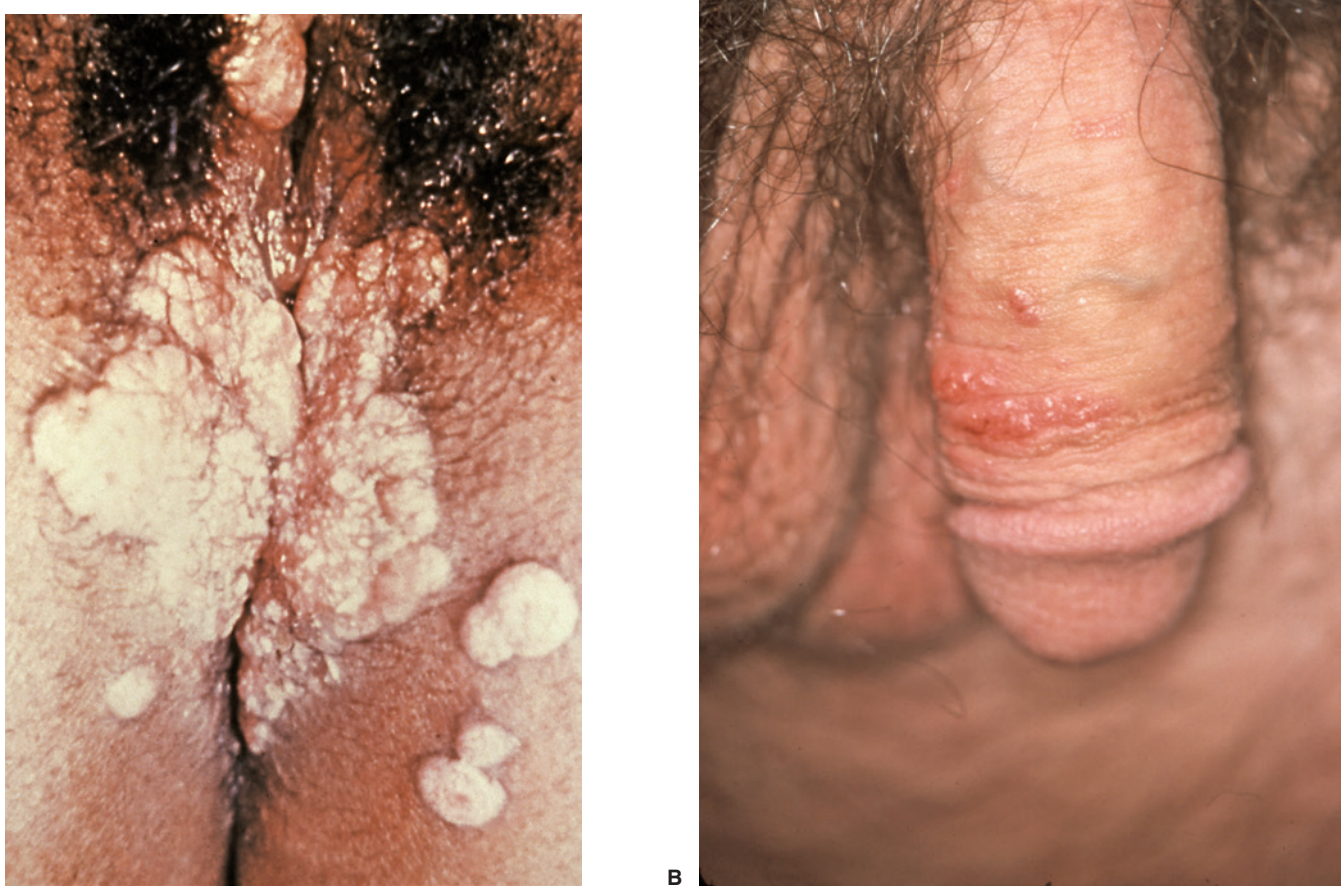
Although some people with HPV may not have manifestations, others exhibit characteristic lesions: single or multiple painless, soft, moist, pink or flesh-coloured swellings in the vulvovaginal area, perineum, penis, urethra, anus, groin or thigh (see Figure 49.2). In women, the growths may be in the vagina or on the cervix and be apparent only during a pelvic examination.

The four types of genital warts are:

1. *Condyloma acuminata:* cauliflower-shaped lesions that appear on moist skin surfaces such as the vagina or anus.
2. *Keratotic warts:* thick, hard lesions that develop on keratinised skin such as the labia major, penis or scrotum.
3. *Papular warts:* smooth lesions that also develop on keratinised skin.
4. *Flat warts:* slightly raised lesions, often invisible to the naked eye, that also develop on keratinised skin.

INTERPROFESSIONAL CARE

Treatment is directed at removal of warts, relief of symptoms and health education to reduce the risk of recurrence and future transmission. Infection with HPV is considered chronic. Research has shown that for about 90% of women, cervical HPV becomes undetectable within 2 years (CDC, 2015).



A B

FIGURE 49.2 ■ Genital warts (*condyloma acuminata*) on the *A*, vulva and *B*, penis

Sources: A, CDC/Richard Deitrick; *B*, CDC/Dr M. F. Rein.

Genital and anal warts are diagnosed primarily by clinical appearance. An HPV DNA test is specific for diagnosis in women. There are currently no HPV tests for men.

Medications

Topical agents can be used to treat genital warts and include podofilox and imiquimod (both can be applied by the person), or podophyllin and trichloroacetic acid (which is administered by health professionals). Podophyllin is contraindicated during pregnancy and can have side effects ranging from nausea, diarrhoea and lethargy, to paralysis and coma (see the following 'Medication administration' box). As already mentioned, Gardasil® is a vaccine developed to prevent genital warts, precancerous genital lesions and cervical cancer due to HPV. It is administered by three intramuscular injections given over a 6-month period. As HPV is so closely associated with cervical cancer, an Australian federal advisory panel recommended that the vaccine be targeted at females aged 9 to 26.

Other treatments

Genital warts may also be removed by cryotherapy, electrocautery, laser vaporisation or surgical excision. Carbon dioxide laser surgery is becoming increasingly common for removal of extensive warts.

Nursing care

Health promotion activities should include information about the causes, treatments and prevention of HPV infections.

Nursing diagnosis and interventions

Nursing interventions primarily involve health education, and discussions on stigma, fear and anxiety. People are often acutely embarrassed.

Knowledge deficits

HPV is spread by contact with infectious lesions or secretions. Up to 70% of genital warts are spread by people who do not know they have the infection. Although there is currently no known cure, it is essential to prevent secondary infections.

- Discuss the need for prompt treatment, and the necessity for sexual abstinence until lesions have healed or using a condom while lesions are present. *This reduces the risk of reinfection and further transmission of the disease. Using condoms promotes the regression of HPV lesions in both men and women.*
- Discuss the increased risk of cervical cancer and the importance of having a Pap smear. *Understanding the risk, the person will be more motivated to seek screening.*
- Stress the importance of thorough handwashing. *Handwashing is essential to prevent the spread of HPV.*

Fear

Surgery engenders some degree of fear in most people: fear of the procedure itself and/or of pain and possible complications. Surgery or cryotherapy in the genital area involves all of these fears plus fear of possible impaired sexual function, as well as stigma.

- Provide opportunities for the person to express their fears and feelings about the procedure. Explain the procedure, approximate recovery time, possible complications and ways to avoid them, and ways to cope with complications that do occur. *Knowing what to expect reduces the person's fear and helps them feel a greater sense of control.*

MEDICATION ADMINISTRATION The person who has genital warts

TOPICAL APPLICATIONS

Podophyllin
Trichloroacetic acid

Although cryotherapy using liquid nitrogen or a cryoprobe is commonly used to treat genital warts, podophyllin preparations are sometimes used. Podophyllin is applied topically to the warts once a week for 3–5 weeks.

Podophyllin is contraindicated during pregnancy; the alternative is cryotherapy. Podophyllin is also contraindicated in cervical, urethral, oral or anorectal warts. It is important to avoid contact of podophyllin resin with the eyes. Wear appropriate personal protective equipment (PPE) when applying podophyllin.

Adverse effects of podophyllin include local irritation, severe ulceration of surrounding tissue, nausea, diarrhoea, lethargy, paralysis and coma.

Nursing responsibilities

- Establish baseline data, including mental status, vital signs and weight.
- Document and report any existing lesions (genital, anal or oral).
- Cover the tissue surrounding the warts with a protective barrier cream or a paste of baking soda and water to protect the tissue from the caustic treatment solution.

Health education for the infected person and their significant others

- Wash off the treated area thoroughly within 1–4 hours after the first application; gradually increase this period to 6–8 hours after the second and subsequent applications.
- Return for regular treatment until warts are gone.
- Refer partners for examination and any necessary treatment.
- Report any adverse effects (nausea, diarrhoea, local irritation, lethargy, numbness).
- Avoid sexual activity until the person and their partners have been free of disease for 1 month.
- Use condoms to prevent future infections.
- Return for an annual Pap smear.

- Explain that the procedure is performed with a local anaesthesia. *Being awake during surgery gives the person a greater sense of partnership in the treatment process.*

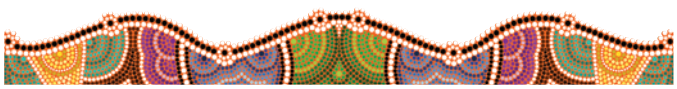
Anxiety

The woman with an HPV infection faces an increased risk of infection of her neonate during delivery. The neonatal infection can range from asymptomatic to widely disseminated fatal disease. Transmission occurs during passage through the birth canal. The risk is highest during the first episode of infection.

- Discuss with women of childbearing age that caesarean delivery can prevent transmission of infection to the neonate. In women without manifestations of recurrence, vaginal delivery is possible. *Understanding that infection of the neonate can be prevented helps relieve anxiety.*

Community-based care

Health education emphasises the need for the infected person and their infected sexual partners to return for regular treatment until lesions have resolved and to use condoms to prevent reinfection. Because of the increased risk of cervical cancer, annual Pap smears are essential for infected women and girls.



THE PERSON WITH A VAGINAL INFECTION

The vagina may be infected by yeasts, protozoa or bacteria. These infections can be sexually transmitted, but the male partner does not usually have manifestations of the infection. Risk factors include the use of hormonal contraceptives or broad-spectrum antibiotics, obesity, diabetes, pregnancy, unprotected sexual activity and multiple sexual partners, particularly if unprotected sex has been practised. Manifestations of vaginal infections are outlined in Table 49.2.

Preventive measures include education about the necessity of high standards of personal hygiene and safe sex practices. Women need to avoid frequent douching and wearing nylon underwear, lycra and/or tight jeans or trousers. Unprotected sexual activity, particularly with multiple partners, increases the risk of vaginal infections.

Pathophysiology and manifestations

Alterations in pH, changes in the normal flora and low oestrogen levels are conducive to the development of vaginal infections. When conditions are favourable, microorganisms invade the vulva and vagina.

Bacterial vaginosis

Bacterial vaginosis (non-specific vaginitis) is the most common cause of vaginal infection in women of reproductive age. *Gardnerella vaginalis* is one of the causative organisms, but others are also implicated. The relationship of sexual activity to this infection is not clear. The primary manifestation is a vaginal discharge that is thin and greyish-white, and has a foul, fishy odour. Complications include pelvic inflammatory disease, preterm labour, premature rupture of the membranes and postpartum endometritis. The infection is treated with oral or intravaginal antibacterial agents.

Candidiasis

Candidiasis (moniliasis or yeast infection) is caused by the organism *Candida albicans*, which has several strains of different virulence. Candida organisms are part of the normal vaginal environment in up to 50% of women, causing problems only when they multiply rapidly. When increased oestrogen levels, antibiotics, diabetes mellitus, faecal contamination or other factors alter the normal vaginal flora, the organism proliferates, resulting in a yeast infection. The manifestations include an odourless, thick, cheesy vaginal discharge

TABLE 49.2 Vaginal infections

INFECTION	TYPE OF DISCHARGE	TYPICAL MANIFESTATIONS	NURSING CARE
Candidiasis (*Monilia*, yeast)	Thick white patches adhering to cervix and vaginal wall, resembling cottage cheese; little odour	Itching of vulva and vaginal area, redness, painful intercourse	Teach perineal hygiene and proper use of vaginal applicators. Instruct the person to complete the entire treatment.
Simple vaginalis (bacterial vaginosis, *Gardnerella vaginosis*)	Thin, white, 'milk-like', or grey with fishy odour, especially when mixed with potassium hydroxide	None to mild itching or burning in vulvar area; clue cells on microscopic examination	Educate the person about proper perineal hygiene. Instruct the person to complete treatment. Inform the person about the relationship of infection to pelvic inflammatory disease.
Trichomoniasis	Frothy, yellow or white, foul odour	Burning and itching of vulva	Provide health education about perineal hygiene.
Atrophic vaginitis (senile vaginitis)	Thin, opaque discharge, occasionally blood tinged, odourless; pale, smooth, thin, dry vaginal walls	Painful intercourse, itching, vaginal dryness	Inform the person about symptoms of menopause and sexual techniques to minimise trauma.

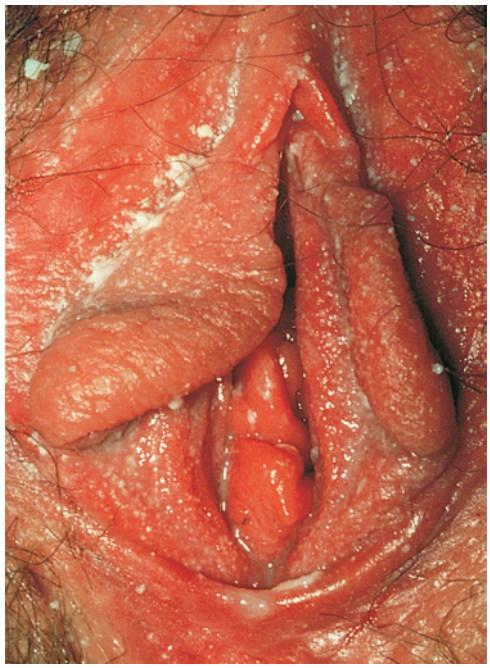


FIGURE 49.3 ■ Yeast infection on female genitalia

Source: Custom Medical Stock Photo, Inc.

(see Figure 49.3). This is often accompanied by itching and irritation of the vulva and vagina, dysuria and dyspareunia. Uncircumcised men may develop a yeast infection over the glans penis, manifested by itching and dysuria. The infection is treated with oral medications or, for women, intravaginal antifungal agents.

Trichomoniasis

Trichomoniasis is caused by *Trichomonas vaginalis*, a protozoan parasite. It is the most common STI worldwide, but is curable. It affects mostly women and is common in Australia. Women who have unprotected sex with multiple partners are at higher risk. Rates of trichomoniasis are increasing in some Aboriginal communities (Australian Sexual Health Alliance (ASHA), 2015). Symptoms usually appear within 5 to 28 days of exposure. It most commonly infects the vagina in women and the urethra in men. Most men are asymptomatic, but when symptomatic may complain of dysuria and urethral discomfort. Women have a frothy, green-yellow vaginal discharge with a strong fishy odour, often accompanied by itching and irritation of the genitalia. A woman with HIV who becomes infected has an increased risk of transmitting HIV to her sexual partner.

Trichomoniasis is treated with a single oral dose of metronidazole or tinidazole. These antibiotics are effective against anaerobic bacteria and certain parasites. Anaerobic bacteria are single-celled, living organisms that thrive in environments in which there is little oxygen (anaerobic environments). They selectively block some of the functions within the bacterial cells and the parasites, resulting in their death.

INTERPROFESSIONAL CARE

Interprofessional care focuses on identifying and eliminating the infection and preventing recurrence.

Diagnosis

Diagnostic tests vary with the suspected organism. Cervical cultures are used to diagnose the causative organism. *Trichomonas* is identified by microscopically examining a specimen of vaginal discharge in saline. Ten per cent potassium hydroxide is used to identify spores and filaments of candida. Diagnostic tests are described in Chapter 46.

Medications

Pharmacological treatments vary with the organism. Sexual partner/s of a woman who has a *Trichomonas* infection must also be treated to prevent reinfection. Some antifungal agents are available without prescription, which can sometimes lead to self-medication with the incorrect agent, or allow repeated infections to go unreported.

Nursing care

Engaging the infected woman in health education and, if necessary, her sexual partner/s to comply with the treatment regimen and using safer sex practices, can prevent future transmission of the infection. Careful history taking may also reveal high-risk sexual practices that require intervention, particularly if the woman has had repeated infections. The initial presenting symptom for many HIV-positive women is vaginal candidiasis which may not respond to over-the-counter treatments. Treatment with some antibiotics destroys normal vaginal flora, also resulting in superinfection with yeast.

Nursing interventions

Although each care plan must be individualised, priorities for care that often apply to women with vaginal infections are *Health education* and *Acute pain*.

Health education

Health education focuses on eradicating the infection, preventing further disease transmission and relieving discomfort associated with the condition. Educating the woman and her partner/s about safe sex and improved genital hygiene practices can reduce the incidence of recurrence.

Many women are unaware of the causes of vaginal infections and the self-care measures to prevent and treat these infections. If possible, both the woman and her sexual partner/s should be provided with information.

- Explain the transmission of the infection and check that you are understood. Many infections are transmitted most easily during menstruation; some can also be transmitted by towels or other inanimate objects, or by certain types of

BOX 49.2 Self-care comfort measures

- Do not wear pantyhose, nylon or lycra; wear loose-fitting trousers, shorts, skirts or dresses.
- Double-rinse underwear; do not use fabric softener.
- Do not use bubble bath, perfumed soaps or perfumed feminine hygiene products.
- Use 100% cotton menstrual pads and/or tampons.
- Use unscented toilet paper.
- Use a water-soluble lubricant for intercourse.
- Apply ice or a frozen gel pack wrapped in a towel to the vulva after intercourse to relieve burning. Ensure the towel is cleansed properly or disposed of.
- Rinse vulva with cool water after voiding and intercourse.

sexual activity. *An open discussion of disease transmission and prevention with the woman and her partner/s can reduce the risk of reinfection.*

- Explain the need to complete the entire course of treatment. *Many infections are asymptomatic in one partner. Incomplete treatment allows for recurrence of the infection and reinfection of the partner.*

Acute pain

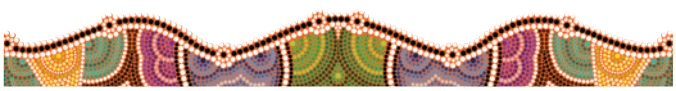
The symptoms of vaginitis can include dysuria, painful excoriation or ulceration of tissue, and painful intercourse (dyspareunia). Often these symptoms can be relieved by relatively simple self-care measures. See Box 49.2 for additional comfort measures.

- Suggest the use of cool compresses. *Cool compresses relieve itching.*
- Recommend warm, not hot, sitz baths to alleviate discomfort. *Sitz baths cleanse the perineal area and the warmth is soothing to inflamed, irritated skin and membranes.*
- Wear cotton underwear. *Cotton absorbs moisture and allows better air circulation than other types of material.*
- If infected with *Trichomonas*, avoid sexual contact until treatment is completed. *Treatment of the infected woman and her partner/s, as well as sexual abstinence, are necessary to facilitate healing and to prevent reinfection.*

Community-based care

Health education

Health education focuses on eradicating the infection, preventing further disease transmission and relieving discomfort associated with the condition. Educating the woman and her partner/s about safe sex and improved genital hygiene practices can reduce the incidence of recurrence.



THE PERSON WITH CHLAMYDIA

Chlamydia is a group of STIs caused by *Chlamydia trachomatis*, a bacterium that behaves like a virus, reproducing only within the host cell. The bacterium is spread by any sexual contact and to the neonate by passage through the birth canal of an infected mother. The infections caused by chlamydia include acute urethral syndrome, non-gonococcal urethritis, mucopurulent cervicitis and pelvic inflammatory disease.

Chlamydia is the most commonly reported bacterial STI in Australia, with 355 cases per every 100 000 people each year (Department of Health, 2014c). Of that number, 81% of reported cases occur in people aged between 15 and 24. Risk factors for chlamydia are listed in Box 49.3.

Because chlamydia is asymptomatic in most women until the uterus and fallopian tubes have been invaded, treatment may be delayed, resulting in devastating long-term complications. Nearly one-third of men with urethral chlamydia are also asymptomatic. Chlamydia is a leading cause of preventable blindness in the newborn.

Pathophysiology

C. trachomatis is an intracellular bacterial pathogen that resembles both a virus and a bacteria. The organism enters the body as an elementary body, a form in which it is capable of entering uninfected cells. The infection begins when the organism enters a cell and changes into a reticulate body. The reticulate body divides within the cell, bursting the cell and infecting adjoining cells.

Manifestations

The incubation period is from 1 to 3 weeks. Chlamydia may be present for months or years without producing noticeable symptoms in women. Chlamydia typically invades the same target organs as gonorrhoea (cervix and male urethra) and results in similar manifestations (dysuria, urinary frequency and discharge). People with the infection may be asymptomatic. However, they are still potentially infectious.

Complications

If a chlamydial infection in women is not treated, it ascends into the upper reproductive tract, causing such complications as pelvic inflammatory disease, which includes endometritis and salpingitis. Chronic pelvic pain may result. These infections are

BOX 49.3 Higher risk factors for chlamydial infection

- Young sexually active poeple
- Previous history of STI
- Homosexually active men and men who have sex with men (MSM)
- Indigenous Australian people
- Oral contraceptive use
- Unprotected sexual activity
- Multiple sexual partners

a major cause of infertility and ectopic pregnancy, which are potentially life threatening. Complications of chlamydial infections in men include pelvic inflammatory disease, epididymitis, prostatitis, sterility and Reiter's syndrome. Reiter's syndrome is a form of reactive arthritis; it is relatively uncommon but can be a debilitating syndrome that follows a gastrointestinal or genitourinary infection. Routine screening for sexually active adolescents and young adults has been suggested by the CDC to minimise these serious complications in asymptomatic people.

INTERPROFESSIONAL CARE

C. trachomatis is treated with medications to eradicate the infection. Its prevalence, particularly in younger populations, makes widespread screening necessary if the disease is to be controlled. Because chlamydia is often asymptomatic, treatment is often begun on a presumptive basis.

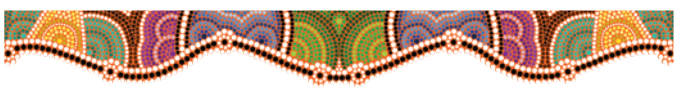
Diagnosis

The diagnostic tests that may be ordered include Gram stain of discharge from the female endocervix and urethra or from the male urethra to look for polymorphonuclear leucocytes (considered evidence of infection).

Tests for antibodies to chlamydia, such as the direct fluorescent antibody (DFA) test and an enzyme-linked immunosorbent assay (ELISA), as well as polymerase chain reaction (PCR) or ligase chain reaction (LCR) tests, are highly sensitive and specific tests performed on cervical and urethral swab specimens. Nucleic acid amplification tests (NAATs), also performed on cervical and urethral swab specimens, are currently the diagnostic method of choice.

Medications

The antibiotic recommended by the CDC for chlamydial infections in men and non-pregnant women is azithromycin, orally in a single dose, or doxycycline, orally for 7 days. All sexual partners must be treated simultaneously or prior to resuming sexual intercourse.

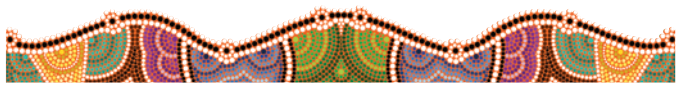


Nursing care

Nursing care of the person who has chlamydia focuses on eradication of the infection, prevention of future infections and management of any chronic complications. Nursing priorities and care for the person who has chlamydia are the same as for anyone with any STI. Interventions are similar to those discussed later in the chapter for gonorrhoea and previously for genital herpes.

Community-based care

Health education for the person who has chlamydia centres on the need to comply with the treatment regimen, to refer partners for examination and necessary treatment, and the use of condoms to avoid reinfection. If the infection has progressed to pelvic inflammatory disease (discussed later), the person will require additional information on self-care and health promotion. The CDC recommends annual screening for chlamydia for people who are young, sexually active and do not use condoms correctly with every act of sexual intercourse.



THE PERSON WITH GONORRHOEA

Gonorrhoea, also known as 'GC' or 'the clap', is caused by *Neisseria gonorrhoeae*, a Gram-negative diplococcus. In Australia, gonorrhoea is the second most commonly reported STI. Global estimated incidence of gonorrhoea is 62 million infected people annually (WHO, 2013).

In Australia in 2010, more than one-third (36%) of all gonorrhoea diagnoses were in Aboriginal and Torres Strait Islander people. The rate of diagnosis was more than 26 times that for the non-Indigenous population (University of New South Wales (UNSW), 2013). Other risk factors include residing in large urban areas, being transient, early onset of sexual activity, multiple serial or consecutive sexual partners, illicit drug use, alcohol misuse, prostitution and previous gonorrhoeal or concurrent STIs.

Pathophysiology

The causative organism of gonorrhoea is a pyogenic (pus-forming) bacterium that causes inflammation characterised by purulent exudate. Humans are the only host for the organism. Gonorrhoea is transmitted by direct sexual intercourse and during delivery as the neonate passes through the birth canal. The portal of entry can be the genitourinary tract, eyes, oropharynx, anorectum or skin. The incubation period is 2 to 7 days after exposure. The organism initially targets the female cervix and the male urethra. Without treatment, the disease ultimately spreads widely to other organs. In men, gonorrhoea can cause acute, painful inflammation of the prostate, epididymis and periurethral glands, and can lead to sterility. In women, it can cause pelvic inflammatory disease, endometritis, salpingitis and pelvic peritonitis.

Manifestations

Manifestations of gonorrhoea in men include dysuria and serous, milky or purulent discharge from the penis. Some men also experience regional lymphadenopathy. About 20% of men and 80% of women remain asymptomatic until the disease is advanced. Women with symptoms experience dysuria, urinary frequency, abnormal menses (increased flow or dysmenorrhoea), increased vaginal discharge and dyspareunia (difficult or painful sexual intercourse).

Anorectal gonorrhoea is seen most often in people who practise anal sex. The manifestations include pruritus,

mucopurulent rectal discharge, rectal bleeding and pain, and constipation. Gonococcal pharyngitis occurs primarily in people after oral sex (fellatio) with an infected partner. The manifestations include fever, sore throat and enlarged lymph glands.

Complications

The complications of untreated gonorrhoea in both men and women may be permanent and serious. They include:

- pelvic inflammatory disease in women, leading to internal abscesses, chronic pain, ectopic pregnancy and infertility
- blindness, infection of joints and potentially lethal infections of the blood in the newborn, contracted during delivery
- epididymitis and prostatitis in men, resulting in infertility and dysuria
- spread of the infection to the blood and joints
- increased susceptibility to and transmission of HIV.

INTERPROFESSIONAL CARE

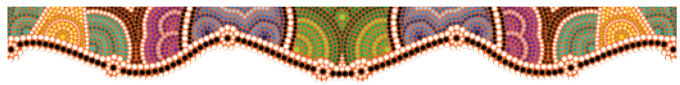
The goals of treatment for the person who has gonorrhoea include eradication of the organism and any coexisting disease, and prevention of reinfection or transmission. It is important to emphasise the importance of taking all medications as prescribed and abstaining from sexual contact until the infection is cured in both the person and their partner/s. Condom use to prevent future infections is essential, particularly for pregnant women whose partner/s may be infected.

Diagnosis

Diagnosis of gonorrhoea is based on cultures from the infected mucous membranes (cervix, urethra, rectum or throat), examination of urine from an infected person and a Gram stain to visualise the bacteria under the microscope. Testing for other STIs (especially chlamydia and syphilis) at the same time is recommended. Pregnant women are routinely screened during their first prenatal visit. Diagnostic tests are described in Chapter 46.

Medications

Because of the many penicillin-resistant strains of *N. gonorrhoeae*, alternative antibiotics, such as ciprofloxacin (Cipro) or ofloxacin (Floxin) are used to treat gonorrhoea. Fluoroquinolone therapy (such as with ciprofloxacin or levofloxacin) is often prescribed because it is inexpensive, oral and single dose. Because of increased prevalence of fluoroquinolone-resistant *N. gonorrhoeae* in Asia, the Pacific Islands and California in the US, this therapy is no longer recommended for use in treating gonorrhoea in those areas. A single dose of oral azithromycin (Zithromax) or a 7-day course of oral doxycycline (Vibramycin, Vivox) is usually added to treat any coexisting chlamydial infection. All sexual partners also need to be treated within 60 days of diagnosis of the infection.



Nursing care

In planning and implementing care for the person who has gonorrhoea, the nurse considers the possible coexistence of other STIs such as syphilis and HIV, the impact of the disease and its treatment on the person's lifestyle, and the likelihood of noncompliance. A nursing care plan for the person who has gonorrhoea is included below.

Nursing interventions

Nursing priorities discussed in this section focus on noncompliance with treatment and social isolation.

Noncompliance

Although one-time treatment with the recommended antibiotic is highly effective in curing gonorrhoea, noncompliance with the doxycycline regimen may leave any coexisting chlamydial infection unresolved. Noncompliance with recommendations for abstinence, follow up or condom use fosters a high rate of reinfection. Failure to refer partners for examination and treatment also leads to reinfection.

- Reinforce the need to take all medications as directed and to keep follow-up appointments to be sure no reinfection has occurred. Discuss the prevalence of gonorrhoea and the potential complications if it is not cured. *The person who understands the complications of incomplete or failed treatment is more likely to comply with the medication regimen.*
- Discuss the importance of sexual abstinence until the infection is cured, referral of partners and condom use to prevent reinfection. *Understanding that cure is possible and reinfection is avoidable helps the person cope with the disease and its treatment, and is likely to increase compliance.*
- Explain to women that condoms must be used during treatment, even if other methods of birth control are used. *Oral contraceptives increase the alkalinity of the vaginal pH, facilitating the growth of the gonococcal bacteria, and intrauterine devices alter the endometrial barrier, favouring persistent gonococcal infections.*

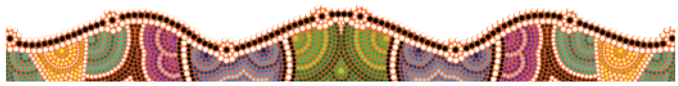
Social isolation

Diagnosis of any STI can make people feel 'dirty', ashamed and guilty about their sexual behaviours, and unworthy of being with others.

- Provide privacy, confidentiality and a safe, non-judgmental environment for expression of concerns. Support the person to gain an understanding that gonorrhoea is a consequence of sexual behaviour, not a punishment, and that it can be avoided in the future. *Being treated with respect and privacy helps the person realise that the disease does not change an individual's worth as a person. This knowledge enhances the person's ability to relate to others.*

Community-based care

Health education focuses on supporting people to understand the importance of (1) taking any and all prescribed medication, (2) referring sexual partners for evaluation and treatment, (3) abstaining from all sexual contact until the person and their partners are cured, and (4) using a condom to avoid transmitting or contracting infections in the future. Individuals also need to understand the need for a follow-up visit 4 to 7 days after treatment is completed.



NURSING CARE PLAN The person with gonorrhoea

Joaddie Deall is a single 26-year-old Assistant in Nursing who lives in a high-density metropolitan apartment. Ms Deall is currently dating Angus Penting, who lives nearby. Ms Deall visits her gynaecologist because her periods have become irregular and she is experiencing pelvic pain. She also complains of an abnormal amount of vaginal discharge. Recently she has developed a sore throat. The pelvic pain has begun to disrupt her sleeping pattern and she is concerned that she might have cancer because her mother recently died of ovarian cancer.

ASSESSMENT

When Ms Deall arrives for her appointment at the gynaecologist's office, Bradley Terr, the Nurse Practitioner (NP), respectfully and non-judgmentally interviews her. Mr Terr undertakes a thorough medical and sexual history, including gathering information about Ms Deall's menstrual periods, pain associated with urination or sexual intercourse, urinary frequency, most recent Pap smear, birth control method, history of STI and drug use, and types of sexual activity. Ms Deall tells the NP her symptoms and her concerns about possible ovarian cancer. She also indicates that she is taking oral contraceptives and therefore sees no need for Angus to use a condom. She believes their relationship to be monogamous.

Physical examination reveals both pharyngeal and cervical inflammation and lower abdominal tenderness. Her temperature is 37.2°C. There are no signs or symptoms of pregnancy.

A Pap smear is ordered and cultures of the cervix, urethra and pharynx to evaluate for gonorrhoea and chlamydial infection. Mr Terr, the NP, takes blood for a white cell count. Test results are positive for gonorrhoea and negative for chlamydia. The white cell count is slightly elevated, indicating possible salpingitis. Because Angus has been Ms Deall's only sexual partner, it is clear that he is the source of infection and also requires treatment.

DIAGNOSES

- *Acute pain* related to the infectious process.
- *Anxiety* related to fear about possible cancer.
- *Low self-esteem* related to shame and guilt because of having an STI.
- *Altered sexuality patterns* related to the impaired relationship and fear of reinfection.

PLANNING

- Establish a therapeutic relationship that is respectful and non-judgmental.
- Discuss with Ms Deall which tests and investigations will be ordered and why.
- In collaboration with Ms Deall, set goals and priorities for care and treatment.

Expected outcomes

- A therapeutic relationship is established and Ms Deall feels as though she was treated with respect and dignity.
- Ms Deall receives education regarding tests and investigations and understands why they have been ordered.
- A care plan is jointly established and agreed upon.
- Ms Deall experiences relief of pain, indicating that the infection has been eradicated.
- Ms Deall understands that she has nothing to be ashamed of and that she was wise to seek treatment as soon as her symptoms occurred.
- Ms Deall understands her diagnosis and the implications of the illness.
- Ms Deall understands the need for her and her partner to use condoms during future sexual activity.

IMPLEMENTATION

- Administer ceftriaxone IM.
- Emphasise the need for regular Pap smears and pelvic examinations because of the family history of ovarian cancer.
- Discuss feelings and concerns about the diagnosis of gonorrhoea. Stress that such a diagnosis is not a reflection on her worth as a person.
- Educate how to talk with sexual partner/s about condom use.

EVALUATION

A week later, during her follow-up visit, Ms Deall states that she is feeling much better and sleeping well at night since the pain has ended. She has terminated her relationship with Mr Penting and is considering joining a health club in the hope of increasing her level of fitness and perhaps meeting someone new.

CRITICAL THINKING IN THE NURSING PROCESS

1. In what ways are Ms Deall's manifestations related to the infectious process of gonorrhoea?
2. Should the nurse have suggested that Ms Deall also be tested for HIV? Why or why not?
3. Develop a care plan for Ms Deall related to *Social isolation*.

REFLECTION ON THE NURSING PROCESS

1. Why is it so important that a therapeutic relationship be established and that the nurse is non-judgmental and respectful?
2. Which aspects of health education should the nurse focus on?

THE PERSON WITH SYPHILIS

Syphilis is a complex systemic STI caused by the spirochaete *Treponema pallidum*. It can infect almost any body tissue or organ and is transmitted from open lesions during any sexual contact (genital, oral–genital, oral–anal, or anal–genital). The organism is highly susceptible to heat and drying, but can survive for days in fluids; thus, it may also be transmitted by infected blood or other body fluid such as saliva. The incubation period ranges from 10 to 90 days, averaging 21 days. If not treated appropriately, syphilis can lead to blindness, paralysis, cardiovascular damage and death. Syphilis often occurs with one or more other STIs, such as HIV/AIDS or chlamydial infection.

Famous people suspected of having syphilis include Charles VIII of France, Adolph Hitler, Mussolini and Leo Tolstoy. Those suspected of dying from the disease include Christopher Columbus, George Washington, Napoleon Bonaparte and Franz Schubert. It is posited that 'the pox', as syphilis is sometimes called, was spread worldwide by Christopher Columbus and his men.

Although the rate of syphilis infection has reduced, it remains a significant problem in certain geographical regions and specific populations. Rates remain high in many urban centres, with higher infection rates found in illicit drug users, people who are transient and people who are homeless. The incidence of primary and secondary syphilis is highest in people 20 to 39 years of age, with the incidence in women decreasing. However, the rate of syphilis in men having sex with men (MSM) is increasing (CDC, 2015).

In 2012, syphilis notifications were approximately four times higher in Aboriginal and Torres Strait Islander people than in the non-Indigenous population. The number of people diagnosed with syphilis was 27 per 100 000 Aboriginal and Torres Strait Islander people, while the number of people diagnosed in the non-Indigenous population was 7 per 100 000 (UNSW, 2013).

Pathophysiology

Any break in the skin or mucous membrane is vulnerable to invasion by the spirochaete. Once it has entered the system, the spirochaete is spread through the blood and lymphatic system. Congenital syphilis is transferred to the foetus through the placental circulation.

Manifestations

Syphilis is generally characterised by three clinical stages: primary, secondary and tertiary. Each stage has characteristic manifestations (see Table 49.3). The person who has syphilis also may experience a latency period when no signs of the disease are evident.

TABLE 49.3 Manifestations of syphilis

SYSTEM	PRIMARY	SECONDARY	TERTIARY
Reproductive	Genital chancre (may be internal in female)	Condyloma lata	
Integumentary		Rash on palms of hands and soles of feet	Granulomatous lesions involving mucous membranes and skin
Gastrointestinal	Loss of appetite Oral mucous patches		
Neurological		Asymptomatic Meningitis Headache Cranial neuropathies	Asymptomatic Tabes dorsalis Neurosyphilis Seizures, hemiparesis, hemiplegia Personality changes, hyperactive reflexes, Argyll Robertson pupil, decreased memory, slurred speech, optic atrophy
Musculoskeletal		Arthralgia Myalgia Bone and joint arthritis Periostitis	Gummas
Cardiovascular			Aortic insufficiency Aortic aneurysm Stenosis of openings to coronary arteries
Renal		Glomerulonephritis Nephrotic syndrome	
Other	Regional lymphadenopathy	Generalised lymphadenopathy Fever Hepatitis Malaise Alopecia	

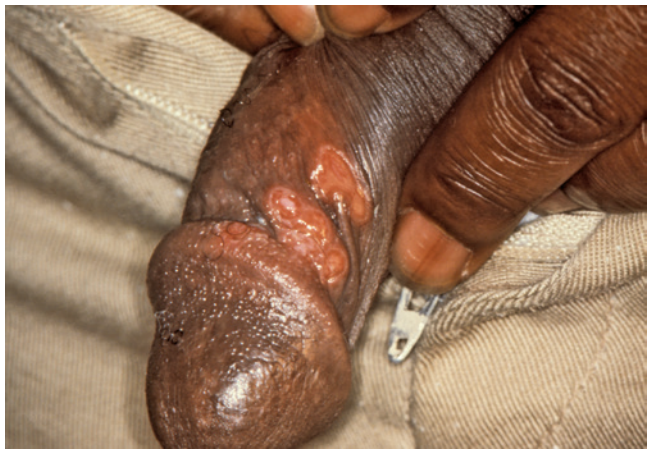


FIGURE 49.4 ■ Chancre of primary syphilis on the penis

Source: CDC/M. Rein.

Primary syphilis

The primary stage of syphilis is characterised by the appearance of a **chancre** (see Figure 49.4) and by regional enlargement of lymph nodes; little or no pain accompanies these warning signs. The chancre appears at the site of inoculation (such as the genitals, anus, mouth, breast, fingers) 3 to 4 weeks after the infectious contact. In women, a genital chancre may go unnoticed, disappearing within 4 to 6 weeks. In both primary and secondary stages, syphilis remains highly infectious, even if no symptoms are evident.

Secondary syphilis

Manifestations of secondary syphilis may appear any time from 2 weeks to 6 months after the initial chancre disappears. Symptoms can include a skin rash, especially on the palms of the hands or soles of the feet; mucous patches in the oral cavity; sore throat; generalised lymphadenopathy; condyloma lata (flat, broad-based papules, unlike the pedunculated structure of genital warts) on the labia, anus or corner of the mouth; flu-like symptoms; and alopecia. These manifestations generally disappear within 2 to 6 weeks and an asymptomatic latency period begins.

Latent and tertiary syphilis

The latent stage of syphilis begins 2 or more years after the initial infection and can last up to 50 years. During this stage, no symptoms of syphilis are apparent and the disease is not transmissible by sexual contact. It can be transmitted by infected blood. Therefore, all prospective blood donors are screened for syphilis. In two-thirds of all cases, the latent stage persists without further complications. Unless treated, the remaining one-third of infected people progress to late-stage or tertiary syphilis. In the presence of HIV infection, disease progression seems to be more rapid.

Two types of late-stage syphilis occur. Benign late syphilis, of rapid onset, is characterised by localised development of infiltrating tumours (*gummas*) in skin, bones and liver, generally responding promptly to treatment. A more insidious onset involves a diffuse inflammatory response that involves the central nervous and cardiovascular system. Though the disease can still be treated at this stage, much of the cardiovascular and central nervous system damage is irreversible.

INTERPROFESSIONAL CARE

The goals of treatment are to inactivate the spirochaete and provide health education about how to prevent reinfection or further transmission. Treatment includes antibiotic therapy and identification and referral of partners for testing and treatment if necessary, follow-up testing and education about condom use to prevent reinfection of self and transmission of disease to partners. In addition, people with syphilis should also be screened for chlamydial infection and advised to have an HIV test.

Diagnosis

Diagnosis of syphilis is complex because it mimics many other diseases. A careful history and physical examination are obtained, as well as laboratory evaluations of lesions and blood. Diagnostic tests are described in Chapter 46.

People with syphilis become positive about 4 to 6 weeks after infection. However, these tests are not specific for syphilis and other diseases may also cause positive results. Additional tests are required for a definitive diagnosis.

The FTA-ABS (fluorescent treponemal antibody absorption) test is specific for *T. pallidum* and can be used to confirm findings. It may be used for people whose clinical picture indicates syphilis but who have negative venereal disease results. In immunofluorescent staining, a specimen is obtained from early lesions or aspiration of lymph nodes and is specially treated and examined microscopically for the presence of *T. pallidum*. Dark-field microscopy involves examining a specimen from the chancre for the presence of *T. pallidum* using a dark-field microscope.

Medications

The treatment of choice for all stages of syphilis is penicillin G, given intramuscularly (IM) in a single dose. People who are allergic to penicillin are given oral doxycycline or tetracycline for 28 days.

Treatment of syphilis may result in a severe reaction called the *Jarisch–Herxheimer reaction*, involving fever, musculoskeletal pain, tachycardia and sometimes hypotension. This is not a reaction to the penicillin itself, but to the sudden and massive destruction of spirochaetes by the penicillin and the resulting release of toxins into the bloodstream. The Jarisch–Herxheimer reaction generally begins within 24 hours of treatment and subsides in another 24 hours. Treatment should not be discontinued unless symptoms become life threatening.

Nursing care

In planning and implementing nursing care for the person who has syphilis, the nurse needs to consider the person's age, their lifestyle, access to healthcare and their educational

level. A nursing care plan for a person who has syphilis is included below.

Nursing diagnoses and interventions

Nursing priorities discussed in this section focus on the *Risk of injury*, *Anxiety* and *Low self-esteem*.

Risk of injury

If syphilis is not diagnosed and treated promptly and effectively, it can have devastating effects on all body systems, particularly the neurological and cardiovascular systems, eventually leading to a painful death.

- Educate the person about the importance of taking any prescribed medication. *Taking the prescribed antibiotic is important to ensure eradication of the infecting organism.*
- Encourage referral of any sexual partners for evaluation and any necessary treatment. *Without treatment of all partners, reinfection can occur or the disease may be transmitted to other people through sexual activity.*
- Educate about why the person needs to abstain from sexual contact until they and their partner/s are cured, and why they should use condoms to prevent future infections. *Abstinence until the organism is eradicated prevents reinfection. Condoms provide barrier protection, reducing the risk of infection during sexual activity.*
- Emphasise the importance of returning for follow-up testing at 3- and 6-month intervals for early syphilis and at 6- and 12-month intervals for late latent syphilis. *Follow-up testing is performed to ensure eradication of the disease.*
- Provide information about manifestations of reinfection. *Successful treatment of the disease does not prevent possible subsequent infections.*

Anxiety

The diagnosis of syphilis understandably causes the person significant levels of anxiety, not only about personal wellbeing but also about the wellbeing of partners and, in the expectant woman, her foetus.

- Emphasise that syphilis can be effectively treated, preventing the serious complications of late-stage disease. *This information provides a sense of control and can help decrease anxiety.*
- Educate the pregnant woman about taking medications as directed and that returning each month for follow-up testing will help ensure the wellbeing of her baby. *Knowing that treatment can reduce the risk to her baby relieves anxiety and possibly increases compliance.*

Low self-esteem

Living with any chronic disease can be damaging to someone's self-esteem. However, the person who has syphilis needs additional support to cope with the stigma of this kind of disease. Unfortunately, people who are most affected by STIs often lack family and other social support networks.

- Create an environment where the person feels respected and safe to discuss questions and concerns about the disease and its effect on their life. *Being treated with respect helps enhance self-esteem.*
- Provide privacy and confidentiality. *People are often embarrassed to discuss the intimate details of their sex lives.*
- Let the person know that the nurse and other healthcare providers care about them and the successful treatment of their disease. *Feeling valued enhances self-esteem.*

NURSING CARE PLAN A person who has syphilis

Nate Elgmisson, aged 24, works as a butcher at a local supermarket. For the past year he has shared a house with Callie Lincoln, who is 5 months pregnant with his child. Although he intends to marry Callie before the baby is born, he has continued to have a sexual relationship with a previous partner named Joshua Sorronsoin. His sexual activities with Joshua have increased in frequency as Ms Lincoln's pregnancy has advanced. Recently, Mr Elgmisson has noticed a swelling in his groin and a sore on his penis.

ASSESSMENT

When Mr Elgmisson comes to the community health centre, he is interviewed by the Nurse Practitioner (NP), Mary Pertolloti. Mary establishes a therapeutic relationship and gathers a thorough medical and sexual history, including gaining information about drug use, allergies, difficulty with urination, urinary frequency, itching or discharge from the penis, recent sexual activities, precautions taken against infection, history of STIs and sexual function. Mary determines that Mr Elgmisson has been having unprotected sex with both Ms Lincoln and Mr Sorronsoin. Nate thinks that Joshua is not having sex with anyone except him but he is not sure.

Physical assessment reveals a classic syphilitic chancre on the shaft of the penis and regional lymphadenopathy. A specimen of exudates from the chancre is sent for dark-field examination. Ms Pertolotti discusses with Mr Elgmisson the likelihood that he has syphilis and the need to tell both Ms Lincoln and Mr Sorronsoin so that they can be tested and, if necessary, treated. Ms Pertolotti also suggests that Mr Elgmisson be tested for HIV since he has been having unprotected sex with two people, at least one of whom may be sexually active with other partners. He agrees and blood is drawn for an ELISA test. Dark-field analysis of the chancre exudate confirms the diagnosis of syphilis; the ELISA results are negative for HIV.

DIAGNOSES

- *Risk of injury* to the person, his partners and the infant, related to the disease process.
- *Lack of knowledge* about the disease process, its transmission and the need for treatment.
- *Anxiety* related to the effects of the infection on the unborn child and discussing the diagnosis with partners.
- *Possible relationship breakdown* related to diagnosis of syphilis and non-monogamous sexual activity.

NURSING CARE PLAN A person who has syphilis (continued)

PLANNING

- Establish a therapeutic relationship based on respect and a non-judgmental attitude.
- Explain the diagnosis and what having this illness means for the person.
- Establish a collaborative care and treatment plan.
- Explain tests and interventions (e.g. the ELISA test).
- Collaboratively identify coping strategies.

Expected outcomes

- Therapeutic relationship and rapport established.
- Prompt treatment will cure the syphilis.
- The person understands the need to abstain from sexual contact during treatment, complete all medications, return for follow-up visits and use condoms to prevent reinfection.
- The person can identify strategies to cope with the effect of diagnosis and treatment on their relationship/s.
- Decreased anxiety following health and psychosocial education and treatment.
- Penicillin administered.
- Follow-up appointments made.
- Health education material provided and any questions and concerns answered and clarified.
- Sexual partners notified that they need to attend the clinic for testing.

IMPLEMENTATION

- Administer IM injection of penicillin G.
- Discuss the importance of abstaining from sexual activity until Mr Elgmission and his partners are cured, and of using condoms to prevent reinfection.
- Explain the need to return for follow-up testing in 3 months and again at 6 months. Provide a copy of the STI prevention checklist, and document that reminders need to be sent at 3- and 6-month intervals.
- Notify sexual partners that they need to be tested.
- Provide counselling about the effect of the disease on relationships.
- Provide health education regarding the importance of treatment to the health of the infant.

EVALUATION

At the 3-month follow-up visit, the chancre on Mr Elgmisson's penis has healed and he reports that he is using a condom whenever he has sex. Ms Lincoln also tested positive for syphilis and negative for HIV, so she, too, is given penicillin G and verbal and written follow-up instructions, including follow up until the infant is born. The couple is meeting every second week with a relationship counsellor and say that, although strained, the relationship is improving. Mr Sorronsoin has received similar test results and is given a prescription for doxycycline because he is allergic to penicillin.

CRITICAL THINKING IN THE NURSING PROCESS

1. Which manifestations might a person with early syphilis experience?
2. List some appropriate questions for taking a sexual history when you suspect the presence of one or more STIs.
3. How might you support Mr Elgmisson to help him break the news of his diagnosis to his sexual partners, especially Ms Lincoln?

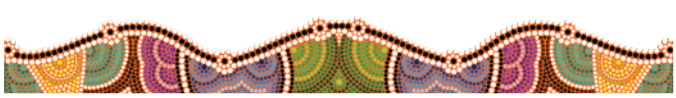
REFLECTION ON THE NURSING PROCESS

1. Which relationship issues will the couple have to deal with?
2. Would your thoughts about Mr Elgmisson's behaviour affect the way you interact with him?

Community-based care

Health and psychosocial education is an essential part of nursing care for someone who has an STI, and syphilis is no exception. The nurse emphasises that syphilis is a chronic disease that can be spread to others even though no symptoms are evident. The following topics need to be discussed:

- taking all prescribed medication
- referring sexual partners for evaluation and treatment
- abstaining from all sexual contact as recommended by CDC guidelines
- using a condom to avoid transmitting or contracting infections in the future
- the need for follow-up testing (at 3 and 6 months for people with primary or secondary syphilis, and at 6 and 12 months for those with late-stage disease). If the person is also HIV positive, follow-up visits are recommended 1, 2, 3, 6, 9 and 12 months after treatment.



THE PERSON WITH PELVIC INFLAMMATORY DISEASE

Pelvic inflammatory disease (PID) is a term used to describe infection of the pelvic organs, including the fallopian tubes (*salpingitis*), ovaries (*oophoritis*), cervix (*cervicitis*), endometrium (*endometritis*), pelvic peritoneum and the pelvic vascular system. PID can be caused by one or more infectious agents, including *N. gonorrhoeae*, *C. trachomatis*, *Escherichia coli* and *Mycoplasma hominis*. *N. gonorrhoeae* and *C. trachomatis* are responsible for as much as 80% of PID; dual infection with both agents is common.

Pelvic inflammatory disease is not a reportable disease in Australia. It is estimated that 1 million women experience PID each year. As a result of the infection, more than 100 000 women become infertile, and a large proportion of the ectopic pregnancies occurring each year are the result of PID (CDC, 2015). The disease may also cause pelvic abscesses and chronic abdominal pain.

Sexually active women aged 16 to 24 years are most at risk. Risk factors include a history of sexually transmitted infection (especially gonorrhoea and chlamydia), bacterial vaginosis, multiple sexual partners, douching and previous PID. Barrier contraceptive devices such as condoms reduce the risk of PID.

Prognosis depends on the number of episodes, promptness of treatment and modification of risk-taking behaviours. Prevention includes health education, especially for young women, regarding the causes and transmission of infection and methods of self-protection, such as avoiding unprotected sexual activity.

Pathophysiology

Pelvic inflammatory disease is usually polymicrobial (caused by more than one microbe) in origin, with *N. gonorrhoeae* and *C. trachomatis* being common causative organisms. Pathogenic microorganisms enter the vagina and travel to the uterus during intercourse or other sexual activity. They can also gain direct access to the uterus during childbirth, insertion of intrauterine devices (IUD), termination of pregnancy or surgery of the reproductive tract. The organisms ascend from the endocervical canal to the fallopian tubes and ovaries.

Manifestations

Manifestations of pelvic inflammatory disease include fever, purulent vaginal discharge, severe lower abdominal pain and painful cervical movement. However, the manifestations may be so mild that the infection is not recognised.

Complications

Complications include pelvic abscess, infertility, ectopic pregnancy, chronic pelvic pain, pelvic adhesions, dyspareunia and chronic pelvic pain. Abscess formation is common.

INTERPROFESSIONAL CARE

The goals of treatment are to eliminate the infection and prevent complications and recurrence. The physical examination may reveal abdominal, adnexal and cervical pain.

Diagnosis

Tests used in the diagnosis of PID may include a full blood count with differential, which will show a markedly elevated white blood cell count and an increased sedimentation rate. If a laparoscopy or laparotomy is done, it may reveal inflammation, oedema or hyperaemia of the fallopian tubes or tubal discharge and, possibly, generalised pelvic involvement, abscesses and scarring.

Medications

Combination antibiotic therapy with at least two broad-spectrum antibiotics, administered IV or orally, is the typical treatment for PID. If PID is not acute, outpatient antibiotic therapy is prescribed. In acute cases, the person may be hospitalised. Analgesics are given, and antibiotics and fluids are administered intravenously. Commonly prescribed antibiotics include parental ceftriaxone, cefoxitin or clindamycin, plus gentamicin or doxycycline. Nursing implications for antibiotics are discussed in Chapter 11.

Surgery

A drain may be inserted into an abscess, if present, and any adhesions may be removed. If the person does not respond to conservative therapy, surgical removal of the uterus, fallopian tubes and ovaries may be necessary.

Nursing care

The goals of nursing care are to treat the infection and to prevent complications, such as scarring and infertility. The person who is hospitalised maintains bed rest in the semi-Fowler's position to promote drainage and to localise the infectious process in the pelvic cavity.

Nursing interventions

Nursing priorities that apply to the person with PID include: *Risk of injury* and *Health education*.

Risk of injury

Pelvic inflammatory disease can have severe, even life-threatening, complications. Scarring of fallopian tubes can lead to ectopic pregnancy or pelvic abscess. Infertility is a common complication, as are recurrent or chronic PID, chronic abdominal pain, pelvic adhesions, premature hysterectomy and depression. The woman who has severe infection and manifestations may be hospitalised for treatment.

- Administer antibiotic therapy and monitor closely for adverse effects. *Antibiotics used in acute PID are potent agents; some can have serious side effects.*
- Practise thorough handwashing and strict adherence to universal precautions when handling perineal pads and linen. Appropriate disinfection of bedpans, toilet seats, linen and utensils is also important. *These practices help avoid transmitting the infection to others.*

Health education

Pelvic inflammatory disease is most common in young women, who often do not understand their own anatomy and physiology or sexually transmitted infections. Diagnosis and treatment of PID provides an opportunity to increase that understanding, thereby preventing complications and recurrent infection.

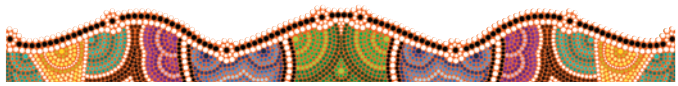
- Explain how infection is spread and which measures to take to prevent future infection. *Understanding can improve compliance with treatment regimens and perhaps change high-risk behaviour.*
- Explain the need to complete the treatment regimen and the importance of follow-up visits. If the person or their partner/s fail to take all of the medication as prescribed, the infection may not be completely cured. *Noncompliance and recurrence are common, particularly if follow-up appointments are not kept.*
- Provide education about proper perineal care, especially wiping from front to back. *This reduces transmission of faecal organisms to reproductive tissues and reduces the incidence of urinary tract infections.*
- Caution the person about using tampons. Educate the person about why it is important to change tampons or pads at least every 4 hours. *Menstrual flow and other discharges provide a favourable environment for microorganisms to multiply.*
- Provide information about safe sex practices and family planning. Support the person to understand that they need

to remove diaphragms within 6 hours after use. IUDs are contraindicated. Latex condoms offer the most effective protection against infection. *These measures help prevent recurrence of infection.*

- Educate the person to recognise and report any unusual vaginal discharge or odour to the healthcare provider. *Treatment is most effective early in the disease process.*

Community-based care

Provide general information related to STIs. Discuss ways to eradicate the infection and prevent recurrence, and support the person to identify strategies to deal with the physical and psychosocial implications of treatment, including possible infertility. Inform the person that the patency of the fallopian tubes can be evaluated after several menstrual cycles; this delay allows for complete resolution of the inflammatory process.



CHAPTER HIGHLIGHTS

- Sexually transmitted infections (STIs) are infections transmitted by sexual contact, including vaginal, oral and anal intercourse. STIs affect women and infants more than men, and are more common in people who have multiple sex partners, abuse drugs or are of a lower socioeconomic status.
- STIs can coexist in the same person and are transmitted by sexual contact. Effective treatment mandates that all sex partners be treated. Most STIs can be prevented by using latex condoms.
- Genital herpes, caused by an infection with an HSV virus, is a commonly occurring STI in teens and young adults. There is currently no cure and treatment is primarily symptomatic. Nursing care is directed towards relieving the pain of the lesions, mitigating sexual dysfunction and relieving anxiety.
- Genital warts, caused by the human papillomavirus (HPV), are a chronic, incurable STI. They are manifested by warts of various forms or may be present without manifestations. Infection with HPV poses a major risk of cervical cancer. A vaccine against the virus has been developed and is recommended.
- Urogenital infections include vaginal infections (bacterial vaginosis, candidiasis and trichomoniasis), chlamydia, gonorrhoea, syphilis and pelvic inflammatory disease (PID).
- Chlamydia, occurring most in young adults under age 25, is a bacterial infection that can spread to the uterus and fallopian tubes in women, causing pelvic inflammatory disease, infertility and ectopic pregnancy. Untreated chlamydia in men may result in epididymitis, prostatitis, sterility and Reiter's syndrome.
- Gonorrhoea (caused by a bacteria) and syphilis (caused by a spirochaete) affect both men and women and may infect the newborn as it moves through the birth canal in an untreated woman. Syphilis, if untreated, exists in the body in three stages, with the third stage lasting up to 50 years. Both of these STIs are treated with antibiotics. Nursing care focuses on education, preventing injury from complications, relieving anxiety and supporting self-esteem.
- Pelvic inflammatory disease is an infection of the female pelvic organs and may be caused by one or more infectious agents. Sexually active young women between the ages of 16 and 24 are most at risk. The prognosis depends on the number of episodes, promptness of treatment and modification of risk-taking behaviours. The goals of nursing care are to treat the infection and prevent complications.

CONCEPT CHECK

1 Which population is most often affected by STIs?
1. men
2. women and infants
3. adolescent males
4. older adults

2 Which of the following statements indicates that a person understands how they should treat an STI?
1. 'My sex partner and I must both take medications.'
2. 'I know I can never have sex again.'
3. 'I will douche after every sexual encounter with my partner.'
4. 'My sex partner does not have an infection, so won't need medications.'

3 When providing a man with information about using condoms to prevent STI, which topics should be included? (Select all that apply.)
1. Use a new condom with each sex act.
2. Ensure a small amount of air is in the tip.
3. Use oil-based lubricants, such as petroleum jelly.
4. Handle carefully to ensure no damage.
5. Withdraw when the penis is erect.

4 You are assessing a young male. He has both blisters and ulcerations on the shaft of his penis. What is he most likely to have contracted?
1. chlamydia
2. gonorrhoea
3. genital warts
4. genital herpes

5 Of the following statements about genital warts, which one is not true?
1. The infection is caused by a yeast organism.
2. The infection can be spread by any type of intercourse.
3. The infection may be transmitted to the foetus.
4. The infection cannot be cured.

6 You are counselling a young woman with an HPV genital infection. Which screening test would you recommend she have every year?
1. breast exam and mammogram
2. stool for occult blood
3. full blood count (FBC) to detect anaemia and infection
4. pelvic exam and Pap smear

7 Which of the following symptoms would most commonly be elicited as part of a health assessment for a woman who has a vaginal infection?
1 pain
2 itching
3 nausea
4 diarrhoea

8 When providing health education to a woman with an STI who has severe genital discomfort, what is one simple recommendation that may relieve her discomfort?
1 Wear nylon pantyhose.
2 Cut fingernails short.
3 Wear cotton underwear.
4 Don't have sex anymore.

9 The infective organism responsible for gonorrhoea initially targets which body parts?
1 male urethra and female cervix
2 female vulva and vagina
3 male prostate
4 male and female external genitalia

10 When providing health education to a person with syphilis, what would you say?
1 Syphilis is caused by a virus.
2 Syphilis is a local genital infection.
3 Syphilis is a systemic infection.
4 Syphilis has no effect on the developing foetus.

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UNIT 13 BUILDING CLINICAL COMPETENCE

Responses to altered reproductive function

CLINICAL SCENARIO

- You have been assigned to work with the following four people for the 0700 shift on a medical–surgical hospital unit. Significant data obtained during handover are as follows:
- David Young, a 24 year old, is admitted with a diagnosis of sickle cell crisis. Vital signs are T 37.6°C, P 86, R 10 and shallow, BP 142/84. He is complaining of chest pain, back pain and priapism. He has a PCA with morphine sulfate for pain.
- Victor Windsor, a 77 year old, is 3 days postoperative for transurethral resection of the prostate (TURP). Vital signs are T 37.2°C, P 84, R 16, BP 155/86. His urinary catheter was removed at 0600. He is complaining of dribbling of urine after attempting to void.
- Tina Morriss is 19 years old and newly admitted from the emergency department with complaints of severe lower abdominal pain, purulent vaginal discharge and cervical pain on pelvic exam. Her vital signs are T 39.3°C, P 86, R 22, BP 115/70. She is to be started on ceftriaxone and gentamicin IVI as soon as possible.
- Rose Getz, a 36 year old, has a history of uterine fibroids. She had a uterine fibroid embolisation yesterday. Her vital signs have remained stable throughout the night. Her discharge is written and she is in a hurry to go home so that her husband can get to work.

Critical thinking questions

1 In which order would you visit these people?

1. _____
2. _____
3. _____
4. _____

2 Which top two nursing priorities would you choose for each of the people presented above? Explain the rationale for your choices.

	Nursing Priority #1	Nursing Priority #2
David Young		
Victor Windsor		
Tina Morris		
Rose Getz		

3 Mr Young asks the nurse about complications due to priapism because he often has this problem with sickle cell crises. Which complication does the nurse discuss with him?

1. severe phimosis
2. erectile dysfunction
3. penile cancer
4. urinary retention

4 Mr Windsor suddenly develops nausea and vomiting. His BP is 170/100, P is 58 and he is confused. What is the probable cause of these manifestations?

1. allergic reaction to the bladder irrigating fluid solution
2. anxiety reaction to having prostate surgery
3. absorption of isotonic bladder irrigating fluids
4. hypovolaemic shock complication of the surgery

5 Which of the following is *most* important to educate Ms Morriss about pelvic inflammatory disease?

1. Take all medication as prescribed.
2. Wipe from front to back after urinating.
3. Use latex condoms when having sex.
4. Change tampons or pads every 4 hours.

6 Prior to surgery, Mrs Getz's haematocrit was 34% and haemoglobin was 10 g/dL. She was instructed to increase iron foods in her diet. Which foods are high in iron?

1. beef, eggs, brown rice
2. green, leafy vegetables, chicken
3. fish, broccoli, kidney beans
4. liver, cheese, asparagus

7 Which is a priority nursing *intervention* for a young woman with endometriosis?

1. *Acute pain* as manifested by severe abdominal cramping
2. *Knowledge deficit*: lack of understanding about endometriosis
3. Treatment for partners
4. *Risk of anxiety* related to loss of reproductive function

8 A prescription for tadalafil is ordered for a man with erectile dysfunction. The man indicates he understands about the medication when he says:

1. 'I will take tadalafil just before intercourse to increase my sex drive.'
2. 'I will consult with the doctor before taking other medications.'
3. 'I can take a second tablet the same day if the first one doesn't work.'
4. 'This tablet will relieve all of my sex problems.'

9 Which laboratory studies would you expect to be conducted to diagnose syphilis? (Select all that apply).

1. VDRL
2. Gram stain
3. RPR
4. DFA
5. tissue culture
6. FTA-ABS

10 Which should you say to someone who is receiving radiation therapy for prostate cancer?

1. Eat more red meat to maintain protein for healing.
2. Avoid close contact with pregnant women, infants and children.
3. Avoid sexual contact to prevent stress from dysfunction.
4. Sleep alone for the first night after radiation therapy.

11 Which symptom would be most significant when assessing someone with possible cervical cancer?

1. vaginal discharge
2. weight loss
3. pain in back and thighs
4. vaginal bleeding

12 A sexually active woman is being treated for gonorrhoea. Which method of birth control do you advise is safest to use?

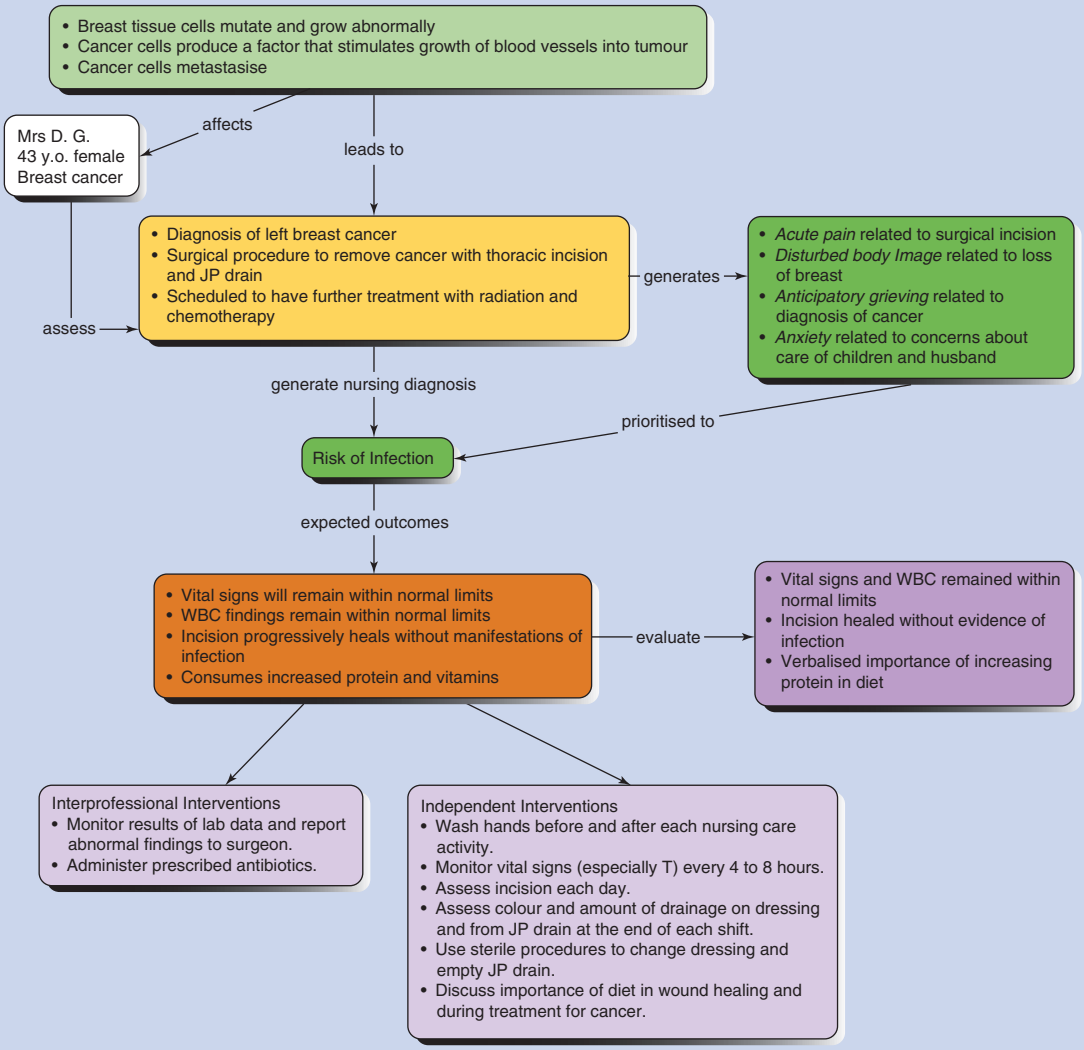
1. oral contraceptives
2. intrauterine device
3. condom
4. oestrogen patch

CASE STUDY

Delaine Grivstasis, a 43-year-old married housewife with two children, had a routine annual mammogram that revealed a mass in the left breast. She has a family history of breast cancer, with her mother and a maternal aunt having breast cancer. She was admitted to outpatient surgery for an incisional biopsy, with results of invasive lobular carcinoma. An MRI was performed, which indicated that four axillary glands have been infiltrated with cancer. She was admitted to the hospital and a modified radical mastectomy on the left side is performed. Postoperatively, she is admitted to the chemotherapy unit. Vital signs are T 36.9°C, P 65, R 14, BP 110/68. She has a dressing on the left chest that is dry and intact, and the Jackson–Pratt drain is intact for suction. Her left arm is elevated on two pillows. The decision has been made to start radiation and chemotherapy treatments as soon as possible.

The pathophysiology of breast cancer is a mutation of breast tissue cells related to hormones and mutations of tumour suppressor genes. Abnormal cell growth occurs. Cancer cells create a factor that stimulates blood vessels to grow into the tumour. Cancer cells invade the blood vessels and travel through the bloodstream or through the lymphatic system to other sites. Manifestations of breast cancer include non-tender lump in the breast, abnormal nipple discharge, rash around the nipple area, nipple retraction, oedema, dimpling of the skin or change in position of the nipple. Some women report a burning or stinging sensation in the breast or nipple pain. Complications of breast cancer are metastasis to bone, brain, lung, liver, skin and lymph nodes, and death.

Based on the surgery of modified radical mastectomy and treatment with radiation and chemotherapy, the nursing diagnosis of *Risk of infection* is an appropriate consideration for planning care for Mrs Grivstasis.



UNIT 14

SPECIAL TOPICS IN MEDICAL–SURGICAL NURSING

CHAPTER 50

MENTAL HEALTHCARE IN THE AUSTRALIAN CONTEXT

LORNA MOXHAM, PAUL ROBSON, SHANE PEGG

KEY TERMS

LEARNING OUTCOMES

- Understand the complexity of definitions related to mental health and mental illness.
- Understand the specialised role of the mental health nurse.
- Appreciate the importance of the Recovery approach within mental healthcare.
- Identify legislation pertaining to the provision of care for people who have a mental illness.
- Understand the symptoms and signs of psychotic and non-psychotic disorders.

CLINICAL COMPETENCIES

- Conduct a mental state assessment (MSA) on a person who is thought to have a mental health issue.
- Use an evidence-based approach to design interventions which promote Recovery.
- Determine priority nursing care, based on assessed data, to select and implement individualised nursing interventions for people who have a mental health issue.
- Provide skilled mental health nursing care for people who live with a mental illness.
- Integrate mental health nursing care into an interprofessional focus for people who live with a mental illness.
- Provide education to decrease the stigma associated with mental illness.

In Australia, and internationally, mental health problems are becoming increasingly common—as many as one in five people are now thought to have a mental health issue in Australia. Internationally, using the United Kingdom as an exemplar, the Mental Health Foundation suggests that up to one in four people will experience some kind of mental health problem in the course of a year.

As a consequence, mental health knowledge and skills, sometimes referred to as mental health literacy, are necessary for nurses working in all areas of nursing practice. Nurses may meet and nurse people as well as their families in a variety of environments such as the emergency department, drug and alcohol services, surgical and medical units, critical care units (CCUs), maternity, outpatients, day surgery, baby health clinics, schools, GP surgeries, paediatrics, intensive care units (ICUs), community health, sexual health clinics, pathology, x-ray departments and rural health.

HOLISTIC CARE

Even though nurses may not be undertaking practice within a specialised mental health setting, it is highly likely that they will provide nursing care for a person who is living with a mental health issue. It may not be the actual person who is the recipient of direct care, but it may be their partner, aunt, uncle, wife, husband, brother, sister, daughter, son, mother, father, grandfather or grandmother. Nurses are beholden to provide the best care they can to all the people they interact with in their practice. This often includes family members and significant others. In the mental health setting, the involvement of carers and family is very important and significant others should be involved in all steps of the nursing process wherever possible. Nurses provide holistic care, across the entire lifespan, which means people are cared for in all aspects of their life and body, and without judgment. Nurses should not 'split' people into physical, social, spiritual or mental health parts—nursing does not compartmentalise. Nurses are there to provide holistic healthcare, to all people in all circumstances, in the safest and best way possible. This is not always easy and nurses cannot be expected to be experts in every nursing specialty. Importantly though, nor do they need to be.

Nurses do, however, need to have an understanding of many areas of nursing. In order to have a balanced understanding of mental illness/mental health, it is important to be aware that there are a number of different types of knowledge bases that contribute to such an understanding. Figure 50.1 demonstrates where our **knowledge resource base** comes from. In the mental health context, the knowledge resource base is founded on a recognition of diversity, mental health literacy, an enriched range of services and supports, and social acceptance and inclusion.

As you can see from the figure, there are a number of different ways that nurses can acquire knowledge about mental health issues. Despite the recognition of multiple ways of knowing and doing, mental health service provision in Australia is still largely based on the medical model. However, with more consumers and their carers gaining a legitimately stronger voice, a social position is being strengthened as nurses continue to advocate for a more holistic approach. As Figure 50.1 suggests, nurses need to appreciate that a balanced understanding of a person's mental health is not only attained through medical or clinical knowledge. This is important as mental healthcare needs to be seen through many different lenses and treatment for people who are living with a mental illness should not be restricted to the administration of medication/s.

Because of the widespread nature of mental health issues, it is necessary to identify some trends around mental health and illness across Australia (see the 'Fast facts' box below).

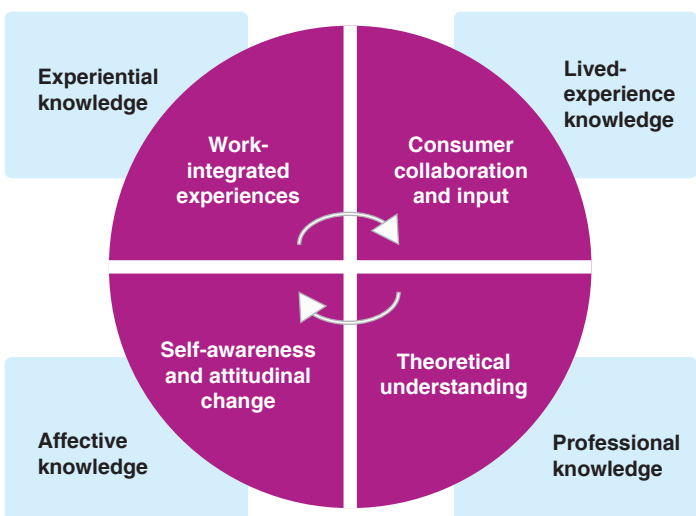


FIGURE 50.1 ■ Knowledge resource base

FAST FACTS

- One in five Australians (20%) will experience some form of mental illness each year. Of these, three out of every 10 will be seriously affected.
- In their lifetime, approximately 45% of Australians aged 16 to 85 years will experience a common mental-health-related condition such as depression, anxiety or a substance use disorder.
- In Australia, approximately 13% of the total burden of disease in Australia is associated with mental and behavioural disorders, placing it third behind only cancer and cardiovascular disease.
- Approximately 31% of Australians receiving a disability support pension were assessed as having a primary medical condition under the category of 'psychological or psychiatric'.
- Depression and anxiety are the most prevalent mental disorders experienced by Australians with over 14% reporting having some form of anxiety disorder. Significantly, depression is predicated to be one of the world's largest health problems by 2020.
- Mental illness affects young people. Around 14% of 12–17 year olds and 27% of 18–25 year olds experience a mental illness in any given year. At least one-third of young people have had an episode of mental illness by the age of 25 years.
- Around 1 million Australian adults and approximately 100 000 young people live with anxiety and depression each year. On average, one in five people will experience depression in their lives—one in four females and one in six males—while many younger people (200 000) are now presenting with aggressive behaviours.
- The majority of mental illnesses begin between the ages of 15 and 25 years. This poses a significant threat to our nation's workforce capacity and economic prosperity.
- The 2007 National Survey of Mental Health and Wellbeing revealed that 11.7% of Australians who had a long-term mental disorder also reported having a physical condition.
- Approximately two-thirds of people with a mental illness do not receive any treatment in a 12-month period.
- Estimates suggest that up to 75% of people presenting with alcohol and drug problems also have additional mental health problems.
- Reports indicate that up to 85% of homeless people have a mental illness.

Sources: Australian Institute of Health and Welfare (AIHW) (2015). *Mental health services in Australia*. Retrieved from http://mhsa.aihw.gove.au/home; Department of Social Security (2014). *Characteristics of disability support pension recipients*. Canberra: Department of Social Security; Institute for Health Metrics and Evaluation (2013). *The global disease burden: Generating evidence, guiding policy*. Retrieved from www.healthmetricsand evaluation.org/gbd/visualizations/country; Mental Health Council of Australia (2012). Mental health fact *sheet: Statistics on mental health in Australia*. Retrieved from www.mhca.org.au/documents/StatisticsonMHinAustralia.pdf.

MENTAL HEALTH AND MENTAL ILLNESS

Mental health and mental illness are difficult terms to define; in fact, there are no universally agreed definitions. While such ambiguity can be frustrating, there are many reasons for this. One key reason arises from the impossibility of separating mind from body; another results from the many unique and individual ways that humans express themselves. Mental illness, as an expression, is also culturally linked and determined, so what might be deemed mental illness in one culture certainly might not be in another.

While it is difficult to arrive at succinct, yet meaningful, definitions of mental illness and mental health, many attempts have been made to conceptualise them. These include:

- the medical model (which emphasises processes of diagnosis and treatment and is based on traditional reductionist models of science)
- the psychoanalytical or psychodynamic approach
- neurobiological theories
- cognitive behavioural perspectives
- lay views
- the legal framework
- lived experience perspectives
- sociological stances (including social causation, social reaction (labelling theory), holism and the social model of disability).

Just as the definitions of mental health and mental illness are ambiguous, so too are the causes and associated nursing care and medical treatments. This makes mental health nursing both complicated and challenging. Modern multidisciplinary mental health practice synthesises the application of understandings that are derived from the various conceptual approaches listed above. Despite this, treatment and management approaches remain heavily dominated by medical models and medical discourse. This is because psychiatrists, as specialist medical practitioners, are trained to identify sick individuals (diagnose), predict the future course of their illness (provide a prognosis), speculate about the cause (identify aetiology), then prescribe a response to the condition, either to cure it or ameliorate its symptoms (medicate).

Diagnosis

The diagnosis of mental illness in Australia occurs as a result of an assessment process. The American Psychiatric Association (APA) established a set of diagnostic criteria, known as the **DSM (*Diagnostic and Statistical Manual of Mental Disorders*)**. The DSM is used in Australia to diagnose a person with signs of mental illness. The DSM 5 was released in May 2013.

There is also another set of diagnostic criteria in use. This is the **International Classification of Diseases (ICD)**, which is the global standard to report and categorise diseases, health-related conditions and external causes of disease and injury. The ICD that specifically relates to mental healthcare is the ICD-10—Classification of Mental and Behavioural Disorders. Classification systems are constantly revised. The current version is the ICD-10: Version 2010. The eleventh revision of the ICD is underway and it is expected to be presented to the World Health Assembly some time in 2017. The current version, ICD-10, is available in the six official languages of the World Health Organization (WHO),

which are Arabic, Chinese, English, French, Russian and Spanish, as well as in 36 other languages (WHO, 2012a).

The Australian version, the ICD-10-AM or, more accurately, the *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification*, serves Australia's particular needs and supports the national collection of data relevant to its population health.

Psychiatrists are in powerful and dominant positions within mental health settings (they are often the directors of services at local, state, territory and national levels). As such, medical opinion remains at the fore of any debate about mental health problems. As a result of these powerful influences, psychiatrists principally view people through an illness lens when they encounter variations in conduct or when they have to assess someone who is 'different'. The concept of illness—and its associated language, symptoms, therapies and the people who are living with the illness, who are invariably known as 'patients'—legitimises and perpetuates the prevailing public perceptions and attitudes, often at the expense of other possible frameworks.

It is therefore fair to say that significant debate still exists about what constitutes mental health or mental illness. One of the places where discourse occurs is the **Australian College of Mental Health Nurses (ACMHN)**. This college is the peak professional body for mental health nurses in Australia and is overseen by a Board of Directors that is elected by college members every 2 years. The ACMHN is recognised as representing the specific interests of mental health nurses in Australia. The ACMHN hosts an annual international conference at which issues such as mental health, mental illness, treatment, policy and particularly mental health nursing practice are discussed and debated.

Definitions

Although there is no specific universal definition, mental health can be described as a positive state in which the person is responsible, self-directive, displays self-awareness and is generally accepted within a group. Numerous factors impact on mental health and include inherited characteristics, childhood nurturing and life circumstances. The nature/nurture debate continues in the field of mental health.

Nature refers to inherited genes and characteristics that are totally out of one's control. Nurture refers to parent–child interactions, sibling interactions and early communication patterns. Positive childhood nurturing refers to the child feeling loved, secure and accepted.

Life circumstances from birth also influence one's mental health. These include socioeconomic status, quality of relationships, physical health, housing and educational success or failure. If influencing factors are positive, a person is more likely to experience positive mental health and a sense of wellbeing.

Mental health is generally defined by essential characteristics rather than a set of statements about a state of health. In order to define mental health, comparisons are often made between mental health and mental illness. This is done in an attempt to distinguish the 'normal' from the 'abnormal'. There is therefore a lot of confusion about what mental health and mental illness are, and how they differ. In the absence of a universal definition, Figure 50.2 describes the factors that influence a person's mental health, while Table 50.1 offers a set of indicators for mental health.

Stigma

Stigma is the single most difficult issue that people with a mental illness face. When society or communities consistently stigmatise people with a mental illness, such attitudes become internalised. Internalised stigma is a process whereby people with a mental illness endorse stereotypes, anticipate social rejection, consider stereotypes to be self-relevant and believe they are devalued members of society. Sensationalist media headlines lead to a perpetuation of such stigma (see Box 50.1).

As the scenario in Box 50.1 demonstrates, *reactions* as a result of this event were considered over the top. Thanks to sensationalist media reporting, which still persists, common public perception remains that innocent individuals may become the victim of random acts of violence perpetuated by people with mental health issues.

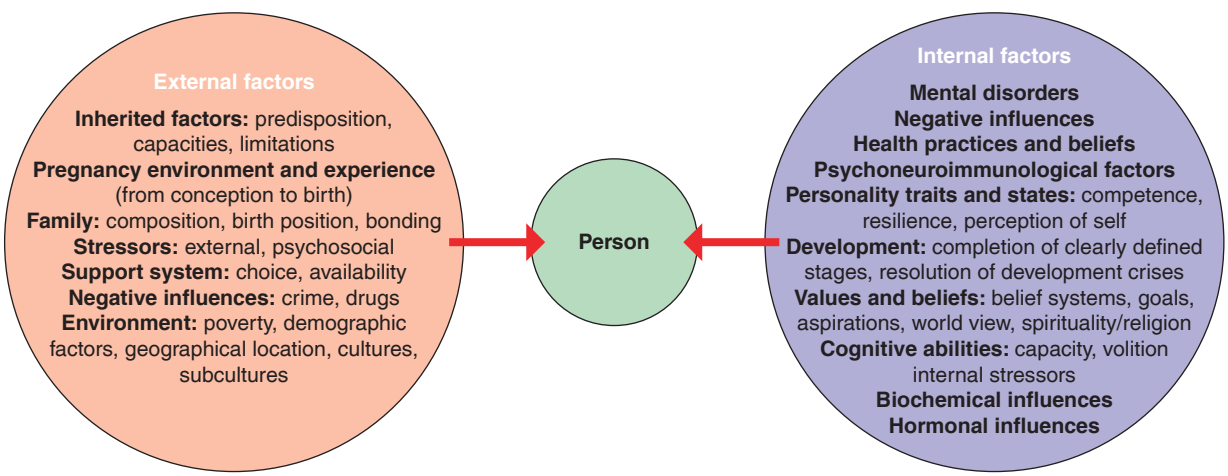


FIGURE 50.2 ■ Influencing factors for mental health or illness

Table 50.1 Components of mental health

SELF-AWARENESS AND DEVELOPMENT	ENGAGEMENT WITH AND RESPECT FOR OTHERS	ADAPTABILITY AND RESILIENCE	CLARITY AND INSIGHT
Attain and maintain positive self-esteem: • Self-concept • Self-image • Self-esteem Involve self in purposeful, meaningful life work Maintain reasonable expectations concerning self and others Seek self-actualisation Learn from experiences Use talents to fullest Maintain wholesome values and belief system Develop and demonstrate appropriate sense of humour Attain self-defined spirituality	Respect societal rules and sanctions Accept self and others as uniquely different but humanly similar Engage in play Relate to others: • Form relationships • Maintain close, meaningful, loving, adaptive relationships • Work and play well with others • Be intimate, appropriately and selectively • Respond to others in need • Feel and exhibit compassion and empathy towards others • Demonstrate culturally and socially acceptable interpersonal interactions • Manage interpersonal conflict constructively • Give and receive gracefully • Learn from and teach others • Function interdependently	Adapt to social environment Function interdependently Return to usual or higher function after crises Be optimistic Be resilient Find beauty, joy and goodness in self, others and the environment Appreciate life Negotiate each development stage Cope with internal and external stressors in constructive and adaptive ways	Ability to: • Demonstrate mental and physical competence and skills • Perceive self, others and events correctly • Recognise own strengths, weaknesses, capabilities and limitations • Separate fantasy from reality Think clearly: • Problem solve • Use good judgment • Reason logically • Reach insightful conclusions
SELF-CONTROL	**INDEPENDENCE**	**SELF-EXPRESSION**	**PHYSICAL HEALTH**
Control impulses and behaviour Delay gratification	Accept responsibility for actions Function independently	Be creative Express emotions Exhibit congruent thoughts, feelings and behaviours	Presence of anatomical and physiological components necessary to function in the world Absence of signs and symptoms of mental disorder Freedom from excessive mental and emotional disability and pain

There is a general belief that people who have a mental illness are dangerous. This perception underpins some of the most destructive stereotypes. Research has demonstrated that people with a mental illness are no more violent than members of society who do not have a mental illness. They are often frightened and are among the most vulnerable members of society. That is not to say, though, that they do not get frustrated and angry with things that happen as part of everyday life (as we all do). The media, however, have promulgated negative stereotypes. Public perception is largely influenced by media sources and negative portrayals have negative impacts on people who live with a mental illness and their family.

Most people who have a mental illness do not need inpatient treatment. If they do, admissions to mental health inpatient units are often brief. Effective treatments, mostly developed in the 1950s and early 1960s, have removed the need for isolation and confinement in asylums—a common occurrence in the past (see Figure 50.3). The attitude of family, friends and the community towards people who have a mental illness plays a critical part in determining their quality of life. Attitudes that people, including nurses, hold have a major effect on how the people feel about themselves and on their recovery. Many people with a mental illness face isolation and discrimination simply for having an illness. This is often not so for people who have a physical illness. People who have cancer, diabetes, asthma, multiple sclerosis, Alzheimer's disease, Parkinson's disease and heart diseases such as cardiomyopathy are often met with sympathy, but people who are living with a mental illnesses are often the butt of jokes, innuendos and scaremongering.

Who would get your donation? An organisation that works with people who have cancer, or one that works with people who have a mental illness? Why is it that people donate more for physical conditions when mental health problems affect at least 25% of the population?

The role of the mental health nurse

The role of the mental health nurse is multifaceted, specialised and dynamic. Mental health nurses' skills, and the roles that they perform, vary according to the context of practice and the level of education, training and experience of the individual nurse. Fundamentally, mental health nurses provide nursing care for people whose lives are affected by mental illness (see Figure 50.4). Nursing care includes the promotion of mental health, the prevention of mental illness and the minimisation of the problems associated with mental illness on individuals, their families and the community. The ACMHN describes mental health nursing as a specialised field of nursing which focuses on meeting the mental health needs of the person in partnership with family, significant others and the community in any setting.

BOX 50.1 The story of a person with a mental illness

Consider the case of a person in Queensland, Australia. The person, who was described on the front page of the *Courier-Mail* (a Queensland newspaper) as 'a paranoid schizophrenic who hacked off his clergyman father's head' (*Courier-Mail*, 23 January 2002) left his ward, which was within the grounds of a psychiatric rehabilitation facility, without permission. Eighty-four hours later, a tired, hungry and thirsty man quietly returned to his ward after having spent the entire time under a cricket shed in the grounds of the facility.

In the meantime, following what was reported as 'extensive searching', a maelstrom erupted; the resultant media coverage could be described as nothing less than a feeding frenzy. The man appeared to represent everything that society feared about mental illness—namely, that all people with mental illness are dangerous. In a sense, this man became stigma personified and for weeks people in Queensland were bombarded with news of other mentally ill people on the run. 'Dangerous', 'violent', 'criminal', 'escapees' and 'killer' were all used to describe people who had a mental illness but were currently, and for numerous valid reasons, not being detained in psychiatric hospitals at all. The relentless media barrage, particularly the insinuation that members of the general public were in danger from these 'killers at large', resulted in a reaction few would have expected. First, an inquiry was held to quantify how many 'patients' who were deemed to be criminally insane were absent without leave (AWOL) from mental health facilities. Recommendations from this inquiry included the 'capture' of these so-called AWOL patients. Surprisingly and controversially, given the ethical nature of the issue, the then state premier ordered the release of photographs of 15 'patients' who were described as AWOL; these pictures were published in the *Courier-Mail*. Second, any patient then under a forensic order who was living in the community, despite the fact that their leave had been granted by the treating psychiatrist and the then Patient Review Tribunal (now the Mental Health Review Tribunal), were taken back to hospital under an involuntary treatment order (ITO). Many people who were well and residing in the community were returned to hospital as a result of this unprecedented over-reaction. Others feared that they, too, would soon be 'taken back to hospital'. Would anyone contemplate doing this to people who have a physical illness?

The tragedy of this 'escape' and its aftermath reached far beyond the city where it occurred. It was acutely felt by people living with mental health issues across the country. The stigma extended to families and their friends, many of whom have also struggled with the discrimination that having a family member with mental health issues brings.

See also 'Risky news, madness and public crisis: A case study of the reporting and portrayal of mental health and illness in the Australian press' by W. Blood & K. Holland (2004). *Journalism*, *5*, 323–342, http://jou.sagepub.com/cgi/reprint/5/3/323.pdf, for an excellent analysis of the reporting of this event.

Mental health nursing has long been recognised as one of the three specialist nursing areas. Psychiatric nurses had separate registration or endorsement in Australia until recently. Previously in Australia, students could enter directly into a 3-year hospital-based mental health nursing course, graduating with a certificate in

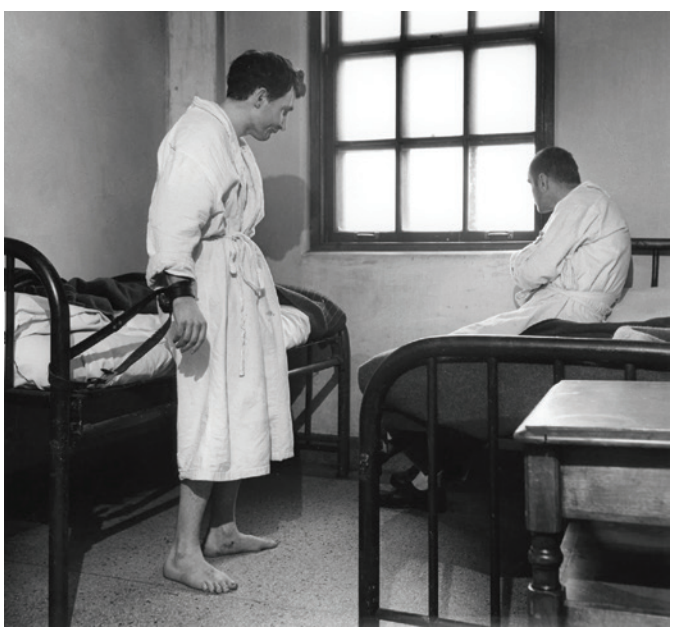


FIGURE 50.3 ■ Isolation and confinement in asylums was common treatment for mental illness prior to the 1950s

Source: © Photo Researchers/Getty Images.

FIGURE 50.4 ■ Mental health nurses provide care for people whose lives are affected by mental illness

Source: Monkey Business Images/Shutterstock.

psychiatric nursing and registering as a Registered Psychiatric Nurse (RPN). This changed when nursing education moved en masse to the higher education sector in 1985. Today, baccalaureate programs (BN) prepare the nurse for 'beginning level' in all health settings, but many BN students have only limited clinical placements in mental health settings and some get no placements at all in mental health. Some education providers have a 'major' in mental health nursing, where the student nurse can start to specialise, usually in the second year of their BN. For the most part, specialist mental health nursing is now a postgraduate area where students can undertake a graduate certificate (GradCert), graduate diploma (GradDip), master's, professional doctorate or PhD.

Mental health nursing in practice

Mental health nurses can aptly be described as the eyes and ears of the multidisciplinary team. Nurses interact with people on a continual basis, observing ADLs and social functioning, monitoring effects (positive and negative) of medication and treatment, and working closely with people and their significant others in a therapeutic manner towards Recovery. Such a therapeutic relationship places nurses in an excellent position to work in collaboration with the person and their family to provide care, such as:

- planning, implementing and evaluating interactions to help the person achieve the highest possible quality of life using a Recovery focus (see below)
- monitoring the person's response to nursing and other interventions, including medication efficacy
- the establishment and maintenance of the therapeutic relationship.

Mental health nursing is founded upon a **therapeutic relationship**, a rapport based on empathy and trust, with the person and family. There is increasing evidence suggesting that a positive therapeutic relationship contributes significantly to recovery. Mental health nurses, who are central in forming and maintaining the therapeutic relationship, therefore play a pivotal role in the *recovery* of a person who is living with a mental illness. The essence of mental health nursing is therefore not in the tasks that are performed, but in the positive relationships the nurse develops with the person and their family. Nursing focus should not be on the illness (the diagnosis that has been given) but on how the person manages on a daily basis. People living with a mental illness are more focused on day-to-day living than the label they have been given.

AREAS OF PRACTICE Within an inpatient unit in many Australian settings there are high-dependency units (for people who are even more acutely unwell). These can be equated to critical or intensive care units in general hospitals. **Acute inpatient units** could be described as the mental health services equivalent of high-dependency nursing. Mental health nurses in these settings need highly developed expertise and skills in observation, assessment, negotiation, de-escalation and milieu management.

Intake team or *acute care team nurses* are often considered the gatekeepers to mental health services. This is also acute care nursing. In these settings the mental health nurse provides short-term interventions and requires highly specialised skills in assessment, psychopharmocology and monitoring. Mental health nurses on these teams often do their 'work' via the telephone, when people call mental health services in crisis. It takes special expertise to be able to conduct an accurate assessment without face-to-face interaction. The role of mental health nurses in this setting is also to be aware of and to liaise with other services and agencies.

Community care coordinators are mental health nurses who undertake their craft by providing continuing care and follow up of people in the community. They used to be called 'case managers', but consumers say, and rightly so, they are not 'cases' to be 'managed'. Care coordinators often have many people to work with and undertake duties such as medication administration, management and monitoring. Assisting people by finding accommodation and employment options, as well as linking people in with therapeutic leisure and recreation activities, are also part of the role. **Community mental health nurses** also provide family support and work with the broader community in mental health promotion and education.

Forensic mental health nurses practise in a range of settings that include courts, police custody centres, prisons, secure hospitals and the community. Practice standards for forensic mental health nurses can be viewed in the *Forensic Mental Health Nursing: Standards of Practice 2012*, which can be accessed at <www.forensicare.vic.gov.au>.

Consultation liaison (CL) nurses have different approaches depending on the setting in which they are practising. This area of mental healthcare is rapidly expanding as a result of the number of people within generalist care settings who also have a mental illness. One approach is having CL nurses in the emergency department (ED) who provide specialist care for someone in the ED with a mental health problem. Another CL approach is where the CL nurse provides care to people who are admitted to a general hospital for a non-psychiatric condition. The CL nurse in this instance usually gets a referral from their general nursing or medical colleagues and provides direct consultation with the person or indirect consultation through support, education and advice for colleagues. CL nurses have high-level assessment and interpersonal skills and broad nursing knowledge that enable them to rule out physical causes and work with complex health issues.

STIGMA AND THE ROLE OF THE NURSE As Box 50.1 shows, the general public continues to receive a very distorted and one-sided picture of people with mental health problems. Sensationalist reporting exacerbates discriminatory attitudes and stigmatising attitudes that society in general holds towards people with mental health problems. Public stigma robs people who live with a mental illness of many opportunities. Nurses need to discuss mental illness openly with family, friends and colleagues. When nurses think about and describe mental illness in similar terms to other illnesses or conditions, this helps educate the community to overcome discrimination and negative attitudes based on misconceptions and stereotypes. Box 50.2 responds to some common misconceptions about mental illness.

Because it is common for individuals to accept and internalise negative stereotypes it is a prime responsibility of nurses to advocate for people who have a mental illness. This means dispelling myths and challenging discrimination. We need to tell people that

BOX 50.2 The facts about mental illness and violence

Are people with a mental illness violent?

Having a mental illness does not mean someone will be violent. People receiving treatment for a mental illness are no more violent or dangerous than anyone else. To make this clear, it has been calculated that the lifetime risk of someone with an illness such as schizophrenia seriously harming or killing another person is just 0.005%. It is much more likely that someone with a mental illness will hurt themselves or be hurt by someone else.

Is there a link between mental illness and violence?

The great majority of people with a mental illness are not violent at all. Research suggests they are more likely to be victims of violence. Violent behaviour is slightly more likely among people with a psychotic illness, especially in the first episode of illness. It may also be associated with other factors, including the type of person they were before the illness and if they have been violent in the past. It is also associated with drug use such as ice.

Does having an illness such as schizophrenia mean someone will be violent?

Violence is not a symptom of psychotic illnesses, such as bipolar disorder and schizophrenia.

There is a slightly increased possibility someone with a psychotic illness may be violent if they are not receiving treatment, have a previous history of violence or are abusing alcohol or drugs.

Symptoms of psychotic illnesses may include frightening hallucinations and delusions, as well as paranoia. This means there is a small chance someone who is experiencing them may become violent when they are scared and misinterpret what is happening around them. If a person is being effectively treated for psychotic illness and is not abusing alcohol or drugs, there is no more risk they will be violent than anyone else.

Who is most likely to be violent in our society?

Research by the Australian Institute of Criminology shows that the vast majority of violence is committed by males aged 18 to 30 years. This is more likely when someone has exhibited highly aggressive behaviours in the past and has some history of alcohol or drug abuse. People in this group are far more likely to be violent than someone with a mental illness.

Where do people learn about mental illness?

The media plays a big part in the way we think about mental illness. Sadly, media often make the link between mental illness and violence appear much stronger than it is. There is actually a weak link between mental illness and violence, but many people wrongly believe all people who have a mental illness are violent.

What can be done to help?

Mental health workers, people with a mental illness and their families all agree the most important step is making sure people receive effective treatment. Mental health workers need to know who is most at risk of being violent or of being a victim of violence. Making sure people receive the right treatment as quickly as possible requires early intervention, especially in the first episode of illness, and ongoing treatment for as long as required. It is also important for everyone to understand that mental illness is not a choice and could happen to anybody.

Source: SANE Australia: www.sane.org. Reproduced with permission of SANE Australia, the national mental health charity.

jokes about mental illness are not funny, names that are negative are not acceptable and that perpetuating myths is harmful.

On 21 January 2009, one of the largest ever programs to reduce stigma and discrimination against people with mental health disorders was launched. It was called 'Time to Change' and was UK based. The initiative was funded with £18 million and was run by three charities: Mental Health Media, MIND and Rethink. While the initial campaign concluded in September 2011, two of the charities involved, MIND & Rethink Mental Illness, have continued to evolve the campaign. Importantly, the campaign continues to advocate for changes in community and individual behaviour and not just attitudes towards people with mental illness. Equality and respect incorporate tolerance, active listening, empathy, compassion, safety, trust, diversity and cultural competence. Nurses should develop these virtues and practise them every day, both in and out of the work environment.

MENTAL HEALTH PROMOTION AND THE ROLE OF THE NURSE Nurses play an important part in promoting mental health. This can be achieved by nurturing healthy attitudes and coping mechanisms through early childhood and adult life. Nurses can empower people by helping them develop appropriate and effective ways to deal with trauma in relationships, situations and events. All nurses, no matter what their discipline area, should provide high-quality support and treatment services that enable people with a mental illness to participate fully in all aspects of their life (Recovery-focused care). Even when not on duty, nurses can encourage and assist friends and relatives who may have a mental illness to seek care and obtain treatment. The nurse can then follow up by encouraging engagement and compliance with the treatment regimen, just as they would with a person who has a physical illness, disease or injury that requires nursing and medical care.

Nurses can also encourage, support and undertake research related to mental health. This evidence base will assist the community to understand causes of mental illness; how these illnesses affect people, their families and the community as a whole; and how they can be prevented.

Mental health legislation

In 1901 Australia became a federation: a country with local, state, territory and federal governments. Australia has many state and territory differences and these occur in school curricula and holidays, number plates, pension entitlements and laws. Often terminology is also different between jurisdictions. Definitions about mental health and mental illness also differ between jurisdictions. These definitions are outlined in the

BOX 50.3 State and territory mental health legislation

Australian Capital Territory—*Mental Health (Treatment & Care) Act 1994*. Available at www.legislation.act.gov.au/a/1994-44/20100701-44521/pdf/1994-44.pdf

New South Wales (NSW)—*Mental Health Act 2007.* Available at www.austlii.edu.au/au/legis/nsw/num_act/mha2007n8155.pdf

Northern Territory—*Mental Health and Related Services Act 2004.* Available at www.health.wa.gov.au/mhareview/resources/legislation/NT_Mental_Health_Act.pdf

Queensland—*Mental Health Act 2000.* Available at www.legislation.qld.gov.au/LEGISLTN/CURRENT/M/MentalHealthA00.pdf

South Australia—*Mental Health Act 2009.* Available at www.legislation.sa.gov.au/lz/c/a/mental%20health%20act%202009/current/2009.28.un.pdf

Tasmania—*Mental Health Act 2013.* Available at www.thelaw.tas.gov.au/tocview/index.w3p;cond=all;doc_id=2%2B%2B2013%2BAT%40EN%2BSESSIONAL;histon=;prompt=;rec=;term=mental%20health%20act

Victoria—*Mental Health Act 2014.* Available at www.legislation.vic.gov.au/domino/web_notes/ldms/pubstatbook.nsf/f932b66241ecf1b7ca256e92000e23be/0001F48EE2422A10CA257CB4001D32FB/$FILE/14-026aa%20authorised.pdf

Western Australia—*Mental Health Act 2014.* Available at www.mentalhealth.wa.gov.au/Libraries/pdf_docs/Mental_Health_Act_2014.sflb.ashx

various **Mental Health Acts** (see Box 50.3) and further demonstrate the complex nature of mental illness and mental healthcare and treatment. Why is it that the states and territories do not differ in their approach to broken legs, appendicitis or aneurysms? Why is mental healthcare different?

Why is mental illness treated so differently?

The various definitions indicate that what *is not* a mental illness is just as important as what *is* a mental illness. This is indicative of the complexity of defining a mental illness; in fact, some of the mental health legislation does not actually define mental illness.

Although mental health legislation across various Australian jurisdictions has similar intent, the differences create major challenges for mobile populations, including nurses. Mental health nurses need to understand and implement different Mental Health Acts as they move across borders when changing jobs (now made easier with a single point of national registration). Subtle differences also make it more complicated for people who are managing their own journey of recovery, particularly if they also wish to travel interstate and are subject to an order set down within a Mental Health Act.

The concept of **Recovery** is a recognised and accepted paradigm that has significant implications for people who have mental health problems, their carers, mental health professionals and mental health services. Traditional models of mental health service delivery were not focused on Recovery; rather, they focused on containment and control of the individual. The notion of Recovery is embedded in the very personal journey of the individual, which is, of course, highly unique. Research has shown that people with a mental illness can and do recover but Recovery does not necessarily mean a cure; nor does it necessarily mean a return to a pre-illness state. Hope energises the Recovery process and lays the groundwork for the healing process to begin.

Recovery

Mental healthcare in Australia and many countries internationally now incorporates Recovery models of care in practice. The Australian Government is committed to the notion of Recovery as a process not an end point. The following information about the Recovery model was sourced from the excellent booklet *Sharing responsibility for recovery: Creating and sustaining recovery oriented systems of care for mental health,* www.health.qld.gov.au/mentalhealth/docs/recovery.pdf.

Recovery, as it is used in mental health, is the journey towards a new and valued sense of identity, role and purpose outside the parameters of mental illness. Recovery is about living well despite the limitations resulting from the illness, its treatment and personal and environmental conditions. Mental health services that are Recovery focused are underpinned by a fundamental shift in power, attitude and beliefs regarding the prognosis for a person who has been diagnosed with a mental illness. A Recovery approach emphasises mutual respect and collaboration between providers and service users as opposed to individual dependency on the system and a profound loss of personal autonomy. Recovery-focused service providers focus their attention on creating environments that nurture recovery and then on facilitating the process of recovery, rather than determining its direction. Recovery is considered a journey not an outcome.

Recovery is viewed as a journey that is a unique and personal experience for each individual (see Figure 50.5). It has often been said to be about gaining and retaining hope, understanding of one's abilities and limitations, engagement in an active life, personal autonomy, social identity, meaning and purpose in life, and a positive sense of self. Essentially, the personal view of Recovery is about a life journey of living a meaningful and satisfying life (NSW Consumer Advisory Group, 2012).

Helen Glover's 2012 model reflects the efforts that people undertake in their personal Recovery journeys through a set of five processes:

1. *from passive to active sense of self:* moving from the passive position of being a recipient of services to reclaiming one's strengths, attributes and abilities to restore recovery
2. *from hopelessness and despair to hope:* moving from a position of hopelessness and despair to one of hope
3. *from others' control to personal control and responsibility:* moving from others taking responsibility for recovery to the person taking, holding and retaining responsibility

FIGURE 50.5 ■ The concept of Recovery

Source: A national framework for recovery-oriented mental health services: Guide for practitioners and providers, Figure 2, p. 12 © 2013 Commonwealth of Australia.

4. *from alienation to discovery:* 'finding meaning and purpose in the journey; doing more of what works and less of what does not work; learning from experiences and incorporating those lessons into the present; acknowledging that journeys always have something to teach us and contribute to our sense of discovery'
5. *from disconnectedness to connectedness:* moving from an identity of illness or disability to an appreciation of personal roles and responsibilities and to 'participating in life as a full citizen and not through the powerlessness of illness' (NSW Consumer Advisory Group, 2012).

HOPE **Hope** is the foundation or guiding principle of Recovery. Hope is the belief that things do not have to remain the same and that change for the better can and does happen. Hope instils the belief that it is possible for someone to have a meaningful life, despite living with a mental illness. An approach based on hope concentrates on a person's strengths, with a focus on the future. Nurturing hope celebrates all successes rather than expecting and insisting on large and rapid change. Setting smaller goals means they are realistic and much more likely to be achieved. Positive reinforcement occurs because change has happened. This in turn provides future hope. Hope is about being positive and is important for the person to hold on to, but is equally important for others to have for the person. The positive impact that the hope of others can have on an individual with mental illness, when all seems bleak, cannot be underestimated. What do we have if we have no hope?

CONSIDERATION FOR PRACTICE

Mental health nurses are the holders of hope. When a person expresses little or no hope, offer to hold their hope in your hands. Tell them you will care for it and nurture it and when they are ready, you will hand it back.

Source: © S. Dashkevych/Shutterstock.com.

ACTIVE SENSE OF SELF For people who are living with a mental illness, sometimes their sense of self can become lost and their identity can revolve around their illness. It is very important that mental health nurses do not perpetuate this. This can happen when they refer to the person as 'the schizophrenic' or 'the depressive'. This kind of language is easy to fall into, but what it does is put the illness first, in front of the person. It is very disempowering language and serves to help destroy an already fragile sense of self. It is also stigmatising.

Nurses need to remember that having an illness is only one part of a person. Having a positive self-image comes from being treated as a whole person instead of just a part—this is called an **active sense of self**. Imagine if you were to be compartmentalised by only being referred to as a nurse or as a student. There is far more to you than simply being a nurse or a student and it devalues the complex person you are if only a single component of you is referred to. Recovery, then, goes beyond just merely functioning. It is the development of meaningful existence and a sense of purpose. We all need a reason to get up in the morning and our sense of purpose is developed through things such as relationships and work. Renowned psychiatrist Victor Frankl's premise is that 'man's search for meaning' is the primary motivation of his life.

DISCOVERY In mental health, **discovery** means that people with a mental illness deserve equality and respect. People who are living with a mental illness struggle every day with stereotypes that are perpetuated in the media (see Mindframe: <www.mindframe-media.info>). They are the most stigmatised and discriminated group in society. Unconditional positive regard is what nurses should afford people.

CONNECTEDNESS Recovery is a social process and correlates to the strength of people's social networks. It involves being with other people and reconnecting with the world. There are very few human beings who really enjoy being alone. Of course, there are times when we enjoy solitude, but by and large humans are social creatures and want the company of others. Indeed, humans actually need human company (interpersonal relationships) in order to be mentally healthy. To make social connections, social roles must be established. This is often very hard for the person who has a mental illness as society is not very accepting of '**differentness**' and, as we have already discussed, subscribes to myths and stereotypes that are simply untrue.

In fostering social connectedness, issues relating to community integration and full community participation need to be considered. Community integration is a concept drawn from the larger disability and civil rights movements and is founded upon the belief that all people have a right to full community participation and full community membership. This includes jobs, relationships with people who don't have a mental illness and appropriate and affordable housing.

PERSONAL RESPONSIBILITY It is the people who are affected by the mental health problem who hold the key to their own recovery, not service providers. Service providers such as mental health nurses need to ensure that people with mental health problems are recognised as whole people who are self-determining, able to make their own choices and able to take **personal responsibility** and live with the consequences of their decisions. Service providers, who are often risk averse, can find handing responsibility to the person a challenge. Recovery occurs when people are empowered to take ownership and when they can take an active role in their own recovery process. **Empowerment** is vital for correcting the learned helplessness that many people experience as a result of long-term interactions with a mental health system. The importance of people participating in their own healthcare has long been recognised as an essential element of effective service provision. This right is included in the World Health Organization Declaration of Alma-Ata (1978). It is also consistent with the principles stated in the *National Standards for Mental Health Services* and the former *National Action Plan on Mental Health 2006–2011*. These documents each served to highlight the need for care to be focused on the needs of the individual, rather than the service. The National Action Plan aimed to improve mental health and facilitate recovery from illness through a greater focus on promotion, prevention and early intervention; improved access to mental health services, including in Indigenous and rural communities; more stable accommodation and support; and meaningful participation in recreational, social, employment and other activities in the community. Improving the care system would also involve a focus on better coordinated care and building workforce capacity.

In 2012, The Council of Australian Governments (COAG) released the *Roadmap for National Mental Health Reform*. This document, now commonly referred to as simply 'The Roadmap', reaffirmed a commitment by state and territory governments to support the federal initiatives with a shared vision for mental health reform for the next 10 years. The creation of a COAG Working Group on Mental Health Reform, to oversee the implementation of new governance and accountability arrangements, has also been charged with development of the next version of the National Mental Health Plan (House of Representatives Standing Committee on Health and Ageing (HRSCHA), 2013). The current National Action Plan seeks to improve mental health and facilitate recovery from illness through a greater focus on promotion, prevention and early intervention strategies and related services. As part and parcel of this ongoing process, particular effort has been made to improve access to mental health services, inclusive of Indigenous and rural communities; provide more stable accommodation and support services; and facilitate the opportunity to access a more meaningful range of recreational, social, and employment-related activities in the community. In early 2014, the Australian Government requested the National Mental Health Commission to undertake a wide-ranging review of existing mental health programs and services across the government, non-government and private sectors nationally with a view to identifying ways to deliver services more efficiently and effectively. Concurrently, the Independent Hospital Pricing Authority was charged with the development of a new classification for mental health, to be known as the Australian Mental Health Care Classification (AMHCC). The development of the AMHCC is intended to improve the clinical meaningfulness of the way that mental healthcare services can be classified, leading to improvements in cost prediction, and support the implementation of new models. This has engendered a greater consideration of the best means for building greater workforce capacity. Of course, while much of the attention has been placed on national

initiatives in recent years, it should be equally evident that individual state and territory governments have also implemented their own suite of state/territory-specific mental health action plans and initiatives. Mental health is, and will remain for many years to come, a national health priority area.

The Commonwealth Government, having acknowledged the full significance of the growing national mental health issues, committed to a raft of new and expanded mental health reforms for the period 2011–2016. This represented increased expenditure, most of which was set aside exclusively for early intervention and prevention programs for youth at risk (HRSCHA, 2013). The AIHW estimates that nearly $7.5 billion is now spent annually in Australia on mental-health-related services. While a considerable sum by any standards, it is, however, a lot less than the amount spent on physical illnesses, despite the fact that mental illness affects one in five people. Perhaps we need to ask why governments continue to concentrate their funding on physical illnesses while acknowledging that mental health is a national priority.

Recovery versus rehabilitation

The following sums up the difference between rehabilitation and Recovery:

> *Rehabilitation refers to the services and technologies that are made available to disabled persons so that they may learn to adapt to their world. Recovery refers to the lived or real-life experience of persons as they accept and overcome the challenge of the disability. (Deegan, 1988)*

The aim of rehabilitation is the restoration of function and the minimisation of psychiatric disability. Rehabilitation does this through the development of strengths, restoration of hope, environmental modifications, enhancement of vocational potential and maximisation of social and recreational networks.

Recovery therefore forms the basis upon which rehabilitation services can be developed, but rehabilitation services should not be considered the only vehicle for Recovery. Rather, rehabilitation services are just one component in a framework that ensures hope, respect and pathways to community participation within a comprehensive service system that collectively works towards the individualised goal of Recovery.

Assisting Recovery

People who are recovering have described specific ways others have helped them. These include:

- *ventilation:* being able to converse with others; sharing concerns
- *reality testing:* having people assist in maintaining clear distinctions between reality and distortions of thought
- *material support:* helping with financial, housing and transport problems
- *social approval and integration:* receiving reassurance when people accept them and provide a sense of belonging
- *constancy:* associating with people they knew before they became unwell, connecting current identity with pre-hospital identity and giving roots to existence
- *motivation:* receiving encouragement to achieve higher levels of occupational and social functioning
- *modelling:* observing the behaviour of others and incorporating appropriate behaviour into their own lifestyle
- *symptom monitoring:* having others alert them to manifestations of symptoms
- *problem solving:* discussing problems and getting concrete, solution-focused feedback
- *empathic understanding:* being understood by people important to them
- *reciprocal relating:* becoming an equal partner, able to share and be of assistance to others
- ***insight**:* acquiring more complete and accurate understanding of themselves.

(Glover, 2012)

One of the initial processes that all nurses need to appreciate is the importance of gathering sound data and undertaking an assessment that provides enough information to establish a beginning path of action. Probably the most common of these, and one that all nurses should have a good understanding of and be competent in, is the mental state assessment.

ASSESSING PEOPLE FOR SIGNS OF MENTAL ILLNESS

MENTAL STATE ASSESSMENT

A **mental state assessment (MSA)** or examination (MSE) is a systematic clinical assessment that provides a picture of what a nurse observes during an interaction or interview with a person (see Box 50.4). The MSA is for that particular interaction only—a person's mental state is subject to change. Do you have the same thoughts, feelings and behaviours every day?

An MSA is not only undertaken by specialist mental health nurses or psychiatrists. Nurses use their skills to conduct an MSA in many healthcare settings. In fact, you are probably applying some of the elements of a mental state assessment already. When you first meet someone you 'assess' them, when you decide who to sit next to on the bus or train or in the movie theatre; when you see an old friend after a time apart, you check out what they are wearing, how they are behaving, what sort of things they are talking about, how their mood seems. All of these things are elements of a mental state assessment.

The MSA is just one part of the bigger picture. A more complete, holistic (biopsychosocial) assessment needs to be undertaken while being mindful of the context of the person's presentation and their history. Think of someone's life as a jigsaw—it is made up of many pieces. A full mental health or psychiatric assessment will include information about why a person is being seen, any significant mental health or medical history, current medications, drug or alcohol use, family history and current family circumstances, developmental history,

BOX 50.4 Components of a mental state assessment

Identifying information
Name
Age
Gender
Occupation
Cultural affiliation
Religion
Next of kin

Presenting problem
Why has the person presented?
What do they see as the problem?

Appearance
Hygiene
Grooming and dress
Posture
Eye contact
Scars/marks/tattoos/piercing/needle sites
Appearance versus stated age
Overall appearance

Behaviour
Tics/tremors
Calm
Agitated
Hyperactive
Rigidity
Facial movements
Unusual movements or gestures
Catatonia

Speech (assessment of the person's ability to communicate vocally)
Tone
Rapid/slow
Pressured
Loud/soft
Fluency (hesitant, mute)
Repetition

Attitude to interview
Cooperative
Hostile
Friendly
Combative
Aloof
Suspicious
Guarded
Apathetic
Distant

Thought processes
Blocking
Flight of ideas
Clang associations
Tangentiality
Loose associations
Circumstantiality
Echolalia
Word salad
Concrete thinking

Thought content
Delusions
Paranoia
Phobias
Magical thinking
Poverty of speech
Obsessions
Risk
- Self-harm
- To others
- To reputation
- Of dependence/ institutionalisation

Mood (Objective—the person's own view) = What does the person say?
Describe how the person identifies what they feel is happening in their own words; for example:
- Sad
- Elated
- Fearful
- Worried
- Angry
- Guilty
- Happy
- Hopeless
- Irritable
- Mixed (anxious and depressed)

Affect (subjective—the clinician's view) = What do you observe?
Is the person:
- Flat
- Blunted
- Diminished
- Appropriate
- Inappropriate

Perceptual disturbances
Hallucinations
- Visual
- Auditory
  - Command
  - Loud/soft
  - Commenting
  - Discussing
- Tactile
- Olfactory
- Gustatory

Illusions
Depersonalisation

Memory/cognition
Orientation (time, place and person)

Memory (ability to store, retain and recall information, both recent and remote)
General: alertness and cooperation
Attention: WORLD backwards and serial sevens
Language: naming and repetition
Calculation: division and subtraction
Abstraction: proverbs and similarities

Insight and judgment
Awareness of illness
- Do you consider that you are ill in any way?
- Why have you come into hospital?
- Do you have a physical or a mental illness?
- Are you suffering from a mental health problem? What is it?

Correct labelling of abnormality:
- You described several symptoms, namely . . .
- What is your explanation of these experiences?

Willingness to take treatment:
- How do you feel about being in hospital . . . coming to the clinic . . .?
- How do you feel about taking medication?
- Has the medication been helpful?
- Have any other treatments been helpful?
- What helps you remain well?

What supports do you have?

Impulse control
Can the person control urges of anger, laughter, self-harm?

their social situation and current level of functioning, including sleep, self-care, spirituality, diet and sexual health.

People often present for help as a result of the impact that their symptoms are having on their level of functioning. This frequently includes situations where things feel overwhelming such as when a person's mood is so low they wish they could die, or their **anxiety**—excessive worry about everyday life events with no obvious reasons for this worry—has become so great they cannot leave home without having a panic attack. Health practitioners, however, are often focused on the need for a diagnosis. While diagnosis can be important, the 'label' is not what the person is concerned with. People with a mental illness, like all of us,

generally just want their life to improve. The focus is different, but hopefully everyone is aiming for the same thing. The needs of the person seeking help should be at the forefront of all nursing care.

The process of assessment is in itself an intervention. This is because the nurse is spending time engaging with the person. Within an MSA there are formal terms and processes that we will now examine. A framework for conducting the assessment is also outlined. There are many assessment tools in use throughout Australia and New Zealand. The following outlines the main components. It is beholden upon the nurse conducting an MSA to make the person feel comfortable about expressing their thoughts and feelings without fear of negative judgment. A conversational approach, rather than a set of formal closed questions, is likely to make the person feel calmer and less anxious. This will result in greater rapport with the nurse and therefore better information. Nurses should use open-ended questions and be conscious of their own non-verbal responses and body language. People can tell if you have a genuine interest in them or are just 'going through the motions', and people with a mental illness are no less sensitive to this. Validating feelings is always helpful. Where possible, sit with the person and/or their family.

The assessment process cannot be rushed. Often people who have a mental illness feel anxious, afraid and vulnerable, and may fear that nurses will judge them negatively. The more patience and respect the nurse shows to the person and their family, the quicker the nurse will gather the information and the more accurate it will be. Accurate information is needed to form the basis of a comprehensive and holistic assessment.

TYPES OF MENTAL ILLNESS

The brevity of this chapter does not allow for an in-depth examination of the various types of mental illness. Broadly speaking, mental illness falls into two large groups—psychotic disorders and non-psychotic disorders.

Psychotic disorders

Psychosis describes a state in which a person's reasoning and thinking (**cognition**) are distorted, leading to a loss of contact with reality. Psychosis, like most mental illnesses, is episodic in nature. People who experience psychotic disorders are not out of touch with reality all the time. Instead, when someone is experiencing psychosis, they are having what is known as a psychotic episode. Such an occurrence describes the state of mind that the person is currently in.

A psychotic episode can occur as part of a number of different disorders—for example, schizophrenia; as part a drug-induced state; in affective illnesses such as depression or mania; and where there is an established physically defined brain disease or impairment, including brain tumours or even epilepsy.

A psychotic episode is a frightening and confusing event for the person experiencing it, and for their carers and friends. Approximately three in every 100 people will experience a psychotic episode at some time, and a psychotic episode usually first occurs between the ages of 15 and 35 years. Of this group, fewer than 4.5 people per 1000 will seek contact with specialised public mental health services. Sometimes a psychotic episode has a rapid onset (a few days), but often the onset of a psychotic episode is more gradual, over a period of weeks or months. When the onset is more gradual, individuals and their families will often report that things 'weren't quite right' for some time before the psychotic episode. A psychotic episode always requires treatment, and studies have shown that the earlier that treatment is obtained, the better the outcome for the person (healthdirect Australia, 2016).

The main distinguishing feature of a psychotic disorder is the person's loss of contact with reality. The person may not be aware of this, however. Two of the key symptoms that a nurse might observe in a person who is experiencing a psychotic disorder are delusions and hallucinations.

Delusions

Delusions are fixed false beliefs that are not a normal part of the person's cultural or religious environment and that cannot be altered by logic. A person who has a delusion is absolutely convinced that what they think is true. It cannot be argued with. A nurse who tries to dispel the delusion and argues with the person runs the risk of becoming incorporated into the delusion and being perceived as combative. Delusions are often complex and can be difficult to comprehend, especially when they incorporate very unusual beliefs. The authors have seen people with delusions who believe that they have chips inserted into their brains to track their thoughts and feelings, or that they were attached to a genetic line of descendants from gangsters located in spy organisations, or that they are working for prime ministers, ASIO or presidents. Delusions that involve the belief that the person is being followed, controlled or watched are common. Such beliefs can be very distressing and cause the person to act in ways they would not normally act. There are many different types of delusions. Some include:

- *delusions of grandeur:* beliefs of great importance or extraordinary powers or abilities (e.g. the person believes they are royalty, the president or Superwoman)
- *somatic delusions:* beliefs that something odd is happening to their body, that they have a disease or that part of their body is missing or has been changed in some way, despite medical evidence to the contrary (e.g. the person believes they have no stomach)
- *delusions of reference:* beliefs that the behaviour of others, events, objects or information such as television, songs or newspapers contain messages that are specifically meant for them (e.g. the person believes that car number plates send special messages)
- *delusions of persecution/paranoia:* beliefs of being pursued, watched, tracked or followed (e.g. the person believes that a government agency is following them).

Hallucinations

A **hallucination** is an alteration in perception. It occurs when a person experiences a sensation of something that is not objectively present. A person can experience hallucinations in any of the senses—auditory (hearing), visual (sight), olfactory (smell), tactile (touch) and gustatory (taste).

- *Auditory:* the most common type of hallucination is auditory and relates to hearing something that others cannot. The sounds (voices, music, tapping etc) can be 'inside' or outside their head. They are real to the person and should never be dismissed as a joke. What the person hears can be of any nature. Hearing voices can be pleasant but can also

be frightening, threatening or critical. The commentary may be about what the person is doing; voices that are telling them they are wrong or that they should be doing something else, or that they are worthless. On the other hand, the voices may be kind, comforting or neutral. Sometimes voices command a person to do something. The name given to this is *command hallucination*s and, depending on what the voices are commanding the person to do, they can pose a threat to the safety of the person or to others. It is very important that the nurse establishes what it is these particular voices are commanding the person to do. Refer back to the MSA and note that when assessing perceptual disturbances, nurses are encouraged to ascertain the content of the voices. Yet other voices may repeat the person's thoughts. Sometimes the person may hear two or more voices having a conversation about them, or several voices at once shouting or yelling at them. Sometimes it may not be a voice/s. People have described hearing an orchestra, birds, animals, drums and all manner of sounds. As with voices, these sounds are not imagined and brain-imaging studies suggest that voices arise from parts of the brain that are ordinarily involved in perceiving spoken speech.

- *Visual:* things or people are seen that cannot be seen by others. Hallucinations are not illusions. An accurate assessment (MSA) will ascertain the difference. (An **illusion** is a distortion of sensory perception and, therefore, reality. It is a misinterpretation of a true sensation and is shared by most people.)
- *Olfactory:* if a person experiences olfactory hallucinations, they smell things that others cannot. These smells may be good or bad and can have an effect on what the person eats.
- *Gustatory:* if a person experiences gustatory hallucinations, food may taste as if it is bad, contaminated or poisoned. If this is the case, the nurse has to be very skilful in order to get the person to have enough sustenance.
- *Tactile:* with this type of hallucination, sensations are experienced that have not actually occurred or are sensed differently from the way they usually are—for example, the person may feel as though spiders, bugs or ants are crawling over their skin, or that they are being touched by someone. Tactile hallucinations are said to be common in people who use the methamphetamine ice.

Types of psychotic disorders

DRUG-INDUCED PSYCHOSIS Using drugs, coming down from them, or withdrawing from certain drugs can precipitate psychotic symptoms. In particular, it is common to see people who have a drug-induced psychosis present with delusions and hallucinations. Sometimes these symptoms wane within a few days as the effects of the substances wear off, and the person may not experience another episode unless they continue to use drugs. However, for some people the effects can last for a long time, even after abstinence. If someone has an underlying vulnerability to a psychotic disorder such as schizophrenia, the psychotic episode can last much longer, and may be triggered by the initial substance use. Drugs such as amphetamines and ice are often linked to psychotic episodes. The link between cannabis (THC) and mental illness is becoming increasingly obvious.

ORGANIC PSYCHOSIS The importance of a medical clearance for possible physical causes of what may appear to be a mental health issue cannot be underestimated. Sometimes a physical injury or an illness that affects the brain can be the cause of an organic psychosis. Such conditions include brain tumours and infections, Alzheimer's disease, delirium, cardiovascular accident and some vitamin deficiencies. Treating the underlying physical problem is the main treatment.

BRIEF REACTIVE PSYCHOSIS A traumatic or very stressful event in someone's life, such as the death of a loved one, a physical or sexual assault, or a natural disaster, can cause a psychotic episode. During a brief reactive psychosis the symptoms can be severe and appear quickly. Symptoms can also rapidly abate.

DELUSIONAL DISORDER In a delusional disorder the main sign is a strong belief in something that is not true, with few if any hallucinations (see above for examples of delusions). People who have a delusional disorder may not experience any other symptoms of mental illness. If the delusions do not affect their lives as such, they can usually continue to manage their responsibilities, and their behaviour may not be very different from usual. Some delusions may not interfere with life as much as others. For example, a delusion that all women who have purple hair, wear green Versace jeans and are very tall are dangerous is not likely to cause alarm because meeting a person who fits this description is not very likely. However, if a person believes that men under 25 who drive white cars are following them, this is likely to have an impact on the person's life because it is highly probable they will encounter this scenario. The effect is dependent on the nature and content of the delusion.

SCHIZOPHRENIA **Schizophrenia** is a mental illness that affects approximately one person in every 100. People who live with this psychotic disorder often experience hallucinations, delusions and thought disorder (see Figure 50.6). The onset of schizophrenia may be rapid, with acute symptoms developing very quickly, or it may be slow and develop over many months or even years. Some people experience only one episode but for others it is a lifelong condition. First onset is usually in adolescence, but it can also occur for the first time in older people. Schizophrenia refers to a change in a person's mental function. A person who has schizophrenia does not have a 'split personality' although this is how this condition is often described, especially in the media. People with schizophrenia may however experience changes to their own personality as a result of disturbed perceptions and thoughts.

There are different types of schizophrenia but it normally has three phases:

1. *Prodromal phase:* early warning signs are present; something is 'not quite right'.
2. *Acute phase:* symptoms of a psychosis are observed, such as hallucinations and/or delusions and/or thought disorder.
3. *Recovery phase:* people return to their day-to-day lives, with the help of treatment.

FIGURE 50.6 ■ Some people with schizophrenia experience hallucinations and delusions

Source: © Triangle Images/Getty Images.

BIPOLAR AFFECTIVE DISORDER Bipolar affective disorder is the name given to what was previously known as manic depressive psychosis. Bipolar affective disorder aptly describes the recurrent episodes of 'up and down' mood swings. These can range from elation and excitement to sadness and depression. When people experience such extremes in mood it can be difficult for them to manage day-to-day responsibilities such as work, finances and family commitments. Episodes of mania (see Box 50.5) and episodes of depression (see Box 50.6) can last for a short time (days) or for a longer time (months). There are different types of bipolar disorder.

People who have bipolar disorder often experience psychotic episodes. The symptoms experienced usually match the person's current mood. If a person is depressed they might hear voices that tell them that they are horrible or no good, or direct the person to hurt themselves. A person who is manic, however, might believe that they are very special and can do remarkable things (delusions of grandeur).

Some of the famous people said to live/have lived with bipolar disorder include Adam Ant, Mark Twain, Catherine Zeta-Jones, Patty Duke, Andrew Johns, Vivien Leigh, Edgar Allen Poe, Nina Simone, Jean-Claude Van Damme, Sidney Sheldon, Sinéad O'Connor, Mel Gibson and Richard Dreyfuss.

BOX 50.5 Symptoms of mania

- An elated, happy mood or an angry, irritated mood
- Increased energy or activity levels
- Decreased sleep/less need for sleep/difficulty getting to sleep
- Increased talking, and talking louder and faster than usual
- More thoughts, and faster thinking than usual
- Overly ambitious plans and reckless behaviour (e.g. spending too much money or engaging in careless or potentially dangerous behaviour)
- Grandiose delusions

BOX 50.6 Symptoms of depression

- Sadness or depressed mood
- Lack of motivation and energy
- Changes in sleep patterns—sleeping more or less than usual
- Changes in appetite—eating more or less than usual
- Hopeless and helpless feelings
- Less interest in and less enjoyment from usually enjoyed activities
- Feelings of guilt and worthlessness
- Depressive delusions

PSYCHOTIC DEPRESSION People who are experiencing psychotic depression may have many of the same symptoms that people with bipolar disorder experience when they are depressed (see Figure 50.7). People who have psychotic depression, though, do not experience periods of elation or mania. When people have psychotic depression they tend to experience symptoms that match their mood; for example, they might hear voices that tell them to kill themselves, or voices that make fun of them, or they may experience delusions.

Non-psychotic disorders

Throughout life we have all experienced strong feelings of sadness, tension or fear. For some people, though, these feelings can become so troubling and overpowering that they have difficulty coping with everyday activities, even those that they have been successfully managing and coping with for years. The processes of going to work, enjoying leisure time and maintaining relationships become so intense that they are unable to carry them out.

FIGURE 50.7 ■ Highly successful individuals, like Andrew Johns the former NRL player, are not immune from depression. It is beholden upon the nurse conducting an MSA to make the person feel comfortable expressing their thoughts and feelings without fear of negative judgment

Source: © Jonathan Carroll/Australian Associated Press Pty Ltd.

Such all-consuming feelings are not often evident to others. For the person who is experiencing them, though, they can cause considerable personal distress and anguish. Such mental illnesses are labelled as non-psychotic illnesses. They are a common experience for many people and the list below demonstrates how widespread and prevalent these illnesses are. It is highly likely that someone with one of these illnesses will require care within the general nursing setting. Nurses themselves can also live with these types of illnesses every day. Remember, if mental illness affects one in five Australians, it is highly likely that someone you know, are related to, study with or work with has a mental health issue.

- Adjustment disorder
- Anxiety disorders
- Post-traumatic stress disorder
- Phobias
- Obsessive compulsive disorder
- Dissociative disorders
- Eating disorders:
  - Bulimia
  - Anorexia nervosa
- **Impulse control** disorders:
  - Pyromania
  - Kleptomania
  - Pathological gambling
- Mood disorders
- Sexual disorders:
  - Paedophilia
  - Necrophilia
  - Fetishism
- Sleep disorders:
  - Night terror disorder
  - Insomnia
  - Narcolepsy
- Sexual dysfunctions:
  - Gender identity disorder
  - Sexual aversion disorder
  - Premature ejaculation
- Somatoform disorders:
  - Hypochondriasis disorder
  - Pain disorder
- Substance disorders:
  - Substance abuse
  - Substance dependency
- Personality disorders:
  - Antisocial personality disorder
  - Narcissistic personality disorder
  - Borderline personality disorder.

Most non-psychotic illnesses can be effectively treated, usually with a combination of medication and psychotherapies. Treatment helps the person understand their illness and gain insight into what exacerbates their symptoms. It also assists with illness management and recovery.

The most common type of non-psychotic disorder is depression. Depression is so commonplace that the WHO (2012b) predicts that by the year 2020 it will be the second main contributor to the world's disease burden.

Depression

We have all felt sad or 'blue' at some time in our lives. This experience of having a low mood can result from something we have experienced: a loss, relationship difficulties, trauma or an event to which we can attribute feeling down. Sometimes, though, people experience low moods for no apparent reason at all. Such feelings are normal and are not cause for concern. However, feeling down can become an illness. Indications that a person may have a depressive illness include when a low mood becomes very severe and lasts for 2 weeks or more and when the low mood interferes with the person's ability to function.

Depression affects nearly all physical and psychological functions. A person with depression may have difficulty getting out of bed, not care for their ADLs, experience loss of appetite, have difficulty coping at work, have constipation (related to not eating; lack of mobility), have difficulty in concentrating and sleeping and suffer with memory loss. Many famous people are said to live/have lived with depression: Schumann, Virginia Woolf, Rachmaninov, Tchaikovsky, Adam Duritz, Billy Joel, Buzz Aldrin, Drew Carey, Kurt Cobain, Vincent van Gogh, Rodney Dangerfield, Winston Churchill, Sheryl Crow, Jeff Kennett and Rosie O'Donnell, to name but a few.

Signs of a depressed mood include:

- apathy and indifference
- lowered energy levels
- reduced motivation
- feelings of dejection and despondency
- lowered pain tolerance and new aches and pains
- reduced libido
- changes in appetite or weight
- reduced ability to control emotions (labile crying, anger outbursts)
- poor concentration and memory
- changes in sleep patterns
- reduced capacity to enjoy life—nothing is pleasurable
- suicidal thoughts.

The above list is not exhaustive and having one or two symptoms is not necessarily indicative of a depressive illness. The nurse needs to ascertain thoughts of self-harm which then need to be further explored so that risk can be properly assessed.

Treatments in mental health

Treatments for mental illness are as varied and diverse as the illnesses themselves but largely fall into two categories: physical treatments and psychological therapies.

Physical treatments

There are a number of types of physical treatments, the most common of which is medications.

MEDICATIONS One of the roles of a mental health nurse is to administer, monitor and evaluate drug therapy. Medications are used widely for many different types of mental health issues. Medications fall broadly into a number of categories, including antidepressants, anxiolytics, antipsychotics and

FIGURE 50.8 ■ Types of medications: *A*, an antidepressant, *B*, an antipsychotic

Sources: A, © Najlah Feanny/Corbis; *B*, © Chris Gallagher/Getty Images.

mood stabilisers (see Figure 50.8). The mental health nurse needs to engage with the person regarding their pharmacological treatment and provide psychoeducation with the aim of medication management.

BRAIN STIMULATION THERAPIES Brain stimulation therapies involve activating or touching the brain directly with electricity, magnets or implants. These therapies are usually focused on mood and anxiety-type disorders. Brain stimulation therapies include vagus nerve stimulation, repetitive transcranial magnetic stimulation, magnetic seizure therapy and deep brain stimulation. Some of these are relatively new and are currently considered experimental methods.

ELECTROCONVULSIVE THERAPY (ECT) is a type of brain stimulation therapy that is worthy of particular discussion because of its controversial past. It is considered particularly useful for someone who is suffering with severe depression or life-threatening mania. Before ECT is administered, the person is sedated with a general anaesthetic and given a muscle relaxant. This prevents jerking and movement during the procedure. As with all general anaesthetics the person's breathing, heart rate and blood pressure are closely monitored throughout the procedure.

Electrodes are placed on the person's temples, either bilaterally or unilaterally, and an electric current is applied through the brain. The person under anaesthesia does not feel this impulse, although a convulsion is induced. In contemporary practice, this convulsion is barely visible and lasts from a few seconds to usually less than 1 minute. This is in stark contrast to the early days of such treatment where the person often thrashed about violently on the procedure table. The most common side effects associated with ECT are headache, upset stomach and muscle aches. Some people may also experience memory problems, especially around the time of the treatment. People may also have trouble remembering information learned shortly after the procedure, but this difficulty usually disappears over the days and weeks following the end of an ECT course. It is possible that a person may have gaps in memory over the weeks during which he or she receives treatment. ECT is usually done in a course of treatments that are individualised for the person.

Psychological therapies

Psychological therapies can also contribute to the Recovery journey of a person who has a mental illness. The most important of these is the development and maintenance of a therapeutic relationship with the care coordinator. The importance of this relationship is the subject of many research papers written by mental health service users, their carers and clinicians.

COGNITIVE BEHAVIOURAL THERAPY (CBT) Along with a therapeutic relationship, CBT can also be useful. CBT aims to work closely with the person in order for them to see the links between their moods and their thinking. The objective is therefore to get the person to think in ways that are more positive.

INTERPERSONAL THERAPY (IPT) IPT aims to help the person identify current life factors that are leading them to experience a mental illness. IPT is founded upon the assumption that a person's vulnerabilities lead them to an illness. The aim is to strengthen the vulnerable parts of the person's personality.

NARRATIVE THERAPY (NT) NT places its main emphasis on a person's strengths. Narrative therapists listen closely to the person's life story and help them recognise times when they have solved problems with a positive outcome. Past situations and circumstances are thus learned from and built upon, ready to be used in future situations.

All therapies and treatments work best when the person is accepting of and seeking treatment for their mental health issues. See Box 50.7 for the SANE checklist to determine whether someone should see a mental health practitioner. Psychological therapies in particular will not work if the person does not trust the therapist. Once again, the importance of developing and maintaining a therapeutic relationship is at the fore.

BOX 50.7 A SANE checklist

Are you worried about someone whose behaviour has changed? If someone you know has become confused, avoids people or has developed strange ideas that are not shared by others, it is important they talk to a mental health nurse or doctor to get help. The reason for this change may be that they have an illness.

Checklist

Encourage someone to seek help if anything on this checklist describes how they feel or act:

- They stop talking to family and friends.
- They become afraid or suspicious for no reason.
- They sleep poorly or often are awake all night.
- They develop strange ideas.
- They hear voices no one else can hear.
- They feel they have special powers.
- They have difficulty concentrating.
- They say or write things that don't make sense.
- They abuse drugs or alcohol.

How to get help

- Encourage the person to see a doctor.
- Offer to go with them, as a support.
- Ask for a longer appointment so there is sufficient time to explain concerns.
- Suggest you write some notes together to help explain things to the doctor.
- If the person is reluctant to seek help, visit the doctor yourself to ask for advice.

CHAPTER HIGHLIGHTS

- Mental disorders account for an estimated 11% of the disease burden worldwide and this is projected to rise to 15% by the year 2020.
- In Australia, mental disorders are the third leading cause of overall disease burden, accounting for 13% of total burden and 27% of years lost due to disability.
- Mental illness represents the largest single cause of disability, accounting for almost 25% of the burden of non-fatal disease.
- It is imperative that all nurses have the skills and knowledge to provide safe and effective care for the large number of people affected by mental illness.
- The care that people who have a mental illness receive should be no less evidence based than the care that is provided to people who have a physical illness or injury.
- Nurses' attitudes towards people who have a mental illness should be respectful and non-judgmental; the nurse should show positive regard and be professional at all times.

CONCEPT CHECK

1 There are two main diagnostic criteria used internationally:
   1 DSN and ICD
   2 DSM and ICC
   3 DSM and ICD
   4 DMM and ICD

2 The American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders:*
   1 assists with diagnosis
   2 sets nursing priorities
   3 establishes appropriate treatment regimes
   4 ensures accurate epidemiological data is collected

3 Antonio has altered thought processes. An appropriate nursing response would be to:
   1 explore his delusions with him
   2 redirect his attention back to the here and now
   3 point out gently the way in which his thinking is altered by giving examples
   4 ensure you speak loudly enough so that he can hear you above his hallucinations

4 Bipolar disorder is:
   1 a non-reactive set of depressive symptoms
   2 a common disorder in young males
   3 a psychotic disorder
   4 a hereditary mental illness

5 People with bipolar disorder have:
   1 delusions and spatial disruption
   2 positive and negative symptoms
   3 mood swings
   4 hallucinations and thought blocking

6 Bipolar disorder is often written as:
   1 BD
   2 BPD
   3 BiPD
   4 BAD

7 Manic symptoms associated with bipolar disorder include:
   1 little eye contact
   2 speaking one's mind
   3 no time for conversations
   4 feeling really good

8 The following is not an example of a perceptual disturbance:
   1 delusion
   2 seeing things
   3 hearing voices
   4 feeling things on your skin

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CHAPTER 51

COMMUNITY CARE

JENNY DAY, ANN TAYLOR, SHARYN HUNTER, SARAH JEONG, ISABEL HIGGINS

KEY TERMS

biomedical model of health 1878
biopsychosocial model of health 1878
community 1873
consumer-directed care 1875
distal determinants 1881
health determinants 1878
health education 1884
health promotion 1884
intermediate determinants 1881
intersectorial 1875
primary care 1882
primary healthcare 1882
proximal determinants 1881
social determinants of health 1878
social model of health 1878
transdisciplinary 1882

LEARNING OUTCOMES

- Describe the background and contemporary context of community care in Australia.
- Differentiate the health models underpinning and influencing community care services in Australia.
- Compare individualist and structuralist–collectivist approaches to health promotion.
- Discuss the evidence for social determinants of health as factors that affect the health of communities.
- Differentiate between primary healthcare and primary care and identify their relationships to community care approaches. Explain the role and contribution of nurses in community care, including intersectorial partnerships.

CLINICAL COMPETENCIES

- Understand the health of communities in conjunction with the health of individuals.
- Apply the social determinants of health in relation to the health of individuals and communities.
- Collaborate with health and non-health professionals intersectorially to promote health, prevent illness/injury and restore health for individuals and communities.
- Utilise culturally safe approaches to care within primary healthcare and primary care services.
- Utilise partnership approaches to facilitate consumer-directed care.

In Australia, community care, or primary care, and public health are terms that are used to refer to organisations which provide health-related services in the community. These organisations aim to support and improve the health of communities by providing a universal point of access to the health system, promoting health and preventing illness, providing high-quality healthcare across the lifespan and ensuring continuity of care (Australian Productivity Commission, 2015). The roles nurses play in these organisations are complex and diverse, ranging from early childhood clinics, occupational health services, chronic disease management and palliative care to providing education on healthy lifestyle and prevention of disease or injury. Also important is the role of nurses in identifying and addressing social, environmental and political factors that impact on the health of individuals and communities as a whole (Schofield et al., 2011). The integral role of nurses in achieving the aim of community care is well acknowledged, as is the importance of nursing's future role in health care (Adrian, 2009; Department of Health and Ageing, 2009a; 2010).

This chapter provides an introduction to the concepts that are important to understanding community care services in Australia, as well as exemplars which illustrate nursing practice in this sector. While reading this chapter, it will be important to appreciate the interrelated nature of each concept in order to understand the contribution community care organisations make to the health of people and populations, and how community care services articulate with, but differ from, those provided by the acute care sector. Also important is remembering that community care organisations are characterised by their diversity, ranging from state- and federal-government-funded health services to church organisations, private businesses and volunteer organisations. Many of these organisations are not recognised as formal healthcare organisations but nevertheless play a vital role in developing and supporting community health in Australia (Australian Productivity Commission, 2015). In addition, key nursing concepts, such as culturally safe care and critical thinking, apply equally to nursing practice in community and hospital contexts.

To begin to understand how community care organisations work and the concepts that guide nurses working in this sector, we first need to clarify what is meant by the term community.

WHAT IS A COMMUNITY?

When considering communities and their health it is helpful to reflect on what is meant by the term **community** (see Figure 51.1). While this term has different meanings, two common meanings used in relation to health are 'community as locality' and 'community as relationships' (Taylor, Wilkinson & Cheers, 2008). Community as locality refers to the

FIGURE 51.1 ■ Community: places where people live and relate

Source: Jenny Day.

geographical location that people share with others (e.g. town, city or suburb), reflecting the role of people's environment and its resources on health (Heller cited in Taylor et al., 2008). Community as relationships refers to the human interactions and social connections that unite people (Heller cited in Taylor et al., 2008). Different communities of people therefore have different attributes, making it important not to assume communities are the same, perhaps thinking they have close-knit relationships or shared values and interests (Talbot & Verrinder, 2013). Also important is acknowledging that each community is likely to have subcommunities, such as school communities and inner- and outer-town communities, based on relationships, interests or localities, creating a complex and dynamic whole. Based on these meanings, the health of a community is a synthesis of people interacting or relating within their locality (McMurray, 2011).

Communities show qualities that indicate their health, many of which are detailed in Box 51.1. These qualities provide a guide for nurses who work in community organisations and reveal an orientation that considers health for the community rather than for each individual.

COMMUNITY CARE IN AUSTRALIA

Since 1973, when a formalised program to address the health of communities was established (McDonald & Smith, 2001; Coombs, 1985), seven goals have guided community health services: prevention, self-help, participation, integration, area responsibility, accountability and teamwork (Division of Health Services Research, 1975, cited in Owen et al., 2008). These goals continue to guide services; however, the current National Primary Health Care Strategic Framework (Department of Health and Ageing, 2013) promotes a stronger emphasis on the empowerment of individuals and communities to improve health literacy and healthy lifestyle choices. Also advocated is flexibility of health services to meet the needs and preferences of individuals and communities (Department of Health and Ageing, 2009b).

Community groups of interest to community services and the focus of their service activities are shown in Table 51.1. These groups and activities describe how different people within communities might need different community services.

To achieve the goals of community health and to meet the needs of groups within the population, community care services are provided in a variety of settings, by a range of people and organisations, and are funded through a range of different sources (Department of Health and Ageing, 2008). Complex networks of organisations, which together support the health of the community and its people, are therefore evident. This network involves interaction between community care organisations and those of the acute or hospital system, as well as interaction between formal health services and other organisations which address the social determinants of health or public health and safety. Figure 51.2 describes service network connections between general and specialist community services and acute care services.

The complex funding structure and areas of responsibility of Australia's health system is illustrated in Figure 51.3. This figure shows the web of health system services, including community and public health services. Note that health services comprise a mix of public and private sector involvement, with funding from the Australian Government, the state and territory governments and private funders. The total government expenditure on community and public health in 2011–2012 was $140.2 billion (Australian Institute of Health and Welfare (AIHW), 2014a, p. 47). This represents 9.5% of

BOX 51.1 Community alert: qualities of healthy communities

- Clean and safe physical environment
- Peace, equity and social justice
- Adequate access to food, water, shelter, income, safety, work and recreation for all
- Adequate access to healthcare services
- Opportunities for learning and skill development
- Strong, mutually supportive relationships and networks
- Workplaces that are supportive of individual and family wellbeing
- Wide participation of residents in decision making
- Strong local cultural and spiritual heritage
- Diverse and vital economy
- Protection of the natural environment
- Responsible use of resources to ensure long-term sustainability

Source: Ontario Healthy Communities Coalition (n.d.). *What makes a healthy community?* Retrieved from www.ohcc-ccso.ca/en/what-makes-a-healthy-community.

These principles have been adapted from the work of Trevor Hancock and Leonard Duhl. They are discussed in more detail, with references to earlier documents, in a report Dr Hancock prepared for the Senate Subcommittee on Population Health in March 2009 entitled *Act locally: Community-based population health promotion.*

TABLE 51.1 Community groups: needs and services

COMMUNITY GROUP	SERVICE ACTIVITY
Community members not at risk of ill health/injury	Health promotion
Community members at risk of developing a health problem	Prevention and early detection
Community members who present with a health problem	Assessment and investigation that does not require access to special technology
Community members with a confirmed problem	Community treatment
Community members with chronic consequences arising from a health problem	Continuing care

Source: K. Eagar et al. (2008). *Community health at the crossroads: Which way now? Final report of the NSW Community Health Review.* Wollongong: Centre for Health Service Development, University of Wollongong.

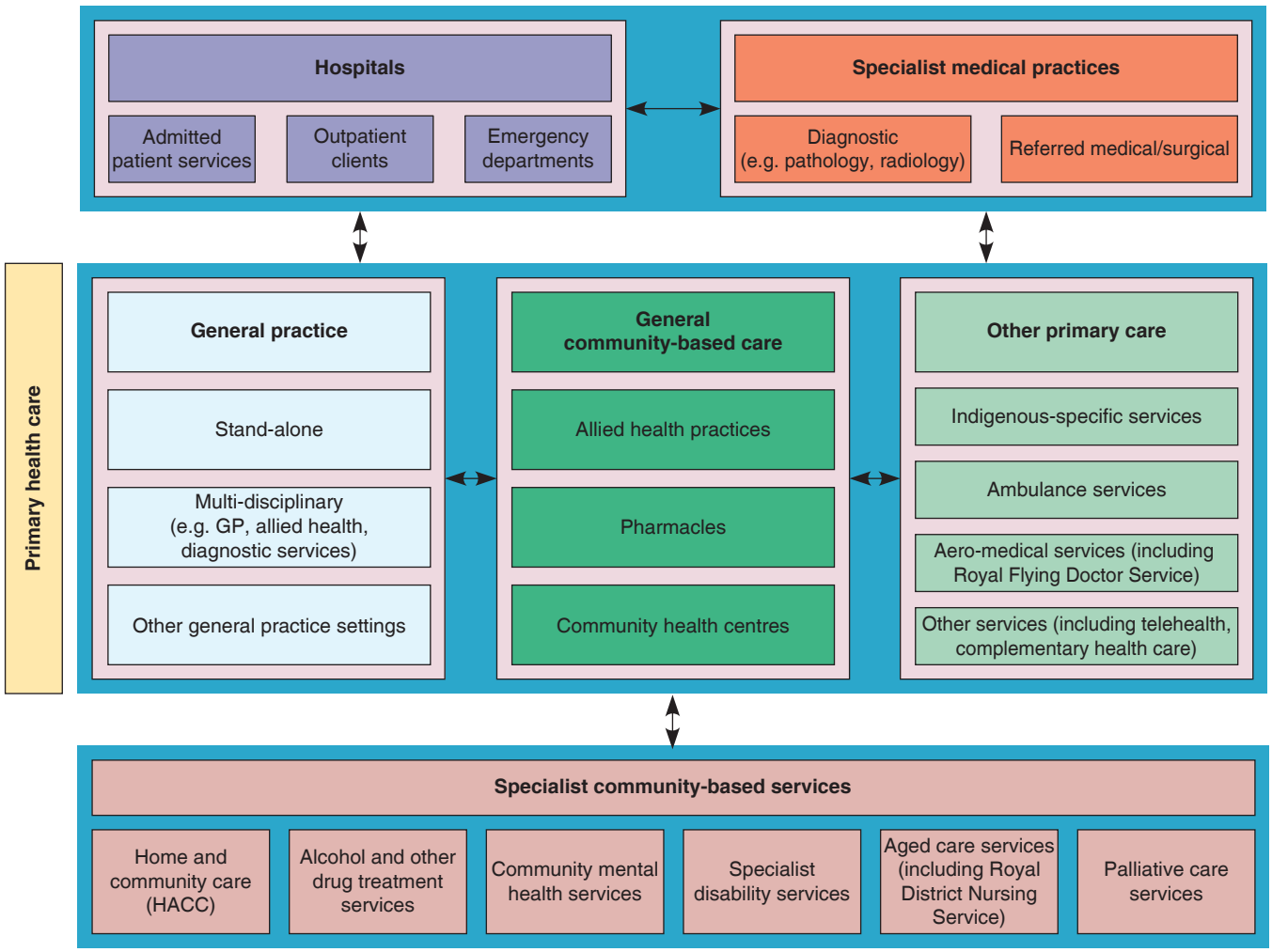

FIGURE 51.2 ■ Community health service network connections

Source: Australian Institute of Health and Welfare (AIHW) (2014a). *Australia's health 2014.* Chapter 8: Treating and preventing ill health. Retrieved from http://www.aihw.gov.au/australias-health/2014/preventing-ill-health/#t3. © Australian Institute of Health and Welfare.

gross domestic product (GDP). It is concerning that the cost of health has grown faster than the population has grown. It must be remembered, however, that formal health services are only one component of the network of organisations within communities which aim to promote health.

To be effective, community health services need to work in partnership with health and other services within the community to ensure the health of the population. They therefore work with many government and non-government organisations and services which are funded outside the health system, yet which make significant contributions to the health of the population, particularly disadvantaged groups (e.g. people who are homeless). This is known as **intersectorial** collaboration. Examples of these organisations include religious organisations; specific interest groups such as Diabetes Australia, Australian Breastfeeding Association and SIDS and Kids; and income-support organisations. Some other partners that are needed to develop and sustain healthy communities are listed in Box 51.2. Importantly, however, there is increasing emphasis on working in partnership with the people, or consumers, who use community services. This shift responds to consumers' calls for models of care which are consumer driven and oriented and is reflected in the introduction of services underpinned by **consumer-directed care** (CDC) philosophies. These CDC services aim to increase consumer power/control over care, increase consumer satisfaction with care and improve consumer health outcomes—both functional and social (Low et al., 2012). In Australia, community aged care CDC refers to enhancing service users' autonomy by enabling and optimising consumer goal setting and control over the care they receive and the providers who deliver their services; the opposite of agency directed or controlled care which has operated in the past (Low et al., 2012).

Community-based programs based on the CDC model have been available in the Australian disability sector for some time. These have stemmed from disability self-advocacy and empowerment movements. They have developed considerable support from consumers with a disability (Low et al., 2012; Aged and

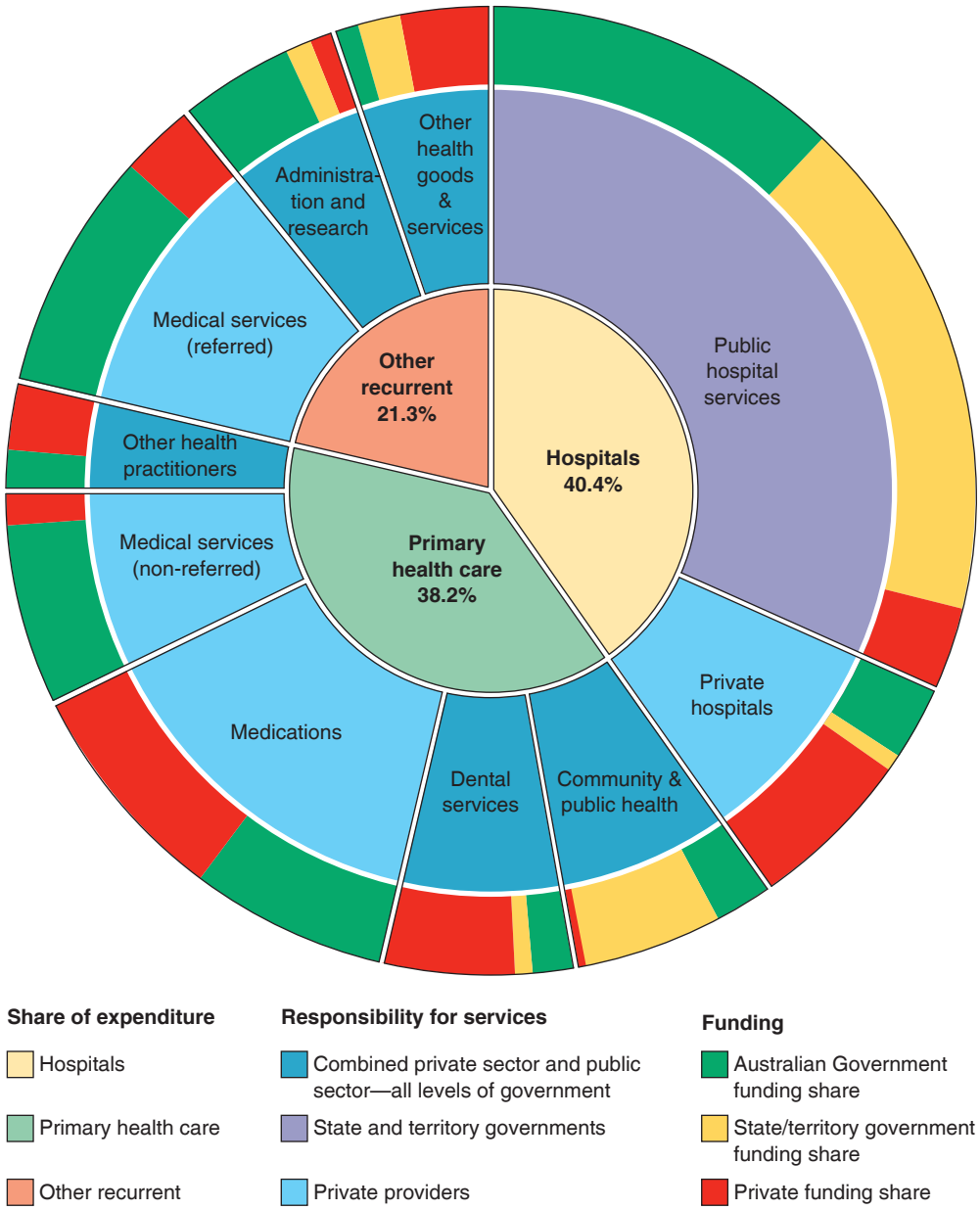

FIGURE 51.3 ■ Australian health services—funding and responsibility

Source: Australian Institute of Health and Welfare (AIHW) (2014a). *Australia's health 2014*. Australia's health series no 14. Cat no. AUS 178. Canberra: AIHW, p. 34. Reproduced with permission.

Community Services Australia (ACSA), 2008). More recently, CDC has been introduced to some community-based services for older people, aiming to deliver increased consumer engagement, power over the design of care packages and the direction of service arrangements (with a corresponding increase in consumer choice), responsibility for making decisions and managing the outcome of service provision (Low et al., 2012). The change that this approach can make is illustrated by Anna's story in Box 51.3.

Community care initiatives based on CDC have similarities with acute health care initiatives based on Standard 2: Partnering with Consumers, as detailed in the Australian National Safety and Quality Health Service Standards (Australian Commission on Safety and Quality in Health Care (ACSQHC), 2012), depending on where consumer involvement fits along the continuum of participation. However, community-based aged care and disability service CDC extend consumer choice, control and responsibility further, placing care outcomes in the hands of consumers by enabling consumer decision making regarding care goals, which services are provided, who provides them and how each consumer's individual service budget is spent (Low et al., 2012).

The role and contribution of non-health organisations and the services they provide are important. Many of the major

BOX 51.2 Possible partners in the healthcare continuum

Community health partners include, but are not limited to, the following:

Health promotion
Public health units, local government, schools, other state government departments, non-government organisations (NGOs), community groups and consumers.

Prevention and early detection
Schools, GPs, Nurse Practitioners, other state government departments, NGOs, community groups and consumers.

Assessment and investigation
GPs, Nurse Practitioners, allied health professionals, community nurses (general, child and family, and mental health), schools, other state government departments, Commonwealth-funded services such as aged care assessment teams (ACATs), NGOs and consumers.

Community treatment
GPs, practice nurses, Nurse Practitioners, allied health professionals, community nurses (general, child and family, and mental health), pharmacies, NGOs and consumers.

Continuing care
GPs, practice nurses, Nurse Practitioners, hospitals, other state government departments (particularly ageing, disability and home care, community services, housing and police), home and community care (HACC) services and Commonwealth-funded services such as community aged care packages (CACPs), NGOs/charities and consumers.

Source: Adapted from *Community health at the crossroads: Which way now? Final report of the NSW Community Health Review* by K. Eagar et al. (2008). Centre for Health Service Development, University of Wollongong, p. 7.

issues for the future attainment of community health involve strategies that promote effective links within and beyond the services of the health system, so that a reorientation of health services and expenditure to primary health can be achieved (Adrian, 2009; Eagar, et al., 2008). Also important is recognition that people within communities will move between community organisations and acute care services at some time in their life, further highlighting the need for partnerships between services in each setting and the contribution each makes to the health of individuals and communities across the lifespan. While community organisations and the services they deliver are important, they cannot function without each other to achieve health for people and communities.

The complex nature of the community health system is also characterised by a tension between providing a comprehensive, participatory and health-promoting approach, and a need to provide within a community direct care and medical interventions that are efficient and sustainable (Nesbit & Allen, 2011). Compounding this complexity is a view that community care services should provide the full range of services from prevention to palliation where people live, and at the same time cater to the needs of hospitals under stress from the impact of factors such as the increasing incidence of chronic diseases and their complications (e.g. diabetes and respiratory diseases) (Eagar et al., 2008).

The model of health embraced by community organisations is reflected in the types of services they provide. The following section discusses different models of health and raises implications for how health is addressed, with a particular focus on community health.

MODELS OF HEALTH AND COMMUNITY CARE

Each community organisation is based on a model of health which influences how healthcare is defined and provided. It also affects when and how the factors that affect health are addressed. In the following sections three models of health are

BOX 51.3 Consumer-directed care in practice

When Anna accessed a CDC-based home care package the focus of her services changed. She was able to identify her own needs and goals, including social isolation. Together with her case manager, Anna decided that involvement in a Lifebook project—a short biography and digital photo book—while receiving community visits from community volunteers might be a good option to increase her social interaction.

Initially, Anna was reluctant to have her private life story divulged to anyone. However, over the course of the project she developed a rapport with and trusted the volunteers who visited. When her completed Lifebook was given to her, Anna decided she would present her Lifebook. She spoke with clarity, expressing for the first time without tears that she did not wish her husband back to suffer more; he would have been 92. She had cared for him at home and done all she could. It seemed to those present that Anna was not just telling her life story, but engaging in a process that enabled her to name and share her grief.

Through the support she receives with her CDC package, Anna has not only reduced her social isolation but also her inner isolation. Anna is now ready to engage more fully with the life she is living.

Source: Adapted from Anna Bermarija, HomecareToday website. *Consumer stories*. Retrieved from www.homecaretoday.org.au/consumer/consumer-stories.

discussed and presented sequentially, representing a continuum which illustrates a move from medical to social orientations to health, and individual to population health concerns. While there are many other models of health, those presented here are a useful introduction to community organisations and the services they provide. The use of different models across health organisations does, however, mean that there can be tensions around the focus of healthcare for individuals, the interventions used and how care is ultimately delivered. In community care, the biomedical and social models of health are often integrated (Talbot & Verrinder, 2013).

The biomedical model of health

The **biomedical model of health**, also known as the medical model, defines health as the absence of disease (Germov, 2014). In this model there is a focus on 'objective specific findings that are interpreted as causal factors of a disease or disorder that needs to be eliminated by medical interventions to cure the patient' (Lundstrom, 2008, p. 393). Health is primarily considered to be an individual responsibility determined by biology and poor lifestyle choices (Engel, 1977; George, 1998). In services that prioritise this model, the psychological and social dimensions of health are not priority concerns for organisations or practitioners and may be overlooked. In contrast, in community and primary care settings the psychological and social dimensions of health are always important considerations.

This model of health is most evident in Australian acute care services, and those community care services which provide primary care (see discussion of primary care and primary healthcare later in this chapter), and is usually the main model used in general medical practice (Adrian, 2009). It is argued that community care services need to move beyond this model and embrace psychological and social dimensions of health as important considerations (Adrian, 2009; Keleher, Parker et al., 2007; World Health Organization (WHO), 2012).

As a reaction to the dominance of biomedicine and its orientation to health, a biopsychosocial movement has developed (Engel, 1977). This is a modification of the biomedical model which incorporates the concept of holistic wellbeing. It can be seen as an intermediate or middle-of-the-road model which lies between the biomedical and social models of health.

The biopsychosocial model of health

The **biopsychosocial model** defines health as holistic wellbeing within an individual (Taylor, 2011). It argues that a focus on disease is too narrow, and that health services need to include psychological and social functioning and social and personal resources, as well as physical capacities (Engel, 1977; Taylor, 2011). In this model, health is a dynamic concept influenced by multiple interacting factors that change over time. It reasserts a connection between mind and body, seeing the individual's mental state as influencing biological processes and thus susceptibility to disease (Engel, 1977; Taylor, 2011). However, it shares many key assumptions and characteristics of the biomedical model, including the focus on individual diagnosis and treatment, so that it is individualistic even if it includes emotional and social factors.

The social model of health

The **social model of health**, also referred to as new public health or the social ecological model (Richmond & Germov, 2014), sees health as multifaceted, with a focus on social rather than biological determinants of health. This model emphasises health equity and prevention of illness or injury, as well as collaboration and empowerment (Talbot & Verrinder, 2013).

> *The social model locates people in social contexts, conceptualises the physical environment as socially organised, and understands ill health as a process of interaction between people and their environments. (Broom cited in Germov, 2014, p. 16)*

The social model focuses on the interrelationship between human interaction, social organisation and natural environment and sees health as a resource within individuals, communities and the population as a whole. It underpins many health policies on a national and global scale. For instance the World Health Organization (WHO) Commission on Social Determinants of Health's 2008 report 'Closing the gap in a generation: Health equity through action on the social determinants of health' (2008b), argues that the 'high burden of illness . . . arises in large part because of the conditions in which people are born, grow, live, work and age—conditions that together provide the freedom people need to live lives they value' (WHO, 2008b). In the Commission's view the biomedical approach, while important, downplays the significance of influences on health that lie beyond the health sector—otherwise known as social determinants of health (AIHW, 2014a; Keleher & Murphy, 2004). Also ignored in the biomedical approach are values, vested interests, politics and context (Baum, 2008a; Sax, 1990).

The social model of health makes a link between 'private troubles', such as obesity, diabetes or unemployment, and 'public issues', such as transport options, food availability, government policy and globalisation (Mills, 1959 cited in Germov, 2014, p. 7). This alerts us to the fact that individuals often share problems, including health problems. Problems then may have a common cause, which may be solved through collective action. Health and illness, in this view, are not simply an individual concern.

In the following section the social determinants of health (those that align with health viewed through the social model of health) are discussed.

SOCIAL DETERMINANTS AND COMMUNITY HEALTH

The AIHW (2014a) calls the factors that affect the health of people '**health determinants**'. There is a growing body of evidence which shows that health determinants are the key to understanding the health of a community, promoting health and providing healthcare (Baum, 2008b; Wilkinson & Marmot, 2003). In particular, the **social determinants of health** are central to understanding the health of a community and community organisations which embrace the social model of health, discussed earlier in the chapter. In the social model, the focus is on social rather than biological health determinants.

Social determinants of health '. . . are the conditions in which people are born, grow, live, work and age, and include the health system. These circumstances are shaped by the distribution of money, power and resources at global, national and local levels' (WHO, 2012). The social determinants are increasingly recognised as the key to the health of people and communities because of the evidence that social conditions, specifically where people are born, live and work, determine people's chances of good health and a long life (WHO, 2012). It is interesting to note that despite the increasing levels of affluence in modern communities, people who have less still have poorer health (Wilkinson & Marmot, 2003). Figure 51.4 shows that in most age groups Australians with the lowest socioeconomic status die at a higher rate than Australians with fewer disadvantages. These differences are explained by the social determinants approach.

There are several ways of thinking about social determinants of health (Cannon, 2008). For instance, Wilkinson and Marmot (2003) refer to a list of 10 social determinants (see Box 51.4), whereas others have developed models to describe the social processes and elements that influence health. An example of one of these models is shown in Figure 51.5. All such models attempt to map a number of common and interconnected dimensions which operate as an intricate web of influence on people and their lives, acting on individuals and the population as a whole (Hetzel et al., 2004).

Early life factors

Early life factors influence a person's experience of health. For example, an adult's health is adversely influenced by physiological or emotional deprivation in early childhood and/or before birth (Wilkinson & Marmot, 2003). When a child is emotionally deprived, brain development can be adversely affected which in turn can lead to low levels of educational attainment, problem behaviour and social exclusion in later life (Cannon, 2008). Physical deprivation such as lack of nourishment in a child is associated with decreased development and function of major organs, which increases the risk of ill health in later life (Wilkinson & Marmot, 2003).

Some models include genetics as a social determinant of health because social factors interact with each other and with the individual's genetic composition. Others exclude genetics on the grounds that even though a person's genetic susceptibility to disease produces ill health, this health change is not a product of the social environment.

BOX 51.4 WHO social determinants of health

- Social gradient
- Stress
- Early life
- Social exclusion
- Work
- Unemployment
- Social support
- Addiction
- Food
- Transport

Source: R. Wilkinson & M. Marmot (2003). *Social determinants of health: The solid facts* (2nd ed.). Denmark: WHO. Retrieved from www.euro.who.int/__data/assets/pdf_file/0005/98438/e81384.pdf.

Income and socioeconomic conditions

Income and socioeconomic conditions are one of the key social determinants of health (Hetzel et al., 2004). People with higher incomes are often healthier and live longer lives than people who have middle and lower incomes (AIHW, 2014a; Marmot, 2002; Wilkinson & Marmot, 2003). Health and wellbeing

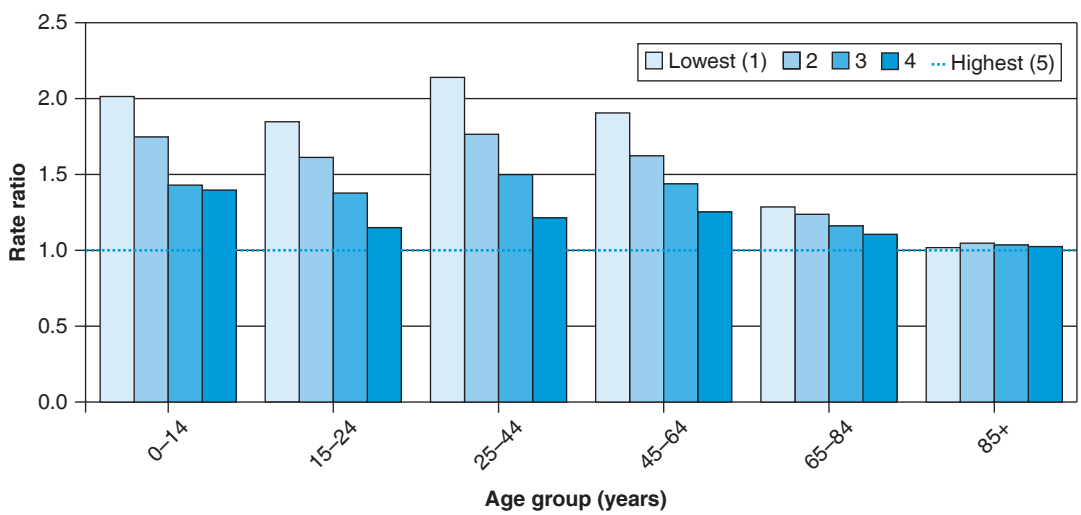

FIGURE 51.4 ■ Comparison of age-standardised mortality rates across socioeconomic groups, by age group, 2009–2011

Source: AIHW (2014b). *Mortality inequalities in Australia 2009–2011.* Summary Bulletin 124. Retrieved from www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=60129548364. Reproduced with permission.

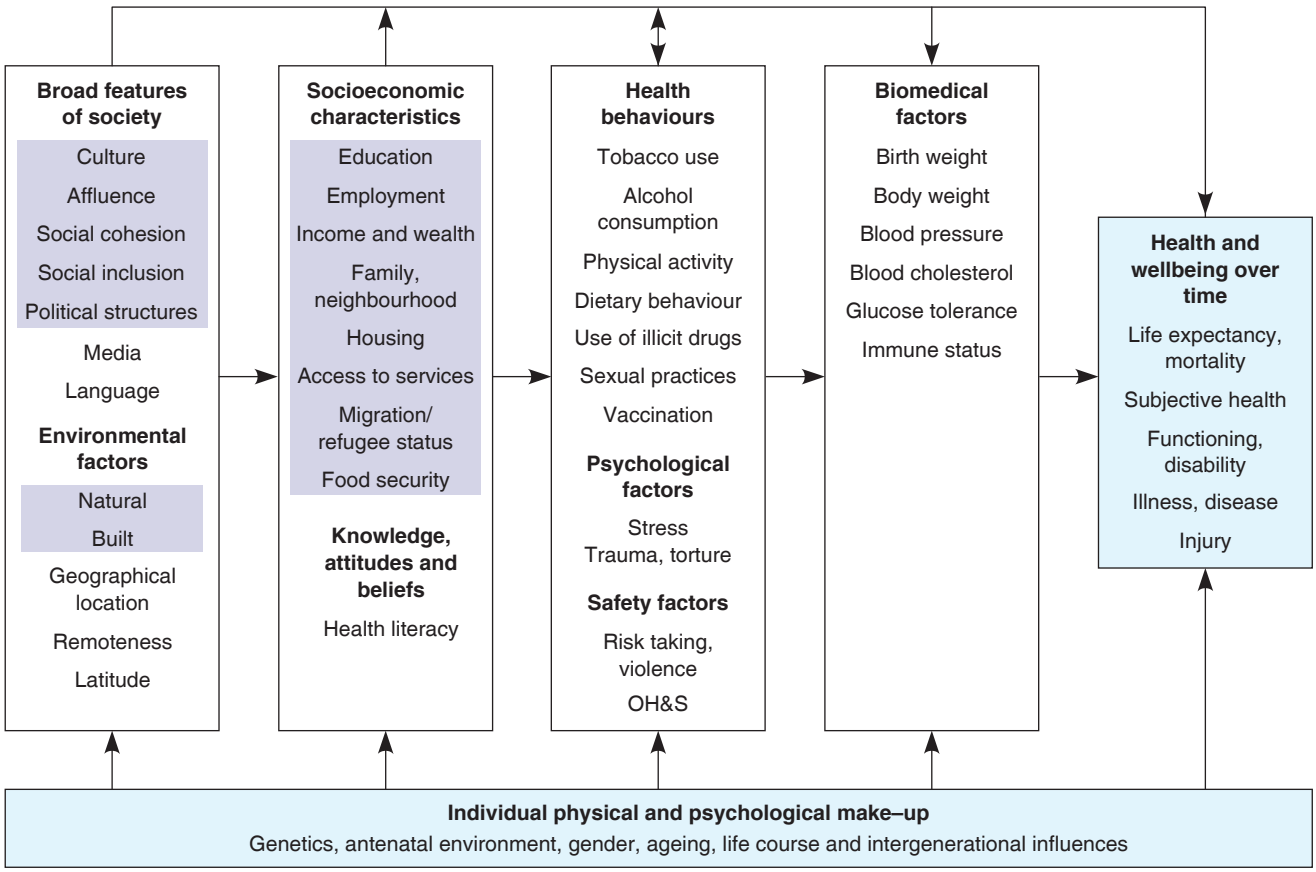

FIGURE 51.5 ■ A framework for the determinants of health. Purple shading highlights selected social determinants of health

Source: AIHW (2014a). *Australia's health 2014.* Australia's health series no. 14. Cat. no. AUS 178, Figure 1.1. Canberra: AIHW.

therefore improve as people's income and social position move upwards—a concept known as the social gradient (Hetzel et al., 2004). The more people live in difficult financial and social circumstances, the greater the ongoing wear and tear they endure, and the less chance they have of continuing to healthy older age (Wilkinson & Marmot, 2003). Income and social conditions act in relation to one another and, at times, it is not only a lack of material resources that affects health but also the social (dis)advantages associated with these conditions (Hetzel et al., 2004).

Living conditions

Living conditions have a significant impact on the experience of health by individuals and communities. The following are important aspects of people's living conditions:

- affordability of housing
- degree of overcrowding in housing
- design of cities
- liveability of communities outside major metropolitan areas
- degree of pollution—air and noise
- availability of transport options
- availability and pricing of nutritious food
- exposure to marketing of nutrient-poor foods
- affordability of utilities (e.g. electricity and water) (Cannon, 2008; Wilkinson & Marmot, 2003).

The cost of living is increasing in Australia, particularly for housing, food and utilities (Cannon, 2008). As a result, people reliant on government benefits and on low incomes are finding their living conditions being eroded and are at an increased risk of ill health.

Other factors

Levels of education, health literacy, behaviours and lifestyle factors are intrinsically linked to health. Education provides the basis for employment and generating an income. Education also enables the person to engage with information about health and to take the appropriate actions to maintain their health (Cannon, 2008). This engagement is referred to as health literacy. When a person does not have a sound level of education they are more likely to be health illiterate. Education and health literacy also influence a person's behaviours and lifestyle. With good levels of education and health literacy there is an increased likelihood that behaviours or lifestyle factors that adversely impact on health are not disregarded (Cannon, 2008). A person's low social position has also been shown to be associated with unhealthy behaviour. Regular tobacco use, poor food choices and low exercise levels are significantly greater in the lowest socioeconomic groups in Australia (AIHW, 2014a; Friel, 2009, p. 3).

The equitable distribution of resources, such as health services, is another key social determinant of health (WHO, 2008a). Access to and use of health services influences the

health of a person and a community. Comprehensive accessible healthcare should be available to everyone in the Australian community on the basis of need, not the ability to pay. However, access to health services is affected by many factors including socioeconomic status, geographical location, cultural background and intellectual or other disabilities. While most Australians benefit from our universal healthcare system, equity of access to healthcare remains an issue for immigrants, asylum seekers, refugees and Indigenous Australians (Adrian, 2009; Cannon, 2008). For example, access to health services is lower for Indigenous people than non-Indigenous people because of proximity, availability, cultural appropriateness, transport availability, health insurance, health services affordability and proficiency in English (AIHW, 2014a; Thomson, 2004). The relationship between socioeconomic status and access to health services, and ultimately to health, is well established. Indigenous Australians who have low socioeconomic status have reduced access to nutritious food, education and healthcare (AIHW, 2014a). They also live in risky environments and participate in unhealthy behaviours. All these social factors interact and contribute to poor health.

Racism, discrimination and culture are also known factors that affect the health of people and communities, as these influence basic levels of security, safety, hygiene, housing, nourishment, knowledge, employment, medical care, community support and social participation (Paradies, Harris & Anderson, 2008). Henry, Houston & Mooney (2004) reported that racism is present in the Australian healthcare system; for example, differences in treatment regimens, inadequate funding to reduce language barriers and a lack of understanding about the different constructs of health for Indigenous people. It is important to note that Australians from culturally and linguistically diverse (CALD) backgrounds also experience racism and discrimination, which contributes to their ill health. Racism and discrimination place significant pressure on people by creating barriers to education, employment and culturally appropriate and specific healthcare (Cannon, 2008; Hetzel et al., 2004). Many people are culturally, socially and economically disadvantaged simply because of cultural difference. Cultural difference may place people at increased risk of disease for a range of reasons including identity, misunderstanding and perceived threats to personal safety.

The concept of cultural safety (see Chapter 1) is an attempt to respond to many of these factors. Cultural safety is concerned with developing ways of addressing the 'social, [economic], political, historical and [often] emotional reasons' for illness and disease in marginalised and Indigenous cultural communities (Papps & Ramsden, 1996, p. 492). Practising cultural safety means ensuring quality in healthcare through culturally appropriate communication and ensuring equity and access to health services. It implores us to reflect on our attitudes and values, respect difference, recognise power differentials in relationships and act upon these. The idea of cultural safety 'assumes that each healthcare relationship between a professional and a consumer is unique, power-laden and culturally dyadic' (Kearns & Dyck in Papps & Ramsden, 1996, p. 494). The tenets of cultural safety help people to feel safe; there is no 'assault, challenge or denial of . . . identity' (Williams, 1999, p. 213). Cultural safety is about respect, shared meaning, knowledge and experience.

Social interaction and support at an individual and community level increase a sense of belonging and social connectedness that has been linked to positive physical and psychological health (Cannon, 2008; Labonte, 1997). Social exclusion leads to loss or lack of connection to the community in which a person lives. This exclusion is linked to psychological stress, depression, smoking, use of illicit drugs, and alcohol misuse and dependence (AIHW, 2010). Therefore, when engagement and interaction between individuals and communities is lacking, people experience ill health (Cannon, 2008; Hetzel et al., 2004). To facilitate interaction and engagement, health services need to embrace body, mind, spirit, land, environment, culture, customs and socioeconomic status, and be accessible to all individuals and communities as close as possible to where they live (Adrian, 2009).

Levels of determinants and interventions

Some social determinants of health have a more direct effect on health than others and hence they can be viewed on a continuum from proximal to distal (Keleher & MacDougall, 2015). **Distal determinants** of health tend to be stable and concern historical, national, institutional, political, legal and cultural factors; for example, anti-discrimination law, changes in taxation or changes to the provisions of government income support. The influence of distal determinants is often mediated through more proximal determinants (Keleher & MacDougall, 2015). **Intermediate determinants** concern community infrastructure, personal wealth or access to resources, and natural, physical and built environments. This level also includes access to healthcare and health systems (Keleher & MacDougall, 2015). **Proximal determinants** have a more direct impact on health, and include lifestyle and behavioural factors as well as underlying health conditions (Keleher & MacDougall, 2015).

Different community organisations address the determinants of health in different ways. Health promotion based on the social model of health focuses on distal or upstream factors and seeks to address working conditions or the effects of rapid social change. Examples of this include campaigns for legislation on food labelling, changes in environmental legislation such as those that have banned smoking from public places or, on a smaller scale, the introduction of harm-reduction strategies, such as needle-exchange programs and early intervention programs for families. Health promotion based on the biomedical model concentrates on proximal or downstream factors such as screening for high cholesterol levels and treating people with lifestyle changes or drugs. Organisations using the biopsychosocial model, the middle-of-the-road model of health that includes psychological and social factors, often focus prevention on intermediate factors or midstream factors.

PRIMARY HEALTHCARE AND PRIMARY CARE

Community organisations use approaches to care which complement the model and definition of health and orientation

to social determinants that has been embraced. Two common approaches to community care in Australia are **primary healthcare** and **primary care**. Each of these approaches describes in more detail how health is achieved for individuals and communities through the activities of community organisations. It is important to note, however, that while the terms 'primary care' and 'primary healthcare' are used widely and are at times interchangeable, primary care and primary healthcare approaches reflect different views of health and service provision (Adrian, 2009; Keleher, 2001). Calling 'primary care' 'primary healthcare' is therefore like calling an apple an orange and can understate the contribution services outside those of the health system (e.g. local councils and churches) make to the health of individuals and communities (Adrian, 2009). It is also important to remember that the underpinning beliefs about health that guide community services may be different from those that underpin medical and surgical or acute hospital services.

Primary healthcare

The philosophy of primary healthcare originates from the WHO. As an agency of the United Nations, the WHO was established to address international health issues and aims to achieve worldwide health (Talbot & Verrinder, 2013). In 1978, 134 nations attended the WHO International Conference on Primary Health Care as a response to growing concern about the world's health in terms of its people and healthcare systems (Talbot & Verrinder, 2013; WHO, 2008a). This conference was marked by the Declaration of Alma-Ata, a declaration that articulates primary healthcare as the guiding philosophy for health development across the world and as a vision for the future (Nesbit & Allen, 2011; Talbot & Verrinder, 2013; WHO, 2008a). Improved health outcomes, reduced costs and a reduction in inequalities have all been associated with having a strong primary healthcare system (Macinko, Starfield & Shi, 2003).

Primary healthcare is intended to be a comprehensive approach to health which forms the nucleus of the health system and the social and economic development of the community (WHO, 1978). This approach to health challenges the prevailing focus on finding a cure, a focus characteristic of current health systems, and calls for a focus on anticipating and preventing health problems (Nesbit & Allen, 2011). It reaffirms that health and wellbeing are dependent on a broad range of interrelated social factors (e.g. socioeconomic, cultural and political environment) and that health is a fundamental human right (Talbot & Verrinder, 2013; WHO, 2008a).

Primary healthcare is based on the social model of health and an understanding of the social determinants of health, both discussed previously (Keleher, 2001). It is defined by the WHO as:

> *essential health care based on practical, scientifically sound and socially acceptable methods and technology . . . made universally accessible to individuals and families in the community through their full participation and at a cost that the community and country can afford to maintain at every stage of their development in the spirit of self-reliance and self-determination. (WHO, 1978, p. 1)*

The goal of primary healthcare, underpinned by the principles of social justice, empowerment/community participation and equity, is 'Health for All' (Talbot & Verrinder, 2013; WHO, 1978). It advocates community services that promote community participation, and ensures access to health-related services for all people and the provision of services at a cost that is affordable and as close as possible to where people live (WHO, 1978). The philosophy of primary healthcare is also underpinned by the tenets of cultural safety. Guided by a primary healthcare philosophy, community organisations work in partnership with people and communities to enable and empower change that is local, affordable and sustainable (Talbot & Verrinder, 2013). The focus is on addressing the root cause of health problems or the social determinants of health experienced by people and communities, reducing the effects of disadvantage and health inequality (Keleher, 2001; Talbot & Verrinder, 2013). The focus of primary healthcare is broad and is therefore at times also referred to as comprehensive primary healthcare. The characteristics of primary care and primary healthcare are listed in Table 51.2.

To practise in a way consistent with primary healthcare, community organisations need to focus on addressing social determinants of health (Adrian, 2009) and to encompass strategies that address the underpinning principles noted previously, including cultural safety. The minimum activities for primary healthcare described by the WHO (1978) are listed in Box 51.5. Intersectorality, a strategy to address social determinants of health and structural barriers to health, refers to collaborative **transdisciplinary** partnerships, with the needs of the community dictating which are the most appropriate services and service relationships (Adrian, 2009). Transdisciplinary care goes beyond past models of multidisciplinary or interprofessional care and facilitates intersectorial relationships which value expertise in areas beyond those addressed by traditional health disciplines. This might include partnerships with services that provide education, transport or income support, reflecting an understanding of the social determinants of health. These approaches are central to primary healthcare and have been identified as key processes for closing the gap between the health of Indigenous and non-Indigenous people in Australia (Adrian, 2009). An exemplar of an Australian community service based on the philosophy of primary healthcare is detailed later in this chapter.

Primary care

While primary healthcare has been advocated by the WHO (1978) as a way to restore the health of communities across the world, another approach has evolved in developed countries such as Australia. This alternative approach returns the focus of health services to the absence of disease (Awofeso, 2004; Cueto, 2004) and to the provision of a first point of contact and entry into the health system (Cueto, 2005; Keleher, 2001). It is known as primary care or selective primary healthcare, reflecting a more selective view of health and service orientation than primary healthcare as advocated by the WHO (Nesbit & Allen, 2011). Although there has been considerable focus on primary care services provided by general medical practitioners in Australia (Australian Productivity Commission, 2015), primary care is also practised in Australian communities by other health professionals

TABLE 51.2 Characteristics of primary care and primary healthcare

CHARACTERISTIC	PRIMARY CARE	PRIMARY HEALTHCARE
Synonym	Selective primary healthcare	Comprehensive primary healthcare
Main aim	Reduction of specific disease—technical focus	Improvement in overall health of the community and individuals; health for all as overall social and political goal
Model of health	Biomedical	Social
Sectors involved	Strong focus on health sector—very limited involvement from other sectors	Involvement of other sectors is central
Strategies	Focus on curative care, with some attention to prevention and promotion	Comprehensive strategy with curative, rehabilitative, preventive and health promotion that seeks to remove root causes of health
Planning and strategy development	External, often 'global', programs with little tailoring to local circumstances	Local, reflecting that community priorities are professional and 'on tap not on top'
Participation	Limited engagement, based on terms of outside experts and tending to be sporadic	Engaged participation that starts with community strengths and the community's assessment of health issues, is ongoing and aims for community control
Engagement with politics	Professional and claims to be apolitical	Acknowledges that primary healthcare is inevitably political and engages with local political structures
Forms of evidence	Limited to assessment of disease-prevention strategy based on traditional epidemiological methods, usually conducted out of context and extrapolated to situation	Complex and varied research methods including epidemiology and qualitative and participatory methods

Source: Adapted from Table 1 in F. Baum (2007). Health for all now! Reviving the spirit of Alma Ata in the twenty first century. An introduction to the Alma Ata Declaration. *Social Medicine*, *2*(1), 34–41.

BOX 51.5 Minimum activities for primary healthcare

- Education concerning prevailing health problems and the methods of preventing and controlling them.
- Promotion of food supply and proper nutrition.
- Provision of an adequate supply of safe water and basic sanitation.
- Provision of maternal and child healthcare, including family planning.
- Immunisation against major infectious diseases.
- Prevention and control of locally endemic diseases.
- Appropriate treatment of common diseases and injuries.
- Provision of essential drugs.

Source: World Health Organization (1978). *The Declaration at Alma-Ata, International Conference on Primary Health Care*. Retrieved from www.who.int/publications/almaata_declaration_en.pdf.

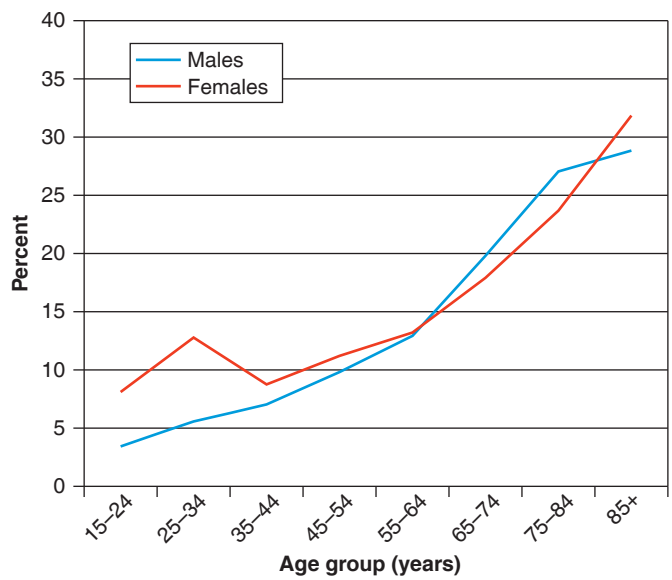

FIGURE 51.6 ■ Proportion of people aged 15 to 85+ years visiting a general practitioner 12 or more times over the previous 12 months

Source: Australian Bureau of Statistics (ABS) (2013). *Patient experiences in Australia: Summary of findings, 2012–13*. Retrieved from www.abs.gov.au/ausstats/abs@.nsf/Lookup/4839.0main+features32012-13. © Commonwealth of Australia.

including Nurse Practitioners, practice nurses, midwives and allied health professionals (Department of Health and Ageing, 2008). Figure 51.6 shows the number of visits made to general practitioners by people aged 15 to over 85 years in a 12-month period. How healthy a person considers themselves to be is related to the number of times they see their GP. Those who considered their health to be 'fair' or 'poor' were more likely to see a GP more often than those who believed their health was 'good', 'very good' or 'excellent'. In addition, the frequency of GP visits was related to the presence of long-term health conditions.

Primary care services are based in communities and focus on a person's specific disease or illness using a disease-management orientation (Nesbit & Allen, 2011). Services

are usually controlled by the healthcare provider (Nesbit & Allen, 2011) rather than developed in partnership with the community, and often use intermittent management or single occasions of service. Visits are often based on appointment times initiated by the consumer (e.g. Medicare short consultation) and providers strategically manage follow-up appointments (e.g. screening or acute diagnostic test results, assisted chronic disease management). Exemplars of Australian community services based on primary care are detailed later in this chapter.

In contrast to primary healthcare, primary care services are usually based on the biomedical model of health (Keleher, 2001), discussed previously, and place minimal emphasis on social determinants, empowerment, partnership or community engagement (Keleher, 2011; Talbot & Verrinder, 2013). However, at times community organisations also display an holistic orientation to individual care within a primary care framework. While not as comprehensive as primary healthcare, this variation addresses some social aspects of heath at an individual rather than a community level.

The characteristics of primary care are listed in Table 51.2 above and can be contrasted with those listed for primary healthcare. The table shows primary care's reliance on medical interventions and professional control, with little local community engagement, minimal involvement across sectors and minimal attention to structural factors affecting health (Baum, 2007). While it is recognised that primary care has led to health gains for some, to improve the health of all members of the community health needs to be contextualised within its social, cultural, economic and environmental context (Adrian, 2009).

HEALTH EDUCATION AND HEALTH PROMOTION

Health promotion, illness/injury prevention and health education are the cornerstones of primary healthcare and community care. **Health promotion**, incorporating illness/injury prevention, is described in the Ottawa Charter for Health Promotion as the process of enabling people to increase control over and improve their health (WHO, 1986). **Health education** is often used interchangeably with health promotion; however, health education refers to an individualist model which focuses on the individual and individual strategies to improve health by increasing knowledge or influencing attitudes to alter behaviours and lifestyle, such as health counselling and health education (WHO, 2012). Primary healthcare, which focuses on the determinants of health, is aligned with population-focused strategies for health promotion, known as structuralist–collectivist health promotion (SCHP), a model that involves community engagement and capacity building.

Individualist health promotion

The individualist model of health promotion focuses on changing individual behaviour and lifestyle, and rests on the assumption that individuals harm their health by engaging in unhealthy living (i.e. smoking, eating fatty foods, not exercising and drinking alcohol). It aims at improving health by focusing on risk factors, particularly those identified as precursors to disease (Duckett, 2004; Richmond & Germov, 2014). In this model, disease and illness are seen as a consequence of failure to comply with healthy lifestyle choices; in an individualist society this is difficult to challenge because it appears to make sense. However, it is a single-cause approach which emphasises individual choices associated with risk factors rather than the underlying social conditions that influence choice and behaviours (Povlsen & Borup, 2011). Most factors affecting illness and health lie outside the control of individuals, and lifestyles are powerfully influenced, if not fully determined, by the social organisation in which they are embedded (Richmond & Germov, 2014; George, 1998; Underwood, Owen & Winkler, 1986).

Individualist health promotion (IHP) is promoted as being successful and cost effective, but adequate evaluation is lacking (Travers, Martin-Khan & Lie, 2009). Few programs succeed for any length of time and many are either unsuccessful or cater only for the well motivated (e.g. weight loss programs). Exaggerated in this case is the ease with which individuals make connections between knowledge and behaviour, when in reality there is a complex interaction between knowledge, attitude and practice/behaviour change (Talbot & Verrinder, 2013). Education alone does not necessarily lead to behaviour change and approaches that rely only on education underestimate the impact of social factors on health and illness or injury.

While IHP is a dominant model and can be useful, it is too narrow when considering health through a social model, as in primary healthcare services. It also risks leading to victim blaming; if an individual is sick, it is because he or she has not complied with health advice. In short, if people took better care of themselves they would not have the health problems they have. The person who develops lung cancer and is also a smoker is an obvious example. IHP programs do nothing to alter the structural causes or determinants of ill health or injury, and can be paternalistic and patronising (Talbot & Verrinder, 2013) (i.e. increased health education, knowledge and/or awareness do not equal structural change). For example, people on low incomes cannot afford more nutritious food, irrespective of their health education. In this example, it is not so much ignorance that is the problem but inadequate employment opportunities and/or welfare allowances. Healthy choices are not easy choices for the disadvantaged; people with little choice about their lives often regard immediate comfort to be more important than end-stage health. All these situations highlight issues and concerns for healthcare which is experienced as culturally safe.

Health promotion should aim to change the social situations framing individual decision making, particularly in terms of income, employment and housing. In particular, attention to the social determinants of health can bridge the disparity between 'knowing' and 'doing' by empowering people to make healthier choices. This can involve small-scale local interventions as well as broader national or global social change. Table 51.3 lists the characteristics of individualist health promotion, including its relationship to models of health and primary healthcare.

TABLE 51.3 individualist and structuralist–collectivist health promotion

	INDIVIDUALIST HEALTH PROMOTION (IHP)	STRUCTURALIST–COLLECTIVIST HEALTH PROMOTION (SCHP)
Model of health	Biomedical or biopsychosocial model	Social model
Target level	Individuals Operates at point of entry, screening, advice Downstream determinants	Community Active care recipients and communities, social change Upstream determinants
Focus of education	Individual focus, expert-led, passive person Lifestyle, risk taking	Participatory, legislation, bureaucratic intervention Community engagement, advocacy, enabling, mediating, organisational development, builds healthy policies
Aim	Persuade individual change Improve physiological risk factors and personal behaviours	Invoke population change Address determinants of health, redress inequities
Examples	Healthy eating campaigns Safe-driving campaigns Brief intervention strategies Quit campaign	Needle exchange Legislation (e.g. banning smoking in public places, compulsory wearing of seat belts) Health services offered by Indigenous health workers working with Indigenous communities
Relationship to primary healthcare	Selective primary healthcare or primary care	Comprehensive primary healthcare or primary care

Sources: Based on K. Richmond & J. Germov (2014). A sociology of health promotion. In J. Germov (ed.) *Second opinion: An introduction to health sociology* (5th ed.) (pp. 464–483). Melbourne: Oxford University Press; and H. Keleher, C. MacDougall & B. Murphy (eds) (2007). *Understanding health promotion* (pp. 29–32). Melbourne: Oxford University Press.

Structuralist–collectivist health promotion

In accordance with the Ottawa Charter, and in alignment with the WHO primary healthcare approach, health priorities should be community-based, reflect the concern of particular communities and be 'bottom up' (WHO, 1978; 1986). Often, though, structuralist–collectivist health promotion (SCHP) is less about structural change and more about health education in disguise by imposing policies or legislation on communities without adequate consultation. A barrier to SCHP approaches is that some interventions are highly politicised (such as food advertising to children) (Richmond & Germov, 2014).

The Ottawa Charter (WHO, 1986) aims to integrate individualist and SCHP approaches by focusing on 'positive' health, improving the settings for decision making on health and rewarding health professionals for their involvement in health promotion. Figure 51.7 illustrates a continuum of health-promotion approaches and Table 51.3 contrasts characteristics of individualist health promotion and SCHP. The 'Translation to practice' box below describes the development of a community care model, based on CDC, which led to educational resources aimed at supporting implementation of the model in community practice. For health education to be effective, culturally safe approaches are needed.

COMMUNITY CARE EXEMPLARS

To cater for the diverse communities and complex health needs that characterise Australian communities, a broad range of community care services have been needed, many of which are characterised by innovative approaches underpinned by primary care or primary healthcare. The goal is to improve the health of people, families or communities and to provide healthcare from conception to death in a range of community settings. These services care for people and communities within their usual environments, including homes, caravans, rural and outback campsites, schools and workplaces. Detailed below are examples of some Australian community nursing services. Each is located in the community and demonstrates aspects of primary care or primary healthcare. They aim to meet the specific health needs of their population and each contributes to ensuring the health of its community in a different way. Reading each exemplar should promote reflection on the key concepts discussed earlier in this chapter and how these are enacted in practice by community nurses.

Caravan Park Project

The Caravan Park Project (Baker cited in Adrian, 2009) commenced in 2000 and is an example of a nurse-led primary healthcare strategy in rural New South Wales which aims to develop health equality. It exemplifies an innovative and sustained response to the social determinants of health impacting on the health of residents of the caravan park, both children and adults. This example illustrates ways of practising which acknowledge and work with cultural dimensions of the health of a community and demonstrates the remarkable resilience, leadership and commitment of the nurse leading the project.

The community of interest for this project was the residents of a local caravan park (see Figure 51.8). Like similar caravan parks in rural and urban locations, the residents came from diverse and difficult backgrounds, including homelessness or transient low-cost accommodation, prison and juvenile services, mental health facilities or refuges. These backgrounds placed residents at risk of poor health, a situation that was confirmed

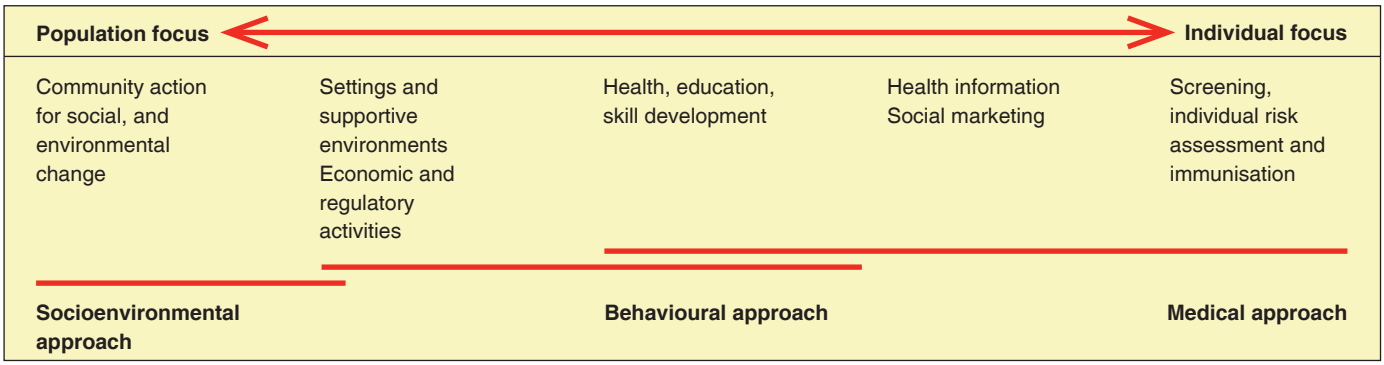

FIGURE 51.7 ■ Continuum of health-promotion approaches

Sources: Figure 1.6 in L. Talbot & G. Verrinder (2010). *Promoting health: The primary health care approach* (4th ed.). Sydney: Elsevier Churchill Livingstone; adapted from R. Labonte (1992). Heart health inequalities in Canada: Models, theory and planning. *Health Promotion International, 7*(2), pp. 119–21.

by a high number of referrals for residents of the park to health professionals—referrals which were difficult to follow up due to factors such as poor or no access to telephones, being away from their caravan when health professionals called to visit and a lack of resident trust in government-based services.

At the outset, a presence of health professionals was established onsite by running a van-based child and family health clinic (Baker cited in Adrian, 2009). This facilitated contact between health staff and residents, allowed an understanding of residents' health issues to be developed and enabled consideration of how social determinants were acting on this community. During this time a picture emerged of a highly disadvantaged community with the following characteristics:

- largely reliant on government benefits for income
- poor functional literacy
- frequent domestic violence
- high levels of substance use and high rates of hepatitis C infection
- social isolation with poor self-esteem
- poor school attendance by the children

TRANSLATION TO PRACTICE Applying evidence about CDC to community aged care practice

Between 2010 and 2012 a major trial of aged care CDC in Australia, the People at Centre Stage (PACS) project, addressed obstacles for successful CDC reported previously by international researchers, and gathered evidence about CDC implementation in Australia (Ottmann & Mohebbi, 2014). In the PACS trial a 'case management led capacity building and restorative health approach' (p. 600) was applied in conversations with older people to set health priorities and goals, and to support three levels of self-direction: level 1, where the older person participated in care planning; level 2, where the older person assumed both care planning and care coordination responsibility; and level 3, where the older person performed the activities of level 2 but also administered and budgeted care using a voucher option with a small cash component. Overall the PACs model resulted in most older people reporting increased satisfaction with care and decision processes, improved health, sense of engagement and community connectedness (Ottmann, Laragy & Allen, 2012, p. 24).

Subsequently the evidence obtained from the PACS trial led to the development of practical resources and tools for community aged care providers delivering CDC-based services to older Australians. These materials, referred to as the 'choices' model of CDC, provide services with materials to address the distinctive needs of older Australians while implementing CDC, including restorative health and capacity building; goal-directed care planning; individualised budgets; self-direction and mentoring; enabling risk; community connectors; and planning ahead. The 'choices' materials can be accessed through the Home Care Today website (www.choicesinagedcare.com.au/training/).

IMPLICATIONS FOR NURSING

- To provide CDC, nurses need to work in partnership with individuals to enable consumer choice and control about all aspects of their care.
- Community care nurses need to work with older individuals to identify their care goals as well as choose care options that match the individual's preferences for community care.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. How might person-centred care be different from consumer-directed care?
2. Why might consumer direction and control be important for community-based individuals?
3. How can nurses enable consumer choice and control of community care?

Sources: Ottmann, G., Laragy, C. & Allen, J. (2012). *People at Centre Stage: Evaluation Summary Report.* Melbourne: UCCO/Deakin University QPS; Ottmann, G. & Mohebbi, M. (2014). Self-directed community services for older Australians: A stepped capacity-building approach. *Health and Social Care in the Community*, *22*(6), 598–611. Doi: 10.1111/hsc.12111.

FIGURE 51.8 ■ Caravan park as a community

Source: © KateTilmouth/Shutterstock.com.

- a strong history of childhood sexual assault, abuse and/or neglect
- failure to access mainstream health services or general practitioners (Baker cited in Adrian, 2009).

With funding from New South Wales Health and Primary Healthcare and Partnerships, an innovative outreach project involving an early childhood nurse, a project officer (Registered Nurse) and a registered psychiatric nurse was developed based on the social model of health/social determinants of health and advocacy (Baker cited in Adrian, 2009). The main aims were to reorient health services to meet the needs of caravan park residents, to build capacity and partnerships with other health and intersectorial services (e.g. Centrelink, local community businesses, schools, charities and education providers), to strengthen existing partnerships, to increase resident literacy and to strengthen resident capacity for community action. Interventions included advocacy for improved living conditions in the park, particularly as children were over-represented for admission to hospital with respiratory conditions exacerbated by damp and musty van accommodation. An onsite needle and syringe program was also established in the outreach van and disposal bins placed in the communal toilet blocks to reduce the number of childhood presentations at the emergency department due to needle-stick injuries. Also negotiated were the supply of full-size refrigerators to enable better food purchases and safe storage of food. Health and intersectorial services were lobbied to reorient their services to enable resident access, and entrepreneurial partnerships were nurtured with private businesses. These helped to raise funds to enable the resident children to participate in sport, dance classes and school excursions, and also to resolve transportation barriers. Onsite services were then extended further to include paediatric clinics and a volunteer public health physician.

After 6 months the caravan park's culture began to change, developing a sense of community (Baker cited in Adrian, 2009). Residents attended antenatal care, TAFE and school. As confidence felt by residents grew, the project team members linked residents back into mainstream health and social services. The project continues to evolve and has been successful in delivering high-quality health services on minimal funding to highly disadvantaged members of the community. The holistic approach has made changes that remove barriers; barriers which make it impossible for disadvantaged members of this community to live in ways that support health and wellbeing. The approach used during this project demonstrates service development and provision using a primary healthcare approach.

Practice nursing

Practice nurses (see Figure 51.9) deliver primary care in a general medical practice context within communities and are a relatively new area of clinical practice in Australia (Australian Nursing Federation (ANF), 2011b). This role has developed due to a combination of factors including inadequate numbers of general practitioners in the primary care sector, a renewed focus on health promotion and chronic disease management, and population ageing (ANF, 2011b). It recognises the role nurses play in ensuring the health of people in a community. A significant driver for nurse employment in general practice, particularly for the development of the clinical nursing role in the management of chronic disease, has been the Australian Government Practice Nurse Incentive Scheme (Australian General Practice Network (AGPN), 2009). The number of general practices employing practice nurses has risen to 63.3% in 2012, compared with 56.9% in 2009 and 58% in 2007 (Australian Medicare Local Alliance, 2012). The number of practice nurses employed was 10 693 in 2012 (Australian Medicare Local Alliance, 2012), with a clear trend that practices are employing more than one practice nurse (AGPN, 2009).

Practice nursing involves care for a broad range of community members from all stages of life and from backgrounds that reflect the diversity of the community population (ANF, 2011b). While practice nurses work in different relationships

FIGURE 51.9 ■ Nurses work in community-based general practices in Australia

Source: © Life in View/Science Photo Library.

with general medical practitioners, many adopt an approach based on primary care rather than task delegation (Keleher, Joyce et al., 2007). While the role varies according to the profile of people attending the general medical practice, adopting a primary care approach allows the practice nurse to play a key role in health promotion, health maintenance and illness prevention through the provision of information, education and care to community members who attend the practice (ANF, 2011b). Also important is working collaboratively with others in the practice and the wider community (ANF, 2011b). The roles that nurses undertake in Australian general medical practices are listed in Table 51.4. Although these mainly illustrate a biomedical orientation to service provision and, hence, an approach consistent with primary care rather than primary healthcare, some practice nurses provide care in ways that also reflect a biopsychosocial understanding of health.

Managing acute illness at home

Community nurses work in a range of contexts, including people's homes, and can be called domiciliary or generalist community nurses. For the community nurse, contact with people is initiated for many reasons. In the following exemplar, nursing assistance has been sought as a consequence of an acute illness and demonstrates a nursing focus beyond the individual to the family, and to health beyond the immediate health issue. Assessing the needs of the person in their own environment and within their normal family relationships highlights the importance of being holistic in the way health is considered. The following narrative provides an example of primary care which extends beyond the biomedical model.

Jodi, a community nurse working in a new local multipurpose centre, has been asked to see Mr Louis Spelt, aged 72 years. The reason given for the referral from a hospital ward is care of a wound with a vacuum-assisted closure (VAC) system and continuous IV antibiotic therapy administered via a Baxter pump into a peripheral intravenous catheter (PIC) line. Mr Spelt has returned home from the local private hospital following revision surgery of his tibia. He originally had a benign tumour removed near his knee but an infection developed and required further removal of bone, and debridement of the original wound.

Jodi arrives at Mr Spelt's cottage and notices it has four steps at the entry. Mr Spelt's wife answers the door and invites her into their home. Jodi finds Mr Spelt dressed in his dressing gown and waiting in the living room. The room is very comfortable but is

TABLE 51.4 Roles of nurses in general practice

ROLE	ACTIVITIES
Clinical nursing services	Triage and individual assessment Clinical care (e.g. wound care) Diagnostic services Clinical data management
Health promotion and chronic disease management	Health screening Immunisation Health check reminders (e.g. Pap smear) Individual health education Outreach services (e.g. visiting elderly people unable to visit the practice) Acute and chronic disease management (e.g. diabetes and asthma management)
Coordinating person services	Working with GPs to plan and manage individual care Liaising with allied health and community care services Coordinating delivery of healthcare services Ensuring continuity of care Facilitating effective communication between individuals and healthcare providers Individual advocacy
Promoting person, carer and community wellbeing	Providing education and health information Delivering specific programs Engaging in community development Educating about self-care
Managing clinical standards and legislative requirements	Infection control and sterilisation Monitoring incidence of infectious disease Records management Occupational health and safety Participating in accreditation processes Maintaining medical supplies
Management of human and material resources	Optimising the use of professional resources Building the practice base Building practice capacity to adapt to change Maximising financial efficiency

Source: Adapted from Australian Nursing Federation (2011b). *A snapshot of general practice nurses in Australia: Fact sheet no. 7.* Retrieved from www.anf.org.au/pdf/Fact_Sheet_Snap_Shot_General_Practice_Nurses.

cluttered. Jodi proceeds to complete her assessments and, in consultation with Mr Spelt and his wife, identifies that Mr Spelt will need nursing care every morning to renew the Baxter infusion and to monitor the VAC dressing, also allowing time for carer support for Mrs Spelt and health education as their situation changes.

During the holistic assessment Mrs Spelt assists, describing herself as 'Louis's full-time carer at the moment. He really can't do much for himself.' Before Jodi completes the consultation she is able to discuss with Mr Spelt some of his other needs and what support Mrs Spelt needs in her caring role. Jodi discusses pain management and completion of activities of daily living. Mr Spelt is mobilising with a single crutch and has an exercise program that he doesn't think is important. Jodi and the Spelts review the program and work out an achievable action plan for them to follow. Jodi also discusses ways of reducing the risk of falls, including the removal of some occasional tables in the living room. She further expresses a concern about the entry steps but Mrs Spelt assures her that Louis would only use them when she was there to assist.

One week passes and Jodi arrives to change the VAC dressing for the second time. Jodi no longer changes the Baxter unit daily as she has been able to educate Mr and Mrs Spelt to attend to this independently; every morning after his shower they do this together. Mrs Spelt is proud that she can help and they are pleased because once the Baxter is changed they are free to continue their day and no longer need to wait for the nurse to arrive. Although there is still the dressing, life is returning to normal for them.

Today while Jodi is changing the VAC dressing, she reflects on the changes that have occurred over the past week. Mr Spelt met her at the door and was dressed in casual clothes ready for a trip to his daughter's house. He was relaxed and chatting about a cruise they were going to book once his leg had healed. The living room was free of clutter and Mrs Spelt was hanging out the washing.

Jodi's narrative shows how individual and family wellbeing are facilitated despite significant illness. Mr Spelt's recovery has been supported by the community nurse's actions, for them as individuals and as a couple. The community nurse's role has been described as one where '. . . you are very much involved in a person's life, not just their condition . . . In this kind of nursing one must consider all of the influences in a client's situation' (Adrian, 2009, p. 43).

THE WAY FORWARD

It has been argued that revision and reorientation of the Australian healthcare system towards a more comprehensive primary healthcare approach is required in response to increasing social and demographic factors, such as health system burdens from chronic conditions, an ageing population and a need to manage increasing healthcare costs (Parker & Keleher, 2008). The complexity and volume of care delivered also continues to increase due to factors such as reduced hospital stay duration, increased focus on ageing in place and care at home, and de-institutionalisation processes (Department of Health and Ageing, 2008). Specific drivers underpinning this reform in NSW are detailed in Box 51.6. Government attention has therefore become focused on the need to develop capacity in the prevention and management of chronic conditions (Parker & Keleher, 2008). Several health reform strategies have ensued, including:

- National Primary Healthcare Strategic Framework
- National Maternity Services Plan
- Nursing and Midwifery Consensus View: Primary Health Care in Australia
- National Health and Hospitals Reform Commission (NHHRC).

The goals of recent health reform have been a reorientation of services towards increased primary healthcare and include increased service access, strengthened prevention and early intervention, improved management of chronic conditions, integrated delivery and cross-disciplinary team-based care (Keleher, Parker et al., 2007). Central to these developments is a desire to shift the emphasis of the system from narrow hospital-based care, treatment and cure of already established disease to the promotion of health across the lifespan; prevention of injury/disease; effective management of chronic disease; and the reduction of health inequalities (Adrian, 2009; Department of Health and Ageing, 2013;

BOX 51.6 Drivers for change for community care

- There is demand for greater provision and variety of primary health services.
- The behaviour and attitudes of people influence their health. Prevention can be encouraged in the community.
- The environment of infancy and childhood influences the development of personality. This should be emphasised in community health and welfare service.
- One-quarter of the total population suffers from chronic disease which will never be cured.
- One person in every 10 is disabled by chronic disease. One-third of those with chronic disease are under the age of 60, and many have psychological and social disabilities. Disability in older people increases with age. Four out of 10 of those aged 75 and over are limited in one or more of their daily activities.
- About 2% of the population have some form of serious mental illness and more than this have a psychological disorder. Many people have coexisting emotional and physical disorders. They require more support, alleviation, special training, rehabilitation and care in the community than is available at present.
- Early discharge programs are moving more people requiring acute and post-acute care into the community.
- There are many problems where the medical and social aspects are so interrelated that they require teams of health and welfare professionals to assist.

Source: Community health: The evidence base. A report for the NSW Community Health Review by A. Owen et al. (2008). Wollongong: Centre for Health Service Development, University of Wollongong, p. 1.

Keleher, Parker & Francis, 2010). This inherently emphasises primary care and illness/injury prevention, making them linchpins of the health system (Keleher et al., 2010). This improvement aims to counteract a natural health system tendency away from primary healthcare (WHO, 2008a) and to increasingly locate care in community settings (Keleher et al., 2010). To achieve this outcome more nurses will be needed, with nurses working in diverse community roles and in ways that harness the benefits of a primary care orientation to health (Adrian, 2009).

Nurses, who are socially mandated to improve population health, hold views complementary to the philosophies of primary care and primary healthcare, and are ideally placed to contribute to this reform and the health of communities (Eagar et al., 2008). While there are already over 29 000 nurses employed in Australian primary healthcare settings (ANF, 2011a), further development of community nursing roles is needed to support the required shift in health system orientation to primary healthcare (Keleher, Parker et al., 2007). Choosing to work as a nurse in a community organisation is an exciting decision and a step towards a challenging and rewarding career. In community care, the community as a whole and the person are both privileged, no matter whether they are a child, a family, a work team or a school. It is important to acknowledge that community members have the potential to make their own decisions about their health and healthcare. The challenge is to work in a sustained way with individuals, groups and organisations to assist community members, their families and community groups to prevent illness and injury, and to lead healthy and rewarding lives. Optimising health for the community is about ensuring a quality of life that is consistent with the individual's and community's needs and values, and is within the WHO's framework for 'Health for All' (WHO, 1978).

CHAPTER HIGHLIGHTS

- The factors that determine the health of a community include societal, environmental and socioeconomic factors, as well as the knowledge, values, beliefs and health behaviours of individuals and the community as a whole.
- Different models of health underpin the way community organisations contribute to the health of individuals and communities.
- In Australia, community services are underpinned by the biomedical, psychosocial and social models of health. Those services underpinned by a social model of health will approach health differently from acute care services based on the biomedical or psychosocial model.
- Community care is delivered in a diverse range of settings, to diverse populations and by a range of different people/services.
- Cultural safety is integral to community care in all settings.
- Community care is provided in partnership with the person and their family, and a range of government and non-government organisations.
- Health promotion and health education are based on different models of health and contribute in different ways to achieving community health. Structural–collectivist approaches to health promotion support primary healthcare.
- Many community organisations in Australia approach community care service provision using a primary care approach rather than a primary healthcare approach.

CONCEPT CHECK

1 Social determinants of health are:
1. all biological and environmental conditions that affect the health of people and communities
2. the conditions in which people are born, grow, live, work and age
3. the biological factors that cause ill health in people
4. none of the above

2 Which factors are considered important social determinants of the health for an individual or group in a community? (Select all that apply.)
1. presence of disease
2. unemployment
3. attitudes
4. genetics
5. life skills

3 Primary healthcare is described by the World Health Organization as essential healthcare based on practical, scientifically sound and socially acceptable methods, which is affordable and is delivered to individuals and families in the community.
1. true
2. false

4 Match the following terms to as many models as needed for each term.

Terms
- a Primary healthcare
- b Primary care

Models
1. Social model of health
2. Biomedical model of health
3. Biopsychosocial model of health

5 What are the principles that underpin a primary healthcare orientation to community health? (Select all that apply.)
1. social justice
2. medical expertise
3. equity
4. responsiveness to need
5. community participation

6 Which of the following are characteristics of primary care? (Select all that apply.)
1. reduction of specific disease and a technical focus
2. based on the biomedical model of health
3. involvement of sectors other than health
4. focus on curative care, with some attention to prevention and promotion
5. limited community engagement

7 Which of the following are characteristics of primary healthcare? (Select all that apply.)
1 improvement in overall health of the community and individuals, and 'Health for All' as overall social and political goal
2 based on the socioecological model of health
3 strong focus on health sector with very limited involvement from other sectors
4 comprehensive strategy with curative, rehabilitative, preventive and health promotion that seeks to remove root causes of health
5 engaged participation that starts with community strengths and the community's assessment of health issues, is ongoing and aims for community control

8 Which of the following are drivers to reorienting community service organisations towards primary healthcare? (Select all that apply.)
1 One-quarter of the total population suffers from chronic disease which will never be cured.
2 Medical and social aspects of health are so interrelated that they require teams of health and welfare professionals to assist.
3 Behaviour and attitudes of people influence their health. Prevention can be encouraged in the community.
4 Demand for greater provision and variety of primary health services.

9 In health, two common ways of understanding what is meant by 'community' are:
1 community as relationships
2 community as politics
3 community as locality
4 community as health

10 On the continuum of interventions using social determinants, which of the following levels of health determinant have a more direct effect on the health of an individual?
1 distal and proximal determinants
2 proximal and intermediate determinants
3 distal and intermediate determinants
4 none of the above

11 Intersectorial refers to:
1 the way health professionals interact within community organisations
2 the way health professionals interact within their own organisation
3 the way health professionals interact with non-health organisations
4 none of the above

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USEFUL WEBSITES

Australian Government Department of Social Services, a national service that supports both consumers and home care providers to work together to implement consumer-directed care through home care packages: http://www.homecaretoday.org.au/

Australian Health Promotion Association: www.healthpromotion.org.au

Australian Indigenous Health Infonet: www.healthinfonet.ecu.edu.au/

Australian Indigenous health promotion knowledge network: www.indigenoushealth.med.usyd.edu.au/links.htm

Australian Institute of Health and Welfare—*Australia's health 2014*: Chapter 8 Preventing illness: www.aihw.gov.au/australias-health/2014/preventing-ill-health/#t3

Australian Nursing Federation Fact Sheets: www.anf.org.au/html/publications_factsheets.html

Australian Primary Health Care Research & Information Service: www.phcris.org.au/

Australian Women's Health Network: www.awhn.org.au

Centre for Culture, Ethnicity and Health: www.ceh.org.au

Community Health Nurses, Western Australia: www.chnwa.org.au

Department of Health: www.health.gov.au

National Health and Medical Research Council: www.nhmrc.gov.au

Palliative care: www.palliativecare.org.au/

World Health Organization: www.who.int/en

CHAPTER 52

NURSING CARE OF PEOPLE IN REGIONAL, RURAL AND REMOTE AREAS OF AUSTRALIA

LEEANNE HEATON

KEY TERMS

LEARNING OUTCOMES

- Discuss the challenges faced in providing nursing care in regional, rural and remote communities.
- Discuss the rewards of nursing in regional, rural and remote areas.
- Identify the determinants of regional, rural and remote classification in Australia.
- Identify the determinants of regional, rural and remote health.
- Describe nursing in remote Indigenous communities with regard to cultural awareness, cultural safety and the rights of people living in these communities.
- Describe the role of the nurse in regional, rural and remote healthcare.
- Discuss the barriers to healthcare as identified by nurses, other health professionals and consumers in regional, rural and remote communities.

CLINICAL COMPETENCIES

- Use evidence-based practice guidelines and clinical assessment skills to inform clinical decision making.
- Provide timely and accurate person-centred assessment.
- Incorporate an understanding of the local determinants of healthcare provision in regional, rural and remote areas into quality nursing care.
- Use evidence-based nursing practice to provide quality nursing care in acute and non-acute situations, including health promotion and prevention strategies.
- Incorporate the principles of primary healthcare into the provision of nursing care in regional, rural and remote settings.
- Provide culturally safe nursing care.
- Use professional communication skills to develop therapeutic relationships and establish professional boundaries when working in regional, rural and remote communities.
- Use assessment findings to determine initial nursing care, referral and transfer as deemed necessary.
- Recognise and work within the interdisciplinary team locally and in distance mode.
- Incorporate all available resources, including technological resources, to maximise health outcomes.
- Recognise the special considerations for people from regional rural and remote areas in the discharge planning process.

Note on clinical competencies

This chapter relates regional, rural and remote nursing in Australia to the Nursing and Midwifery Board of Australia (NMBA, 2016) *National Registered Nurse Standards for Practice (2016).* The competencies of the Registered Nurse practising in regional, rural and remote Australia are directly linked to all of the domains of professional practice, critical thinking and analysis, provision and coordination of care, and collaborative and therapeutic practice. Specifically, regional, rural and remote nursing practice in Australia is clearly aligned with the guidelines of all of the individual elements as identified in the NMBA Standards for Practice.

The terms 'regional', 'rural' and 'remote' invoke impressions of distance but in reality the communities, people and available health services in these areas vary greatly. So, too, do the responsibilities, roles and philosophies employed when providing nursing care for people in these areas. Therefore, any discussion about regional, rural and remote nursing requires acknowledgement of the fact that there is no single definition of 'regional', 'rural' or 'remote', and awareness of the specific areas being discussed to ensure accurate and consistent representation.

Understanding regional, rural and remote nursing is not exclusively the domain of the local workforce, health department officials or government policy advisers. All nurses, including those working in large metropolitan areas, need to possess a sound knowledge of the associated challenges and barriers. Nurses provide a greater percentage of healthcare than any other health-related discipline, and regional, rural and remote nursing encompasses a vast range of nursing skills, including the ability to accurately identify the health needs of people in these communities and to refer and/or transfer people to major centres as appropriate. Nurses working in referral centres also face unique challenges when caring for, and planning discharge for, people who usually reside in a regional, rural or remote area.

This chapter discusses the nursing care of people in regional, rural and remote areas from two perspectives: first, the challenges, opportunities and rewards associated with nursing in regional, rural and remote Australia; and second, the provision of nursing care for people from rural and remote areas in regional or metropolitan areas.

CHALLENGES AND REWARDS

Nurses work in remote areas for a range of reasons, and quite often the reason that brings them to the community may be the reason they leave. This can include employment for their partner, family commitments, end of employment contract and change in life expectations. Leaving small communities can be a distressing time for the nurse, colleagues and the community members. This can be compounded if there is difficulty in filling the vacant nursing position.

Working in regional, rural and remote areas can be extraordinarily challenging while at the same time being immensely rewarding, quite often for the very same reasons. Nursing practice in regional, rural and remote areas is holistic in the truest sense of the term. A nurse who works in a small community for many years may well provide nursing care for several generations of a family, providing holistic care from the cradle to the grave.

THE DIFFERENCES BETWEEN 'REGIONAL', 'RURAL' AND 'REMOTE'

One of the most complex tasks when discussing regional, rural and remote issues relating to any discipline is to identify and apply common definitions. To assist in providing a consistent guide, the Australian Government uses three systems that incorporate many of the factors impacting on an individual's access to health services as a result of their location. These systems are the Accessibility/Remoteness Index of Australia (ARIA+) (University of Adelaide, 2015), the Rural, Remote and Metropolitan Areas (RRMA) Classification (Australian Institute of Health and Welfare (AIHW), 2015a) and the Australian Statistical Geography Standard (ASGS) (Australian Bureau of Statistics (ABS), 2014a).

There is a complex method of determining remoteness areas (RAs) in Australia. In 2011, the remoteness structure of the Australian Statistical Geography Standard (ASGS) was introduced by the ABS to assist in publishing a diverse range of social and demographic statistics (ABS, 2014a). The ASGS divides each state and territory into regions according to the access to services in the localities (ABS, 2014a). The University of Adelaide is responsible for the Accessibility/Remoteness Index of Australia (ARIA+), which measures the remoteness of one point to another based on physical road distance to the nearest urban centre in each of the five size classes: major cities, inner regional, outer regional, remote and very remote (University of Adelaide, 2015). The ARIA+ information is placed into 1 km grids of Australia. These grids are then overlayed onto the ASGS statistical area (SA) levels, determining the remoteness areas (RAs) (ABS, 2014a). Awareness of these classifications is necessary for nurses for a range of reasons. Possibly of greatest importance is the statistical evidence that people who live in remote and very remote Australia have overall poorer mortality (up to 1.4 times higher) and morbidity than those who live in major cities (AIHW, 2014). It is statistically significant that Aboriginal and Torres Strait Islander people make up a higher percentage of the remote area population than of the urban population. Indigenous people in regional, rural and remote areas are also over-represented in poorer health outcomes data overall, which is consistent with the lower life expectancy rates for Indigenous people in Australia (AIHW, 2014). Furthermore, it has been proposed that the lower life expectancy of Indigenous people living in remote areas is more suggestive of their Indigenous status than their locality.

The Council of Remote Area Nurses of Australia (CRANAplus) explained that working in remote areas can present its own set of challenges (CRANAplus, 2015). This can be due to factors such as geography causing difficult access, extreme distance and variations in weather conditions. The population is scattered, with people from different cultures and groups who have challenging health conditions. Lack of resources and a highly itinerant group of health professionals also impact on delivery of quality health services to people in regional, rural and remote areas of Australia (CRANAPlus, 2015).

Regional, rural and remote nursing

In Australia, nursing is one of the most stable and equally distributed healthcare disciplines, with many nurses working in a diverse range of settings within regional, rural and remote areas. However, the nursing workforce in very remote Australia is like the rest of Australia in that it is ageing. Of the remote area nurses (RANs) working in very remote areas, 66.6% are aged 50 years or over, despite the average age of Registered Nurses being 44.2 years, and the national percentage of nurses aged 50 years or over being 39%. Men working in very remote areas account for 15% of the nursing and midwifery workforce (AIHW, 2012).

FAST FACTS

- Approximately 69% of the Australian population resides in major cities (ABS, 2015).
- Approximately 2.3% of the population lives in remote or very remote Australia (ABS, 2015).
- The Northern Territory has the largest percentage of people (57%) living in outer regional areas (including Darwin), and remote (20%) and very remote (22%) areas (ABS, 2015).
- The life expectancy for Aboriginal and Torres Strait Islander men is estimated to be 10.6 years lower than for non-Indigenous men (ABS, 2014b).
- The life expectancy for Aboriginal and Torres Strait Islander women is 9.5 years lower than for non-Indigenous women (ABS, 2014b).
- The mortality rate of Indigenous Australians was nearly twice that of non-Indigenous Australians and five times higher for Indigenous people aged 35 to 44 years (AIHW, 2015b).
- The rate of dying due to a land transport accident was more than four times higher in remote and very remote areas than in major cities (AIHW, 2014).
- Homelessness is an issue related not only to urban living. There are high rates of homelessness in regional and rural areas. The homelessness rate for Indigenous people in 2011 was nearly 14 times the rate for non-Indigenous people and is often due to severely overcrowded dwellings (AIHW, 2015b).
- Of the proportion of people living in remote areas, the Indigenous population is very high, with 45% living in very remote and 16% in remote areas (AIHW, 2015b).
- The nursing workforce in Australia has increased by 13% from 2005.
- In 2013, there were 1111 full-time equivalent nurses per 100 000 people in outer regional areas compared with 1264 nurses working in very remote areas (AIHW, 2015c).

Over 43% of RANs in very remote areas are in working in Indigenous communities in primary healthcare settings (Lenthall et al., 2011). Many remote communities are relying on health professionals who have not trained in Australia, and on locums or fly-in/fly-out healthcare providers who do not have an understanding of cultural awareness, cultural safety and/or the rights of people living in these communities (Ware, 2013).

CRANAplus describes RANs as working in a multitude of settings such as Indigenous communities, outback and isolated towns, farm communities, islands, tourist locations, mines and railways. They must have skills that facilitate a primary healthcare response ranging from covering an emergency to administering a variety of medications to providing health education. The scope of practice required of an RAN can be quite diverse and, depending on a number of factors, can become quite broad when required (CRANAplus, 2015). There is a great deal of information about the health of people living in remote areas, particularly about Indigenous people who have complex medical issues, but who are less able to access equitable health services.

PRIMARY HEALTHCARE AND REGIONAL, RURAL AND REMOTE NURSING

Over recent decades, the World Health Organization (WHO) has outlined global health strategies, from the early primary healthcare models to the current more specific strategies designed to address the underlying factors that affect health outcomes. This is hoped to achieve defined healthcare goals. These healthcare goals, as defined by the WHO, are further supported by the Millennium Development Goals (MDGs) initiated by the United Nations (WHO, 2015).

The delivery of any health service based on **primary healthcare** principles includes acknowledgement of the individual and often specialised needs of the community. This is achieved through consultation, optimising availability of and accessibility to services, and adopting an interdisciplinary approach which addresses all aspects of healthcare, from prevention and early detection to curative, rehabilitative and palliative services. These strategies require a 'whole of government' approach, coordinated and in collaboration with community organisations to ensure that changes in the global provision of healthcare acknowledge the rights of individuals and communities to have equitable access to quality healthcare. The implementation process of primary healthcare models in regional, rural and remote areas in Australia has proven to be more lengthy than anticipated. However, throughout this process the role of the nurse in these areas has evolved to what it is today, becoming increasingly recognised as that of an advanced clinical practitioner.

The Medical Specialist Outreach Assistance Program (MSOAP) was initially established in 2000. In the 2011–2012 Budget, the Australian Government announced it would provide funding to establish the Rural Health Outreach Fund consolidating the MSOAP, MSOAP ophthalmology expansion, MSOAP maternity services expansion, rural women's GP service and National Rural and Remote Health Kimberley Paediatric Outreach Program. This funding is an attempt to improve health outcomes for people living in regional, rural and remote locations by supporting the delivery of outreach health activities (Department of Health, 2014a).

Primary healthcare and social determinants of health

The WHO (2012) has determined that one of the most important ways in which the global community can affect health inequity is to mobilise governments and political bodies to address the inequity from a social determinants perspective. *Social determinants* is a broad term that describes the main factors influencing the quality of preventive and curative healthcare available to all people. As well as the persuasive power of global economics, national, state and local politics and economics influence the access to and quality of healthcare services. To achieve the goals outlined by the WHO (2012), governments, policy advisers and budgetary committees must be prepared to invest in the future health of all of their citizens. The realities of distance impacting on access to more

comprehensive healthcare services cannot be changed—Australia is a vast country—but what can be changed is the ability of people who live and work in regional, rural and remote areas to access healthcare services and the effort it takes to do so. Economic viability and sustainability are immense challenges to implementing higher-level health services in 'the bush', a colloquial term for non-urban areas. The health demands of people in regional, rural and remote areas will never be as great as those in urban centres where a much greater percentage of the population resides. As such, most health services are concentrated in urban areas. This approach not only significantly impacts on people who live in regional, rural and remote areas, but also has significant implications for the clinical practice of health professionals.

The role of the education provider should also not be overlooked. Institutions providing courses for health professionals must address the health issues and concerns inherent in these communities. Graduates need to be encouraged to work in regional, rural and remote areas, and having a clinical placement opportunity in the bush can facilitate this (Hart et al., 2014). There are a number of scholarship or intern schemes for all health disciplines which aim to recruit and retain staff in regional, rural and remote areas. Some examples of these are the opportunities provided by the Australian College of Nursing (ACN) for nurses to participate in a regional, rural or remote placement. Most state health departments also offer such opportunities. Medical students are offered support from the Medical Rural Bonded Scholarship scheme, and the Services for Australian Rural and Remote Allied Health (SARRAH) organisation offers scholarships for allied health professional students to attend placement in rural settings. As with most of the organisations that offer scholarships, these are often funded by the Department of Health. Many education providers also offer courses which incorporate regional, rural and remote health in Australia. Most universities offer placements for regional, rural and remote health. These include Central Queensland University, University of Wollongong, University of New South Wales, Charles Darwin University, Charles Sturt University, Flinders University, Deakin University and Curtin University. The importance being placed on regional, rural and remote considerations is demonstrated by the forming of a number of centres for rural health at various universities throughout Australia. The staff in these centres perform research into issues such as mental health, Indigenous health, aged care, health workforce and health and wellbeing for people living in regional, rural and remote communities.

INDIGENOUS HEALTH CONSIDERATIONS IN REGIONAL, RURAL AND REMOTE AREAS

The Indigenous people of Australia are over-represented in mortality and morbidity statistics (AIHW, 2015b). These poorer health outcomes are an indicator of very complex issues relating to Australia's First Nation peoples, requiring greater consideration than a simple set of health problems. There are a range of strategies in place throughout Australian states and territories which aim to return control to Indigenous communities through approaches that are culturally appropriate. One of these is the Closing the Gap campaign (Council of Australian Governments (COAG), 2008) which highlights many of the social issues impacting on health equity. An agreement was made in 2008 between all tiers of the Australian Government to work with Aboriginal and Torres Strait Islander communities to reduce the disparity in healthcare outcomes with the non-Indigenous Australian population. The first two points agreed to by COAG were to 'close the gap in life expectancy within a generation and halve the gap in mortality rates for Indigenous children under five in a decade' (COAG, 2008, p. 14). The four other points made in this agreement relate to improving the education and employment of Indigenous people (COAG, 2008).

The current estimate of the differences in life expectancy of Aboriginal and Torres Strait Islander people compared with non-Indigenous Australians is 10.6 years for a male and 9.5 years for a female (AIHW, 2015b). Information relating to the indicators that may cause a reduction in life expectancy are the principal reason for admission to hospital, numbers of current daily smokers, average daily alcohol consumption, levels of obesity, levels of physical activity and ability to access healthcare compared with need (COAG, 2008).

The *Closing the gap in a generation* (WHO, 2008) report calls for health equity through action on the **social determinants of health**. The social determinants of health are the circumstances relating to dealing with illness. The WHO (2012) states that regardless of where people are born, live or work, or who the government that rules on the day is, all people are entitled to equality in healthcare services. Low levels of health literacy are also seen as an issue that affects the health and wellness of lower socioeconomic groups, with many of these groups living in regional, rural and remote areas (Australian Commission on Safety and Quality in Health Care (ACSQHC), 2015).

Some of the relevant issues include improving access to health prevention and screening strategies, improving access to quality health services and improving the recruitment and retention of **Indigenous health** workers and professionals. To impact on the disparity requires a multi-level, multifaceted and interdisciplinary approach across many diverse professions, only some of which are formally defined as health professions. Such an inclusionary approach includes government policy and budgetary advisers, strategic planners, community agencies, community liaison workers and community representatives, all of whom are essential members of the decision-making process and can affect change in healthcare services (ACN, 2011).

Working in an Indigenous community

Nurses working in Indigenous communities must earn acceptance and respect. This respect is often earned through listening to, and really understanding (i.e. hearing the words and the feelings) what the community identifies as the issues relating to healthcare, and what it considers appropriate solutions to be. The nursing workforce is a facilitator of community ownership

of health through a consultative process underpinned by the principles of reciprocity, respect, equality, responsibility and integrity (ACN, 2011). This approach goes at least some way to ensuring that the community members are not passive recipients of care and enables the health service provider to address the overarching health issues by actively addressing community-identified needs. It is essential that a consultative, respectful approach be taken to ensure that health strategies are embedded within the community structure in a sustainable manner.

The process of consultation aligns with the principles of respect and acknowledgement of traditional beliefs and practices. It also underpins the development of sustainable healthcare based on prevention of illness through education and compliance through understanding. The long-term effects of implementing a community-driven healthcare service include improved mortality and morbidity statistics, a reduction in the representation of Indigenous people in relation to poor health outcomes and the ongoing world presence of a traditional culture for generations to come. Additionally, active consultation and consistent demonstration of cultural respect are safe cultural practice. All health professionals working in Indigenous communities should complete professional development courses in cultural awareness, cultural safety and cultural security prior to going to the community (SARRAH, 2015).

Aboriginal or Torres Strait Islander Health Workers (ATSIHWs) employed in each Indigenous community are the foundation of the health service (see Figure 52.1). The ATSIHWs, as respected community members, often live within the community and have established links that are invaluable to engaging members of the community in healthcare issues. The professional relationship between the nurses and the health workers is a symbiotic one, in which each role relies upon the other to achieve stated outcomes. In summary, a mutually respectful, collaborative, collegial interdisciplinary team approach underpins the success of health initiatives in Indigenous communities (National Aboriginal & Torres Strait Islander Health Worker Association (NATSIHWA), 2012).

FIGURE 52.1 ■ An Aboriginal and Torres Strait Islander Health Worker

Source: © Clive Hyde/Northern Territory Government. AAP Pty Ltd.

THE REGIONAL, RURAL AND REMOTE NURSING WORKFORCE

The roles of nurses in regional, rural and remote communities differ from the roles of their metropolitan counterparts. However, regional, rural and remote nurses can also find that their practice can vary greatly from location to location, depending on the economic, geographical, social, spiritual, cultural and political identity of the individual community. Regional, rural and remote nurses can experience feelings of isolation and stress related to community demographic, lack of multidisciplinary and interdisciplinary support, resource un/availability and the nurses' in/ability to meet the complex health requirements of the communities in which they work. The specific needs of the community, exacerbated by the degree of isolation that can be felt, require nurses to adopt more flexible and creative approaches to clinical problem solving. Such autonomous practice environments may further contribute to the development of professional skills and support the development of an advanced practice or Nurse Practitioner role.

Globally, the nursing workforce is an ageing workforce. Recent statistics from AIHW (2015c) indicate that the average age of nurses working outside major cities is slightly higher than that of their metropolitan counterparts. Detailed statistics of the **regional, rural and remote health workforce** show that the number of clinicians in remote and very remote areas is increasing, particularly for general practitioners (GPs), whose rate is higher than the national average of 108 per 100 000 population (AIHW, 2015c). However, the number of practising GPs is not sufficient for demand in regional, rural and remote areas and this implies an increased workload for the nurses in those communities (Health Workforce Australia (HWA), 2012). The gap in health service provision is often met by experienced nurses adopting a greater role in local health services. Historically, nurses and GPs in rural and remote areas work collaboratively. The loss of professional support may lead to increased feelings of isolation, scope of practice issues and ethical dilemmas for the nurses. However, the lack of GPs in rural and remote areas and the requirement for nurses to adopt greater roles has contributed to the ongoing demand for an increased scope of practice for rural and remote nurses, advanced practice nurses and suitably qualified Nurse Practitioners (HWA, 2012). These roles are supported by local health workers (NATSIHWAH, 2012). The number of registered Aboriginal and Torres Strait Islander health practitioners in Australia rose from 265 in 2012 to 310 in 2013, with the majority of staff working in remote/very remote areas. These were mainly women aged 55 and over (AIHW, 2015c).

It is a common generalisation that people residing in regional, rural and remote areas are resilient and somewhat hardened: 'making do' and 'getting on'. It is also commonly accepted that these traits extend to the nurse in these areas, who does tend to 'soldier on' and does what has to be done with often limited resources. However, for some nurses, the stressors associated with practising in such difficult circumstances can lead to burnout. This can occur in health professionals due to an overwhelming anxiety resulting from the ongoing

expectations and responsibility to patients (Remote Area Health Corp (RAHC), 2015). While burnout is not specific to rural and remote practice, it is unfortunately common in nurses who try to work harder and longer to fulfil the expectations of what can be sometimes unrelenting community demands. This can lead to increasing job dissatisfaction, increased stress levels, work/life imbalance, professional relationship breakdown, personal relationship breakdown and physical deterioration. Such professional side effects have the further potential to negatively impact on nurses in small communities, exacerbated by their high visibility and the overlap between personal and professional identity (RAHC, 2015) (see Figure 52.2).

Recruiting new nurses to rural and remote areas can be challenging for a number of reasons, such as a lack of connection with the community, a change in lifestyle, worries about physical security and a lack of personal support. New graduates may also not possess the advanced clinical competencies required to practise in a regional/rural/remote setting that has limited resources (Hart et al., 2014; Bennett et al., 2012). See the accompanying 'Translation to practice' box.

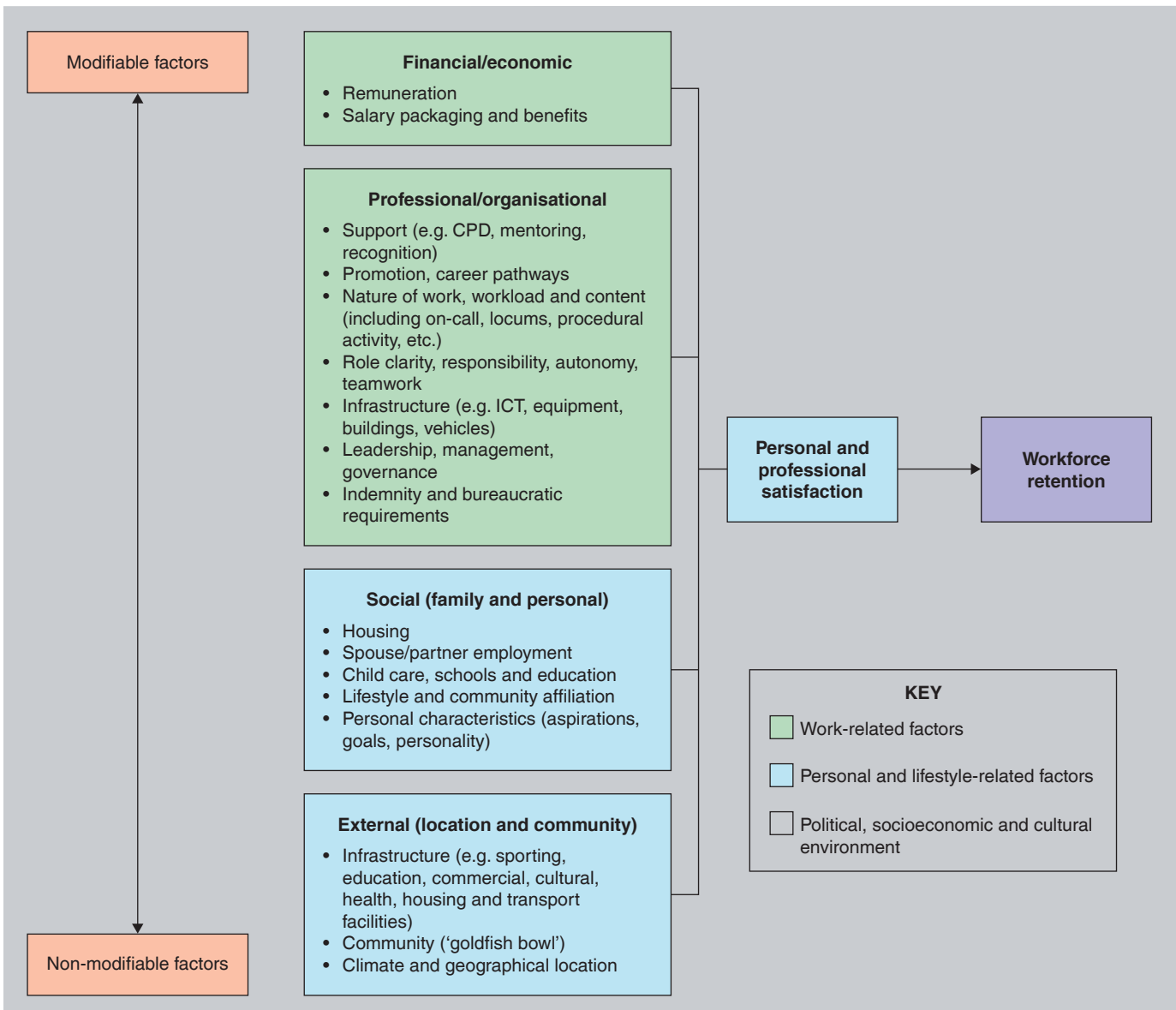

FIGURE 52.2 ■ Factors affecting retention of health workers in rural and remote areas

Source: J. Humphreys, J. Wakerman, D. Pashen & P. Buykx (2009). *Retention strategies & incentives for health workers in rural & remote areas: What works?* The research reported in this paper is a project of the Australian Primary Health Care Research Institute, which is supported by a grant from the Australian Government Department of Health. The information and opinions contained in it do not necessarily reflect the views or policies of the Australian Primary Health Care Research Institute or the Australian Government Department of Health. Retrieved from aphcri.anu.edu.au/sites/aphcri.jagws03.anu.edu.au/files/research_project/292/international_retention_strategies_research_pdf_10642.pdf.

TRANSLATION TO PRACTICE New nursing graduates working in rural communities

Bennett et al. (2012) reported that factors such as personal and professional expectations, unusual workloads and varying levels of support available to staff can cause staff to leave regional, rural and remote areas. Anderson (2012) supported this by explaining that issues such as lack of cultural awareness, inadequate resources, role confusion and limited social support make working in regional, rural and remote areas challenging. Although it is recognised that new graduate nurses may find the transition into the workplace challenging, with ongoing support from employers obstacles can be overcome. The researchers found that issues such as understanding expectations, having social support and reviewing workloads should be an integral part of educating and preparing nurses for practice in regional, rural and remote communities. With the current workforce shortages in regional, rural and remote areas, it is imperative that these recommendations are acknowledged and incorporated in education programs and recruiting processes.

IMPLICATIONS FOR NURSING

- Resource planning by health services can incorporate these findings to individualise the health service to meet the specific needs of the communities. This will also have a significant impact on the skill-mix considerations for staffing health facilities.
- These findings should be considered by nursing professional bodies when addressing the professional representation and development needs of the nurses.
- Undergraduate education providers and the providers of ongoing professional development should consider professional requirements, attributes expected of graduates on completion of their undergraduate degree and curriculum and program design.

CRITICAL THINKING IN PERSON-CENTRED CARE

Some critical-thinking skills linked to consideration of working in a regional, rural or remote area relate to the ability of the nurse to self-assess and identify areas of professional strengths and areas requiring further development.

1. What do you identify as the individual strengths that would support your ability to practise in a regional, rural or remote setting?
2. In which areas of your professional practice do you require further enhancement prior to embarking on a nursing career in regional, rural or remote areas?
3. What do you see as the advantages of engaging with the community to identify the attributes of the nurses working within the community?

Competency to practise

Nurses working in regional, rural and remote areas must possess highly developed skills related to physical assessment, especially those related to traumatic injury. This skill level must include the ability to perform the assessment, note and interpret abnormalities, initiate treatment, initiate communication with emergency transport and personnel as required, and refer to other health professionals locally if available. The nurse in this setting will also initiate access to professional guidance from a distant provider as deemed necessary. Great responsibility is placed on nurses in these situations, with the experience of the practitioner performing the assessment a major factor. The accuracy of initial assessment and timing of referral and transfer can make a major difference in the health outcome. Accuracy of initial assessment, timing of referral, and timing of transfer directly affect the prognosis, with the experience of the practitioner performing the initial physical assessment being a major factor in determining outcomes (Centre for Remote Health, 2015).

Regional, rural and remote areas are not all the same, and the individual characteristics of the region, including the main industries, will contribute to the healthcare requirements of that region. For example, some regional, rural and remote areas rely on farming and agriculture for their local economy, while others may have a mixture of agriculture and mining (see Figure 52.3), or tourism and ecotourism.

FIGURE 52.3 ■ Mining is one of the main economic activities for rural and remote communities

The Nursing and Midwifery Board of Australia provides the opportunity for nurses in isolated areas to be endorsed to administer, obtain, possess, prescribe or supply a scheduled medicine as an isolated or rural practice nurse.

ROLE OF THE NURSE IN REGIONAL, RURAL AND REMOTE AUSTRALIA

The role of nurses in regional, rural and remote areas of Australia is as vast and divergent as the country that surrounds them. The following areas of practice demonstrate the diversity of nursing roles which combine to make up the multifaceted approach to healthcare that these nurses engage in.

Health promotion

Health outcomes are the ultimate deciders, but community education and awareness also reflect the success of health promotion strategies. The role of the nurse is extremely valuable in activities such as multimedia campaigns promoting early detection of cancer by means of screening (e.g. for breast, prostate or bowel cancer) which can result in an increased number of people presenting to screening services. Regional, rural and remote nurses liaise with health service providers and the community; advocate for individuals and for the community regarding access, which may include, for example, the arrival of a breast screen bus; coordinate the local application of service provision and accessibility for community members (ramps, signs); actively promote the event; recruit from the targeted population and facilitate follow-up care as necessary. When the initial health promotion strategy has been completed, the nurse, as part of the interdisciplinary team, is required to evaluate the program and ensure that people requiring further investigation or treatment gain referral to the appropriate service.

Regional, rural and remote nurses actively participate in health promotion in individual, small group or community sessions, or by providing education sessions in the hospital or community setting. Furthermore, it is extremely difficult for nurses in small communities not to be identified as a nurse, so often their professional expertise and advice will be sought during their off-duty hours (e.g. at the local shop), extending their ongoing commitment to the provision of accurate health advice.

Governments and health providers organise national and state-wide rollouts of specific health promotion campaigns that include smaller regional/rural/remote communities. Some of these campaigns will be specifically aimed at residents of country areas; for example, the *Close the Gap* campaign directed at Indigenous health inequality, and the Care for Kids' Ears campaign aimed at highlighting the risk of hearing loss associated with ear disease in Aboriginal and Torres Strait Islander people. Nurses in these communities are actively involved in the planning, implementation and evaluation of the programs. This has further implications for practice and ongoing professional development, and also for the workload of nurses, requiring that the nurses have up-to-date knowledge of the campaign content and the health promotion approach.

Screening and prevention strategies

Health screening programs globally are based on the principle of early detection leading to early intervention, which leads to improved health outcomes for individual people and communities. As previously stated, residents in regional, rural and remote areas of Australia have statistically poorer outcomes for many diseases. This may be partially due to a lack of participation in screening programs, which in turn may be due to a lack of awareness or inability to access screening points. There are a number of reasons—not only confined to regional, rural and remote communities—why people do not participate in screening programs. However, when geographical and cultural isolation are factored in, Australia suffers from a 'tyranny of distance', and there is an increased risk of non-participation. In many cases, strategic planning and service development address this through specialised, localised screening and detection programs, bringing the screening points to the community where at all possible. For example, in sparsely populated areas in Australia, mobile women's health nurses ensure that all women have access to regular, timely screening for breast and cervical cancers, among other health screening services. This model of health screening has also been used in other preventive health strategies, some of which are provided by community organisations as a not-for-profit community service.

Acute assessment

Nurses intending to work in regional, remote and rural settings need to be cognisant of their individual scope of practice and seek to address limitations prior to embarking upon this field of nursing. It should be acknowledged that while nursing education provides a basis for generalist nursing, it is untenable for any single program to cover all discipline perspectives in equal depth. For some graduates, this may necessitate gaining additional experience and education in areas such as aged care, paediatrics, people who live with mental health problems, or primary health screening.

Nurses working in small communities, either in health centres or in hospitals, must be competent to practise in emergency situations. In many instances, the nurse will be working alone or with only limited support from other health professionals. Some of the skills required in emergency situations are outlined below.

Unexpected illness or trauma are two of the main reasons why people living in sparsely populated areas seek healthcare. Road trauma, workplace injuries, mental health and farm-related injuries require the nurse on duty to have a high level of competence in acute health assessment, including physical and mental health assessment, and an in-depth understanding of the particular type of injuries and presentations associated with these conditions. Injuries received in motor vehicle accidents can range from minor to life threatening, requiring immediate intervention. In a regional, rural or remote area, the availability and timely response of emergency assistance can be varied at best. Therefore, many regional, rural and remote residences are equipped with emergency medical kits, provided through organisations such as the Royal Flying Doctor Service (RFDS). Medical advice can also be sought through radio or telephone contact: a suitably qualified professional will instruct the person at the site of the emergency what to do. Nurses in regional, rural and particularly remote areas can be called upon to receive the person in the local health service clinic or hospital; or to assist in the **retrieval** process; or they can be called upon in their role as neighbour and community member.

Nurses working in regional, rural and remote areas must be prepared to provide care for people with a broad spectrum of clinical presentations including mental illness. In Australia, in 2007, 45% of the population aged between 16 and 85 years stated that they had been affected by anxiety, mood (affective) or substance

use disorder (ABS, 2012), so the probability that the nurse will be confronted by a person with a mental health issue is very high. While it is not feasible for any one person to know each and every possible clinical presentation, nurses must be able to identify deviations from normal and seek appropriate assistance in a timely manner to ensure optimal outcomes for the person presenting to them for help. In all areas of nursing practice, irrespective of the context, assessment forms the cornerstone of best practice. In regional, rural and remote areas, enhanced assessment skills are the basis upon which management of the person and significant clinical decisions are made. Astute assessment provides the nurse with the requisite decision-making capacity. It is essential to be able to assess, plan, implement and evaluate treatment based on psychological, physiological and pathophysiological knowledge. Nurses are expected to identify and interpret data before responding appropriately. The presenting problem of the person will guide the order and priority of the type of assessment to be undertaken, but will not limit the extent to which the nurse will perform a full physical and psychological assessment. For example, while some individuals will not present with any obvious physical injury or physiological dysfunction, they may exhibit signs of distress, altered mental health state and/or drug and alcohol issues. Acute presentation of injuries or illnesses acquired may be further complicated by co-morbidities or by combinations of two or more problems (e.g. trauma and drug or alcohol problems). In addition to the varied physical and psychological presentations, nurses will be required to provide care to people who come from diverse cultural backgrounds and from across the lifespan.

Emergency nursing care

Triage

In remote communities and health sites across Australia, some degree of emergency care for communities is provided. The health outcomes are influenced in part by the large distances to travel, the time taken for retrieval and emergency stabilisation, and the emergency skills of the local practitioners (HWA, 2011). For **triage in regional, rural and remote areas**, nurses may use the Australasian Triage Scale (ATS, see Table 52.1) but this may be quickly followed by the nurses themselves initiating emergency treatment. In situations such as this, the ATS is applied according to nurse waiting times.

The ATS categories determine the urgency with which the person's presenting problem must be treated. In regional, rural and remote areas, the deterioration of the person's condition must be considered with regard to the response time of appropriate personnel and the needs of the individual to be triaged. Therefore, the triage category may be higher in anticipation of the challenges of assembling qualified personnel and required equipment. The triage categories are based on the presumption of morbidity if treatment is not initiated within the identified time to treatment. The categories also include behavioural or mental health disorders, which pose a threat of danger to self or others.

By nature, triage is necessarily contingent upon context; in such settings the attending nurse will use advanced decision-making processes to determine the person's need for emergency care and put in place the resources available to expedite time-critical interventions.

PRIMARY SURVEY A primary survey is focused on identifying life-threatening conditions and the need for emergency first aid. In the regional, rural and remote context, nurses may be called to a scene to provide assistance or they may be the only staff member in the local healthcare facility. In these situations, having the means by which to summon assistance is also a priority, requiring the nurse to be competent at emergency transmission via the telephone or by radio.

Understanding the principles of Danger, Response, Send for help, Airway, Breathing, Circulation, Disability (neurological) and Exposure is essential in the provision of safe and effective care. To provide appropriate and expedient care the nurse must possess effective assessment skills and be able to interpret findings accurately, in addition to having a working knowledge of all resources available. This includes awareness of where to locate, and how to use, emergency resuscitation equipment.

Some members of the community require special consideration during primary survey and physical assessment. Care of children in emergent situations requires more than merely size modification of adult assessment and management. Children are more susceptible to heat loss, and their fluid and medication requirements are different from adults. During the primary survey, nurses must incorporate knowledge of the physical, cognitive, emotional and behavioural stage of development into the interpretation of findings. Likewise, the older adult has altered states resulting from the normal ageing processes or disease processes which will influence the interpretation of assessment findings. This group is also at higher risk of complications from co-morbidities or as a result of concurrent treatment regimens. The older adult and the child are only two examples that demonstrate the breadth of clinical practice needed by nurses working in regional, rural and remote areas.

SECONDARY SURVEY A secondary survey is not performed until the primary survey has been completed, findings have been interpreted and documented, and the prioritised interventions implemented. It is generally accepted that the secondary survey entails a comprehensive head-to-toe physical assessment. This physical assessment includes inspection, palpation, auscultation and percussion of the body systems.

Physical assessment is not the only form of assessment that nurses working in regional, rural and remote areas must be proficient in performing. Mental health assessment is also frequently performed, and requires the nurse to possess a sound understanding of assessment processes, manifestations of mental illness and treatment strategies. True holistic person-centred care includes acknowledgement of the whole person as well as the lifestyle factors influencing the person's health and wellbeing.

Depression has been recognised as a major health issue in all Australian communities and is a health priority area throughout Australia. Global statistics of suicide in young men living in rural areas show consistently higher rates in this demographic than in the general population (Jones et al., 2015; Kennedy et al., 2014; Hirsch & Cukrowicz, 2014). There are a range of factors that may influence the rates of suicide in young men living in regional, rural and remote areas, including mental illness, physical illness, financial stress, isolated residence, increased

TABLE 52.1 The Australasian Triage Scale

ATS CATEGORY	RESPONSE	DESCRIPTION OF CATEGORY	CLINICAL DESCRIPTORS (INDICATIVE ONLY)
Category 1	Immediate simultaneous assessment and treatment	**Immediately life threatening** Conditions that are threats to life (or imminent risk of deterioration) and require immediate aggressive intervention.	• Cardiac arrest • Respiratory arrest • Immediate risk to airway—impending arrest • Respiratory rate < 10/min • Extreme respiratory distress • BP < 80 (adult) or severely shocked child/infant • Unresponsive or responds to pain only (GCS < 9) • Ongoing/prolonged seizure • IV overdose and unresponsive or hypoventilation • Severe behavioural disorder with immediate threat of dangerous violence
Category 2	Assessment and treatment within 10 minutes (assessment and treatment often simultaneous)	**Imminently life threatening** The individual's condition is serious enough or deteriorating so rapidly that there is the potential of threat to life, or organ system failure, if not treated within 10 minutes of arrival or **Important time-critical treatment** The potential for time-critical treatment (e.g. thrombolysis, antidote) to make a significant effect on clinical outcome depends on treatment commencing within a few minutes of the individual's arrival in the ED or **Very severe pain** Humane practice mandates the relief of very severe pain or distress within 10 minutes	• Airway risk—severe stridor or drooling with distress • Severe respiratory distress • Circulatory compromise — Clammy or mottled skin, poor perfusion — HR < 50 or > 150 (adult) — Hypotension with haemodynamic effects — Severe blood loss • Chest pain of likely cardiac nature • Very severe pain—any cause • BSL < 2 mmol/L • Drowsy, decreased responsiveness any cause (GCS < 13) • Acute hemiparesis/dysphasia • Fever with signs of lethargy (any age) • Suspected meningococcaemia • Acid or alkali splash to eye—requiring irrigation • Major multi-trauma (requiring rapid organised team response) • Severe localised trauma—major fracture, amputation • High-risk history: — Significant sedative or other toxic ingestion — Significant/dangerous envenomation — Severe pain suggesting PE, AAA or ectopic pregnancy • Behavioural/psychiatric: — violent or aggressive — immediate threat to self or others — requires or has required restraint — severe agitation or aggression
Category 3	Assessment and treatment start within 30 minutes	**Potentially life threatening** The patient's condition may progress to life or limb threatening, or may lead to significant morbidity, if assessment and treatment are not commenced within 30 minutes of arrival or **Situational urgency** There is potential for adverse outcome if time-critical treatment is not commenced within 30 minutes or Humane practice mandates the relief of severe discomfort or distress within 30 minutes	• Severe hypertension • Moderately severe blood loss any cause • Moderate shortness of breath • SAO_2 90–95% • BSL > 16 mmol/L • Seizure (now alert) • Any fever if immunosuppressed (e.g. oncology patient, steroid prescription) • Persistent vomiting • Dehydration • Head injury with short LOC—now alert • Moderately severe pain—any cause—requiring analgesia • Chest pain likely non-cardiac and moderate severity • Abdominal pain without high-risk features—moderately severe or patient age > 65 years • Moderate limb injury—deformity, severe laceration, crush • Limb—altered sensation, acutely absent pulse • Trauma—high-risk history with no other high-risk features • Stable neonate • Child at risk of abuse/suspected non-accidental injury

(continued)

TABLE 52.1 The Australasian Triage Scale (continued)

ATS CATEGORY	RESPONSE	DESCRIPTION OF CATEGORY	CLINICAL DESCRIPTORS (INDICATIVE ONLY)
			• Behavioural/psychiatric: — very distressed, risk of self-harm — acutely psychotic or thought disordered — situational crisis, deliberate self-harm — agitated/withdrawn — potentially aggressive
Category 4	Assessment and treatment start within 60 minutes	**Potentially serious** The individual's condition may deteriorate, or adverse outcome may result, if assessment and treatment is not commenced within 1 hour of arrival in ED. Symptoms moderate or prolonged. or **Situational urgency** There is potential for adverse outcome if time-critical treatment is not commenced within 1 hour or **Significant complexity or severity** Likely to require complex work-up and consultation and/or inpatient management or Humane practice mandates the relief of discomfort or distress within 1 hour	• Mild haemorrhage • Foreign body aspiration, no respiratory distress • Chest injury without rib pain or respiratory distress • Difficulty swallowing, no respiratory distress • Minor head injury, no loss of consciousness • Moderate pain, some risk features • Vomiting or diarrhoea without dehydration • Eye inflammation or foreign body—normal vision • Minor limb trauma—sprained ankle, possible fracture, uncomplicated laceration requiring investigation or intervention • Normal vital signs, low/moderate pain • Tight cast, no neurovascular impairment • Swollen 'hot' joint • Non-specific abdominal pain • Behavioural/psychiatric: — Semi-urgent mental health problem — Under observation and/or no immediate risk to self or others
Category 5	Assessment and treatment start within 120 minutes	**Less urgent** The individual's condition is chronic or minor enough that symptoms or clinical outcome will not be significantly affected if assessment and treatment are delayed up to 2 hours from arrival or **Clinico-administrative problems** Results review, medical certificates, prescriptions only	• Minimal pain with no high risk features • Low-risk history and now asymptomatic • Minor symptoms of existing stable illness • Minor symptoms of low-risk conditions • Minor wounds—small abrasions, minor lacerations (not requiring sutures) • Scheduled revisit (e.g. wound review, complex dressings) • Immunisation only • Behavioural/psychiatric: — Known patient with chronic symptoms — Social crisis, clinically well individual

GSC = Glascow Coma Scale; BSL = blood sugar level; PE = pulmonary embolism; AAA = abdominal aortic aneurysm; LOC = level of consciousness

Source: Australasian College for Emergency Medicine (ACEM) (2005). *G24 Guidelines for the Implementation of the Australasian Triage Scale in Emergency Departments.* Revised November 2013. Retrieved from www.acem.org.au/getattachment/d19d5ad3-e1f4-4e4f-bf83-7e09cae27d76/G24-Implementation-of-the-Australasian-Triage-Scal.aspx.

availability of lethal means, male stereotyping and reluctance to seek help. These issues are often exacerbated by variations in mental health services in these areas (Jones et al., 2015; Kennedy et al., 2014; Hirsch & Cukrowicz, 2014).

The *2015 Australian National Drug Strategy Household Survey* (AIHW, 2013) revealed that people living in remote and very remote areas were more likely to smoke, drink at risky levels and use cannabis and methamphetamines. These data provide essential background knowledge for nurses working in these areas, to ensure that they possess the critical-thinking ability to assess each individual situation. The data may be relevant to opportunistic intervention and treatment, early detection and accurate monitoring and management of symptoms.

TYPES OF QUESTIONING In emergency situations, specific closed questioning is often appropriate. For example, when the person is short of breath and complaining of pain, the nurse should use closed questions to extract the relevant information in the shortest time (see Box 52.1 for examples).

Open questions require the person to respond in more than one word, allowing greater depth of information to be obtained (see Box 52.2). Open questions are the style predominantly used for mental health assessments where in-depth information is required in order to gain a comprehensive understanding of the presenting problem or issue. Try not to ask two questions as one, such as 'What car were you driving and was it yours?' These types of questions are confusing and should be asked separately.

BOX 52.1 Closed questions

- Where is the pain?
- On a scale of 1–10, how much does it hurt?
- Does your arm hurt?
- Does it hurt when you breathe in?
- Does it hurt when you breathe out?
- Is the pain any better now?
- Have you had this pain before?

BOX 52.2 Open questions

- Can you describe what the pain is like?
- Tell me what were you doing when this pain started?
- What have you done since the pain started?
- Which types of pain relief have you tried?

In all aspects of nursing care in all settings—metropolitan, regional, rural or remote—nurses must be proficient communicators to gain relevant information for care planning and to establish a supportive relationship with the person, their family and/or their carers. In some cases, if the person or their significant other is distressed, the presence of the nurse to listen and provide support through silent empathy is invaluable.

Establishing boundaries

As previously mentioned, nurses working in small regional, rural and remote communities are less anonymous than their urban counterparts, a consequence of which may be that a more considered commitment to the protection of each person's privacy and confidentiality is required. Personal communication can be either a barrier or a bridge in these communities. One of the most important factors in establishing a professional therapeutic relationship is that of trust. The person must feel comfortable discussing their personal information and health history with the nurse providing care. This can be difficult in small communities where the nurse may know the person through social and community interactions. While it is also possible for this to occur in larger towns or cities, it is far more likely to occur in communities where the population is smaller and the possibility of crossing paths in various roles is greater. For example, the person may be the manager of the local bank which holds the mortgage over the nurse's property. As in all professional interactions, the person requires reassurance that they will be treated with dignity, integrity and professionalism in the course of receiving healthcare—expectations which may be intensified and become challenging in smaller communities. It is thought that some people in small communities have sought healthcare away from their home areas to minimise the chance of knowing their care provider socially. Therefore, it is imperative that the nurse is able to reassure the person by verbal and nonverbal means during any interactions that they will uphold confidentiality at all times.

Subjective data on individuals are obtained through a range of clinical approaches which are each contingent upon the clinical context specific to the person. Both over-familiarity and failure to acknowledge the person may be perceived as equally offensive. The degree of familiarity will depend on each individual and the type of healthcare they require. Nurses will have to use their judgment and exercise it for each individual circumstance. One way to approach this is for the nurse to actually ask the person what degree of familiarity they would like. An example of this would be: 'If I see you in the shops how would you like me to behave? Do you want me to say hello or would you prefer I just went about my business without acknowledging you?' Establishing mutually agreed upon boundaries will prevent uncomfortable future situations.

The type and manner of the interview will depend largely on the information required. If the person is already known to the nurse, the beginning phase may be briefer. In many cases, it may be advisable to approach the interaction as if beginning a new relationship. Gaining the relevant information should not be influenced by previous interactions because each occasion where the person is seeking healthcare is a new and slightly different occasion. Closing the interview may involve reference to seeing the person in the community, but should clearly reinforce the respect for confidentiality.

Teleconsultations/videoconsultations

The *Millennium Development Goals Report 2015* (United Nations, 2015) states that with regard to achieving the millennium goals related to the improvement of mortality and morbidity statistics, the global use of mobile communication services has been growing at a fast rate and is seen to be a major contributor to the success of the initiative. Access to information has the ability to increase the knowledge of individuals and communities, leading to improved, informed decision making and, ultimately, improved health outcomes—empowerment through knowledge. The objectives of these goals continues with 17 new sustainable goals being implemented to be developed by 2030 (United Nations, 2015).

In modern Australia, the availability of telecommunications services and the internet is growing. The introduction of the Australian National Broadband Network (NBN) is assisting to bridge the gap of telecommunication access. However, in many rural and remote areas these services are limited and unreliable. The ability to phone in or dial in to include a specialist in the assessment of, or discussion with, a person in a regional, rural or remote area has the potential to be an immensely valuable tool. Expert opinion remains divided, with some people viewing **telehealth** as a time-efficient, labour-efficient and cost-effective method of delivering health services to the bush, for which long-term investment in the system is extremely worthwhile. The monetary savings can include the cost of a videolink instead of the cost of the person and carer travelling to the service or their appointment, and their accommodation. That said, patient transport schemes do not cover all expenses. They merely subsidise the person's costs and most people who have to travel to access healthcare are financially disadvantaged. The humanitarian savings of a reliable system are immeasurable: the assumption is that the person is not removed from their home area; they remain with family, friends and support networks; and they do

not have the burden of finding someone to care for family, property, animals or business for the period of their absence.

From a nurse's perspective, having access to clinical expertise while geographically isolated is reassuring. An example of this is the access to emergency mental healthcare in rural and remote emergency departments in rural Australia. Staff at rural locations have access to a telepsychiatry program to assist with emergency mental health issues (Saurman, Kirby & Lyle, 2015).

The introduction of telehealth to provide specialist advice to people living in regional, rural or remote areas of Australia has been quite an achievement. This service facilitates specialist care for an individual so that they do not have to leave their local area. A **referral** to a specialist can be from a nurse or medical practitioner, midwife, practice nurse or Aboriginal or Torres Strait Islander Health Worker on behalf of a medical practitioner (Department of Health, 2015). Among other potential benefits, telehealth enhances a sense of professional support for nurses and other health professionals in regional, rural and remote settings.

The provision of professional support not only provides timely guidance but may well impact on the recruitment and retention issues surrounding nursing in regional, rural and remote areas. This is especially so as nurses in these areas report a perception of professional isolation as a contributing factor to leaving these roles (Bennett et al., 2012). The provision of professional support is extended to include online access to professional databases for current publications and best practice guidelines. This access relies heavily on the motivation of the practitioner to access, interpret and adopt current practice standards, which is more difficult for nurses who are already mentally, emotionally and physically fatigued. Many professional groups and colleges now offer online professional development opportunities for nurses in a vast range of specialties. However, the lack of reliability of access and the potential cost associated with such programs may preclude use by some regional, rural and remote nurses.

The uptake of telehealth has been a major boost for people living in regional, rural and remote locations. The ability to access specialist health professionals in metropolitan and city locations is helpful for people suffering health conditions as well as the health professionals situated in those areas. Emergency mental health, rheumatoid arthritis and oncology clinic consultations are just some examples of effective service provision by telemedicine (Saurman, Kirby & Lyle, 2015: Sabesan et al., 2014: Roberts et al., 2012). The Royal Flying Doctor Service (RFDS) also provides medical consultation to people living or travelling in regional, rural and remote Australia (RFDS, 2012a).

Liaison and advocacy

Nurses working in regional, rural and remote areas of Australia must possess a high standard of interpersonal skills and be able to communicate effectively with their community, nursing colleagues, members of the interdisciplinary team, local health service providers, tertiary referral centres, retrieval teams and other organisations involved in the transfer of people in their care (e.g. the RFDS). In remote Indigenous communities, local health workers form the backbone of the health service, integrating community knowledge, cultural knowledge and health knowledge into individualised person-centred care. It is therefore imperative that nurses in these areas work closely with other health workers to ensure that the healthcare provided is culturally safe and acceptable to the community.

Referral and retrieval

Preparing a person for referral to a metropolitan or city health service is a part of the daily routine for regional, rural and remote nurses, especially when dealing with acute illness or trauma.

Public hospitals in central Australia are noticeably absent, but the RFDS is notable for its broad range of services throughout Australia, including the sparsely populated areas. The RFDS began in 1928 when Reverend John Flynn initiated a flying medical service to isolated inland communities (RFDS, 2012a). The service has continued to grow and is funded by the Australian Government to provide aeromedical transport and healthcare services to people living in regional, rural and remote areas of Australia. The RFDS provides a 24-hour medical evacuation service staffed by highly skilled flight nurses and medical practitioners, primary and community healthcare clinics. It also provides remote consultations via phone or radio by medical practitioners or nurses, and supplies medical chests for emergency use (RFDS, 2012b). Although access to services in regional, rural and remote regions is improving due to the National Strategic Framework for Rural and Remote Health, the RFDS can provide invaluable support for practitioners where there are gaps in service provision. In many cases, the RFDS provides essential lifelines for the practitioners and communities (Department of Health, 2012).

Correctly following transfer and retrieval guidelines and policy is extremely important to ensure that the process proceeds smoothly. Such professional actions may not be noticeable until after the transfer is complete. All centres have criteria for transfer, clearly articulated in policies and procedures, often with quick reference guides for use in emergent situations. The process usually follows a flowchart or document with a 'checkbox' format. The accurate handover of relevant information is essential to the provision of appropriate care during the transfer and on arrival at the destination. In some cases, the nurse completing the checklist will be the nurse escorting the person to the referral centre. However, in other instances, the transfer will be by outside organisations such as ambulance services, emergency services or the RFDS. Nurses working in these areas must be fully aware of these transfer procedures prior to an emergency, as it is not the right time to be reading policies when dealing with a critically ill or injured person who requires immediate attention.

Discharge planning and continuum of care

When providing nursing care to a person in any health facility in any community, it is imperative that **discharge planning** is incorporated into all aspects of person-centred care. Commencement of discharge planning at the initial point of contact is a fundamental nursing consideration, whether the person presents with a chronic condition or acute illness, or as a result of trauma. The role of the nurse is to assist the person to return to their home having regained their former level of independence if possible, or having achieved an optimal level of independence or mental health recovery resulting from their altered physical, psychological or cognitive ability.

Therefore, the discharge of a person within a regional, rural or remote community or back to a rural or remote community from an urban setting must be done proficiently and acknowledge the individuality of the person and their needs. It must also take into account the resources and support available in their community. If the discharge process is not performed appropriately, the chance of readmission is increased by limited availability of follow up, limited contact with health professionals and limited access to healthcare facilities.

Provision of care for the person from a regional, rural or remote area

In all hospitals in every metropolitan or city centre in Australia, nurses will be providing care for people who have had to travel, sometimes long distances, for treatment. These people have a unique set of challenges for inpatient treatment, upon discharge and for the **continuum of care**. The following considerations relate to nursing and discharging people who are from regional, rural and remote areas.

Considerations

The person is likely to feel isolated and lonely. Their support systems are often hundreds of kilometres away, which will impact on their mental health and their ability to heal. When the person is from a regional, rural or remote area, discharge planning becomes even more significant in the overall care of the individual. When preparing to transfer a person back to their home area, or to discharge them to travel home, nurses must ensure that they take all aspects of transport into consideration (see Box 52.3).

The financial costs associated with transport must be incorporated into care planning and discharge planning. All states and territories have implemented a state-specific model of patient travel subsidy, identified by different titles according to whichever state or territory is the place of origin. In contemporary healthcare, with the continuous state of reduced bed availability, it is not uncommon for people to be transferred across jurisdictional borders to receive specialised medical care.

In some cases, the person may be required to fund their return home and claim expenses at a later date. Claiming back the costs may be time consuming and exhausting for a person who may still be recuperating from significant illness or injury. Therefore, the cost of transport becomes increasingly important. New South Wales has the Isolated Patients Travel and Accommodation Assistance Scheme (IPTAAS) (NSW Government, 2015a). This scheme is for people who have to travel significant distances to access specialist medical treatment which is not available locally (NSW Government, 2015a). Many people from the Northern Territory are transported to Adelaide for tertiary-level healthcare. The cost of getting back home is supported through the Patient Assistance Travel Scheme (PATS). This scheme or others like it are available to all people living in rural and remote Australia in each state and territory (Department of Health, 2014b). The cost of transport for a carer is often covered by the transport scheme, but this can be variable. If there is more than one support person, individuals may be required to pay the full costs. For example, this may be a problem if the person has a great distance to travel, is required to change modes of transport at some stage during the journey and the carer is not physically able to assist. In these cases, there is an increased need for the assistance of another support person or carer. In the case of the transfer of a child, there may be two parents to accompany the child, one of whom will travel with the child regardless of transport and the remaining parent who will need to organise their own transport and perhaps that of other children returning home. This may also occur when an older parent is travelling with their spouse and an adult child is required to be a further support person. There are support groups or volunteer organisations that can assist with emergency funding in times of financial difficulty for people and their families. Transport for treatment or returning home from treatment is deemed eligible for this purpose in many instances.

The person returning home will also need to have adequate supplies of medications and medical supplies, as access to a supplier may be limited (National Rural Health Alliance Inc., 2014). Contact should also be made with the pharmacy in the person's local area to give sufficient time for ordering and receiving supplies.

BOX 52.3 Considerations when discharging or transferring a person

Transport availability

What type of transport is available?
- Private motor vehicle
- Aircraft (commercial)
- Aircraft (specialist provider)
- Bus
- Train
- Marine transport (some island communities are only accessible by water)

How frequently does this service run?
- Some services run only twice a week or weekly
- On demand

Will this cost money?
- How much?
- Who will pay?

Is the person able to travel via this mode of transport?

Can they spend long periods of time sitting?
Are they able to mobilise as necessary?
Can they board the transport?
Do they require a stretcher/lift to board?
If travelling by air, can the person tolerate the pressure changes?
Can this mode of transport accommodate the person's needs?

Carer support

Can the person travel unaccompanied?
Will the person require a nursing or medical escort?
Can the carer/escort travel with the person?

In many regional, rural and remote areas of Australia, residents and services are quite often restricted by seasonal weather patterns. For example, in the northern areas of Australia, the wet season between November and March can significantly impact on the mobility of the population and access to services. The coming of the rains usually brings great anticipation to the residents of the outback, especially those reliant on the water for crops, stock, replenishing water supplies and rejuvenating the bush flora. However, while the rains are essential to the lifeblood of the community, the associated flooding or limiting of access can range from being mildly frustrating to intensely problematic (see Figure 52.4). The lack of access can affect the ability of the person requiring healthcare to access the relevant services, and it can limit the ability of the health service to discharge the person.

Community care

Hospitals are fast becoming places for people who are treated for acute illnesses and conditions. Many people (under 65 years of age, or Aboriginal and Torres Strait Islander people under 50 years of age) are now treated at home for conditions that restrict their normal daily living or who are living with a disability. There are also services for frail older people with dementia, those who are financially disadvantaged or those in remote or isolated areas still living in their home (NSW Government, 2015b). There are a range of resources available through community care services such as in-home services, community-based services, clinical services, respite, counselling and carer support. People who are having difficulty with their daily activities of living and are at risk of losing their independence are eligible for these services.

FIGURE 52.4 ■ Rain is necessary to survival but it can isolate people and communities. Children often make the most of being restricted to the home area

Source: Adele Baldwin.

In 2010 the Productivity Commission launched a public enquiry regarding long-term disability care and support services. The National Disability Insurance Scheme (NDIS) was established in 2011 when COAG agreed to the need for reform for disability services (Queensland Government, 2015). The service provides referrals and support services for people with a disability so that they can achieve their goals and be in a position to enjoy their lives (NDIS, 2015).

TRANSLATION TO PRACTICE The perceptions of people living in regional, rural and remote areas

The reduced level of services available for residents of regional, rural and remote communities who required cancer treatment was the subject of a study by Moorin et al. (2012). The study highlighted that, unbelievably, some older people who live in remote areas of Western Australia accept that services are not available to them due to isolation, and that cancer is part of growing older. Of particular concern is evidence to support that up to 12% of Indigenous Australians living in regional rural and remote areas have difficulty with transport to cancer services compared with only 4% of non-Indigenous Australian people (Moorin et al., 2012). This discrepancy has also been highlighted for mental health services for older people living in rural areas in South Australia (Henderson et al., 2014). The difficulties associated with travel for medical consultation or for long-term treatment highlight the need for continuity of care across health services and the need for consideration of this to be incorporated into the care received in the referral centre and the support services available.

IMPLICATIONS FOR NURSING PRACTICE

Research projects such as these provide significant evidence of the importance of both physical and psychosocial support to the experience of, and recovery from, serious illness. The provision of support measures adapted for use in regional, rural and remote areas is not insurmountable.

The difficulties associated with living in regional, rural and remote areas are exacerbated in times of extreme vulnerability related to serious illness. The role of the nurse can impact positively and result in improved quality of life for people and their families.

CRITICAL THINKING IN PERSON-CENTRED CARE

1. Using your current knowledge about holistic person-centred care, how can a perceived lack of psychosocial support influence the person's response to treatment?
2. As a nurse working with people in this or similar situations, what are some strategies you could implement to meet the psychosocial needs of the person and their families?
3. People in these situations require significant support from their local community, but confidentiality is often a concern. Can you identify some community-based initiatives that would contribute to the overall wellbeing of these people?

Sources: Moorin et al. (2012). Challenging the perceptions of cancer service provision for the disadvantaged: Evaluating utilisation of cancer support services in Western Australia. *Support Care Cancer*, *20*(8), 1687–1697; Henderson et al. (2014). Meeting unmet needs? The role of a rural mental health service for older people. *Advances in Mental Health*, *12*(3), 182–191.

CHAPTER HIGHLIGHTS

- The formal acknowledgement of the specialised nature of regional, rural and remote nursing is driving the adoption of greater clinical responsibility and advanced practitioner roles.
- Community-driven healthcare addressing the needs of the community, as identified by the community, is a key feature of healthcare provision in regional, rural and remote areas.
- Equity in access to quality healthcare in a timely manner is the foundation for improving health status in regional, rural and remote communities.
- Health promotion, including early detection and prevention strategies, is a vital component in regional, rural and remote healthcare provision.
- Incorporating rurality into the provision of nursing care is not the sole domain of the regional, rural and remote nursing workforce.
- Factors impacting on the continuum of care for people living in regional, rural and remote areas must be addressed within the care and management provided by all nurses, regardless of location.

CONCEPT CHECK

1 When triaging a person, the primary survey includes:
1. rescue, retrieval and response
2. assessment of danger, response, send for help, airway, breathing, circulation, disability, exposure
3. calling for immediate transfer
4. assessment of available resources

2 The 2013 National Drug Strategy Household Survey showed alcohol consumption at which level for people living in regional, rural and remote areas?
1. low
2. moderate
3. moderate–high
4. risky–high risk

3 The considerations for discharge planning for people from a regional, rural or remote area:
1. are the same as those for all people regardless of location
2. include access to follow-up care and support services
3. include the assumption that the person will return home by the same means as they arrived
4. are the domain of the home health service

4 The challenges faced by regional, rural and remote communities in accessing healthcare:
1. are driven only by economic forces
2. are contingent upon the local weather at the time
3. are the same as those faced by all communities in Australia
4. represent a multi-level, multifaceted integration of diverse influences

5 Community engagement with rural and remote health services is vital because:
1. the community should like whoever is working for them
2. it makes the people feel better even though the health service knows best
3. the community will vote for the government that gives the most money
4. the sustainability of a service depends on community involvement

6 Three of the five principles of working with Indigenous people are:
1. reciprocity, respect, equality
2. spirituality, respect, equality
3. economics, culture, demographics
4. spirituality, culture, demographics

7 Nurses working in regional, rural and remote areas are predisposed to burnout as a result of:
1. community support
2. the opportunity to develop a broad range of clinical skills
3. the building of professional relationships with a smaller group of co-workers
4. longer work hours because the available resources are limited

8 Establishing and maintaining a therapeutic relationship with a person is:
1. easier in a small community because the nurse knows everyone already
2. more difficult in a small community where everyone knows everyone else
3. easier in a small community because the patients are the same ones all the time
4. more difficult in a small community because nurses cannot ethically provide care for people they already know

9 Clinical competency in rural and remote areas is:
1. directly linked to all domains of the NMBA *National Registered Nurse Standards for Practice (2016)*
2. directly linked to all domains of the National Health and Medical Research Council guidelines
3. not very important because all of the high-level cases are transferred to larger centres
4. defined by a specific set of regional, rural and remote competencies

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UNIT 14 BUILDING CLINICAL COMPETENCE

Special topics in medical–surgical nursing

CASE STUDY

A Registered Nurse with 2 years' postgraduate experience commenced working at a small rural hospital 2 weeks ago. This is her second afternoon shift in charge of the hospital's 10 beds, of which five are occupied by long-term residents. A very experienced Enrolled Nurse is working alongside her and has been a great source of advice and information on where to find things and who to contact.

The dreary day outside has suddenly turned violent with gale-force winds, intermittent hail and torrential rain. The lights flickered into a brief moment of total darkness before the hospital generators kicked in. The ageing phone system is not working, so it appears that the Registered Nurse and the Enrolled Nurse are isolated at this time.

At first the knock on the door sounds like trees or doors banging in the wind, but when the Registered Nurse goes to investigate, there are three young men at the door in various states of distress and with obvious physical injuries. One of the young men appears to have a head injury, is being held up by his mates and is losing consciousness as the nurse watches. The Registered Nurse calls out to the Enrolled Nurse who brings the trolley to transport the man into the assessment area. The man with the head injury is now not responding to verbal commands and requires assistance to maintain his airway. As the communication lines are down, and the man's mates are injured, the Enrolled Nurse runs to the nearby doctor's residence to request medical assistance.

Critical thinking questions

1. If you were the Registered Nurse in this situation, how do you think you would react?
2. How would you ensure that you are still working within your scope of practice?
3. What are some of the ethical considerations of this case study? Does this constitute a professional dilemma? How did you come to this conclusion?
4. What do you think are some of the contributing factors to the above decisions being made?
5. If you think that this is not the right way to address this situation, what could the nurses have done differently?

CASE STUDY 2

Maryanne Freeman, aged 69 years, and her husband James, 72 years, prepared to travel from their home in Remoteville to the nearest metropolitan hospital in Regionalton, where Maryanne is to undergo removal of a cataract and implantation of an intraocular lens to her right eye. Maryanne has a history of type 2 diabetes and osteoarthritis. James has a history of pacemaker insertion and congestive cardiac failure, and he and Maryanne manage independently. The couple are fortunate that the RFDS clinic run is able to transfer them to Regionalton. While Maryanne is hospitalised, James is able to stay in hospital accommodation.

The operation goes smoothly and Maryanne remains as an inpatient for 3 days postoperatively. Maryanne is seen by the ophthalmic surgeon on the third morning and is cleared for discharge. The attending nurse provides education to both Maryanne and James, instructing them on how to instil eye drops, the need for Maryanne to cover her eyes at night and the need to continue wearing dark glasses. James becomes concerned and asks the nurse how they will get back home. The nurse contacts the hospital social worker who books two bus tickets for the journey and a taxi voucher for transfer from the hospital to the bus depot. James then asks if they could go back with the RFDS as the road journey is a 10-hour trip and he is concerned that Maryanne cannot sit that long on the bus. The social worker does not think that returning with the RFDS is possible. As the bus was to depart at 7 am, James became quite anxious that they would miss it and that they would need to leave the hospital at 6 am before Maryanne had had breakfast. The pharmacy organised discharge medications the night before.

The bus departed at 7 am and the couple endured the 10-hour journey home.

Critical thinking questions

1. What are some of the practical concerns with this case study?
2. Were James's concerns valid and/or acknowledged?
3. Was the discharge planning individualised for Maryanne?
4. Can you identify any other considerations the nurse and the social worker should have included in their provision of care?

APPENDIX STANDARD PRECAUTIONS

Standard precautions are safe work practices that are able to be applied to all patients regardless of their known or presumed infectious status. Standard precautions are minimum requirements for the control of infection in all settings and all situations, including those where a high risk of infection transmission exists, and are designed to protect both patients and healthcare workers.

Standard precautions comprise the following measures:

- handwashing
- use of appropriate personal protective equipment (PPE)
- immunisation of healthcare workers
- use of aseptic technique to reduce patient exposure to microorganisms
- management of sharps, blood spills, linen and waste to maintain a safe environment
- routine environmental cleaning.

Standard precautions apply to (1) blood; (2) all body fluids, secretions, and excretions except sweat, regardless of whether or not they contain visible blood; (3) non-intact skin; and (4) mucous membranes. These precautions are specifically designed for hospitals; however, they may also be implemented in extended and long-term care facilities, and to a more limited extent in providing home care or in other community-based care settings.

Handwashing

Handwashing is the single most important strategy to reduce the risk of infection. Handwashing comprises mechanical activity, use of soap and water, rinsing and drying to reduce the number of microorganisms on hands.

Wash hands with soap and water when visibly dirty or contaminated with proteinaceous material, or visibly soiled with blood or body fluids. An alcohol-based hand rub can be used for routine hand antisepsis in clinical situations if hands are not visibly soiled.

Handwashing should occur:

- before and after having direct contact with patients
- after removing gloves
- before handling an invasive device for patient care, regardless of whether or not gloves are used
- after contact with body fluids or excretions, mucous membranes, non-intact skin, or wound dressings
- if moving from a contaminated body site to a clean body site during patient care
- after contact with inanimate objects (including medical equipment) in the immediate vicinity of the patient.

Gloves

Gloves protect skin from direct contamination with blood and body fluids. The use of gloves within a healthcare setting is recommended to (a) prevent microorganisms that may be infecting, carried or present on healthcare workers' hands from being transmitted to patients and from one patient to another; and (b) to reduce the risk of transmission of an infection from the patient to the healthcare worker. The following guidelines apply:

- Gloves are not a substitute for handwashing.
- Hands should always be washed following glove removal.
- Gloves should be changed between patients, when punctured or torn, and between different procedures on the same patient.
- Gloves are not to be washed or disinfected between patients.

Wear clean, non-sterile gloves when touching blood, body fluids, secretions, excretions and contaminated items. Sterile gloves should be worn for all sterile procedures. Gloves should also be worn for all invasive procedures such as performing venipuncture or other vascular or surgical procedures. Gloves should always be removed promptly after use, and hands should always be washed immediately after the removal of gloves.

Mask, eye protection, face shield

When a splash or spray to the face is anticipated, both protective eyewear and a fluid-repellent surgical mask should be worn, or a full-face shield. Eye protection should always be used during procedures that have the potential to splash or spray the face. These should be able to be cleaned routinely and when visibly soiled.

A fluid-repellent surgical mask should be worn in conjunction with eye protection when the potential exists for splashes or sprays to the face. Masks should always be removed and discarded when the procedure is complete or if the mask becomes wet or visibly soiled.

Gown

Wear an unsterile gown or plastic apron (clean, disposable) to protect your skin and prevent soiling of clothing when there is potential for contamination with blood or body fluids, secretions or excretions. Remove soiled gowns promptly, washing your hands immediately after gown removal. Gowns should always be changed between patients.

Equipment

Handle used patient-care equipment that is soiled with blood, body fluids, secretions and excretions in a way that prevents exposing your skin and mucous membranes, contaminating your clothing and transferring microorganisms to other patients or environments. Ensure that reusable equipment is cleaned and appropriately reprocessed according to the facility's guidelines before using for the care of another patient.

Environmental control

Follow hospital procedures for routine care, cleaning and disinfecting environmental surfaces, beds, bed rails, bedside equipment and other frequently touched surfaces. Routine environmental cleaning of healthcare facilities is required to minimise the number of microorganisms in the environment. Microorganisms are unable to multiply on clean, dry surfaces.

Linen

All used linen should be handled with care to avoid dispersion of microorganisms into the environment and to avoid contact with clothing. Appropriate PPE should be used during the handling of soiled linen. Linen soiled with body fluids or substances should be placed into leak-proof laundry bags for safe transport. Hands should always be washed following the handling of used linen.

Occupational health and blood-borne pathogens

Contaminated sharps pose the greatest risk to healthcare workers of exposure to blood-borne viruses. They should be handled with due care.

Sharps handling and disposal

- All staff should be educated with regard to the safe use, handling and disposal of sharps.
- People using a sharp object are responsible for safely disposing of the item, preferably directly at the point of use. The task should not be delegated.
- Contaminated sharps should not be broken or bent, removed from disposable syringes prior to disposal, or passed by hand between healthcare workers.
- Avoid re-sheathing and passing needles; utilise safety devices—for example, retractable-needle/syringes, blood-taking devices.
- In the case of inappropriately disposed sharps, a sharps container should be taken to the location, the sharp handled and disposed of in a manner to avoid injury, and hands washed following disposal. Sharps should be disposed of in a labelled, puncture-resistant container that conforms to Australian Standards specifications.

Patient placement

Place patients who contaminate the environment or who do not (or are not expected to) assist in maintaining appropriate hygiene or environmental control (e.g. an ambulatory, confused patient with faecal incontinence) in a private room.

Sources: Centers for Disease Control and Prevention (2002). Guidelines for hand hygiene in health-care settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Taskforce. *MMWR*, 51 (RR-16), 1–56; Hospital Infection Control Practices Advisory Committee (1997). *Part II. Recommendations for isolation precautions in hospitals*. Atlanta: Public Health Service, US Department of Health and Human Services, Centers for Disease Control and Prevention; Queensland Health (2008). *Elements of infection control*. Retrieved from www.health.qld.gov.au/chrisp/ic_guidelines/sect2_elements.pdf; World Health Organization (2007). *WHO guidelines on hand hygiene in health care*. Retrieved from http://whqlibdoc.who.int/publications/2009/9789241597906_eng.pdf.

GLOSSARY

abrasion Partial-thickness denudation of an area of integument, generally resulting from falls or scrapes.

absence seizure (petit mal seizure) A type of generalised seizure characterised by a sudden brief cessation of all motor activity accompanied by a blank stare and unresponsiveness.

accommodation The ability of the eye to adjust to variations in distance.

achalasia Absence of peristalsis of the oesophagus and high gastro-oesophageal sphincter pressure resulting in dilation and loss of tone in the oesophagus.

acidosis The condition in which the hydrogen ion concentration increases above normal (reflected in a pH below 7.35).

acid A substance that releases hydrogen ions in solution.

acne Disorder of the pilosebaceous (hair and sebaceous gland) structure, resulting in eruption of papules or pustules.

acoustic neuroma (schwannoma) Benign tumour of cranial nerve VIII.

acquired immune deficiency syndrome (AIDS) A specific group of diseases or conditions that are indicative of severe immunosuppression related to infection with the human immunodeficiency virus.

acquired immunity Immunity developed after exposure to a pathogen. See *active immunity*.

acromegaly Meaning literally 'enlarged extremities', this is a condition resulting from excessive growth hormone secretion during adulthood.

actinic keratosis Also called senile or solar keratosis, this is an epidermal skin lesion directly related to chronic sun exposure and photo damage.

active immunity Production of antibodies or development of immune lymphocytes against specific antigens.

active sense of self The development of meaning, purpose and direction in one's life.

active transport Movement of molecules across cell membranes and epithelial membranes against a concentration gradient; requires energy.

acute care The provision of care for conditions of rapid onset that last a relatively short time and are self-limiting.

acute coronary syndrome (ACS) A general term used to describe the effects of coronary heart disease, including angina and myocardial infarction.

acute gastritis A benign, self-limiting disorder associated with ingestion of gastric irritants such as aspirin, alcohol, caffeine or foods contaminated with certain bacteria.

acute illness An illness that occurs rapidly, lasts for a relatively short time and is self-limiting.

acute inpatient unit A ward in a mental health facility that admits people who are generally very mentally unwell.

acute kidney failure Abrupt onset of kidney failure, often reversible.

acute kidney injury (AKI) Kidney injury characterised by a rapid onset of symptoms that are potentially reversible with prompt intervention that addresses the initial cause of the injury.

acute lymphoblastic leukaemia (ALL) Abnormal proliferation of lymphoblasts in the bone marrow, lymph nodes and spleen; the most common type of leukaemia in children and young adults.

acute myeloblastic leukaemia (AML) Uncontrolled proliferation of myeloblasts (*granulocyte precursors*) and hyperplasia of bone marrow and the spleen; the most common acute leukaemia in adults.

acute myocardial infarction (AMI) Necrosis (death) of myocardial cells.

acute pain Usually temporary, localised and of sudden onset; it lasts for less than 6 months and has an identifiable cause, such as trauma, surgery or inflammation.

acute renal failure See *acute kidney failure*.

acute respiratory distress syndrome (ARDS) Non-cardiac pulmonary oedema and progressive refractory hypoxaemia.

acute tubular necrosis (ATN) A syndrome of abrupt and progressive decline in tubular and glomerular function.

adaptive immune response A specific and systemic immune response initiated by and directed against particular antigens.

addiction Dependency on a drug, which can be either physical or psychological.

Addisonian crisis A life-threatening response to acute adrenal insufficiency; occurs in about 25% of patients.

Addison's disease A rare endocrine disorder wherein the adrenal glands produce insufficient steroid hormones.

adrenal crisis A constellation of symptoms that indicate severe adrenal insufficiency caused by insufficient levels of the hormone cortisol.

advance directive Also called a *living will*, this is a document in which a client formally states preferences for healthcare in the event that he or she later becomes mentally incapacitated and names a person who has durable power of attorney to serve as a substitute decision maker to implement the patient's stated preferences.

aesthetic surgery See *cosmetic surgery*.

affect Refers to the experience of feeling or emotion.

afterload The force the ventricles must overcome to eject their blood volume; the pressure in the arterial system ahead of the ventricles.

agnosia The inability to recognise one or more subjects that were previously familiar; agnosia may be visual, tactile or auditory.

agranulocytosis Severe neutropenia, with less than 200 cells/μm.

albuminuria Albumin (protein) in the urine.

alcohol An organic compound obtained by substituting a hydroxyl group for a hydrogen on a hydrocarbon.

alcoholic cirrhosis (Laënnec's cirrhosis) The end result of alcoholic liver disease.

alkalis Substances that accept hydrogen ions in solution.

alkalosis The condition where the hydrogen ion concentration decreases below normal (reflected in a pH above 7.45).

alleles Different forms of a gene or DNA occupying the same place on a pair of chromosomes; an allele for each gene is inherited from each parent.

allergy A hypersensitivity response to environmental or exogenous antigens.

allodynia Pain due to stimulus that normally does not evoke pain.

allografts Grafts between members of the same species but who have different genotypes and HLA antigens. See also *homograft*.

alopecia Loss of hair; baldness.

Alzheimer's disease (AD) A form of dementia characterised by progressive, irreversible deterioration of the general intellectual functioning.

amenorrhoea Absence of menstruation.

amphetamine A psychostimulant drug that is known to produce increased wakefulness and focus in association with decreased fatigue and appetite.

amputation Partial or total removal of a body part.

amyotrophic lateral sclerosis (ALS) Progressive, degenerative neurological disease characterised by weakness and wasting of the involved muscles, without any accompanying sensory or cognitive changes; also called *Lou Gehrig's disease*.

anaemia An abnormally low number of circulating red blood cells, haemoglobin concentration or both.

anaesthesia State produced by medications given intravenously, intraspinally, subcutaneously or by inhalation to create temporary partial or total loss of sensation and consciousness in a person for invasive procedures such as surgery or painful diagnostic tests.

analgesic A medication that reduces or eliminates the perception of pain.

anaphylactic shock Shock resulting from a widespread hypersensitivity reaction (called *anaphylaxis*). The pathophysiology in this type of shock includes vasodilation, pooling of blood in the periphery and hypovolaemia with altered cellular metabolism.

anaphylaxis An acute systemic type I response that occurs in highly sensitive persons following injection of a specific antigen.

anaplasia The regression of a cell to an immature or undifferentiated cell type.

anasarca Severe, generalised oedema.

androgens Hormones synthesised in the testes, ovaries and adrenal cortex that promote expression of male sex characteristics.

anergy Inability to react to specific antigens.

aneurysm Abnormal dilation of a blood vessel, commonly at a site of a weakness or tear in the vessel wall.

angina pectoris (angina) Chest pain resulting from reduced coronary blood flow that causes a temporary imbalance between myocardial blood supply and demand.

angioma (haemangioma) Benign vascular tumour.

anion gap The difference between the sum of two measured anions, chloride and bicarbonate, and the principal measured cation, sodium.

ankylosing spondylitis A chronic inflammatory arthritis that primarily affects the axial skeleton, leading to pain and progressive stiffening and fusion of the spine.

anorexia Loss of appetite.

anorexia nervosa An eating disorder characterised by a body weight less than 85% of expected for age and height and an intense fear of gaining weight.

anorgasmia Absence of orgasm.

anosmia Inability to smell.

anthropometric measurements Measurement of height, weight, triceps skin folds and midarm circumference.

antibodies Immunoglobulin molecules that bind with an antigen to inactivate it.

antibody-mediated (humoral) immune response Activation of B cells to produce antibodies to respond to antigens such as bacteria, bacterial toxins and free viruses.

anticipatory grieving A combination of intellectual and emotional responses and behaviours by which people adjust their self-concept in the face of a potential loss.

antigen A substance capable of evoking a specific immune response; usually a protein, which the body recognises as foreign, causing an immune response to be stimulated.

anxiety An unpleasant feeling that is typically associated with uneasiness, apprehension, fear or worry.

aortic valve The semilunar valve between the left ventricle of the heart and the aorta in the heart. It prevents blood from flowing backwards into the ventricle.

aortitis Inflammation of the aorta, usually the aortic arch.

aphasia Defective or absent language function.

apical impulse A normal, visible pulsation (thrust) in the area of the midclavicular line in the left fifth intercostal space. It can be seen on inspection in about half of the adult population.

aplastic anaemia A condition manifested by failure of the bone marrow to produce all three types of blood cells.

apnoea Cessation of breathing lasting from a few seconds to a few minutes.

appendectomy Surgical removal of the appendix.

appendicitis Inflammation of the vermiform appendix.

apraxia The inability to carry out a motor pattern (such as drawing a figure) even when strength and coordination are adequate.

areflexia Lack of normal reflexes.

arrhythmia Abnormal heart rate or rhythm.

arterial blood gas (ABG) A laboratory test used to evaluate acid–base balance and gas exchange.

arteriovenous (AV) malformation A congenital intracranial lesion, formed by a tangled collection of dilated arteries and veins, that allows blood to flow directly from the arterial into the venous system, bypassing the normal capillary network.

arthralgia Joint pain.

arthritis Joint inflammation.

ascites Excess fluid in the peritoneal cavity.

asphyxiation Oxygen deprivation.

asthma Chronic inflammatory disorder of the airways that is characterised by recurrent episodes of wheezing, breathlessness, chest tightness and coughing.

astigmatism A condition that develops with abnormal curvature of the cornea or eyeball, causing the image to focus at multiple points on the retina.

ataxia Uncoordinated, irregular gait and muscle movement; weakness.

atelectasis Collapse of lung tissue following obstruction of the bronchus or bronchioles.

atherosclerosis A form of arteriosclerosis in which deposits of fat and fibrin obstruct and harden the arteries.

ATODS Alcohol tobacco and other drugs services.

atopic dermatitis (eczema) Common inflammatory skin disorder of unknown cause.

atrial kick Delivery of an additional bolus of blood to the ventricles resulting from atrial systole; occurs just prior to ventricular systole.

atrial natriuretic peptide (ANP) A hormone released by atrial muscle cells in response to distension from fluid overload.

aura Sensation preceding generalised seizure activity; may be a vague sense of uneasiness or an abnormal sensation.

auscultatory gap A temporary disappearance of sound between the systolic and diastolic blood pressure.

Australian College of Mental Health Nurses Peak professional college for mental health nurses in Australia.

Australian Organ and Tissue Donation and Transplantation Authority Act 2008 Legislation that requires people to be informed about their options related to organ donation.

autografting Transplanting of the person's own tissue; the most successful type of tissue transplant.

autoimmune disorder Failure of the immune system to recognise itself, resulting in normal host tissue being targeted by immune defences.

autonomic dysreflexia Exaggerated sympathetic response that occurs in people with spinal cord injuries at or above the T6 level.

autosome A single chromosome from any one of the 22 pairs of chromosomes not involved in sex determination (X or Y); humans have 22 pairs of autosomes.

azotaemia Increased blood levels of nitrogenous waste products.

B lymphocytes (B cells) Bursa-equivalent lymphocytes responsible for synthesising humoral antibody.

bacterial vaginosis Non-specific vaginitis.

bactericidal agent Capable of killing an organism without immune system intervention. These include the penicillins, cephalosporins and aminoglycoside antibiotics.

bacteriostatic agent Inhibits the growth of a microorganism, leaving its destruction to the host's immune system.

bacteriuria Bacteria in the urine.

balanced suspension traction Traction in which several forces of pull work in unison to raise and support the person's injured extremity off the bed and pull it in a straight fashion away from the body.

balloon tamponade The application of pressure to stop oesophageal bleeding using an inflatable balloon.

bariatrics The branch of healthcare that deals with the causes, prevention and treatment of obesity. The term *bariatrics* was created around 1965, from the Greek root *bar-* ('weight' as in barometer), suffix *-iatr* ('treatment', as in pediatrics), and suffix *-ic* ('pertaining to'). The field encompasses dieting, exercise and behavioural therapy approaches to weight loss, as well as psychotherapy, pharmacotherapy and surgery.

basal cell carcinoma (BCC) Epithelial tumour that is believed to originate either from the basal layer of the epidermis or from cells in the surrounding dermal structures. These tumours are characterised by an impaired ability of the basal cells of the epidermis to mature into keratinocytes, with mitotic division beyond the basal layer.

basal metabolic rate (BMR) Test to measure the energy used when the body is at rest; rarely used due to the availability of more accurate thyroid tests.

base excess (BE) A calculated value also known as buffer base capacity. Base excess reflects the degree of acid–base imbalance by indicating the status of the body's total buffering capacity.

bases (or alkalis) Substances that accept hydrogen ions in solution.

Bell's palsy (facial paralysis) Disorder of the facial nerve (seventh cranial nerve), characterised by unilateral paralysis of the facial muscles.

benign prostatic hyperplasia (BPH) Enlargement of the prostate gland.

benzodiazepines Minor tranquillisers belonging to the sedative-hypnotic group of drugs that have a CNS depressant effect through action at the gamma aminobutyric acid (GABA) receptor sites.

bereavement The time of mourning experienced after a loss.

bile A greenish, watery solution containing bile salts, cholesterol, bilirubin, electrolytes, water and phospholipids.

biliary colic A severe, steady pain in the epigastric region or upper right quadrant of the abdomen.

binge-eating disorder An eating disorder characterised by recurrent episodes of eating an excessive amount of food during a defined period of time, and a sense of lack of control over eating during binge episodes.

biofeedback An electronic method of measuring autonomic physiological responses, such as brain waves, muscle contraction and skin temperature, and then 'feeding' this information back to the person.

biofilms Polymicrobial microbial communities which proliferate and are encased in a protective glycocalax matrix.

biological terrorism Use of an aetiological agent (disease) to cause harm or kill a population, food and/or livestock.

biomedical model of health A model of health which mainly focuses on biological health determinants and broadly views health as the absence of disease. This model is also known as the medical model.

biopsychosocial model of health The biopsychosocial model of health broadly views health as individual holistic wellbeing.

bioterrorism Use of an aetiological agent (disease) to cause harm or kill a population, food and/or livestock.

biotherapy Treatment that modifies the biological processes that result in malignant cells, primarily through enhancing the person's own immune responses.

bivalving Process of splitting a cast down both sides to alleviate pressure on the injured extremity.

blood clot See *thrombus*.

blood flow The volume of blood transported in a vessel, in an organ or throughout the entire circulation over a given period of time.

blood pressure The tension or pressure exerted by blood against arterial walls.

blunt trauma The type of trauma that occurs when there is no communication from the damaged tissues to the outside environment.

body mass index (BMI) Used to identify excess adipose tissue, BMI is calculated by dividing the weight (in kilograms) by the height (in metres squared, m^2).

bone marrow transplant (BMT) Infusion of bone marrow cells to restore bone marrow function after chemotherapy or radiation; allogeneic BMT uses donor bone marrow cells from a donor; autologous BMT uses the person's own bone marrow.

borborygmi Excessive loud and hyperactive bowel sounds.

botulism A severe, life-threatening form of food poisoning caused by *Clostridium botulinum*.

brachytherapy A type of radiation therapy in which the source of radiation is placed directly into or adjacent to the tumour, a technique that delivers a high dose to the tumour and a lower dose to normal tissue.

bradycardia A heart rate of less than 60 beats per minute.

bradykinesia Slowed movements due to muscle rigidity.

bradypnoea Abnormally low respiratory rate.

brain abscess Infection with a collection of purulent material within the brain tissue.

brain death The cessation of cerebral blood flow with global brain infarction and permanent loss of all brain function.

brain death criteria Clinical signs used to determine whether a comatose person is brain dead.

breakthrough pain A sudden flare or increase in pain despite comfort with or without baseline analgesia.

bronchiectasis Permanent abnormal dilation of one or more large bronchi and destruction of bronchial walls, usually accompanied by infection.

bronchitis Inflammation of the bronchi.

bruit An adventitious sound heard during auscultation; of venous or arterial origin.

buffer A substance that prevents major changes in pH by removing or releasing hydrogen ions.

bulimia nervosa An eating disorder characterised by recurring episodes of binge eating followed by purge behaviours such as self-induced vomiting, use of laxatives or diuretics, fasting or excessive exercise.

burn An injury resulting from exposure to heat, chemicals, radiation or electric current.

burn shock Hypovolaemic shock resulting from the shift of a massive amount of fluid from the intracellular and intravascular compartments into the interstitium following burn injury.

bursitis Inflammation of the bursa.

cachectic The state of very poor health and malnourishment in a person.

cachexia The wasted physical appearance characteristic of cancer and other chronic illnesses. It is characterised by rapid depletion of the body's protein, particularly in skeletal muscle, with less rapid loss of fat.

caffeine A bitter, white crystalline xanthine alkaloid that is a psychoactive stimulant drug.

calculi An abnormal concentration in the body, commonly called a stone; occur in the kidneys, ureters, bladder or urethra.

cancer A family of complex diseases with manifestations that vary according to body system and type of tumour cells involved; marked by uncontrolled growth and the spread of abnormal cells.

cancer (palliative) pain A common condition of people suffering with advanced cancer, it is often persistent and arises from a number of factors.

candidiasis Infection of mucous membranes caused by *Candida albicans*, a yeast-like fungus.

cannabis The general name given to the psychoactive substances found in the marijuana plant, *Cannabis sativa*, the main active constituent being delta 9-tetra-hydrocannabinol (THC).

carbuncle A group of infected hair follicles.

carcinogen Cancer-causing agent.

carcinogenesis The production or origin of cancer.

carcinoma A tumour arising from epithelial tissue.

cardiac arrest Sudden failure of the heart to pump.

cardiac cycle The contraction and relaxation of the heart during one heartbeat.

cardiac index Cardiac output adjusted for body size.

cardiac output (CO) The amount of blood pumped by the ventricles into the pulmonary and systemic circulations in 1 minute.

cardiac rehabilitation A long-term program of medical evaluation, exercise, risk factor modification, education and counselling designed to limit the physical and psychological effects of cardiac illness and improve the person's quality of life.

cardiac reserve The ability of the heart to respond to the body's changing need for cardiac output.

cardiac tamponade Compression of the heart due to pericardial effusion, trauma, cardiac rupture or haemorrhage.

cardiogenic shock Shock that occurs when the heart's pumping ability is compromised to the point that it cannot maintain cardiac output and adequate tissue perfusion.

cardiomegaly Enlargement of the heart.

cardiomyopathy Primary abnormality of the heart muscle that affects its structural or functional characteristics.

cardiovascular disease (CVD) Generic term for disorders of the heart and blood vessels.

carpal spasm When the person's hand and fingers contract due to decreased calcium levels.

carpal tunnel syndrome Compression of the median nerve as a result of inflammation and swelling of the synovial lining of the tendon sheaths.

carrier Any individual who carries a single copy of an altered gene or mutation for a recessive condition on one chromosome of a chromosome pair and an unaltered form of that gene on the other chromosome; a carrier generally is not affected by the gene alteration; on average, each person in the general population is a carrier of five or six gene mutations for recessive disorders.

catabolism Biochemical process involving the breakdown of complex structures into simpler forms.

cataract Opacification (clouding) of the lens of the eye.

cell cycle The four phases that occur during growth and development of a cell.

cell-mediated (cellular) immune response Direct or indirect inactivation of antigen by lymphocytes.

cellulitis A localised infection of the dermis and subcutaneous tissue.

central nervous system depressants Drugs that can be used to slow down brain activity.

central obesity Obesity characterised by a waist-to-hip ratio of greater than 1 in men or 0.8 in women.

central pain Related to a lesion in the brain that may spontaneously produce high-frequency bursts of impulses that are perceived as pain.

cerebral concussion Transient, temporary, neurogenic dysfunction caused by mechanical force to the brain.

cerebral contusion Bruise on the surface of the brain.

cerebral oedema An increase in the volume of brain tissue due to abnormal accumulation of fluid.

cerumen Earwax.

chalazion Granulomatous cyst or nodule of the eyelid.

chancre Hard, syphilitic primary ulcer.

cheilosis Chemical peeling; the application of a chemical to produce a controlled and predictable injury that alters the anatomy of the epidermis and superficial dermis.

chemotherapy Cancer treatment involving the use of cytotoxic medications to decrease tumour size, adjunctive to surgery or radiation therapy; or to prevent or treat suspected metastases.

chlamydia A group of syndromes caused by *Chlamydia trachomatis*, a bacterium that behaves like a virus spreading within a host cell; spread by sexual contact and to the neonate by passage through the birth canal of an infected mother.

cholecystectomy Removal of the gallbladder.

cholecystitis Inflammation of the gallbladder, usually associated with stones in the cystic or common bile duct.

cholelithiasis Formation of stones (calculi) within the gallbladder or biliary duct system.

cholera Acute diarrhoeal illness caused by certain strains of *Vibrio cholerae*.

chorea Jerky, rapid, involuntary movements.

chromosome Genetic material carried by each cell; found in the cell nucleus.

chronic bronchitis Excessive secretion of bronchial mucus characterised by a productive cough lasting 3 or more months in 2 consecutive years.

chronic disease A disease involving a long course in its development or its symptoms.

chronic gastritis Disorders characterised by progressive and irreversible changes in the gastric mucosa.

chronic illness Illness that lasts for an extended period of time, usually greater than 6 months.

chronic kidney disease Kidney injury in a person with a glomerular filtration rate (GFR) < 60 mL/min/1.73 m^2 for > 3 months with or without evidence of kidney damage OR evidence of kidney damage (with or without decreased GFR) for > 3 months including microalbuminuria, proteinuria, glomerular haematuria or any anatomical or pathological abnormality.

chronic kidney failure Progressive kidney tissue destruction with loss of entire nephron unit and function; kidney mass decreases, and glomerular filtration, tubular secretion and reabsorption deteriorate.

chronic lymphocytic leukaemia (CLL) Proliferation and accumulation of small, abnormal, mature lymphocytes in the bone marrow, peripheral blood and body tissues; least common type of the major leukaemias.

chronic myelogenous leukaemia (CML) Abnormal proliferation of all bone marrow elements, usually associated with a chromosome abnormality (the Philadelphia chromosome).

chronic obstructive pulmonary disease (COPD) Chronic airflow obstruction due to chronic bronchitis and/or emphysema.

chronic otitis media Condition involving permanent perforation of the tympanic membrane, with or without recurrent pus formation and often accompanied by changes in the mucosa and bony structures (ossicles) of the middle ear.

chronic pain Prolonged pain, usually lasting longer than 6 months. It is not always associated with an identifiable cause and is often unresponsive to conventional medical treatment.

chronic renal failure See *chronic kidney failure*.

chronic sorrow A cyclical, recurring and potentially progressive pattern of pervasive sadness experienced in response to continual loss, throughout the trajectory of an illness or disability.

chronic stump pain The result of neuroma formation, causing severe burning pain.

chronic venous insufficiency A chronic disorder of inadequate venous return.

Chvostek's sign Contraction of the lateral facial muscles in response to tapping the face in front of the ear; caused by decreased blood calcium levels.

chyme Thick, fluid mixture of food and gastric juices formed in the stomach during the digestive process.

circulating nurse Assists scrub nurses and surgeons during surgery.

cirrhosis A progressive, irreversible disorder, eventually leading to liver failure; the end stage of chronic liver disease.

claudication Cramping, aching pain in the calves, thighs and buttocks that occurs with a predictable level of activity and is relieved by rest.

client A term used instead of 'patient' that is based on a philosophy that individuals are active participants in health and illness as well as consumers of healthcare services.

clinical governance A system of policies, processes and accountabilities that is directed at improving patient safety and the quality and effectiveness of patient care within a health service.

clinical pathway A healthcare plan designed to provide care with a multidisciplinary, managed action focus; developed for specific diagnoses, usually those that are high volume, high risk and high cost.

clinical reasoning The process by which nurses (and other clinicians) collect cues, process the information, come to an understanding of a person's problem or situation, plan and implement interventions, evaluate outcomes, and reflect on and learn from the process.

closed fracture (simple fracture) Break in continuity of bone with skin still intact.

clubbing Enlargement and blunting of the terminal portion of the fingers; associated with chronic hypoxaemia.

cluster headache A form of vascular headache predominantly experienced by men aged 20 to 40. The headache typically begins 2 to 3 hours after the person falls asleep.

coagulation The process of creating a fibrin meshwork that cements blood components together to form an insoluble clot.

cocaine An illegal drug extracted from a cocoa leaf that is white, odourless, and takes the form of a crystalline powder.

code of ethics An established and agreed-on group of principles of conduct that provide a professional framework.

coeliac disease (coeliac sprue, non-tropical sprue) Chronic hereditary disorder characterised by sensitivity to the gliadin fraction of gluten, a cereal protein.

cognition The ability to process information and apply knowledge.

cold sore See *herpes simplex*.

cold zone Considered the 'safe zone' during a disaster, it is adjacent to the warm zone and is the area where a more in-depth triage of victims would occur; survivors may find shelter in this area and command and control vehicles would be found here as well as the emergency transport vehicles.

colectomy Surgical removal of the colon.

collateral channels Connections between small arteries.

collateral vessels Accessory pathways connected to the smaller arteries in the coronary system.

colorectal cancer Malignant tumour arising from the epithelial tissues of the colon or rectum.

colostomy Ostomy made in the colon.

comedones Non-inflammatory acne lesions.

communication The exchange of information between two or more people, groups or entities. It involves verbal and written exchanges, as well as body language, attitude and tone (Nadzam, 2009).

community A collection of people who share some attribute of their lives.

community-based care Centres on individual and family healthcare needs. The nurse practising community-based care provides direct services to individuals to manage acute or chronic health problems and to promote self-care. The care is provided in the local community, is culturally competent and is family centred.

community mental health nursing nurse A branch of mental health nursing in which the nurse works with consumers who are living in the community. It often involves case management.

community nursing Care directed towards a specific population or group within the community; primary, secondary or tertiary care may be provided to individuals or groups.

compartment A space enclosed by a fibrous membrane or fascia.

compartment syndrome Condition in which excess pressure constricts the structures within a compartment and reduces circulation to muscles and nerves.

concussion Injury resulting from a violent jar, shake or impact with an object.

conjunctivitis Inflammation of the conjunctiva.

consanguinity Related by having a common ancestor; close blood relationship.

conscious sedation Anaesthesia that provides analgesia and amnesia, but in which the person remains conscious. People are able to breathe independently and are cardiovascularly stable.

consciousness A condition in which a person is aware of self and environment and is able to respond appropriately to stimuli; full consciousness requires both normal arousal and full cognition.

constipation The infrequent (two or fewer bowel movements weekly) or difficult passage of stools.

consultation liaison A specialist mental health nurse who is the interface between medicine and psychiatry.

consumer-directed care A model of service delivery designed to give more care choice and flexibility to consumers. This model provides consumers with more control over the types of care and services they access, and the delivery of those services, including who delivers the services and when they are delivered.

contact dermatitis Type of dermatitis caused by a hypersensitivity response or chemical irritation.

continuous renal replacement therapy (CRRT) A form of haemodialysis in which blood is continuously circulated through a highly porous haemofilter from artery to vein, or vein to vein.

continuum of care The provision of ongoing quality healthcare in acute and community settings to optimise quality of life for people, underpinned by an interprofessional consultative team approach.

contractility The inherent capability of the cardiac muscle fibres to shorten.

contracting The negotiation of a cooperative working agreement between the nurse and person that is continuously renegotiated.

contracture Permanent shortening of connective tissue.

contralateral deficit Manifestations of a stroke on the side of the body opposite the side of the brain that is damaged.

contusion Superficial tissue injury resulting from blunt trauma, such as a kick or blow from an object, that causes the breakage of small blood vessels and bleeding into the surrounding tissue.

conventional weapons Weapons such as bombs and guns that are used more frequently than non-conventional terrorist weapons.

convergence Moving inward of the eyes to see an object close to the face.

co-occurring disorders Concurrent diagnosis of a substance use disorder and a psychiatric disorder. One disorder can precede and cause the other, such as the relationship between alcoholism and depression.

cor pulmonale Condition of right ventricular hypertrophy and failure that results from longstanding pulmonary hypertension.

core competencies Standards that a profession agrees are essential for a person to be deemed competent in their field.

corneal reflex Closure of eyelids (blinking) due to corneal irritation.

corneal ulcer Local necrosis of the cornea, may be caused by infection, exposure trauma or the misuse/overuse of contact lenses.

coronary heart disease (CHD) Heart disease caused by impaired blood flow to the myocardium.

coryza (rhinorrhoea) Profuse nasal discharge.

cosmetic surgery (aesthetic surgery) One of two fields within plastic surgery. Cosmetic surgery enhances the attractiveness of normal features.

crackles Discontinuous lung sound heard by auscultation; can be fine or coarse. Produced by air passing over airway secretions or the opening of collapsed airways.

creatinine The end product from the breakdown of creatine phosphate in muscles.

crepitation A grating sound heard on movement of a joint.

Creutzfeldt–Jakob disease (CJD, spongiform encephalopathy) Rare, progressive neurological disease that causes brain degeneration without inflammation.

critical pathway See *clinical pathway*.

critical thinking Self-directed thinking that is focused on what to believe or do in a specific situation.

Crohn's disease (regional enteritis) Chronic, relapsing inflammatory disorder affecting the gastrointestinal tract.

crossing over A process that occurs during meiosis in which homologous maternal and paternal chromosomes break and exchange corresponding sections of DNA and then rejoin; this process can cause an exchange of alleles between chromosomes and provides human diversity.

cryosurgery The destruction of tissue by cold or freezing with agents such as fluorocarbon sprays, carbon dioxide snow, nitrous oxide and liquid nitrogen.

cultural competence Practising in a way that demonstrates the importance of social and cultural influences on patients' health beliefs and behaviours, and devising interventions that take these issues into account.

cultural safety The effective nursing practice of a person or family from another culture, as determined by that person or family.

culture A learned world viewpoint or paradigm shared by a population or group and transmitted socially. It influences values, beliefs, customs and behaviours, and is reflected in the language, dress, food, materials and social interactions of a group.

curettage The removal of lesions with a curette, a semi-sharp cutting instrument.

Curling's ulcers Acute ulcerations of the stomach or duodenum that form following a burn injury.

Cushing's syndrome A chronic disorder in which hyperfunction of the adrenal cortex produces excessive amounts of circulating cortisol or adrenocorticotropic hormone (ACTH).

Cushing's ulcers Stress ulcers occurring as sequelae of head injury or central nervous system surgery.

cutaneous melanoma See *malignant melanoma*.

cyanosis A bluish discolouration of the skin and mucous membranes due to oxygen deficiency.

cyst A sac containing fluid or semisolid fluid.

cystectomy Complete surgical removal of the urinary bladder and adjacent muscles and tissues.

cystic fibrosis (CF) Inherited disorder of the exocrine glands that results in the secretion of abnormal amounts of mucus.

cystitis Inflammation of the urinary bladder.

cysts of the skin Benign closed sacs in or under the skin surface that are lined with epithelium and contain fluid or a semisolid material. Epidermal inclusion cysts and pilar cysts are the most common types.

cytokines Hormone-like polypeptides produced primarily by monocytes, macrophages and T cells. Cytokines act as messengers of the immune system, facilitating communication between the cells to adjust or vary the inflammatory reaction or to initiate immune cell proliferation and differentiation.

dawn phenomenon A rise in blood glucose between 4 am and 8 am that is not a response to hypoglycaemia.

day surgery units/centres Facilities where surgery is performed and the person is discharged on the same day.

death Irreversible cessation of circulatory and respiratory functions or irreversible cessation of all functions of the entire brain, including the brainstem.

death anxiety Worry or fear related to death or dying.

debridement Process of removing dead tissue from a wound.

decerebrate posturing Abnormal posture with the neck extended; the jaw clenched; arms pronated, extended and close to the sides; legs extended and feet plantar flexed. Results from lesions of the midbrain, pons or diencephalons.

decorticate posturing Abnormal posture with the upper arms close to the sides; the elbows, wrists and fingers flexed; the legs extended and internally rotated; and the feet plantar flexed. Results from lesions of the corticospinal tracts.

deep venous thrombosis or deep vein thrombosis (DVT) Blood clot (thrombus) formation and inflammation within a deep vein, usually in the pelvis or lower extremities; a common complication of hospitalisation, surgery and immobilisation.

dehiscence An unintended separation of wound margins due to incomplete healing.

dehydration Loss of water.

delayed healing Healing that occurs at a slower rate than expected.

delegation To effectively assign appropriate work activities to other members of the healthcare team. When the nurse delegates nursing care activities to another person, that person is authorised to act in the place of the nurse, while the nurse retains the accountability for the activities performed.

delirium tremens (DT) A medical emergency usually occurring 3 to 5 days following alcohol withdrawal and lasting 2 to 3 days; characterised by paranoia, disorientation, delusions, visual hallucinations, elevated vital signs, vomiting, diarrhoea and diaphoresis.

delusion A fixed false belief that is firmly sustained despite what constitutes incontrovertible and obvious proof or evidence to the contrary. The belief is not one ordinarily accepted by other members of the person's culture or subculture.

dementia A global impairment of cognitive function that usually is progressive and may be permanent; interferes with normal social and occupational activities.

demyelination Destruction or removal of the myelin sheaths of nerves.

depolarisation The rapid inflow of sodium ions, causing an electrical change in which the inside of a cell becomes positive in relation to the outside.

dermatitis Acute or chronic inflammation of the skin characterised by erythema and pain or pruritus.

dermatome Area of skin innervated by cutaneous branches of a single spinal nerve.

dermatophytes Fungi that cause superficial skin infections.

dermatophytoses Superficial fungal infection of the skin; also called *ringworm*.

determinants of health Factors that influence health in either a positive or a negative way. Some of these function on an individual level (e.g. health behaviours such as smoking or exercise, or our genetic make-up). Others function at a broader societal level, such as the availability of health services, vaccination programs or clean drinking water and healthy food.

diabetes insipidus The result of antidiuretic hormone insufficiency.

diabetes mellitus (DM) Group of chronic disorders of the endocrine pancreas, all categorised under a broad diagnostic label. The condition is characterised by inappropriate hyperglycaemia caused by a relative or absolute deficiency of insulin or by a cellular resistance to the action of insulin.

diabetic ketoacidosis (DKA) A form of metabolic acidosis induced by stress in a person with type 1 diabetes mellitus.

diabetic nephropathy A disease of the kidneys characterised by the presence of albumin in the urine, hypertension, oedema and progressive renal insufficiency.

diabetic neuropathies Disorders of the peripheral nerves and the autonomic nervous system manifesting one or more of the following: sensory and motor impairment, muscle weakness and pain, cranial nerve disorders, impaired vasomotor function, impaired gastrointestinal function and impaired genitourinary function.

diabetic retinopathy The collective name for the changes in the retina that occur in the person with diabetes. The retinal capillary structure undergoes alterations in blood flow, leading to retinal ischaemia and a breakdown in the blood retinal barrier.

***Diagnostic and Statistical Manual of Mental Disorders* (DSM)** A manual that is published by the American Psychiatric Association and provides common language and standard criteria for the classification of mental disorders.

dialysate Dialysis solution.

dialysis The diffusion of solute molecules across a semipermeable membrane from an area of higher concentration to one of lower concentration.

diaphoresis Copious production of sweat.

diarrhoea An increase in the frequency, volume and fluid content of the stool.

diastolic blood pressure The minimum pressure maintained by elastic arterial walls during diastole (cardiac relaxation) to maintain blood flow through capillary beds; averages 80 mmHg in a healthy adult.

differentiation A process occurring over many cell cycles that allows cells to specialise in certain tasks.

'differentness' Being different from another person or group of people.

diffuse brain injury (DBI) A brain injury from a high-speed acceleration–deceleration accident with widespread disruption of axons in the white matter.

diffuse oesophageal spasm Non-peristaltic contraction of oesophageal smooth muscle.

diffusion The process by which solute molecules move from an area of high solute concentration to an area of low solute concentration to become evenly distributed.

dilemma A choice between two unpleasant, ethically troubling alternatives.

diplopia Unilateral or bilateral double vision.

dirty bomb Consists of a conventional explosive such as trinitrotoluene (TNT) packed with radioactive waste by-products from nuclear reactors that discharge deadly radioactive particles into the environment. See also *radiological dispersion bomb*.

disability The degree of observable and measurable impairment.

disaster Event that requires extraordinary efforts beyond those needed to respond to everyday emergencies.

discharge planning A planned process beginning with the person's initial presentation, considering the needs of the unique individual, based upon the availability of, and access to, support and resources. This process ensures that these needs are met through ongoing assessment and consultation involving the relevant healthcare professionals, client, family, carers and community services.

discovery Encompasses equality and respect. Equality is the belief that all people ought to be treated equally. Respect is esteem for, or a sense of the worth or excellence of, a person, a personal quality, ability or a manifestation of a personal quality or ability.

disease Literally meaning 'without ease', this term describes alterations in structure and function of the body or mind. Diseases may have mechanical, biological or normative causes.

dislocation Separation of contact between two bones of a joint.

dissection (aortic) A life-threatening emergency caused by a tear in the intima of the aorta with haemorrhage into the media.

disseminated intravascular coagulation (DIC) A disruption of haemostasis characterised by widespread intravascular clotting and bleeding; a syndrome that develops as a complication of many other disorders.

distal determinants Determinants of health which tend to be stable and concern historical, national, institutional, political, legal and cultural factors.

distributive shock Also called *vasogenic shock*, this includes several types of shock that result from widespread vasodilation and decreased peripheral resistance.

diverticula Sac-like projections of mucosa through the muscular layer of the colon.

diverticulitis Inflammation in and around the diverticular sac; typically affects only one diverticulum, usually in the sigmoid colon.

diverticulosis Indicates the presence of diverticula.

DNA-based tests Tests that incorporate new, sophisticated technology that permits the examination of the DNA itself, obtained from blood, bone marrow, amniotic fluid, fibroblast cells of the skin or buccal cells from the mouth.

dominant A characteristic or gene that is apparent even when the relevant gene is present in only one copy; a person with a dominant gene usually expresses that gene trait.

Do-Not-Resuscitate (DNR) order Usually written by the physician for the person who has a terminal illness or is near death, this order is usually based on the wishes of the person and family that no cardiopulmonary resuscitation be performed for respiratory or cardiac arrest.

Down syndrome A human genetic disease caused by the presence of an extra chromosome 21; characterised by mental retardation and heart and respiratory defects.

dual diagnosis The coexistence of substance use/dependence and a psychiatric disorder in one individual (used interchangeably with *dual disorder* and *co-occurring disorders*).

dual disorder See *dual diagnosis*.

dumping syndrome Complication of partial gastrectomy characterised by nausea, weakness, sweating, palpitation, syncope, sensation of warmth and occasionally diarrhoea.

duodenal ulcers Peptic ulcer disease affecting the duodenum.

dwarfism A medical disorder, the term being used to describe a person of short stature.

dysarthria Difficulty speaking.

dysfunctional uterine bleeding (DUB) Vaginal bleeding that is usually painless but abnormal in amount, duration or time of occurrence.

dysmenorrhoea Pain associated with menstruation.

dyspareunia Painful intercourse.

dysphagia Difficulty swallowing.

dysphonia Change in the tone of voice.

dysplasia The loss of DNA control over differentiation occurring in response to adverse conditions.

dyspnoea Difficult or laboured breathing.

dysrhythmia See *arrhythmia*.

dysuria Painful urination.

ecchymosis A flat, irregularly shaped lesion of varying size with no pulsation; caused by blood collecting under the skin.

ectopic beats Impulses originating outside normal conduction pathways of the heart.

ejection fraction (EF) The percentage of total blood remaining in the ventricle at the end of diastole (relaxation); normal is 50–70%.

electrical bone stimulation Application of electrical current at the fracture site to treat fractures that are not healing appropriately. The electrical stress increases the migration of osteoblasts and osteoclasts to the fracture site. Mineral deposition increases, promoting bone healing.

electrocardiography The graphic recording of the heart's electrical activity detected and recorded through electrodes placed on the surface of the body.

electrolytes Substances that dissociate in solution to form charged particles called ions.

electrosurgery The destruction or removal of tissue with high-frequency alternating current.

embolic CVA Cerebrovascular accident (CVA) occurring when a blood clot or clump of matter travelling through the cerebral blood vessels becomes lodged in a vessel too narrow to permit further movement.

embolism Sudden obstruction of a blood vessel by debris.

emergency Encompasses an unforeseen combination of circumstances calling for immediate action for a range of victims from one to many.

emphysema Destruction of the walls of the alveoli, with resulting enlargement of abnormal air spaces.

empowerment Developing confidence in one's own capacities.

empyema Accumulation of purulent exudate in the pleural cavity.

encephalitis An acute inflammation of the parenchyma of the brain or spinal cord.

end-of-life care Care provided in the final weeks of life when death is imminent.

endocarditis Inflammation of the endocardium.

endogenous insulin The insulin the pancreas makes.

endometriosis A condition in which multiple, small implants of endometrial tissue develop throughout the pelvic cavity.

endoscopy Inspection of organs or cavities of the body using an endoscope.

endotoxins Found in the cell wall of Gram-negative bacteria, endotoxins are released only when the cell is disrupted. They act as activators of many human regulatory systems, producing fever, inflammation and potentially clotting, bleeding or hypotension when released in large quantities.

end-stage kidney disease (ESKD) The final stage of chronic kidney failure in which the kidneys are unable to excrete metabolic wastes and regulate fluid and electrolyte balance adequately; characterised by a glomerular filtration rate of less than 5% of normal.

enduring power of attorney A document that can delegate the authority to make health, financial and/or legal decisions on a person's behalf. It must be in writing and must state that the designated person is authorised to make healthcare decisions.

enophthalmos Sunken appearance of the eyes.

enteral nutrition Administration of liquid nutritional formulas to meet kilojoule and protein needs in people unable to consume adequate food; also called *tube feeding*.

enucleation Surgical removal of an eye.

epicondylitis (tennis elbow, golfer's elbow) Inflammation of the tendon at its point of origin into the bone.

epididymitis Infection or inflammation of the epididymis.

epidural haematoma (extradural haematoma) A collection of blood between the dura and the skull.

epilepsy Chronic seizure activity.

epistaxis Nosebleed.

equianalgesia Equal analgesia; used when referring to the doses of various opioid analgesics that provide approximately the same pain relief.

erectile dysfunction Inability of the male to attain and maintain an erection sufficient to permit satisfactory sexual intercourse.

erosive gastritis See *stress-induced (erosive) gastritis*.

erysipelas Infection of the skin most often caused by group A streptococci.

erythema A reddening of the skin.

erythropoiesis Red blood cell production.

eschar Hard, leathery crust that covers a burn wound and harbours necrotic tissue.

escharotomy Surgical removal of eschar from the torso or extremity to prevent circumferential constriction.

ethics Principles of conduct. Ethical behaviour is concerned with moral duty, values, obligations and the distinction between right and wrong.

euthanasia From the Greek for 'painless, easy, gentle or good death', now commonly used to signify a killing prompted by a humanitarian motive.

euthyroid The state of having normal thyroid gland function.

eventually fatal condition Any illness in which it is expected that death will be a direct consequence.

evisceration Protrusion of body contents through a surgical wound.

exacerbation A period during chronic illness in which symptoms reappear.

exfoliative dermatitis Inflammatory skin disorder characterised by excessive peeling or shedding of skin.

exogenous insulin The insulin people inject or infuse via an insulin pump.

exophthalmos Protrusion of the eyeballs.

exotoxins Soluble proteins secreted into surrounding tissue by the microorganism. Exotoxins are highly poisonous, causing cell death or dysfunction.

external otitis Inflammation of the ear canal.

extracapsular fractures Fractures of the trochanteric region.

extracorporeal shock wave lithotripsy (ESWL, transcutaneous shock wave lithotripsy) Non-invasive technique for fragmenting kidney stones using shock waves generated outside the body.

faecal impaction A rock-hard or putty-like mass of faeces in the rectum.

faecal incontinence Loss of voluntary control of defecation.

faecalith A hard mass of faeces.

family Two or more persons joined by emotional closeness and shared bonds and who identify themselves as being part of a family.

fascial excision (fasciectomy) Process of excising the wound to the level of fascia.

fasciculations Involuntary twitching.

fat embolism syndrome (FES) Characterised by neurological dysfunction, pulmonary insufficiency, and a petechial rash on the chest, axilla and upper arms due to fat globules lodged in the pulmonary vascular bed or peripheral circulation.

fibrocystic changes (FCC) Physiological nodularity and breast tenderness that increases and decreases with the menstrual cycle.

fibroid tumours (uterine leiomyomas) Solid, pedunculated benign tumours.

fibromyalgia (fibrositis) A common rheumatic syndrome characterised by musculoskeletal pain, stiffness and tenderness.

filtration The process by which water and dissolved substances (solutes) move from an area of higher hydrostatic pressure to an area of lower hydrostatic pressure.

fistula Abnormal opening or passage between two organs or spaces that are normally separated, or an abnormal passage to the outside of the body.

flaccidity Decreased muscle tone in disease or trauma of the lower motor neurons.

flail chest Free-floating segment of the chest wall, resulting from two or more consecutive ribs fractured in multiple places.

flap A piece of tissue whose free end is moved from a donor site to a recipient site while maintaining a continuous blood supply through its connection at the base or pedicle.

flatus Gas in the digestive tract.

fluid resuscitation Replacement of the extensive fluid and electrolyte losses associated with major burn injuries.

fluid volume deficit (FVD) A decrease in intravascular, interstitial and/or intracellular fluid in the body.

fluid volume excess (FVE) Excess extracellular fluid resulting from retention of both water and sodium in the body.

focused assessment A physical assessment that concentrates on the part of the body that may be affected by disease or injury.

folic acid deficiency anaemia An anaemia resulting from folic acid deficiency, a necessary nutrient for DNA synthesis and red blood cell maturation.

folliculitis Bacterial infection of the hair follicle, most commonly caused by *Staphylococcus aureus*.

forensic mental health A subspecialty of mental health in which scientific and clinical expertise is applied in legal contexts, combining civil, criminal, correctional and legislative matters.

fracture A break in a bone, usually due to trauma.

freestanding outpatient surgical facilities Surgical units independent of a hospital with or without financial connections to a hospital or healthcare organisation.

friction rub The sound heard when two dry surfaces are rubbed together.

frostbite An injury of the skin from freezing.

full-thickness avulsion injuries Injuries that result in loss of all of the layers of the skin, causing fat and muscle to be exposed.

full-thickness burn A burn that involves all layers of the skin, including the epidermis, dermis and epidermal appendages.

fulminant hepatitis Hepatitis with a rapid and severe onset and course.

furuncle Often called a boil, but also an inflammation of the hair follicle.

fusiform excision The removal of a full thickness of the epidermis and dermis, usually with a thin layer of subcutaneous tissue.

galactorrhoea Lactation not associated with pregnancy or nursing.

gamma hydroxybutyrate A dissociative anaesthetic agent; another of the newer drugs diverted to illicit use.

gastric lavage Irrigation of the stomach with large quantities of normal saline.

gastric mucosal barrier A protective barrier consisting of lipids, bicarbonate ions and mucous gel that protects the stomach lining from the damaging effects of gastric juices.

gastric outlet obstruction Obstruction of the pyloric region of the stomach and duodenum that impairs gastric outflow; a potential complication of peptic ulcer disease.

gastric ulcers Ulcers of the stomach lining, usually in the lesser curvature and antrum; more common in older adults.

gastritis Inflammation of the stomach lining.

gastroduodenostomy (Billroth I) Excision of the pylorus of the stomach with the anastomosis of the upper stomach to the duodenum; commonly used partial gastrectomy procedure.

gastroenteritis Inflammation of the gastrointestinal tract; not a specific disease, but a group of syndromes or a collection of related manifestations.

gastrojejunostomy (Billroth II) Subtotal excision of the stomach with closure of the duodenum and side-to-side anastomosis of the jejunum to the stomach; commonly used partial gastrectomy procedure.

gastro-oesophageal reflux Backward flow of gastric contents into the oesophagus.

gastro-oesophageal reflux disease (GORD) Causes heartburn, usually after meals, when bending over or reclining.

gastroparesis Slowed gastrointestinal motility, which causes early satiety.

gene A sequence of DNA on a chromosome that represents a fundamental unit of heredity; occupies a specific spot on a chromosome (gene locus).

gene expression When the protein product of a gene is visible (e.g. through the presence of a body structure or identifiable through biochemical tests such as insulin or phenylalanine levels).

general anaesthesia Deep sedation, which includes analgesia and muscle paralysis. This type of anaesthesia requires respiratory maintenance without the aid of the person's respiratory musculature.

genetic locus The term used to describe a gene's location on a specific chromosome.

genetics The scientific study of heredity and hereditary variation.

genital herpes (herpes simplex genitalis) An infection of the external genitalia caused by herpes simplex genitalis; transmitted by vaginal, anal or oral–genital contact.

genital warts (condyloma acuminatum, venereal warts) A sexually transmitted condition caused by the human papillomavirus.

genomics The study of whole sets of genes and their interactions.

genotype The genes and the variations therein that a person inherits from his or her parents.

germ cells Cells that give rise to a sperm or egg.

gigantism Occurs when growth hormone hypersecretion begins before puberty and the closure of the epiphyseal plates, leading the person to become abnormally tall.

gingivitis Inflammation of the gums, characterised by inflammation, redness and bleeding.

gland Tissue that synthesises hormones.

glaucoma Condition characterised by increased intraocular pressure of the eye and a gradual loss of vision.

glomerular filtration rate (GFR) The rate at which plasma is filtered through the glomeruli of the kidney.

glomerulonephritis Inflammation of the capillary loops of the glomeruli.

glossitis Inflammation of the tongue.

glucocorticoid A group of hormones secreted by the adrenal cortex; they regulate carbohydrate levels in the body.

gluconeogenesis Formation of glucose from fats and proteins.

glucose-6-phosphate dehydrogenase (G6PD) anaemia Anaemia due to a hereditary defect in red blood cell metabolism.

glucosuria Excessive glucose in urine.

glycogenolysis Breakdown of liver glycogen to glucose.

goitre An enlarged thyroid gland. Enlargement results from both inadequate and excessive synthesis of thyroid hormones.

gonorrhoea (GC, clap) An infection caused by *Neisseria gonorrhoeae* that is transmitted by direct sexual contact or by delivery of a neonate by an infected mother.

gout A syndrome that occurs from an inflammatory response to the production or excretion of uric acid resulting in high levels of uric acid in the blood (hyperuricaemia) and in other body fluids, including synovial fluid.

Grave's disease Caused by a defect in immunoregulation in genetically predisposed individuals, leading to production of thyroid-stimulating antibodies.

grief The emotional response to loss and its accompanying changes.

grieving The internal process the person uses to work through the response to loss.

Guillain–Barré syndrome (GBS) Acute demyelinating disorder of the peripheral nervous system characterised by progressive, usually rapid muscle weakness and paralysis.

gynaecomastia Breast enlargement in men.

haemangioma See *angioma*.

haemarthrosis The collection of blood in the elbow joint.

haematemesis Blood in the vomit.

haematochezia Blood in the stool.

haematoma A contusion with a large amount of bleeding.

haematopoiesis Blood cell formation.

haematuria Blood in the urine.

haemianopia Loss of half of the visual field of one or both eyes.

haemiparesis Weakness of one side of the body.

haemiplegia Paralysis in one-half of the body vertically.

haemodialysis A procedure in which electrolytes, waste products and excess water are removed from the body by diffusion and ultrafiltration as blood passes by an artificial semipermeable membrane outside the body.

haemodynamics Study of the forces involved in blood circulation.

haemoglobin The oxygen-carrying protein within red blood cells; composed of the haem molecule and globin, a protein molecule.

haemolysis The process of red blood cell destruction.

haemolytic anaemia Premature destruction (lysis) of red blood cells.

haemophilia A group of hereditary clotting factor disorders that lead to persistent and potentially severe bleeding.

haemophilia A (classic haemophilia) The most common type of haemophilia, caused by clotting factor VIII deficiency.

haemophilia B (Christmas disease) Haemophilia caused by factor IX deficiency.

haemoptysis Bloody sputum.

haemorrhage Rapid or excessive bleeding.

haemorrhagic CVA (intracranial haemorrhage) Cerebrovascular accident (CVA) occurring when a cerebral blood vessel ruptures.

haemorrhoids (piles) Clusters of dilated veins in swollen anal tissue.

haemostasis Control of bleeding.

haemothorax Blood in the pleural space.

halitosis (bad breath) A common condition caused by an increase in sulfur-producing bacteria in the oral cavity.

hallucination An alteration of perception in the absence of a stimulus.

hallucinogens Drugs that produce hallucinations.

hallux valgus (bunion) The enlargement and lateral displacement of the first metatarsal.

hammer toe (claw toe) The dorsiflexion of the first phalanx with accompanying plantar flexion of the second and third phalanges.

handicap The total adjustment to disability that limits functioning at a normal level.

harm minimisation A way of reducing the impact of drug- and/or alcohol-related harm to individuals and the community through a range of cost-effective public health policies, strategies and practices.

Hashimoto's thyroiditis An autoimmune disorder caused by the development of antibodies that destroy thyroid tissue.

hazardous materials Substances that pose a potential risk to life, health or property if they are released because of their chemical, biological or physical nature.

health As defined by the World Health Organization 'A state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity.'

health determinants Factors that affect the health of people.

health education Individualistic strategies to improve health, often by increasing knowledge or influencing attitudes to alter behaviours and lifestyle. Health education is often used interchangeably with health promotion although they are not the same.

health maintenance Care focused on supporting people to manage their health and medical conditions in a way that maintains their current health and prevents further deterioration.

health promotion Any activity undertaken for the purpose of achieving a higher level of health and wellbeing.

healthcare-associated infection (HAI) An infection contracted during residence in a hospital or extended care facility.

healthcare surrogate An individual selected to make medical decisions when a person is no longer able to make them for themself.

heart block A block in the normal conduction pathways.

heart failure Inability of the heart to pump adequate blood to meet the metabolic demands of the body.

heave An excessive thrust.

hemianopia The loss of half of the visual field of one or both eyes.

hemiparesis Weakness of the left or right half of the body.

hemiplegia Paralysis of the left or right half of the body.

hepatic encephalopathy Altered consciousness, mentation and motor function affecting people with cirrhosis.

hepatitis Inflammation of the liver, usually caused by a virus; may be acute or chronic.

hepatorenal syndrome Kidney failure accompanied by azotaemia, sodium retention, oliguria and hypotension in people with cirrhosis and ascites.

hernia A defect in the abdominal wall that allows abdominal contents to protrude out of the abdominal cavity.

herniated intervertebral disc Rupture of the cartilage surrounding the intervertebral disc with protrusion of the nucleus pulposus.

herpes simplex (fever blister, cold sore) Acute viral infections of the skin and mucous membranes caused by two types of herpes virus: HSV I and HSV II.

herpes zoster (shingles) Viral infection of a dermatome section of the skin caused by varicella zoster, the same herpes virus that causes chickenpox.

heterograft (xenograft) Skin obtained from an animal, usually a pig.

heterozygous Non-identical copies of a particular gene (different alleles) on the paired chromosomes.

hiatal hernia Protrusion of part of the stomach through the oesophageal hiatus of the diaphragm into the mediastinal cavity.

hirsutism Increased growth of coarse hair, usually on the face and trunk.

histocompatibility The ability of cells and tissues to survive transplantation without immunological interference by the recipient.

Hodgkin's disease A single lymph node or chain of nodes which spreads to adjoining nodes. Involved lymph nodes contain *Reed–Sternberg cells* (malignant cells) surrounded by host inflammatory cells. These malignant cells secrete inflammatory mediator substances, attracting inflammatory cells to the tumour site. They may invade almost any tissue in the body.

holistic healthcare Care in which all aspects of a person (physical, psychosocial, cultural, spiritual and intellectual) are considered as essential components of individualised care.

home care Services for people who are in need of treatment or support to function effectively in the home environment.

homeostasis The body's tendency to maintain a state of physiological balance in the presence of constantly changing conditions.

homograft (allograft) Human skin that has been harvested from cadavers.

homologous chromosomes Chromosomes that are members of the same pair and normally have the same number and arrangement of genes; usually one copy is from the mother and the other copy is from the father.

homonymous hemianopia Impaired vision or blindness in one side of both eyes.

homozygous Identical copies of a particular gene (same alleles) on both paired chromosomes.

hope A belief in a positive outcome related to events and circumstances in one's life; the foundation of recovery from mental illness.

hordeolum (sty) Staphylococcal abscess that may occur on either the external or internal margin of the lid.

hormone Chemical messengers secreted via body fluids that have specific targets where they increase or inhibit organ functions.

hospice The delivery of care for terminally ill people either in healthcare facilities or in the person's home.

hospice care A special component of home care, designed to provide medical, nursing, social, psychological and spiritual care for terminally ill people and their families. Hospice care relies on a philosophy of relieving pain and suffering and allowing the person to die with dignity in a comfortable environment.

hot zone The site of a disaster where a weapon was released or where contamination occurred.

human genome The total amount of the DNA (genes) in an individual's cells.

human immunodeficiency virus (HIV) Virus responsible for AIDS.

Huntington's disease Progressive, degenerative, inherited neurological disease characterised by increasing dementia and chorea; also called *chorea*.

hydrocoele Fluid-filled mass within the scrotum.

hydrocephalus An abnormal accumulation of cerebrospinal fluid within the cranial vault and dilation of the ventricles.

hydronephrosis Distension of the urinary tract with urine behind an obstruction.

hydroureter Distension of the ureter with urine.

hypercapnia Increased blood levels of carbon dioxide.

hyperglycaemia Elevated blood glucose levels (above 126 mg/dL), which causes osmotic diuresis and, if chronic, damages vessel epithelium and renal glomeruli.

hyperopia (farsightedness) The condition in which the eyeball is short, causing the image to focus behind the retina.

hyperosmolar hyperglycaemic state (HHS) A condition of very high blood glucose with adequate insulin to prevent ketosis, but which does cause diuresis.

hyperparathyroidism Results from an increase in the secretion of parathyroid hormone, which regulates normal serum levels of calcium and phosphate.

hyperplasia An increase in the number or density of normal cells.

hypersensitivity Exaggerated response of the immune system to an antigen.

hypertension Excess pressure in the arterial portion of systemic circulation.

hyperthyroidism A disorder caused by excessive delivery of thyroid hormone to the peripheral tissues. Also called thyrotoxicosis.

hypertrophic scar Overgrowth of dermal tissue that remains within the boundaries of the wound.

hypervolaemia Excess intravascular fluid.

hyphaema Bleeding into the anterior chamber of the eye, possibly as the result of blunt eye trauma.

hypoglycaemia Low blood glucose levels; deficiency of blood sugar.

hypoparathyroidism A condition that results from abnormally low parathyroid hormone levels, causing hypocalcaemia and an elevated blood phosphate level.

hypothyroidism A disorder that results when the thyroid gland produces an insufficient amount of thyroid hormone.

hypovolaemia Decreased circulating blood volume.

hypovolaemic shock Shock caused by a decrease in intravascular volume of 15% or more. This form of shock is caused by the loss of whole blood, blood plasma or extracellular fluid.

hypoxaemia Decreased oxygen concentration in the blood, measured by PaO_2.

hypoxia Insufficient supply of oxygen to the tissues.

ichthyosis An inherited dermatological condition in which the skin is dry, fissured and hyperkeratotic; the surface of the skin has the appearance of fish scales.

ileostomy An ostomy made in the ileum of the small intestine.

illness A highly personal state in which the person feels unhealthy or ill; may or may not be related to disease.

illness–wellness continuum A continuum representing health as a dynamic process, with high-level wellness at one extreme of the continuum and death at the opposite extreme.

illusion A distortion of the senses.

immunity The protection of the body from disease.

immunocompetent Possessing an immune system that can identify antigens and effectively destroy or remove them.

immunocompromised Possessing an immune response that has been weakened by a disease or an immunosuppressive agent.

immunoglobulin (Ig) A protein that functions as an antibody.

immunosuppression Inability of the immune system to respond to an antigen. Occurs in response to disease or medications; may be intentional to prevent rejection of transplants or a side effect of some medications.

impairment A disturbance in structure or function resulting from physiological or psychological abnormalities.

impetigo Infection of the skin caused by either *Staphylococcus aureus* or beta-haemolytic streptococci.

impotence Inability to achieve or maintain an erection.

impulse control The ability to control behavioural impetuosity.

incident pain A type of breakthrough pain that is predictable because it is associated with movement such as turning or coughing.

increased intracranial pressure (IICP, intracranial hypertension) Sustained elevated pressure (10 mmHg or higher) within the cranial cavity.

independent nursing care Care provided by nurses within the scope of their practice without the direction or supervision of a physician.

Indigenous health A broad term which generally refers to the health status and health outcomes of the Aboriginal and Torres Strait Islander population of Australia.

infection Colonisation by and multiplication of an organism within a host. The host can be any organism capable of supporting the nutritional and physical growth requirements of the microorganism—for example, humans.

inflammation A complex, non-specific, adaptive response to injury that brings fluid, dissolved substances and blood cells into the interstitial tissues where the invasion or damage has occurred.

inflammatory bowel disease (IBD) Chronic inflammation of the bowel common to a group of conditions that includes Crohn's disease and ulcerative colitis.

influenza Highly contagious viral respiratory disease characterised by coryza, fever, cough and constitutional manifestations such as headache and malaise.

informed consent Disclosure of risks associated with the intended procedure or operation to the client. The language of the document varies according to statutory and common law of each state.

inhalants Inhaled solvents categorised into three types: anaesthetics, volatile nitrites and organic solvents.

innate immunity Specific and non-specific responses that prevent or limit the entry of invaders into the body, thereby limiting the extent of tissue damage and reducing the workload of the adaptive immune system.

insight The degree to which a person has an understanding of their illness or disorder.

instrument nurse The nurse primarily responsible for technical skills, manual dexterity and in-depth knowledge of the anatomic and mechanical aspects of a particular surgery. The instrument nurse handles sutures, instruments and other equipment immediately adjacent to the sterile field.

insulin A hormone that facilitates entry of glucose into fat and muscle cells for energy.

insulin reaction Hypoglycaemia in people with type 1 diabetes mellitus.

intermediate determinants Determinants of health which concern community infrastructure, personal wealth or access to resources, natural, physical and built environments. This level also includes access to healthcare and health systems.

International Classification of Diseases (ICD) Classification of diseases, functioning and disability.

interprofessional care Care provided by an interprofessional team where two or more professions work together as a team with a common purpose, commitment and mutual respect (Freeth et al. 2005, cited in Dunston et al., 2009, p. 6).

intersectorial Working with more than one sector of society to take action on an area of shared interest, such as health.

intracerebral haematomas A collection of blood in the brain tissue, most often located in the frontal or temporal lobes.

intracranial aneurysm Saccular outpouching of a cerebral artery that occurs at the site of a weakness in the vessel wall.

intracranial hypertension See *increased intracranial pressure*.

intracranial pressure (ICP) The pressure within the cranial cavity, usually measured as the pressure within the lateral ventricles.

intraductal papilloma A tiny wart-like growth on the inside of the peripheral mammary duct that causes discharge from the nipple.

intraoperative phase The time during surgery, from beginning to end.

iron deficiency anaemia The most common type of anaemia; results from inadequate iron for optimal red blood cell formation.

irritable bowel syndrome (IBS) A motility disorder of the gastrointestinal tract characterised by alternating periods of constipation and diarrhoea.

ischaemia Deficient blood flow to tissue.

ischaemic Deprived of oxygen.

islets of Langerhans Hormone-producing cells (alpha cells, beta cells and delta cells) scattered through the pancreas.

isograft Tissue transplant where the donor and recipient are identical twins.

jaundice Yellow-to-orange colour visible in the skin and mucous membranes; most often the result of a hepatic disorder.

joint arthroplasty Reconstruction or replacement of a joint.

Kaposi's sarcoma (KS) A vascular malignancy (a tumour of the endothelial cells lining small blood vessels) that presents as vascular macules, papules or violet lesions affecting the skin and viscera. It is often the presenting symptom of AIDS.

keloid Elevated, irregularly shaped, progressively enlarging scar arising from excessive amounts of collagen in the stratum corneum during scar formation in connective tissue repair.

keratin A fibrous, water-repellent protein that gives the epidermis its tough, protective quality.

keratitis Inflammation of the cornea.

keratosis Any skin condition in which there is a benign overgrowth and thickening of the cornified epithelium.

ketamine A central nervous system depressant, best described as a dissociative anaesthetic agent; one of the newer drugs to migrate into the illicit drug-using world.

ketoacidosis A condition of very high blood glucose and insufficient insulin that results in accumulation of ketones and fatty acids in the blood and urine and diuresis.

ketonuria The presence of ketones in the urine.

ketosis An accumulation of ketone bodies produced during the oxidation of fatty acids.

kidney failure A condition in which the kidneys are unable to remove accumulated metabolites from the blood, resulting in altered fluid, electrolyte and acid–base balance.

kidney replacement therapy Therapy provided through haemodialysis, peritoneal dialysis or kidney transplantation.

kidney transplant The surgical insertion of a functioning kidney.

kindling Long-term changes in brain neurotransmission that occur after repeated detoxifications.

kinesthaesia The ability to perceive movement and sense of position.

Klinefelter's syndrome A syndrome in which males have an extra X chromosome in most of their cells (also known as the XXY condition).

knowledge resource base Ways in which knowledge is developed.

Korotkoff's sounds Sounds heard during auscultation of blood pressure.

Korsakoff's psychosis Secondary dementia caused by thiamine (B_1) deficiency that may be associated with chronic alcoholism; characterised by progressive cognitive deterioration, confabulation, peripheral neuropathy and myopathy.

Kussmaul's respirations Deep, rapid respirations associated with compensatory mechanisms.

kwashiorkor protein energy undernutrition (protein energy malnutrition, PEM) Chronic protein deficiency with adequate kilojoules to meet body needs.

kyphosis Exaggerated thoracic curvature of the spine common in older adults.

labyrinthectomy Surgical removal of the labyrinth.

labyrinthitis Inflammation of the inner ear.

laceration Open wound that results from sharp cutting or tearing. Injuries to the integument are at risk of contamination from dirt, debris or foreign objects.

lacunar strokes Thrombotic stroke of smaller cerebral blood vessels that causes tissue to slough off, leaving a small cavity in the brain tissue.

laminectomy Removal of the lamina of the vertebrae.

laparoscopic cholecystectomy Removal of the gallbladder using an endoscope.

laryngectomy Removal of the larynx.

laryngitis Inflammation of the larynx.

leiomyoma See *fibroid tumours*.

leucocytes Also called white blood cells, these are the primary cells involved in both non-specific and specific immune system responses. These cells isolate the infecting organism or injury, destroy pathogens and promote healing.

leucocytosis An increase in the number of leucocytes in the blood (above 10 000/mm^3), usually caused by infection.

leucopenia Abnormal decrease of circulating leucocytes, usually below 5000/mm^3; occurs when bone marrow activity is suppressed, or when leucocyte destruction increases.

leucoplakia Formation of white patches or spots on the mucous membranes or tongue; these lesions may become malignant.

leukaemia ('white blood') A group of chronic malignant disorders of white blood cells (WBCs) and WBC precursors; characterised by replacement of bone marrow by malignant immature WBCs, abnormal immature circulating WBCs and infiltration of malignant cells into other tissues.

libido Instinctual drive associated with sexual desire.

lichen planus Benign inflammatory disorder of the mucous membranes and skin.

life-limiting illness Any illness where it is expected that death will be a direct consequence of the specified illness.

lift A more sustained thrust than normal.

lipoatrophy Atrophy of subcutaneous tissue.

lipodystrophy Hypertrophy of subcutaneous tissue that may result if the same injection sites are used repeatedly, especially with pork and beef insulins.

liposuction A method of changing the contours of the body by aspirating fat from the subcutaneous layer of tissue.

lithiasis Stone formation.

lithotripsy Crushing of renal calculi.

liver transplantation Surgery to remove a diseased liver and transplant a healthy liver (whole or segment) from another person.

lobectomy Surgical removal of tumours in a single lobe of lung.

locked-in syndrome Person is alert and fully aware of the environment, but is unable to communicate through speech or movement as a result of blocked efferent pathways to the brain.

lordosis Increased lumbar curve.

loss An actual or potential situation in which a valued ability, object or person is inaccessible or changed so that it is perceived as no longer valuable.

lower body obesity (peripheral obesity) A waist-to-hip ratio of less than 0.8.

lung abscess Localised area of lung destruction or necrosis and pus formation.

lung compliance Distensibility of the lungs.

lyme disease An inflammatory disorder caused by a spirochete, *Borrelia burgdorferi*, which is transmitted primarily by ticks.

lymphadenopathy The enlargement of lymph nodes (over 1 cm) with or without tenderness. It may be caused by inflammation, infection or malignancy of the nodes or the regions drained by the nodes.

lymphangitis Inflammation of a lymphatic vessel.

lymphocytes Lymphocytes account for 20–40% of circulating leucocytes. Lymphocytes are the principal effector and regulator cells of specific immune responses.

lymphoedema Extremity oedema due to accumulated lymph; may be primary or secondary, resulting from inflammation, obstruction or removal of lymphatic vessels.

lymphoid tissues Connective tissues containing lymphocytes; include tissues of the bone marrow, thymus, lymph nodes and spleen.

lymphomas Malignancy of lymphoid tissue.

macrophages Monocytes mature into macrophages after settling into tissue. Macrophages are large phagocytes. They are important in the body's defence against chronic infections.

macular degeneration Destructive changes in the macula due to injury or gradual failure of the outer pigmented layer of the retina (the retinal layer adjacent to the choroid), which removes cellular waste products and keeps the retina attached to the choroid.

major trauma Serious single-system injury (such as the traumatic amputation of a leg) or multiple-system injuries. Also known as multiple trauma.

malabsorption A condition in which nutrients are ineffectively absorbed by the intestinal mucosa, resulting in their excretion in the stool.

malignant hypertension A hypertensive emergency, marked by a diastolic pressure greater than 120 mmHg.

malignant melanoma (cutaneous melanoma) Skin cancer that arises from melanocytes.

malignant pain Pain associated with a life-threatening illness such as cancer but not limited to cancer pain.

malnutrition Inadequate nutrient intake to meet body needs; may include deficiency of major nutrients (kilojoules, carbohydrates, proteins and fats) or micronutrients such as vitamins and minerals.

manifestations Signs and symptoms of a disease or condition caused by alterations in structure or function.

man-made disasters Either accidental or intentional, they are complex emergencies, technological disasters, material shortages and other disasters not caused by natural hazards.

marasmus (protein energy undernutrition) Insufficient protein and kilojoule intake to meet metabolic needs.

mass casualty incidents Situations in which 100 or more casualties are involved, significantly overwhelming available emergency medical services, facilities and resources.

mastoidectomy Surgical removal of infected mastoid air cells.

mastoiditis Bacterial infection of the mastoid process.

maturity-onset diabetes of the young (MODY) Diabetes in young obese adults.

mean arterial pressure (MAP) The average pressure in the arterial circulation throughout the cardiac cycle; the product of cardiac output and systemic vascular resistance (SVR).

medical–surgical nursing The health promotion, healthcare and illness care of adults, based on knowledge derived from the arts and sciences and shaped by knowledge (the science) of nursing.

meiosis A modified type of cell division in sexually reproducing organisms consisting of two rounds of cell division but only one round of DNA replication. It results in cells with half the number of chromosome sets as the original cell.

melanin Skin pigment that forms a protective shield to protect keratinocytes and nerve endings in the dermis from the damaging effects of ultraviolet light.

melaena Black, tarry stool that contains blood.

memory The ability to store, retain and recall information.

Ménière's disease Chronic disorder of unknown cause characterised by recurrent attacks of vertigo with tinnitus and a progressive unilateral hearing loss.

meningitis Inflammation of the meninges of the brain and spinal cord.

menopause Permanent cessation of menses.

menorrhagia Excessive or prolonged menstruation.

menstrual cycle Cyclic build-up of the uterine lining, ovulation and sloughing of the lining occurring approximately every 28 days in non-pregnant females.

menstruation Periodic shedding of the uterine lining in a woman of childbearing age who is not pregnant.

Mental Health Act Mental health legislation.

mental state assessment (MSA) A clinical assessment that describes the sum total of the examiner's observations at the time of the interview or interaction.

metabolic syndrome A cluster of manifestations often associated with type 2 diabetes. Includes hypertension, visceral obesity, low high-density lipoproteins, high triglycerides, elevated C-reactive protein and fasting glucose > 110mg/dL.

metabolism Consisting of the breakdown of complex structures into simpler forms to produce energy (catabolism) and the combination of simpler molecules to produce and maintain more complex structures necessary to living organisms (anabolism).

metaplasia A change in the normal pattern of differentiation such that dividing cells differentiate into cell types not normally found in that location in the body.

metastasis Secondary tumour; the process by which spreading of malignant neoplasms occurs; the transfer of disease from one organ or part to another not directly connected with it.

metrorrhagia Bleeding between menstrual periods; may be caused by hormonal imbalances, pelvic inflammatory disease, cervical or uterine polyps, uterine fibroids, or cervical or uterine cancer.

microalbuminuria Protein in the urine.

micturition Releasing urine from the urinary bladder (voiding).

mild concussion Brain trauma resulting in a brief loss of consciousness that lasts from seconds to hours.

minor trauma Injury to a single part or system of the body, usually treated in the hospital or emergency department.

mitigation The action taken to prevent or reduce the harmful effects of a disaster on human health or property; it involves future-oriented activities to prevent subsequent disasters or to minimise their effects.

mitochondria Mitochondria provide the energy a cell needs to move, divide, produce secretory products and contract.

mitosis A process of nuclear division in eukaryotic cells conventionally divided into five stages: prophase, prometaphase, metaphase, anaphase and telophase. Mitosis conserves chromosome numbers by allocating replicated chromosomes equally to each of the daughter nuclei.

mitral valve (bicuspid valve) Valve between the left atrium and ventricle in the heart; prevents blood from flowing backwards into the atrium.

monosomy (monosomic) When one member of the chromosome pair is missing—for example, in Turner syndrome (45, X0).

mood The way in which a person describes their feelings at a particular time.

morbid obesity Weight greater than 100% over ideal body weight.

Morton's neuroma A tumour-like mass formed within the neurovascular bundle of the intermetatarsal spaces.

mosaicism A chromosome variation or abnormality that occurs after fertilisation during mitosis at an early cell stage, so that not all cells are affected with the variation; for example, a child who is mosaic for Down syndrome will have some cells with two copies of chromosome 21 and some that have an extra chromosome 21.

mourning The actions or expressions of the bereaved, including the symbols, clothing and ceremonies that make up the outward manifestations of grief.

multifactorial Health conditions determined by multiple factors, including genetic and environmental factors, each having an additive effect.

multiple casualty incidents Incidents in which more than 2 but fewer than 100 persons are injured.

multiple myeloma A malignancy in which plasma cells multiply uncontrollably and infiltrate the bone marrow, lymph nodes, spleen and other tissues.

multiple sclerosis (MS) A chronic degenerative disease of the central nervous system primarily affecting the white matter.

multiple trauma Most often the result of a motor vehicle crash, this type of trauma requires immediate intervention specifically focused on ensuring survival.

murmurs Sounds made by turbulent blood flow through the heart.

muscular dystrophy (MD) A group of inherited muscle diseases that cause progressive muscle degeneration and wasting.

myasthenia gravis Chronic, progressive neuromuscular disorder characterised by fatigue and severe weakness of skeletal muscles.

myocarditis Inflammatory disorder of the heart muscle.

myopia (nearsightedness) A condition in which the eyeball is elongated, causing the image to focus in front of the retina instead of on it.

myringotomy Incision of the tympanic membrane.

myxoedema Systemic condition that develops from inadequate levels of thyroid hormone.

myxoedema coma A life-threatening complication of longstanding, untreated hypothyroidism usually triggered by an acute illness or trauma.

naevi (moles) Flat or raised macules or papules with rounded, well-defined borders.

natural disasters Disasters caused by acts of nature or emerging diseases. Some are unexpected, and some are predictable through advanced meteorological technologies.

natural killer cells (NK cells, null cells) Large, granular lymphocytes (found in the spleen, lymph nodes, bone marrow and blood) that provide immune surveillance and resistance to infection and play an important role in the destruction of early malignant cells.

nausea An unpleasant sensation usually followed by vomiting.

necrosis Tissue cell death.

neglect syndrome (unilateral neglect) A disorder of attention. In this syndrome, the person cannot integrate and use perceptions from the affected side of the body or from the environment on the affected side and, hence, ignores that part.

neoplasm A mass of new tissue (a collection of cells) that grows independently of its surrounding structures and has no physiological purpose.

nephrectomy Removal of the kidney.

nephrotic syndrome A condition marked by massive proteinuria, hypoalbuminaemia, hyperlipidaemia and oedema.

neurogenic bladder Dysfunctional urinary bladder due to lesion of central or peripheral nervous system.

neurogenic shock Shock resulting from an imbalance between parasympathetic and sympathetic stimulation of vascular smooth muscle. If parasympathetic overstimulation or sympathetic understimulation persists, sustained vasodilation occurs and blood pools in the venous and capillary beds.

neuropathic pain Pain caused by a lesion or dysfunction in the nervous system from the primary afferent conducting mechanism to the central nervous system.

neuropathy Damage to peripheral nerves causing hyper- or hyposensation and leading to pain and injury.

neutropenia A decrease in circulating neutrophils.

nicotine An alkaloid found in the nightshade family of plants (*Solanaceae*) which constitutes approximately 0.6–3.0% of the dry weight of tobacco, with biosynthesis taking place in the roots and accumulating in the leaves.

nociception The physiological processes related to pain perception.

nociceptors Sensory nerve fibres that conduct pain impulses from the periphery to the central nervous system.

nocturia Voiding two or more times at night.

node Elements of the immune system connected by lymphatics; upregulates immune function; does not synthesise hormones.

non-conventional terrorist weapons Chemical, biological and nuclear weapons of terrorism; used less frequently than conventional terrorist weapons.

nondisjunction An error in meiosis or mitosis in which members of a pair of homologous chromosomes or a pair of sister chromatids fail to separate properly from each other.

non-Hodgkin's lymphoma (NHL) Lymphoid tissue malignancies that do not contain Reed–Sternberg cells.

non-union A state that exists when the ends of a fracture fail to heal together.

normal sinus rhythm (NSR) Normal heart rhythm, in which impulses originate in the sinus node and travel through normal conduction pathways without delay.

nosocomial Pertaining to or occurring in a hospital.

nosocomial infection Infection contracted during residence in a hospital or extended care facility.

nuclear terrorism Use of a nuclear device to cause mass murder and devastation.

nursing process The series of critical thinking activities nurses use as they provide care to clients; this logical approach to care ensures that clients receive comprehensive and effective care.

nutrients Substances found in food that are used by the body to promote growth, maintenance and repair.

nutrition The process by which the body ingests, absorbs, transports, uses and eliminates food.

nystagmus Rapid involuntary eye movements.

obesity An excess of body fat (adipose tissue).

obstructive shock Shock caused by an obstruction in the heart or great vessels that either impedes venous return or prevents effective cardiac pumping action.

occult bleeding Hidden bleeding.

occult blood Blood that is not obvious on examination from a non-specific source, with obscure signs and symptoms. It may be detected by means of a chemical text or immunological reaction. Occult blood is often present in the stools of people with gastrointestinal lesions.

oedema Accumulation of fluid in the body's tissues; an excess accumulation of fluid in the interstitial space.

oesophageal varices Enlarged, thin-walled veins that form in the submucosa of the oesophagus.

oesophagojejunostomy Removal of the entire stomach with anastomosis of the distal oesophagus to the jejunum.

oestrogen Hormone produced by the ovary.

oligomenorrhoea Scant menses.

oliguria Urine output of less than 400 mL in 24 hours.

oncogene Gene capable of triggering cancerous characteristics.

oncology The study of cancer.

onycholysis The separation of the distal nail plate from the nail bed.

onychomycosis A fungal or dermatophyte infection of the nail plate.

opioids A chemical that works by binding to opioid receptors, which are found principally in the central nervous system and the gastrointestinal tract.

orchitis Infection or inflammation of the testicle.

orthopnoea Difficulty breathing when supine.

orthostatic hypotension A decrease in systolic blood pressure of more than 10 to 15 mmHg and a drop in diastolic blood pressure on standing.

osmosis The process by which water moves across a selectively permeable membrane from an area of lower solute concentration to an area of higher solute concentration.

ossification The process of bone formation.

osteitis deformans See *Paget's disease of bone*.

osteoarthritis (degenerative joint disease) The most commonly occurring of all forms of arthritis. This disease is characterised by loss of articular cartilage in articulating joints and hypertrophy of the bones at the articular margins.

osteomalacia (adult rickets) Metabolic bone disorder characterised by inadequate mineralisation of bone matrix.

osteomyelitis Infection within the bone that can lead to tissue death and necrosis.

osteophytes Bony outgrowths often called 'joint mice'.

osteoporosis Literally defined as 'porous bones', a metabolic bone disorder characterised by loss of bone mass, increased bone fragility and an increased risk of fractures.

osteotomy An incision into or transection of the bone.

ostomy General term for an operation in which an artificial opening is created.

otitis externa Inflammation of the ear canal, commonly known as *swimmer's ear*.

otitis media Inflammation or infection of the middle ear.

otorrhoea Leakage of cerebrospinal fluid through the ear.

otosclerosis Abnormal bone formation in the osseous labyrinth of the temporal bone causing the footplate of the stapes to become fixed or immobile in the oval window. The result is a conductive hearing loss.

ovarian cycle The female cycle that occurs from puberty until menopause in which the production of ova occur.

oxyhaemoglobin The combined form of haemoglobin and oxygen; found in arterial blood, it carries oxygen to body tissues.

pacemaker A pulse generator used to provide an electrical stimulus to the heart when the heart fails to generate or conduct on its own a rate that maintains the cardiac output.

$paCO_2$ Partial pressure of carbon dioxide in arterial blood.

Paget's disease of bone (osteitis deformans) A skeletal disorder that results from excessive osteoclastic activity. Paget's disease is characterised by bone deformity, especially of the long bones of the lower limbs, the pelvis, the lumbar vertebrae and the skull.

pain A subjective response to both physical and psychological stressors.

pain tolerance The amount of pain a person can endure before responding to it.

palliative care An area of care that has evolved out of the hospice experience, but exists outside of hospice programs and is not restricted to the end of life. Palliative care is focused on the relief of physical, mental and spiritual distress for individuals who have an incurable illness and is used earlier in the disease experience than hospice care. The goal of palliative care is to prevent and relieve suffering by early assessment and treatment of pain and other physical, psychosocial and spiritual needs to improve the person's quality of life.

pallor Lack of colour; paleness of skin.

pancreatitis Inflammation of the pancreas.

pannus Granulation tissue that forms in joints affected by rheumatoid arthritis and leads to the formation of scar tissue that immobilises the joint.

PaO_2 Partial pressure of oxygen in arterial blood.

papilloedema Swelling of the optic nerve.

paracentesis Aspiration of fluid from the peritoneal cavity.

paralytic ileus Impaired propulsion or forward movement of bowel contents.

paraplegia Paralysis of the lower portion of the body, sometimes involving the lower trunk.

parasites Organisms that live within, on or at the expense of the person.

parenchyma The key elements of an organ essential to its functioning, as distinct from the capsule that encompasses it and other supporting structures.

Parkinson's disease (PD) Progressive, degenerative neurological disease characterised by non-intention tremor, bradykinesia and muscle rigidity.

paronychia An infection of the cuticle of the fingernails or toenails.

paroxysmal Abrupt onset and termination.

paroxysmal nocturnal dyspnoea (PND) Attacks of acute shortness of breath that occur at night, waking up the person.

partial gastrectomy Removal of a portion of the stomach, usually the distal half to two-thirds.

partial seizures Seizures that involve a restricted part of one cerebral hemisphere; may be simple partial (without loss of consciousness) or complex partial (with loss of consciousness).

partial-thickness burn Burn that involves the entire dermis and the papillae of the dermis (superficial partial-thickness burn) or extends into the hair follicles (deep partial-thickness burn).

passive immunity Temporary protection—provided by antibodies produced by other people or animals—against disease-producing antigens. Protection is gradually lost when these acquired antibodies are used up either by natural degradation or by combining with the antigen.

pathogens Virulent organisms rarely found in the absence of disease.

patient A person who is waiting for or undergoing medical treatment or care. The term used interchangeably with *client* in the Australian nursing context.

patient-controlled analgesia (PCA) A pump with a control mechanism that allows the person to self-manage pain.

pediculosis An infestation with lice, parasites that live on the blood of an animal or human host.

pediculosis capitis An infestation with head lice.

pediculosis corporis An infestation with body lice.

pediculosis pubis An infestation with pubic lice (often called 'crabs').

pelvic inflammatory disease (PID) A term used to describe infection of the pelvic organs.

pemphigus vulgaris Chronic disorder of the skin and oral mucous membranes characterised by vesicle (blister) formation.

penetrance The percentage or likelihood that an individual who has inherited a gene mutation will actually express the disease signs and symptoms in his or her lifetime.

penetrating trauma Occurs when a foreign object enters the body causing damage to body structures.

penicillin-resistant *Streptococcus pneumoniae* (PRSP) Infection transmitted by droplets from the respiratory tract; requires transmission-based droplet precautions.

peptic ulcer An ulcer that occurs in any area of the gastrointestinal tract exposed to acid-pepsin secretions, including the oesophagus, stomach or duodenum.

peptic ulcer disease (PUD) A break in the mucous lining of the gastrointestinal tract where it comes in contact with gastric juice.

perforation Penetration of ulcer through mucosal wall.

pericarditis Inflammation of the pericardium.

perioperative nursing A specialised area of nursing practice that incorporates the three phases of the surgical experience: preoperative, intraoperative and postoperative.

peripheral obesity Obesity characterised by a waist-to-hip ratio of less than 0.8, more commonly seen in women.

peripheral vascular disease (PVD) Impaired blood supply to peripheral tissues, particularly the lower extremities.

peripheral vascular resistance (PVR) The opposing forces or impedance to blood flow as the arterial channels become more and more distant from the heart.

peristalsis Alternating waves of contraction and relaxation of involuntary muscle.

peritoneal dialysis Procedure in which electrolytes, waste products and excess water are removed from the body by diffusion using the peritoneum surrounding the abdominal cavity as the dialysing membrane.

peritonitis Inflammation of the peritoneum.

pernicious anaemia Anaemia resulting from failure to absorb dietary vitamin B_{12} due to lack of intrinsic factor.

persistent (chronic) pain Ongoing and prolonged pain, not always associated with an identifiable cause but often arising from an acute cause.

persistent vegetative state (PVS) Condition of complete unawareness of self and the environment.

personal protective equipment (PPE) Equipment used for the protection of personnel including gloves, masks, goggles, gowns and biological disposal bags (red bags); may also include hoods, helmets, headgear and impermeable suits.

personal responsibility (empowerment) Admitting responsibility for choices made.

person-centred care A holistic approach to the planning, delivery and evaluation of healthcare that is grounded in mutually beneficial partnerships between healthcare professionals, patients and families. Person-centred care is underpinned by the principles of trust, empathy, dignity, autonomy, respect, choice, transparency and desire to help individuals lead the life they want.

pertussis (whooping cough) A highly contagious acute upper respiratory infection cause by the bacterium *Bordetella pertussis*.

phagocytosis A process by which a foreign agent or target cell is engulfed, destroyed and digested. Neutrophils and macrophages, known as phagocytes, are the primary cells involved in phagocytosis.

phantom limb syndrome (phantom pain) A confusing pain syndrome that occurs following surgical or traumatic amputation of a limb. The person experiences pain in the missing body part even though there is complete mental awareness that the limb is gone.

pharmacogenetics The study of how genetic factors influence drug action.

pharyngitis Acute inflammation of the pharynx.

phenotype The expression of a person's entire physical, biochemical and physiological make-up, as determined by the individual's genotype and environmental factors.

pheochromocytoma Tumours of chromaffi in tissues in the adrenal medulla. These tumours, which are usually benign, produce catecholamines (adrenaline or noradrenaline, also known as epinephrine and norepinephrine) that stimulate the sympathetic nervous system.

phimosis Constriction of the foreskin so that it cannot be retracted over the glans penis.

plasmapheresis (plasma exchange) Removal of the plasma component from whole blood.

plastic surgery The alteration, replacement or restoration of visible portions of the body, performed to correct a structural or cosmetic defect.

platelets (thrombocytes) Cell fragments that have no nucleus and cannot replicate.

pleural effusion Collection of excess fluid in the pleural space.

pleuritis Inflammation of the pleura.

pneumonectomy Removal of an entire lung.

pneumonia Inflammation of the lung parenchyma (the respiratory bronchioles and alveoli).

pneumothorax Results when air enters the pleural space due to blunt and penetrating injuries to the chest.

polycystic kidney disease (PKD) A hereditary disease characterised by cyst formation and massive kidney enlargement.

polycythaemia (erythrocytosis) Excess red blood cells characterised by a haematocrit higher than 55%.

polydipsia Excessive thirst.

polymorphisms DNA sequences that have many forms but give the genetic 'directions' for the same thing.

polymyositis A systemic connective-tissue disorder characterised by inflammation of connective tissue and muscle fibres leading to muscle weakness and atrophy.

polyp Mass of tissue that arises from the bowel wall and protrudes into the lumen.

polyphagia Excessive eating.

polysubstance abuse The simultaneous use of many substances.

polyuria A condition where increased blood volume increases renal blood flow and the hyperglycaemia acts as an osmotic diuretic, thereby increasing urine output.

portal hypertension Elevated pressure in the portal venous system that causes rerouting of blood to adjoining lower pressure vessels.

portal systemic encephalopathy Impaired consciousness and mental status due to the accumulation of toxic waste products in the blood (ammonia in particular) as blood bypasses the congested liver.

positioning Positioning exposes the operative site in conjunction with access for anaesthesia administration. Proper positioning is imperative to prevent injury to the person.

post-acute care See *acute care*.

post-concussion syndrome Persistent headache, dizziness, irritability, insomnia, impaired memory and concentration and learning problems following a concussion; may last for several weeks or up to 1 year.

postoperative phase Period when a procedure or surgery has been completed and the person is recovering from the stress associated with the surgery.

postpoliomyelitis syndrome A complication of a previous infection by the poliomyelitis virus.

preload The amount of cardiac muscle fibre tension or stretch that exists at diastole, just before ventricular contraction.

premenstrual syndrome (PMS) Complex of symptoms characterised by irritability, depression, oedema and breast tenderness preceding the monthly menses.

preparedness Having a comprehensive disaster plan in place that coordinates efforts among many people, agencies and levels of government.

preoperative phase Time when preparation of the person for surgery is conducted and completed.

presbycusis Age-related loss of the ability to hear high-frequency sounds; may occur because of cochlear hair cell degeneration or loss of auditory neurons in the organ of Corti.

presbyopia Impaired near vision resulting from a loss of elasticity of the lens related to ageing.

pressure injury Ischaemic lesion of the skin and underlying tissue caused by external pressure that impairs the flow of blood and lymph.

pretibial myxoedema Also known as thyroid dermopathy, pretibial myxoedema refers to lesions of the skin resulting from the accumulation of hyaluronic acid, as a result of thyroid disease.

priapism Sustained, painful erection that lasts at least 4 hours and is not associated with sexual arousal.

primary care Community-based day-to-day healthcare provided by a healthcare provider who acts as the first contact, and principal point of continuing care, for people within the healthcare system, as well as coordinating other specialist care (e.g. general practitioner). Primary care services focus on a person's specific disease or illness using a disease or biomedical management orientation.

primary healthcare (PHC) Essential healthcare based on practical, scientifically sound and socially acceptable methods and technology made universally accessible to individuals and families in the community through their full participation and at a cost that the community and country can afford to maintain in order to prevent illness in the population for which it is provided. A sustainable, community-driven and evidence-based healthcare approach underpinned by the social, cultural, economic and practical determinants of the community.

primary hypertension (idiopathic, essential) A persistently elevated systemic blood pressure.

primary polycythaemia (polycythaemia vera) A neoplastic stem cell disorder characterised by overproduction of red blood cells and, to a lesser extent, white blood cells and platelets.

primary prevention Activities directed toward the protection from or avoidance of potential health risks.

primary survey An initial assessment of a person to determine if there is serious compromise to airway, breathing or circulation.

professional boundaries A health professional's individual determination of the limits of the therapeutic relationship.

progesterone Hormone produced by the ovary; works with oestrogen to control the menstrual cycle.

proptosis Forward bulging of one or both eyes.

prostatitis Inflammation of the prostate gland.

protein energy malnutrition (PEM) The state of decreased body pools of protein with or without fat depletion or a state of diminished functional capacity, caused at least partly by inadequate nutrient intake relative to nutrient demand and/or which is improved by nutritional repletion.

protein-kilojoule malnutrition Deficient protein and kilojoules to meet metabolic needs.

proteinuria Abnormal proteins in the urine.

proximal determinants Determinants which have a more direct impact on health, and include lifestyle and behavioural factors as well as underlying health conditions.

pruritus Subjective itching sensation producing an urge to scratch.

psoriasis Chronic, non-infectious skin disorder that is characterised by raised, reddened, round circumscribed plaques covered by silvery white scales.

psychogenic pain Pain that is experienced in the absence of any diagnosed physiological cause or event.

psychosis A mental health condition in which there is a loss of contact with reality.

psychostimulants A diverse group of natural and synthetic drugs with a wide range of psychological and physical effects, including euphoria, increased energy and irregular heartbeat.

ptosis Drooping of the eyelid.

pulmonary oedema An abnormal accumulation of fluid in the interstitial tissue and alveoli of the lung.

pulmonary embolism Sudden occlusion of a pulmonary artery resulting in disruption of blood supply to the lung parenchyma.

pulmonary hypertension Condition in which the pulmonary arterial pressure is elevated to an abnormal level.

pulmonic valves One of the semilunar valves, separating the ventricles from the great vessels.

pulse Rhythmic pressure wave that can be felt over an artery.

pulse deficit Condition in which the radial pulse is less than the apical pulse, indicating weak, inefficient left ventricular contractions.

pulse pressure The difference between the systolic and diastolic blood pressure.

puncture wound Wound that occurs when a sharp or blunt object penetrates the integument.

pupillary light reflex Reflex in which the pupil contracts in response to a bright light.

pyelonephritis Upper urinary tract inflammation affecting the kidney and renal pelvis.

pyoderma Purulent bacterial infection of skin.

pyuria (bacteriuria) Pus in the urine.

quadriplegia See *tetraplegia*.

quality assurance The process of ensuring quality control activities that evaluate, monitor or regulate the standard of services provided to the consumer.

rabies Viral (rhabdovirus) infection of the central nervous system transmitted by infected saliva that enters the human body through a bite or an open wound.

radiation sickness One of the results of DNA mutation inside cells exposed to ionising radiation.

radiation therapy Therapy that uses radiation to kill a tumour, to reduce its size, to decrease pain or to relieve obstruction.

radiological dispersion bomb Also called a 'dirty bomb', consists of a conventional explosive such as trinitrotoluene (TNT) packed with radioactive waste by-products from nuclear reactors that discharge deadly radioactive particles into the environment.

Raynaud's disease (Raynaud's phenomenon) Disorders characterised by episodes of intense vasospasm in the small arteries and arterioles of the fingers and possibly the toes.

reactive arthritis (Reiter's syndrome) An acute, non-purulent inflammatory arthritis that complicates a bacterial infection of the genitourinary or gastrointestinal tracts.

rebound tenderness Pain that occurs with withdrawal or release of pressure applied during abdominal palpation.

recessive A characteristic that is apparent only when two copies of the gene encoding it are present, one from the mother and one from the father.

reconstruction The recovery aspect of disaster response; during this period restoration, reconstitution and mitigation take place.

recovery A recognised and accepted paradigm, related to a personal journey, that has significant implications for people who have mental health problems, their carers, mental health professionals and mental health services.

recovery (stage of disaster) The stage where restoration, reconstitution and mitigation take place. See also *reconstruction*.

red blood cell (RBCs, erythrocytes) Blood cells shaped like a biconcave disc that contain haemoglobin required for oxygen transport to body tissues; the most common type of blood cell.

referral Timely consultation and handing over of clinical care of people to the appropriate personnel and facility for ongoing management.

referred pain Pain that is perceived in an area distant from the site of the stimuli.

reflex sympathetic dystrophy A group of poorly understood post-traumatic conditions involving persistent pain, hyperaesthesias, swelling, changes in skin colour and texture, changes in temperature and decreased motion.

reflux, urinary Backflow of urine towards the kidneys.

refraction The bending of light rays as they pass from one medium to another medium of different optical density.

refractory period A period in which myocardial cells are resistant to stimulation.

regional anaesthesia Anaesthesia that desensitises the area to be operated but does not involve the full central nervous system or cause sedation.

regional, rural and remote health workforce A statistical representation of nurses working in identified rural and remote areas which includes the number of currently employed nurses, acknowledging their age, experience and qualifications.

regurgitation (valvular) Backflow of blood through an incompletely closed valve into the area it just left.

rehabilitation The process of learning to live to one's maximum potential with a chronic impairment and its resultant functional disability.

Reiter's syndrome See *reactive arthritis*.

remission A period in which symptoms are not experienced even though the disease is still clinically present.

renal artery stenosis Narrowing of the renal artery.

renal colic Acute, severe, intermittent pain in the flank and upper outer abdominal quadrant generally associated with acute obstruction of a ureter and resulting ureteral spasm.

renal failure See *kidney failure*.

renal impairment (decreased renal reserve) A glomerular filtration rate of approximately 50% of normal with normal blood urea nitrogen (BUN) and serum creatinine levels.

renal insufficiency A glomerular filtration rate of 20–50% of normal with azotaemia and some manifestations of renal failure.

renal replacement therapy (RRT) See *kidney replacement therapy*.

renal transplant See *kidney transplant*.

renin–angiotensin mechanism Method of controlling the glomerular filtration rate by releasing chemicals that cause intense vasodilation of the afferent arterioles. Conversely, an increase in the flow of filtrate results in the promotion of vasoconstriction, decreasing the glomerular filtration rate.

repolarisation Restoration of the resting cell membrane potential following generation of an action potential.

respiratory failure Inability of lungs to oxygenate the blood and remove carbon dioxide adequately to meet the body's needs, even at rest.

respite care Short-term or intermittent home care, often using volunteers. These services exist to give the primary caregiver some relief from the burden of full-time care.

response Occurs in the emergency stage of disaster response, after the impact of the disaster event has occurred, the community has been rapidly assessed for damage, and the types and extent of injuries suffered as well as the immediate needs of the community have been determined.

reticular activating system (RAS) A system of reticular neurons within the reticular formation that passes steady streams of impulses through thalamic relays in order to stimulate the cerebral cortex into wakefulness.

retinal detachment Separation of the retina or sensory portion of the eye from the choroid.

retinitis pigmentosa Hereditary degenerative disease characterised by retinal atrophy and loss of retinal function progressing from the periphery to the central region of the retina.

retractions A pulling in of the tissue of the precordium; a slight retraction just medial to the midclavicular line at the area of the apical impulse is normal and is more likely to be visible in thin people.

retrieval Specialised transfer of people with needs exceeding the capacity of their current location to a clinical facility providing a higher level of specialised healthcare.

retrograde ejaculation Seminal fluid discharged into the bladder.

reverse triage Working from the principle of the greatest good for the greatest number, reverse triage is an 'upside-down triage' used in mass casualty events in which the victims who are most severely injured, requiring extensive resources with little chance of surviving, are treated last.

rheumatic disorders Refers to diseases of the muscles and bones as well as the joints.

rheumatic fever A systemic inflammatory disease caused by an abnormal immune response to pharyngeal infection by group A beta-haemolytic streptococci. The condition is characterised by acute inflammation, joint pain, fever and cardiac valve scarring.

rheumatic heart disease (RHD) Slowly progressive valvular deformity following acute or repeated attacks of rheumatic fever; characterised by rigid and deformed valve leaflets; fused valve commissures and fibrosis of chordae tendineae.

rheumatoid arthritis A chronic systemic autoimmune disease that causes inflammation of connective tissue, primarily in the joints.

rhinitis Inflammation of the nasal cavities.

rhinoplasty Surgical reconstruction of the nose.

rhinorrhoea Leakage of cerebrospinal fluid through the nose.

risk factors Defined as individual or environmental variables that are related to the increased likelihood that a negative outcome will occur.

ruptured disc See *herniated intervertebral disc*.

salmonellosis A form of food poisoning caused by ingestion of foods contaminated with one or more varieties of *Salmonella* bacteria.

sarcoidosis Systemic disease characterised by granulomas in the lungs, lymph nodes, liver, eyes, skin and other organs.

sarcoma A tumour arising from supportive tissues.

scabies Parasitic infestation caused by the mite *Sarcoptes scabei*.

schizophrenia A mental disorder characterised by abnormalities in the perception or expression of reality.

sciatica Pain over the sciatic nerve.

scleroderma Hardening of the skin; a chronic condition characterised by the formation of excess fibrous connective tissue and diffuse fibrosis of the skin and internal organs.

sclerotherapy The removal of benign skin lesions with a sclerosing agent that causes inflammation with fibrosis of tissue.

scoliosis A lateral curvature of the spine.

scope of practice Referred to when nurses and employers are deciding what constitutes nursing duty.

scrub person Prepares the sterile field, surgical supplies and equipment for surgical procedures; also assists surgeon and physician assistant by passing instruments, suctioning blood and maintaining the sterile field.

seborrhoeic dermatitis Common and chronic inflammatory disorder of the skin that involves the scalp, eyebrows, eyelids, ear canals, nasolabial folds, axillae and trunk. The cause is unknown.

sebum An oily substance secreted from sebaceous glands; softens and lubricates the skin and hair and also decreases water loss from the skin in low humidity. Sebum also protects the body from infection by killing bacteria.

secondary hypertension Elevated blood pressure resulting from an identifiable underlying process.

secondary prevention Activities designed for early diagnosis and treatment of disease or illness.

secondary survey A head-to-toe assessment of a person that includes all body systems.

seizure An episode of excessive and abnormal discharge of electrical activity within the central nervous system.

semen Contains sperm and fluids secreted by the male reproductive system glands.

seminoma A tumour from seminal or germ tissue.

septic arthritis The type of arthritis that develops when a joint space is invaded by a pathogen.

septic shock One part of a progressive syndrome called systemic inflammatory response syndrome. Beginning with an infection, septic shock progresses to bacteraemia, then sepsis, then septic shock and finally multiple organ failure syndrome.

septicaemia Systemic disease associated with the presence of bacteria or their toxins in the blood.

seroconversion Antibody response to a disease or vaccine.

serum biocarbonate (HCO_3^-) Reflects the renal regulation of acid–base balance. It is often called the metabolic component of arterial blood gases.

severe acute respiratory syndrome (SARS) Lower respiratory illness of unknown aetiology; spread by close person-to-person contact.

sex chromosome A chromosome responsible for determining the sex of an individual.

sexually transmitted infection (STI, sexually transmitted disease, venereal disease) Any infection transmitted by sexual contact, including vaginal, oral and anal intercourse.

shigellosis (bacillary dysentery) An acute bowel infection caused by microorganisms of the *Shigella* genus.

shingles See *herpes zoster*.

shock A clinical syndrome characterised by a systemic imbalance between oxygen supply and demand. This imbalance results in a state of inadequate blood flow to the peripheral tissues, causing life-threatening cellular dysfunction, hypotension and oliguria.

sickle cell anaemia A hereditary, chronic haemolytic anaemia characterised by episodes of sickling, during which red blood cells become abnormally crescent shaped.

sickle cell crisis Severe episodes of fever and intense pain that are the hallmark of sickle cell anaemia.

sinusitis Inflammation of the mucous membranes of one or more of the sinuses.

Sjögren's syndrome An autoimmune disorder that causes inflammation and dysfunction of exocrine glands throughout the body.

skeletal traction Application of a pulling force through placement of pins into the bone.

skin graft Surgical method of detaching skin from a donor site and placing it in a recipient site, where it develops a new blood supply from the base of the wound.

skin tags Soft papules on a pedicle.

skin tear A traumatic wound occurring principally on the extremities of older adults, as a result of friction alone or shearing and friction forces that separate the epidermis from the dermis (partial-thickness wound), or which separate both the epidermis and the dermis from underlying structures (full-thickness wound).

skin traction Traction in which the cradle-like sleeve placed around the extremity exerts its pulling force through the person's skin.

sleep apnoea Absence of airflow through the upper airways for 10 or more seconds.

social determinants of health The social factors that influence the health status and health outcomes of individuals and communities.

social model of health A model of health which views health as multifaceted, focusing on social rather than biological determinants of health. This model emphasises health equity and prevention of illness or injury, as well as collaboration and empowerment. The social model of health is also referred to as new public health or the social ecological model.

solvents Solvents produce a depressant effect on the central nervous system and comprise a range of products producing vapours which, when inhaled through the nose or mouth, may cause an intoxicated feeling and lead to an altered state of consciousness.

somatic cell Any cell in the body that is not a sex cell (ova and sperm).

somatic pain Pain arising from nerve receptors originating in the skin or close to the surface of the body.

Somogyi phenomenon A morning rise in blood glucose to hyperglycaemic levels following an episode of nocturnal hypoglycaemia and a counter-regulatory hormone response.

spasticity Increased muscle tone in disease of the corticospinal motor tract.

speech The manner in which words are articulated.

spermatocoele A mobile, usually painless mass containing dead spermatozoa that forms in the epididymis.

spinal cord injury Injury to spinal cord, usually due to trauma and classified according to systems.

spinal cord tumours Benign or malignant, primary or metastatic tumour of the spinal cord.

spinal shock Temporary loss of reflex function below the level of injury.

splenomegaly Enlargement of the spleen.

sprain Tearing or stretching of a ligament that results from a twisting motion.

sprue A chronic primary disorder of the small intestine in which the absorption of nutrients, particularly fats, is impaired.

squamous cell cancer (SCC) A malignant tumour of the squamous epithelium of the skin or mucous membranes.

squamous cell carcinoma Malignant tumour of the squamous epithelium of the skin or mucous membranes.

standard A statement or criterion that can be used by a profession and by the general public to measure quality of practice.

starvation Inadequate dietary intake; the condition of being without food for long periods of time.

status asthmaticus Severe, prolonged asthma that does not respond to routine treatment. Without aggressive therapy, status asthmaticus can lead to respiratory failure with hypoxaemia, hypercapnia and acidosis.

status epilepticus Continuous seizure activity with only very short periods of calm occurring between intense and persistent seizures.

steatorrhoea Greasy, frothy, yellow stools resulting from excess fat in the faeces.

stem cells (haemocytoblasts) Bone marrow precursor cells for all blood cells.

stem cell transplant (SCT) Infusion of donor stem cells to replace the recipient's blood cell lines (white blood cells, red blood cells and platelets).

stenosis Condition where valve leaflets fuse together and are unable to open or close fully.

stigma Severe social disapproval.

stoma Surface opening.

stomatitis Inflammation of the oral mucosa.

straight traction A pulling force applied in a straight line to the injured body part resting on the bed.

strain Stretching or tearing of muscle fibres that results in bleeding into the tissues.

stress incontinence Loss of usually less than 50 mL of urine occurring with increased abdominal pressure.

stress-induced (erosive) gastritis Inflammation and superficial erosions of the gastric mucosa that may occur as a complication of other life-threatening conditions such as shock, severe trauma, major surgery, sepsis, burns or head injury.

striae A line above or below tissue that differs in colour and texture from surrounding tissue.

stridor High-pitched, harsh inspiratory sound indicative of upper airway obstruction.

stroke (brain attack, cerebrovascular accident, CVA) A condition in which neurological deficits occur as a result of decreased blood flow to a focal (localised) area of brain tissue.

stroke volume (SV) The amount of blood pumped into the aorta with each contraction of the left ventricle.

subacute thyroiditis Inflammation of the thyroid gland.

subdural haematoma A localised mass of blood that collects between the dura mater and the arachnoid mater.

subluxation Partial separation (or dislocation) of the bones of a joint.

substance dependence A severe condition occurring when the use of a chemical substance is no longer under an individual's control for at least 3 months. Continued use of the substance usually persists despite adverse effects on the person's physical condition, psychological health and interpersonal relationships (used interchangeably with 'addiction').

substance-related disorder Described as being maladaptive patterns of substance use leading to clinically significant impairment or distress.

substance use The use of any chemical in a fashion inconsistent with medical or culturally defined social norms despite physical, psychological or socially adverse effects.

sudden cardiac death (SCD) Unexpected death occurring within 1 hour of the onset of cardiovascular symptoms.

sundowning A behavioural change in Alzheimer's disease characterised by increased agitation, time disorientation and wandering during afternoon and evening hours.

superficial burn Burn involving only the epidermal layer of the skin; most often results from damage from sunburn, ultraviolet light, minor flash injury (from a sudden ignition or explosion) or mild radiation burn associated with cancer treatment.

surfactant A lipoprotein produced by the alveolar cells; interferes with adhesion of water molecules, reducing surface tension and helping to expand lungs.

surge capacity The healthcare system's ability to rapidly expand beyond normal services to meet the increased demand for qualified personnel, medical care and public health in the event of a large-scale disaster.

surgery An invasive medical procedure performed to diagnose or treat illness, injury or deformity. Although surgery is a medical treatment, the nurse assumes an active role in caring for the person before, during and after surgery.

surgical debridement The process of excising a wound to the level of fascia (fascial excision) or sequentially removing thin slices of a burn wound to the level of viable tissue (sequential excision).

surveillance Collecting and analysing data to establish a baseline and determine a point at which there is a change or trend in the health of the population.

syndrome of inappropriate ADH secretion (SIADH) Characterised by high levels of antidiuretic hormone (ADH) in the absence of serum hypo-osmolality, and most often caused by the ectopic production of ADH by malignant tumours.

synovitis Inflammation of the synovial membrane lining the articular capsule of a joint.

syphilis A sexually transmitted infection caused by a spirochete that may invade almost any body tissue or organ. It enters the body through a break in the skin or mucous membranes and can be transferred to the foetus through the placental circulation.

systemic lupus erythematosus (SLE) A chronic, inflammatory immune complex connective tissue disease.

systemic sclerosis (scleroderma) Hardening of the skin; a chronic disease characterised by the formation of excess fibrous connective tissue and diffuse fibrosis of the skin and internal organs.

systole A phase during which the ventricles contract and eject blood into the pulmonary and systemic circuits.

systolic blood pressure This arterial pressure wave produced by ventricular contraction (systole) averages 120 mmHg in healthy adults.

T lymphocytes (T cells) Type of lymphocyte that matures in the thymus gland.

tachycardia A heart rate exceeding 100 beats per minute.

tachypnoea Abnormally rapid respiratory rate.

telehealth A broad term that refers to the use of technology to contribute to the provision of healthcare, usually at a distance.

tendonitis Inflammation of a tendon.

tension headache Poorly localised headache characterised by ill-defined bilateral head aching, tightness, pressure or a vice-like feeling.

tension pneumothorax A condition in which an injury to the chest allows air to enter but not escape the pleural cavity.

terrorism An action, or threat of action, that causes harm or interference and is made with the intention of advancing a political, religious or ideological cause.

tertiary prevention Activities designed to restore individuals with disabilities to their optimal level of functioning.

test sensitivity How specifically a test identifies (positive test result) individuals who are affected and/or who have a disease phenotype.

test specificity How specifically a test does not identify (negative test result) individuals who are unaffected or do not have a disease phenotype.

testicular torsion Twisting of the testes and spermatic cord.

testosterone Male hormone produced in the testes.

tetanus Disorder of the nervous system caused by a neurotoxin elaborated by *Clostridium tetani*.

tetany Tonic muscular spasms.

tetraplegia (formerly called quadriplegia) Injury to cervical segments of the cord thus impairing function of the arms, trunk, legs and pelvic organs.

thalassaemia An inherited disorder of haemoglobin synthesis in which either the alpha or beta chains of the haemoglobin molecule are missing or defective.

therapeutic relationship A relationship that aims to empower the person with the knowledge and ability to recover from their illness.

third spacing The accumulation and sequestration of trapped extracellular fluid in an actual or potential body space as a result of disease or injury.

thoracentesis Invasive procedure in which fluid (or occasionally air) is removed from the pleural space with a needle.

thoracotomy Incision of the chest wall to gain access to the lung for surgery.

thought content The actual content of what a person is thinking.

thrill Palpable vibration over the precordium or an artery.

thromboangiitis obliterans (Buerger's disease) An occlusive vascular disease involving inflammation, spasm and clot formation in small- and medium-sized peripheral arteries.

thrombocytopenia A platelet count of less than 100 000 per millilitre of blood.

thromboembolus A thrombus that breaks loose from the arterial wall.

thrombophlebitis See *venous thrombosis*.

thrombotic cerebrovascular accident Cerebrovascular accident caused by occlusion of a vessel by a thrombus (a blood clot) on the interior wall of an artery.

thrombus A blood clot that adheres to a vessel wall.

thyroid gland A gland situated at the front of the throat which secretes thyroid hormones (thyroxine (T_4) and triiodothyronine (T_3)) to regulate the body's metabolic process.

thyroid storm or crisis An extreme state of hyperthyroidism that is rare today because of improved diagnosis and treatment methods. Also called thyroid crisis.

thyroidectomy A procedure performed to treat cancer of the thyroid.

thyroiditis Inflammation of the thyroid gland.

thyrotoxicosis See *hyperthyroidism*.

tidal volume (TV) The amount of air (approximately 500 mL) moved in and out of the lungs with each normal, quiet breath.

tinea capitis A fungal infection of the scalp.

tinea corporis A fungal infection of the body.

tinea pedis A fungal infection of the toenails and feet.

tinnitus Perception of sound such as ringing, buzzing or roaring in the ears.

titrate To determine the concentration of (a solution) by titration or perform the operation of titration.

titration Administration of analgesics in small increasing or decreasing increments.

tolerance A cumulative state in which a particular dose of a chemical elicits a smaller response than before. With increased tolerance, the individual needs higher and higher doses to obtain the desired effect.

tonic–clonic seizures Alternating contraction (tonic phase) and relaxation (clonic phase) of muscles during seizure activity.

tonsillitis Acute inflammation of the palatine tonsils.

tophi Small white nodules in subcutaneous tissue composed of urate deposits resulting from gout.

total colectomy Surgical removal of the entire colon.

total gastrectomy Removal of the entire stomach.

total hip arthroplasty (THA) Replacement of both the femoral head and the acetabulum.

total parenteral nutrition (TPN) Intravenous administration of carbohydrates (high concentrations of dextrose), protein (amino acids), electrolytes, vitamins, minerals and fat emulsions.

toxic epidermal necrolysis (TENS) Rare, life-threatening disease in which the epidermis peels off the dermis in sheets, leaving large areas of denuded skin.

toxic megacolon A condition characterised by acute motor paralysis and dilation of the colon.

toxic multinodular goitre A tumour characterised by small, discrete, independently functioning nodules in the thyroid gland tissue that secrete excessive amounts of thyroid hormone.

trachoma A chronic conjunctivitis caused by *Chlamydia trachomatis,* and a significant preventable cause of blindness worldwide.

traction The application of a straightening or pulling force to return or maintain the fractured bones in normal anatomic position.

transcutaneous electrical nerve stimulation (TENS) A unit that consists of a low-voltage transmitter connected by wires to electrodes that are placed by the person as directed by the physical therapist. The person experiences a gentle tapping or vibrating sensation over the electrodes. The person can adjust the voltage to achieve maximum pain relief.

transdermal Medication absorbed through the skin without injection.

transdisciplinary Transdisciplinary approaches in health involve partnerships and strategies that cross many health and other

professional discipline boundaries to create a holistic approach, addressing multiple influences.

transfusion An infusion of blood or blood components.

transient ischaemic attack (TIA) Brief period of localised cerebral ischaemia that causes neurological deficits lasting for less than 24 hours.

transjugular intrahepatic portosystemic shunt (TIPS) Used to relieve portal hypertension and its complications of oesophageal varices and ascites. A channel is created through the liver tissue using a needle inserted transcutaneously; an expandable metal stent is inserted into this channel, to allow blood to flow directly from the portal vein into the hepatic vein, bypassing the cirrhotic liver. The shunt relieves pressure in oesophageal varices and allows better control of fluid retention with diuretic therapy. Generally it is used as a short-term measure until a liver transplant is performed.

translocation The joining of a part of or a whole chromosome to another separate chromosome.

trauma An injury to human tissues and organs resulting from the transfer of energy from the environment.

traumatic brain injury (TBI) A traumatic insult to the brain capable of causing physical, intellectual, emotional, social and vocational changes.

tremors Rhythmic movement.

triage Means 'sorting'. Triage is a continuous process in which client priorities are reassigned as needed treatments, time and the condition of the clients change.

triage in rural, regional and remote areas Clinical determination of the acuity of the presenting health problems for people seeking healthcare in rural, regional and remote areas.

trichomoniasis A sexually transmitted disease (STD) caused by a parasite passed from person to person.

tricuspid valve A valve between the right atrium and ventricle of the heart; prevents blood from flowing backwards into the atrium.

trigeminal neuralgia (tic douloureux) A chronic disease of the trigeminal cranial nerve (cranial nerve V) that causes severe facial pain.

triglycerides Molecules of glycerol with fatty acids used to transport and store fats in body tissues.

trisomy Possessing three chromosomes instead of the usual two as in trisomy 21 or Down syndrome.

Trousseau's sign Contraction of the hand and fingers in response to occlusion of the blood supply by a blood pressure cuff; caused by decreased blood calcium levels.

tuberculosis (TB) Chronic, recurrent infectious disease caused by *Mycobacterium tuberculosis*; usually affects the lungs, although any organ can be affected.

tumour marker A protein molecule detectable in serum or other body fluids. This marker is used as a biochemical indicator of the presence of a malignancy.

Turner's syndrome A chromosomal abnormality in which all or part of one of the sex chromosomes is absent.

tympanoplasty Surgical reconstruction of the middle ear.

type 1 diabetes One of two types of diabetes characterised by the destruction of beta cells, usually leading to absolute insulin deficiency.

type 1 diabetes mellitus The result of pancreatic islet cell destruction and a total deficit of circulating insulin.

type 2 diabetes One of two types of diabetes the characteristics of which may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance. There is no immune destruction of beta cells.

type 2 diabetes mellitus Results from insulin resistance with a defect in compensatory insulin secretion.

ulcer A lesion of the skin or mucous membranes.

ulcerative colitis Chronic inflammatory bowel disorder of the mucosa and submucosa of the colon and rectum.

ultrafiltration Removal of excess body water using a hydrostatic pressure gradient.

unilateral neglect State in which a person is unaware of and inattentive to one side of their body.

upper body obesity (central obesity) Excess intra-abdominal fat characterised by a waist-to-hip ratio greater than 1 in men and 0.8 in women.

uraemia Literally, 'urine in the blood'; the syndrome or group of symptoms associated with end-stage renal failure.

urea An end product of protein metabolism and, along with water, the main constituent of urine.

ureteral stent Thin catheter inserted into the ureter to provide for urine flow and ureteral support.

ureteroplasty Surgical repair of a ureter.

urgency A sudden, compelling need to urinate.

urinary calculi Calculi or 'stones' in the urinary tract.

urinary diversion Procedure to provide for urine collection and drainage following cystectomy. The most common urinary diversion is the ileal conduit.

urinary drainage system The ureters, urinary bladder and urethra.

urinary incontinence Involuntary urination.

urinary retention Incomplete emptying of the bladder.

urolithiasis Development of stones within the urinary tract.

urticaria Hives.

vaccine Suspensions of whole or fractionated bacteria or viruses that have been treated to make them non-pathogenic.

Valsalva manoeuvre Closing the glottis and contracting the diaphragm and abdominal muscles to increase intra-abdominal pressure to facilitate expulsion of faeces.

valvular heart disease Interference of blood flow to, within and from the heart.

vancomycin intermediate-resistant *Staphylococcus aureus* (VISA) A form of *S. aureus* with intermediate resistance to vancomycin.

varicocoele Dilation of the pampiniform venous complex of the spermatic cord.

varicose veins Irregular, tortuous veins with incompetent valves.

vasectomy Sterilisation procedure in which a portion of the spermatic cord is removed.

vasoconstriction Smooth muscle contraction that narrows the vessel lumen.

vasodilation Smooth muscle relaxation that expands the vessel lumen.

vasogenic shock See *neurogenic shock*.

venous stasis Occurs when venous blood collects and stagnates in the lower leg.

venous thrombosis (thrombophlebitis) Blood clot (thrombus) formation on the wall of a vein, accompanied by inflammation of the vein wall and obstructed venous blood flow.

vertigo Sensation of whirling or rotation.

very-low-kilojoule diet (VLKD) A protein-sparing modified fast (1700 to 3500 kilojoules/day or less) under close medical supervision that may be used to treat significant obesity.

vesicoureteral reflux Condition in which urine moves from the bladder back towards the kidney.

visceral pain Pain arising from body organs. It is dull and poorly localised because of the low number of nociceptors.

vital capacity The sum of TV (tidal volume) + IRV (inspiratory reserve volume) + ERV (expiratory reserve volume); approximately 4500 mL in healthy people.

vitamin B_{12} deficiency anaemia Anaemia due to inadequate vitamin B_{12} consumption or impaired absorption.

vitiligo Abnormal loss of melanin in patches.

volatile acids Acids eliminated from the body as a gas.

Volkmann's contracture A common complication of elbow fractures; can result from unresolved compartment syndrome. Arterial blood flow decreases, leading to ischaemia, degeneration and contracture of the muscle.

vomiting The forceful expulsion of the contents of the upper gastrointestinal tract resulting from contraction of muscles in the gut and abdominal wall.

von Willebrand's disease The most common hereditary bleeding disorder, caused by a deficit of or defective von Willebrand factor.

warts (verrucae) Lesions of the skin caused by the human papillomavirus.

weaning Process of removing the person from ventilator support and re-establishing spontaneous, independent respirations.

Weber test A test of hearing; a vibrating tuning fork is placed on the midline of the top of the head and the person is asked to describe where the sound is heard. Normally, sound is heard equally in both ears.

wellness A state of wellbeing; engaging in attitudes and behaviours that enhance quality of life and maximise personal potential.

Wernicke's encephalopathy Caused by thiamine (B_1) deficiency and characterised by nystagmus, ptosis, ataxia, confusion, coma and possible death. Thiamine deficiency is common in chronic alcoholism.

wheeze Continuous, musical sound caused by narrowing of the lumen in a respiratory passage.

white blood cell (WBCs, leucocytes) The blood cells that contribute to the body's defence against microorganisms.

wild-type gene The most common type of gene; designated as normal.

withdrawal Cessation of use of a substance to which an individual has become addicted.

withdrawal symptoms Constellation of signs and symptoms that occurs in physically dependent individuals when they discontinue drug use.

xenograft A transplant from an animal species to a human.

xeroderma A chronic skin condition characterised by dry, rough skin.

xerosis Dry skin.

xerostomia Excessive dryness of the mucous membranes (due to chemotherapy or radiation).

X-linked dominant Any gene found on the X chromosome or traits determined by such genes; also refers to the specific mode of inheritance of such genes. One altered gene on an X chromosome in a male can produce disease, such as haemophilia.

X-linked recessive The result of an altered gene on the X chromosome.

Zollinger–Ellison syndrome Peptic ulcer disease caused by a gastrinoma or gastrin-secreting tumour of the pancreas, stomach or intestines.

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